Psychodermatology in Clinical Practice

Anthony Bewley
Peter Lepping
Ruth E. Taylor
Editors

Springer

Psychodermatology in Clinical Practice

Anthony Bewley • Peter Lepping
Ruth E. Taylor
Editors

Psychodermatology in Clinical Practice

Springer

Editors
Anthony Bewley
Royal London Hospital
Queen Mary University of London
London
UK

Ruth E. Taylor
Queen Mary University of London
London
UK

Peter Lepping
Wrexham Maelor Hospital Liaison Service
(BCULHB)
Wales, Wrexham
UK

Centre for Mental Health and Society,
Bangor University
Wales, Bangor
UK

Mysore Medical College and Research
Institute
Karnataka
India

ISBN 978-3-030-54306-8 ISBN 978-3-030-54307-5 (eBook)
https://doi.org/10.1007/978-3-030-54307-5

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Dedications

Dr. Bewley would like to dedicate this book to the memory of Rose Griffin, an extraordinarily selfless and loving individual, and to Sol.

Prof. Lepping would like to dedicate this book to his wife Ursula, thank you for everything. He would like to thank Prof. Vimal Sharma and Prof. Rob Poole for their kind mentorship over the years.

Dr. Ruth Taylor would like to dedicate this book to her husband Dr. Nicholas Moran for his unfailing support in all areas of life and for his love of life in all its eccentricities. Also, to her parents Alec and Elizabeth Taylor who fostered a love of life and learning, and to her children Hannah Austin and Mehetabel Moran who make everything worthwhile.

Preface to the Handbook of Psychodermatology

This volume is intended to be an easy hands-on guide to the basic management of patients with psychocutaneous disease. It is not meant to be a comprehensive volume discussing, in depth, the full literature in the diagnosis, management and pathoaetiology of psychodermatological disease. So, we have attempted to keep discussions and references to a minimum. And we have attempted to provide an accessible algorithmic approach to the diagnosis, assessment and basic management of psychodermatological conditions. Readers, we hope, can access the material in this book whilst seeing patients in clinic, and apply the algorithms to the management of very real patients. The book chapters are separated into principles of psychodermatology, individual illness and lastly concepts useful in clinics. We are aware that working with patients who have psychodermatological disease is not without its risks to the clinicians and healthcare professionals.

We have demonstrated that working in psychodermatology carries a very real risk of complaints, abuse, referrals to regulatory bodies and even death threats. Whilst working in these clinics is challenging, it makes such a big difference to the patients who will engage with healthcare professionals who take up this challenge. But it is very much a multidisciplinary team which works best in the psychodermatology clinics, and the interdependence of that team and the skills each member contributes is crucial. We would, then, like to thank all our colleagues who have helped develop this book. The authors of each chapter have provided really helpful and, where possible, evidence-based guidance for clinicians and healthcare professionals. And all the staff who have worked with each and every chapter author and editor, we believe, are to be celebrated and championed for their contribution to the management of potentially vulnerable patients with complex disease. We are grateful to all of our colleagues for all their advice and help in producing this book.

London, UK — Anthony Bewley

Acknowledgements

Thanks to all colleagues and authors.
Thanks to all patients who have helped review material in this book.
Thanks to Springer for publishing the book.

Contents

About the Authors

Alia Ahmed is a Consultant Dermatologist with a special interest in the psychological impact of skin disease. In addition to her NHS work, Dr. Ahmed is an honorary lecturer in Psychodermatology at the University of Hertfordshire. She is active in research and has presented internationally, as well as having several peer-reviewed publications and a research fellowship with the UK Dermatology Clinical Trials Network. Dr. Ahmed provides holistic care to her patients and strongly believes the interaction between the brain, skin and mind is key to achieving healthy skin.

Rukshana Ali (BSc (Hons), MSc, PGDip, DClinPsy) qualified with her doctorate from Royal Holloway, University of London, and holds a Postgraduate Diploma in Cognitive Behavioural Therapy. She currently works as the lead Clinical Psychologist in Paediatric Dermatology at St Thomas Hospital, St John's Institute of Dermatology, in London. She has worked across different clinical areas within the NHS and private practice and has contributed to several publications examining the role of psychology in long-term health conditions. She has a special interest in the impact of skin disease and psychological well-being in children.

Ilknur K. Altunay works as the head of the Department of Dermatology and Venereology Clinic, Psychodermatology Liaison Unit at the University of Health Sciences, Şişli Hamidiye Etfal Training and Research Hospital in Istanbul. She regularly sees patients with psychocutaneous disease with a clinical psychologist and psychiatrists for diagnosis and therapy and currently is a member of the executive committee of ESDAP (European Association of Dermatology and Psychiatry). She is also interested in clinical dermatology and wound care.

Janet Angus is currently a Consultant Dermatologist at Bristol Royal Infirmary. She qualified in 1991 in Manchester and was previously a Consultant Dermatologist in Nottingham. Her specialist interest is psychodermatology, and she runs a psychodermatology service for the South West.

Iyas Assalman works as a Consultant General Adult Psychiatrist in London, for East London Foundation Trust. He is an Honorary Clinical Senior Lecturer with the Centre for Psychiatry, Wolfson Institute of Preventative Medicine, Queen Mary University of London in the UK.

Benjamin Baig is a Consultant Liaison Child Psychiatrist at South London and Maudsley NHS Foundation Trust and a Senior Lecturer at the Institute of Psychiatry, King's College London. He holds CCTs in both general adult and child and adolescent psychiatry. He has completed an MPhil in Schizophrenia Genetics and PhD in Depression in Inflammatory Bowel Disease and is a Fellow of the Higher Education Academy. He has held academic positions at the University of Edinburgh and the University of Malawi and is Deputy Director of International Affairs at the Royal College of Psychiatrists.

Richard Barlow MRCP is a specialist registrar (trainee) in dermatology in the West Midlands, UK.

Susannah Baron is a Consultant Dermatologist working at the St John's Institute of Dermatologists. She specialises in paediatric dermatology and has established a national multidisciplinary paediatric psychodermatology service at St Thomas' Hospital. Susannah's other field of expertise is in the management of severe eczema in children, focusing on the psycho-social impact on children and families, and is involved in population-based research and clinical trials. Susannah is Treasurer of the British Society of Paediatric Dermatology and co-chair of Psychodermatology UK, whose aim is to raise awareness of the psychosocial impact of skin disease and promote research and multidisciplinary management.

Anthony Bewley is a Consultant Dermatologist at Barts Health NHS Trust and Honorary Professor at Queen Mary University London. He has a special interest in psychodermatology and has published extensively in international peer-reviewed journals on a range of different areas within dermatology. He is the co-editor of *Practical Psychodermatology* (Wiley, 2014) and associate editor of *Rooks Textbook of Dermatology* (Wiley 2016); he was chair of Psychodermatology UK (2008–2020) and is Secretary and President Elect of the European Society for Dermatology and Psychiatry (ESDaP; www.psychodermatology.net). He has established training schools for healthcare professionals in psychodermatology.

Elaine N. Clarke is a PhD candidate in the Department of Psychology at the University of Sheffield. She has experience of providing CBT-based interventions to adults with depression and anxiety disorders and is a qualified supervisor for low-intensity therapists. She has interests in the psychosocial aspects of long-term health conditions, mental health and the development of psychological interventions.

Dimitre Dimitrov works as a Dermatologist for the Ministry of Presidential Affairs in the UAE. He is an Honorary Consultant Dermatologist at the Dermatology department, Sheikh Khalifa Medical City, Abu Dhabi, UAE. Psycho-dermatology is his main interest: recently, he conducted research into the stigmatisation experience among patients with psoriasis.

Dr. Dimitrov is registered with the GMC and also practises as a Locum Consultant Dermatologist in the UK.

Christina George is a consultant dermatologist working at the Imperial College Healthcare NHS Trust in London. She has a special interest in psoriasis and the psychosocial impact of this condition on her patients.

Maria-Angeliki Gkini is a Consultant Dermatologist at Barts Health NHS Trust, London, UK, and Consultant Dermatologist and Venereologist in Athens, Greece. She has a special interest in hair disorders and psychodermatology.

Jon Goulding is a consultant dermatologist at University Hospitals Birmingham NHS Foundation Trust, working at Solihull and Good Hope Hospitals. Undergraduate training was undertaken at Edinburgh and Oxford Universities, with higher specialist training in London and the West Midlands.

Dr. Goulding has established a fully integrated, multidisciplinary psychodermatology service at Solihull Hospital, working together with colleagues in clinical psychology and liaison psychiatry. He has written widely in the peer-reviewed medical literature and is actively engaged in research and teaching in this field. He sits on the executive committee for Psychodermatology UK and is a member of the European Society for Dermatology and Psychiatry.

Victoria Joliffe is a Consultant Dermatologist with a special clinical interest in hair disorders. She was Educational Lead for the British Hair and Nail Society and lectures internationally about hair disorders.

Ahmed Kazmi MCBhB, MRCGP, MRCGP, FRACGP, DRCOG, DFSRH, DCH, DGM, Dip Clin Derm, is a GP and dermatology specialty doctor based in London. He trained at the University of Birmingham and undertook his junior doctor training at Imperial Deanery London and his GP training in Warwickshire. Dermatology is currently his main focus and he is working towards becoming a consultant. His experiences of holistic care and managing mental health in primary care have been invaluable in his psychodermatology practice. His main special interests are medical education and psychodermatology. He regularly lectures and teaches on general and psychodermatology subjects, and when he is not in the dermatology outpatient department he can be found performing stand-up comedy in Australia.

Jonathan Kentley is a dermatology specialist registrar at Chelsea and Westminster Hospital. He has a particular interest in psychocutaneous medicine and has previously worked as a clinical research fellow in the specialist psychodermatology clinic at the Royal London Hospital.

Alex Laird is a medical herbalist running the UK's only herbal medicine dermatology clinic at Whipps Cross University Hospital and a clinic and food medicine workshops at Breast Cancer Haven, London. She has published research and is a university visiting lecturer. Alex is the author of *Root to Stem*—a seasonal guide to natural recipes and remedies for everyday life (Penguin 2019). She founded the charity Living Medicine (www.livingmedicine.org) that teaches people how to use foods and herbs in healthcare.

Peter Lepping works as a Liaison Psychiatrist in Wrexham, Wales, for Betsi Cadwaladr University Health Board. He is an Honorary Professor with Bangor University in Wales (Centre for Mental Health and Society) and Mysore Medical College and Research Institute in India. He is an international expert on delusional infestation and runs a specialist clinic with the School of Tropical Medicine in Liverpool.

Tabi Leslie is a general adult and paediatric Consultant Dermatologist at the Royal Free Hospital, London, where she runs a specialist urticaria service. Her academic and clinical interests include urticaria, angioedema, mastocytosis, mast cell disorders and itch. Dr. Leslie is the Honorary Secretary at the British Association of Dermatologists (BAD). She sits on the Council of the Royal Society of Medicine (RSM) Dermatology Section and is President of the RSM Clinical Immunology and Allergy (CIA) section. Dr. Leslie has been an elected Dermatology Board member for the European Academy of Allergy and Clinical Immunology (EAACI) and is Secretary of the EAACI Chronic Urticaria in Children (CU-Kids) Task Force. Dr. Leslie is involved in the European and British guidelines for pruritus and for urticaria.

Anna V. Michenko is a Consultant Dermatologist in the Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia.

Padma Mohandas qualified from the University of Mysore in India and pursued further medical training in the UK initially in medicine and general practice. After completing a master's in clinical Dermatology in 2015 with distinction from King's College London she decided to pursue a career in dermatology and completed specialist dermatology training in the East Midlands at Nottingham University Hospital and the University Royal Derby and Burton Hospitals. She was appointed as Consultant Dermatologist at Bart's Health London in 2020 and has a special interest in psychodermatology running a dedicated complex needs clinic at the Royal London Hospital alongside the Dr. Bewley, Taylor and Ahmed.

Dmitry V. Romanov is a professor of the Department of Psychiatry and Psychosomatics, Sechenov First Moscow Medical University (Sechenov University), and is a leading research associate of the Department of Boundary Mental Conditions and Psychosomatic Disorders, Mental Health Research Center, Moscow, Russia. He also works as a consulting psychiatrist in Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology where he deals with patients suffering from psychodermatological conditions as a member of a joint research group. He is a well-known expert in psychodermatology in Russia.

Reena Shah (BSc (Hons), MSc, DClin Psych, CPsychol) is a Chartered Senior Clinical Psychologist who works for the Central and North West London Trust. She specialises in treating dermatology patients. Privately she is a Consultant Clinical Psychologist at the Dermatology Clinic, Harley Street. Dr. Shah is on the advisory board for the BAD psychodermatology group, is a member of the All-Party Parliamentary Group on Skin and is on the executive committee for Psychodermatology UK and in the NICE Guidelines for Acne Vulgaris and BAD Clinical Guideline Developmental group for vitiligo and delusional infestation. Dr. Shah is a lecturer at the University of Hertfordshire on the MSc Dermatology programme, and she teaches on the postgraduate Diploma for Clinical Dermatology and the annual SpR training on Psychodermatology.

Kirsty E. Smith works as a postgraduate Clinical Teaching Fellow at Wexham Park Hospital, England. She graduated in 2017 from Oxford University and completed her foundation training programme in South West England. She has been enjoying pursuing an interest in dermatology under the supervision of Dr. Alia Ahmed.

Tanyo Tanev is a cognitive-behavioural psychotherapist and hypnotherapist in Stara Zagora, Bulgaria. He has an interest in eclectic psychotherapy, particularly the integration of mindfulness, meditation, and stress-reduction techniques. He studies the relation of adult attachment in psychodermatology patients and their diagnosis.

Ruth E. Taylor trained in psychiatry in Manchester and then as a senior trainee at the Maudsley and the Institute of Psychiatry where she completed her PhD. Dr. Taylor is a Senior Lecturer in Liaison Psychiatry at Barts and the London Medical School, Queen Mary University. She runs a joint psychodermatology clinic and a liaison psychiatry clinic at the Royal London and has both a clinical and research interest in psychodermatology and in somatisation and medically unexplained symptom syndromes. Dr. Taylor has published widely on psychodermatology in international peer-reviewed journals. She is the co-editor of *Practical Psychodermatology* (Wiley, 2014) and authored the psychiatric assessment chapter in *Rooks Textbook of Dermatology* (Wiley 2016). She was co-chair of Psychodermatology UK (2008–20) and is now a member of the executive committee of Psychodermatology UK. Dr. Taylor Lectures on the Mind and Skin Master's degree at the University of Hertfordshire, and teaches on UK psychodermatology training courses for dermatologists.

Andrew R. Thompson is the Programme Director on the South Wales Clinical Psychology Doctorate based at Cardiff University. He is a Consultant Clinical Psychologist and Honorary Professor of Clinical Psychology. He has a long-standing research interest in psychodermatology and more broadly in understanding the psychosocial issues associated with conditions that affect appearance. Over the years, he has provided NHS clinical psychology services in adult mental health and clinical health psychology and has led a novel NHS psychodermatology service.

Part I

Principles

Psychodermatology History and Examination

1

Ruth E. Taylor

A full psychodermatological assessment requires both an assessment of the skin condition and of any mental health disorder. The most common situation will be where the patient is being seen by a dermatologist with an interest and some expertise in psychodermatology but who is not a psychiatrist. This chapter will therefore focus on advice tailored to that situation.

Initial Assessment

General Considerations in Conducting a Psychodermatological Assessment

The initial assessment is crucial as it is the best opportunity to engage the patient with an approach that embraces their psychosocial well-being as well as their physical health. However, it must be borne in mind that the vast majority of patients who attend a dermatologist will be expecting an assessment of their skin, not their mental health. There are various ways in which mental and physical disorders may interact in skin clinic presentations (Box 1.1). It is useful to consider into which of the categories the patient falls in tailoring one's approach to the patient. Those with primary psychiatric disorders presenting via the skin may or may not have insight into their illness, and where insight is lacking engagement with a psychosocial agenda can be more challenging. Advice on the initial engagement of the patient with a psychosocial agenda is given in the *Approaches to patient* chapter. A thorough

R. E. Taylor (✉)
Centre for Psychiatry, Wolfson Institute of Preventive Medicine, Barts & The London School of Medicine and Dentistry, Queen Mary University of London, London, UK
e-mail: r.e.taylor@qmul.ac.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_1

Box 1.1 Relationship of Physical and Mental Health Disorder in Patients Presenting in Psychodermatology

Primary dermatological disorders exacerbated by stress	Secondary psychiatric disorder caused by primary skin condition	Primary psychiatric disorder manifesting via skin
• Psoriasis • Eczema • Alopecia • Acne rosacea • Urticaria	• Depression • Anxiety • Body image disorder • Social anxiety • Hypochondriacal anxiety (illness anxiety)	• Delusional infestation • Body dysmorphic disorder • Neurotic excoriation • Dermatitis artefacta • OCD spectrum disorders, e.g. trichotillomania • Somatization Disorders

history and physical examination is the starting point of any initial assessment, as it would be in any new dermatology patient.
The main aspects of a successful assessment can be summarised as follows:

- Make the patient aware that you are interested in treating them holistically.
- Ensure the clinic room is suitable in terms of privacy and safety. There should be an unobstructed exit for the clinician. There should preferably be an alarm button to raise assistance if needed. The usual coming and going of other staff common in dermatology clinics needs to be minimised so that patients can discuss issues which they may feel to be embarrassing or stigmatising.
- Inform the patient that there is a close relationship between the skin and the mind. Mental ill health can impact on the skin and skin disorders can have a big impact on mental health.
- Emphasise that you want to assess their skin disorder AND understand the effect that it is having on their emotional and social well-being.
- Inform patients the information shared is confidential, but some details may be shared with other health care professionals, e.g. letters to GP.
- Avoid sharing unnecessary details. Make sure any information in a letter has been shared with the patient (with the exception of illnesses where full disclosure of the diagnosis is better done gradually). It is good practice to copy all letters to patients to reinforce information and messages given in the consultation. (See *Approaches to patient* for more detail on conversations about letters and how to write them).
- Book longer appointment times (45 min new patients, half an hour follow-up), and make the patient aware you have extra time to complete a comprehensive assessment.
- If no additional time is available when you encounter the patient make them aware you can book further appointments in order to complete the assessment.
- At the first visit, make sure you have thoroughly heard the patient's story. If the patient feels listened to and that you have understood their skin condition, they are much more likely to engage.
- Allow patients to ventilate frustration and anger at their condition and care that has been offered previously.

- Do not be dismissive, however bizarre and unusual the patient's symptoms and signs might be.
- See chapter on *Approaches to patient* for general guidance to aid the engagement of patients with psychosocial assessment.

Psychodermatological Assessment

Psychodermatological assessment will cover a medical history and a psychiatric history (some overlap in these). See Box 1.2 for contents of full psychocutaneous history and Box 1.3 for the mental state examination. In practice, it will usually not be possible to take a full psychiatric and medical history at the first visit and priority areas are outlined below. Other relevant areas of psychiatric history can be covered subsequently or when the patient is seen by a psychiatrist. Please see Chap. 2 on Approaches to patient for more detail on assessing particular mental health presentations, e.g. risk assessment, depression, psychosis, delusional disorder, etc.

Box 1.2 Psychocutaneous History

Presenting Complaint: identify what the patient feels is the main problem—this may differ from the view of the referrer.

History of Presenting Complaint: when was onset? Establish any triggers or life events around the time of onset, current triggers to symptoms, any previous episodes, related disorders in close others?

Nature of Symptoms: look for unusual sensations: burning, crawling, stinging, electric shocks.

Note Distribution of Skin Symptoms: dermatomal, non-organic patterns, patterns indicating self-infliction, e.g. in accessible areas or worse where dominant hand can reach, etc.

Review the Presence of Any Psychiatric Symptoms: mood, anxiety, obsessional, psychotic. See Chap. 2 'Approaches to patient' for detailed suggestions of how to enquire about and assess these symptoms.

Previous Medical History: Psychiatric and Physical

Note any episodes of mental illness needing treatment in primary care, secondary care, involvement of community mental health team, psychology services, any admissions to mental health bed voluntary or involuntary.

History of Previous or Current Substance Misuse: Enquire about substance misuse, ask about dependence symptoms, use screening tool—see below, consider urine screen—see below.

Current Medication:

- Previous and current skin treatments.
- Current and previous psychotropic medications including depot antipsychotics and St John's Wort.
- Note any medications that can affect mental state (prescribed or non-prescribed, e.g. bought on the internet).

- Opiate or amphetamine/stimulant use may be linked to delusional infestation.
- Dopaminergic medication, e.g. in treatment of Parkinson's, can be linked to delusional infestation.
- Medications with strong anticholinergic property, e.g. amitriptyline, can cause visual hallucinations.
- Steroids can cause mood change, depression, mania or other psychosis.
- Use of any mood stabilisers lithium or anticonvulsants suggesting bipolar illness. Need for caution in giving antidepressants in such patients—requires a specialist referral.

Family history of both physical and mental health problems. NB history of severe OCD in BDD patients. Note if family history of bipolar illness: caution using antidepressants, would require a specialist referral, would not be appropriate for a dermatologist to start antidepressant in patient at risk of manic episode. NB any family history of suicide which is a risk factor for suicide.

Personal History: Childhood, Schooling, Occupation, Relationship/marital history, reproductive history in women

Children: even if short of time this must be covered in order to consider if there are child safeguarding issues.

Present Social Circumstances and Social Support: Must always be covered at first visit even if short of time as would be part of risk assessment.

Premorbid personality: can be very helpful to understand if current symptoms are part of a new onset of illness or part of long-term personality traits. Patients with emotionally unstable personality disorder (EUPD) may be at particular risk of self-harm, or dermatitis artefacta. See chapter on personality disorder.

Box 1.3 The Mental State Examination

Appearance and Behaviour

Speech

Mood: subjective and objective

Thought: Form and Content (including delusions)

Perception (e.g. tactile, olfactory, auditory and visual hallucinations)

Cognitive Assessment: including orientation, attention and concentration, registration and short-term memory, recent memory, remote memory, intelligence, abstraction

Suicidal and homicidal ideation, intent or plans (risk) (see approaches to patient chapter)

Insight—this is very important to assess as it determines the approach to take in engaging a patient with a psychosocial agenda.

The reader is referred to an undergraduate psychiatry text for detailed information on how to do a basic mental state assessment.

Please see Chap. 2 Approaches to patients for detailed information on how to assess psychiatric symptoms: mood, anxiety, obsessional, psychosis and risk assessment.

Time may be limited and it may be impossible to cover the full psychocutaneous history at the initial assessment, and for some areas, e.g. personal history, it is reasonable to leave it to subsequent follow-up visits. However, there are certain areas which MUST be covered at the initial visit as follows:

Medical History Priority Areas at Initial Assessment

- As with any new patient take a full Medical History both skin-related and of other medical conditions.
- Ensure that the patient feels that all their concerns are addressed.
- Assess patients' health beliefs. What do they think is wrong and what has caused it? Physical/psychological cause?
- Address any fears or anxieties, e.g. re cancer or specific diseases.
- Ask about family history (skin and other disorders), and illness in close others. This is important for understanding heritable disorders and what may have triggered illness anxiety in patients.
- Ask about previous experience with health care providers, e.g. patients may have been ridiculed in the past or falsely reassured and a serious disorder was missed. Such experiences will undermine the ability to trust health professionals.
- Take a detailed medication history including non-prescribed medication bought in health food shops (e.g. St. John's wort), or over the internet. Note medication affecting mental state—see Box 1.2. Note any psychotropic medication and ask why they are being taken. For instance, patients taking lithium are likely to have bipolar illness. Sodium valproate lamotrigine and carbamazepine could either be taken as an anticonvulsant or mood stabiliser. Pregabalin is used as an anticonvulsant, for neuropathic pain and in chronic anxiety.

Psychiatric History Priority Areas at Initial Assessment

- A risk history is important: Enquire about mood and biological symptoms (sleep, weight, appetite). Where there is low mood move to enquiry about suicidal feelings and behaviour (ideation, intent, plans). Also consider the risk to children in the care of the patient or any other risk to others, e.g. health care professionals, especially where the patient has delusional beliefs.
- Assess the impact of mood or other psychiatric symptoms on function in everyday activities: work, child care, household chores, etc.

- Ask about any history of mental disorder and use different words to ensure patients realise what sort of events you want to know about, e.g. depression, nerves, breakdowns, stress, difficult losses, psychosis, memory problems.
- Ask specifically about contact with mental health services, including community mental health teams, psychology, counselling services, primary care services, memory clinics, etc. Do they have a key worker or support worker? These support networks can be helpful in helping the patient deal with their skin condition.
- Always take a detailed history of the use of alcohol, recreational drugs and non-prescription medication, including looking for signs of dependence. The CAGE is a useful screen for alcohol dependence (see Box 1.8 below).
- Take a detailed family history of psychiatric disorder. It is especially important to be aware of a strong family history of bipolar illness. (If present it requires caution in using antidepressants to treat depression, needs close monitoring, and will need psychiatric referral.) NB in risk assessment: family history of suicide.
- Personality disorders are persistent inflexible traits in mood, behaviour, and ways of relating to others which begin in adolescence or early adulthood and result in disruption of the ability to function in work and social relationships. It is not likely to be possible to do a full assessment at the initial visit but it may be worth asking patients who have had contact with psychiatric services what diagnoses they have been given and taking note if the patient has been told they have a personality disorder. (See chapter on personality disorder.)

Physical Examination and Investigation Is Crucial at the Initial Assessment: See Box 1.4 for Suggested Investigations

- Examine the skin comprehensively and perform a relevant physical examination. This helps the patient to feel that their skin disorder is being understood, evaluated thoroughly and taken seriously. It also means that organic skin or systemic disorders will not be missed. Patients with delusional infestation will often have previously had the experience of their skin problem being dismissed without thorough evaluation. For this reason, physical disorders can be missed in psychodermatology patients. Physical examination can also be part of fulfilling a patient's need for care, and help build a trusting rapport.
- Examine carefully for signs indicative of psychiatric disorder: self-neglect/poor hygiene, factitious lesions, picking or plucking of hair or skin indicating OCD spectrum disorders, poor dentition—(can indicate drug use), needle tracks. See Box 1.5.
- Look for evidence of self-treatment: steroid atrophy or dermatitis from irritant agents.
- It may be appropriate to use a dermatoscope to look at affected areas.
- A skin biopsy may be indicated. Time constraints may mean this has to be deferred to a second visit.

Box 1.4 Baseline Investigations at Initial Visit (See Also Individual Disorder Chapter for Specific Tests for Particular Disorders)

Blood Tests: Full blood count and haematinics and iron, B12 and folate, urea and electrolytes, glucose, liver function tests, renal function, thyroid function tests, c reactive protein and erythrocyte sedimentation rate, human immunodeficiency virus, syphilis serology.

Urine drug screen looking for opiates, amphetamines and recreational drugs.

Send off any specimens brought by patient for microscopy and culture.

Consider skin scrapings.

Consider skin biopsy.

Consider MRI brain—see text.

Consider EEG—see text.

Consider cognitive assessment, e.g. mini-mental state, ACE-R.

Box 1.5 Physical Findings During Examination Indicating Psychocutaneous Disorder

Poor self-care and dishevelled appearance may indicate depression/psychosis/cognitive decline.

Dermatitis neglecta may indicate poor self-care.

Lice or scabies tracks indicating poor self-care.

Linear tears, unusual shaped erosions or burns may indicate factitious disorder or NB in children be aware of the possibility of physical abuse.

Stretch marks/skin atrophy from steroid overuse.

Bitten nails–OCD tendencies.

Nail or hair dystrophy indicating nutritional deficit.

Areas of excoriation, scarring, erosions, non-healing ulcers, secondary infection of wounds may indicate skin picking disorder or delusional infestation or dermatitis artefacta.

Wearing of unusual clothing, e.g. scarf over face, large hat drawn down or very heavy make-up may indicate body image problems which could be part of body dysmorphic disorder.

- Sometimes patients may demand a skin biopsy but the dermatologist may not feel this is indicated. In this situation, it may be appropriate to take a skin scraping. Skin scrapings can be helpful in delusional infestation if taken from an area where the patient feels particular ‘parasite activity’.
- Take any specimens the patient offers, examine them visually and, if possible, with a dermatoscope, and send them for laboratory examination. Carefully examine any videos, photos or literature the patient may bring. It is crucial the

patient feels understood and taken seriously. Provide patients with pots in which to bring further specimens if they wish, though discourage patients from actively excoriating or picking at their skin to produce specimens.

- Demonstrating an open mind and willingness to look at any evidence in the form of specimens or photographs will help build trust and rapport and aid engagement.
- Perform relevant baseline blood tests. There can be many underlying organic disorders that may be producing the physical symptoms. The tests used should be guided by history and examination findings (see individual disorder chapters for suggestions of relevant investigations). Raised eosinophil counts may indicate a systemic infestation, whilst raised CRP and white cell counts may indicate an acute infectious process. See box 1.4 for suggested baseline investigations and the relevant disorder chapters.
- A urine sample for drug screening is invaluable. The patient can be openly asked to provide a sample, explaining that this is routine practice. The use of non-prescribed drugs is often discovered this way and can open a channel to discuss what is being used, how this may be relevant, and offer appropriate services to address this should the patient wish to do so.
- Consider the need for cognitive assessment. Delusional infestation is sometimes seen in early dementia. This has to be handled sensitively. It is usually possible during the conversation to elicit whether a patient is fully orientated and has any obvious memory problems. Ask patients if they have any such problems. Where indicated a cognitive screening tool such as the MMSE or equivalent should be employed. This may be best done at subsequent visits when the patient is more engaged, and it is usually best to present this as a routine screen performed on all patients.
- Consider the need for a brain MRI. This may not be something to discuss at the first appointment but if it is indicated discuss and offer it at follow-up. Where patients have very bizarre symptoms or delusions it is important. Temporal lobe epilepsy can produce unusual sensory symptoms, and brain lesions can be primary causes of the delusional infestation. An EEG may be indicated.
- A neurological examination may be indicated. Particularly look for any signs of Parkinsonian symptoms on a patient where you may be considering using an antipsychotic as such medication can exacerbate these symptoms. It is important to check for this at follow-up where patients (especially elderly patients) have been started on antipsychotics.

Use of Questionnaires in Psychocutaneous Clinics

- Advantages—used prior to seeing the patient it may enable the patient to divulge information about their mental state without feeling stigmatised or threatened. The clinician can then pick up on this information. It can enable the clinician to focus psychological assessments on high-risk patients. Some specific screens can help a dermatologist pick up psychiatric disorders, e.g. CAGE questionnaires to screen for alcohol, MMSE for cognitive impairment, HADS for depression and anxiety.

- Disadvantages—They are merely screening tools and do not give a diagnosis. It may reveal more psychosocial morbidity than the clinician can deal with in the time available. Clinicians should do basic psychosocial assessment in all patients, not just those scoring high on screens. Some screening tools are easy to manipulate by patients.
  See box 1.6 for useful and widely used questionnaires to assess mental health.

Box 1.6 Useful and Widely Used Questionnaires

- Anxiety and Depression Scale (HADS). (Zigmond, AS; Snaith, RP (1983). 'The hospital anxiety and depression scale'. *Acta Psychiatrica Scandinavica* 67 (6): 361–370.)
  Fourteen items questionnaire (seven items for depression, seven for anxiety). Cut-off score of 8/21 for a potential clinical case of either anxiety or depression)
  Can indicate whether the patient is potential case and whether the main issue is low mood or anxiety or a mixture.
- Patient Health Questionnaire for depression (PHQ2) (Box 1.7) Arrol B et al. Validation of the PHQ-2 and PHQ-9 to screen for major depression in the primary care population. Ann Fam Med 2010;8(4):348–353.
- A widely used screen for cognitive impairment is the Mini-Mental State Examination (MMSE) (Folstein MF, Folstein SE, McHugh PR (1975). "Mini-mental state'. A practical method for grading the cognitive state of patients for the clinician'. *Journal of Psychiatric Research* 12 (3): 189–98.
- Addenbrookes Cognitive Examination Revised ACE-R another cognitive assessment tool. E. Mioshi et al Int J Geriatr Psychiatry 2006.
- The CAGE questionnaire to screen for alcohol dependence. Box 1.8.

Box 1.7 Patient Health Questionnaire for Depression PHQ 2

Over the past 2 weeks how often have you been bothered by any of the following problems?

1. Little interest or pleasure in doing things.
2. Feeling down depressed or hopeless.

0 = Not at all, 1 = several days, 2 = More than half the days, 3 = Nearly every day

A score of 2 or more has a 86% sensitivity and a 78% specificity for depression.

Box 1.8 The CAGE Questionnaire

CAGE is an acronym that makes the four questions easy to remember. Each letter represents a specific question:

1. Have you ever felt you should *cut* down on your drinking?
2. Have people *annoyed* you by criticising your drinking?
3. Have you ever felt bad or *guilty* about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (*eye-opener*)?

Answers Yes = 1 No = 0 Score 2–3 indicates heavy alcohol use or dependence.

Practice Points

- At the initial visit address patient expectations and ensure the patient understands that they will be treated holistically, that there are important links between mind and skin, and that they will receive a psychosocial as well as physical assessment.
- Ensure there is sufficient time and an appropriate safe, private setting in which to allow adequate psychosocial assessment.
- The psychocutaneous specialist should be trained in psychological as well as physical assessment.
- Ensure the patient's whole story is heard at the initial visit, that their experience and symptoms are validated and taken seriously.
- It is vital that there is a thorough initial physical work up so that no organic disorder is missed, and the patient has confidence in their treatment plan.
- Engagement of the patient is crucial and this is achieved through a true bio-psychosocial approach, which is informed, expert, honest, non-judgemental and empathic.
- Simultaneous treatment of the skin and mental state is vital for engagement and successful management of the psychocutaneous disorder.
- A true multidisciplinary team and close liaison between primary and secondary care are necessary for the successful management of the psychocutaneous disorder.

Approaches to Patients

2

Ruth E. Taylor

This chapter will discuss some general considerations and advice on carrying out mental health assessments embedded in dermatology settings. The approach will be different depending on the service model being employed. The following models are commonly employed in the UK and elsewhere in Europe (Boxes 2.1, 2.2, 2.3, and 2.4):

Box 2.1 Joint Psychodermatology Clinics (Currently Still Rare Worldwide)
All patients both new and follow-up are seen jointly by both a psychiatrist and dermatologist at the same time. Typically, 45–60 min for new patients and 30 min for follow-up.

Benefits: Enables delivery of truly holistic care.
- Very acceptable to the vast majority of patients.
- Reduces stigma of being singled out to be 'sent' to see psychiatrist.
- Increases engagement of patient with mental health assessment and treatment.
- Enables true joint management.

Drawbacks: More expensive as it needs two specialists in the clinic, though there is evidence that it can be cost-effective.
- Can be difficult to fund/deliver with administrative boundaries between physical and mental health care providers.
- Need to carefully consider clinical governance and record keeping issues between physical and mental health care providers.

R. E. Taylor (✉)
Centre for Psychiatry, Wolfson Institute of Preventive Medicine, Barts & The London School of Medicine and Dentistry, Queen Mary University of London, London, UK
e-mail: r.e.taylor@qmul.ac.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_2

Box 2.2 Dermatologist with special interest and training in psychodermatology runs specialist psychodermatology clinic

Dermatologist runs specialised psychodermatology clinic with longer new patient and follow-up slots. Typically, 45–60 min for new patients and 30 min for follow-up. Refers to separate liaison psychiatry and psychology sessions that can be run within the same department, either at same time or at another time

Benefits: Easy to engage patients, and patients who would not go to see a psychiatrist can undergo a psychological assessment.

Holistic management of skin and mental health problems treated together.

Drawbacks: Time consuming therefore expensive in specialists' time.

Requires dermatologists with appropriate training and expertise.

No immediate access to psychiatric advice, unless this can be negotiated with liaison psychiatry colleagues. Some barrier to patients seeing mental health professionals as they have to attend a separate appointment, and they may decline to do so.

Box 2.3 Dermatologist does brief screening/psychosocial exploration as part of usual dermatology clinic, then refers patient to liaison psychiatry and/or psychology sessions run within same department

Benefits: Less costly, integrated within usual clinic.

There is a dedicated mental health service for dermatology patients with appropriate expertise.

Enables good liaison communication both ways between dermatologists and mental health specialists.

Drawbacks: Insufficient time to conduct an adequate psychiatric assessment.

Patients may fail to attend further mental health appointment after need for assessment /treatment is identified due to time/logistics/stigma.

Likely to have high rates of non-attendance in mental health sessions.

Dermatologist is not involved in further assessment /treatment, so loses the opportunity to gain skills in this area.

Box 2.4 Dermatologist does brief screening/psychosocial exploration as part of usual dermatology clinic, then refers patient to generic liaison psychiatry services and/or psychology service. This is the standard process in most places
Benefits: None.
Drawbacks: As in Box 2.3.

Introductions and Scene Setting for the Patient

Whatever model is employed, it is important that patients are properly introduced to the professionals who are seeing them, whether this is a dermatologist, a dermatologist with a special interest in psychodermatology, a psychiatrist, a psychologist or a clinical nurse specialist. The patient should understand who they are seeing, what that person's role and area of expertise are, and what the aim of the consultation is to be. It is also helpful for patients to be told in advance how long the consultation will be. Patients will engage much more easily in both a physical and mental health assessment if they are clear what the aim of the assessment is, and that there will be sufficient time to address both their physical and mental health concerns.

Opening Up a Psychosocial Agenda in a Dermatology Consultation

Consultations in dermatology are frequently brief with skin and physical health-focused history and examination. There are usually a lot of people around and in and out of the clinic room. If the clinician plans to open up the agenda to explore mental health issues it is necessary to make some adjustments. It is important to remember that patients will have certain expectations of what their dermatologist will discuss and examine, and they may be surprised by an unexplained change of agenda. Some patients, particularly those who have no insight into their mental health problems and believe their skin disorder to be entirely physical, may not respond well to their dermatologist changing the focus from the skin to psychological health. However, there are certain approaches that can help with the engagement of all patients:

Consultation Environment Factors

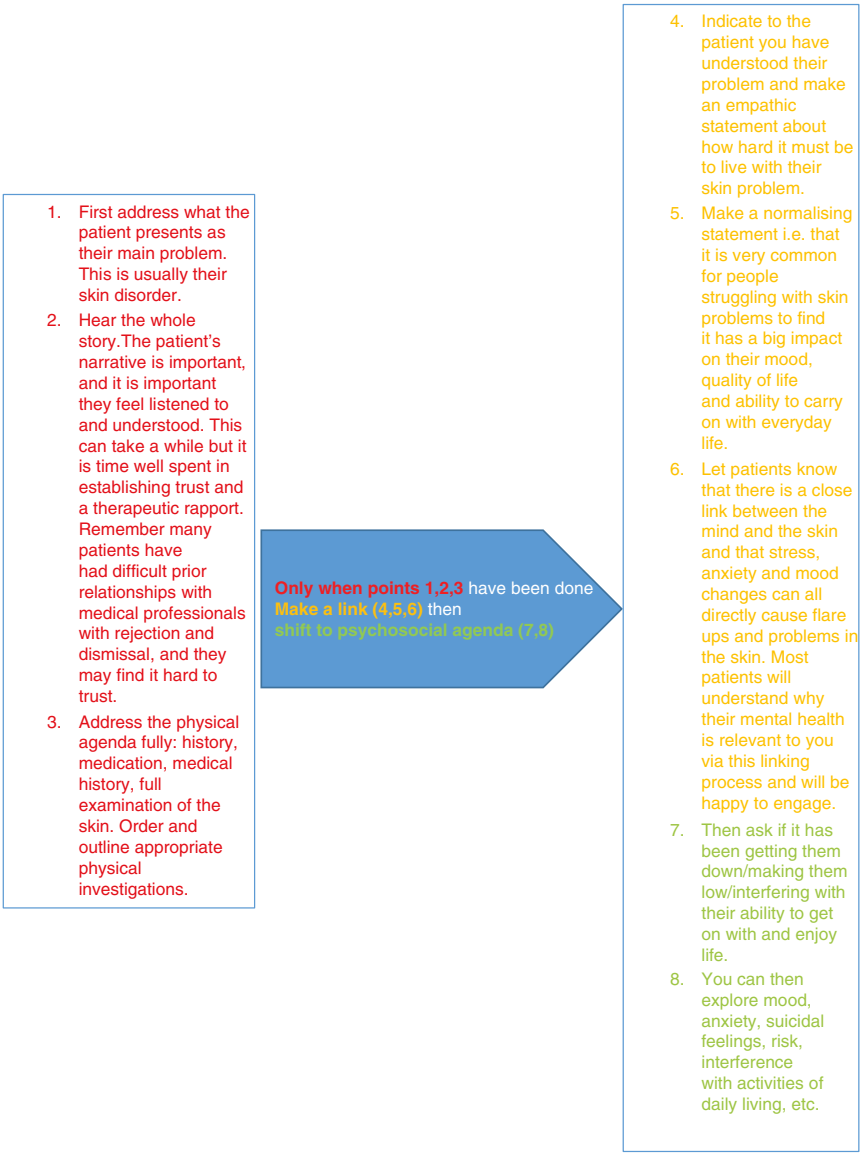
- Consider privacy issues: try to reduce the traffic of staff in and out.
- Safety: ensure the room is set up such that the clinician can exit safely.
- Time: if you are running a specialist psychodermatology clinic you will book longer appointment times, typically 45–60 min for a new patient and 30 min for follow-up. In a general clinic, consider asking a patient to come back and booking two or three clinic slots in order to have time to address psychological health. Inform the patient upfront how long the consultation will last and that follow-up appointments are possible if not all aspects of the patient's problems can be addressed in the initial consultation.

A Note on Record Keeping and Confidentiality Issues

The details of this will depend on the liaison model being used and the legal requirements of the respective country. However, there are some general considerations.

- Mental health information is often more sensitive than physical health information and patients may feel much more sensitive about how the information is recorded and who will have access to it. For example many patients feel uncomfortable with GP reception and administration staff (whom they may know personally in their own communities) potentially reading very sensitive information about their mental health and relationships.
- Patients are much more likely to feel comfortable sharing sensitive details if there has been an explicit discussion of what will be recorded, where, in how much detail, and who will have access to it.
- Staff such as psychologists and psychiatrists seeing patients may be employed by a separate care provider and not the acute general hospital. The mental health care provider may have its own separate record keeping system, activity recording, and clinical governance system. They may be required to keep a full separate medical record including such things as risk assessment. This can pose a problem, and there is not a catch-all solution as it will depend on the local requirements and situation. Any solution to this problem will require negotiation and discussion as to how a record is kept by both the physical health and the mental health care provider without the need for time-wasting and costly duplication.

Behaviours and Techniques with Which to Engage Patients in Discussing Their Psychological Health



Useful Interview Techniques

- Be open, empathic, non-judgemental, maintain non-threatening eye contact.
- Use transition statements, e.g. 'Now I have understood a bit about your problem, I am going to ask about your family'. Patients are then not surprised by a change in the line of questioning.
- Use normalising statements. These can be useful if you need to ask about issues that may be stigmatising or where patients may feel defensive and as though the questions imply something undesirable about them (e.g. recreational drug use, alcohol history, cognitive impairment, hallucinations). A normalising statement is something along the lines of 'We have to check certain things with all patients whether it is relevant or not', or 'I need to run through some routine questions about alcohol use', 'I need to ask about use of any non-prescribed drugs' or 'I need to do some brief tests of your memory, we have to check this in everyone'. Other types include normalising for certain problems, e.g. 'many people with similar problems to yours also experience X'.
- Use open questions. E.g. 'How have you been in your mood'? or 'How have you been feeling in yourself?', rather than 'Have you felt depressed'?
- Avoid loaded questions.
- Use summarising and clarifying statements, check you have understood correctly. This has the added advantage of giving the patient an idea of their own problems in a structured way.
- Check patients' comfort zones if they are starting to discuss something difficult, check they are ok with this, let them know they do not need to tell you all the details if they are not comfortable doing so. Indicate that they can talk about it another time or you can refer them to speak with someone else. This is very important if patients have alluded to issues of abuse of any sort. Be aware patients may answer questions as you are an authority figure and they feel they have to answer, but later they may feel very vulnerable and exposed. Being aware of this and using the above technique should avoid the patient feeling uncomfortable afterwards about how much they have shared.

Correspondence and Communication of Mental Health Assessments in the Joint Psychodermatology Setting (Where This Model Is Possible)

- Mental health staff and dermatologists are often working for different health care providers.
- There needs to be service level agreements between providers about who is responsible for funding and providing administrative support: administering the clinic, bookings, patient appointment letters, writing clinic letters, etc.
- Similar considerations apply to letter writing and communicating with the GP and other professionals. The professionals involved need to work out an efficient way of creating relevant correspondence without duplicating one another and

having the right balance of sharing appropriate information whilst avoiding sharing sensitive details unless it is vital.

- Many mental health and acute medical care providers require that all correspondence is copied to patients. In the case of mental health assessments, it is important that anything in the letter will not come as a surprise to the patient. It is also important to check the patient is happy to have a copy of the letter - some patients may decline if they are concerned someone else will open their mail and read sensitive information. If a particular diagnosis or formulation has not yet been shared with the patient it should not be put in a letter. This can be a particularly difficult area when dealing with patients with dermatitis artefacta, factitious disorder or delusional patients such as those with a delusional infestation or the delusional subgroup of body dysmorphic disorder. In those patients, gradual or limited exploration of the diagnosis with the patient is acceptable. See Box 2.5 for some suggestions on how to write GP letters in the psychodermatology clinic.

Box 2.5 Some Suggestions for How to Write GP Letters

Where the patient has a diagnosis, which cannot immediately be shared with them, e.g. dermatitis artefacta, factitious disorder, or they are delusional with no insight, letters to the GP which are also copied to the patient have to be written to avoid saying anything which has not been shared with the patient. Otherwise, any developing trust and therapeutic rapport will be lost the moment the patient reads the letter.

The solution is usually to avoid writing the diagnosis, e.g. factitious disorder, delusional infestation, and simply record in the letter what the patient has told you: e.g. patient believes they have mites burrowing under the skin, the patient feels unhappy that the shape of their nose is triangular, or such like. Patients' behaviour can also be described; e.g. they are throwing away their sheets every week, they check reflective surfaces constantly, they spend 3 hours putting on make-up, etc. The clinician can then make a factual statement of examination findings, record investigations and results when available. It is usually then clear to other professionals that there is a mismatch between the patient's perception of their situation and that of the clinician. In some instances, an additional letter to the GP or referring doctor that is not disclosed to the patient may be feasible. Treatment and its purpose can be recorded, e.g. topical creams to moisturise and containing antibacterial properties, low dose neuroleptics (this term may be preferred to antipsychotics) to reduce crawling and biting sensations in the skin, thus reducing itch and helping sleep. Obviously, all this will also have been discussed with the patient but the letter will serve as a reminder of the treatment rationale, and therefore has potential therapeutic value. It can also be helpful for patients to show to pharmacists as the latter may not dispense neuroleptics unless they understand the rationale for them.

If it is necessary to make the GP aware of suspicion of factitious disorder it may be best to do this by phone or with an additional letter. This diagnosis may need to be discussed with the patient at some point but it is important it is not done too soon, if there is any diagnostic uncertainty, and if there is not a good rapport with the patient.

Different health systems in different countries may have different legal requirements around doctors' letters which obviously need to be taken into account.

Why Are Mental Health Disorders Missed in Dermatology Outpatients?

- Often missed due to context: brief consultations, lots of staff in and out, the focus is on examining skin and brief dermatologically focused history.
- Doctors and nurses may not ask about mood due to fear of opening 'can of worms'.
- Perceived stigma of mental illness limits its discussion by doctors and patients.
- Patient may feel mood symptoms not relevant, 'nothing the doctor can do'. Also patient may be concerned that discussing mental health problems may distract the doctor from addressing physical symptoms thoroughly and physical symptoms will be ignored.
- Staff may feel depressive/anxious reaction is normal and inevitable: 'I would be depressed if that happened to me'; but not all dermatology patients are depressed or anxious!
- Common physical symptoms of depression such as poor sleep, loss of appetite, tiredness can be due to the physical illness.

Factors to Look Out for in Dermatology Patients Which May Suggest There Is an Underlying Psychiatric Disorder

- Distress about skin disease very severe.
- Mood change persistent (>2 weeks) and not responsive to the environment.
- Failure to adjust to illness-exaggerated perception of altered body image, feel ugly and disfigured out of proportion to objective assessment. Difficulty adhering to treatments, overwhelmed.
- Physical function poorer than expected, failure to continue or resume social and work roles.
- Recovery slower than expected, rehabilitation difficult. Patients may be very avoidant of social situations, going out in public, returning to work, etc., even after skin improves.
- Dermatologic non-disease, e.g. burning sensations are frequently associated with depression.

If any of the above are observed there is a need to actively look for an underlying psychiatric disorder.

Approaches to Patient in Specific Clinical Situations: Some Questions to Use and Things to Notice

Assessing Mood

- Subjective: How the patient feels in their own words. Fed up, sad, etc.
- Objective: Observe and record objective indicators of mood during the interview such as body language, behaviour and facial expression, e.g. weeping, sad expression, laughing, irritable, etc. Biological symptoms can be included here.
- Open Question First: 'How have you been feeling in yourself …….or feeling in your mood….. or feeling in your spirits (or try all three) recently'?
- If there is no clear response ask a more closed question: 'Have you been feeling at all low, sad or miserable recently?' or 'Have you been feeling at all depressed'?
- How bad has it been—look for a pervasive low mood. Note variability and reactivity.
- Tearfulness present or not.
- Negative cognitions such as hopelessness, worthlessness, guilt. Yes to all these indicates higher suicide risk.
- Anhedonia (inability to enjoy things one normally enjoys), alexithymia (inability to express one's emotions).
- Diurnal variation of mood (in depression, mood is often lower in the morning).
- Biological symptoms such as poor sleep, early morning wakening, loss of appetite, weight change, loss of libido.
- Assess severity: persistence, lack of variability, limiting social function, diurnal variation of mood and biological symptoms all indicate more severe depression.

Assessing Anxiety

There are two main components of anxiety:

1. *Cognitive: Anxious ruminations*
2. *Autonomic Symptoms of Anxiety*: Palpitations, tachycardia, paraesthesias, dizziness, cold clammy hands, sweating, hot and cold spells, frequency of urine, diarrhoea, nausea and blepharospasm. Increased muscle tone producing shakiness, tremor, trouble swallowing, lump in throat, muscular aches, excessive tiredness. These symptoms can be worsened by hyperventilation, which can also lead to dizziness, perioral and limb paraesthesias and muscular spasm.

   Asking About Anxiety:

- Have you had problems with feeling anxious/scared/ nervous/fearful? Try different words.
- Have you found yourself worrying constantly? Having thoughts and worries which go round and round in your head?
- If they say yes, ask how it makes them feel in their body? (open question). If no useful response, go on to ask if they have any of the specific autonomic symptoms of anxiety listed above.
- Ask if they have panic attacks; if they say yes, ask them to describe them. If they do not know what they are, describe a panic attack as a sudden feeling of fear or anxiety where they may feel they cannot breathe, the heart is racing, they feel sweaty or shaky and as though they may pass out or collapse, and they may feel they have to get out of the situation they are in.
- If they do have anxiety, ask if this is constant which hints towards generalised anxiety, or in relation to a specific situation which is phobic anxiety (fear of spiders, etc.).

Assessing Obsessional Symptoms

Features of Obsessional Phenomena

- Obsessional thoughts are repeated stereotyped intrusive thoughts or images which cannot be stopped, though they may be resisted.
- Recognised as patients' own thoughts.
- The motor act often accompanying an obsession is called a compulsion, e.g. handwashing, checking locks.
- Obsessional Rumination: repeating the same stereotyped thought over and over.
- Magical Thinking: The patient links two events, knowing that the connection is senseless (e.g. If I do not see three red cars today, my children will come to harm).
- Obsessional Images: repeated similar image in mind.
- Obsessional thoughts are often egodystonic, e.g. the religious person who has blasphemous thoughts. They are recognised as irrational by the patient, the patient usually tries to resist them, and the resistance causes anxiety, which is relieved by a ritualistic act.

Asking About Obsessional Symptoms:

- Before asking about unusual symptoms like obsessional symptoms you may want to make an orientating statement like 'you may find some of these questions a bit unusual and they don't apply to everyone, but I need to ask them just to check whether you have had any of these experiences'.
- 'Sometimes people find they have to keep checking everyday things even though they know they have done them, for example checking light switches, gas taps, locks. Do you ever have problems like this'?
- 'Are you someone who is unusually tidy and orderly and you find you have to keep things in a special order for example ornaments, clothes or papers'?

- 'What about being unusually clean and finding that you have to either wash your hands very frequently or clean things in your house excessively'?
- If present, check frequency severity and impact on function.

Obsessional Disorders in the Skin Clinic

Skin picking disorders and trichotillomania: these can be driven by obsessional thoughts, e.g. repeated thought that something needs extracting from under the skin before the area will heal. The picking or plucking can be a compulsion: there will be a strong compulsion to pick driven by ideas that this is needed for healing or to relieve the compulsive drive. The patient experiences anxiety when the compulsion is resisted, then relief immediately afterward, though they will often then experience shame and guilt about having picked.

Body dysmorphic disorder is thought to lie on the obsessive-compulsive disorder spectrum. The patient may experience repeated obsessional thoughts about the appearance of a part of their body with a strong drive to repeat behaviours such as checking in a mirror or trying to alter a part of the body, e.g. arranging the hair, covering blemishes, etc.

Assessing Psychosis in the Skin Clinic

What is psychosis?

The defining symptoms of psychosis are:

- Loss of contact with reality.
- Hallucinations: auditory, visual, tactile, olfactory or gustatory.
- Delusions: These are fixed, usually false, unshakeable beliefs held with subjective conviction and usually despite evidence to the contrary. They are not explained by a patient's usual cultural or religious concepts. The intensity of the delusional belief can be variable.
- Loss of insight.

Types of Psychotic Illness

- Schizophrenia.
- Mania.
- Psychotic depression.
- Schizoaffective disorder.
- Organic psychosis.
- Delusional disorder.

Any of the above can be seen but the commonest psychotic presentation in skin clinics is *delusional disorder*. The ICD 11 criteria for this disorder is that the main features include non-bizarre delusions, the criteria for schizophrenia are not met, there is no auditory or visual hallucination, though olfactory and tactile

Box 2.6 Some Common Psychoses Seen in Skin Clinics

- Delusional infestation.
- Morgellons: Disorder often suggested by patients following research on internet. Patient sees hairs often coloured on or growing out of the skin, and there may be a variety of non-specific neuropsychiatric symptoms, e.g. fatigue, headache, poor concentration. Not scientifically accepted as a diagnostic entity. The new term suggested in Morgellons is 'unexplained dermopathy'.
- Body dysmorphic disorder (psychotic subtype). Patient has delusions about abnormalities of appearance in a particular part of their body, e.g. believes there are big scars, pores, or swelling of nose, eyes, chin, etc, when no such abnormality is objectively apparent or if it exists it is very minimal.
- The belief of abnormal smell (cachosmia).

hallucinations can occur, mood episodes may occur but are brief compared to the duration of the delusion, the disturbance is not secondary to drugs, alcohol or any general medical condition. There can be a variety of types of delusion, e.g. erotomanic, grandiose, jealous, persecutory or somatic such as infestation. In the skin clinic, somatic delusional disorders are commonly seen. The delusions remain focused around the main somatic theme, and the rest of the personality can be remarkably intact in comparison to schizophrenia, where there is often a general deterioration of cognition, affect and personality. See Box 2.6 for common psychoses seen in skin clinics.

Risk Assessment

- Always consider the risk to self, others and of neglect.
- Must always do a risk assessment: two main forms of risk to consider.
  1. Risk of patients harming themselves. This can include:
     (a) Suicide risk in patient.
     (b) Risk of other self-harm: e.g. damage to skin driven by abnormal beliefs or picking. There may be a risk of self-mutilation in patients with body dysmorphic disorder.
  2. Risk of patient harming others, e.g. risk to children in their care, inability to adequately parent, shared delusional beliefs involving children, for example in delusional infestation patient may expose the child to harmful 'treatment', e.g. with bleach or disinfectant (be aware of child protection guidelines), very rarely *Münchausen by proxy*. Consider the risk to other clinicians in patients with delusional disorders. Patients may threaten the plastic surgeon to obtain a cosmetic procedure.

Box 2.7 Statistical Population Risk Factors for Suicide

Older age (any sex)
Middle-aged and young men
Male
Previous attempts, especially with violent methods
Psychiatric history
Family history of suicide or suicide in close other
Unemployment
Poor physical health, especially chronic pain
Recent loss/bereavement
Living alone
Alcohol/drug misuse

Assessment of Suicide Risk

- You must ask about thoughts of suicide and self-harm.
- There is no evidence that asking about suicidal ideas increases the risk of suicide; in fact the opposite is the case. You will reduce the risk of suicide by asking patients about suicidal ideas.
- Be aware of the risk factors for suicide (Box 2.7) and assess the patient for these risk factors.

How to Ask About Suicidal Ideas?

- Begin with an enquiry about mood and move stepwise into more specific enquiry as appropriate.
- Having established there is low mood or distress, move into enquiry about suicidal feelings: Ask about the future and feelings of hopelessness. 'How do you feel about the future'? 'Do you feel hopeless or do you feel things will improve'?
- Ask about passive suicidal thoughts: 'Has it ever got so bad that you have felt you did not want to carry on'? 'Have you felt that life was not worth living'? 'Have you ever wished you would not wake up in the morning'?
- 'Have you ever thought about acting on those sorts of feelings'? 'Have you thought about doing something to harm yourself'?
- 'What sort of things have you thought about doing'? 'How close have you got to carrying out these thoughts'? 'Have you done anything to harm yourself'?
- If you get affirmatives to the above, spend time exploring the intensity and frequency of the thoughts, the detail of the plans, whether plans have been put in place to say goodbye to others (letters, etc.), organising affairs, e.g. making a will. Detailed plans and preparations increase the risk.

- If there are suicidal feelings, it is important to understand what is keeping the person going, and preventing them from acting on these feelings: 'What has stopped you from acting on these thoughts/feelings'.

Practice Points

- Carefully consider the model of service delivery which is feasible in your service. The optimal model for seeing patients with a primary psychiatric disorder which presents via the skin is joint consultation with a psychiatrist and dermatologist.
- When setting up psychodermatology services, consider issues such as appropriate space, and how to deal with clinical correspondence.
- Patients attending psychodermatology clinics should understand who they are seeing, what the role of each professional is, and should understand how records will be kept and how information sharing with GP and others will function.
- Engagement of patients with the biopsychosocial model is crucial to their management. This engagement is achieved by fully understanding the patient's presenting problem, hearing their story and carrying out a thorough physical assessment BEFORE then moving to the psychosocial agenda. This move can be facilitated by using normalising and empathic statements to encourage patients to see the link between their skin and their emotional state.
- Clinicians running psychodermatology clinics must be trained in psychological assessment and learn appropriate communication skills which maximise their ability to elicit psychological symptoms and so understand their patients holistically.
- Clinicians seeing psychodermatology patients must always carry out risk assessments and be skilled in how to do this.

Capacity

3

Peter Lepping

Introduction

Most medical health systems consider the patient capacity to be a mainstay of medical practice. It is assumed that the capacitous patient can give valid consent which allows doctors to perform investigations, interventions and treatments. Normally, capacity is assessed by clinicians, whilst competence to give consent is judged by a court of law. In legal situations, judges will often consider the clinical opinion/s on capacity when giving a verdict on competence. From a day to day point of view most health legislations have provisions that allow treatment in the patient's best interest if they lack capacity. Most countries have gone down the path of individualised and capacity based health care to different degrees. What all have in common is the aim to protect patients against decisions made on their behalf which they would not approve of if they could have their say. Many authors go back to the Prussian Philosopher Immanuel Kant and his idea of deontology. Kant claimed that ethics is the belief that people's actions are to be guided by moral laws and that these moral laws are universal. At the time it stood in sharp contrast to absolutist regimes. Later universal declarations of human rights promoted through the United Nations have gradually led to a strengthening of the legal position of consent in most countries. In theory, autonomous decision-making should be independent of any outside and undue influence. In reality, many authors have argued that this is difficult and not realistic in many situations. However, many medico-legal theories and legal provisions consider autonomy to be one of the cornerstones of ethical decision-making in medicine.

P. Lepping (✉)
Wrexham Maelor Hospital Liaison Service (BCULHB), Wales, Wrexham, UK

Centre for Mental Health and Society, Bangor University, Wales, Bangor, UK

Mysore Medical College and Research Institute, Karnataka, India
e-mail: peter.lepping@wales.nhs.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_3

Having Capacity

Most legal provisions across the world assume that patients have capacity until proven otherwise. Many assert that patients should not be considered incapacitous merely because of certain diagnoses or characteristics. This is particularly important when assessing patients with dementia, learning disabilities, or acquired brain injuries. These are patient groups where incapacity can more commonly be found. However, capacity always relates to the decision that has to be made. In accordance with the sliding scale approach to capacity, some decisions require more capacity than others because they have potentially more severe consequences for the patient. It is also important to consider that capacity can be temporarily impaired and it may be possible to wait for a patient to regain capacity before making a decision. Reasons for a temporary impairment may be an acute medical illness, acute intoxication or brain injury (including strokes or other temporary neurological disorders). It is therefore important to make sure that the level of capacity required to consent is in keeping with the importance of the question. To consent to have bloods taken, for example, does not require the same level of capacity as the consent to, or refusal of, a potentially life-threatening operation. Some legal frameworks are relatively clear about how to assess capacity. What most have in common is that at the end of the assessment a binary decision of yes or no is required, which may be controversial or difficult to do in some cases. In fact, research suggests that whilst clinicians are good at identifying cases of clear incapacity, they miss many cases where incapacity is present but not completely obvious. Every country will have legal provisions that allow doctors to investigate and treat patients if they give valid consent. However, the burden of proof of what makes consent valid may be different in different legislations.

Lack of Capacity

There is a lot of research evidence suggesting that clinicians tend to overestimate their patient's capacity (Lepping and Raveesh 2014). A recent meta-analysis looking at the prevalence of lack of capacity in a variety of medical and psychiatric settings showed that 34% of medical patients and 45% of psychiatric patients lacked decision-making capacity (Lepping et al. 2015). The studies included in the meta-analysis incorporated inpatients and outpatients. Although the patient groups were heterogeneous, all studies used validated capacity assessment tools. It showed the high prevalence of incapacity in medical settings which is in contrast to many clinicians' assumptions and assessments. It should alert clinicians to the possibility that they may assume valid consent from their patients even though the patient lacks the capacity to give such consent.

Practice Point
Evidence suggests that clinicians overestimate patients' capacity.

Patients at Risk of Incapacity

Whilst most legislation requires clinicians to assume capacity, there are some patient groups that are known to be more at risk of lacking capacity than others. Patients with dementia and a mini mental state examination (MMSE) score of 20 or below are very unlikely to have full capacity to make decisions. Those with learning disabilities, delirium, acute mania or psychosis and neurological illnesses including stroke also have a higher prevalence of incapacity. Acute intoxication can often lead to temporary incapacity. Respiratory outpatients seem to have a particularly low prevalence of incapacity. One large study examining the prevalence of incapacity concluded that 26% of medical patients lacked capacity compared with only 3% of the healthy elderly controls. They added that while physicians routinely miss the diagnosis of incapacity, they were usually correct when they made that diagnosis (of incapacity). However, in their study, only 42% of patients with incapacity were correctly recognised as lacking capacity.

Illnesses and states with a high prevalence of incapacity
Delirium
Dementia
Neurological disease including stroke
Mania
Acute psychosis of any cause
Learning disability
Acute intoxication

Capacity in Dermatology

There are no specific studies to date about capacity and incapacity in dermatological inpatients or outpatients. We therefore have to extrapolate from known research. If the same principles apply, patients particularly at risk because of other underlying diagnoses may also lack the capacity to make dermatological treatment decisions. Equally, if a quarter to a third of medical patients lacks capacity this is likely to be the case in dermatological practice. However, there is some evidence that older people are more likely to lack capacity than younger patients which is again an important risk factor when assessing capacity. In dermatology, there are many investigations and treatment decisions where capacity needs to be at a high level because of the seriousness of the potential consequences of the illness and the interventions. This includes investigations and treatment for malignancies and benign cancers but also the use of methotrexate and other medication that have significant potential side effects. Many countries have legislation or protocols about what type of intervention may need formal written consent. However, it is worth remembering that any interaction with a patient requires consent, and therefore the patient has to have the capacity to give that consent. In addition, written consent given by a patient who lacks capacity is invalid, or in other words: null and void. It is therefore

important for doctors to protect themselves and the patient from wrongly assuming that decision-making capacity is present when it is not. Acting in the patient's best interest when the patient lacks capacity should lead to individualised decision-making that protects the patient. At the same time, the legal processes protect clinicians from accusations of paternalistic decision-making (Lepping et al. 2016) and usually allow a degree of coercion to pursue the best interest decision.

Assessing Capacity

Any clinician has to be able to assess capacity. Most legislation requires the person who performs an investigation or treatment to have assessed the patient's capacity to consent. Legal frameworks usually define what is required for a lack of capacity and normally include some kind of disturbance in the functioning of the mind or brain, either temporary or permanent, for incapacity to be a legal entity. In order to have *full* capacity, a person must be able to understand, retain, weigh up and communicate a decision. The following example is based on the principles of the Mental Capacity Act 2005 for England and Wales:

First step	Establish a disturbance in the functioning or the mind or brain
Second step	Understanding the information
	Retaining the information
	Weighing up and believing the information
	Communicate a decision

It is up to the clinician to make sure that any explanation of the planned investigation or treatment is done in a way that the patient can understand it. Most legislation requires clinicians to make every effort to make sure that they facilitate understanding. This can be in the form of pictures that explain the planned intervention or the use of interpreters or relatives in order to explain what is suggested to the patient. If patients have the capacity, they can refuse interventions that a clinician may deem reasonable. Most medical health legislation specifically allows decisions that do not follow medical recommendations as long as the decision-making process was at least to some degree rational and broadly in keeping with the information given to the patient. Communication of any decision is obviously an important part of communication and this may be impaired by language barriers, a need to use sign language, post-stroke or other speech impediments.

There may also be a problem with fluctuating or impaired consciousness. Some of these problems can obviously be more easily rectified than others. In order to assess capacity, a clinician could ask the patient how he or she came to any given conclusion which tests the ability to communicate and the rationale used by the patient. A simple question asking the patient to repeat what has been explained to him or her examines whether they have retained and understood the information given to them.

Capacity can fluctuate. It is important to try and assess capacity when the patient is at his or her best capacity wise. If the patient's capacity fluctuates and they are highly confused intermittently, any decisions the patient makes whilst having capacity must take into account any risks that occur while they lack capacity.

Practice Point
Capacity can fluctuate and is always related to a specific decision.

An Example of Legislation

In England and Wales, the Mental Capacity Act 2005 has given the public and clinicians a legal framework for medical interventions in persons who lack capacity. It allows treatment in a patient's best interest whilst they lack capacity. However, in addition it allows members of the public to prepare for times of incapacity by appointing persons for financial as well as social and health decisions. Such an appointee will be able to speak on behalf of the patient when they lack capacity as long as they have a registered power of attorney. This can only be overridden by the court if the appointee clearly acts against the patient's best interests. As part of the Mental Capacity Act, there are also liberty protection safeguards that are designed to protect patients who need more long-term accommodation decisions or serious medical interventions including longer hospital admissions. The safeguards are aimed at making sure that best interest decisions are made with transparency and individualise the decision-making processes. The legislation also allows for advanced refusals to be made. This may include specific dermatological treatments.

Important Pointers for Dermatologists

Dermatologists ought to be aware that a substantial minority of their inpatients and outpatients may lack capacity. They should be aware that they may themselves overestimate their patients' capacity, and may not sufficiently assess capacity systematically. They should be particularly cautious when dealing with groups that are at high risk of lacking capacity because consent given without capacity is invalid. It is important to be aware of the capacity-related legislation in the country one practises in. Advanced directives or lasting powers of attorney can be excellent tools to have discussions with people who may lack capacity in the future to determine what their wishes are. This then makes any future best interest decision-making much easier. It is important to see capacity legislation as supporting clinicians rather than being a hindrance because it does not just protect patients but also clinicians. If processes of the law are being followed, it is highly unlikely that dermatologists can be challenged for their decision-making in people who lack capacity or people who they wrongly assumed to have the capacity.

Practice Point
Be aware of high-risk groups for incapacity. Have capacity in mind when dealing with any patient.

References

Lepping P, Raveesh BN. Overvaluing autonomous decision making. Br J Psychiatry. 2014;204:1–2.
Lepping P, Stanly T, Turner J. Systematic review on the prevalence of lack of capacity in medical and psychiatric settings. Clin Med. 2015;15(4):337–43.
Lepping P, Palmstierna T, Raveesh BN. Paternalism versus autonomy—are we barking up the wrong tree? Br J Psychiatry. 2016;209(2):95–6.

Protecting the Psychodermatology Health Care Professional

4

Maria-Angeliki Gkini and Anthony Bewley

There are some challenges for health care professionals who work in psychodermatology. These include:

Complaints: Dermatologists who treat psychodermatology patients are at high risk of complaints, abuse and being threatened or stalked, as are other specialties, such as psychiatrists or cosmetic and plastic surgeons. Complaints are very common as well as threats of defamation through the internet and social media platforms.

Trust: Psychodermatology patients struggle to trust their treating physician, leading often to 'doctor shopping', a need for a second opinion and poor adherence to the treatment plan.

Burnout: Burnout in dermatology and specifically in psychodermatology is a reality and needs to be addressed.

M.-A. Gkini (✉)
Barts Health NHS Trust, London, UK
e-mail: margo.gkini@nhs.net

A. Bewley
Barts Health NHS Trust, London, UK

Queen Mary University London, London, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_4

Recommendations:
(a) Development of networks in Psychodermatology.
(b) Regular 'burnout' sessions at national and international meetings.
(c) Regular, sympathetic, confidential assessment for depression, anxiety and burnout for all dermatologists who treat psychodermatology patients.
(d) General Medical Council and other national regulatory bodies need to recognise the challenges of working in this emerging subspecialty of dermatology.
(e) Further training in psychodermatology is required.
(f) Development of regional dedicated psychodermatology clinics.

Introduction

Psychodermatology is an emerging subspecialty of dermatology dealing with the complex interaction between the skin and the mind. Psychodermatology or psychocutaneous medicine deals with two large categories of patients: (a) patients with primary psychiatric disease that develop secondary skin lesions or symptoms and (b) patients with primary dermatological disease that develop significant psychiatric or psychosocial comorbidities due to their skin problem. Both of these categories can be quite challenging in terms of management. In particular, patients with psychiatric disease may refuse a referral to a psychiatrist and frequently seek help from a dermatologist, considering their condition to be of cutaneous origin.

Complaints

Psychiatrists and related healthcare staff are at risk of complaints, abuse and being threatened or stalked, as are other specialties, such as plastic surgeons, especially those performing cosmetic procedures. In a study conducted in Australasia, 20% of cosmetic and plastic surgeons reported having been harassed by patients, especially those with body dysmorphic disorder or other underlying psychopathology. According to Gkini et al. 2019, in a survey performed in psychodermatology, 75% of dermatologists admitted feeling stressed, when dealing with this challenging category of psychodermatology patients. Also, 36% of psychodermatologists had received complaints about misdiagnosis and/or wrong diagnostic tests, which is a much higher percentage of complaints when compared to those generated from routine general dermatology referrals. Complaints were either escalated to local clinical governance teams/chief executives or/and National Regulatory bodies and physicians had to respond formally. Some colleagues were verbally abused by their patients and/or threatened with defamation. All of them reported that Internet abuse (trolling) has been used to threaten and/or defame them, using social media platforms, such as YouTube or Facebook.

Patients with delusional beliefs may be more demanding than other general dermatology and psychodermatology patients. In particular, managing patients with delusional infestation (DI) may invoke risks to dermatologists' personal and professional well-being. Dermatologists, who manage patients with DI, need to be aware of the specific personal risks, including complaints, referral to regulatory body, threatening behaviour, stalking and (very rarely) the risk of a violent attack. Regulatory and defence bodies appear to at least partly recognise the risks and offer guidance for doctors who are stalked. Due to the risks implicated, dermatologists may choose to refer psychodermatology patients to other healthcare professionals to obviate the risk to themselves and to achieve the best care for patients who represent a higher risk of complaints/abuse.

Burnout

Burnout is a major issue among physicians. Burnout has been defined as long-term, unresolvable job stress that leads to exhaustion and feeling overwhelmed, cynical detachment from the job, and a diminished sense of personal accomplishment, according to Maslach et al. In Medscape's 2019 report, 44% of physicians reported feeling burnt out; 11% were colloquially depressed and 4% were clinically depressed. In 2019, critical care (48%), neurology (48%), family medicine (47%), obstetrics/gynaecology (46%) and internal medicine (46%) were among the most burnt out specialties. Among the least burned out were plastic surgeons (23%), pathologists (32%) and dermatologists (32%).

Physician burnout affects both the patient and the physician. It has been demonstrated that physician burnout leads to lower patient satisfaction and care as well as a higher risk of medical errors. Direct effects on the physician may include higher employment turnover and an increased risk of addiction and suicide. Other more downstream effects of burnout may involve physicians' families and societal effects when fully trained physicians leave their clinical practice to pursue other careers.

Burnout in Dermatology and Psychodermatology

Working as a dermatologist often entails challenges and frustration. Compared with other specialties, dermatologists are in the middle of the pack, as the percentage of dermatologists who are burned out is less than that of physicians overall (44%) .

Only 34% of dermatologists in Medscape's survey responded that they were very or extremely happy. Dermatologists' rates of reported colloquial and clinical depression are about the same as those of physicians overall (11 and 4% overall). There are many contributing factors to burn out but the most significant include too many administrative tasks and bureaucracy, complying with policies and regulations, and less time for clinical work. The majority of dermatologists do not seek help. Many physicians have rationalised their exhaustion and discontent, noting that other physicians feel it too. Others say that their degree of unhappiness is bad enough to require outside help.

Dermatologists practicing psychodermatology are at a higher risk of burnout compared to general dermatologists. There have been no available data until recently. We recently conducted a European Survey in an attempt to quantify our hypothesis and assess the levels of burnout among psychodermatologists and evaluate potential contributing factors to it (Gkini et al. 2019). Despite the fact that the majority of physicians were working part-time with a mean number of working hours of 32, the mean score for burnout amongst psychodermatologists was high/very high, using the Oldenburg Burnout Inventory. The mean scores for disengagement and exhaustion were also high/very high. In conclusion, treating psychodermatology patients seems to be associated with an increased risk for burnout, disengagement and exhaustion.

Contributing Factors to Burnout and Mental Illness

Work-related factors that can contribute to mental illness in medical staff, including dermatologists include:

(a) Poor quality and/or risky working environments.
(b) Dissatisfaction with the achievable quality of patient care.
(c) Lack of support, e.g. from managers, or socially within the workplace.
(d) Employers' failure to address workplace stressors, e.g. time pressures, excessive workload and absence of support.
(e) Feelings of not being valued as an HCP.

(a) Poor Quality and/or Risky Working Environments
From our survey of European dermatologists who treat patients with DI, most were specialists in psychodermatology who had been trained in larger specialist centres. Further training in risk management for psychodermatology trainees and consultants may be necessary, perhaps with the development of support networks for dermatologists who manage patients with DI Dermatologists, who manage patients with psychocutaneous disease, may need appropriate support themselves. Regulatory bodies should become sufficiently briefed to assess and manage the complaints received from patients with DI. A multidisciplinary approach through a psychodermatology clinic is preferred when treating this challenging category of patients, as it builds a trust relationship between patient and clinician. Further studies with larger sample size and higher response rate are needed to provide objective data about complaints and stalking in psychodermatology, but also in dermatology in general.

(b) Dissatisfaction with Achievable Quality of Care
Another key point is the dissatisfaction with the achievable quality of care. It has become increasingly recognised that the best outcomes for patients with the psychodermatological disease are via a multidisciplinary psychodermatology team. The

exact configuration of the multidisciplinary team is, to some extent, determined by local expertise. Primary and secondary care need to work together consistently because patients with psychocutaneous disease may be 'doctor-shoppers'. Patients may seek repeated consultations in primary care because their disease, they believe, is not being acknowledged, or they may seek repeated referrals to dermatologists and other specialists for the same reasons. It is important for primary and secondary care to have the same agenda in treating patients with psychocutaneous disease, as any difference between approaches will be recognised by patients and will lead to dissatisfaction with the service and disengagement from clinicians. In addition, there is a growing body of evidence that it is much more cost-effective to manage patients with psychodermatological disease in dedicated psychodermatology clinics. Nevertheless, despite this evidence, and the demand from patients (and patient advocacy groups) for the delivery and establishment of psychodermatology services, it is very sporadic globally. Clinical and academic expertise in psychodermatology is emerging in dermatology and other (often peer-reviewed) literature. Healthcare professionals need to be aware of the steps necessary to establish and maintain psychodermatology services (see below). Furthermore, organisations such as the European Society for Dermatology and Psychiatry champion clinical and academic advances in psychodermatology, whilst also enabling training of health care professionals in psychodermatology, which should be mandatory for dermatology training curriculum.

- Psychodermatology Useful references in Clinical Practice: Main Principles.

Marshall C, Taylor R, Bewley A. Acta Derm Venereol. 2016 Aug 23;96(217):30–4

- How to set up a psychodermatology clinic.

Aguilar-Duran S, Ahmed A, Taylor R, Bewley A. Clin Exptal Dermatol 2014 Jul;39(5):577–8.

There are other factors contributing to poor quality of services. Psychodermatology patients are often angry, desperate or fed up, expressing explicitly their bitterness, when they reach the specialist psychodermatology clinic. It is much harder to build a patient-physician trust relationship, especially when the patients lack the capacity to engage in the proposed management plan. Therefore, the consultation time with patients should be much longer than that of general dermatology consultations, with a mean time of almost 40 min (Marshall and Aguilar above). We often find that adherence to treatment is very poor due to the fact that patients are not happy to start on psychotropic medication, as they consider it to be irrelevant or they are scared of the potential adverse events. In a short survey (Pathmarajah Br J Dermatol, Jan 2019), patients seemed to be pleased with the information provided by their treating physician/prescriber, but there was

insufficient or even contradictory counselling by the pharmacy. Pharmacy departments should receive appropriate education to ensure that expectations are met, and a lack of education may in fact explain the ineffectiveness of the counselling as perceived by the patients. A tighter cross-collaboration between physicians, pharmacists and the patient could help to improve adherence behaviour.

Another important component of service quality is the patient's satisfaction with their treating physician. In psychodermatology clinics, patients may struggle to trust their dermatologists. In our research (Stavrou, Br J Dermatol, Oct 2020), 25 and 45% of patients thought that their doctors do not really care or are inconsiderate of their needs respectively; 25% reported limited trust levels towards their physician, and 33% reported a wish for a second medical opinion. These data highlight the challenging levels of distrust towards dermatologists in psychodermatology. Further research is required to improve patient access to comprehensive multidisciplinary psychodermatology services.

(c) Lack of Support

Another key factor affecting health care physicians who practise psychodermatology is the support from managers, clinical governance teams as well as regulatory bodies. In 2013 in the UK, a leading psychodermatologist, addressed a letter to the UK's General Medical Council to discuss whether avoiding sharing the diagnosis of delusional infestation with a patient would be permitted as patients with delusional infestation may lack capacity to make a shared management decision with their treating physician. The reply from the GMC indicated that conveying the diagnosis to the patient follows Good Medical Practice, as stated but the GMC, but that it was very difficult to give guidance about this category of patients.

Clinical governance teams may need to be trained about managing complaints from psychodermatology patients, and to escalate only non-abusive complaints and those where the patient has the capacity to complain. Medical Defence organisations have recently published guidance for physicians who may be the target of stalking, such as General Practitioners and Psychiatrists, but not dermatologists. Further official guidance is required for subjects such as complaints, threats of any origin, verbal/ physical abuse and stalking.

(d) Failure to Address Workplace Stressors

Consultation time is much longer during a psychodermatology clinic, and lack of administrative time can be a crucial factor for burnout. Psychodermatology patients can be very complex and adequate time is required in order to offer an effective holistic approach with satisfactory results. Therefore, employers and commissioners must facilitate the setting up of dedicated comprehensive psychodermatology clinics (www.appgs.co.uk/mental-health-and-skin-disease-report-2020/).

(e) Overlooking Dermatologists' Role

Finally, in large medical centres and hospitals, the dermatologists' role may be overlooked, due to the relatively few emergencies and often modest on-call work hours. Nevertheless, the reality is contradictory, as dermatologists see a significant number of patients, with complicated skin and systemic diseases that cause significant

impairment in the quality of life of patients. It is vital to inform other colleagues, managers and the public about the importance of that work.

Current Situation

The impact and the extent of the psychosocial burden amongst dermatologists who practise psychodermatology have not been researched sufficiently. Pilot studies with a small number of participants have shown that burnout exists in psychodermatology, with levels being high to very high. Contributing factors include working environment, lack of administrative time, absence of available comprehensive psychodermatology clinics, complaints and abuse (verbal, online trolling, physical) by patients as well as lack of support by managers, clinical governance teams and regulatory bodies. Further studies are required from various countries, in order to support psychodermatology working practices. Issues with mental health and the well-being of psychodermatology staff need to be addressed and further investigated and managed. Stigmatisation of physicians with psychosocial comorbidities exists and this is a contributing factor for dermatologists not requesting help and support and needs to be challenged. Treating psychodermatology patients can be challenging and constitutes a risk for personal and professional well-being. Currently, insufficient priority is being given to improving the health and well-being of psychodermatologists within the duty of care that all employers have for their staff. The need for set up of dedicated psychodermatology clinics could contribute positively towards both physicians and patients.

Recommendations

Our recommendations include the development of networks in Psychodermatology. International societies, such as ESDAP (European Society for Dermatology and Psychiatry) and APMNA (Association for Psychoneurocutaneous Medicine of North America), as well as national societies, such as Psychodermatology UK, can serve as a domain for the exchange of views, clinical and academic, in the subspecialty of psychodermatology, leading to the championing of clinical and academic excellence in psychocutaneous medicine.

Regular burn out sessions should be organised at the national and international dermatology meetings as well as in the local work environment, in an attempt to address the relevant issues and offer support to physicians.

Regular, sympathetic and confidential assessments for depression, anxiety and burnout could be offered to all dermatologists who treat psychodermatology patients. Activities and social gatherings within the workplace should be offered so as to improve physicians well-being at work. If there is clinical depression, anxiety or /and suicidal ideation, professional psychiatric and psychological help should be offered in a non-judgmental way, without stigmatisation.

The General Medical Council and other national regulatory bodies need to update about the emerging subspecialty of psychodermatology and familiarise themselves with the challenges faced.

In the workplace, clinical governance teams need to become aware of psychodermatologists' potential personal and professional risks and offer support.

There is increasing demand for formal regional and national clinical networks to identify the training needs of staff. Further training is required for both resident and specialist dermatologists and should be part of the dermatology training curriculum.

Commissioners should encourage employers to prioritise the support of dermatology staff who practice psychodermatology, regarding their mental health and wellbeing through engagement with local initiatives and organisational improvements.

There should be a close collaboration with national dermatology societies for the promotion of dedicated comprehensive psychodermatology services.

Finally, the development of at least regional dedicated psychodermatology services with a trained specialist psychodermatologists and clinical psychologist support should be encouraged.

Bibliography

Bewley A, Taylor R, Reichenberg J, Magrid M. Delusional infestation. In: Bewley A, Taylor R, Reichenberg J, Magrid M, editors. Practical psychodermatology. 1st ed. London: Wiley; 2014. p. 117–26.

de Moll EH. Physician burnout in dermatology. Cutis. 2018;102(1):E24–5.

Gkini MA, Dimitrov D, Tanev T, Chan Y, Taylor R, Bewley AP. Are dermatologists who treat patients with delusional infestation at risk of major complaints and being stalked? J Eur Acad Dermatol Venereol. 2018;32(10):e379–81.

Gkini MA, Hussain K, Taylor R, Bewley A. Burnout in psychodermatology: results from a European Survey. Acta Derm Venereol 2019;8:728.

Leslie Kane MA. Medscape National Physician Burnout, Depression & Suicide. https://www.medscape.com/slideshow/2019-lifestyle-burnout-depression-6011056.

Leslie Kane MA. Medscape dermatologist lifestyle, Happiness & Burnout Report 2019. https://www.medscape.com/slideshow/2019-lifestyle-psychiatrist-6011149.

Nelsen AJ, Johnson RS, Ostermeyer B, et al. The prevalence of physicians who have been stalked: a systematic review. J Am Acad Psychiatry Law. 2015;43:177–82.

Psychopharmacology for Dermatologists

5

Peter Lepping

Introduction

Because of the considerable degree of connection between psychiatry and dermatology, there is a need for dermatologists to be well acquainted with psychiatric medication and diagnosis. This chapter gives an update of psychopharmacology relevant to the dermatologist including important interactions between psychiatric and dermatological medication. As the prevalence of the psychiatric disorder in dermatological outpatients is estimated at 30–40%, dermatologists will often have to diagnose and treat comorbid psychiatric disorders. These are disorders such as anxiety, depression, substance misuse but also psychotic illnesses such as schizophrenia or delusional infestation. A good knowledge of psychiatric medication is therefore essential for dermatologists in order to treat primary or comorbid psychiatric disorders they may find in their patients. This chapter will cover antidepressants, beta blockers, anti-epileptics, benzodiazepines and antipsychotics.

Most guidelines suggest a stepped care approach in order to treat illnesses like anxiety or depression. This usually means that milder forms of the illness can be managed in primary care and without pharmacological approaches whereas moderate and severe forms of the same illness usually require medication in keeping with evidence-based approaches. A good reference guide is the UK NICE guidelines which are freely available on the internet, but many countries have their own guidelines for the treatment of common illnesses such as anxiety, depression, obsessive-compulsive disorder, body dysmorphic disorder, psychotic disorders, delusional

P. Lepping (✉)
Wrexham Maelor Hospital Liaison Service (BCULHB), Wales, Wrexham, UK

Centre for Mental Health and Society, Bangor University, Wales, Bangor, UK

Mysore Medical College and Research Institute, Karnataka, India
e-mail: peter.lepping@wales.nhs.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_5

infestation, social anxiety disorder, post-traumatic stress disorder, generalised anxiety disorder, panic disorder, eating disorders, stroke recovery and premature ejaculation.

Antidepressants

The main classes of antidepressants used today are selective serotonin reuptake inhibitors (SSRI) and tricyclic antidepressants. Whilst SSRI only have an effect on serotonin reuptake, the tricyclic antidepressants are more complex in their pharmacology in that most work on norepinephrine (noradrenaline) as well as serotonin and histamine. Both neurotransmitters are implicated in the aetiology of depression. In addition, the class of serotonin-norepinephrine reuptake inhibitors (SNRI) were specifically developed to work on serotonin and noradrenaline. Other antidepressants like mirtazapine or mianserin have presynaptic anti-histaminic α2-blocker and anti-serotonergic activity. They are pharmacologically related to tricyclic antidepressants, and therefore share their side effect profile. Additional antidepressants include Monoamine oxidase inhibitors (MAOI) which can be reversible or irreversible inhibitors of monoamine oxidase. Other antidepressants that do not fall into any of the above classes of antidepressants include agomelatine (a melatonin agonist), bupropion (a norepinephrine and dopamine reuptake inhibitor and nicotinic receptor antagonist), and reboxetine (a norepinephrine reuptake inhibitor). Whilst SSRI and tricyclic antidepressants are widely used and available, the use of antidepressants depends on traditions and guidelines in different countries as well as cost. As a general rule, there is not much difference in efficacy between newer antidepressants and older antidepressants. Network meta-analyses have shown that there may be slight differences between medications with regard to efficacy and tolerability, and SSRIs are often recommended first-line because statistically they are better tolerated than tricyclic medication. Antidepressants can be used for the treatment of depression, although medication is not recommended by most guidelines for subthreshold or mild depressive episodes. Antidepressants are also used for the treatment of anxiety disorders (including social anxiety, phobias and panic disorder), seasonal affective disorder, obsessive-compulsive disorder, post-traumatic stress disorder, eating disorders and body dysmorphic disorder.

There are a number of differences between SSRI and tricyclic antidepressants. When it comes to choices, any clinician may well want to consult their own national guidelines and recommendations. But as a general rule, tricyclic antidepressants are cheap. The main side effects include sedation, postural hypotension, urinary retention, weight gain, sweating and dizziness. Compared to other antidepressants they are more dangerous in overdose because of their effect on cardiac function. They are not addictive. SSRIs are the recommended first-line treatment for most illnesses where antidepressants are being considered because of their favourable tolerability compared to tricyclic antidepressants. They are not sedating. Common side effects include nausea, agitation, sexual dysfunction and gastric bleeding (particularly in

the elderly). Some people also find that they are more anxious when they first start an SSRI. Although SSRIs are non-addictive some people experience discontinuation symptoms, and a gradual withdrawal is always recommended, particularly in those SSRIs with a short half-life (like paroxetine). Most guidelines recommend the use of antidepressants in conjunction with non-pharmacological therapies such as cognitive behavioural therapy or other psychotherapy. As a general rule, doses used should be higher in anxiety and related disorders than in depression. Some antidepressants are particularly useful in specific conditions, such as the use of clomipramine in obsessive-compulsive disorder.

As an illness *depression* has a relatively high placebo response rate which has made it easy for critics of depression treatments to doubt their efficacy. However, a number of large network meta-analyses have clearly shown the superiority of antidepressant medication over placebo in moderate and severe depressive illness. SSRI and tricyclic medication as well as other antidepressants are therefore part of all depression treatment guidelines (Table 5.1). Most guidelines suggest using a single antidepressant rather than a combination or augmentation because of the usually lower side effect burden. It is normally considered good practice to start with an SSRI because of better tolerability compared to other antidepressants. Adults should respond within 2 weeks, older adults within 4 weeks of initiating medication. Around 60% of patients respond to the first-choice antidepressant. If a switch of antidepressant is necessary the patient should be switched to a different class of antidepressant, for example, from an SSRI to an SNRI or a tricyclic. The UK NICE guidelines advise against the use of dosulepin because the evidence supporting its tolerability relative to other antidepressants is outweighed by the increased cardiac risk and toxicity in overdose. Any switching of antidepressants can usually be done within a week (with the exception of MAOI). Combination therapies or augmentation with non-antidepressant medication would normally be started only in consultation with a psychiatrist. Most guidelines recommend *against* the use of benzodiazepines for depression. Common medications used in augmentation therapy would include lithium, an antipsychotic, a second antidepressant of a different class, or levothyroxine. Such treatment would normally be reserved for severe depression or treatment-resistant depression where a mere change of class of antidepressant has not achieved sufficient efficacy. Evidence suggests that this may be the case in about 10% of patients. Most research suggests that the best efficacy in severe depression is for electroconvulsive therapy (ECT). This must be given under a short general anaesthetic with a muscle relaxant (modified ECT). Whilst the use of ECT has gradually diminished in many developed countries, it remains very common in developing countries because of its efficacy and speed of onset. It is important to remember that most guidelines consider antidepressant treatment to be necessary for 6–12 months after the patient has fully recovered in order to prevent a relapse. In addition, medication can often be used in combination with psychotherapy such as CBT to enhance the efficacy of the overall treatment. This is particularly useful in moderate depression but not in severe depression. Remedies such as St John's Wort are common in some countries. St John's Wort essentially works like an SSRI, has data proving its efficacy when used in high doses, and has significant

Table 5.1 Commonly used antidepressants for the treatment of depressive episodes

Type	Medication and doses	Important side effects	Special comments
SSRI	Citalopram 20 mg daily up to a maximum of 40 mg daily. Escitalopram 10 mg daily up to a maximum 20 mg daily. Fluoxetine 20 mg daily, increased after an interval of 3–4 weeks to a maximum of 60 mg daily. Sertraline 50–200 mg daily	See Table 5.2 for the common side effects of SSRIs	No significant sedation. Have fewer antimuscarinic and cardiotoxic effects than TCAs
SNRI	Venlafaxine XL starting with 75 mg od, increased to at least 150 mg od to use its noradrenergic effect (up to a maximum 375 mg daily but only under specialist psychiatric supervision). Duloxetine 60 mg daily	See Table 5.2 for common side effects of venlafaxine and duloxetine	High doses of venlafaxine should be used only under expert psychiatric supervision. Blood pressure and ECG should be monitored once the dose of venlafaxine is >225 mg daily. No sedation and antimuscarinic effects. Antiemetics may have to be given with Duloxetine
NaSSa	Mirtazapine 15 mg at night, increased to 30 mg within 2–4 weeks according to response; maximum 45 mg daily as a single dose at night or 2 divided doses	Increased appetite and weight gain, oedema, sedation, constipation	Sedation occurs from 15 mg onwards and, being a strong side effect, can be helpful in pruritus
TCA and related	Amitriptyline 75 mg at night to 150 mg daily. Caution above 100 mg. Doxepin 75 mg daily (usual maintenance dose 30–300 mg daily). Imipramine 75 mg daily to be increased to 150–200 mg daily. Trazodone (150–600 mg). Dosulepin 75 mg at night, up to 225 mg daily (not first-line!)	Sedation, giddiness, weight gain, constipation, akathisia antimuscarinic effects, cardiotoxicity, rashes and hypersensitivity reactions, hypomania or mania, confusion or delirium, urinary retention	Doxepin is licensed for use as an antipruritic in eczema. Available as cream (doxepin hydrochloride 5%). TCA overdose can be fatal. NICE guidelines suggest the use of dosulepin should be reserved for specialist treatment because of a possible slight increase in cardiovascular events associated with long-term use

drug–drug interactions, which can lead to liver failure. Many guidelines therefore do not recommend its use.

In people with recurrent depression, antidepressants can be used for maintenance treatment beyond 2 years. The dose of medication at which acute treatment was effective should be maintained. Normally, relapse prevention is used in patients who had two or more episodes of depression in the recent past when they experienced significant functional impairment, for patients who have other significant risk factors of relapse or the consequences of relapse are likely to be severe, for example, suicide attempts, loss of functioning, severe life disruption, or inability to work.

Patients should be discouraged from suddenly stopping antidepressants because of the possibility of discontinuation symptoms, especially when using antidepressants with a short half-life such as paroxetine. Discontinuation symptoms can include restlessness, problems sleeping, feeling unsteady, sweating abdominal discomfort, altered sensations like electric shocks sensations, or altered feelings like irritability, anxiety or confusion. These withdrawal symptoms are usually mild and stop relatively quickly. Some patients, however, may have longer lasting discontinuation problems. Particularly after the use of high doses of antidepressants the medication should be stopped gradually and any sudden cessation should be avoided. It is important to emphasise that discontinuation symptoms occur with many medications and are *not* a sign of dependency.

Antidepressant medication is the standard medication for *anxiety* disorders (Table 5.2). As a general rule, most guidelines recommend the use of SSRIs as first-line pharmacology treatment. The dose usually required in the treatment of anxiety is normally higher than that used in the treatment of depression. Most modern guidelines do *not* recommend the use of benzodiazepines or antipsychotics unless specifically indicated and supervised by a psychiatrist. Alternatives to SSRI include SNRI, tricyclic antidepressants and pregabalin. It is important to note that there have been increasing concerns about the use of pregabalin and gabapentin in anxiety disorders because of the development of tolerance. Antidepressants have the best evidence base for efficacy in anxiety. This includes SSRI and tricyclic antidepressants. Imipramine and clomipramine as well as the SNRI venlafaxine are commonly considered good alternative choices when SSRI has failed. They should be considered if there is no improvement after a 12 week course of an SSRI. It is important to inform patients that at the beginning of a treatment with a serotonergic drug it is possible that anxiety and agitation may increase for the first few days before they get better. There is also a higher risk of discontinuation symptoms when patients suddenly stop their medication because the anxiolytic doses of antidepressants are usually higher than the antidepressant doses. Therefore, sudden cessation is more likely to cause short-lived discontinuation symptoms in anxiety treatment doses compared to antidepressant doses of medication.

When starting antidepressant medication, it is worth explaining to patients that it may take 2 weeks before a response can be expected (up to 4 weeks in the elderly). They are usually taken for at least 6 months beyond the patient's recovery to avoid a relapse. It is important to emphasise that they are not addictive. Antidepressant doses for older adults are generally half of those recommended for adults.

Antipsychotics

Antipsychotics (Table 5.3) are usually classed into first and second-generation antipsychotics. Antipsychotics were first used in the 1950s when chlorpromazine was developed. The advent of second-generation antipsychotics started in 1991 with the introduction of risperidone. Second-generation antipsychotics are sometimes called atypical antipsychotics because they did not produce extrapyramidal side effects in rats. However, in reality, all antipsychotics used today have varying side effect

Table 5.2 Non-benzodiazepines licensed for the treatment of anxiety

Name	Type	Mechanism	Dose	Common side effects	Comments
Escitalopram Citalopram	Antidepressant	SSRI	Escitalopram 10 mg once daily increased to a maximum 20 mg daily For Citalopram 20 mg and 40 mg respectively	Nausea, vomiting, dyspepsia, abdominal pain, diarrhoea, increased agitation, insomnia, hyponatraemia	Abrupt withdrawal can lead to discontinuation symptoms such as headache, anxiety, dizziness, paraesthesias, sleep disturbance, influenza-like symptoms
Paroxetine	Antidepressant	SSRI	20 mg once daily to a maximum of 50 mg daily	Same as above	Same as above, particularly prone to discontinuation symptoms because of short half-life
Venlafaxine XL	Antidepressant	SNRI	75 mg once daily, higher doses have not been proven to be more effective	Gastrointestinal, palpitations, nausea, sweating, increased BP	Discontinue if no response in 8–12 weeks
Duloxetine	Antidepressant	SNRI	Starting from 30 mg once daily up to a maximum of 120 mg/day	Same as in venlafaxine, but nausea is very common	
Buspirone	Anxiolytic	Acts on 5HT1a receptors	5 mg 2–3 times daily, increased to maximum 45 mg daily	Nausea, dizziness, headache, nervousness, excitement, chest pain, confusion, seizures, fatigue and sweating	Performance of skilled tasks may be affected. Short-term use
Propranolol	Antihypertensive	Beta-blocker	40 mg once daily, increased to 40 mg three times daily	Bradycardia, GI disturbances, hypotension, bronchospasm, fatigue, purpura, exacerbation of psoriasis, alopecia, rarely rashes and dry eyes	Does not affect psychological symptoms of anxiety. Only reduces autonomic arousal such as palpitations and tremor
Pregabalin	Antiepileptic		150–300 mg daily with a maximum of 600 mg daily	Dry mouth, constipation, nausea, vomiting, flatulence, oedema, dizziness, drowsiness, memory impairment. Rarely Stevens–Johnson syndrome and pruritus	Avoid abrupt withdrawal, caution in congestive heart failure, renal impairment and pregnancy. Often shows a rapid onset of effect. Addiction potential increasingly recognised.

Table 5.3 Table of commonly used antipsychotics (maximum doses are European norms and relate to the treatment of schizophrenia). Far lower doses and particular caution are needed in dementia and delirium, refer to national guidelines

	Drug	Dose	Side effects and efficacy (+++ highest to – not present)	Notes
Second-generation antipsychotic	Amisulpride	200–1200 mg	Efficacy +++ Prolactin level ++ Sedation – Weight gain – EPSE + QT prolongation +	Suggest dose splitting. Renally excreted. No liver effect. Only dopaminergic.
	Olanzapine	2.5–20 mg	Efficacy +++ Prolactin level + Sedation +++ Weight gain +++ EPSE + QT prolongation +	Risk of metabolic syndrome. Increased risk of cardiovascular events in patients with dementia. Multiple neurotransmitters affected. Used for anxiety and in delirium.
	Risperidone	0.5–6 mg	Efficacy ++ Prolactin level ++ Sedation + Weight gain + EPSE ++ QT prolongation +	Increased risk of cardiovascular events in patients with dementia. Primarily dopaminergic. Used in delirium. Paliperidone is a metabolite of risperidone and also a licensed antipsychotic.
	Quetiapine	50–800 mg	Efficacy + Prolactin level + Sedation ++ Weight gain ++ EPSE + QT prolongation +	Increased risk of cardiovascular events in patients with dementia. Multiple neurotransmitters affected. Often used in anxiety.
	Aripiprazole	5–30 mg	Efficacy + Prolactin level – Sedation + Weight gain – EPSE + QTprolongation–	Increased risk of cardiovascular events in patients with dementia. Long half-life. Partial dopamine agonist.
	Clozapine	100–800 mg	Efficacy +++ Prolactin level – Sedation +++ Weight gain +++ EPSE – QT prolongation +	Used for treatment-resistant schizophrenia. Requires registration and regular blood tests in most countries because of licensing and neutropenia risk. Many drug–drug interactions.

(continued)

Table 5.3 (continued)

	Drug	Dose	Side effects and efficacy (+++ highest to – not present)	Notes
First-generation antipsychotic	Haloperidol	0.5–5 mg	Efficacy ++ Prolactin level ++ Sedation + Weight gain – EPSE +++ QT prolongation ++	Used in dementia and delirium. Primarily dopaminergic. Other first generation antipsychotics include sulpiride, pimozide, promazine and chlorpromazine.

Efficacy relates to efficacy in schizophrenia. *EPSE* Extrapyramidal side effects

profiles, largely depending on the neurotransmitters they work on. The main target for antipsychotic medication is dopamine which explains most of the antipsychotic side effects. However, many other neurotransmitters are also targeted, for example, specific serotonergic, muscarinic, and histamine receptors, GABA receptors, and others. Amisulpride is the only antipsychotic that only has an effect on dopaminergic receptors. Clozapine is the least dopaminergic antipsychotic and has a wide range of target receptors.

Various new network meta-analyses have shown the variance of side effect profiles between the antipsychotics. The main potential side effects include extrapyramidal side effects such as stiffness or akathisia (restless legs) and Parkinsonian symptoms. Other significant potential side effects include weight gain, sedation, prolactin increase and QTc prolongation. Some antipsychotics lead to metabolic changes including those in glucose tolerance and in cholesterol levels. Significant differences also exist between the efficacy of various antipsychotics. Clozapine is accepted as the most efficacious antipsychotic followed by amisulpride and olanzapine. Details about efficacy and side effect profiles are well described in published network meta-analyses. It is now accepted that there is no particular class effect that differentiates first and second-generation antipsychotics, although amongst second-generation antipsychotics the variability between side effect profiles and efficacy is higher than amongst first-generation antipsychotics. Various studies looking at efficacy and the clinical relevance of the published data for antipsychotics in the treatment of schizophrenia have shown that there is definite efficacy above a placebo effect. However, for many patients, the efficacy overall is modest rather than high, as is the case for most drugs used in medicine. Increasingly, antipsychotics have been used well beyond the treatment of schizophrenia. Many have licences for the treatment of mania, maintenance treatment for bipolar affective disorder, behavioural challenges in dementia, and as an adjunctive treatment in depression. They are also used off licence in the treatment of mono-delusional disorders such as delusional infestation, and in the treatment of anxiety. Use lower doses of antipsychotics in the elderly and very low doses if used in elderly patients with cognitive impairment.

The current evidence for the use of antipsychotics outside the treatment of schizophrenia and in particular for delusional infestation is very limited and the

clinical relevance and efficacy remain to be confirmed in future research. Antipsychotics have shown disappointing results in research on the treatment of delirium and alcoholic hallucinosis. Outside the treatment of schizophrenia, any judgement on effect sizes and long-term efficacy of antipsychotics should be treated with caution. This is particularly the case with a view that some antipsychotics can have significant side effects for some people including catastrophic weight gain and heavy sedation. In addition, clozapine is only licensed in most countries when the patient is registered and regularly examined for early detection of agranulocytosis.

Mood Stabilisers

Lithium, anti-epileptics and antipsychotics are commonly used as mood stabilisers for people with bipolar affective disorder. All of them can also be used as an adjunct in treatment of resistant depression. Lithium is the most effective mood stabiliser to prevent episodes of mania and depression in patients with bipolar affective disorder. However, lithium has a small therapeutic window and regular blood tests are necessary to make sure that the medication is being used within its therapeutic boundaries. Most countries suggest that lithium levels ought to be between 0.4 and 1.0 to be therapeutic. In higher doses, patients can show signs of lithium intoxication. In addition, long-term use can have negative effects on the kidney and the thyroid gland, for which it is also necessary to monitor the patient carefully. Anti-epileptics such as carbamazepine, sodium valproate (or valproate semisodium) and lamotrigine are commonly used as mood stabilisers. There are obvious problems with such medications in women of childbearing age. However, anti-epileptics are a common second line mood stabiliser. Adding antipsychotics such as olanzapine, quetiapine or aripiprazole as a mood stabiliser in bipolar affective disorder has become more common. They are also sometimes used in difficult to treat depressive episodes. The evidence for efficacy is still scanty, but some antipsychotics have a licence for the adjunctive treatment of unipolar depression.

Benzodiazepines

Benzodiazepines are usually recommended for short-term use only. The shorter the half-life the more addictive they are, which is a particular problem in the case of lorazepam and alprazolam. Benzodiazepines are usually used in acute stress reactions because of their sedative and anxiolytic properties. Particular concerns are with benzodiazepines in people who have a history of addiction because they are particularly at risk of developing tolerance and drug-seeking behaviour. Another risk factor is that benzodiazepines can cause falls in the elderly. Some research has suggested a possible association between the development of dementia and long-term use of benzodiazepines. Other studies suggest that the risk of benzodiazepine abuse may be overestimated in primary care where many people seem to be able to take the medication without the development of tolerance and withdrawal symptoms. Certainly, long-term use of high dose benzodiazepines is never desirable and

can cause significant cognitive problems. Most guidelines today suggest a very gradual reduction of benzodiazepines if people have been on them for a long time. The aim would be to avoid long-term use from the beginning by emphasising to patients that they are strictly for short-term use.

Summary

Medication is commonly used to treat a variety of psychiatric illnesses. Dermatologists should be able to use standard antidepressants and antipsychotics to treat primary and secondary psychiatric disorders in psychodermatology. It is worth familiarising oneself with at least one antidepressant and one antipsychotic in order to treat depression, anxiety and delusional infestation. Local psychiatric colleagues or pharmacists can always be approached to advise further on medication. Medication is proven to be effective and mostly well tolerated in depression, anxiety and psychotic illnesses. It is used in conjunction with psychological therapies in some illnesses.

Psychological Interventions

6

Reena Shah

Introduction

There is a bi-directional process between skin conditions and psychological distress, with an overlapping biological mechanism associated with inflammation. Psychological distress can be manifested in a number of ways, including through the skin and cutaneous disease with organic or physical causes which can cause psychological distress (i.e. low-self-esteem, social anxiety, depression, stress). A review of the literature shows the relationship between various skin disorders and mood disorders indicating that there are various biological pathways that explain the biological relationships (such as hypothalamic–pituitary–adrenal axis hyperactivity, glucocorticoid receptor desensitisation and sympathetic nervous system activation). There is also a plethora of evidence to show that psychological distress can cause skin disorders and inflammation.

Prevalence

Approximately 30% of those living with a skin condition experience clinical levels of psychological distress. Eighty-five percent of those report that the psychological impact is the main component of their skin condition. High rates of suicidal ideation have also been reported, with 8.6% of outpatients with skin conditions and in particular, 7.2% of those with psoriasis and 5.6% of those with acne, which is higher than in general medical patients.

R. Shah (✉)
Central and North West London NHS Foundation Trust, University of Hertfordshire, Hatfield, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_6

Efficacy of Psychological Interventions

Over the years, studies have looked at the effectiveness of psychological interventions in dermatology but there is lack of randomised-controlled trials. Lavda et al. (2012) conducted a meta-analysis that included 22 studies. They showed that psychological interventions were beneficial for people with skin conditions; effect sizes suggested that interventions had a medium effect on the severity of the condition and psychosocial outcomes and a medium-to-large effect was seen on itch/scratch reactions. Recent therapeutic guidelines recommended the use of psychological interventions in routine practice. It is useful for clinicians to have an awareness of different evidence-based psychological approaches to draw on in the assessment process so they can refer on for appropriate psychological intervention. Dermatologists and nurses can utilise basic psychological techniques such as relaxation or habit reversal therapy within their practice. This would be in line with the stepped care model for providing psychosocial interventions for patients (with mild to moderate psychological distress) and then referring on for more complex issues as per patient need. Developing a therapeutic relationship and conducting a brief psychological assessment can be an intervention in itself, which would dramatically increase the quality of clinical care for patients with skin disease.

Principles of Skin Care Regimes

Finding the most beneficial skin care regimen is key for the patient to manage their skin condition effectively. It can be helpful to motivate and reassure the patient to not give up if one cream does not work and highlighting that it may take months of 'trial and error' to find the most effective treatment strategy. Adapting the skin care regime to suit the patient's daily schedule can also increase success. Promoting simple techniques to reduce stress can benefit the patient such as: good sleep hygiene, deep breathing, having a healthy work-life balance and rest, 'me time' and promoting a healthy and positive lifestyle. Supportive, social networks with others who experience the same difficulties can contribute to better treatment outcomes. Therefore, introducing support groups, such as The National Eczema Society, Eczema Outreach (EOS) and/or increasing psychoeducation via relevant websites (e.g. www.skinsupport.org.uk) or giving leaflets from specific charities, such as The Vitiligo Society and The Psoriasis Association, can also be helpful.

Mode of Therapy

Principles and Set Up

Psychological therapy can be conducted either on a one-to-one, couple, family or group basis with people of all ages. Usually in group therapy, the content is organised around one type of problem (such as anxiety or social skills) or type of condition (such as eczema or dermatillomania) and in general have around 6–8 patients per group. Depending on the patients' difficulty and goals, the clinician's skills and the

service remit, this can govern which mode of treatment is offered. However, the psychology service set up can also vary from service to service and some have a pathway to follow, for example, patients attend first a group, then telephone sessions and then individual/couple or family face-to-face sessions. Whereas other services offer mode of therapy based soley on the individual's needs and the clincian's experience.

Benefits and Disadvantages

There are disadvantages as well as benefits of the different modes of therapy, for example in group therapy, patients are given the opportunity to share and explore their experiences with one another, which can help to reduce stigma, to normalise the problem/condition and to feel supported. Patients often encourage one another, model positive strategies, decrease feelings of isolation and increase self-confidence which would not feature in individual therapy. However, in individual therapy patients have the opportunity to explore deep-rooted issues where more complex problems can be explored and therapy can be tailored to meet the individual's needs.

Treatment Pathway/Stepped Care Model

Principles

Studies have shown that a high percentage of patients perceive the severity of their skin disorder and its impact on their life as more distressing than the objective severity of their disorder. Therefore, it is key to explore the patients' perceptions of their disorder. Within a psychological assessment, completing baseline objective measures can be useful (such as the Dermatology Life Quality Index (DLQI, Finlay and Khan 1994), Patient Health Questionnaire (PHQ-9, Kroenke et al. 2001), a brief measure for General Anxiety Disorder (GAD-7, Spitzer et al. 2006), as well as specific assessments such as the Cardiff Acne Disability Index (CADI, Motley & Finlay 1992), or a vitiligo-specific quality-of-life instrument (VitiQoL, Lilly et al. 2013). This can help to ascertain the severity of the psychological distress and the impact on their daily life. A 2018 study suggested that it would be useful for dermatologists to detect patients at risk of psychological problems by using a simple psychological outcome measure and subsequently refer them for psychological consultation (Panebiano et al. 2018). Skin disorders have historically been treated with medicine. However, it is known that reducing stressors and psychological factors can reduce flare-ups of skin conditions. The gold standard treatment in psychodermatology is to embed a psychologist in dermatology services, however, this is not always possible due to lack of resources and finances.

Stepped Care

The stepped care model is a system of delivering and monitoring treatments, so that the most effective and least resource-intensive treatment is delivered to patients first, then stepping up to intensive and specialist services as clinically relevant, see Fig. 6.1.

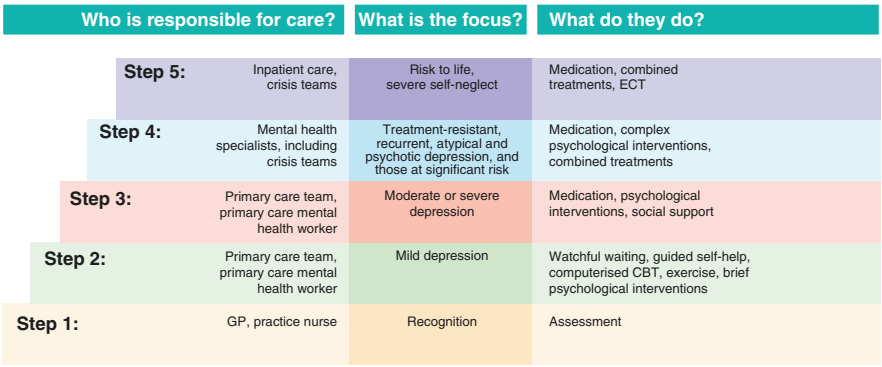


Fig. 6.1 An example of a stepped care model. Taken from https://wellbeinginfo.org/self-help/mental-health/stepped-care/

Within a psychological service, this entails increasing time with the patient and the therapy process, and increasing intensity based on severity. Firstly, offering behavioural treatments such as self-help or guided self-help, which includes behavioural activation and exposure and response therapy (stage 2). The next stage is offering CBT (group or 1:1), and/or other therapies such as Mindfulness, ACT or Systemic Family Therapy (stage 3–4).

What Can Dermatologists Do?

Dermatologists can offer self-help tips depending on the patients concerns and depending on the dermatologist's remit. They can either offer strategies that they feel comfortable with (such relaxation, stress management strategies, thought challenging, motivational interviewing approaches to increase motivation to change), or they can refer to local primary care mental health services, a psychologist, or to a specific psychodermatology service. Within all types of consultations and therapy sessions, managing patient and clinician expectations is helpful to improve and enhance success. Being mindful of what these are can positively influence the experience and dynamic between the clinician and patient. Exploring expectations can be useful, as it helps to set boundaries and create a safe non-judgemental space for the patient to explore their emotions fully.

Self-Help Approaches

Self-Help

Self-help is most beneficial to those with mild to moderate difficulties. It aims to increase patient knowledge and helps them to gain skills in how to better self-manage and overcome their psychological distress and/or skin condition. The self-help leaflet

can be based on either a specific skin condition, or a certain type of psychological approach to help a mood disorder associated with a skin condition (e.g. Hudson et al. 2020; Shah et al. 2014). Providing self-help has shown to reduce the time needed in individual therapy at a later stage and often patients have reported that further support is not necessary. Computer or literature-based self-help can be cost and resource effective. Patients who are engaged in this way are more likely to have better compliance and adherence to regimens. In addition, self-help material alongside a plan of when to use it has shown to reduce psychological distress in relation to skin disease and increases adherence (Shah et al. 2014). Specialists nurses can offer guided self-help which has shown to be more effective than self-help alone, which can be attributed to repeated reassurance and encouragement and maintaining hope.

- *Stage 1-signposting—websites, support groups and books, leaflets.*
- *Stage 2-psychoeducation, leaflets, books.*
- *Stage 3-guided self-help.*

Psychological Approaches/Therapies

Behavioural Therapies

Behavioural therapies aim to change behaviour/s that are unhelpful (sometimes habitual), external factors that increase stress. It is based on the premise that all behaviours are learnt. There are different types of therapies such as behavioural activation and exposure response prevention.

Behavioural Activation

Behavioural Activation is when the patient approaches activities that they were previously avoiding, using activity schedules. The aim is to slowly increase the time spent doing one activity with the hope to increase the number of positively rewarding activities. The rationale is that the difficulty (such as anxiety or depression) is a consequence of avoiding particular activities or situations. Specific goals are set for the week and the patient works towards meeting those goals.

Exposure Response Prevention

Exposure response prevention is when the patient faces their fear/s (such as fear of socialising or swimming) and they let the negative thoughts occur without challenging them. Within this the patient learns relaxation training and uses systematic desensitisation. This is a technique in which a hierarchy (listing anxiety-producing triggers from least to most distressing) is created with the patient. The exposure component helps the patient learn to tolerate increasing levels of distress with respect to situations that they fear. They expose themselves to the thoughts, objects, images, situations that make them feel anxious. The response prevention part involves the patient making a choice not to do the 'neutralising' behaviour/s (to make them feel better; e.g. to avoid going on holiday or swimming or to leave a party early) once the anxiety has been triggered.

Habit Reversal Therapy

Habit reversal therapy is a behavioural approach for patients with eczema or those who pick their skin. It aims to help patients to stop scratching or picking. See chapter 28 for more details.

Cognitive Behavioural Therapy (CBT)

Cognitive behavioural therapy (CBT) looks at the link between thoughts, emotions, physiology and behaviour. There is a plethora of research to show the effectiveness of CBT for a variety of skin conditions. When people feel distressed, they may fall into unhelpful patterns which worsen how they feel. CBT helps to recognise the problematic thinking styles (e.g. 'I am ugly) or behaviours (e.g. avoidance of socialising) and works on present symptoms (e.g. racing heart, sweating, shaking). It aims to challenge and alter difficult thoughts and unhelpful behaviours to more adaptive ways of thinking and behaving, which in turn impacts on the patient's physical feelings and emotions.

How patients cope with their skin condition also impacts the way they deal with their negative feelings. Explaining the link between the mind and the skin when exploring these factors in therapy can influence the dermatological and psychological treatment goals. Within sessions, there are numerous techniques that the therapist utilises such as eliciting thoughts, exploring thinking styles and emotions, cost and benefit analysis, problem-solving, event scheduling, promoting self-reward and positive affirmations, relaxation training, behavioural activation and thought diaries.

Figure 6.2 shows an example CBT model for someone with a skin condition. It shows how our underling core beliefs influence the way we think about ourselves,

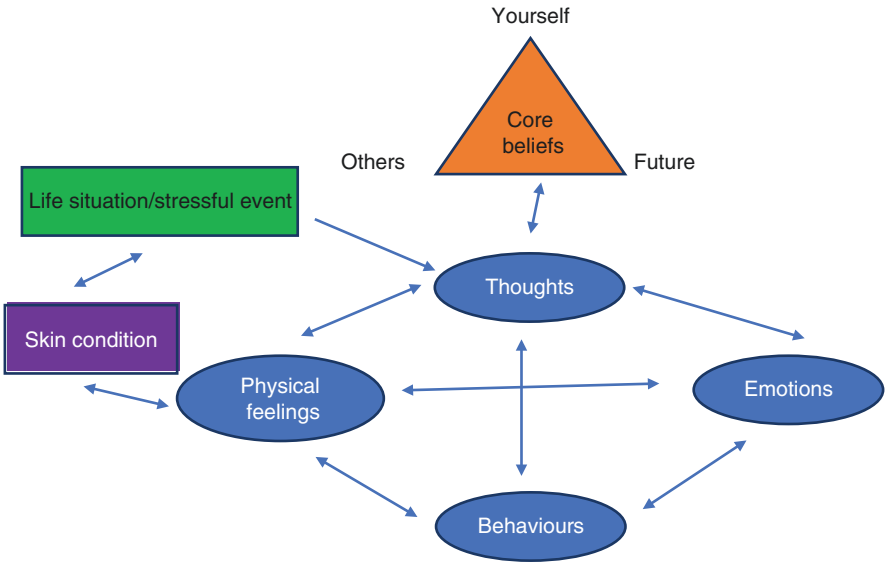


Fig. 6.2 Example CBT model

others and the world, which then impacts on the way we cope with life events/stressful situations. This influences how we cognitively process the situation and our thoughts, physical feelings, emotions and behaviours. Living with a skin condition adds an extra component that can exacerbate and/or precipitate stressful situations, given the biological link between stress and the skin.

There are particular models of CBT that are helpful to address underlying difficulties that may cause stress and consequently skin flare-ups (e.g. CBT for social anxiety or Body Dysmorphic Disorder). The approaches have specific evidence-based strategies. They help the psychological difficulties, mediated primarily by fear and avoidance, which are perpetuated by dysfunctional thoughts.

Relaxation

The link between the mind and the skin and the vicious cycle of stress and exacerbation of skin disease is well known. In particular, the relationship between stress and flare-ups of skin conditions (such as psoriasis, eczema and acne): activation of the stress HPA axis is known to affect the skin. Therefore, teaching patients simple strategies, that can be incorporated into daily life to reduce physiological arousal associated with stress and anxiety can be useful.

One strategy is slow deep breathing (which is the first skill to learning relaxation effectively). The purpose of slow deep breathing is to regulate physiology, therefore learning and teaching relaxation can help to reduce stress and worry. It also distracts from unhelpful thoughts and gives the experience of having some control. Once the basic skill of slow deep breathing has been established, the next step is to add in other components such as colour breathing, counting or external visualisation. If practiced regularly, the new skill can be effective in stressful situations. This provides the patient with a discreet coping strategy that can be used anywhere to help reduce the potential consequence of a stressful event as well as a flare-up. Facilitating relaxation with an imagery component is an effective method to lower state anxiety levels and itchy sensations. Studies have shown that relaxation can have a positive effect on many skin disorders, and with practice, the strategy can become automatic rather than consciously applied. Free patient handouts can be accessed online (e.g. www.getselfhelp.co.uk/docs/relaxation).

Trauma-Based Therapy

Trauma-based therapy looks at background factors from childhood or significant life events that cause distress in the present day. People who experience adverse childhood life events that are abusive or stress-filled are more likely to develop a range of physical health problems and social problems in adulthood. Patients often relate the exacerbation of the skin condition and psychological distress to memories or flashbacks from a trauma. Facilitating trauma therapy can work on reducing flashbacks of abuse (including the impact of them on daily life) and

stressful triggers, helping to reduce the psychological burden that is perpetuating the skin disorder (such as dermatillomania, acne excoriee or psoriasis). For some patients, their psychological difficulties predate the onset of the skin condition. Therefore, at times the predisposing skin condition could be viewed as an expression of their psychological problems, such as trauma, abuse, retirement or bereavement. When working with these patients, concentrating on the underlying problem rather than their skin condition can help reduce the severity of the skin condition by resolving psychological distress.

Schema Therapy

Schema therapy is an attachment-based therapy. It is an approach that combines CBT, Gestalt experiential therapy and psychoanalytical thinking. Schemas are a way of referencing the way in which we understand the world, a lens in which we use to view experiences (e.g. 'I am loveable'). We all have schemas, positive and negative, schema therapy has identified 18 core schemas. However, maladaptive schemas about ourselves and others (e.g. 'I am not loveable', 'I am not safe') are developed when childhood needs are not met.

Schema therapy aims to help people change longstanding patterns of thinking and acting, i.e. the maladaptive schemas. The approach looks to change the self-defeating core themes that are consistently repeated in the patient's life. The role of early maladaptive schemas of patients and the link to psychological distress in people with skin disorders is relatively new. An initial study (Mizara et al. 2012) linked some of the 18 schemas to skin disorders (specifically in eczema and psoriasis). The authors postulated that six early maladaptive schemas are reported by patients with skin disease: (1) emotional deprivation, (2) social isolation, (3) defectiveness/shame, (4) failure, (5) vulnerability to harm and (6) subjugation. Here, vulnerability to harm and defectiveness/shame predicted anxiety and vulnerability to harm and social isolation predicted depression. Overall the therapy helps to achieve change and improve adjustment in living with a skin disorder. Patients learn more adaptive ways of coping and relating to others and, consequently become less susceptible to psychological distress.

Acceptance and Commitment Therapy (ACT)

Acceptance and Commitment Therapy (ACT) is a behavioural therapy about taking values-guided action, by accepting the thought rather than challenging them and then to defuse it using techniques such as mindfulness. These are based on acceptance and commitment to values-based living. The patient uses their core values to guide, motivate and inspire behavioural change whilst engaging in the six core processes of ACT, see Fig. 6.3. They learn to accept what is out of their control and commit to taking action. The aim is to help create a meaningful life, while

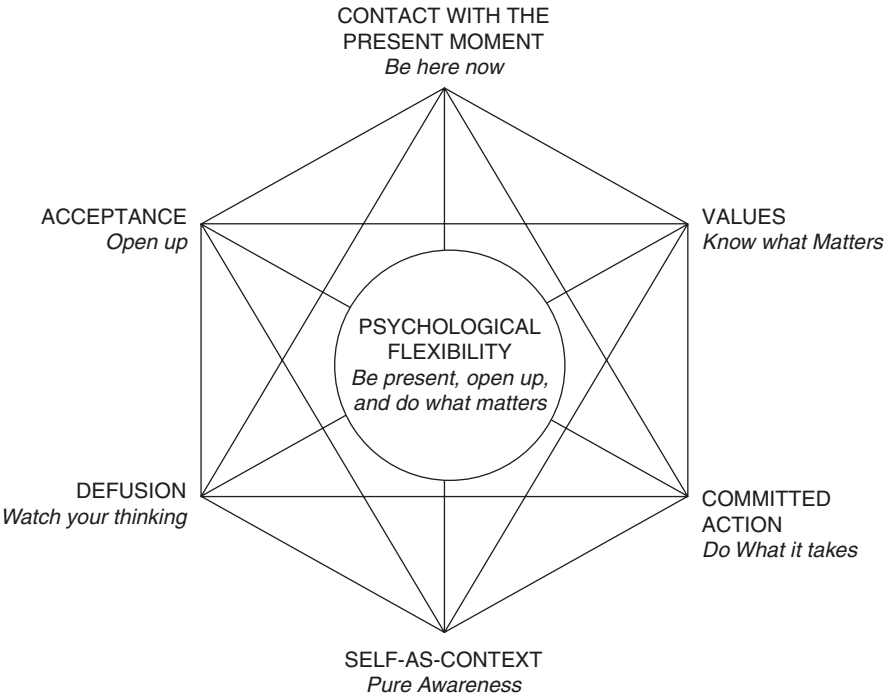


Fig. 6.3 ACT hexaflex—the six core therapeutic processes of ACT

accepting the pain that life inevitably brings. ACT has been shown to be effective for increasing psychological resilience for patients and significant reductions in measures of depression, anxiety and avoidance for skin pickers.

Mindfulness

Mindfulness is learning to disengage with thoughts and concentrate on the present moment. To be able to sit with distress and become aware of patterns in the mind. For example, to notice an urge to pick and to avoid self-critical thoughts without judgement. Whilst mindfulness is an ancient form of Buddhist meditation, it has become increasingly popular in current day therapies; within psychological therapy for skin conditions and especially via Apps, such as Headspace or Calm. Mindfulness is 'cultivating our ability to pay attention in the present moment'. It helps the individual to alleviate distress by disengaging from their automatic negative thoughts. Mindfulness facilitates acceptance of the situation in the present moment, whilst taking mindful action toward desired change. As with many new strategies, practice is the key to developing the skill. Over time the patient learns to cope with automatic thoughts and become more tolerant of stress. This is turn reduces the effect of

stress on the skin condition. Research has shown that delivering a brief audio mindfulness intervention during ultraviolet light therapy can increase the resolution rate of psoriatic lesions in patients with psoriasis. It has also been shown to be effective for those with dermatillomania. Studies show that mindfulness can change brain structures. The outcome of consistent regular practice can eventually lead to new automatic authentic changes, which lead to permanent behaviour modification.

Systemic Family Therapy

Systemic family therapy is a therapy that does not directly work on the mental illness within the patient. It aims to help the person mobilise the strengths of their relationships, to make the psychological difficulties less problematic. There is an understanding that psychological difficulties are due to past and present relationship problems and develop in the context of family and social relationships. Also that reciprocal dynamics in the family influence the problem/s.

One person can hold the stress, anxiety or anger for example, but focussing on the family dynamics and relationships in therapy can reduce stress in the individual, see Fig. 6.4 below.

One idea in SFT is that problems have a dual construction; they do not exist only within an individual, but rather are a product of the interactions between people and wider systems, such as communities and cultures. Within psychodermatology this approach links the patient's skin story with how patients are often influenced by other people's opinions (i.e. the problem of stigma within communities).

Figure 6.5 shows a formulation created for a patient who had psoriasis. The patient's psychological distress was rooted in adverse past experiences, perpetuated by negative thoughts and were precipitated and perpetuated by past and present relationship dynamics and interactions. She had a traumatic history of sexual abuse, being bullied and not being accepted by her parents or first husband. When she disclosed to her first husband that she had been abused, he left her, which perpetuated

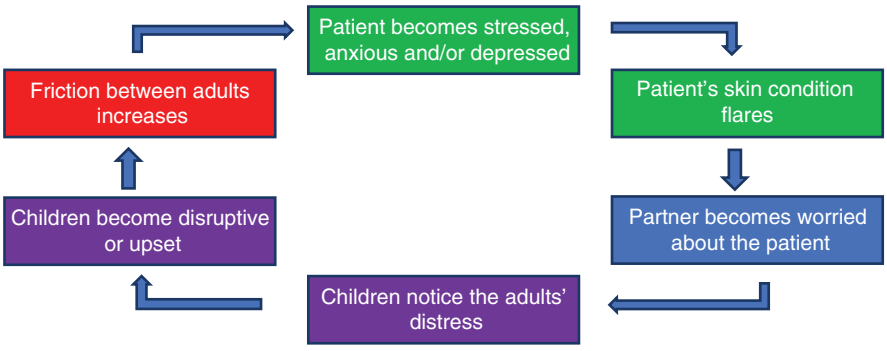


Fig. 6.4 Systemic family therapy diagram adapted for skin (taken from—http://www.svhf.ie/systemic-family-therapy.html)

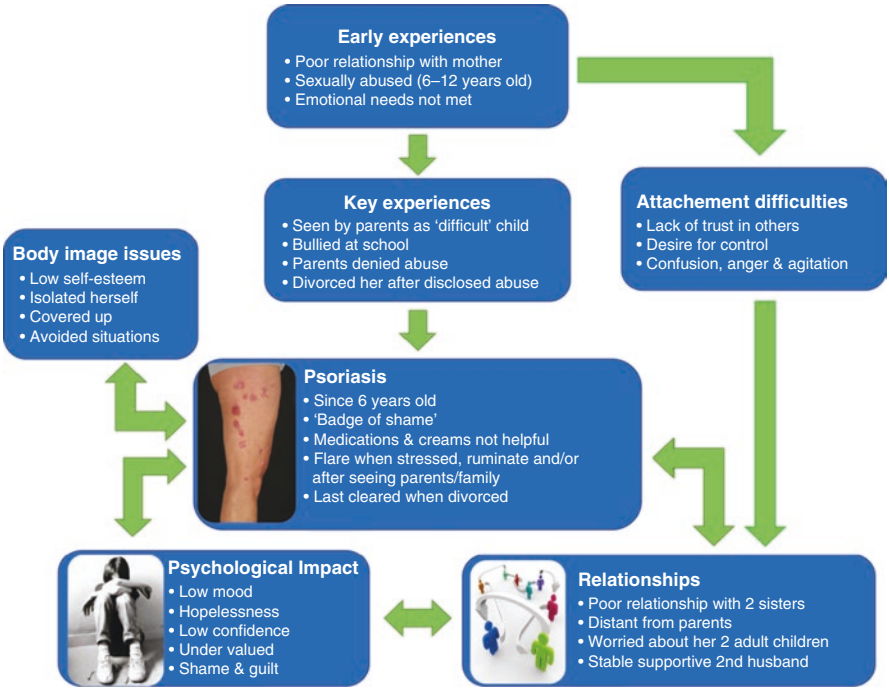


Fig. 6.5 Psychological formulation of the patient's experience and how the factors were related to each other (taken from Shah and Bewley 2014)

her shame about the abuse and exacerbated the psoriasis. She had difficult relationships throughout life as a consequence, due to a lack of trusting others and an underlying anger towards those she loved. She lived with periods of depression and shame which correlated with the inflammation of psoriasis. Using SFT, the difficulties that she had in her relationships were addressed. Helping her work on her difficult emotions and her interactions within her family and wider system, had an indirect positive effect on her skin (given the link between stress and the skin).

Overview of Talk Therapies

In this chapter, we have looked at various psychological approaches. When thinking about which therapy to offer, a thorough assessment is required and then a psychologist would also create a formulation for the patient (see Fig. 6.6). It is important to consider the impact on the quality of life and daily functioning and the level of distress and risk. The need for more complex psychological therapies is dependent on the severity of psychological distress. Table 6.1 gives an overview of the types of therapies that could be considered for different types of skin conditions.

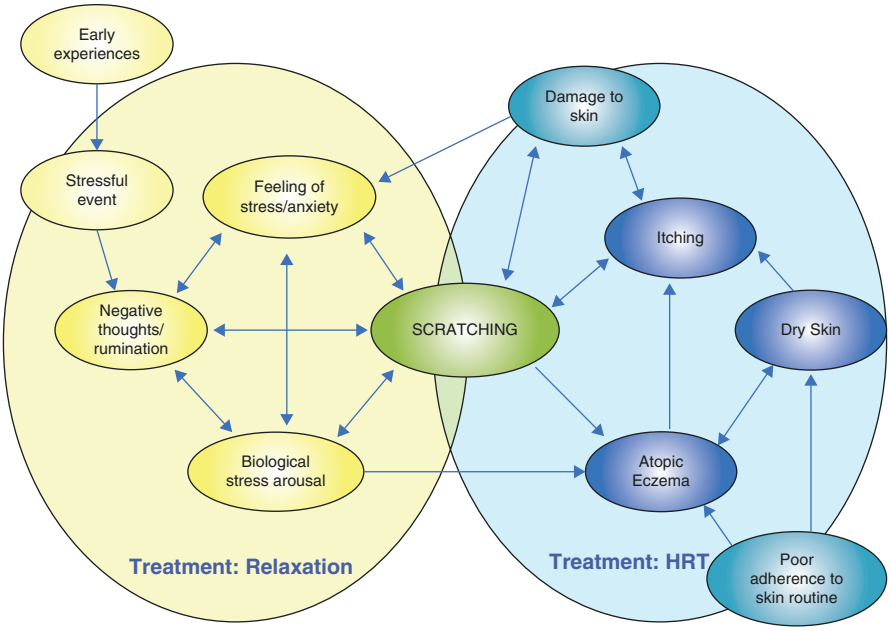


Fig. 6.6 Psychological formulation showing the links between scratching and stress and the mode of therapy used to help the difficulties. Shah and Bewley (2014)

Table 6.1 Overview of different talk therapies and which may be considered for each skin problem grounded on evidence-based and practice-based evidence

	Behavioural therapies	Cognitive behavioural therapy	Systemic family therapy	Schema therapy	Trauma-based therapies
Efficacy (evidence and practice based)	Eczema, psoriasis, vitiligo, acne, nodular prurigo, dermatillomania	Eczema, psoriasis, vitiligo, acne, dermatillomania, trichotillomania, vulvodynia, body dysmorphic disorder, delusional infestation	Psoriasis, eczema, vulvodynia, vitiligo, dermatitis artefacta	Psoriasis, eczema, acne, rosacea, urticaria pigmentosa	Acne excoriee, Dermatillomania, psoriasis, BDD, dermatitis artefacta
Time frame	Here and now symptoms	Here and now symptoms	Relationships, interpersonal issues, here and now symptoms	Here and now symptoms, adjustment issues	Past issues, current relationships
Duration	Short-term	Short-term	Mid-range	Short-term	Long-term

Formulation

Principles

Formulation is a method that Psychologists use to conceptualise the person's problem/s and situation presented to them to help gain an understanding of the individual's needs. Clinical Psychologists are trained in multiple models, which they can draw upon to develop an individual understanding of the patient's predisposing, precipitating, perpetuating, presenting and protective factors. Therefore, therapy is tailored to meet the patient's goals (based on evidence-based and practice-based evidence). This method sometimes requires the therapist to integrate different psychological approaches within the therapy. An example of this is shown below where after the assessment, a formulation was completed and two approaches were used within therapy: habit reversal therapy (HRT in diagram) and relaxation/mindfulness to enhance outcome (Fig. 6.6).

Consequences for Clinical Set Ups

Having a psychologist dedicated to working in dermatology can be helpful and recommended as gold standard as they are able to work with complex histories and problems. The cost and clinical benefits are also undeniable. One UK study (Shah 2018) showed that 86% of patients were discharged from the dermatology service after completing psychological therapy. This was a significant increase in the discharge rate in the service. The results on clinical utility indicated that there was a statistically significant improvement in patients' psychological distress; anxiety, depression, appearance-related concern, and QoL scores. Savings were calculated because patients who were successfully discharged no longer sought dermatological services at the same rate as before they were seeing a psychologist. The cost-benefit analysis showed that for the successful patients, for each year of a 5-year period, the projected savings from not providing additional dermatological services would accumulate at £19,370/year savings to the service. Studies have demonstrated significant cost-savings across a range of psychocutaneous diagnoses, (e.g. Golding et al. 2017).

Practice Points

- When assessing patients for levels of psychological distress, using standardised measures such as the PHQ-9, GAD-7 or DLQI can be helpful, (see Shah 2014 for a list of measures).
- Allow time for psychoeducation: explaining the link between the mind and skin and the importance of considering psychology. This can help to reduce the stigma of mental health, fear of the unknown and the potential high

level of shame about their difficulties. For example, providing a leaflet about how skin disorders can affect emotional wellbeing can be helpful.
- Understanding the benefit of psychological interventions will help with the assessment process to increase the use of psychological strategies in routine practice or to refer on as appropriate.
- Dependent on the severity of psychological distress, look at the stepped care model and where to refer. Psychologists offer consultation to bring a different perspective to complex cases.
- Offer relaxation as a baseline strategy to help reduce stress.

References

Finlay AY, Khan GK. Dermatology life quality index (DLQI): a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994;19:210–6.

Golding GMR, Harper N, Kennedy L, Martin KR. Cost-effectiveness in psychodermatology: a case series. Acta Derm Venereol. 2017;97:663–4.

Hudson MP, Thompson AR, Emmerson L-M. Compassion-focused self-help for psychological distress associated with skin conditions: a randomized feasibility trial. J Psychol Health. 2020;35(9):1095–114.

Kroenke K, Spintzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13.

Lavda AC, Webb TL, Thompson AR. A meta-analysis of the effectiveness of psychological interventions for adults with skin conditions. Br J Dermatol. 2012;167:970–9.

Lilly E, Lu PD, Borovicka JH, Victorson D, Kwasny MJ, West DP, Jundu RV. Development and validation of a vitiligo-specific quality-of-life instrument (VitiQoL). J Am Acad Dermatol. 2013 Jul;69(1):e11–8.

Mizara A, Papadopoulos L, McBride SR. Core beliefs and psychological distress in patients with psoriasis and atopic eczema attending secondary care: the role of schemas in chronic skin disease. Br J Dermatol. 2012;166(5):986–93.

Motley RJ, Finlay AY. Practical use of a disability index in the routine management of acne. Clin Exp Dermatol. 1992;17:1–3.

Panebiano A, Sampogna F, Lemboli ML, Sobrino L, et al. A screening programme for dermatologists as a guide to request psychological consultation in routine clinical practice. Eur J Dermatol. 2018;28(3):326–31.

Shah R. Psychological assessment and interventions for people with skin disease. In: Bewley A, Taylor RE, Reichenberg JS, Magid M, editors. Practical psychodermatology. 1st ed. Chichester: Wiley; 2014.

Shah R. Impact of collaboration between psychologists and dermatologists: UK hospital system example. Int J Women's Dermatol. 2018;4(1):8–11.

Shah R, Bewley A. Psoriasis: 'the badge of shame'. A case report of a psychological intervention to reduce and potentially clear chronic skin disease. Clin Exp Dermatol. 2014;39:600–3.

Shah R, Hunt J, Webb TL, Thompson AR. Starting to develop self-help for social anxiety associated with vitiligo: using clinical significance to measure the potential effectiveness of enhanced psychological self-help. Br J Dermatol. 2014;171(2):332–7.

Spitzer RL, Kroenke K, Williams JB, et al. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166:1092–7.

Cognitive Behavioural Therapy for Skin Conditions

7

Elaine N. Clarke and Andrew R. Thompson

Introduction

What Is CBT?

Cognitive behavioural therapy (CBT) is an umbrella term used to cover a variety of different therapies that focus on alleviating distress by working to change both internal cognitive processes and external behaviours. Like other approaches to psychological intervention, formal CBT psychotherapy requires the application of empathy and other 'non-specific' therapeutic skills such as active listening and the ability to build a therapeutic alliance with the patient. Indeed, CBT has at its heart the goal of the practitioner/therapist working in collaboration with the patient, usually in such a way as to foster both parties making guided discoveries of changeable factors that may be serving to maintain distress. For example, during CBT, the person living with psoriasis and experiencing low mood will explore how their mood interacts with specific thoughts and behaviours. This guided discovery occurs not just during the therapy session or whilst reading the self-help materials provided, but, crucially, also during guided exercises and practices. For example, Fig. 7.1 shows an excerpt from a typical diary used to identify the relationship between thoughts, feelings and behaviours.

E. N. Clarke
Department of Psychology, University of Sheffield, Sheffield, UK

A. R. Thompson (✉)
South Wales Clinical Psychology Doctorate based at Cardiff University, Cardiff, UK
e-mail: thompsona18@cardiff.ac.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_7

Situation Where were you, what was going on?	Emotion What did you feel?	Unhelpful thoughts and thinking patterns –be specific
Saw my psoriasis in the mirror	**anxious**	**What if people at work notice**

Fig. 7.1 Example of a thought record

CBT Protocols

CBT has developed such that there are now evidence-based 'protocols' involving specified techniques and procedures for treating many types of psychological presenting problems or disorders. Such protocols typically incorporate behavioural interventions that draw heavily on learning theory (such as exposure and response prevention) but also use insight-based approaches that focus on drawing an individual's attention to the role played by cognitive and affective factors such as thought content, cognitive processes, attentional biases and emotional states.

CBT and Skin Conditions

For people with skin conditions experiencing psychological distress, a variety of CBT approaches can be used as valuable additions to dermatology treatment. In this chapter, we will briefly outline the impact that skin conditions can have on psychosocial functioning, differentiate between CBT offered at different steps of mental health treatment, and detail how a range of specific CBT-based approaches can be used to treat the psychological distress associated with skin conditions.

CBT Techniques

Whilst some CBT informed 'techniques', such as habit reversal and relaxation can (and should) be incorporated into routine dermatology practice, CBT psychotherapy and the use of in-depth CBT protocols requires delivery by highly specialist accredited practitioners (usually CBT therapists accredited by an internationally recognised professional body or clinical psychologists who have completed an accredited training course). However, in order to be able to refer patients for CBT interventions, it is essential that healthcare clinicians working with dermatology patients are able to assess for the presence of forms of psychological distress that are likely to be amenable to treatment with this approach. Consequently, this chapter will have as a theme running through the 'practice points' tips as to how to identify psychological distress quickly during routine consultations with

dermatology patients. Further information on the assessment of psychological distress associated with dermatological conditions can be viewed for free by UK healthcare practitioners on the Health Education England e-learning portal (https://portal.e-lfh.org.uk/) and can be purchased by practitioners from other countries via integrity (https://www.eintegrity.org/).

The Impact that Skin Conditions Can Have on Psychosocial Functioning

Prevalence of Distress in Skin Conditions

Many people with skin conditions manage extremely well without psychological support: in the UK, around half of the population experience, a skin condition each year and the majority of these people cope well. Nevertheless, around 10–15% of patients with skin conditions experience clinically significant distress. However, objective skin condition severity is not a good predictor of psychological distress: clinician severity ratings tend to be only weakly associated with psychological distress, while patient severity ratings are more strongly associated with distress. It is not uncommon for there to be a poor agreement between clinician and patient assessments of disease severity. These discrepancies suggest that psychological factors are particularly important in the development and maintenance of distress associated with skin conditions.

Practice Point
It is worthwhile establishing the patient's view on the severity of their condition as an initial gauge of their distress: 'How severe do you rate [name of skin condition] on a 1–10 scale?'. If the patient's rating is significantly higher than the clinician's rating, then there be a need to assess further about the impact of the condition.

Impact

The impact of skin conditions can be considerable, for example, psoriasis has been found to have a similar impact on health-related quality of life as diseases such as cancer, arthritis and heart disease. Skin conditions can affect many areas of life, including work/school, leisure, personal relationships and socialising. Some skin conditions cause physical symptoms that are difficult to live with, such as pain, itch or skin flaking. These symptoms can, in turn, cause difficulty in sleeping and tiredness, which is known to be a risk factor for the development of illnesses such as depression and anxiety. Furthermore, some patients may engage in unhelpful coping strategies such as avoidance of exercise or excessive use of alcohol. Indeed, there is some evidence that some skin condition populations have higher levels of

substance use and clearly this has the propensity to adversely affect both the skin condition itself, general physical, and mental health. Treatment of the skin condition can also be problematic, as the treatments may be time-consuming and/or unpleasant. The skin condition can affect the individual's view of themselves and trigger thoughts of being 'unattractive' or worries of being 'rejected' by other people. Regrettably, negative reactions from others, such as staring and negative comments, are not uncommonly experienced by people with skin conditions. As such, stigmatisation poses an additional burden that may require learning and rehearsal of strategies to manage other peoples' reactions and such strategies can be built into CBT treatment protocols.

Practice Point
A significant minority of individuals find that their skin condition negatively affects their wellbeing and psychosocial functioning. Therefore, it is essential to acknowledge and validate this by routinely asking all patients directly about potential psychosocial impacts of their disease e.g. 'It isn't unusual for [name of skin condition] to have an impact on how people feel; how is it affecting you?'

Overlapping Categories of Psychological Distress

The relationship between skin conditions and psychological distress can be classified into three overlapping categories: primary psychological; 'psychophysiologic'; and secondary psychological. CBT can play a role in supporting adjustment in conditions found within all three of these categories. CBT can be the main form of treatment for presentations within the primary psychological category of conditions. In primary psychological conditions, the aetiology of the presenting problem is psychological, for example, as in trichotillomania. There are established CBT protocols that have been developed for treating trichotillomania and typically involve behavioural techniques associated with habit reversal.

In secondary psychological conditions, the aetiology of the complaint is a known skin disease (e.g. vitiligo, nodular prurigo, etc.) yet the presence of the condition can understandably be associated with significant psychological distress. CBT treatment protocols are not well established for the treatment of secondary distress related to skin disease. However, the protocols developed and tested for treating anxiety and depression are highly modifiable and there is emerging evidence that they are effective with dermatology patients.

Some skin conditions can also be considered to have a 'psychophysiologic' element, in which the physical symptoms are exacerbated by inflammation associated with stress, which can, for example, be the case in psoriasis or atopic dermatitis. Indeed, there is evidence that inflammation can play an important role in mental health. There are several well established CBT protocols used for stress reduction that are likely to be beneficial to patients living with skin disease, and relaxation/stress reduction strategies can be easily added into most CBT treatment protocols.

The Different Types of CBT

Background

CBT is a collection of therapeutic approaches that has at its heart an understanding that people do not simply respond to a situation, but rather respond on the basis of both the situation and their interpretation and physical reactions to that situation. Figure 7.2 shows a diagram of the simple 'five areas model' used as a starting point

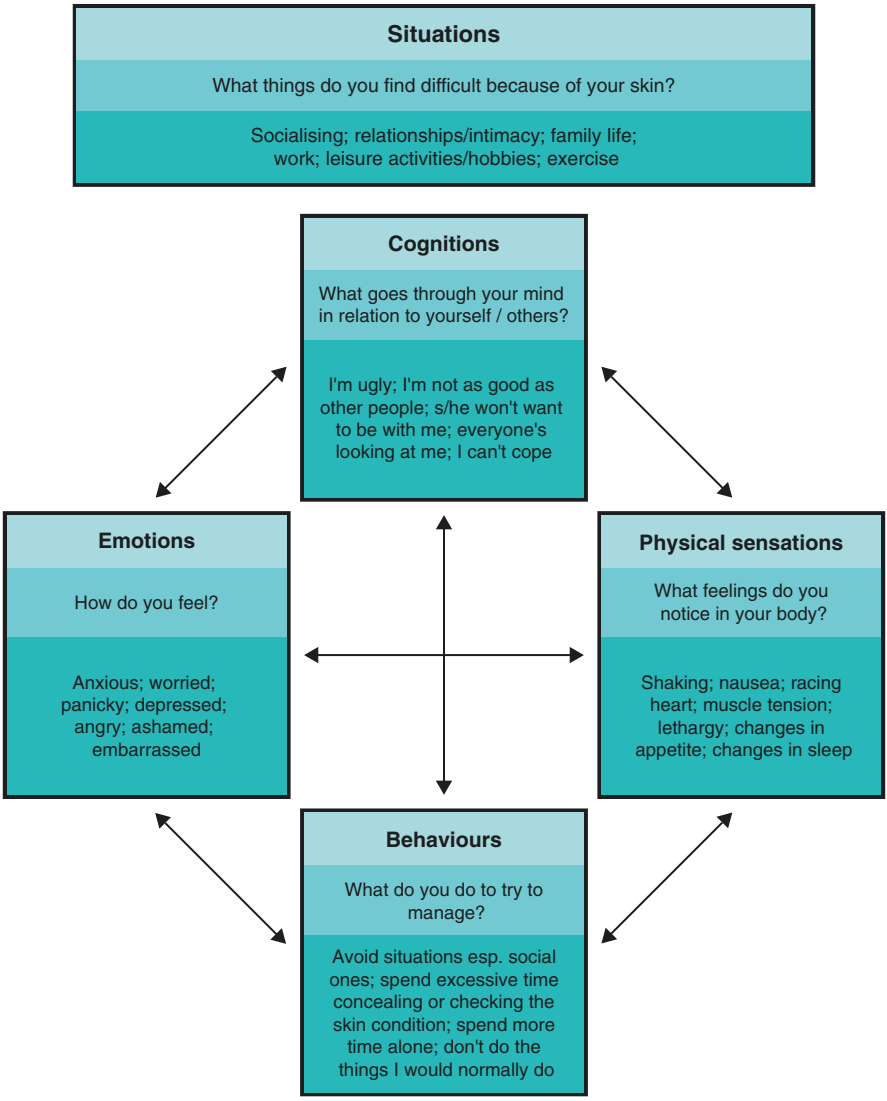


Fig. 7.2 Diagram of CBT five areas model, with example information gathering questions and responses that indicate the potential benefit of a CBT approach

in many CBT protocols. Figure 7.2 includes examples of questions that can be used to reveal information relating to each specific 'area'.

How CBT Works

CBT draws heavily on cognitive neuroscience and learning theory, which has identified how patterns of responding can be learnt and maintained. It seeks to use a guided approach to support patients to recognise for themselves the role played by the different 'areas'. This involves the patient developing an ability to stand back from the presenting problem and to try out new ways of responding. In traditional CBT this typically involves engaging in exposure and carrying out 'behavioural experiments' to experientially test unhelpful thoughts or assumptions and to learn via experience that anticipated adverse consequences do not occur at all or with a much lesser frequency than expected. The patient is also guided to challenge or 'restructure' unhelpful thought content (e.g. 'I'm ugly') and recognise unhelpful cognitive processing associated with specific thought content (for example, labelling oneself as ugly is an example of 'self-criticism' and 'over generalisation').

Practice Point
For some individuals, cognitive (e.g. thoughts and thinking styles) and behavioural (e.g. actions) factors exacerbate the distress associated with the skin condition. Therefore, it can be useful to directly ask patients about the content of their thoughts and investigate how their thoughts might influence their behaviour and mood as shown in Fig. 7.2.

Development of CBT Over Time

CBT has sometimes been described as developing in 'waves', with the first wave occurring in the 1950s and 1960s being focused primarily on techniques drawn from behaviour therapy and learning theory, and the second wave which arose in the 1970s and 1980s being focused on developing protocols that sought to identify and modify cognitive processes and thought content. The most recent 'wave' of CBT has placed emphasis on assisting people to reconnect with the present moment and to learn how to reconnect with values and aspects of their lives that typically become lost in the maelstrom of psychological distress. These so-called 'third wave approaches' include adapted forms of CBT to include mindfulness and Acceptance and Commitment Therapy (ACT). Both these approaches differ from earlier forms of CBT in placing more emphasis on decoupling from cognitive processes and tolerating affect and thought content as opposed to seeking to directly restructure them. They also place a large amount of emphasis on fostering a non-judgemental stance towards oneself and others. ACT also places emphasis on context and behaviour.

Mindfulness-Based Cognitive Therapy

Mindfulness-based cognitive therapy (MBCT) has been trialled with a limited number of skin conditions, that thus far show promising results: reliable and/or clinical changes in social anxiety for treatment completers, and participants rating MBCT as satisfactory and beneficial. However, methodological limitations mean that further research on third-wave CBT interventions for skin conditions is required before they can be included in clinical guidelines. As yet there has only been limited investigation of ACT for treating skin related distress.

CBT Approaches Within Stepped Care

Stepped Care

In some countries, guidelines for the treatment of depression and anxiety disorders follow a stepped care model, in which the least restrictive treatment that is likely to improve mental health is first recommended, and then more intensive treatment is provided as necessary if the patient remains significantly distressed. Figure 7.3 shows how CBT approaches can be used with patients at all steps of the stepped care model.

As mental and physical health problems can adversely affect each other, we recommend that CBT interventions for people with skin conditions acknowledge the presence and impact of the skin condition. At steps one and two, CBT-based interventions would benefit from the inclusion of skin-specific self-help materials. Normalising information, such as reading about examples of other people with skin conditions, can be valuable for reducing shame and promoting engagement in interventions.

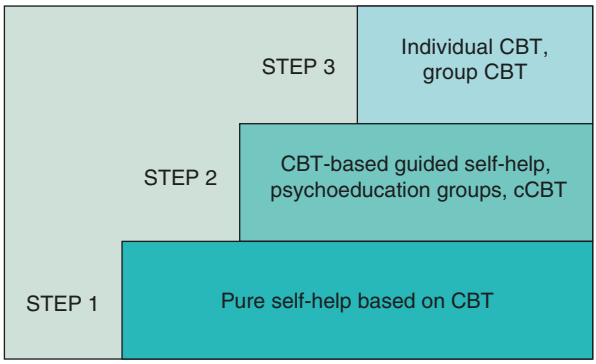


Fig. 7.3 CBT approaches within the stepped care model

Practice Point
CBT-based interventions can be delivered in a variety of formats, depending on the patient's needs and preferences. Some patients may be put off by the idea of receiving 'therapy' or attending 'a group', so it is useful to alert patients to the fact that CBT-based interventions do not solely consist of face-to-face psychotherapy, but can involve having access to self-help materials and e-support resources.

Pure Self-Help Based on CBT

'Step one' is the recognition, assessment and active monitoring of the mental health problem in primary care or an outpatient clinic. The CBT-based interventions at this step consist of 'pure' self-help, where distress has been recognised by a healthcare professional, and the patient has been given relevant self-help information to use independently. CBT-based self-help incorporates CBT formulations to help individuals understand how their thoughts and behaviours may be maintaining distress, and suggests the use of specific CBT techniques to facilitate change. Pure self-help is modestly clinically effective for anxiety and depression, with CBT-based self-help being more effective than educational self-help. There are a plethora of CBT-based self-help books and websites available, but clinicians need to be careful to direct patients to appropriate and reputable resources. Some examples have been provided at the end of this chapter. Several CBT-based self-help interventions for people with skin conditions have been trialled in recent years, although none are yet widely available. CBT-based interventions have been adapted for people with skin conditions with the inclusion of specific CBT techniques for the management of appearance related distress and common symptoms associated with skin disease such as itch. Studies have found some promising results, such as reductions in anxiety, depression, stress, shame, skin complaints and improvements in quality of life. However, further development work is needed before CBT-based self-help interventions for skin conditions are ready to be used in health services. In particular, studies of self-help interventions have reported high attrition rates and/or unexpected null findings on certain outcome measures. This suggests that further development work is needed to improve the acceptability of the interventions. Furthermore, caution must be exercised with self-help interventions as for some individuals, self-help increases awareness of psychological distress but does not provide enough support to make improvements. This is more likely to be the case for individuals whose psychological issues are more severe and/or longstanding.

CBT-Based Guided Self-Help, Psychoeducational Groups, cCBT

Patients who need additional support should be 'stepped up' with a referral (or self-referral) to mental health services. 'Step two' interventions, also known as 'low-intensity' interventions, involve limited contact time with a therapist or qualified mental health practitioner, typically over 6–8 sessions. Low-intensity CBT interventions can be delivered in a variety of formats, such as psycho-education groups, computerised CBT (cCBT), and individual or group 'guided self-help', in which patients receive advice on the use of self-help materials. Guided self-help has been shown to be more effective for depression than pure self-help. Low-intensity interventions are a cost-effective way of delivering treatment to a large number of patients and e-interventions can enable patients to access treatment despite geographic or time restrictions.

High-Intensity CBT

At step three, interventions are described as 'high intensity', as they involve more therapist contact time, typically up to 12–20 weekly sessions, with an accredited cognitive behavioural therapist or clinical psychologist. This form of CBT often involves individually tailoring treatment protocols and additional consideration of earlier experiences (including adverse childhood experiences that may have shaped patients underlying assumptions about the world, themselves, and other people).

Practice Point
Consider using brief screening tools such as the PHQ-2 and GAD-2. Where patients are exhibiting positive signs of distress consider using the full versions of these measures and where people score in the severe range consider referral for high-intensity CBT and consider other medications. For people in the mild range, consider referring to low-intensity CBT.

Signposting Patients to Places Where They Can Gain some Access to CBT and Mindfulness-Based Self-Help Information

Practice Point
Some CBT and mindfulness-based self-help techniques can be found on the following websites. Consider alerting patients to these sites.

- *Skin Support Website*
  Self-help website providing emotional support and information for people living with skin conditions—www.skinsupport.org.uk.
- *Patient Information Leaflets by St John's Institute of Dermatology*
  Leaflets about various skin conditions and dermatology treatments/procedures—https://www.guysandstthomas.nhs.uk/our-services/dermatology/patients/patient-leaflets.aspx.
- *Changing Faces*
  Charity supporting people who live with visible differences has lots of self-help information available—https://www.changingfaces.org.uk/.
- *Self-Help Access in Routine Primary Care (SHARP)*
  Short CBT-based self-help leaflets on a range of subjects including anxiety, depression, stress and physical health problems—https://www.primarycare-self-help.co.uk/.
- *Northumberland, Tyne and Weir NHS Foundation Trust Leaflets*
  CBT-based self-help booklets on a variety of mental health issues and difficult life events—https://web.ntw.nhs.uk/selfhelp/.
- *Mindfulness Resources*
  Free-to-download mindfulness meditation exercises—http://www.freemindfulness.org.

Part II

Specific Illnesses

Dermatitis Artefacta

8

Padma Mohandas

Definition

Dermatitis Artefacta (DA) or artefactual skin disease (ASD) is a rare primary psychiatric disorder that manifests on the skin. The term 'artefact' is derived from the Latin words "arte" (art, handicraft) and "factum" (to make). The condition is a self-induced dermatosis and lesions are produced either consciously or in a dissociated state. It is a poorly understood entity, often presenting to different disciplines before a diagnosis is made. Patients are usually referred to Dermatology services as they (patients and family) are often unable to acknowledge the psychosocial underlay for their physical presentations.

Classification

The current psychiatric nosology in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) refers to DA as a *factitious disorder*, under 'Somatic symptoms and related disorders' (American Psychiatric Association 2013). DA is defined as an induction of injury or disease in the absence of obvious external rewards or gain and with the intention of assuming a sick role.

P. Mohandas (✉)
Bart's Health, London, UK
e-mail: pmohandas@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_8

Epidemiology

DA is rare in routine dermatological clinics and the true incidence of this condition is unknown. However, various studies report values ranging from 0.04 to 1.5% of patients seen in dermatology clinics (Koblenzer 1996; Mohandas and Bewley 2013), though it is seen more frequently in psychodermatological services (1/3000) (Mohandas and Bewley 2013). Female patients out number male presentations by a ratio of 4:1 (Koblenzer 1996; Mohandas and Bewley 2013). DA can present at any age, the youngest reported self-inflicted patient being 8 years old (Alcántara Luna and García Bravo 2015). There is one case report that outlines child abuse by the mother on her infant at 4 weeks of age (Munchausen's by proxy) (Meadow 1977) The peak incidence is in young adulthood age ranges in various studies from 8 to 86 years mean age overall of 31 years (Lyell 1979). We have not found any distinction in socio-economic status or education levels in our patient population.

Etiopathology

The physical manipulation of the skin in DA usually occurs in a dissociated state (Gupta and Gupta 1993). Dissociative states tend to produce behaviour that is impulsive and automatic, whereas the self-injurious behaviour is carried out with full awareness and recollection. DA is less responsive to behaviour directed therapy as opposed to skin picking disorders or trichotillomania, as the latter groups of individuals are aware of their behaviour (Gupta et al. 2017). Dissociative features may also be indicative of more stress and trauma with complex psychiatric comorbidity. Patients may have been exposed to abusive situations which may be physical, emotional or sexual. In such cases, the skin signs may be an indication that the patient is experiencing extreme stress outside their coping capacity and are likely to require psychiatric intervention.

Practice Point
Always ask the patient about abuse; physical, emotional and sexual.

Clinical Presentations

DA can present in a manner of ways; lesions as a rule are found on parts accessible to the patient (Table 8.1). It is worthwhile noting the handedness of the patient as lesions can appear more on one side than the other. The lesions usually have a bizarre appearance, tend to erupt suddenly on previously normal skin, and can occur overnight. Patients are usually vague about their disorder and recount the classic 'hollow history' (Gandy 1953), meaning that they are unable to describe in detail when their lesions appear or how they develop. The lesions may appear or are

Table 8.1 Common sites of artefactual skin lesions

Site
Head and neck—scalp, face
Lower limbs—shins, thighs
Upper limbs—forearms, wrists, upper arms
Torso—chest, breasts, abdomen, shoulders
Genitalia

Table 8.2 Clues to the diagnosis of DA

• Hollow history
• Indifferent patient
• Lesions fully form without precursor lesions
• Sites of lesions may be predicted by patients
• Usually geometric bizarre shaped
• Present in accessible areas, oddly distributed
• Usually present in visible areas
• Suspect historical or current sexual abuse if on breasts or genitalia
• Poorly healing/ recurrent ulceration despite the absence of organic pathology

'discovered', often on waking. Lesions usually appear at an identical stage in development, in crops or groups, more often symmetrically. There is usually a lack of disease progression history. By contrast, there is a prolonged and elaborate description of the complications and the failure to heal. Characteristically, established lesions may undergo sudden deterioration at the same time as new areas appear. Rarely do patients use more than one method to produce lesions. Patients may show a 'belle indifference' to their predicament as part of a dissociative state and manifest a nonchalance transmitted through an enigmatic 'Mona Lisa smile' (Bewley et al. 2014) (Table 8.2). Patients are more often passive than aggressive, even though they have a widespread disfigurement. However, considerable anger is well recognised by parents, carers, spouses or partners, who complain at the incompetence of doctors they have seen in the past. Patients and their relatives may consume huge amounts of medical resources to seek the cause and resolution of the 'problem'. Relatives are usually convinced that the patient is genuinely ill. Official complaints to hospital management have been documented (Bewley et al. 2014).

The most common presentations seen are as follows.

Excoriations These are the commonest and easiest of lesions to identify and are usually inflicted by fingernails, although a variety of implements may be used. Distinguishing DA from neurotic excoriations (e.g. Acne Excoriee) can be challenging, however, patients presenting with the latter usually recognise that they have picked or scratched their skin. Excoriations in DA are not usually preceded by a sensation of itching and are prone to infection.

Ulcers These are also common and appear in varying stages of development due to the method of production, e.g. due to constant picking, application of caustic substances, burns (Fig. 8.1). They may also exhibit an unusual morphology such as linear or other geometric patterns. Look out for the 'drip' sign (caused by splashes from caustic agents). Underlying osteitis or necrosis may also develop in severe cases.

Blisters Another frequently occurring presentation, whereby blisters are produced usually by spraying aerosols at close range (Fig. 8.2). Friction between skin surfaces is another mode of blister production.

Purpura/Bruising These lesions are rarer and organic causes (e.g. bleeding/clotting disorder) must be ruled out before reaching a diagnosis of DA (which may be caused by pinching or suction).

Abscesses These are usually found on the arms, thighs or breast; the history could present as recurrent breast abscesses without underlying pathology alongside failure to respond to surgical therapy (Rodríguez-Pichardo et al. 2010). (Do think about a history of sexual abuse in these patients).

Fig. 8.1 Showing ulceration over the left hand. *Photograph courtesy Dr A Shum, Derby*

Fig. 8.2 Showing blistering over the right arm

Fig. 8.3 Showing pink pigmentation simulating gout. *Photograph courtesy Dr K Warrier Nottingham*

Panniculitis Chronic indolent lesions are more of a challenge. They may present as panniculitis (inflammation of the subcutaneous tissue) which is tender and may necrose to discharge purulent material. This picture can be the result of covert injections of milk, oil and other substances.

Pigmentation on Skin These externally induced marks, usually pink or red, may simulate a condition the patient may be aware of through prior contact or knowledge about the condition (Fig. 8.3).

Common sites of involvement in DA are the arms, hands, abdomen, anterior thighs and breasts. Facial lesions are usually seen on the cheeks, neck and chin. There is usually an absence of involvement in inaccessible areas of the body, for example, the centre of the back. DA should also be considered in non-healing wounds in an otherwise healthy individual. Factitious interference is much more common than genetic immune deficiencies. The growth of unusual bacteria (e.g. faecal bacteria) should also arouse suspicion and can be diagnosed on laboratory

investigations (Table 8.3). Table 8.4 outlines the Psychiatric differential for self-induced lesions.

Practice Point
Perform a thorough examination using a dermatoscope if required to assess skin paying attention to the sites most affected. Patients will appreciate the extra time spent on this simple intervention.

Table 8.3 Differential dermatological diagnosis for DA

• Autoimmune bullous disease (bullous pemphigoid, cicatricial pemphigoid, linear IgA disease
• Porphyria Cutanea Tarda
• Pyoderma Gangrenosum
• Vasculitis
• Connective Tissue disorder
• Cutaneous lymphoma
• Folliculitis (bacterial/ fungal)
• Granulomatous infection (mycobacterial, deep fungal)
• Folliculitis decalvans
• Arthropod bites
• Dermatitis herpetiformis
• Pinch purpura

Table 8.4 Differential psychiatric diagnosis for DA

Condition	Distinguishing features for DA
Self-harm	Patients deliberately cause harm, often with suicidal intent. Intention is not to feign illness but a 'cry for help'.
Self-mutilation	Maybe unintentional seen in patients with severe learning disabilities/ neurological patients.
Skin picking disorders	Damage skin to relieve tension, patients usually admit to this.
Skin damage due to psychosis	Damages skin in response to hallucinations or delusions, e.g. Delusional infestation
Skin damage due to body dysmorphic disorder	Damage to skin secondary to overvalued ideas of perceived imperfection.
Malingering	External motive present e.g. pecuniary gain.
Dermatitis simulata	Individuals (usually children) apply pigments to simulate skin disease.
Dermatological pathomimicry	Individuals aggravate an existing dermatosis, so the disease looks like an exacerbation of an established skin condition with none of the bizarre physical signs.
Dermatitis neglecta	Patients self-neglect and develop a build-up of keratin and debris that form a thick crust.
Munchausen's and Munchausen's by proxy	With Munchausen's the patient repeatedly and deliberately acts as if he or she has a physical or mental illness when not really sick. Munchausen syndrome by proxy is a mental illness and a form of abuse. The caretaker of a child, most often a mother, either makes up fake symptoms or causes real symptoms to make it look like the child is sick.

Rarely full thickness skin loss and severe scarring may require specialist Plastic surgery input, there are even case reports of amputation (Bewley and Taylor 2016).

Table 8.5 outlines the main complications reported in the literature.

Psychopathological Factors and Comorbidities

Physical manipulation of the skin can be mediated by a range of psychiatric conditions from body image issues to Delusion Infestation. Twenty per cent of patients with DA have a somatization disorder and hypochondriasis. These are co-morbid with anxiety, depression and substance abuse. Borderline personality disorder is a factor for chronicity in both sexes (Gandy 1953).

Table 8.6 outlines the psychosocial precipitants commonly observed in patients with DA.

Evaluation and Diagnosis

The establishment of a supportive and non-judgemental physician–patient relationship is paramount to the management of DA. Without a rapport between the patient (parent, in the case of children) and physician, an honest discussion will be challenging. The diagnosis is often not very difficult to make and may be quite obvious on presentation. Nevertheless, it is essential to exclude organic disease.

Table 8.5 Physical complications of DA

• Pigmentation disturbances.
• Scarring.
• Cutaneous infection.
• Osteomyelitis.
• Fistulae.

Table 8.6 Psychosocial factors that may have precipitated skin disorder

Children/adolescent	Bullying, Family upheaval (divorce, separation), Emotional neglect,
	Physical/Sexual abuse, Exam stress, Bereavement, Adoption, Fostering
Adults	See above causes (for events which may have occurred in childhood)
	Relationship breakdown, Bereavement, Financial crisis, Employment
	Issues e.g. bullying, discrimination.
	Social isolation, Depression, Anxiety
	Body dysmorphic disorder, Personality disorders

Investigations

In the vast majority of cases, investigations are unremarkable and do not shed much light on the external aetiology of the skin signs. Organic skin disease should however be ruled out in the first instance (e.g. immunobullous disorder, trigeminal trophic syndrome on facial lesions), which not only helps the clinician but also in some way the patient and their family to understand that there is no worrying primary skin disease which is the problem. It can also help turn the consultation to other factors that can influence skin health such as stress or psychological trauma. There must also be careful clinicopathological co-relation when interpreting histology results. The pitfall of over-diagnosing organic disease when the history and clinical features are suggestive of DA must be kept in mind. Table 8.7 below outlines the main investigations that are performed depending on clinical presentation.

For this and a range of other reasons, the author recommends that dermatitis artefacta is managed in a psychodermatology MDT setting (Bewley et al. 2014). A skin biopsy may provide essential supportive information and help to exclude organic disease (Fig. 8.4).

Depending on the type of injury and the duration of the damage/healing process, there may be a variety of histological patterns, some of which may possibly mimic other skin disorders. It is crucial therefore, that the clinician informs the dermatopathologist about their suspicions (based on history or clinical findings) when sending in biopsy specimens. In general, findings that should prompt consideration of the diagnosis dermatitis artefacta include prominent epidermal damage, multinucleated keratinocytes or deformed keratinocyte nuclei, as well as an inadequately mild inflammatory, infiltrate in early lesions. Absence of the stratum corneum without any substantial inflammatory reaction may also be suggestive of an external skin factitious disorder (Gutierrez et al. 2016).

Occasionally, patients will embed needles completely into their skin to produce recurrent abscesses. If suspected an X-ray will visualise these. Wooden skewers may also be inserted; these are best seen through MRI scans (Gandy 1953).

Table 8.7 Suggested initial investigations for managing DA

Investigations	Request
Skin swab	Microscopy/culture and sensitivity
Blood tests	Full blood count/Renal/Liver / Thyroid function
Clotting screen	Bruising/petechial manifestations
Skin biopsy	Histology/IMF E.g. to rule out bullous disease

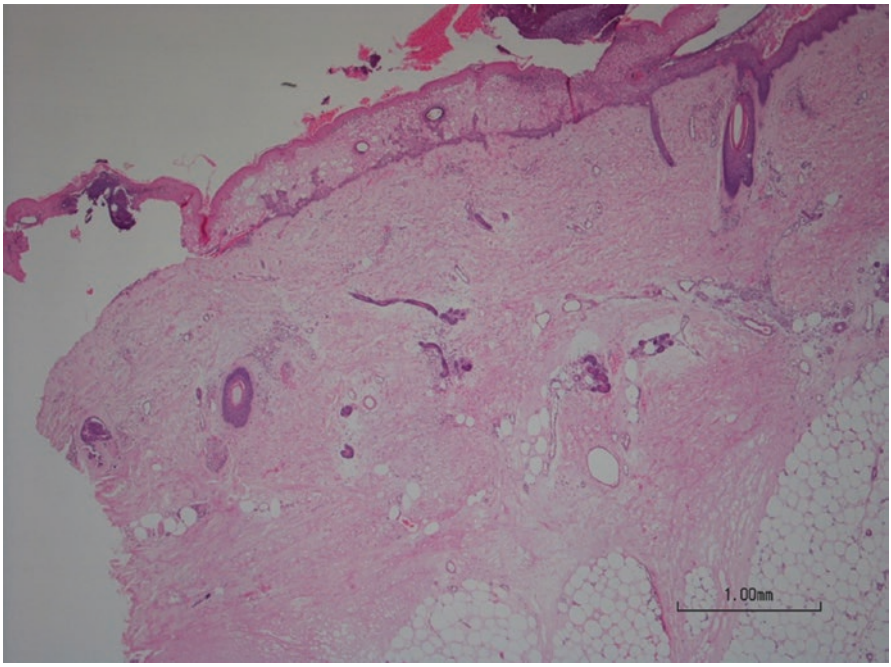


Fig. 8.4 Histology from the patient in Fig. 8.1 showing necrosis of the epithelium with re epithelisation of the basal layer with necrosis of the underlying collagen involving the full thickness of biopsy with focal areas of fat necrosis. Features suggestive of thermal injury. *Image courtesy Dr Rand Hawari, Derby*

Management

There are two main aims of therapy (Fig. 8.5) 1. Addressing the cutaneous component of the problem. 2. The psychosocial aspects of the problem.

1. The skin damage may be extensive, disfiguring and infected, with the potential for scarring and dyspigmentation. As a consequence, the process of tissue repair may take time, but this can be used as an opportunity for patient–clinician relationship building. It is important that the physician has a helpful, non-aggressive, sympathetic approach. For superficial erosions, a combination of bland emollients and short course topical corticosteroids (TCS) may be helpful. If there is clinical evidence of infection, a combination of TCS with antibiotics may also help with the skin symptoms.

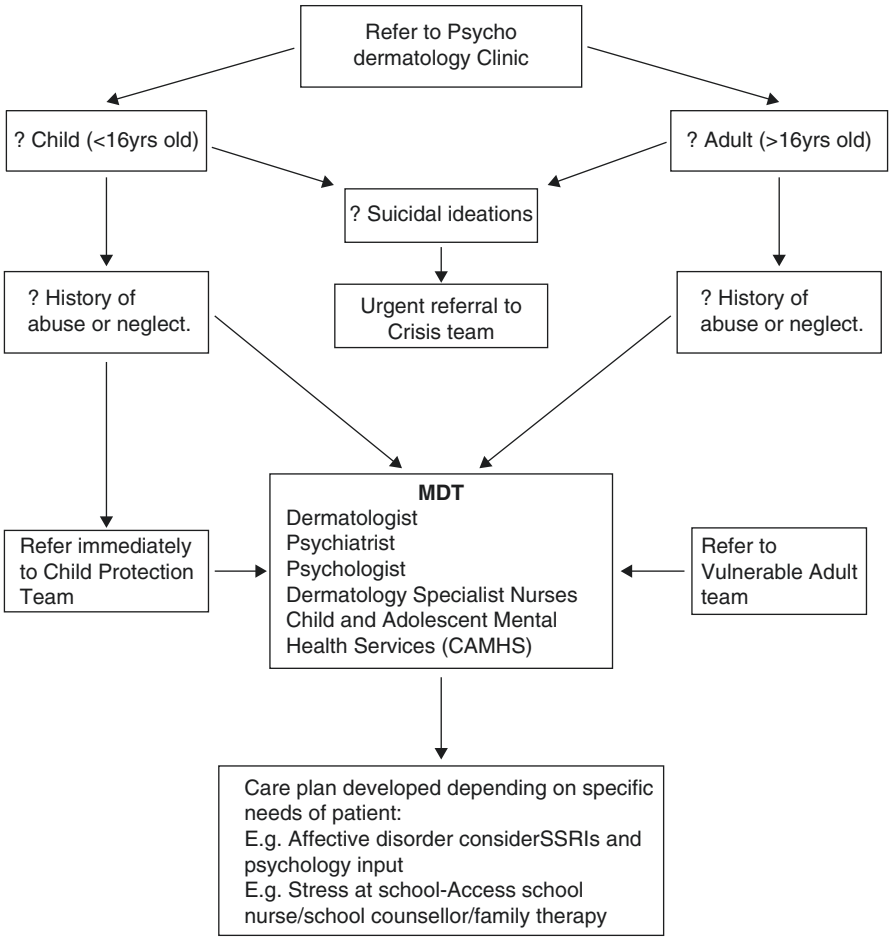


Fig. 8.5 Algorithm for managing DA (Mohandas, Shah and Bewley 2013)

Occlusion may be of benefit if there are deep gouged ulcerations, as not only will this promote healing but it may also physically prevent further external skin damage from taking place. However, patients can be quite contriving and find other ways of producing skin lesions on uncovered sites.

2. Learning about life events/changes in circumstances that surround the appearance of the skin lesions may provide insight into the patient's initial psychological state. Building rapport and trust may not be achieved in a single consultation; therefore, it is important that continuity of care can be provided, and it may take many sessions before a positive change is seen. Evaluating mental health and signposting to the appropriate service must be undertaken (Table 8.7).

   Patients may attend with rather unlikely explanations for their condition; this should be taken as an opportunity to discuss wider issues of the skin and the psyche. A few phrases that may help are outlined below (Table 8.8).

Table 8.8 Consultation strategies for DA patients

Consultation strategies
Diffusing anger and suspicion—'*Shall we start afresh and see how we can help you?'*
Introducing stress as a possible factor—' *It is important to look at all aspects of what has happened or is happening in your life', 'are you able to tell me a bit more about……'*
Taking your time—'*Sometimes it is not obvious as to what exactly causes certain symptoms and it may take time to get better*'.
Introduce dissociation as a possible cause—'*Sometimes people do things that they are completely unaware of for example sleepwalking. Is this something that you think might be happening to you and your skin'?*

DA or Malingering?

Malingering is a term that indicates the production or feigning of a symptom for secondary gain (e.g. financial or social incentives) and can present to any speciality (Bewley and Taylor 2016). In dermatology, patients may intentionally aggravate pre-existing skin diseases or produce skin lesions de novo. Some typical examples are self-inflicted manipulation of prurigo nodularis to avoid issues at school, such as bullying or exams. Malingering must be differentiated from deliberate nonadherence with medical treatments leading to persistence or deterioration of the of disease. Most cases of nonadherence are not hidden from medical professionals. Some cases of hidden nonadherence can however be supported by social incentives and therefore share some characteristics with malingering: for example, a parent who deliberately neglects their child's eczema for pecuniary gains.

Prognosis

The overall prognosis of DA can be divided into that of paediatric patients (generally self-limiting and good prognosis with appropriate support and psychological interventions) and adult patients (maybe more protracted, chronic and recurrent). There is one longitudinal study by Sneddon (Sneddon and Sneddon 1975) who described the cases of 43 patients of which 33 were traced. Around 60% recovered and of those who did, two main factors of disease resolution were identified which still holds good today. Firstly, emotional maturation helped, especially in the paediatric population, secondly resolution of the adverse social situation. Those who had unresolved psychological trauma continued to be symptomatic (Sneddon and Sneddon 1975; Gupta and Gupta 2003).

References

Alcántara Luna S, García Bravo B. Dermatitis artefacta in childhood: a retrospective analysis of 44 patients, 1976–2006. Pediatr Dermatol. 2015;32(5):604–8. https://doi.org/10.1111/pde.12625.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013.

Bewley A, Taylor RE. Psychodermatology and psychocutaneous disease. Rook's Textbook of Dermatology, Ninth Edition. 2016; pp 1–45.
Bewley AP, Taylor R, et al. Practical psychodermatology: dermatitis artefacta. Hoboken: Wiley; 2014.
Gandy DT. The concept and clinical aspects of factitial dermatitis. South Med J. 1953;46(6):551–4.
Gupta MA, Gupta AK. Dermatitis artefacta and sexual abuse. Int J Dermatol. 1993;32:825–6.
Gupta MA, Gupta AK. Psychiatric and psychological co-morbidity in patients with dermatologic disorders: epidemiology and management. Am J Clin Dermatol. 2003;4:833–4.
Gupta MA, Jarosz P, Gupta AK. Posttraumatic stress disorder (PTSD) and the dermatology patient. Clin Dermatol. 2017;35:260–6.
Gutierrez D, Schowalter MK, Piliang MP, Fernandez AP. Epidermal multinucleated keratinocytes: a histopathologic clue to dermatitis artefacta. J Cutan Pathol. 2016;43:880–3.
Koblenzer CS. Neurotic excoriations and dermatitis artefacta. Dermatol Clin. 1996;14:447–55.
Lyell A. Cutaneous artifactual disease. A review, amplified by personal experience. J Am Acad Dermatol. 1979;1:391–407.
Meadow R. Munchausen syndrome by proxy. The hinterland of child abuse. Lancet. 1977;2:343–5.
Mohandas P, Bewley A, Taylor R. Dermatitis artefacta and artefactual skin disease: the need for a pscyho dermatology multidisciplinary team to treat a difficult condition. Br J Dermatol. 2013;169(3):600–6. https://doi.org/10.1111/bjd.12416.
Rodríguez-Pichardo A, Hoffner MV, García-Bravo B, Camacho FM. Dermatitis artefacta of the breast: a retrospective analysis of 27 patients (1976–2006). J Eur Acad Dermatol Venereol. 2010;24:270–4.
Sneddon I, Sneddon J. Self-inflicted injury: a follow-up study of 43 patients. Br Med J. 1975;3(5982):527–30.

Psoriasis

9

Christina George and Anthony Bewley

Aetiology

Psoriasis is a common autoimmune inflammatory condition of the skin, affecting approximately 125 million people worldwide, approximately 2–3% of the total population.

It is a multisystem condition which poses an increased risk of psoriatic arthritis, inflammatory bowel disease, cardiovascular disease, diabetes and lymphoma. It is known to cause substantial disability, akin to those caused by conditions such as cancer, arthritis, heart disease and diabetes. Psoriasis is associated with profound psychosocial comorbidity with a burden that extends well beyond the physical signs and symptoms. Psoriasis not only results in these negative psychosocial consequences, but paradoxically can be exacerbated by them, leading to a complex and destructive negative cycle affecting the skin and mental health.

Psychiatric Comorbidities

Psychosocial comorbidity is common in patients with psoriasis, and the most common of these include anxiety and depression, suicidal ideation and substance misuse.

C. George
Charing Cross Hospital, London, UK

A. Bewley (✉)
Barts Health NHS Trust, London, UK

Queen Mary University of London, London, UK
e-mail: anthony.bewley@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_9

Anxiety and Depression (Fig. 9.1)

There is a significant prevalence of anxiety and depression in patients with psoriasis. Studies have shown that between 10% and 40% of patients experience depression, with a 72% higher prevalence in those with more severe disease. Approximately 31% of patients experience symptoms of anxiety. Psoriasis has been shown to cause greater psychological distress than those with fungal infections and vitiligo. A study by Fortune et al. in 2000 showed that 38% of psoriasis patients have features of pathological worry, and 25% fulfilled the criteria for generalised anxiety disorder. Interestingly, this seems to be irrespective of symptoms or their frequency. Anxiety appears to be related to concerns regarding societal beliefs, and worry that their own anxiety was the main cause of their psoriasis. In women, pathological worry appears to be more prevalent and is not always related to the degree of skin severity.

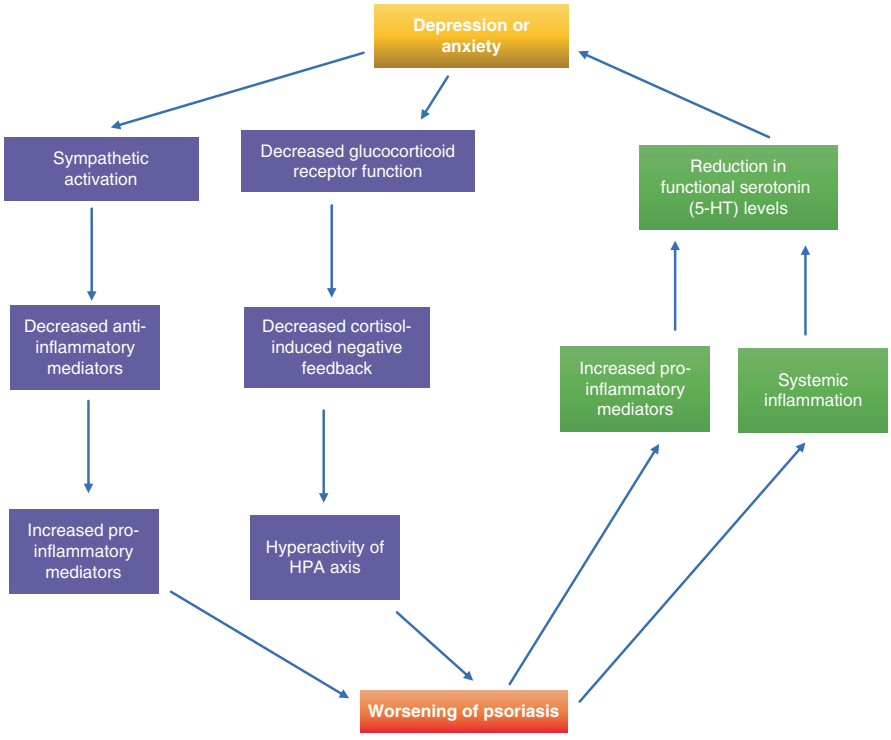


Fig. 9.1 The relationship between psoriasis and depression or anxiety *(from El Sayed et al. 2018). 5-HT* 5-hydroxytryptamine (serotonin), *ACTH* adrenocorticotropic hormone, *CC16* uteroglobin, *CRH* corticotropin-releasing hormone, *HPA* hypothalamic–pituitary–adrenal. https://www.emjreviews.com/dermatology/article/beneath-the-skin-the-relationship-between-psychological-distress-and-the-immune-system-in-patients-with-psoriasis/

The Brain–Skin Axis (Table 9.1)

Whilst it is known that patients with psoriasis may experience mood disturbance as a consequence of the impact on their skin, it is also well recognised that psychological distress can have a negative pathological impact on cutaneous stability, often resulting in worsening of the condition. There is evidence that psychological distress can both drive skin and systemic inflammation as well as being a cause (Table 9.1); in practice, this can result in a vicious cycle of poor skin condition and mental health (Fig. 9.1).

Hypothalamic–Pituitary–Adrenal (HPA) Axis (Fig. 9.2)

The mechanism by which activation of the HPA axis could lead to worsening psoriasis remains yet to be fully elucidated. It is known that stress results in activation of the hypothalamic–pituitary axis (HPA); this causes an upregulation of Corticotrophin-releasing hormone (CRH), resulting in a downstream increase in adrenocorticotropic hormone (ACTH), glucocorticoids and neuropeptide mediators. The effect of the glucocorticoids is to inhibit IL-12, IFN-γ and TNF (via T-Helper 1 cells), and to upregulate IL-4, IL-10 and IL-13 (via T-Helper 2 cells), as well as resulting in a general shift from TH-1 to TH-2 regulated immune profile. CRH itself also stimulates a pro-inflammatory response which is likely to contribute to a cumulative inflammatory effect. In addition, neuropeptides such as Substance-P (SP) and Nerve growth Factor (NF) are involved in communication between the neuronal and immune system. The skin is responsive to the central stress response via a peripheral HPA axis and therefore it is pathophysiologically feasible that a central stress response could result in an effect on the skin.

Paradoxically, the peripheral HPA axis in the skin may directly affect the central HPA axis. IL-1 and IL-6 (upregulated in psoriasis) are known to upregulate the expression of CRH which in turn activate the central stress response, and other pro-inflammatory cytokines are also activated in the skin, all of which result in central HPA activation which could induce symptoms including depression and anxiety.

Sympathetic–Adrenal–Medullary Axis

The sympathetic–adrenal–medullary axis is activated as a fast-response to stressful stimuli. This axis activation results in an increase in catecholamines such as noradrenaline which decrease anti-inflammatory mediators and active pro-inflammatory mediators. CD4+ lymphocytes are activated and lymphocytes are trafficked into the skin. These effects are associated with psoriatic plaque formation.

Brain Changes

Patients with psoriasis have higher rates of depression and anxiety than those with other severe skin conditions, suggesting that there may be physiological brain changes specific to psoriasis which may account for the significant psychosocial morbidity found specifically in this condition.

Table 9.1 The relationship between psychological distress and inflammation

Inflammation causing psychological distress	Psychological distress causing inflammation
Mice models exposed to pro-inflammatory IL-1 have exhibited more symptoms of depression, and those treated with anti-17A were less likely to have these symptoms.	Human studies have shown increased levels of IL-1B, IL-6 and TNF-a following psychological distress Stress is also known to increase levels of pro-inflammatory markers IL-1 and IL-6 in animal models, which correlate with increased symptoms of depression These inflammatory markers have been linked to psoriatic plaque formation
In cancer patients exposed to pro-inflammatory interferon +/− IL-2, there was greater psychological distress, suggesting that these pro-inflammatory cytokines drive psychological distress.	Hypothalamic–Pituitary Axis (HPA) hyperactivity, resulting in higher levels of Corticotropin-Releasing Hormone (CRH) is commonly seen in depression. CRH stimulates pro-inflammatory cytokines (IL-6 and IL-11). Psoriatic plaques express higher levels of CRH than in unaffected skin, suggesting a possible causal association.
Inflammation can lead to a reduced level of functional serotonin, increases its breakdown and inhibits serotonin receptors. This powerful combination of actions could explain the reason why depression in these cases can be resistant to treatment.	HPA hyperactivity seen in depression results in higher baseline cortisol levels; however, this results in a saturation of the anti-inflammatory mineralocorticoid receptors (which exhibit their action via a negative feedback loop), resulting in a reduced ability to regulate corticosteroid levels. This effect results in desensitisation to the anti-inflammatory effects of cortisol. Patients with psoriasis who identify stress as a trigger for their skin to flare, show blunted cortisol levels in response to stress, which may be a consequence of this desensitisation. This leads to increased CRH via a negative feedback loop, resulting in pro-inflammatory effects.
Treatment of inflammation, for example, in patients taking anti-TNF treatment, has shown a significant improvement in depression when compared to placebo, irrespective of clinical severity. This finding suggests that treating systemic inflammation is important in treating the psychological distress, and thus may indicate a causal factor.	Psoriasis patients have been shown to have higher levels of noradrenaline in response to stressful situations. Noradrenaline is pro-inflammatory via IL-6 and TNF-a which could explain why psoriasis flares in response to stress.
Treatment of psoriasis with anti-IL-23 and IL-12 biologics has also shown a significant improvement in symptoms of depression	In a group of patients whose psoriasis was felt to flare due to stress, those whose depression responded to tricyclic antidepressant medication, exhibited a reduction in pro-inflammatory mediators such as IL-6, TNF-a and IL-1B. Similar findings have been demonstrated in those treated with other classes of antidepressant medication.

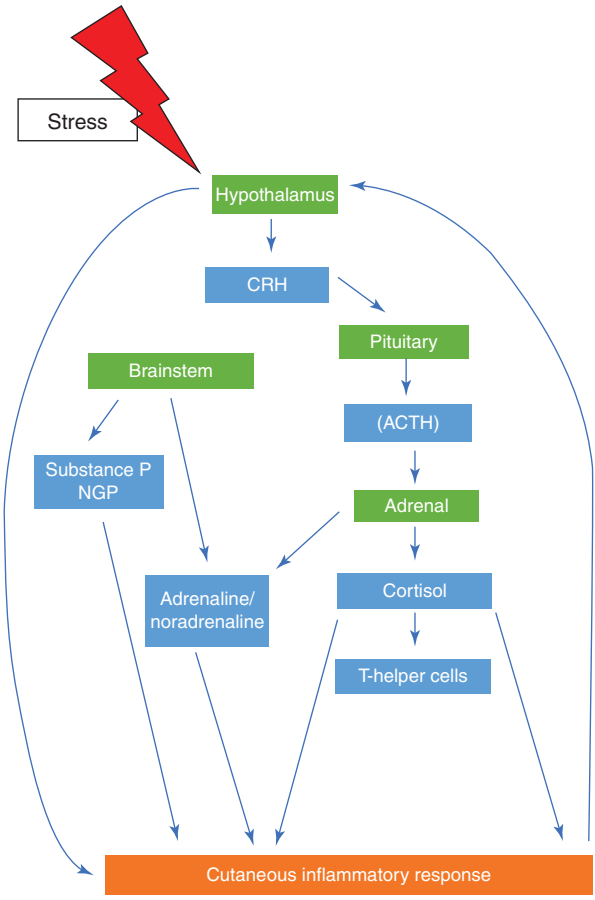


Fig. 9.2 The brain–skin axis. *ACTH* adrenocorticotropic hormone, *NGF* nerve growth factor, *SP* substance-P, *Th 0-2* T-helper: 0-2 cells, *CRH* corticotropin-releasing hormone, ? upregulation of CRH by the cutaneous inflammatory response

Research performed by Kleyn et al. using functional magnetic resonance imaging (MRI) studies have shown brain changes which are specific to patients with psoriasis. In one study, 26 male patients (13 with psoriasis and 13 controls) were shown images of disgusted faces. Those with psoriasis had significantly less activity in the bilateral insular cortex (which is known to be activated in response to the feeling and observation of disgust); the authors hypothesise that this may reflect a learned coping mechanism to self-protect from exposure to disgusted facial expressions. Interestingly, more recent work by this group investigating potential neuroinflammatory changes using positron emission tomography (PET) scanning found no significant differences in neuroinflammatory signals between psoriasis patients and controls. This may suggest that the brain is protected from the effects of peripheral inflammation via the blood-brain barrier, or it might be that this mode of imaging was not sensitive enough to pick up inflammatory changes. This is a relatively new and unknown area of interest and remains the subject of ongoing research.

Suicide

Despite a scarcity of high-quality evidence, patients with psoriasis seem to have a higher risk of suicidal ideation, and of attempts and completed suicide.

One study has shown up to a 44% higher rate of suicide compared with the general population. In those with severe disease, there is reported to be a 69% greater likelihood of attempting suicide, and a 30% greater likelihood of completing suicide, despite controlling for possible confounders.

Alcohol and Smoking Misuse

There is a high rate of alcoholism and alcohol misuse in patients with psoriasis, and this appears to positively correlate with the severity of psoriasis. The reasons for this are many but in part are seen to be a coping mechanism for dealing with the distressing psychosocial effects of this condition. In a large cohort of psoriasis patients who were admitted to hospital in Finland between 1973 and 1995, there was a higher than expected mortality rate in the psoriasis cohort, and the highest rates were alcohol related.

Alcohol can have a direct effect on the skin, with higher intake resulting in greater severity, and abstinence resulting in improvement and sometimes even remission. Excess alcohol can also complicate suitability for systemic treatment and adherence to treatment, and therefore patients should be encouraged to limit their intake to healthy quantities.

Smoking has been reported to be twice as prevalent in psoriasis patients compared to controls, and there appears to be an increased standardised mortality ratio in alcohol and smoking-related causes. One study has shown a trend towards a higher risk of psoriasis in current smokers and drinkers, and a higher still trend in those with a higher usage.

Practice Point
The prevalence of psychosocial comorbidities in patients with psoriasis is high. The reasons for this are complex and may be due to organic physiological brain and hormonal changes, as well as due to the effect of the condition itself. Clinicians should actively seek out signs of psychosocial comorbidity when treating these patients.

Quality of Life

Many patients with psoriasis report a significant impact on their quality of life. The World Health Organization (WHO) describes the quality of life as *'an individual's perception of their position in life in the context of culture and value systems in*

which they live and in relation to their goals, expectations, standards and concerns … It is a concept affected by the person's physical health, psychological state, personal beliefs, social relationships and relationship to salient features in their environment.'

Physical Factors (Table 9.2)

The physical factors in psoriasis can have a severely detrimental effect on the quality of life, with studies reporting that two-thirds of patients feel the negative physical impact of psoriasis in their everyday lives. This rises to up to 80% in those with severe disease.

The physical symptoms of their skin are a significant factor in the negative effects on quality of life, and these include itching, pain, irritation and the functional inability of the hands and feet.

In addition, the physical impact of co-existing psoriatic arthritis has the potential to cause devastating effects on quality of life. Other associated comorbidities such as obesity, metabolic syndrome and autoimmune conditions such as Crohn's disease may all result in physical and consequent psychosocial morbidity.

Pruritus

Pruritus remains an under-recognised symptom in psoriasis, yet its prevalence and effect are substantial. Studies report that between 67% and 77% of patients with psoriasis have symptoms of pruritus which are significant and arise on a daily basis, and 92% have had pruritus at some point. Interestingly the magnitude of pruritus does not appear to always correlate to clinical severity. It is exacerbated by heat, skin dryness, sweating and importantly, stress. There is a known association between pruritus in psoriasis and the risk of depression, again leading to a negative cycle of worsening psoriasis and mental health. In a survey of 104 patients with psoriasis, 30% of patients reported pruritus to be the worst physical factor, a symptom that is often under-estimated in this condition.

Table 9.2 Physical symptoms of psoriasis

Skin symptoms: • Itching • Skin shedding • Tightness • Redness • Dryness • Bleeding • Pain
Functional impairment: • Self-care • Activities of daily living • Occupational factors
Sexual dysfunction
Sleep disturbance

Functional Impairment

Functional impairment in psoriasis is common, and particularly seen when it affects the palms and soles; the consequent physical disability from pain results in higher levels of functional impairment. This in addition to nail involvement has been shown to limit the ability to self-care and perform basic activities of daily living. These restrictions result in psychological distress and isolation.

These physical factors can have significant sequelae, ranging from the inability to carry out simple activities of daily living, through to occupational difficulties which can be so severe as to render patients unable to work. These effects can exacerbate the condition and can lead to social isolation and a downward spiral of psychological distress and worsening of the skin.

Sexual Dysfunction

Sexual dysfunction in psoriasis is common, and psoriasis is reported to interfere with sexual relations in 35–50% patients. This appears to be more prevalent in female patients and can manifest in a number of ways. The physical involvement of the genital skin can make sexual intercourse painful or uncomfortable. A large study of 354 patients revealed that 39% patients experienced pain, 42% dyspareunia and 32% worsening of genital psoriasis after intercourse. The psychological effect of the skin being affected, not only the genital skin but generalised psoriasis, can make it difficult for patients to enter relationships, due to self-consciousness or fear of stigma. This is another way in which social isolation can ensue resulting in mental health decline. In addition, psoriasis causes a decrease in libido in a large proportion of patients. Those who report sexual dysfunction as a result of psoriasis have more symptoms of depression. It appears that psoriasis has a profoundly negative impact on sexual health and satisfaction.

Sleep Disturbance

Sleep disturbance is common and variable in psoriasis, with reports ranging from 5.9% to 44.8% prevalence. The reasons for insomnia include innate disturbance in thermoregulation due to psoriasis, physical symptoms of the condition which themselves cause poor sleep, the negative psychiatric sequelae in which sleep is often disturbed, and finally the higher prevalence of comorbid conditions.

The skin has an important role in mediating core body temperature and acts as a primary circadian mediator to reduce this temperature at night as part of normal sleep initiation. The normal and physiological reduction in core body temperature occurs due to a drop in metabolic heat generation, increase in blood flow to the skin and distal vascular dilatation; these result in the dissipation of heat and increase in transepidermal water loss. In psoriasis, thermoregulation via the skin is impaired, and therefore sleep initiation may be compromised as a result.

Cutaneous symptoms including pruritus and pain are well recognised in psoriasis (see section “Physical Factors”), and pruritus is often said to be worse towards the

end of the day. This symptom is also regulated by circadian mechanisms and the threshold for symptoms is lower in the evening due in part to a reduction in cortisol levels, increase in temperature and reduced epidermal barrier function. This therefore manifests as an exacerbation of cutaneous symptoms at night which cause disturbed sleep.

Many of the associated comorbid conditions can also result in sleep disturbance; for example, there is a higher prevalence of obstructive sleep apnoea in psoriasis, with studies reporting 36%–81.8% in psoriasis, compared with 2%–4% in the general population. There is also a known increased prevalence of restless leg syndrome (15%–18% in psoriasis patients compared with 5%–10% in the unaffected population). The increased prevalence of psychiatric comorbidity (see section "Psychiatric Comorbidities") is also a significant contributor to problems with sleep.

Psychosocial Factors

The psychosocial aspect of psoriasis has been reported by patients to be one of the worse aspects of their condition, resulting in a severely negative impact on the quality of life (see the section on *Psychiatric Comorbidities)*. The extent to which this occurs differs widely, and does not always correlate with the extent of disease. The psychosocial implications are varied and include negative emotional effects on the self, as well as impacting their interactions with their close and wider social network.

Psychological Factors and Schemas (Table 9.3)

The profound psychological impact of psoriasis is well recognised, and the role of distress in the onset, exacerbation and persistence of the condition is also well established. The common and recurrent patient reported themes in studies include negative effects on self-confidence, feelings of shame, embarrassment and a lack of self-esteem. In a large study of 217 patients, over 50% reported feeling self-conscious around strangers. Research has shown that patients with psoriasis use anticipatory and avoidance behaviours as a coping mechanism.

Schemas are now being recognised as an important part of this psychological sequelae. These are engrained cognitive and emotional patterns which influence the

Table 9.3 Schema in psoriasis

Early maladaptive schemas in psoriasis (Mizara et al. 2012)
Emotional deprivation
Social isolation
Defectiveness
Failure
Vulnerability to harm
Subjugation
Emotional inhibition

individual's approach to life; the early maladaptive schemas (EMS) are those which originate in childhood and develop in adulthood. Schemas are particularly difficult to challenge as they are deeply held beliefs that are consolidated through repeated and often self-fulfilling experiences. A number of these schemas have been described in psoriasis (Table 9.3). EMS are strongly predictive of psychological distress. In particular *vulnerability to harm* and *defectiveness* is predictive of anxiety and *social isolation* and *vulnerability to harm* are predictive of depression.

Interestingly, patient's beliefs about the negative effects of living with psoriasis affect their ability to cope with their condition. This is further complicated by evidence that patient symptoms are more severe when they believe their skin is unsightly, have worries about being excluded, or have feelings of low self-worth.

Social Factors (Table 9.4)

Psoriasis affects many patient's ability to function to their best potential in social environments. This is largely a consequence of the psychological effects of this condition. The fear of stigma plays a large part in this (see below). This results in withdrawal from relationships with family and friends, intimate relationships, interactions with the general public, and can have detrimental effects on study and work. Numerous studies have shown that patients with psoriasis try to hide their psoriasis, and many report avoiding social activities that involve showing their skin such as swimming, with one study quoting that 83% of patients would 'often' or 'always' avoid these situations. Social functioning appears to be more severely affected in psoriasis than in other chronic conditions such as hypertension and arthritis, reflecting the visible nature and stigma associated with this condition.

The ability to work and study can also be severely impacted by psoriasis; of a large survey of 369 patients with psoriasis in the UK, one third attributed not working to their psoriasis, and two studies have shown a lower rate of employment in those with severe psoriasis. In patients with severe psoriasis, up to 26 days working days per year were lost as a result of their disease. Over 17% of 18–54-year-old patients with psoriasis report a psychological impact of psoriasis on their work, and 23% reported that their psoriasis had an impact on the choice of their career. In those who do work, over half report that the quality of their work life is negatively impacted as a result of their psoriasis (Table 9.4).

Table 9.4 Psychosocial impact of psoriasis on quality of life

Negative psychological effects on the patient: • Self-image • Self-esteem • Self-wellbeing • Early maladaptive schemas
Negative effects on social functioning: • Relationships with friends and family • Sexual relations • Day to day encounters with the general public • Occupational effects

Table 9.5 QOL questionnaires

Quality of life indices	Specificity	Areas analysed	Comments
Salford Psoriasis Index (SPI)	Disease-specific	• Clinical extent of psoriasis (PASI score) • Psychosocial disability • Past severity (based on treatment history)	• Physician reported
Dermatology Life Quality Index (DLQI)	Dermatology-specific	• General quality of life in relation to the skin	• Patient reported • Most commonly utilised as a treatment target
Hospital Anxiety and Depression Scale (HADS)	General	• Anxiety and depression	• Patient reported • Provides separate scores for anxiety and depression

Quality of Life Questionnaires (Table 9.5)

Objective measures of quality of life (QOL) are important when assessing psoriasis patients, as the high prevalence of alexithymia (see section "Alexithymia") may render it difficult for clinicians to ascertain the extent of the psychological impact of the disease. As results are not infrequently higher than expected, and not always proportional to the severity of their skin disease, measuring the extent of skin disease is not an accurate surrogate for assessing the quality of life.

High scores on quality of life assessments should prompt the assessing clinician to consider whether the patient may benefit from psychological intervention. Importantly for patients on systemic and biologic therapy, QOL represents important end-points in assessing treatment response and the results of direct therapy.

Quality of life questionnaires in dermatology are generally categorised as general health and dermatology-specific and disease-specific questionnaires. General health questionnaires aim to assess the overall physical and psychosocial factors. Skin-specific questionnaires can be more helpful, and efforts have been made to devise psoriasis-specific questionnaires to generate more relevant and meaningful information; these can be used in conjunction with general health questionnaires to provide a better understanding of the disease impact. Examples of some of the most commonly used questionnaires in psoriasis are listed above (Table 9.5).

Stigma (Table 9.6)

Stigma is defined as '*a mark of disgrace which sets people apart from each other*'.

Many patients with psoriasis report experiencing stigma as a result of their skin, which can have a profound effect on their social interactions and general quality of life. This effect is most pronounced in the 18–45 year old age bracket, correlating with the age in which people are most likely to be socially and professionally active.

Table 9.6 Components of stigmatisation (Ginsburg and Link 1989)

Anticipation of rejection
Feelings of being flawed
Sensitivity to others' attitudes
Guilt and shame
Secretiveness
Positive attitudes

The visible nature of their condition renders patients exposed and vulnerable to external perception and misconceptions. Many patients report experiences of being publicly rejected due to a public belief that the condition is contagious, or simply due to fear or lack of knowledge. The result of this on the patient, are feelings of shame and lack of self-worth, with consequent avoidance, isolation and social withdrawal. In a large study of patients with moderate to severe psoriasis, one quarter reported an episode where someone 'had made a conscious effort not to touch them'. Those with publicly visible affected skin perceive their condition to be more disabling and have higher levels of self-reported physical morbidity.

Ginsburg et al. identified six dimensions to stigmatisation (Table 9.6). There appears to be a significant variation in the frequency with which these feelings are experienced, and contradictory feelings could be experienced simultaneously. The group also investigated predictors for the components of stigma experienced. They found that age of onset, bleeding, employment, duration of experience and rejection were the strongest predictors of stigma. Of these, bleeding was the most strongly predicting factor and correlated highly with stigma. Stigma was also associated with poor adherence to treatment and worsening of psoriasis.

Alexithymia

Alexithymia is the difficulty in identifying, expressing and describing one's feelings.

An observational study measuring alexithymia using the validated Toronto Alexithymia Scale in a large cohort of psoriasis patients showed a 24.8% prevalence in this group (compared to approximately 5–10% in the general population). These patients had more severe disease, significantly reduced quality of life, greater prevalence of anxiety and depression, a higher rate of alcohol dependence, and reduced work productivity. Alexithymia can make it difficult for clinicians to ascertain the true effect of the patient's psoriasis on their life.

Practice Point

There are many external factors that can both cause and result in the worsening of psoriasis; these result in a profound effect on the quality of life. Patient's perceptions of their condition or symptoms do not always correlate with the objective skin severity, and therefore clinicians need to be proactive in enquiring about the psychosocial effects and quality of life.

Treatment

As has been demonstrated, psoriasis is a complex condition with a significant psychological overlay (Fig. 9.3). Therefore, just simply treating the skin is not always sufficient; often a more holistic approach, including a focus on psychological health, is required in order to successfully manage these patients (Table 9.7).

Due to the chronic and relapsing nature of the condition, and the fact that many patients have been undertreated for years, it can be difficult for clinicians to encourage patient adherence and positivity to treatment.

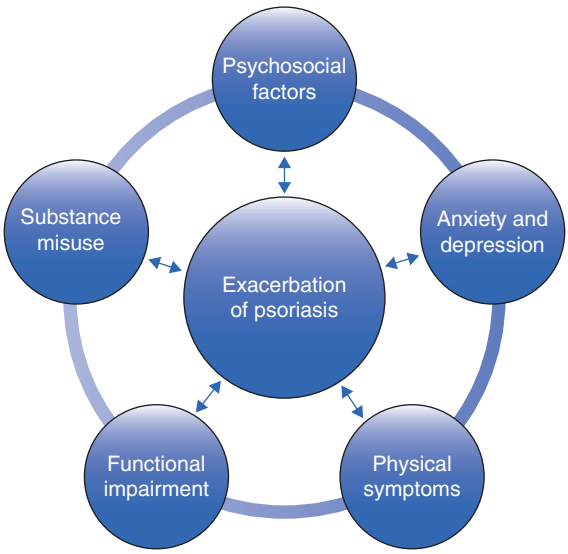


Fig. 9.3 Factors leading to exacerbation of psoriasis

Table 9.7 Treatment options

Treatment	Pros	Cons
Skin directed therapy	• Essential component of treatment approach • Clinical improvement may improve psychosocial function	• Adherence can be variable when there are complex coexistent psychosocial issues • Clinicians may focus on this and neglect holistic approach
Cognitive behavioural therapy	• Can be helpful in combination with standard treatment • Particularly useful to interrupt learnt negative behaviours	• Requires patient and time commitment • Not always accessible
Psychotropic therapy	• Can be a useful adjunct to standard therapy	• Can cause a paradoxical flare of psoriasis

Treatment of the Skin

Treatment of the skin is generally instigated in a stepwise approach and should be tailored to the individual patient depending on the extent of disease, severity and effect on the quality of life. This involves topical treatments, phototherapy, systemic and biologic agents. Further details are outside the scope of this book. There is plenty of evidence from every day clinical practice that when the skin is treated, patients are generally more satisfied and have an improved quality of life. However as mentioned previously, this is not always a predictable response and sometimes quality of life measures reveal that the patient may still be suffering from significant psychological morbidity despite an improvement in their physical health.

Cognitive Behavioural Therapy (CBT)

CBT is a psychological intervention that involves identifying and challenging unhelpful thoughts and behaviours, and learning competing coping mechanisms in order to break the negative cycle. It is well established that stress and distress are frequent exacerbators of psoriasis, but this recognition can also cause patient anxiety which can perpetuate a worsening of their physiological and psychological state. CBT aims to break this cycle. There is evidence that just 6 weeks of weekly CBT sessions combined with standard treatment, versus standard treatment alone, have a significant improvement in the clinical severity of the skin, and improves symptoms of anxiety, depression, stress and disability. In one study, these results persisted at the 6-month follow-up, with 64% of patients achieving a greater than 75% improvement in the clinical extent of their psoriasis, compared with 23% in the control group. Other evidence suggests that CBT is effective at improving anxiety levels but less effective at treating depression. Another study has shown that just seven psychotherapy sessions delivered over 12-weeks resulted in clinical improvement although the perception of stress remained similar. Promising results have also been demonstrated using an internet-based electronic CBT intervention, with an improvement in anxiety and quality of life.

Psychotropic Medication

Psychotropic medication includes any medication which affects the mind, emotions or behaviour. There is a scarcity of high-level evidence for the use of psychotropic medication in psoriasis; however, identifying and treating comorbid psychiatric diagnoses is anecdotally known to be beneficial. In one double-blind placebo-controlled study of 60 patients with psoriasis, patients were randomised to a Moclobemide (a monoamine oxidase inhibitor antidepressant) plus topical corticosteroids, or to topical corticosteroids alone. Those treated with the antidepressant and topical corticosteroids showed improvements in the clinical severity of psoriasis as well as well as depression and anxiety. Another small observational study of

38 psoriasis patients treated with anti-TNFα treatment compared concurrent treatment with Escitalopram (a selective serotonin reuptake inhibitor antidepressant) and psychotherapeutic treatment, compared with psychotherapeutic treatment alone; those treated with Escitalopram plus psychotherapeutic treatment had greater improvements in the clinical severity of their skin, as well as greater reduction in symptoms of anxiety and depression.

Clinicians should however be aware that there are reports of psychotropic medication resulting in flaring or inducing psoriasis, and these include but are not limited to lithium (a well-recognised culprit), fluoxetine (several case reports), and bupropion.

Practice Point

Effective treatment of psoriasis requires a holistic approach to the physical and psychosocial aspects, in order to maximise patient adherence and chance of efficacy.

Bibliography

Elsayed M, Connor CJ. Beneath the Skin: The relationship between psychological distress and the immune system in patients with psoriasis. Dermatology. 2018.

Fortune DG, Richards HL, Main CJ, Griffiths CEM. Pathological worrying, illness perceptions and disease severity in patients with psoriasis. Br J Health Psychol. 2000;5:71–82.

Fortune DG, Richards HL, Griffiths CE, Main CJ. Psychological stress, distress and disability in patients with psoriasis: consensus and variation in the contribution of illness perceptions, coping and alexithymia. Br J Clin Psychol. 2002;41(2):157–74.

Ginsburg IH, Link BG. Feelings of stigmatization in patients with psoriasis. J Am Acad Dermatol. 1989;20(1):53–63.

Gupta MA, Gupta AK. Psychiatric and psychological co-morbidity in patients with dermatologic disorders. Am J Clin Dermatol. 2003;4(12):833–42.

Kurd S, Troxel A, Crits-Christoph P, Gelfand J. The risk of depression, anxiety, and suicidality in patients with psoriasis. Arch Dermatol. 2010;146(8):891–5.

Mizara A, Papadopoulos L, McBride SR. Core beliefs and psychological distress in patients with psoriasis and atopic eczema attending secondary care: the role of schemas in chronic skin disease. Br J Dermatol. 2012;166(5):986–93.

Moon HS, Mizara A, McBride SR. Psoriasis and psycho-dermatology. Dermatol Ther. 2013;3(2):117–30.

Rapp SR, Feldman SR, Exum ML, Fleischer AB Jr, Reboussin DM. Psoriasis causes as much disability as other major medical diseases. J Am Acad Dermatol. 1999;41(3):401–7.

Richards HL, Fortune DG, Griffiths CE, Main CJ. The contribution of perceptions of stigmatisation to disability in patients with psoriasis. J Psychosom Res. 2001;50(1):11–5.

Vos T, Allen C, Arora M, Barber RM, Bhutta ZA, Brown A, Carter A, Casey DC, Charlson FJ, Chen AZ, Coggeshall M. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016;388(10053):1545–602.

Eczema and Psychodermatology

10

Alia Ahmed

Definition of Illness

Atopic eczema is a chronic itchy inflammatory skin condition that occurs mostly in childhood, but can persist into adult life (Darsow et al. 2005; Williams 2005). Impaired epidermal barrier function and an inflammatory infiltrate cause intense pruritus, skin lesions, xerosis and lichenification (Suarez et al. 2012; De Benedetto et al. 2009; Solomon and Beerman 1966). The diagnostic criteria for children are summarised (The UK Refinement of Hanifin and Rajka's diagnostic criteria of atopic dermatitis/eczema):

- An itchy skin condition in the last 12 months

  Plus three or more of:

- Onset below age 2[1]
- History of flexural involvement
- History of a generally dry skin
- Personal history of other atopic disease[2]
- Visible flexural dermatitis as per photographic protocol

A disturbed epidermal barrier leads to increased transepidermal water loss (leading to skin dryness), hyper-irritability, altered sweat delivery and increased

[1] Not used in children under 4 years.

[2] In children aged under 4 years, history of atopic disease in a first degree relative may be included.

A. Ahmed (✉)
King Edward VII Hospital, Frimley Health Foundation Trust, Windsor, UK

Royal London Hospital, Barts Health NHS Trust, London, UK
e-mail: dr.alia.ahmed@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_10

susceptibility to infection (O'Regan et al. 2009). The pathogenesis is multifactorial, including genetic (especially loss-of-function filaggrin mutations resulting in a lack of essential structural proteins making up the epidermis; present in 10% of the western population and 40% of children with moderate to severe eczema) (O'Regan et al. 2009; Palmer et al. 2006; Smith et al. 2006; Paternoster et al. 2012), immunological (through penetration of allergens across a dysfunctional epidermis) (Fallon et al. 2009) and environmental factors (e.g. socioeconomic status, lifestyle factors) (Pastar et al. 2005; Weidinger et al. 2008; Williams 1995). Other environmental factors that contribute to eczema include water hardness, protease-containing detergents and soaps (Cork et al. 2006; McNally et al. 1998; Sherriff et al. 2002).

Classification

1.1 The main classification—there is a lack of consensus on terms used to categorise or phenotype the clinical presentations of eczema. Broadly, eczema can be exogenous (i.e. caused by extrinsic factors) or endogenous (i.e. immunologic). Exogenous eczema often improves on the removal of the exacerbating factor, whereas endogenous causes can have a more chronic course. There are also unclassified presentations.

1.2 Subtypes

Exogenous	Endogenous	Unclassified
Contact dermatitis (irritant and allergic)	Atopic eczema	Lichen simplex chronicus or neurodermatitis
Photodermatitis	Pompholyx or dyshidrotic eczema	Juvenile plantar dermatosis
Phytophotodermatitis	Seborrhoeic dermatitis	
	Discoid or nummular eczema	
	Stasis dermatitis	
	Asteatotic	
	Hand eczema	
	Nodular prurigo	
	Pityriasis alba	

1.3 Historical names—other terms that are synonymous with eczema include atopic dermatitis, dermatitis, neurodermatitis, Besniers prurigo (Williams 2000; Johansson et al. 2004).

Clinical Presentation

General Symptoms

Pruritic skin lesions are the over-riding symptom of atopic eczema. The intense itch is followed by scratching (itch-scratch cycle), which causes damage to the skin barrier and maintains inflammation, leading to chronic changes, such as

lichenification (Lebovidge et al. 2016). The itch-scratch cycle can become difficult to break and may require habit reversal therapy. Itch can also cause sleep disruption, leading to fatigue and irritability. Changes in temperature can aggravate the rash, as can emotional distress, physical exercise and certain clothing (Arden-Jones et al. 2016). Food allergies are present in 35% of infants and children with moderate to severe eczema (Nowak-Węgrzyn and Groetch 2015). Signs of failure to thrive (short stature, low weight, falling off the predicted height and weight centiles) in children on a restricted diet should be monitored. The atopic march describes the development of atopic dermatitis in infancy, followed by allergic rhinitis and asthma in childhood (Bantz et al. 2014). A dysfunctional skin barrier increases the chance of infection through bacterial colonisation and facilitates allergen sensitisation (Boguniewicz and Leung 2011; De Marchi et al. 2015). Environmental allergens like house dust mites and food proteins penetrate the superficial dermis to cause immune sensitisation via antigen-presenting cells, the Th2 pathway is activated, leading to allergic nasal response and airway hypersensitivity, thus exacerbating eczema and leading to food allergies and asthma (Fallon et al. 2009; Bantz et al. 2014; Ersser et al. 2014).

Dermatological Symptoms

Features of the rash include erythema, pruritus, induration, papules/papulovesicular lesions, excoriation and lichenification. The rash can be intermittent. Other associated signs are hyperlinearity of skin, post-inflammatory changes (hypo- or hyperpigmentation), follicular hyperkeratosis (keratosis pilaris), crusting or exudate (indicative of secondary infection) and dry skin (Brown and Reynolds 2006). The patient or their family may also have a history of other atopic conditions (e.g. asthma, allergic rhinitis). The distribution of the rash varies with age, infants (<2 years old) present with an erythematous exudative rash typically on the cheeks, scalp and forehead, this spreads to involve the trunk and extensors, sparing the nappy area (Tareen 2013). In childhood (2–11 years old) the rash localises to flexural surfaces, there may be associated hand or lip dermatitis as well as periorbital symptoms (Tareen 2013). Over the age of 12 years, the rash can be widespread involving the face, neck, trunk, dorsum of feet and hands, and flexures with evidence of lichenification (Tareen 2013). There may be elements of photosensitivity in adulthood (Arden-Jones et al. 2016). People with eczema can describe the sudden deterioration of symptoms, otherwise known as a ‘flare’. During a flare patients will experience increased itching, erythema and induration, larger body surface area (BSA) involvement, and signs of secondary cutaneous infection (e.g. weeping, crusting or blistering).

Psychiatric Symptoms

Psychological/psychiatric comorbidities associated with atopic eczema are listed. Baseline assessments are recommended to identify psychological issues (e.g.

Dermatology Life Quality Index (Finlay and Khan 1994), Hospital Anxiety and Depression Scale (Zigmond and Snaith 1983)).

- Depression (Hashiro and Okumura 1997; Slatter et al. 2009)
- Anxiety (Zigmond and Snaith 1983)
- Social anxiety (Tareen 2013)
- Reduced quality of life (Tareen 2013)
- Psychosomatic symptoms (Finlay and Khan 1994)
- Behavioural problems (Lien et al. 2010)
- Internalising behaviours (panic, anxiety, dizziness, tense, sad, sleeplessness, worthless, blaming oneself, hopeless, burden) (Hashiro and Okumura 1997)
- Conduct problems (Schmitt et al. 2010)
- Emotional problems (Slatter et al. 2009)
- ADHD (Slatter et al. 2009)
- Personality traits (hypersensitive, aggressive, inferiority, insecurity, mood lability, cognitive rigidity, difficulty dealing with anger, hostility in interpersonal relationships) (Tareen 2013)

Systemic Symptoms

Other atopic signs that can accompany the presentation of atopic eczema include conjunctival irritation, keratoconjunctivitis, rhinitis, wheezing and gastrointestinal upset. Dietary and nutritional concerns may lead to failure to thrive in infants, especially if accompanied by genuine food allergies.

Why Is This so Debilitating?

People with eczema report high levels of psychological distress and anxiety (Thompson and Kent 2001; Stangier and Ehlers 2000). Psychological stress is well known as a trigger and aggravating factor for up to 70% of patients with eczema (Morren et al. 1994; Kilpelainen et al. 2002). Poor quality of life and stigmatisation related to atopic dermatitis further impact the perception of stress (Kemp 1999; Schmid-Ott et al. 1999; Kiebert et al. 2002). Stress is a trigger of itch and flares in patients with eczema (Suarez et al. 2012; Oh et al. 2010; Schmid-Ott et al. 2001; Buske-Kirschbaum et al. 2002). Eczema is implicated in negatively affecting the quality of life of both patients and families, more than that associated with other inflammatory conditions such as acne and psoriasis (Lebovidge et al. 2016; Lewis-Jones and Finlay 1995).

The link between emotional stress and acute inflammation can be explained at a biochemical level via the Hypothalamic–Pituitary–Adrenal (HPA) axis. Stress activates this major neuroendocrine system which causes activation and dysregulation of cellular processes that cause or drive skin disease (Kim et al. 2013).

The HPA axis is controlled by a negative feedback system, with corticotropin-releasing hormone (CRH) acting as the principal regulator of the stress response

(Buske-Kirschbaum et al. 2002; Lin et al. 2017). Stress initiates the production of CRH in the hypothalamus, which in turn acts on the anterior pituitary gland to promote the secretion of adrenocorticotrophin (ACTH) resulting in the rapid production of glucocorticoids (e.g. cortisol) by the adrenal glands (Buske-Kirschbaum et al. 2002). Elevated glucocorticoid levels cause tissue damage in the body by driving inflammation, as well as have significant effects on skin barrier function (Buske-Kirschbaum et al. 2002; Lewis-Jones and Finlay 1995). See Fig. 10.1 below.

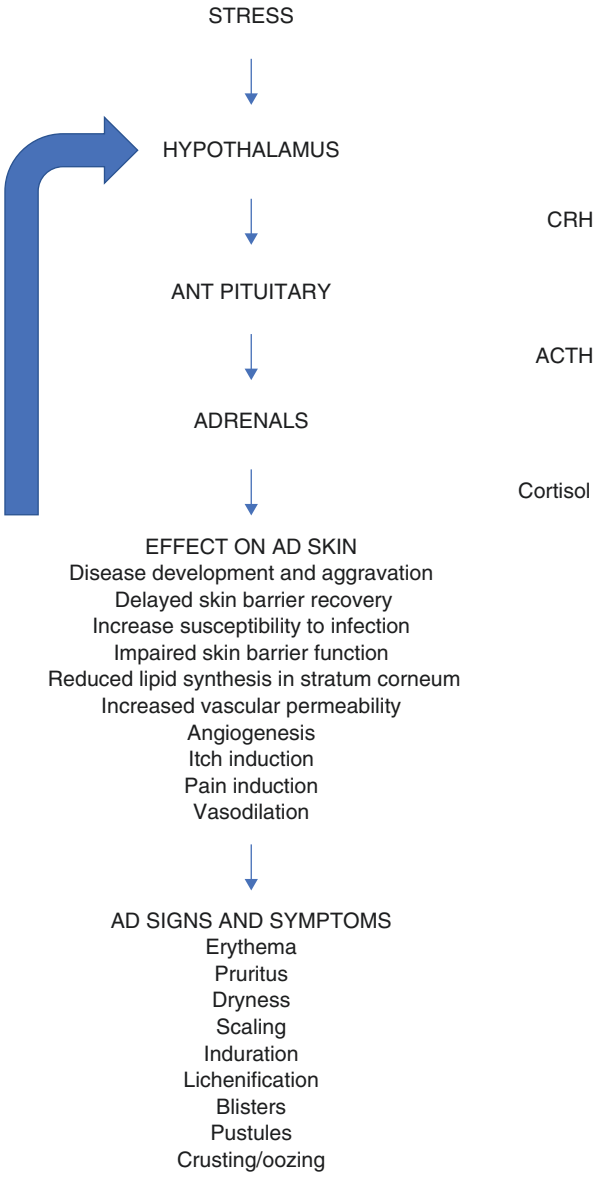


Fig. 10.1 Adreno-hypothalamic-ptuitary axis in patients with atopic dermatitis

Condition	Effect of psychological stress
Atopic dermatitis	Delayed skin barrier recovery (Suarez et al. 2012; Garg et al. 2001) Inhibit lipid synthesis and lamellar body secretion in stratum corneum (Choi et al. 2005) Inhibit antimicrobial peptide production (Aberg et al. 2007) Increase susceptibility to severe skin infection (Garg et al. 2001) IL 4,6,10,13 release from keratinocytes and mast cells (Arck and Paus 2006) Lesion initiation and aggravation (Amano et al. 2008) Increased pruritus (Lee et al. 2006)

Patterns of behaviour (or 'schemas') start forming in childhood and can be adversely affected by life experiences, especially if a person has a chronic skin condition like eczema (Mizara and McBride 2012; Absolon et al. 1997; Zuberbier et al. 2006; Paller et al. 2002). There is evidence to suggest that people with eczema develop maladaptive schemas, that make them vulnerable to negative psychological outcomes (e.g. anxiety and depression) (Mizara and McBride 2012). Identified maladaptive schemas are (Mizara and McBride 2012):

- Emotional deprivation
- Social isolation
- Defectiveness
- Failure
- Dependence
- Vulnerability to harm
- Subjugation
- Insufficient self-control

Childhood behavioural problems are also seen in people with eczema (Absolon et al. 1997), it is possible that having a chronic skin conditions leads to issues with attachment and developing psychological problems in adulthood (Mizara and McBride 2012). A meta-analysis has shown that children and young people with eczema are at higher risk of developing mental health disorders (65.2% more likely) (Xie et al. 2019).

Atopic eczema causes cumulative life course impairment, negatively impacting qualify of life (e.g. employment; personal relationships, and mental health) (Schmid-Ott et al. 2001; Ibler and Jemec 2013; Camfferman et al. 2013; Fishbein et al. 2015; Meltzer and Mindell 2014; Beebe 2011; Chamlin et al. 2004; Alanne et al. 2011; Dahl et al. 1995; Reuveni et al. 1999; Bender et al. 2003). Important factors affecting the quality of life are:

- Sleep disturbance
- Itch
- Socioeconomic cost (Herd 2002)

There is a role for psychological interventions, such as habit reversal and cognitive-behavioural therapy (CBT), in conjunction with medical management of eczema (Ehlers et al. 1995; Bridgett 2014). Options for supporting patients with eczema include patient support groups (e.g. National Eczema Society), online habit reversal resources (e.g. www.atopicskindisease.com), and referral to allied health-care professionals for those struggling despite optimal management.

Epidemiology

No global trend has been established with regard to the epidemiology of children and adults with atopic eczema (Deckers et al. 2012). Atopic eczema is the most common inflammatory condition in children (20% of under 5 year-olds) (Williams et al. 2008; Asher et al. 2006), but less reported in adulthood (3.3%) (Muto et al. 2003), although adults seem to have more chronic and severe disease (Herd et al. 1996). Up to 70% of reported cases are in the under-5 age group (Hanifin and Reed 2007), and the number is rising (two- to threefold increase in prevalence) (Schram et al. 2010). This is suggested to be due to environmental factors, as illustrated by the difference in prevalence between the same ethnic group in rural and urban areas (Muto et al. 2003).

Diagnostic Process

When diagnosing eczema consider:

- Risk Factors
  - Age less than 5 years—associated with more severe disease and the atopic march (Carlsten et al. 2013)
  - Family history of eczema—high concordance in monozygotic twins (77%) (Meagher et al. 2002), sibling prevalence reported as 22–24% (Schultz Larsen et al. 1996)
  - Allergic rhinitis—reported in 50–80% of children with eczema (Spergel and Paller 2003; Paller and Mancini 2016)
  - Asthma
  - Family history of atopy
  - Exposure to cigarette smoke—both active and passive smoke are associated with increased eczema prevalence (Kantor et al. 2016)
- History of pruritus
- Presence of xerosis
- Distribution (e.g. infants show involvement of the cheeks, forehead, scalp, extensors, sparing of the nappy area; children show flexural involvement; chronic eczema can be widespread, involving the neck, trunk and limbs)

- Presence of other cutaneous signs (e.g. erythema, scaling, vesicles, papules, keratosis pilaris, excoriation, lichenification, hypopigmentation)

Eczema is a clinical diagnosis; however some investigations can be useful:

- Serum IgE—may be elevated; however this is a non-diagnostic test.
- Calcium and vitamin D levels—may be reduced in those children with cow's milk protein allergy.
- Allergy testing—common allergens are house dust mite, pollens, grasses, moulds, animal dander and the major food groups (cow's milk protein, soya, fish, eggs, nuts). Food allergy testing should be based on careful history including history of acute reactions and cutaneous response to dietary restriction. True IgE-mediated allergy should prompt removal of the allergen (Bath-Hextall et al. 2009).

Patch testing—may be indicated in difficult to control eczema with a history of exacerbation by exogenous factors.

Differential diagnoses include:

- Seborrhoeic dermatitis—greasy scales affecting the head and neck area predominantly, but can be widespread, nappy area involvement
- Psoriasis—well-demarcated erythematous plaques with a silvery scale, predilection for extensor surfaces
- Irritant contact dermatitis—dry scaly rash, distribution dependent on contact, but in children may include the nappy area, face and extensor surfaces
- Allergic contact dermatitis—well-demarcated erythematous rash with associated papulovesicular lesions and weeping or crusting, may be asymmetric depending on the area of contact
- Scabies—intensely pruritic rash, tends to affect the interdigital area and groin, but can be widespread, there may be a contact history, the rash can be papulovesicular, burrows are visible with dermoscopy, microscopy can aid diagnosis
- Mycosis fungoides/Cutaneous T Cell Lymphoma—well-demarcated erythematous, scaly rash, tends to affect adults >50 years old, skin biopsy is needed to confirm diagnosis

Treatment

Treatment of eczema requires an integrated and comprehensive approach to disease management. First-line treatment is with emollients and topical corticosteroids (TCS). Options for second-line treatment include topical calcineurin inhibitors and phototherapy. Third line treatment includes oral or injectable immunosuppressants.

- *Emollients*

  Emollients are used to hydrate the skin, reduce itch and rebuild the skin barrier. They should be applied liberally and regularly, especially as they have a corticosteroid-sparing effect (Grimalt et al. 2007). The application of emollients should be in the direction of hair growth to avoid folliculitis/occlusive symptoms. Ceramide containing emollients can be helpful for pruritus (Simpson and Dutronc 2011). Types of emollients are:

  Lotion—lighter formulation with higher water content, use for very mild symptoms.

  Cream—more greasy formulation, can be used any time.

  Ointment—very greasy, best used at night or for very dry areas.

- *Anti-Inflammatories/Anti-Pruritics*
  - Topical Corticosteroids (TCS)

    Treat eczema by reducing inflammation and pruritus (Hebert and Nguyen 2019). They should be used during flares to settle acute symptoms, and in some patients are required to maintain long-term control (e.g. twice a week on consecutive nights). The potency of TCS vary, mild to moderate potency is most suitable for sensitive areas (face, neck, genital), moderate to potent TCS are indicated for trunk and limb involvement. Commonly used examples include:

    Low potency—Hydrocortisone 0.5%, 1%, 2%

    Mid-potency—Clobetasone butyrate

    High-potency—Mometasone furoate 0.1%, Betamethasone valerate

    Very high-potency—Clobetasol propionate 0.05%

    During acute flares the use of TCS is indicated, once adequate control has been achieved, the use can be tapered. For example, once nightly use for 1 week (or the amount of time it takes to control symptoms), then alternate nights for 1 week, then twice a week on consecutive nights if required to maintain control. Chronic eczema may require intermittent TCS use, for example, twice a week, this can be accompanied by the concurrent use of non-steroid containing topical treatments (e.g. calcineurin inhibitors).

    Cutaneous side effects of over-use of TCS include atrophy, striae, hypopigmentation and telangiectasias (Bath-Hextall et al. 2009). Systemic side effects are rare but include adrenal suppression, reduced bone density and linear growth and Cushing's syndrome (Bath-Hextall et al. 2009). If the skin is no longer responding to TCS therapy then consider switching to a different TCS as the patient may have developed tachyphylaxis (Bath-Hextall et al. 2009).
  - Topical Calcineurin Inhibitors (TCIs)

    Treat eczema via an immunosuppressive effect. TCIs inhibit T cells, thereby reducing the action of pro-inflammatory cytokines (Carr et al. 2014). Examples of TCIs include:

    Pimecrolimus 1% cream

    Tacrolimus 0.03% ointment

Tacrolimus 0.1% ointment

TCIs are indicated in the management of eczema as maintenance treatment (e.g. three times a week) to control symptoms and reduce the incidence of flares.

During acute flares, the use of topical calcineurin inhibitors can be increased to daily application if tolerated, then reduced to maintenance use for chronic eczema.

Side effects of treatment include burning/stinging at the site of application (tends to resolve after 1 week of repeated application, advise patients to apply to a small area initially), worsening of local herpetic or bacterial infection (advise patients to avoid if active signs of infection). Although there has been concern about the long-term safety and risk of malignancy with the use of topical immunosuppressants by the US Food and Drug Administration (FDA), a randomised controlled trial over 5-years has supported the safety of topical pimecrolimus (Sigurgeirsson et al. 2015).

- *Phototherapy*

  Phototherapy (including UVA1, UVAB, Narrow-band and Psoralen + UVA) can be utilised for eczema that is not responding to topical treatment. It treats eczema through immunosuppression and anti-inflammatory action. There is however lack of robust data evaluating the use of phototherapy in eczema. Adverse effects of phototherapy include acute flare of eczema, sunburn-like reactions, herpetic reactivation (through local immunosuppression), increased risk of skin malignancy.

- *Immunosuppression*

  Systemic immunosuppression is indicated for patients with treatment-refractory eczema. Patients for immunosuppressive therapy require appropriate screens for latent infections (e.g. HIV, Hepatitis B and C, tuberculosis), previous varicella-zoster virus infection (ascertain through clinical history or VZV immunoglobulin G test) and other treatment specific tests (e.g. procollagen-3-N-terminal peptide, thiopurine methyltransferase). The only licensed oral treatment for eczema is ciclosporin; however, others have been used in clinical practice with good effect. Examples include:

  Methotrexate

  Azathioprine

  Mycophenolate mofetil

  The most recent advance in eczema treatment is the use of injectable biologic therapy. There is evidence for the use of Dupilumab, a recombinant human monoclonal antibody that blocks interleukin 4 and 13, both of which are implicated in the pathogenesis of eczema. Dupilumab is approved for the treatment of moderate to severe eczema and has shown favourable outcomes in clinical trials (Beck et al. 2014; Thaçi et al. 2016; Blauvelt et al. 2017; Fleming and Drucker 2018; Kraft and Worm 2017).

- *Oral Corticosteroids*

  In the presence of signs of a flare that is not managed by high to very high-potency TCS, a weaning course of oral corticosteroids can be helpful to control disease in the short-term (e.g. 30 mg daily for a week, then reduce by 5 mg per week).
- *Antibacterial Measures*

  The use of antibacterials should be considered in patients with recurrent flares or acutely infected eczema. Bacterial colonisation can be treated with diluted bleach baths and topical antibiotics. Nasal treatment is indicated for those patients who have a positive nasal swab (most commonly colonised with *Staphylococcus aureus*) (Gong et al. 2006; Dickman et al. 2012; Anderson and Dinulos 2009; Huang et al. 2011). If signs of acute infection are present (e.g. fever, tachycardia, hypotension, deterioration in eczema) oral antibiotic treatment is indicated. Local hospital policies can direct antibiotic choice and duration of use.
- *Exacerbating Factors*

  Minimising or eliminating exacerbating factors can have positive outcomes for patients with eczema.
  - House dust mite—avoid carpets, use dust mite protective bedding, minimise clutter in the immediate environment
  - Grass and tree pollens—dry clothes inside when possible, during high pollen season take antihistamines
  - Animal dander—avoid in the immediate environment (e.g. bedroom)
  - Humidity—address humidity in the immediate environment
  - Extremes of temperature—heat and cold weather can aggravate eczema and necessary precautions should be taken
  - Food allergies—true allergens should be avoided in the diet. Re-introduction of food groups under the supervision of a dietitian can be helpful
  - Irritants—soaps and detergents can aggravate pre-existing eczema
  - Occupational factors—minimise occupational hazards like contact allergens or irritants
  - Clothing—certain clothing should be avoided (e.g. wool)
  - Stress—recognise and minimise stressors (e.g. mindfulness, meditation)
- *Allergies*

  Allergy plans for significant allergies are recommended and may require referral to an allergist. Injectable adrenaline pens are indicated for those patients that have reported significant allergic symptoms (e.g. loss of consciousness, vomiting, breathing difficulty, angioedema).
- *Sleep*

  Issues with sleep are common in patients with eczema. It is important to assess sleep disruption (e.g. difficulty with sleep onset, frequent wakening, feeling tired during the day, signs of itching during the night) and where necessary advise management options including improved sleep hygiene (e.g. establishing

a bed-time routine, having a set time to sleep, relaxation to encourage sleep onset) and sedating antihistamines (reduce night waking and promote sleep onset) (Lio et al. 2014).

- *Itch-Scratch Cycle*

  People with eczema are prone to entering the itch-scratch cycle, which perpetuates and aggravates lesional skin. Addressing the itch-scratch cycle is important in the ongoing management of eczema, especially in those patients who have chronic diseases. Habit reversal therapy is a useful intervention to help identify triggers for itch and teaches alternative behaviours rather than scratching and damaging the skin.

- *Treatment Adherence*

  Treatment of eczema can be complex and there are several adherence issues that have been identified. It is important to educate the patient and their carers on the diagnosis and treatment of eczema. There is a lack of understanding of topical treatments and their application (e.g. how to apply, difficulties with applying treatment, time-intensive) (Beattie and Lewis-Jones 2003). It can also be difficult in some cases to apply treatments correctly, for example, children can lack co-operation, which is frustrating for parents/carers (Fishbein et al. 2015; Santer et al. 2013). In addition, parental concern about the side effects of TCS can result in inadequate treatment (Mizara and McBride 2012; Aubert-Wastiaux et al. 2011). Dietary restrictions due to true food allergies can be difficult to manage without proper guidance, thus leading to exposure to potential exacerbating factors (Bollinger et al. 2006; Springston et al. 2010).

- *Other Adjunctive Treatments*

  Depending on the clinical history and comorbidities, people with eczema may benefit from additional treatments:

  – Antihistamines—sedating antihistamines reduce sleep disruption; second-generation antihistamines can also reduce pruritus in adults with eczema (Kawakami et al. 2006)
  – Antidepressants—indicated in those patients that report clinically significant mood disturbance. Antidepressants can improve coping mechanisms in people with eczema
  – Anxiolytics—symptoms of anxiety can trigger or aggravate existing eczema. By treating anxiety effectively these symptoms can be minimised and consequently improve cutaneous symptoms
  – Combined treatment—indicated for patients with symptoms of both depression and anxiety. Selective serotonin reuptake inhibitors (SSRIs) would be a reasonable choice for the combination treatment of mood and anxiety disorders

- *Multidisciplinary Teams (MDTs)*

  The MDT may contain dermatologists, nurses, psychologists and dietitians, the combined efforts of which can address biological, psychological, dietary and behavioural factors associated with eczema. MDTs can facilitate education and improve knowledge about eczema, promote adherence and self-management,

and facilitate individualised treatment (Barbarot et al. 2013). The involvement of the MDT can also help to identify and address bullying in vulnerable individuals (Lebovidge et al. 2016).

- *Educational Interventions*

  There is evidence for structured parental education interventions in the management of eczema (Grillo et al. 2006; Moore et al. 2009; Niebel et al. 2000; Kupfer et al. 2010). Similarly, there is also some evidence for the use of patient education and cognitive-behavioural therapy in eczema treatment (Ehlers et al. 1995).
- *Psychological Interventions*

  There is some evidence for the use of habit reversal in conjunction with the medical management of eczema (Melin et al. 1986; Norén and Melin 1989). In addition, relaxation training, distraction and stress management have been shown to reduce itch intensity, scratching, and disease severity (Moore et al. 2009; Chida et al. 2007; Bae et al. 2012; Evers et al. 2009).
- *Patient Support Resources*
  - The Home of the Combined Approach (www.atopicskindisease.com)—teaches habit reversal to people struggling to break the itch-scratch cycle
  - The National Eczema Society (UK) (www.eczema.org)
  - Eczema Outreach Support (UK) (www.eos.org.uk)
  - National Eczema Association (USA) (www.nationaleczema.org)
  - The Eczema Society of Canada (www.eczemahelp.ca)
  - Eczema Association of Australasia (www.eczema.org.au)
  - Eczema Apps—EmolliZoo (education for children with eczema), Eczema Tracker (monitors triggers for eczema)
- *Outcome Measures*

  There are some recommended outcome measures for monitoring treatment response in people with eczema (Slatter et al. 2009):
  - Quality of life (e.g. Dermatology Life Quality Index, Infants' Dermatology Quality of Life index, Children's Dermatology Life Quality Index)
  - Disease severity (e.g. validated measures such as SCORing Atopic Dermatitis, Patient Oriented Eczema Measure, Eczema Area and Severity Index)
  - Itch (e.g. Itch Numerical Rating Scale 11 for itch intensity)
  - Treatment adherence (e.g. Medication Event Monitoring System)
  - Sleep improvement (e.g. sleep duration and quality)
  - Long-term control (e.g. Recap of atopic eczema, The Atopic dermatitis control test)

Prognosis

The clinical course of eczema varies and the spectrum of disease ranges from mild to severe. Mild disease can be controlled with the regular emollient application and intermittent TCS use, moderate to severe disease requires additional consideration of second-line treatments. Although 60% of paediatric cases resolve by puberty, in

up to 50% eczema re-occurs (Williams 2005). There is a correlation between serum IgE levels and disease severity (Illi et al. 2004). Poor prognostic factors for eczema include disease severity and early atopic sensitisation (Bath-Hextall et al. 2009).

The psychological burden of eczema is vast and may likely impact several factors, including disease severity, relationships, employment and quality of life (Mizara and McBride 2012). Early access to psychodermatology services may be a way to limit these far-reaching effects and positively impact the prognosis of people with eczema.

References

Aberg KM, et al. Psychological stress downregulates epidermal antimicrobial peptide expression and increases severity of cutaneous infections in mice. J Clin Investig. 2007;117:3339–49.

Absolon CM, Cottrell D, Eldridge SM, Glover MT. Psychological disturbance in atopic eczema: the extent of the problem in school aged children. Br J Dermatol. 1997;137:241–5.

Alanne S, Nermes M, Soderlund R, Laitinen K. Quality of life in infants with atopic dermatitis and healthy infants: a follow-up from birth to 24 months. Acta Paediatr. 2011;100:e65–70.

Amano H, et al. Psychological stress can trigger atopic dermatitis in NC/Nga mice: an inhibitory effect of corticotropin-releasing factor. Neuropsychopharmacology. 2008;33:566–73.

Anderson PC, Dinulos JG. Atopic dermatitis and alternative management strategies. Curr Opin Pediatr. 2009;21(1):131–8.

Arck P, Paus R. From the brain-skin connection: the neuroendocrine-immine misalliance of stress and itch. Immunomodulation. 2006;13:347–56.

Arden-Jones MR, Flohr C, Reynolds NJ, Holden CA. Atopic eczema. In: Griffiths C, Barker J, Bleiker T, Chalmers R, Creamer D, editors. Rook's textbook of dermatology. Oxford: Wiley; 2016.

Asher MI, Montefort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. Lancet. 2006;368(9537):733–43.

Aubert-Wastiaux H, Moret L, Le Rhun A, Fontenoy AM, Nguyen JM, Leux C, et al. Topical corticosteroid phobia in atopic dermatitis: a study of its nature, origins and frequency. Br J Dermatol. 2011;165:808–14.

Bae BG, Oh SH, Park CO, Noh S, Noh JY, Kim KR, et al. Progressive muscle relaxation therapy for atopic dermatitis: objective assessment of efficacy. Acta Derm Venereol. 2012;92:57–61.

Bantz SK, Zhu Z, Zheng T. The atopic march: progression from atopic dermatitis to allergic rhinitis and asthma. J Clin Cell Immunol. 2014;5(2):202.

Barbarot S, Bernier C, Deleuran M, De Raeve L, Eichenfield L, El Hachem M, et al. Therapeutic patient education in children with atopic dermatitis: position paper on objectives and recommendations. Pediatr Dermatol. 2013;30:199–206.

Bath-Hextall F, Delamere FM, Williams HC. Dietary exclusions for improving established atopic eczema in adults and children: systematic review. Allergy. 2009;64:258–64.

Beattie PE, Lewis-Jones MS. Parental knowledge of topical therapies in the treatment of childhood atopic dermatitis. Clin Exp Dermatol. 2003;28(5):549–53.

Beck LA, Thaçi D, Hamilton JD, et al. Dupilumab treatment in adults with moderate-to-severe atopic dermatitis. N Engl J Med. 2014;371(2):130–9.

Beebe DW. Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. Pediatr Clin N Am. 2011;58:649–65.

Bender BG, Leung SB, Leung DY. Actigraphy assessment of sleep disturbance in patients with atopic dermatitis: an objective life quality measure. J Allergy Clin Immunol. 2003;111(3):598–602.

Blauvelt A, de Bruin-Weller M, Gooderham M, et al. Long-term management of moderate-to-severe atopic dermatitis with dupilumab and concomitant topical corticosteroids (LIBERTY AD CHRONOS): a 1-year, randomised, double-blinded, placebo-controlled, phase 3 trial. Lancet. 2017;389(10086):2287–303.

Boguniewicz M, Leung DY. Atopic dermatitis: a disease of altered skin barrier and immune dysregulation. Immunol Rev. 2011;242:233–46.

Bollinger ME, Dahlquist LM, Mudd K, Sonntag C, Dillinger L, McKenna K. The impact of food allergy on the daily activities of children and their families. Ann Allergy Asthma Immunol. 2006;96:415–21.

Bridgett C. Habit reversal therapy:a behavioural approach to atopic eczema and other skin conditions. In: Bewley AP, Taylor RE, editors. Practical psychodermatology. Oxford: Wiley Blackwell; 2014. p. p66–72.

Brown S, Reynolds NJ. Atopic and non-atopic eczema. BMJ. 2006;332(7541):584–8.

Buske-Kirschbaum A, Gierens A, Hollig H, Hellhammer DH. Stress-induced immunomodulation is altered in patients with atopic dermatitis. J Neuroimmunol. 2002;129:161–7.

Camfferman D, Kennedy JD, Gold M, Simpson C, Lushington K. Sleep and neurocognitive functioning in children with eczema. Int J Psychophysiol. 2013;89:265–72.

Carlsten C, Dimich-Ward H, Ferguson A, et al. Atopic dermatitis in a high-risk cohort: natural history, associated allergic outcomes, and risk factors. Ann Allergy Asthma Immunol. 2013 Jan;110(1):24–8.

Carr CW, et al. Factors mediating the impact of chronic pruritus on quality of life. JAMA Dermatol. 2014;150:613–20.

Chamlin SL, Frieden IJ, Williams ML, Chren MM. Effects of atopic dermatitis on young American children and their families. Pediatrics. 2004;114:607–11.

Chida Y, Steptoe A, Hirakawa N, Sudo N, Kubo C. The effects of psychological intervention on atopic dermatitis. A systematic review and meta-analysis. Int Arch Allergy Immunol. 2007;144:1–9.

Choi EH, et al. Mechanisms by which psychologic stress alters cutaneous permeability barrier homeostasis and stratum corneum integrity. J Investig Dermatol. 2005;124:587–95.

Cork MJ, Robinson DA, Vasilopoulos Y, Ferguson A, Moustafa M, MacGowan A, et al. New perspectives on epidermal barrier dysfunction in atopic dermatitis: geneenvironment interactions. J Allergy Clin Immunol. 2006;118(1):3–21.

Dahl RE, Bernhisel-Broadbent J, Scanlon-Holdford S, Sampson HA, Lupo M. Sleep disturbances in children with atopic dermatitis. Arch Pediatr Adolesc Med. 1995;149:856–60.

Darsow U, Lubbe J, Taieb A, et al. Position paper on diagnosis and treatment of atopic dermatitis. J Eur Acad Dermatol Venereol. 2005;19(3):286–95.

De Benedetto A, Agnihothri R, McGirt LY, Bankova LG, Beck LA. Atopic dermatitis: a disease caused by innate immune defects? J Invest Dermatol. 2009;129:14–30.

De Marchi F, Piacentini GL, Piazza M, Sandri M, Boner AL, Peroni DG. Correlation of skin barrier impairment in atopic dermatitis with aeroallergen sensitization. Allergy Asthma Proc. 2015;36:127–33.

Deckers IA, McLean S, Linssen S, Mommers M, van Schayck CP, Sheikh A. Investigating international time trends in the incidence and prevalence of atopic eczema 1990-2010: a systematic review of epidemiological studies. PLoS One. 2012;7(7):e39803.

Dickman M, Dawson AL, Dellavalle RP. The relationship between hygiene and microbial burden in atopic dermatitis risk based on a systematic review. Arch Dermatol. 2012;148(8):936–8.

Ehlers A, Stangier U, Gieler U. Treatment of atopic dermatitis: a comparison of psychological and dermatological approaches to relapse prevention. J Consult Clin Psychol. 1995;63:624–35.

Ersser SJ, Cowdell F, Latter S, Gardiner E, Flohr C, Thompson AR, Jackson K, Farasat H, Ware F, Drury A. Psychological and educational interventions for atopic eczema in children. Cochrane Database Syst Rev 2014, Issue 1. Art. No. CD004054.

Evers A, Duller P, De Jong E, Otero ME, Verhaak CM, Van der Valk P, et al. Effectiveness of a multidisciplinary itch-coping training programme in adults with atopic dermatitis. Acta Derm Venereol. 2009;89:57–63.
Fallon PG, Sasaki T, Sandilands A, Campbell LE, Saunders SP, Mangan NE, et al. A homozygous frameshift mutation in the mouse Flg gene facilitates enhanced percutaneous allergen priming. Nat Genet. 2009;41(5):602–8.
Finlay AY, Khan GK. Dermatology Life Quality Index (DLQI)--a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994;19(3):210–6.
Fishbein AB, Vitaterna O, Haugh IM, Bavishi AA, Zee PC, Turek FW, et al. Nocturnal eczema: review of sleep and circadian rhythms in children with atopic dermatitis and future research directions. J Allergy Clin Immunol. 2015;136:1170–7.
Fleming P, Drucker AM. Risk of infection in patients with atopic dermatitis treated with dupilumab: a meta-analysis of randomized controlled trials. J Am Acad Dermatol. 2018;78(1):62–9;e1.
Garg A, et al. Psychological stress perturbs epidermal permeability barrier homeostasis: implications for the pathogenesis of stress-associated skin disorders. Arch Dermatol. 2001;137:53–9.
Gong JQ, Lin L, Lin T, et al. Skin colonization by Staphylococcus aureus in patients with eczema and atopic dermatitis and relevant combined topical therapy: a double-blind multicentre randomized controlled trial. Br J Dermatol. 2006;155(4):680–7.
Grillo M, Ng M, Gassner L, Marshman G, Dunn S, Hudson P. Pediatric atopic eczema: the impact of an educational intervention. Pediatr Dermatol. 2006;23(5):428–36.
Grimalt R, Mengeaud V, Cambazard F. The steroid-sparing effect of an emollient therapy in infants with atopic dermatitis: a randomized controlled study. Dermatology. 2007;214(1):61–7.
Hanifin JM, Reed ML. Eczema Prevalence and Impact Working Group. A population-based survey of eczema prevalence in the USA. Dermatitis. 2007;18(2):82–91.
Hashiro M, Okumura M. Anxiety, depression and psychosomatic symptoms in patients with atopic dermatitis: comparison with normal controls and among groups of different degrees of severity. J Dermatol Sci. 1997;14:63–7.
Hebert AA, Nguyen QD. Eczema. BMJ best practice: BMJ Publishing Group; 2019.
Herd RM. The financial impact on families of children with atopic dermatitis. Arch Dermatol. 2002;138(6):819–20.
Herd RM, Tidman MJ, Prescott RJ, Hunter JA. Prevalence of atopic eczema in the community: the Lothian Atopic Dermatitis study. Br J Dermatol. 1996;135(1):18–9.
Huang JT, Rademaker A, Paller AS. Dilute bleach baths for Staphylococcus aureus colonization in atopic dermatitis to decrease disease severity. Arch Dermatol. 2011;147(2):246–7.
Ibler KS, Jemec GB. cumulative life course impairment in other chronic or recurrent dermatologic diseases. Curr Probl Dermatol. 2013;44:130–6.
Illi S, von Mutius E, Lau S, et al. The natural course of atopic dermatitis from birth to age 7 years and the association with asthma. J Allergy Clin Immunol. 2004;113(5):925–31.
Johansson SG, Bieber T, Dahl R, et al. Revised nomenclature for allergy for global use: report of the Nomenclature Review Committee of the World Allergy Organization, October 2003. J Allergy Clin Immunol. 2004;113:832–6.
Kantor R, Kim A, Thyssen JP, et al. Association of atopic dermatitis with smoking: a systematic review and meta-analysis. J Am Acad Dermatol. 2016;75(6):1119–25;e1.
Kawakami T, Kaminishi K, Soma Y, et al. Oral antihistamine therapy influences plasma tryptase levels in adult atopic dermatitis. J Dermatol Sci. 2006;43(2):127–34.
Kemp S. Atopic eczema: its social and financial cost. J Pediatr Child Health. 1999;35(3):229–31.
Kiebert G, et al. Atopic dermatitis is associated with a decrement in health-related quality of life. Int J Dermatol. 2002;41:151–8.
Kilpelainen M, et al. Stressful life events promote the manifestation of asthma and atopic diseases. Clin Exp Allergy. 2002;32:256–63.
Kim JE, et al. Expression of hypothalamic-pituitary-adrenal axis in common skin diseases: evidence of its association with stress-related disease activity. Acta Derm Venereol. 2013;93:387–93.
Kraft M, Worm M. Dupilumab in the treatment of moderate-to-severe atopic dermatitis. Expert Rev Clin Immunol. 2017;13(4):301–10.

Kupfer J, Gieler U, Diepgen TL, Fartasch M, Lob-Corzilius T, Ring J, et al. Structured education program improves the coping with atopic dermatitis in children and their parents – a multicenter, randomized controlled trial. J Psychosom Res. 2010;68(4):353–8.
Lebovidge J, Elverson W, Timmons K, Hawryluk E, Rea C, Lee M, Schneider L. Multidisciplinary interventions in the management of atopic dermatitis. J Allergy Clin Immunol. 2016;138:325–34. https://doi.org/10.1016/j.jaci.2016.04.003.
Lee CH, et al. Transepidermal water loss, serum IgE and beta-endorphin as important and independent biological markers for development of itch intensity in atopic dermatitis. Br J Dermatol. 2006;154:1100–7.
Lewis-Jones MS, Finlay AY. The Children's Dermatology Life Quality Index (CDLQI): initial validation and practical use. Br J Dermatol. 1995;132(6):942–9.
Lien L, Green K, Thoresen M. Atopic conditions and mental health problems: a 3-year follow-up study. Eur Child Adolesc Psychiatry. 2010;19:705–13.
Lin T, et al. Association between stress and the HPA axis in the atopic dermatitis. Int J Med. 2017;18:2131.
Lio PA, Lee M, LeBovidge J, Timmons KG, Schneider L. Clinical management of atopic dermatitis: practical highlights and updates from the atopic dermatitis practice parameter 2012. J Allergy Clin Immunol Pract. 2014;2:361–9.
McNally NJ, Phillps DR, Williams HC. The problem of atopic eczema: aetiological clues from the environment and lifestyles. Soc Sci Med. 1998;46(6):729–41.
Meagher LJ, Wines NY, Cooper AJ. Atopic dermatitis: review of immunopathogenesis and advances in immunosuppressive therapy. Australas J Dermatol. 2002;43(4):247–54.
Melin L, Frederiksen T, Noren P, Swebilius BG. Behavioural treatment of scratching in patients with atopic dermatitis. Br J Dermatol. 1986;115(4):467–74. MEDLINE: 3778815
Meltzer LJ, Mindell JA. Systematic review and meta-analysis of behavioral interventions for pediatric insomnia. J Pediatr Psychol. 2014;39:932–48.
Mizara A, McBride SR. Core beliefs and psychological distress in patients with psoriasis and atopic eczema attending secondary care: the role of schemas in chronic skin disease. Br J Dermatol. 2012;166(5):986–93.
Moore EJ, Williams A, Manias E, Varigos G, Donath S. Eczema workshops reduce severity of childhood atopic eczema. Australas J Dermatol. 2009;50(2):100–6.
Morren MA, et al. Atopic dermatitis: triggering factors. J Am Acad Dermatol. 1994;31:467–47.
Muto T, Hsieh SD, Sakurai Y, Yoshinaga H, Suto H, Okumura K, et al. Prevalence of atopic dermatitis in Japanese adults. Br J Dermatol. 2003;148(1):117–21.
Niebel G, Kallweit C, Lange I, Folster-Holst R. Direct versus video-aided parental education in atopic eczema in childhood as supplement to specialty physician treatment. A controlled pilot study [Direkte versus videovermittelte Elternschulung bei atopischem Ekzem im Kindesalter als Erganzung facharztlicher Behandlung Eine Kontrollierte Pilotstudie]. Hautarzt. 2000;51(6):401–11.
Norén P, Melin L. The effect of combined topical steroids and habit-reversal treatment in patients with atopic dermatitis. Br J Dermatol. 1989;121(3):359–66.
Nowak-Węgrzyn A, Groetch M. Nutritional aspects and diets in food allergy. Chem Immunol Allergy. 2015;101:209–20.
O'Regan GM, Sandilands A, McLean WH, Irvine AD. Filaggrin in atopic dermatitis. J Allergy Clin Immunol. 2009;124:R2–6.
Oh SH, Bae BG, Park CO, Noh JY, Park IH, Wu WH, et al. Association of stress with symptoms of atopic dermatitis. Acta Derm Venereol. 2010;90:582–8.
Paller AS, Mancini J. Hurwitz clinical pediatric dermatology: a textbook of skin disorders of childhood and adolescence. 5th ed. Philadelphia, PA: Elsevier Saunders; 2016.
Paller AS, McAlister RO, Doyle JJ, Jackson A. Perceptions of physicians and pediatric patients about atopic dermatitis, its impact, and its treatment. Clin Pediatr. 2002;41:323–32.
Palmer CN, Irvine AD, Terron-Kwiatkowski A, Zhao Y, Liao H, Lee SP, et al. Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis. Nat Genet. 2006;38(4):441–6.

Pastar Z, Lipozencic J, Ljubojevic S. Etiopathogenesis of atopic dermatitis – an overview. Acta Dermatovenerol Croat. 2005;13:54–62.

Paternoster L, Standl M, Chen CM, Ramasamy A, Bønnelykke K, Duijts L, et al. Meta-analysis of genomewide association studies identifies three new risk loci for atopic dermatitis. Nat Genet. 2012;44(2):187–92.

Reuveni H, Chapnick G, Tal A, Tarasiuk A. Sleep fragmentation in children with atopic dermatitis. Arch Pediatr Adolesc Med. 1999;153:249–53.

Santer M, Burgess H, Yardley L, Ersser SJ, Lewis-Jones S, Muller I, et al. Managing childhood eczema: qualitative study exploring careers' experiences of barriers and facilitators to treatment adherence. J Adv Nurs. 2013;69:2493–501.

Schmid-Ott G, et al. Validity study for the stigmatization experience in atopic dermatitis and psoriatic patients. Acta Derm Venereol. 1999;79(6):443–7.

Schmid-Ott G, Jaeger B, Meyer S, Stephan E, Kapp A, Werfel T. Different expression of cytokine and membrane molecules by circulating lymphocytes on acute mental stress in patients with atopic dermatitis in comparison with healthy controls. J Allergy Clin Immunol. 2001;108:455–62.

Schmitt J, Apfelbacher C, Chen CM. Infant-onset eczema in relation to mental health problems at age 10 years: results from a prospective birth cohort study (German Infant Nutrition Intervention plus). J Allergy Clin Immunol. 2010;125(2):404–10.

Schram ME, Tedja AM, Spijker R, Bos JD, Williams HC, Spuls PI. Is there a rural/urban gradient in the prevalence of eczema? A systematic review. Br J Dermatol. 2010;162(5):964–73.

Schultz Larsen F, Diepgen T, Svenson A. The occurrence of atopic dermatitis in North Europe: an international questionnaire study. J Am Acad Dermatol. 1996;34(5 Pt 1):760–4.

Sherriff A, Golding J, Alspac Study Team. Hygiene levels in a contemporary population cohort are associated with wheezing and atopic eczema in preschool infants. Arch Dis Child. 2002;87(1):26–9.

Sigurgeirsson B, Boznanski A, Todd G, et al. Safety and efficacy of pimecrolimus in atopic dermatitis: a 5-year randomized trial. Pediatrics. 2015;135(4):597–606.

Simpson E, Dutronc Y. A new body moisturizer increases skin hydration and improves atopic dermatitis symptoms among children and adults. J Drugs Dermatol. 2011;10(7):744–9.

Slatter MJ, Hetzel S, Essex MJ. Anxiety and depression in adolescents with atopic dermatitis. Brain Behav Immunity. 2009;23:25–6.

Smith FJ, Irvine AD, Terron-Kwiatkowski A, Sandilans A, Campbell LE, Zhao Y, et al. Loss-of-function mutations in the gene encoding filaggrin causes ichthyosis vulgaris. Nat Genet. 2006;38(3):337–42.

Solomon LM, Beerman H. Atopic dermatitis. Am J Med Sci. 1966;252:478–96.

Spergel JM, Paller AS. Atopic dermatitis and the atopic march. J Allergy Clin Immunol. 2003;112(6 Suppl):S118–27.

Springston EE, Smith B, Shulruff J, Pongracic J, Holl J, Gupta RS. Variations in quality of life among caregivers of food allergic children. Ann Allergy Asthma Immunol. 2010;105:287–94.

Stangier U, Ehlers A. Stress and anxiety in dermatological disorders. In: Mostofsky DI, Barlow DH, editors. The management of stress and anxiety in medical disorders. Needham Heights, MA: Allyn & Bacon; 2000. p. 304–33.

Suarez AL, Feramisco JD, Koo J, Steinhoff M. Psychoneuroimmunology of psychological stress and atopic dermatitis: pathophysiologic and therapeutic updates. Acta Derm Venereol. 2012;92:7–15.

Tareen RS. Atopic dermatitis: a psychocutaneous review. In: Tareen RS, Greydanus DE, Jafferany M, Patel DR, Merrick J, editors. Pediatric Psychodermatology: a clinical manual of child and adolescent psychocutaneous disorders. Berlin: De Gruyter; 2013.

Thaçi D, Simpson EL, Beck LA, et al. Efficacy and safety of dupilumab in adults with moderate-to-severe atopic dermatitis inadequately controlled by topical treatments: a randomised, placebo-controlled, dose-ranging phase 2b trial. Lancet. 2016;387(10013):40–52.

Thompson A, Kent G. Adjusting to disfigurement: processes involved in dealing with being visibly different. Clin Psychol Rev. 2001;21:663–82.

Weidinger S, Gieger C, Rodriguez E, Baurecht H, Mempel M, Klopp N, et al. Genome-wide scan on total serum IgE levels identifies FCER1A as novel susceptibility locus. PLoS Genet. 2008;4:e1000166.

Williams HC. Atopic eczema. BMJ. 1995;311(7015):1241–2.

Williams HC. Atopic dermatitis: the epidemiology, causes and prevention of atopic eczema. Cambridge: Cambridge University Press; 2000.

Williams HC. Clinical practice. Atopic dermatitis. N Engl J Med. 2005;352(22):2314–24.

Williams H, Stewart A, von Mutius E, Cookson W, Anderson HR. International Study of Asthma and Allergies in Childhood (ISAAC) PhaseOne and Three Study Groups. Is eczema really on the increase worldwide? J Allergy Clin Immunol. 2008;121(4):947–954. e15.

Xie QW, et al. Risk of mental disorders in children and adolescents with atopic dermatitis: a systematic review and meta-analysis. Front Psychol. 2019;10. Article 1773

Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361–70.

Zuberbier T, Orlow SJ, Paller AS, Taieb A, Allen R, Hernanz-Hermosa JM, et al. Patient perspectives on the management of atopic dermatitis. J Allergy Clin Immunol. 2006;118:226–32.

Somatoform Disorders

11

Iyas Assalman

Definition of the Disorder

Somatoform disorders, also known as somatic symptom disorders, include hypochondriasis. Some significant changes to the terminology have been introduced in ICD-10 and DSM 5. In ICD-11 somatoform disorders are now classed under the term *bodily distress disorder*, which is characterized by the presence of bodily symptoms that are distressing to the individual and excessive attention directed toward the symptoms, which may be manifested by repeated contact with health care providers. All somatoform disorders are characterized by repeated presentations of physical symptoms together with requests for medical investigations, despite repeated negative findings and reassurances by a clinician that the symptoms have no physical basis. Somatization disorder in ICD-10 is defined as presenting with multiple, recurrent and frequently changing physical symptoms of at least 2 years' duration. These are often associated with many fruitless investigations with negative results by a variety of clinicians. Hypochondriasis is classed in ICD-11 under obsessive-compulsive and related disorders. In DSM 5 it has been renamed *illness anxiety disorder* and is characterized by 6 or more months of a general and non-delusional preoccupation with fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms. This preoccupation causes significant distress and impairment in one's life; it is not accounted for by another psychiatric or medical disorder, and a subset of individuals with somatic symptom disorder has poor insight about the presence of this disorder.

I. Assalman (✉)
East London Foundation Trust, London, UK

Queen Mary University of London, London, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_11

Aetiology and Classification

There are various subtypes of somatisation disorders described in the two main diagnostic systems. There are minor differences between ICD-10 and DSM 5. The latest edition of DSM 5 has moved away from the need to have no medical explanation in order to make the diagnosis of *medically unexplained symptoms* and gain access to appropriate treatment. The emphasis now is on symptoms that are substantially more severe than expected in association with distress and impairment. The diagnosis includes conditions with no medical explanation and conditions where there is some underlying pathology, but an exaggerated response.

The major diagnosis in this diagnostic class, Somatic Symptom Disorder (Table 11.1), emphasizes diagnosis made on the basis of positive symptoms and signs (distressing somatic symptoms plus abnormal thoughts, feelings and behaviours in response to these symptoms) rather than the absence of a medical explanation for somatic symptoms. A distinctive characteristic of many individuals with somatic symptom disorders is not the somatic symptoms per se, but instead the way they present and interpret them.

A new category has therefore been created in DSM 5 under the heading *Somatic Symptom and Related Disorders*. This includes diagnoses of Somatic Symptom Disorder, Illness Anxiety Disorder (Table 11.2), Conversion Disorder, Factitious Disorder and a variety of other related conditions. The last three are not classed under somatoform disorders in ICD-10 or ICD-11 (Table 11.3).

Practice Point
The term Hypochondriasis is no longer included in DSM 5. In ICD-11 it is classed under obsessive-compulsive or related disorders.

The aetiology of somatization disorder is unknown, but it is most likely multifactorial including biological, physiological, psychological, social, cultural and iatrogenic factors. The importance and relevance of these factors can be different at

Table 11.1 DSM-5 criteria to make a diagnosis of Somatic Symptom Disorder

DSM-5 Somatic Symptom Disorder criteria
A. One or more somatic symptoms that are distressing or result in significant disruption of daily life.
B. Excessive thoughts, feelings or behaviours related to the somatic symptoms or associated health concerns as manifested by at least one of the following:
(a) Disproportionate and persistent thoughts about the seriousness of one's symptoms.
(b) Persistently high level of anxiety about health or symptoms.
(c) Excessive time and energy devoted to these symptoms or health concerns.
C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months).

Table 11.2 DSM-5 criteria to make a diagnosis of Illness Anxiety Disorder

DSM-5 Illness Anxiety Disorder Criteria
A. Preoccupation with having or acquiring a serious illness.
B. Somatic symptoms are not present or if present, are only mild in intensity. If another medical condition is present or there is a high risk for developing a medical condition (e.g. strong family history is present), the preoccupation is clearly excessive or disproportionate.
C. There is a high level of anxiety about health, and the individual is easily alarmed about personal health status.
D. The individual performs excessive health-related behaviours (e.g. repeatedly checks his or her body for signs of illness) or exhibits maladaptive avoidance (e.g. avoids doctor appointments and hospitals).
E. Illness preoccupation has been present for at least 6 months, but the specific illness that is feared may change over that period of time.
F. The illness-related preoccupation is not better explained by another mental disorder, such as *somatic symptom disorder, panic disorder, generalized anxiety disorder, body dysmorphic disorder, obsessive-compulsive disorder or delusional disorder, somatic type*.

Table 11.3 Diagnostic principles in ICD and DSM

ICD 10	ICD 11	DSM 5
Somatoform disorders	Bodily distress disorder BDO Obsessive-Compulsive Disorder (OCD) or related disorders	Somatic Symptom disorder
Somatoform disorders • Hypochondriacal disorder (including Body Dysmorphic Disorder) • Somatoform autonomic dysfunction • Persistent somatoform pain disorder	Bodily distress disorder • BDO (mild to severe) • Body integrity dysmorphia Obsessive-compulsive disorder or related • OCD • Body dysmorphic disorder • Olfactory reference disorder • Hypochondriasis • Hoarding disorder • Body-focused repetitive behaviour disorders	Somatic Symptom and related disorders • Somatic Symptom disorder • Illness Anxiety disorder • Conversion disorder • Factitious disorder • Psychological factors affecting other medical conditions

different times in the natural course of the illness. For instance, it may be a psychological trauma that precipitates the illness, but iatrogenic factors that maintain the illness.

Prevalence and Age of Onset

The expected prevalence of Somatic Symptom Disorder stated in DSM 5 is higher than that for Somatization Disorder (<1%) but lower than that of Undifferentiated Somatoform Disorder (19%). Both are more common in women.

Somatic symptom disorder usually has an onset before age 30, whereas illness anxiety disorder has a less specific age of onset.

Clinical Features and Presentation

Patients with somatic symptom disorder believe that they have a serious disease that has not yet been detected and they cannot be persuaded to the contrary. Their convictions persist despite negative laboratory results, the benign course of the alleged disease over time, and appropriate reassurances from physicians. Yet, their beliefs are not sufficiently fixed to be delusions.

Practice Point
Somatic symptom disorder is often accompanied by symptoms of depression and anxiety and commonly coexists with a depressive or anxiety disorder.

Patients with illness anxiety disorder (hypochondriasis), like those with somatic symptom disorder, believe that they have a serious disease that has not yet been diagnosed, and they cannot be persuaded to the contrary. Their convictions also persist despite negative laboratory results, the benign course of the alleged disease over time, and appropriate reassurances from physicians. Their preoccupation with illness interferes with their interaction with family, friends and co-workers. They are often addicted to Internet searches about their feared illness, inferring the worst from information (or misinformation) they find there. The feared illness is usually fairly static over time in contrast to the varying aspect of symptoms in somatization (body distress disorder).

Practice Point
Patients with somatic symptom disorder and illness anxiety disorder may maintain a belief that they have a particular disease or, as time progresses, they may transfer their belief to another disease. Illness anxiety disorder shows much less fluctuation in the feared disease.

Deferential Diagnosis and Comorbidity

Somatic symptom disorder must be differentiated from non-psychiatric medical conditions, especially disorders that show symptoms that are not necessarily easily diagnosed (Table 11.4).

Somatic symptom disorder is differentiated from illness anxiety disorder by the emphasis in illness anxiety disorder on fear of having a disease rather than a concern about many symptoms. Patients with illness anxiety disorder usually complain

Table 11.4 Differential diagnosis

Psychiatric	Non-psychiatric medical conditions
• Body dysmorphic disorder • Conversion disorder • Delusional disorder, somatic type • Depression • Dissociative disorders • Dysthymic disorder • Factitious disorder • Generalized anxiety disorder • Malingering • Obsessive-compulsive disorder • Panic disorder • Schizophrenia	• Acquired immunodeficiency syndrome (AIDS) • Degenerative diseases of the nervous system • Endocrinopathies • Multiple sclerosis • Myasthenia gravis • Occult neoplastic disorders • Systemic lupus erythematosus

about fewer symptoms than patients with somatic symptom disorder; they are primarily concerned about being sick.

Practice Point
Illness anxiety disorder must be differentiated from other medical conditions. Too often these patients are dismissed as 'chronic complainers' and careful medical examinations are not performed.

Somatic symptom disorder can also occur in patients with depressive disorders and anxiety disorders. Patients with panic disorder may initially complain that they are affected by a disease (e.g. heart trouble), but careful questioning during the medical history usually uncovers the classic symptoms of a panic attack. Delusional disorder beliefs occur in schizophrenia and other psychotic disorders, but can be differentiated from somatic symptom disorder by their delusional intensity and by the presence of other psychotic symptoms. In addition, schizophrenic patients' somatic delusions tend to be bizarre, idiosyncratic and out of keeping with their cultural milieu.

Practice Point
Patients with body dysmorphic disorder wish to appear normal, but believe that others notice that they are not, whereas those with somatic symptom disorder seek out attention for their presumed diseases.

Practice Point
Illness anxiety disorder can be differentiated from obsessive-compulsive disorder by the singularity of their beliefs and by the absence of compulsive behavioural traits; but there is often an obsessive quality to the patients' fear.

Table 11.5 The primary role of the non-psychiatric specialist is to

- Exclude physical disease and trauma that can be treated medically.
- Make it clear to the patient that he/she does not have the physical disease he/she fears and that there is no indication for any other medical attention.
- There is no medical indication for further diagnostic tests or examinations.
- Coordinate the management with the primary care physician and other doctors that the patient may be in contact with.
- Consider a referral to a psychiatrist for examination or treatment.

Assessment and Treatment

Patients with somatic symptom disorder usually resist psychiatric treatment, although some accept this treatment if it takes place in a medical setting and focuses on stress reduction and education in coping with chronic illness (Table 11.5).

Psychotherapy is an established treatment modality, but it meets with specific challenges in the initial phases, when patients very often find it difficult to accept that a "talking cure" might help with their primarily bodily symptoms and concerns.

Group psychotherapy often benefits such patients, in part because it provides social support and social interaction that seem to reduce their anxiety. Other forms of psychotherapy, such as individual insight-oriented psychotherapy, behaviour therapy, cognitive therapy and hypnosis, may be useful.

Consider treatment with medication; primarily antidepressants. However, an antidepressant with the fewest interactions should be chosen as polypharmacy is common in these patients.

Delusional Infestation

12

Peter Lepping

Definitions

Delusional Infestation: The core and defining symptom of patients with delusional infestation (DI) is the rigidly held believe that they are infested despite a lack of medical evidence for an infestation. The patients also have abnormal sensations of crawling, biting, leaving marks or other symptoms they interpret as evidence of an alleged infestation. Therefore, delusional infestation is diagnosed by (1) the presence of a delusional belief of being infested, (2) sensations on one's body or one's immediate environment of such an infestation, and (3) the absence of medical evidence for an infestation.

Aetiology and Classification

Primary and Secondary DI

Delusional infestation can occur as a primary mono-delusional disorder or in the context of other medical or psychiatric illnesses (Freudenmann and Lepping 2009) as a secondary diagnostic entity (secondary delusional infestation).

P. Lepping (✉)
Wrexham Maelor Hospital Liaison Service (BCULHB), Wales, Wrexham, UK

Centre for Mental Health and Society, Bangor University, Wales, Bangor, UK

Mysore Medical College and Research Institute, Karnataka, India
e-mail: peter.lepping@wales.nhs.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_12

Primary delusional infestation meets the criteria for a delusional disorder in ICD 11 (6A24), or a delusional disorder, somatic type in DSM-5. The symptoms are not a manifestation of another disorder or disease and are not due to the effect of a substance or medication on the central nervous system.

Secondary DI can be secondary to organic physical disease, substance use, or secondary to psychiatric disease. Approximately 60% of patients have the secondary form.

DI secondary to organic physical disease occurs in the context of medical illness, particularly brain disorders such as

- Parkinson's Disease.
- Huntington's Disease.
- Multiple Sclerosis.
- Dementia.
- Brain stem and thalamus atrophy.
- Stroke.
- Neurovascular disorders.
- DI has also been described in general medical conditions with pruritus or paraesthesia, for example, infections such as Tuberculosis, Leprosy or Gonorrhoea.
- Endocrine conditions such as diabetes, hypothyroidism or hypoparathyroidism.
- Oncological conditions as well as vitamin deficiencies.
- Some case reports have mentioned DI in the context of rheumatoid conditions
- Renal failure.
- Sensory deficits.
- Cardiovascular conditions.

- DI can also occur as part of a psychiatric disorder such as schizophrenia, depression and anxiety. Some case reports have described DI in the context of various prescribed medications such as L-DOPA, Methylphenidate, Erythromycin, Clarithromycin and non-steroidal anti-inflammatories.

- Substance misuse disorders are common triggers for DI, especially stimulants. DI can occur in association with cocaine, crack cocaine, amphetamines, cannabis and alcohol use. Drug use is more common in younger, more urban and male DI patients.

Practice Point
Always ask patients about recreational drug usage (some patients will not own up to taking recreational drugs). Ask patients if you can analyse their urine for recreational drugs.

Patho-Aetiology

A number of MRI studies have looked at the aetiology of DI. DI patients show lower grey matter volume in thalamic, striatal, insular and medial prefrontal brain regions in contrast to patients with non-somatic delusional disorder (such as paranoid delusions) and healthy controls. These differences were consistently detected at regional and network levels. This indicates that patients with delusional infestation may have a different aetiology than patients with other mono-delusional disorders, making DI a unique disorder. The available data also support the notion that dysfunctional somatosensory and peripersonal networks could mediate somatic delusions in patients with DI. This would support the idea that errors of probabilistic reasoning are involved in symptom formation in DI, with patients preferring unlikely explanations over likely ones because of changes to neuronal pathways.

Shared Beliefs and Unusual Presentations of DI

Shared Delusional Belief: DI occurs as a shared delusional belief in about 10% of cases. The person with the delusion, called the inducer, is able to persuade other people around him or her that there is an infestation. These people may end up sharing the delusional belief in what is called a shared delusional belief or folie à deux or folie à trois depending on whether two, three or more people are involved in total. In terms of treatment, the primary target for treatment is the inducer. Separation of other involved persons from the inducer is usually enough to treat *their* shared delusional belief.

DI by Proxy: A patient with DI believes that the infestation is in somebody else, for example, a relative, their own child or a pet. This can cause safeguarding concerns if the patient attempts to get rid of the alleged pathogen in a child or pet, for example, by using topical chemicals or pesticides.

Double Delusional Infestation: Double DI is a phenomenon in which patients with the delusional beliefs have symptoms of an alleged infestation themselves, but also believe that another person or pet (the proxy) is affected by the infestation. In the case of a pet or a small child the proxy is not able to express whether they share this delusional belief and it therefore cannot be a shared delusional as defined in ICD 11. It is therefore called double delusional infestation and means that the patient considers symptoms to be present in him or herself and another person or pet who is unable to express whether they are infested or not.

Historical Names for Delusional Infestation (Main Terms Out of Over 30 Terms Used Since 1894)

(a) *Les acarphobes* The first description of delusional infestation goes back to 1894 (Thibierge).
(b) *Dermatozoenwahn* Most famously Ekbom described DI as Dermatozoenwahn in 1938. Harbauer later coined the phrase 'Ekbom syndrome' in 1949.
(c) *Delusions of Parasitosis* Miller and Wilson first described the illness as delusions of parasitosis in 1946, later reaffirmed by Tullett in 1965.
(d) *Delusional Infestation* Freudenmann and Lepping (2009) described the term delusional infestation, which has since become the standard terminology.

Delusional infestation has been accepted as the standard terminology because it best describes the widening of alleged pathogens patients ascribe their symptoms to. It is in keeping with the fluent and ongoing shifts in the delusional theme associated with DI patients and includes any future alleged pathogens (see also Section 'Delusional Themes').

Clinical Presentation

General Considerations

There is no uniformity in the presentations or individual patients with DI. Whilst all patients believe that either they or their immediate environment is infested with an alleged pathogen, the actual reported symptoms can vary quite significantly (Lepping 2015). As a set of minimal criteria for a diagnosis of DI, one would expect

conviction of being infested by pathogens without any medical or microbiological evidence for this. One would expect abnormal sensations in or on the skin, body or in the patient's immediate environment, with or without visual illusions or hallucinations.

Delusional Theme

As is common with any classic delusional beliefs, the delusional theme changes over time. This happened in DI as well. Currently, around 75% of DI patients believe that the alleged pathogen is alive; most patients describing worms, insects, parasites but also mites, fungi, bacteria or vermin. Around a quarter of alleged pathogens are deemed to be non-living (inanimate) organisms such as fibres, threads or strands (This is commonly the case in patients who identify with the invented category of 'Morgellon Disease'). Patients may describe the pathogen as laying eggs or occurring with particular egg cycles. Patients may also describe infestations of their home, car or immediate environment. However, most patients will describe the infestation to be on or underneath their skin or inside their whole body.

Dermatological Symptoms: Patients with DI classically describe itching and changes to their skin. They may also point to alleged canals or tunnels they think the pathogen is creating under their skin. They commonly misinterpret normal, often age-related skin changes as evidence of infestation. They are likely to scratch because of the itching sensation and may interpret scratch marks as signs of infestation. There is commonly evidence of superinfections on damaged skin.

Systemic Symptoms: Patients often complain of general feelings of malaise, tiredness or lethargy. Depending on the type of the alleged infestation, patients often complain of systemic pathogens. They may think they have a worm or parasite infestation that affects the whole body. They may also focus on particular body parts such as intestines, eyes, ears or lungs. They commonly describe elaborate and unusual symptoms that are not in keeping with known infestations.

Environmental Presentations: Patients may complain of infestations of their immediate environment. They may therefore regularly change bedding, clothes or furniture. Occasionally, patients believe that their car, their garden, or their pets may be infested or the source of infestation. In rare cases, patients cite infestation by vermin such as rats or mice.

Specimen Sign

Formerly known as ‘matchbox sign’ this describes the high likelihood that patients bring evidence of the pathogen in a container, fixed on sellotape, or as a digital photo (Figs. 12.1, 12.2, 12.3, and 12.4). In the past, many patients used matchboxes as a container of choice, but this is now rare. Between half and three quarters of patients bring some evidence of their alleged infestation. This should be encouraged because it allows for an examination of the alleged evidence and a subsequent conversation about the results. The most common particles brought in are skin debris, followed by hair. Incidental or actual findings of parasites are extremely rare, and may still not explain the symptoms the patient describes.

Fig. 12.1 Specimen the patients may bring in for examination as their proof of the alleged infestation. Sometimes patients may present with samples of genuine insects which do, however, not explain the patient’s symptoms. Exclusion of a genuine infestation is important

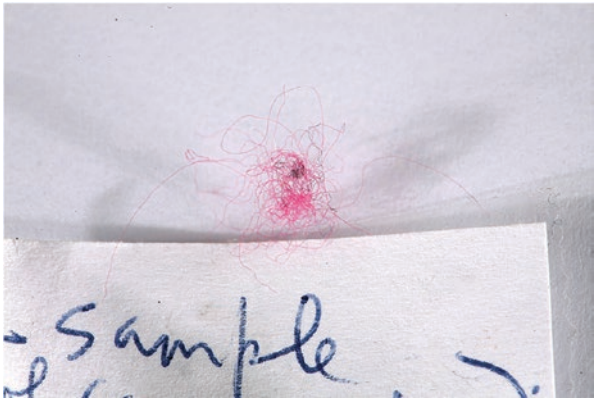


Fig. 12.2 Specimen the patients may bring in for examination as their proof of the alleged infestation. Specimen sign. Some patients present with fibrous material which they find on their body or in their environment

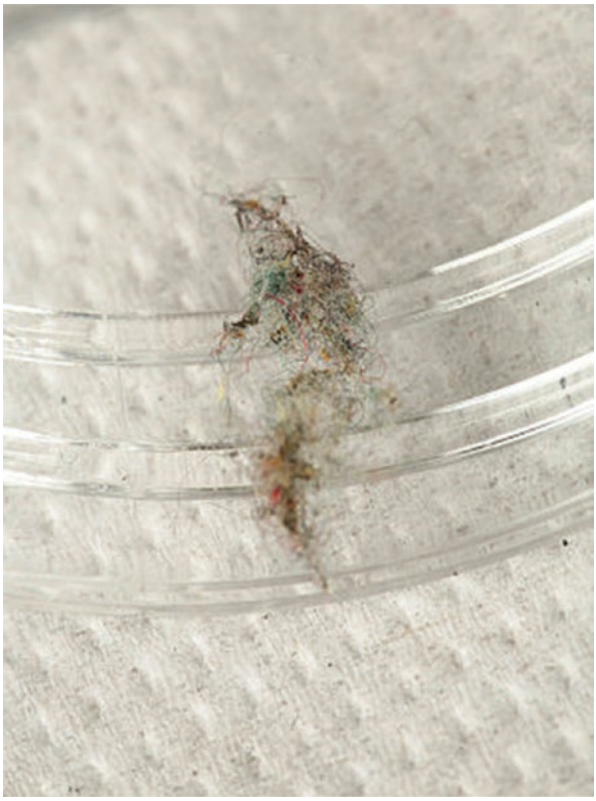


Fig. 12.3 Specimen the patients may bring in for examination as their proof of the alleged infestation. Some patients present with fibrous material which they find on their body or in their environment

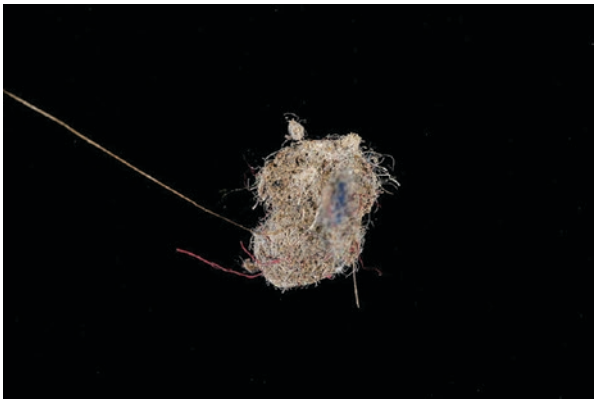


Fig. 12.4 Specimen the patients may bring in for examination as their proof of the alleged infestation. Patients may take their own images of infesting material

Practice Point
Pay attention to any material (specimen) the patient brings. Have a low threshold for sending material to the laboratory for analysis. This will assist engagement of the patient.

Photograph of material provided by patient to support the diagnosis (specimen sign)

Material provided by patient (specimen sign)

Why DI Is so Debilitating

Tenacity of Patients to Prove Their Infestation Patients with DI commonly present after they try to identify the pathogen themselves. They may go through great length to 'catch the parasite'. Some patients resort to tweezers or knives to cut out the alleged pathogen from their skin or body.

Fig. 12.5 Presentation at examination. Patients may present with excoriations and erosions of the skin

Patients Try to Cure Themselves Some use bleach or other industrial chemicals to clean their skin. This can cause further skin irritations.

The Lives of Those Around Them Are Compromised In cases of DI by proxy, it can lead to other people being affected by attempts to rid them from the alleged pathogen.

Skin Damage In combination with scratching, the use of industrial chemicals often causes additional secondary skin irritation or infections (Fig. 12.5).

Social Consequences, Debt and Disability The affected patients usually have a high degree of impairment of quality of life and may spend hours looking for and trying to rid themselves of the pathogen. The illness commonly has a negative effect on relationships with social isolation, divorce, moving house and losing employment being common consequences. It would also not be unusual for a patient to have spent a lot of money on pesticides, private medical consultations, alternative medicines and pest control companies.

Psychosocial Comorbidities The symptoms can cause secondary paranoia, depressive symptoms and despair, which can lead to suicidal and very rarely homicidal thoughts against treating doctors. Patients usually get exasperated with the medical profession for their perceived inability to find the alleged pathogen. Doctor hopping is common and frustration on the side of the patient as well as the clinician is the norm.

Examination
Patients may have no physical signs at all. Or patients may have cutaneous lesions at various stages of the external trauma/healing process e.g.

- Lichenification
- Excoriation
- Linear tears
- Scarring
- Hyperpigmentation

- Hypopigmentation
- Ulceration
- Crusting
- Slough
- Hair loss
- Nail changes

Practice Point
Always make the effort to check the patient's skin thoroughly. Ask the patient to show you where the skin in most affected. This will aid engagement, and allow you to assess appropriate cutaneous treatments.

Epidemiology and Aetiology

Prevalence

Delusional infestation is considered a rare disorder, but real epidemiological data are rare. The most comprehensive study was done in Germany for the index year 1988. Based on cases reported to hospitals and public health services a prevalence of 5.5 cases per one million inhabitants was calculated. However, this rose to 83 cases per million based on a survey of private and specialist practices. The latter figures are in keeping with other available data and would mean an extrapolated annual incident rate of 17 new cases per one million inhabitants. About 90% of patients with DI seek help from dermatologists, while many psychiatrists may not see a single DI patient in the entire career. A large survey amongst British dermatologists showed that they had all seen at least one patient with DI. In Poland, 85% of all dermatologists had seen at least one case. 20% reported to currently treat at least one patient. Psychiatrists are much less likely to see DI patients and psychiatric admissions are rare. One German study gave a rate of 2.5 DI patients per 1000 admissions in Münster, whilst the rate was as low as 0.67 patients per 1000 patients in Bonn, going back to data from the 1970s and 1980s.

Epidemiology

The sex ratio is around 2.5:1 with a preponderance of female patients. In patients below 40 years of age, there can be a preponderance of male patients, possibly because of presentations secondary to substance use. DI patients are sometimes more isolated and sensory impairment is a risk factor. However, particularly in primary delusional infestation, there is no association with any particular social-economic status, education attainment or childhood difficulties. Overall, DI is a rare disease, which disproportionally presents to dermatologists. Patients avoid seeing psychiatrists because they do not believe to be mentally ill.

Course and Duration of Illness

The clinical cause of DI is very variable and can be episodic, periodic or chronic. The peak age at presentation is between 60 and 70. However, DI can occur throughout the whole range of ages. There is a clear tendency for DI patients to be older, with a mean age of around 60 years at presentation. The duration of untreated psychosis varies largely, but on average is around 3½ years. A recent study, combining patients from various specialist clinics, has shown that the prognosis is significantly better with shorter duration of untreated psychosis (Romanov 2018).

Diagnostic Process

General Considerations

Patients come to a clinician with a set of symptoms and their own explanatory model for those symptoms. Getting into any kind of argument around their explanatory model is unlikely to be a successful way forward. It is, however, often possible to agree with the patient that the primary objective should be to get rid of the symptoms. This is usually an important starting point for developing a trustful relationship. As with any delusional disorder, the extent to which a clinician explains their understanding of the symptoms to the patient has a number of ethical considerations. Most people consider it acceptable in delusional disorders to take a gradual approach to any explanatory models in the interest of the patient and symptom reduction. Some specialist clinics point out that there is a variety of potential reasons for a patient's symptoms (including a disorder of the brain), whilst other clinics may take the view of not going into any explanatory models at all but focusing entirely on symptom reduction. It is very important to genuinely keep an open mind as to what the cause of the symptoms is.

Diagnosing Process

The first step in the diagnosis of DI is to verify that the patient is likely to have a delusional belief. It is impossible to exclude a new pathogen with a certainty of 100%, as the history of medicine is full of examples of new diagnoses. However, psychiatrists do not primarily identify delusions by judging the reality or falsity of the content of the belief. In normal practice, they look instead at the patient's explanations and proofs for the belief and their likelihood. In addition, it is important to examine the intensity with which the belief is held (delusional intensity). For a diagnosis of DI the patient has to have a strongly held belief of infestation with an explanatory model that makes an actual infestation highly unlikely. The belief has to be held despite evidence to the contrary, for example, negative specimen or biopsy results. It is important to find out whether the patient would consider alternative explanations for their symptoms. Any symptoms need to be distinguished from formication (the sensation of insects crawling on or underneath the skin), which is a symptom of menopause as well as acute stimulant intoxication and is not usually accompanied by fixed delusions (Fig. 12.6).

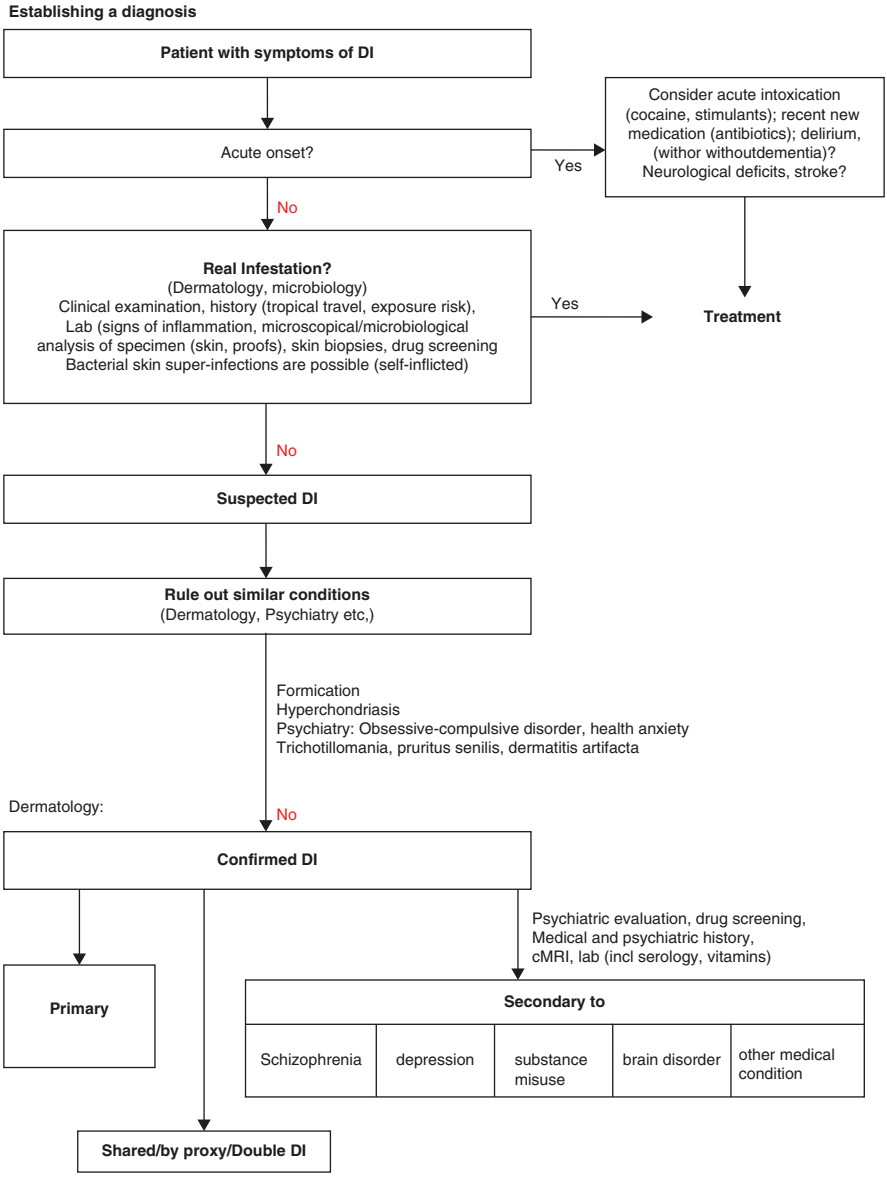


Fig. 12.6 Establishing a diagnosis

Delusional Themes

No delusional themes are stable over time and develop with the prevailing zeitgeist and societal developments. In the case of delusional infestation, patients believed themselves to be infested by scabies, typhoid or the pest in the nineteenth and the early twentieth century, which changed to parasites or insects in the middle of the twentieth

century. Increasingly at the end of the twentieth century, patients believed to be infected with bacteria, fungi, viruses or unknown species (Freudenmann and Lepping 2009). In recent years, non-living organisms have increasingly been named as alleged pathogens including threads, hairs, fibres, and a phenomenon that affected persons call 'Morgellons'. Investigations and research on self-diagnosed patients with 'Morgellons' have never been able to show that it is in fact a new illness, but the conclusion of all major research projects suggest that it is a further development of the delusional theme of infestation. About a quarter of patients now identify with non-living pathogens as the cause of their symptoms. Such development of themes is common in psychiatry. It is also the reason why the terminology has been changed from delusional parasitosis to delusional infestation to encompass any alleged pathogens, past, present and future.

Differential Diagnosis

It is vital to exclude real infestations in all cases. Always examine the patient and any specimens they bring. If necessary, a rigorous exclusion of an infestation by a dermatologist or infectious disease specialist may be indicated. It is important to look at secondary DI by identifying possible primary triggers or illnesses. This includes a history of antibiotics and particularly stimulant drug use. It includes the possibility of psychiatric illnesses such as anxiety and depression, but also dementia, delirium or schizophrenia. Consider medical and neurological conditions, particularly those that may cause itching. These include diabetes, cancer, stroke and thyroid disease. It is advisable to request any investigation that a dermatologist or microbiologist would find reasonable and necessary. Mandatory tests include a full blood count, particularly to look for raised eosinophils, which may suggest an infestation, allergy or hypersensitivity. In addition, mandatory tests include c reactive protein, serum creatinine and electrolytes, liver function, thyroid-stimulating hormone, fasting glucose, and ideally a urine analysis for illicit drug use. In some clinical contexts, it may be useful to consider serology for borrelia, Treponema, hepatitis, HIV, vasculitis screening, allergy testing, vitamin B12 and folate levels. See Fig. 12.6 for pathway.

Differential diagnosis of DI (based on published cases)

1.	Organic or substance-induced DI
1.1	Psychotropic drugs
1.1.1	Cocaine
1.1.2	Crack cocaine
1.1.3	Ecstasy
1.1.4	Amphetamine
1.1.5	L-DOPA
1.1.6	Methylphenidate
1.1.7	Cannabis
1.1.8	Alcohol
1.1.9	Phenelzine with PH
1.1.10	Polysubstance misuse
1.2	Other medications

1.2.1	Corticosteroids
1.2.2	Antibiotics (clarithromycin azithromycin ciprofloxacin erythromycin)
1.2.3	Alpha b2 interferon plus ribavirin
1.2.4	Topiramate
1.2.5	Bromide intoxication
1.3	Brain disorders
1.3.1	Dementia
1.3.2	Cortical atrophy
1.3.3	Parkinson's disease
1.3.4	Huntington's disease
1.3.5	Multiple sclerosis
1.3.6	Brain stem and thalamus atrophy
1.3.7	Torticollis spasmodicus
1.3.8	Vascular/subcortical damage
1.3.9	Other dementia
1.3.10	Stroke
1.3.11	Haemorrhage
1.4	Brain tumours and infections
1.4.1	Pituitary tumour
1.4.2	Craniopharyngioma
1.4.3	Meningitis
1.4.4	Encephalitis
1.4.5	Neurosyphilis
1.4.6	HIV posttraumatic epilepsy
1.4.7	Normal pressure hydrocephalus
1.4.8	Delirium
1.5	General medical conditions
1.5.1	Tuberculosis
1.5.2	Leprosy
1.5.3	Gonorrhoea
1.5.4	Diabetes
1.5.5	Hypothyroidism
1.5.6	Panhypopituitarism
1.5.7	Hyperparathyroidism
1.5.8	Postpartum
1.5.9	Solid tumours, lymphoma
1.5.10	Leukaemia
1.5.11	Anaemia
1.5.12	Vitamin deficiency (B1 B3 folic acid B12)
1.5.13	Systemic lupus erythematosus
1.5.14	Behçet's disease
1.5.15	Renal failure
1.5.16	Cholestasis
1.5.17	Sensory deficits
1.5.18	Cardiovascular conditions (congestive heart failure, absolute arrhythmia, arterial hypertension)
1.5.19	
2.1	Major psychiatric disorders
2.1.1	Schizophrenia

2.1.2	Psychotic depression
2.1.3	Other delusional disorders
2.1.4	Toxic psychosis
2.1.5	OCD
2.1.6	Anxiety
2.2	Similar psychiatric disorders
2.2.1	Trichotillomania
2.2.2	Hypochondriasis
2.2.3	Health anxiety
2.2.4	Burning mouth disorder (glossodynia)
2.3	Psychodermatoses
2.3.1	Dermatitis artefacta
2.3.2	Psychogenic excoriation
2.3.3	Other causes for pruritus such senile pruritus or medication
3.1	Formication
3.1.2	Menopausal formication
3.1.3	Drug induced formication

Treatment

Treatment Pathway for DI

History and examination
Diagnosis

Investigation

Engagement

Assessment of:
Risk
Suicidal and homicidal thoughts and plans
Capacity
Shared delusions
Psychosocial comorbidities

Treatment:
Delusion (with antipsychotics, see 5.2)
Skin
Psychosocial comorbidities
Underlying causes
Risk mitigation

Assessment of:
Progress and efficacy of medication
Adherence
Side effects

Continuation of treatment

General Considerations

The most challenging aspect of the treatment of DI is to engage the patient. This requires the development of trust that the clinician is on the patient's side, without colluding with the delusion, as this would eventually be counterproductive. To build up trust it is important to investigate specimen the patient brings and to be transparent about the results. If the patient has secondary DI it is always essential to treat the underlying illness as well as the delusional belief. It is important to treat any dermatological or psychiatric complications of DI, including superinfections, skin irritation itching, anxiety or depression.

Pharmacological Treatment

The mainstay treatment of DI is antipsychotic medication. Success rates are high if the patient reliably takes the medication. As a general rule, the target dose for any antipsychotic is about a third of the maximum dose used for schizophrenia. We suggest the use of amisulpride, olanzapine or risperidone (named in alphabetical order) as first-line treatment. First-generation antipsychotics, such as haloperidol or sulpiride are second-line options. Pimozide has been suggested for the treatment of DI by some authors in the past, but despite good efficacy, it is less desirable than the alternatives because of its side effect profile, especially in the elderly. Patients should be forewarned that the information leaflets for the medication will mention *schizophrenia* as the main illness, for which the medication is licenced. It is important to explain that the patient does not suffer from schizophrenia, but that this medication is used off licence, and will help to address the specific symptoms the patient has as well as the distress that arises from those symptoms. In specialist clinics, clinicians often compare this with the use of aspirin in low doses in cardiac illnesses and in high doses for pain. Patients usually understand this analogy. However, they are unlikely to try the medication if they are suddenly confronted with an information leaflet that says *schizophrenia.* A typical starting regime would be amisulpride 100 mg twice daily, which could be increased to 200 mg twice daily. Half these doses should be used in the elderly. An alternative would, for example, be risperidone 1 mg at night, which could be increased to 2 or 3 mg at night.

Antipsychotic Treatment for DI

Medication name	Starting dose	Target dose	Common side effects
Amisulpride	100 mg twice daily (50 mg twice daily in the elderly)	200 mg twice daily (100 mg twice daily in the elderly)	Extrapyramidal side effects such as akathisia (restless legs), muscle stiffness, hyperprolactinaemia
Olanzapine	2.5 mg at night	7.5 mg at night (5 mg in the elderly	Sedation, increased appetite, metabolic syndrome, avoid in diabetics

Medication name	Starting dose	Target dose	Common side effects
Risperidone	1 mg at night (0.5 mg at night in the elderly	2 mg at night (1 mg at night in the elderly	Extrapyramidal side effects such as akathisia (restless legs), parkinsonism, muscle aches, sedation, hyperprolactinaemia
Haloperidol	1 mg twice daily (0.5 mg twice daily in the elderly)	3 mg twice daily (2 mg twice daily in the elderly)	Extrapyramidal side effects, sedation, depression
Sulpiride	200 mg twice daily (100 mg twice daily in the elderly)	600 mg twice daily	Extrapyramidal side effect, Hyperprolactinaemia

The side effects listed are in no way an exhaustive list. All antipsychotics can increase the QT-interval.

Non-Pharmacological Interventions

In theory, one could extrapolate from schizophrenia research that psychosocial interventions would potentially be of benefit for this patient group. However, no actual trials or published experiences exist.

Discussing the Diagnosis and Management with a Patient

It is very important to acknowledge the patient's suffering and to show empathy. It is often vital for the development of a therapeutic relationship to help reduce stress. A clinician should paraphrase the patient's symptoms (for example, 'your itching', 'the sensations') instead of reinforcing them by calling them 'infestations', or questioning them. Clinicians should explain the findings of lab results, including the fact that they have not found any pathogens so far. It is important to reassure the patient that their suffering is believed. At some point, one may end up with the need to agree to disagree. This means that the clinician acknowledges that the patient has the right to have a different opinion, but also that he or she should acknowledge that the clinician has the same right. One may want to remind the patient that as a clinician you would like to find out what causes the symptoms, but an open mind is necessary. Possibly options such as a medical illness, a psychiatric illness (paraphrased as 'illness of the brain'), or a hitherto unknown infestation can all be discussed as possibilities. When it comes to the introduction of medication you may want to introduce an antipsychotic as helpful in alleviating the patient's distress and itching. You may want to engage the patient's relatives or friends with the patient's consent in order to reinforce treatment aims and the interpretation of symptoms for the patient. It is important to assess the risk to the patient and others whom the

patient may believe are infested. Depending on the patient's insight, capacity and best interest, it may sometimes be appropriate to gradually discuss details of the patient's illness and treatment rather than all at the first consultation, in order to help engagement.

Practice Point
Start the patient on antipsychotic medication at the first appointment where possible. Do treat the skin at the same time as the delusional belief.

Practice Point
In patients with shared belief, do not try to persuade them that their relative has a psychiatric disease. Treat the index patient with antipsychotics and skin treatment, but treat only the skin of the patient with shared delusions.

Prognosis

Existing Prognosis Data

Very few trials exist in DI. The only two existing RCTs are very small and from the 1980s when pimozide was widely used, which is why it was used in this trial. Both small trials showed a response in most cases with full remission in half. Remission rates are even better in systematic collections of case series, although publication bias cannot be excluded. It is fair to say though that if the patient can be persuaded to take an antipsychotic, response rates are excellent and remission rates are consistently about 50%. In one trial with a depot antipsychotic, remission rates went up to 73%, but obviously compliance is guaranteed in such cases. There seems to be evidence that some patients respond to a change of antipsychotics, but none of these case series tested for compliance by blood level analysis. It appears that whilst response rates are similar between first and second-generation antipsychotics, there could be higher remission rate with first-generation antipsychotics. However, these data are based on case reports only. Most data are available for risperidone and olanzapine with partial or full remission rate of around 70%. Amisulpride is particularly interesting from a theoretical point of view as an exclusively dopaminergic antipsychotic. However, whilst results are promising there is less data available compared to risperidone, olanzapine, or first-generation antipsychotics. It does follow from the existing data that the criterion of remission alone would favour first over second-generation antipsychotics with limited overall available data. However, the other important aspect when choosing any medication is the possibility of side effects, which is generally higher with some of the first-generation antipsychotics. Particularly pimozide has significant side effects in the elderly and is therefore no longer the first-line antipsychotic of choice. Improvements are also significant when

measured on the Clinical Global Impression Score (CGI-S) with average changes of between 2.2 and 3.3 out of 7 in patients who confirm compliance with medication. The average improvement decreases with a longer duration of untreated psychosis, making early intervention more likely to be successful.

Practice Point
Delaying effective treatment leads to worse outcomes.

Duration of Treatment

Little is known about follow-up and duration of treatment but general advice about psychosis would suggest 2 years of treatment after symptoms subside. Some limited case series data suggest that DI relapse rates are in the region of 20% after 1 year of treatment.

Considerations of Insight

It is not necessarily likely that patients regain full insight but the aim for remission should be a full cessation of symptoms. Many patients will suggest that the parasites or pathogens have now gone, but patients may not necessarily have become convinced that they had a psychiatric illness.

Considerations of Capacity

Many patients struggle with the concept that they have a psychiatric illness, because other than the one delusional belief they do not show any symptoms they associate with psychiatric illnesses. However, the delusional belief system makes it difficult for patients to weigh up information in a rational way, as they are convinced that the source of their alleged infestation needs to be found. This impairs the capacity to a point that they may not have treatment decision-making capacity. In such cases, clinicians may have to consider the use of relevant national capacity or mental health legislation. This is particularly the case when there are clear risks to the patient or others.

Practice Point
What to do if the patient is not engaging is a common question with DI patients. It works to keep trying. One may have to agree to disagree and not be too dogmatic about forcing one's own medical explanation on the patient. In severe cases mental health legislation may have to be considered.

Practice Point
If the patient is taking recreational drugs it is important to make the patient aware that this can cause the symptoms. A referral to local substance misuse services is often indicated with the patient's agreement.

Practice Point
If the patient develops extrapyramidal side effects (EPSE) from antipsychotic medication it is often sufficient to add antimuscarinic medication such as procyclidine 5 mg tds or orphenadrine 50 mg tds. Alternatively, a reduction of the antipsychotic dose can reduce EPSE if a dose reduction does not jeopardise efficacy and symptom control.

Additional Guidance

7.1 The British Medical Journal published advice on how to approach delusional infestation as part of their Practice Pointer series, including a podcast (see references).
7.2 The British Association of Dermatologists is due to publish guidelines on delusional infestation in 2021.

References

Freudenmann RW, Lepping P. Delusional infestation. Clin Microbiol Rev. 2009 Oct;22(4):690–732.
Lepping P. podcast delusional infestation. BMJ. 2015 https://soundcloud.com/bmjpodcasts/delisional-infestation or as an article https://www.bmj.com/content/350/bmj.h1328
Romanov DV, Lepping P, Bewley A, Huber M, Freudenmann RW, Lvov AN, Squire SB, Noorthoorn EO. Duration of untreated illness and outcome in delusional infestation. Acta Derm Venereol. 2018 Oct 10;98(9):848–54.

Psycho-Dermato-Oncology: Psychological Aspects of Skin Cancer

13

Andrew G. Affleck and Lesley Howells

The aims of this chapter are

- To describe the patient experience of being diagnosed and treated for skin cancer.
- To suggest ways to assess for distress and psychosocial morbidity.
- To describe the impact on the patient of breaking bad news and provide a best practice model.
- To describe the physical and emotional impact of treatments for skin cancer and the psychological support required.
- To illustrate with case studies specific psychological and emotional issues that may arise and their management with psychological interventions.

Introduction

Skin cancer has not received the same recognition as other types of cancer. Affected individuals may receive dismissive responses from others based on common misconceptions including: skin cancer only happens to people who sunbathe, skin cancer is rare, it can just be cut away, or it is not life threatening or serious. However, skin cancer is not rare and indeed is the commonest form of cancer; incidences are projected to rise by 7% annually. An individual's thoughts, feelings and behaviours form an integral and unique part of their illness experience. When treating a physical disease like skin cancer, busy health care professionals may overlook assessing

A. G. Affleck (✉)
Department of Dermatology, Ninewells Hospital and Medical School, Dundee, UK
e-mail: andrew.affleck@nhs.net

L. Howells
Maggie's Centres, London, UK
e-mail: Lesley.Howells@maggiescentres.org

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_13

and enquiring about the person's subjective experience. People developing a skin cancer form a diverse group with biomedical differences in tumour biology and so a spectrum of morbidity and mortality risk as well as other inter-individual differences that influence their experience and ability to cope (Winterbottom and Harcourt 2004). Individuals with life-threatening tumours may cope better than individuals with biologically benign local skin tumours and so one cannot be judgemental. Most research has been done on melanoma, the commonest life-threatening skin cancer, the incidence of which continues to rise. However, the vast majority of skin cancers arise from keratinocytes to form either basal cell carcinomas and low-risk squamous cell carcinomas (non-melanoma skin cancer) — these types only invade the skin locally and are eradicated by surgical excision. More aggressive skin cancers are rare, but metastases can arise. It is important to give patients accurate clinical information to avoid disproportionate distress; the word 'carcinoma' or 'cancer' can lead to catastrophic thinking in some individuals even though an explanation of the localised nature of the tumour has been emphasised; in this regard, some clinicians prefer using alternative terms —e.g. basal cell epithelioma or indolent growth of epidermal origin.

The principle goals in the treatment of skin cancer are to treat primary cancer, prevent disease recurrence and promote long-term survival. Surgically, physical function is preserved as much as possible and aesthetic outcome optimised by good reconstructive technique. Another important objective that has been neglected is to actively take measures to minimise the patient's distress which can be considered "the 6th vital sign" (Holland and Wiesel 2017). Distress, in the context of skin cancer and skin cancer treatment, can be caused by multiple factors each of which can be managed (Table 13.1). Psycho-dermato-oncology may be defined as 'the assessment and management of distress, psychosocial, emotional and behavioural factors associated with the diagnosis of skin cancer and its treatment'.

The Patient Experience of Being Diagnosed and Treated for Skin Cancer

Distress can occur at any part of the patient's healthcare 'journey'— before diagnosis, after diagnosis, during surgery or other treatment and after treatment. People and their families faced with a sudden life change such as a serious skin cancer diagnosis may experience a series of intense traumatic emotions as they try and adjust to this new reality and attempt to cope with the new challenges cancer creates for relationships, their home and working life, their sense of self and the loss of the future they had assumed for themselves. Emotions can intensify or appear unexpectedly at critical times in the cancer journey, e.g. during the staging process at diagnosis, in preparation and response to potentially disfiguring surgery, at the end of treatment, during survivorship when the person encounters physical late effects and psychological hurdles as they try to regain a sense of normality, upon recurrence and when facing a terminal prognosis.

Table 13.1 Targets of psycho-oncological interventions

1. Physical symptoms
Pain
Fatigue
Sexual function
Insomnia
Cognitive disorders
Symptoms specifically related to cancer type/localisation or form of treatment
2. Emotional problems
Psychiatric comorbidities
Fear of progression
Fear of local recurrence
Fear of new primary skin cancers
Other subthreshold psychological conditions, e.g. demoralisation, irritable mood, hopelessness, health anxiety, illness denial, fear of death
3. Assistance with practical/social problems
Return to work
Financial problems
Travel insurance
Life insurance
Child care
Housing
4. Family problems/support to caregivers
5. Spiritual aspects
Religious concerns
Meaning/personal growth
Death/bereavement
6. Improvement of general health
Lifestyle
Nutrition
Exercise
Stress management
Relaxation
7. Optimising treatment
Adherence to treatment (medical/surgical)
Treatment decisions
Use of analgesics
Use of alternative medicine

Surgery is almost always part of the treatment for skin cancer and a degree of stress is to be expected (Augustin et al. 1999). Tips to help reduce patient anxiety and make the experience as positive as possible during surgery have been described (Shenefelt 2010; Mitchell 2008). Larger surgical defects require more complex reconstructions. Most patients achieve satisfactory functional and aesthetic outcomes, although there are certain procedures that are more likely to result in

significant disfigurement, e.g. removal of whole or part of the nose (Moolenburgh et al. 2009), large excisions of the lip and 2 cm wide scar re-excisions for intermediate/thick melanoma often requiring split-thickness skin graft repair and partial amputation of an ear (Fig. 13.1). The use of a mirror to show the planned size of excision before surgery may help prepare a patient as can the use of clinical photographs of similar reconstructions to the one proposed. How closely the actual size of the scar matches pre-surgery expectations, is associated with the resultant emotional distress. Even smaller surgical excisions with apparent good aesthetic outcomes as judged objectively by the surgeon can cause the individual patient quite marked subjective distress. It is the patients' own perception of the outcome that is most important (Brown et al. 2010). The use of skin camouflage can improve

Fig. 13.1 Visible difference of ear secondary to partial amputation to excise a high-risk squamous cell carcinoma

Fig. 13.2 Visible difference of nose after excision of a basal cell carcinoma and full-thickness skin graft repair

aesthetic outcomes (Figs. 13.2 and 13.3). Caddick et al. (2012) studied psychological outcomes following surgical excision of facial skin cancer and found that although female and younger patients were more vulnerable to anxiety preoperatively, surgical excision of facial skin cancers improved social, emotional and cosmetic well-being. Rhee (2007) also found that the extent of disease was a factor in the quality of life scores. Lesions requiring less extensive reconstruction (e.g. direct closure) were associated with a more positive outlook. This might be explained by the patient's perception that less complex surgery meant less serious disease.

Adjustment to becoming visibly different, e.g. after cutaneous surgery, is a large topic and is reviewed in detail by Thompson and Kent (2001). The psychological impact of scarring includes disruption to activities of daily living; anxiety and depression; isolation; and altered body image (Brown et al. 2010) and cannot be assumed on the basis of age, gender, extent, severity or visibility of the disfigurement. Self-esteem is closely associated with body image and it is not surprising that patients with scars often report anger, frustration and low self-esteem. Chronic low-grade skin cancer precursors, e.g. actinic keratoses can cause a marked visible difference which can be upsetting to affected individuals (Fig. 13.4). Topical treatment with chemotherapy cream causes marked redness, which some people find embarrassing.

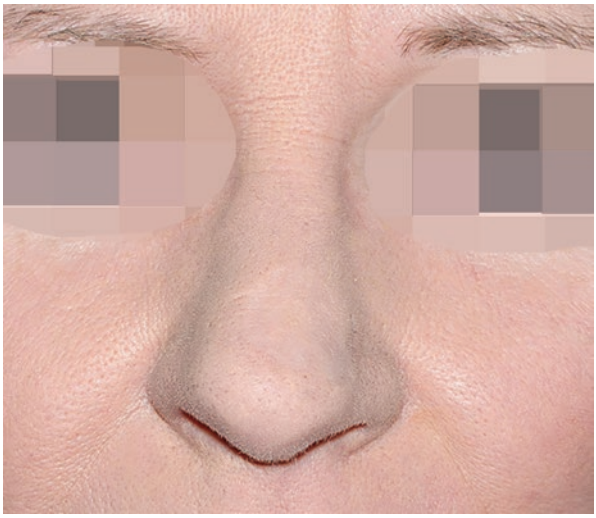


Fig. 13.3 Improvement of appearance and self-confidence with the use of skin camouflage

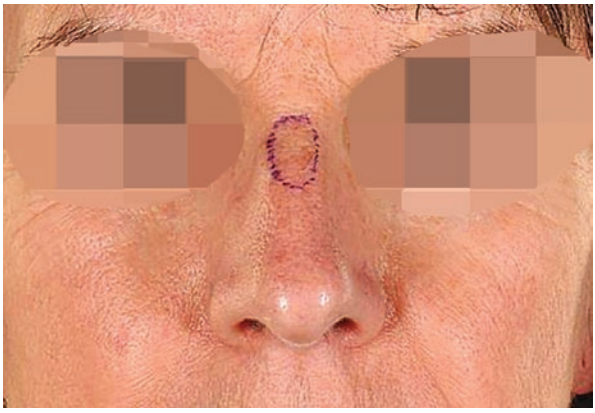


Fig. 13.4 Localised basal cell carcinoma on proximal nasal dorsum marked pre-surgery with catastrophic thinking that extensive subclinical spread is present and all the nose is involved

Winterbottom reported a qualitative study of 16 patients interviewed about their experiences of being diagnosed with skin cancer. Similar themes for melanoma and non-melanoma cancer were identified. Information and knowledge were key themes and influenced experience in different ways. People with melanoma used a wider range of strategies to cope with the diagnosis. Satisfaction with care experienced played a crucial role in minimising the adverse experience for the patients. Increased levels of anxiety were noted in patients who felt their diagnosis had not been explained with clarity. Patients often try to make sense of the disease trying to make a logical explanation as to what the cause might be. A common coping strategy that can enhance self-esteem involves trying to minimise the experience, using comparisons with others who are felt to be worse affected than themselves, comparing their

skin cancer with other serious cancers or diseases. Other coping styles include information seeking or indeed avoidance of information as well as using social support (Roberts et al. 2013). In some people changing behaviour helped them to adapt to the disease, e.g. in minimising sun tanning. Patients often tried to rationalise causes for the condition in an effort to make sense of the diagnosis as a way of restructuring their lives and also possibly to externalise any feelings of guilt that they may have.

Knowledge about patients' experience having skin cancer and different coping strategies enable health professionals to relate to patients better and have productive discussions. Specific concerns raised by patients can be addressed. Common reactions to the diagnosis of skin cancer include shock, fear, uncertainty, worry, guilt, helplessness, gratitude and resentment (Burden-Jones et al. 2010). The emotional impact in response to the challenges cancer brings may be overwhelming to some people; it compromises their ability to function on a day-to-day basis and affects their capacity to hear, retain, understand and act on information. Individuals may feel a loss of control, isolation and helplessness and may struggle with uncertainty, fears about their future and life expectancy. Positive outcomes include adopting a participatory stance, e.g. long-term improved sun-protective measures for self and the need to 'spread the word', advising others to use sunblock and avoid sunburning, a need to live life to the full, encouraging others to have any skin blemishes checked, more empathy towards others who have had cancer, increased awareness of skin with a lower threshold to have any new or changing skin lesion checked, and finding a positive meaning, e.g. deepening of friendships and relationships and coming to terms with their own mortality.

A sensible balance needs to be achieved and some patients initially (and a few continue long-term) adopt extreme measures which are disproportionate and unhelpful and so contribute to decreased life quality, e.g. some people stop going on sunny holidays, constantly checking skin, stopping previously enjoyed outdoor pursuits, and persistent guilty feelings for not having taken more care to protect skin from the sun. Hope, optimism and self-esteem should be maintained (Kneier).

Patients with advanced versus localised disease had more supportive care needs, particularly the amount, quality and timing of melanoma-related information, communication with and emotional support from clinicians (Dunn et al. 2017).

Rarely, an individual may feel pessimistic and without hope and develop depression and suicidal ideation. Completed suicide can arise after the diagnosis of a skin cancer; those with a poor prognosis, low survival rate and limited treatment options tend to be at the highest risk especially in the first 6 months after diagnosis (Wang et al. 2018).

Several different coping strategies exist—more than one may be used at one time and different strategies are used over time. Some strategies are considered better than others in promoting adjustment and reducing distress (Kasparian et al. 2009).

Fig. 13.5 Neglected deeply invasive ulcerated basal cell carcinoma on ear helical rim—cartilage has been destroyed by the tumour

Maldaptive coping is associated with adjustment disorder and can be seen after skin cancer surgery. Denial of illness as a coping mechanism is not uncommon and can lead to neglect of a skin cancer with delay in treatment and so potential adverse outcomes (Flynn 2004) (Fig. 13.5). Catastrophic thinking is not uncommon in relation to fear of recurrence and concern regarding the size of the primary tumour (Figs. 13.6 and 13.7).

Most patients do cope and adjust overtime, putting the experience of having skin cancer behind them and continue with their lives without lasting psychological consequences. Qualitative studies examining patient narratives show common themes, for example, for the rare but life-threatening skin cancer Merkel Cell Carcinoma (Kaufman et al. 2018):

- Several tests and visits needed to establish the diagnosis
- Misdiagnosis
- Lack of information
- Growing painless lump
- Shocked, scared by the diagnosis
- Unaffected daily routine and general physical condition
- Anxiety-related sleeping difficulties
- Fighting the disease
- Role of supportive relatives and friends
- Patients' experience with chemo and radiotherapy and expectations toward a study treatment in Merkel Cell Carcinoma
- Limited perceived efficacy of chemotherapy: success/relapse/failure
- Limited perceived efficacy of radiotherapy: success/relapse/failure
- Tolerability issues
- Disrupted activities
- Expectations towards the study treatment

Fig. 13.6 Healthy scar, left temple, 3 months after cutaneous surgery to excise a BCC. The patient was convinced that there was persistent tumour due to sensory symptoms consistent with neuropraxia causing 'stabbing pains'. A careful explanation and reassurance regarding wide excision margins helped her to understand the nature of her symptoms and spontaneous resolution occurred over the next 3 months

Fig. 13.7 Widespread actinic keratoses impacting on daily quality of life causing frustration and embarrassment

Psychological Intervention

The potential benefits of high-quality psychological care in a cancer setting are clear however, high-quality evidence is lacking (Peters 2012). About one in five people will be at least moderately distressed when diagnosed with skin cancer and among patients with malignant melanoma, the number is higher with one in three patients reporting clinically relevant levels of psychological distress in a systematic review (Kasparian et al. 2009).

With new treatments for metastatic disease, has come a more intensive clinical surveillance program for many high-risk patients, usually including imaging of asymptomatic patients, and this has increased the potential psychological burden. A systematic review of psycho-educational interventions for melanoma survivors identified four psychological intervention studies (amongst a total of 27 studies encompassing educational and psycho-educational interventions), and concluded that significant reductions could be achieved in common psychological symptoms including anxiety, depression and cancer-related distress (McLoone et al. 2013).

However, Dunn et al. (2017) reviewed qualitative and quantitative evidence for psychosocial outcomes for advanced (stage III/IV) melanoma patients. They concluded that high-quality trial evidence is still needed to clarify the impact of treatment innovations for advanced melanoma on patients' psychosocial well-being stating that 'Survivorship research and subsequent translation of that knowledge into programs and services currently lags behind gains in the medical treatment of advanced melanoma, a troubling circumstance that requires immediate and focused attention'.

Fig. 13.8 Holistic patient information leaflets on skin cancer produced by MacMillan

However, whilst awaiting trial evidence, health professionals can still improve their confidence and skills in talking with such patients who may be confused, scared and ashamed of their beliefs and feelings. Good relational skills are needed to help develop a therapeutic alliance with the patient. Effective communication is critical (Fallowfield and Jenkins 1999).

Generally, it is recommended that all patients who show distress, have low levels of social support and experience cancer-related difficulties in daily life, should be offered psychological support regardless of whether they have a diagnosed psychological disorder. The supply of relevant self-help materials can help some patients through the adjustment process, e.g. MacMillan cancer information booklets and online https://www.macmillan.org.uk/ (Fig. 13.8).

Psychosocial and Quality of Life Assessment

Routine screening for distress by the medical team is recommended to identify vulnerable patients and enable the effectiveness of psychosocial support to be enhanced by tailoring it to a person's current need. However, communication about psychosocial issues is delicate. There is evidence that many clinicians do not systematically inquire into the emotional problems of patients, and many clinicians prefer patients to bring up a problem.

On the other hand, patients may have trouble sharing emotional difficulties, and some do not want to address distress at all. A balance is required and an open invitation to patients to discuss matters if and when they wish is desirable. Distress is frequently not voluntarily disclosed to the person's medical team, so potentially alleviating psychosocial support is not provided. A baseline assessment of the patient's mood, ideas and expectations regarding their skin cancer and its treatment is desirable. Talking with patients about their experience and asking about specific concerns and emotional well-being should be routine practice. This empathic interaction is itself an essential element of an effective screening procedure. Screening for distress and difficulty coping related to skin cancer can be done using key questions, simple visual analogue scales and when needed, using other validated tools, e.g. include the 'Distress thermometer' (Cutillo et al. 2017), an ultra-short measure of psychological and practical distress; the Skin Cancer Index (SCI) (Rhee et al. 2007), a skin cancer-specific screening tool; the 'Hospital Anxiety and Depression Scale' (HADS) Zigmond and Snaith (1983) (Stern) which provides clinical cut off points.

A recent review recommended the use of the cancer-specific EORTC QLQ-C30, especially in late stages of disease, and the melanoma-specific FACT-M and skin cancer-specific SCI questionnaires as these instruments have been well validated and used in a number of studies (Chernyshov et al. 2019). Using a screening tool positively influences communications about psychosocial issues and distress.

Some degree of anxiety and stress is common in any individual having skin surgery; it would appear that only a minority are extremely anxious. Patients' desire for psychosocial support is an independent factor and may not correlate with high distress (Buchhold et al. 2016). Patient self-evaluation is an important instrument to identify patients who need psycho-oncological support (Mayer et al. 2017).

The commonest patient-reported reason for refusing psychosocial support services is that they feel no subjective need for such input. Broader reasons include; a preference for self-managing symptoms, not feeling distressed enough, a belief that their distress is not severe enough, a belief that help would be ineffective and receiving sufficient support from family and friends. Another important barrier is context-related when patients report they lack information about the availability of psychological support services.

Both a patient's desire for normalcy and their lack of information about the potential benefits of psycho-oncological treatment could lead patients to refuse psychosocial support. The subjective norms and information deficits of health care professionals also may influence the choice of patients to use psycho-oncological support services.

With a better understanding of the determinants and barriers along the distress screening pathway, access for underserved groups of distressed cancer patients can be increased and resources in psychosocial cancer care utilised efficiently leading to optimal management of patients.

NICE states that

1. 'During follow-up of patients treated for skin cancer there should be provision of psychological and emotional support to patient, carer and family'.
2. 'Those who are directly involved in treating patients should receive specific training in communication and breaking bad news. They have a responsibility for good communication with patients and carers'.
3. 'Skin cancer patients should have access to psychological support services'.
4. 'There should have at least one skin cancer clinical nurse specialist (CNS) who will play a leading role in supporting patients and carers'.
5. 'Skin cancer CNSs who have received training will be better equipped to identify and assist with the management of patients with psychosocial needs'.
6. 'All people with cancer should be offered access to timely and tailored psychosocial support'.

The nature and frequency of follow-up are influenced by the level of distress. Different levels of intervention can be provided by different NHS professionals and supportive care charities such as Maggie's Cancer Centres and Macmillan. Maggie has the advantage of offering all the NICE recommended levels of emotional, social and practical support under one roof adjacent to the NHS Cancer Centres and on a drop-in and more traditional referral basis (www.maggiescentres.org), (Lang-Rollin and Berberich 2018; National Institute for Health and Clinical Excellence 2006).

The NICE model emphasises the importance for all professionals involved in the person's treatment to appreciate their role in enhancing psychological well-being. In response to the challenges of cancer, people differ in their coping style, resilience to adversity and previous potentially debilitating life experience. Many people can be adequately supported through the crucial emphasis by their medical team on high-quality communication, through which concerns are elicited, listened to and acknowledged; bad news is delivered with empathy; complex information is offered in a paced and understandable form; anxieties displayed during surgical procedure are managed, and additional cancer support services (e.g. Maggie's) are signposted.

Screening and psycho-education are also within the remit and expertise of many MDT members. In particular the Clinical Nurse Specialists or Allied Health Professionals can assist in cancer-related problem solving, coaching in lifestyle change post-cancer (e.g. managing the risk of sun exposure) and guidance in the choice and use of psychological self-help materials and resources (e.g. Changing Faces as a support for coping with disfigurement). The medical team is also pivotal in collaborating with psychological specialists in the provision of Psycho-educational group support, particularly in the early stages after diagnosis when

personalised and understandable information is critical in managing anxiety and optimising a person's natural coping mechanism. Patients identified through screening or consultation as particularly vulnerable should be referred for prompt assessment by a psychological specialist (e.g. psychologist, psychiatrist either in the NHS or voluntary sector) to gauge the level of intervention required and determine whether psychotropic medication is indicated. Ideally, this specialist should work in close flexible liaison with the medical team. It is important to note that the level of psychological distress does not necessarily relate to perceived need for professional help, so patients may not seek or accept help even if it is indicated, therefore close monitoring and repeated recommendation is vital to ensure help is available when the person sees the need (e.g. the CNS monitors distress routinely at follow-up clinics).

Breaking Bad News

It is important to optimise initial discussions and engagement when giving a significant diagnosis. Having the person's partner present is helpful as is the provision of a written summary of the key facts and a contact details for a skin cancer clinical nurse specialist.

NICE guidance places particular emphasis on MDT members having training in Breaking Bad News. With its attention to empathy and inclusion of the patient, the SPIKES protocol has contributed immeasurably to professional practice (Baile et al. 2000). This seminal study is highly recommended reading material and is available free open access online at http://theoncologist.alphamedpress.org/content/5/4/302.full.pdf. However, Dean and Willis (2016) suggest that new evidence and changes in the context of care indicate several additions to the six steps of SPIKES.

SPIKES
*S*etting up the interview, assessing patient
*P*erception
*I*nviting patient to clarify how much they wish to be told
give *K*nowledge to the patient, address patient's
*E*motions with empathic responses
*S*ummarise the discussion and form a strategy for ongoing management

SPIKES is particularly notable for looking beyond breaking bad news as a single interview, the availability of multidisciplinary support for patient following the news and opportunity for the health professional to reflect on the emotional impact of having breaking bad news as a routine aspect of their clinical remit.

The PEWTER model also provides a useful mnemonic for defining a framework to communicate significant, life-changing news to a patient effectively (Keefe-Cooperman and Brady-Amoon 2013):

*P*repare—Know what information will be presented and understand how to present it in clear language. Provide an unhurried and uninterrupted meeting with the person(s) receiving the difficult news.

*E*valuate—Assess what the patient and family members already know or suspect and their present psychological and emotional status.

*W*arning—Give the patient an indication that serious news will be presented.

*T*elling—Give the information in a straightforward, nonapologetic calm manner in small pieces at a time. One should pause intermittently to confirm understanding prior to disclosing more information to ensure that the person is not overwhelmed.

*E*motional response—Assess the person's reaction to the news and consider arranging another meeting for further discussion if necessary.

*R*egrouping preparation—Patient/doctor collaboration in response to the news emphasising realistic hope and aims for short and long term. Motivate engagement and identification of new goals.

Specific Forms of Psychosocial Support

It is seldom that a person will have a distinct diagnosable psychological disorder in response to their skin cancer diagnosis. Many cancer fears have a rational basis so traditional psychological approaches have been adapted for this population with an increasing evidence base supporting their effectiveness. The forms of support described are: Psycho-education Groups, and Cognitive Behaviour Therapy (CBT) Techniques, specifically 'Third Wave CBT' techniques which are particularly suited to physical health-related psychological problems. They include Mindfulness, Self-compassion and Acceptance and Commitment Therapy (ACT) (Hulbert-Williams et al. 2018). Most notably ACT techniques formed the key components of the CONQUER FEAR Trial (Butow et al. 2017). ACT is a trans-diagnostic form of

Cognitive Behaviour Therapy that enhances emotional self-regulation, the ability to live with uncertainty and the person's willingness to accept the irrevocable impact of their cancer. An integrative approach enables the therapist to develop specific tools from a range of theories that are often used within a core framework, including CBT and existentialism.

Melanoma survivors may benefit personally from sharing their cancer experience online and professionally facilitated support groups such survivor narratives can motivate behaviour change and facilitate coping among readers (Banerjee et al. 2018).

Psycho-Education

Open disclosure of negative feelings can be therapeutic. Normalisation of these feelings can help in patient adjustment. Psycho-education in individual and group format with people with skin cancer has been found to be acceptable and effective with both melanoma and non-melanoma skin cancers when particular emphasis is placed on building or fine-tuning active Problem-Focused Coping strategies and reducing avoidant coping strategies (Dieng et al. 2017).

Psycho-education is particularly successful in a group format, as the participants become a natural therapeutic support network, becoming role models for each other, exchanging experiences and empathising with each other's challenges. Ideally, the group is facilitated by a psychologist and CNS specialising in skin cancer. There is a different theme each week with open discussion and 'homework'. Handouts and online resources are utilised, and should be offered at key transition points in the cancer journey, for example, at diagnosis and when active treatment is completed. A brief, patient-centered psychological intervention in reducing fear of cancer recurrence comprising a 76-page psycho-educational resource and three individually-tailored, telephone-based sessions with a psychologist was found to have continued benefit 12-months post-intervention (Dieng et al. 2019a, b). The primary outcome was the level of self-reported fear of new or recurrent melanoma using the Severity subscale of the Fear of Cancer Recurrence Inventory (FCRI). Ideally, this type of implementation could be part of routine melanoma care (Kasparian et al. 2016).

Framework for Psycho-Education Group at Diagnosis

- *Health Education specific to skin cancer diagnosis*
- *Stress management techniques (*e.g. *what is stress and why now? Relaxation, Mindfulness techniques and managing anxious thoughts)*
- *How to use Active Coping strategies and Problem Solving techniques (*e.g. *managing relationships at home, with work, with your medical team and GP)*

As treatments become more successful in achieving remission and in managing cancer as a chronic illness, people have to cope with additional psychological, physical and practical challenges, for example, resuming work and family responsibilities that were 'shelved' during treatment; coping with late effects of their cancer

treatment and disfigurement; and navigating existential hurdles to find the means to invest fully in life knowing their life expectancy is limited. For many, cancer is also a catalysis for essential lifestyle change in exercise, nutrition and risk behaviours such as sun exposure. Psycho-education groups post-treatment are designed to assist people make the challenging transition between active treatment and building the life they want beyond cancer.

Framework for Psycho-Education Group at the End of Active Treatment

- Practical and experiential introduction to exercise, nutrition and stress management.
- Managing risk behaviour such as sun exposure.
- Cognitive behavioural therapy techniques to help live with uncertainty and fears of cancer recurrence and look afresh at work, home life and relationships and accommodate changed priorities.
- Training in how to build and utilise effective post-treatment partnerships with medical teams to enable smooth communication to enhance the monitoring of disease recurrence and late effects of treatment (Brown et al. 2019)

Cognitive Behavioural Therapy Based Techniques Including 'Third Wave' CBT

There is an established evidence base for the use of CBT, and increasingly, Third Wave CBT techniques in managing psychological problems associated with cancer when delivered by a Psychologist or accredited Mental Health professional (Hulbert-Williams et al. 2018). The goal of therapy is to help a person perceive their cancer and its irrevocable consequences adaptively; they haven't chosen to have cancer and its trajectory *but* they can choose how to respond and the extent to which it impacts on their life.

In the context of a non-judgemental therapeutic relationship, the patient is encouraged to:

- Connect with personal values as a compass to determine what matters to a person and how they wish to live their life, e.g. in relation to family, friends, work, health and leisure
- Explore how and why a person's psychological response to skin cancer (e.g. fear of recurrence) impacts on their ability to live the life that matters to them.
- Use psychological techniques and metaphor to help them become more self-aware, understand the psychological impact of their skin cancer and respond with greater psychological flexibility, so enabling them to stay connected to their values and live meaningfully despite the implication of their diagnosis. Psychological techniques include present moment awareness exercises, cognitive diffusion techniques, meditation and mindfulness activities for daily life, mindful movement (e.g. yoga), and self-compassion approaches.

- Develop a willingness to be more accepting of uncertainty and change; manage difficult intrusive thoughts, disturbing emotions and sensations; overcome avoidance and commit to living life more aligned with their values.

Case Histories

In this section four case studies will be used to illustrate the common and frequently overlapping forms of distress people experience and introduce the psycho-educational, and psychological therapy support that can be used.

1. Lisa's Story: Nurse-led individual psycho-educational support

   Lisa was diagnosed and successfully treated for BCC associated with previous sunbed use. During a clinic appointment and two follow-up telephone calls, she forms a confiding relationship with her CNS. She describes her guilt and embarrassment about her diagnosis and her frustration with her family who are telling her to 'move on' before she feels she has processed the trauma of having had cancer. She identifies the opportunity to talk in confidence with someone outside her friends and family as central to her improved emotional wellbeing, together with advice about lifestyle changes.
2. Jen's Story: Psycho-educational group intervention

   Jen is a housewife and child-minder in her late 50s with Malignant Melanoma. Her surgery was successful, but she could not receive adjuvant treatment due to a kidney transplant 12 years previously. In response to her diagnosis her mood immediately lowered, she became increasingly irritable and withdrawn. She took immediate and extended leave from her child-minding and gradually stopped activities that previously had given her a sense of accomplishment and pleasure. Her sleep was disturbed with panicky thoughts and agitated pacing. Her thinking was characterised by hopelessness, stating that she considered her life as now 'in limbo' until her eventual death. Jen was encouraged by her CNS to participate in psycho-education groups during and after her treatment to introduce her to others with similar fears, help her develop coping strategies to manage her emotions, particularly fear of recurrence and to build value into her life again by resuming pre-cancer activities that previously gave a sense of self worth. She also cultivated new activities such as a passion for exercise.
3. Sharon's Story: Individual psychological therapy for disfigurement

   Sharon is a 40-year-old single Mum with a son in early adolescence. She has no support from the child's father, struggles financially and has recently been made redundant. A year ago she was diagnosed with Gorlin Syndrome for which she has since undergone three facial Mohs Surgery procedures. Her score on the Skin Cancer Index indicated significant distress associated with her perceived facial disfigurement and fears that her attractiveness will be further compromised by future surgery. She finds the relinquishing of control during surgery frightening and insists on being treated by a trusted surgeon. She obsessively checks for the sign of recurrence and ruminates through the day about potential

lesions. Fearing negative judgment from others, she has become socially isolated with a resultant lowering of mood and despite information to the contrary she fears her cancer will spread to other organs. Sharon was referred to a psychologist by her Consultant Dermatologist after routine screening for distress indicated her vulnerability.

A CBT for disfigurement approach was used. Initial concerns about the impact on attractiveness were addressed through questioning the validity of her self-appraisal and using evidence that contradicted her overarching belief that she was ugly. Resources from the charity 'Changing Faces' (www.changingfaces.org.uk) were pivotal in guiding therapy sessions. The aim was to help Sharon improve her confidence and manage her shameful thoughts around her scarring to overcome her social avoidance so she could live a valued life whilst coping with the often harsh societal norms for attractiveness.

4. Peter's story: individual psychological therapy for fear of recurrence and adjustment to terminal diagnosis

Peter is a senior manager in the public sector diagnosed with malignant melanoma that was initially treated with surgery and adjuvant chemotherapy. As a proud, stoical father of two children in their early teens, he saw his role as protecting his family and so did not disclose troubling thoughts and emotions. He used an avoidant coping strategy by distracting himself from difficult emotions without processing the significance of what he had come through and focused on 'getting back to normal' as quickly as possible. He returned to work without a phased return and resumed his fulltime responsibilities despite profound fatigue, sleeplessness, hypervigilance for any signs of recurrence, intense irritability and anxiety prior to routine monitoring appointments, daily graphic flashbacks to his chemotherapy and intrusive thoughts and images featuring a painful death. He would experience panic attacks when faced with making plans even for events only a few weeks in the future. When the avoidance of future planning became apparent to his manager he eventually sought help to deal with the psychological aftermath of his cancer treatment and uncertain prognosis. He was assessed as experiencing PTSD symptoms with associated fears of progression and depression. After Peter successfully developed strategies for alleviating and managing the symptoms of his PTSD and depression he returned to work and his wife was relieved by how he re-engaged in family life and planned the holidays that had been a valued feature of his pre-cancer life. Within 18 months he was found to have developed lung and brain metastasis and was given a very poor prognosis but also the chance to receive immunotherapy. He now had the task to prepare himself and his family for the uncertainty, physical and emotional impact of immunotherapy and the potential for death within a short timeframe.

Therapy for Peter at this stage was very typical of people on immunotherapy who live with uncertainty. He used 'Third wave CBT' including ACT and Mindfulness techniques to diffuse his troubling thoughts and focus on the 'here and now' through meditation. He read self-help literature, in particular, 'Facing the Storm, Using CBT, Mindfulness and Acceptance to build resilience when your world is falling apart' (Owen 2012) and 'The Reality Slap — How to find

fulfillment when life hurts' (Harris 2012). In the confidential setting of his psychology sessions he expressed extreme emotions (including anger, guilt, grief) and rehearsed conversations he wished to have with his wife. He was introduced to a weekly support group for people with metastatic cancer at his local Maggie's and gained hope through their lived experience that he could still live a meaningful life (Breitbart et al. 2018). He sought advice through Winston's Wish (www.winstonswish.org.uk), a charity to assist families when a parent has a terminal prognosis, and eventually he and his wife felt able to tell their children that although he was being treated successfully with immunotherapy, his condition was serious and ultimately life-limiting.

Practice Points

1. Routine screening for distress is recommended for individuals diagnosed with a skin cancer.
2. Clear communication of significant news (e.g. a new diagnosis of skin cancer) is recommended using best practice principles in breaking bad news and delivering complex information.
3. Support using empathy and avoid making assumptions of emotional impact on the basis of disease severity. Always check for understanding of information provided but also the emotional impact of the information.
4. Help the person tap into existing resilience strategies and reach out where possible to family and friends for support.
5. Liaison with colleagues in clinical psychology and psychiatry is indicated in individual cases.

References

Augustin M, Zschocke I, Godau N, et al. Skin surgery under local anesthesia leads to stress-induced alterations of psychological, physical, and immune functions. Dermatol Surg. 1999;25:868–71.

Baile WF, Buckman R, Lenzi R, et al. SPIKES - a six-step protocol for delivering bad news: application to the patient with cancer. Oncologist. 2000;5:302–11.

Banerjee SC, D'Agostino TA, Gordon ML, et al. "It's not JUST skin Cancer": understanding their cancer experience from melanoma survivor narratives shared online. Health Commun. 2018;33:188–201.

Breitbart W, Pessin H, Rosenfeld B, et al. Individual meaning-centered psychotherapy for the treatment of psychological and existential distress: a randomized controlled trial in patients with advanced cancer. Cancer. 2018;124:3231–9.

Brown BC, McKenna SP, Solomon M, et al. The patient-reported impact of scars measure: development and validation. Plas Reconstr Surg. 2010;125:1439–49.

Brown L, Allan J, Makinson J, et al. Experiences of a cancer post-treatment group intervention and effects on reported well-being, worry, self-efficacy, diet, and activity. Psychooncology. 2019; https://doi.org/10.1002/pon.4994.

Buchhold B, Wiesmann U, Bahlmann J, et al. Psychosocial burden and desire for support in outpatients with skin cancer. J Dtsch Dermatol Ges. 2016;14:405–15.

Burden-Jones D, Thomas P, Baker R. Quality of life issues in nonmetastatic skin cancer. Br J Dermatol. 2010;162:147–51.
Butow PN, Turner J, Gilchrist J, et al. Randomized trial of conquer fear: a novel, theoretically based psychosocial intervention for fear of cancer recurrence. J Clin Oncol. 2017;20(35):4066–77.
Caddick J, Stephenson J, Green L, et al. Psychological outcomes following surgical excision of facial skin cancers. J Plast Reconstr Aesthet Surg. 2012;65:e257–9.
Chernyshov PV, Lallas A, Tomas-Aragones L, et al. Quality of life measurement in skin cancer patients: literature review and position paper of the European Academy of Dermatology and Venereology task forces on quality of life and patient oriented outcomes, melanoma and non-melanoma skin. Cancer. 2019;33:816–27.
Cutillo A, O'Hea E, Person S, et al. The distress thermometer: cut off points and clinical use. Oncol Nurs. 2017;44:329–36.
Dean A, Willis S. The use of protocol in breaking bad news: evidence and ethos. Int J Palliat Nurs. 2016;22:265–71.
Dieng M, Kasparian NA, Mireskandari S, et al. Psychoeducational intervention for people at high risk of developing another melanoma: a pilot randomised controlled trial. BMJ Open. 2017;7(10):e015195. https://doi.org/10.1136/bmjopen-2016-015195.
Dieng M, Smit AK, Hersch J, et al. Patients' views about skin self-examination after treatment for localized melanoma. JAMA Dermatol. 2019a May 15; https://doi.org/10.1001/jamadermatol.2019.0434.
Dieng M, Morton RL, Costa DSJ, et al. Benefits of a brief psychological intervention targeting fear of cancer recurrence in people at high risk of developing another melanoma: 12-month follow-up results of a randomised controlled trial. Br J Dermatol. 2019b Apr 9; https://doi.org/10.1111/bjd.17990.
Dunn J, Watson M, Aitken JF, Hyde MK. Systematic review of psychosocial outcomes for patients with advanced melanoma. Psychooncology. 2017;26:1722–31.
Fallowfield L, Jenkins V. Effective communication skills are the key to good cancer care. Eur J Cancer. 1999;35:1592–7.
Flynn TC. Denial of illness: basal cell carcinoma. Dermatol Surg. 2004;30:1343–4.
Harris R. The reality slap. New York: Constable & Robinson Publishers; 2012.
Holland JC, Wiesel TW. Principles of psycho-oncology. In: Bast RC, Hait WN, Kufe DW, Weichselbaum RR, Holland JF, Croce CM, Piccart-Gebart M, Wang H, Hong WK, Pollock RE, editors. Holland-Frei cancer medicine. Hoboken, NJ: Wiley; 2017.
Hulbert-Williams N, Beatty JA, Dhillon L, et al. Psychological support for patients with cancer: evidence review and suggestions for future directions. Curr Opin Support Palliat. 2018;12:276–92.
Kasparian NA, McLoone JK, Butow PN. Psychological responses and coping strategies among patients with malignant melanoma. Arch Dermatol. 2009;145:1415–27.
Kasparian NA, Mireskandari S, Butow PN, et al. "Melanoma: questions and answers." Development and evaluation of a psycho-educational resource for people with a history of melanoma. Support Care Cancer. 2016;24:4849–59.
Kaufman HL, Dias Barbosa C, Guillemin I, et al. Living with Merkel cell carcinoma (MCC): development of a conceptual model of MCC based on patient experiences. Patient. 2018;11:439–49.
Keefe-Cooperman K, Brady-Amoon P. Breaking bad news in counselling: applying the PEWTER model in the school setting. J Creat Mental Health. 2013;8:265–77.
Lang-Rollin I, Berberich G. Psycho-oncology. Dialogues Clin Neurosci. 2018;20:13–21.
Mayer S, Teufel M, Schaeffeler N, et al. The need for psycho-oncological support for melanoma patients: central role of patients' self-evaluation. Medicine (Baltimore). 2017;96:e7987.
McLoone J, Menzies S, Meiser B, Mann GJ, Kasparian NA. Psycho-educational interventions for melanoma survivors: a systematic review. Psychooncology. 2013;22:1444–56. https://doi.org/10.1002/pon.3165.
Mitchell M. Conscious surgery: influence of the environment on patient anxiety. J Adv Nurs. 2008;64:261–71.

Moolenburgh SE, Mureau MAM, Versnel SL, et al. The impact of nasal reconstruction following tumour resection on psychosocial functioning, a clinical-empirical exploration. Psycho-Oncology. 2009;18:747–75.

National Institute for Health and Clinical Excellence. Guidance on cancer services. Improving outcomes for people with skin tumours including melanoma: the manual. London: NHS; 2006.

Owen R. Facing the storm: using CBT, mindfulness and acceptance to build resilience when your world's falling apart. New York: Routledge; 2012.

Peters EM. Psychological support of skin cancer patients. Br J Dermatol. 2012;167(Suppl 2):105–10.

Rhee J, Matthews A, Neuberg M, et al. The skin cancer index: clinical responsiveness and predictors of quality of life. Laryngoscope. 2007;117:399–405.

Roberts N, Czajkowska Z, Radiotis G, et al. Distress and coping strategies among patients with skin cancer. J Clin Psychol Med Settings. 2013;20:209–14.

Shenefelt PD. Relaxation strategies for patients during dermatologic surgery. J Drugs Dermatol. 2010;9:795–9.

Thompson A, Kent G. Adjusting to disfigurement: processes involved in dealing with being visibly different. Clin Psychol Rev. 2001;21:663–82.

Wang SM, Cheng JC, Weng SC, et al. Risk of suicide within 1 year of cancer diagnosis. Int J Cancer. 2018;15:1986–93.

Winterbottom A, Harcourt D. Patients' experience of the diagnosis and treatment of skin cancer. J Adv Nurs. 2004;48:226–33.

Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361–70.

Body Dysmorphic Disorder (BDD)

14

Dimitre Dimitrov

Body dysmorphic disorder (BDD) is a disabling illness with a high worldwide prevalence (Hong et al. 2018), yet the condition still remains under-recognized and under-diagnosed (Dyl et al. 2006). People suffering from BDD are concerned with minimal or non-existent defects, develop social avoidance and may become housebound or even suicidal (Helwick 2011; Veale et al. 1996a; Phillips et al. 2005; Phillips and Diaz 1997).

BDD is primarily a psychiatric health problem and patients usually consult dermatologists, plastic surgeons, other specialists or general practitioners, but not mental health specialists, as patients firmly believe that their disease is a physical problem (Philips 1996). Even when their problem is recognized as BDD, it is important to be aware that patients may be resistant to engage with mental health professionals and seek psychiatric help. Instead, they may simply consult other dermatologists or plastic surgeons in the battle to achieve the image of 'perfection'. However, once diagnosed, a holistic psycho-dermatological approach, focusing not only on the disease, but also on the patient's psychological, emotional, physical and social needs should be adopted.

Definition

According to DSM-5, the diagnosis of BDD require the following criteria to be fulfilled see Box 14.1 (American Psychiatric Association 2013).

D. Dimitrov (✉)
Ministry of Presidential Affairs and Sheikh Khalifa Medical City, Abu Dhabi, UAE

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_14

Box 14.1 DSM-5 Definition of Body Dysmorphic Disorder

DSM-5 Definition of Body Dysmorphic Disorder(American Psychiatric Association 2013)

A. *Preoccupation* with one or more perceived defects or flaws in physical appearance that are *not observable or appear slight to others*.
B. *At some point during the course of the disorder, the individual has performed repetitive behaviours* (e.g. mirror checking, excessive grooming, skin picking, reassurance seeking) or mental acts (e.g. comparing his or her appearance with that of others) in response to the appearance concerns.
C. The preoccupation causes clinically significant distress or impairment in social, occupational or other areas of functioning.
D. The appearance preoccupation is not better explained by concerns with body fat or weight in an individual whose symptoms meet diagnostic criteria for an eating disorder.

Prevalence

The prevalence of BDD varies in different studies, but all have found that a high percentage of patients with the disorder presented in aesthetic practices. According to previous studies, the prevalence in the general population is 1.7–2.4%, but in the setting of general dermatology and aesthetic practices, it can reach 7–20.3% (Haas et al. 2008; Phillips et al. 2000; Harth et al. 2009).

Veale et al. performed a systematic review and analyzed the weighted prevalence of body dysmorphic disorder in different settings see Table 14.1 (Veale et al. 2016).

A Systematic Review with Meta-Analysis performed by Ribeiro ascertained the prevalence of Body Dysmorphic Disorder in Plastic Surgery and Dermatology patients 15.04% and 12.65% respectively (Ribeiro 2017)

Aetiology and Pathophysiology

Despite its prevalence, the aetiology and pathogenesis of body dysmorphic disorder (BDD) has yet to be fully elucidated. According to the present understanding, various factors play a role in its complex pathological process (Fig. 14.1).

Neurobiological abnormalities are considered to be associated with certain symptoms of BDD. There is evidence that the susceptibility to BDD may be at least partially heritable and BDD might share genetic factors with other conditions from the group of obsessive-compulsive and related disorders. Studies analyzing visual processing among BDD patients found disturbances in visual perception and

Table 14.1 Presents an estimated weighted prevalence of Body dysmorphic disorder in different settings, by Veale et al. (2016) (modified by Dimitrov D)

Weighted prevalence of BDD in different settings	*n* (%) with BDD		
	Total	Female	Male
Adults in the community	13,773 (1.9%)	(2.1%)	(1.6%)
Adolescents in the community	464 (2.2%)	(2.8%)	(1.7%)
Student populations	3516 (3.3%)	(3.6%)	(2.2%)
Adult psychiatric inpatients	788 (7.4%)	(9.6%)	(5.6%)
Adolescent psychiatric inpatients	229 (7.4%)	(6.9%)	(3.5%)
Adult psychiatric outpatients	765 (5.8%)	(6.5%)	(4.6%)
General dermatology outpatient settings	914 (11.3%)	(13.4%)	(14.0%)
Cosmetic dermatology outpatients[a]	60 (9.2%)	–	–
General cosmetic surgery	2291 (13.2%)	(10.9%)	(15.3%)
Rhinoplasty	1001 (20.1%)	(16.7%)	(18.4%)
Orthognathic surgery	259 (11.2%)	(13.2%)	(8.0%)
Orthodontics/Cosmetic dentistry	480 (5.2%)	(7.9%)	(2.5%)
Vulvo-vaginal surgery	49 (18.4%)	(18.4%)	–
All cosmetic surgery and cosmetic dermatology settings[b]	(12.2%)		
Acne clinics[a]	32 (11.1%)	–	–
Female ballet dancers[c]	19 (10.5%)	–	–
Male weight lifters[c]	648 (10.6%)	–	–

[a]A weighted prevalence for males and females could not be calculated as only one study provided details on gender
[b]Estimated prevalence for all 23 cosmetic surgery settings
[c]As these were the only studies of this kind, weighted prevalence could not be calculated

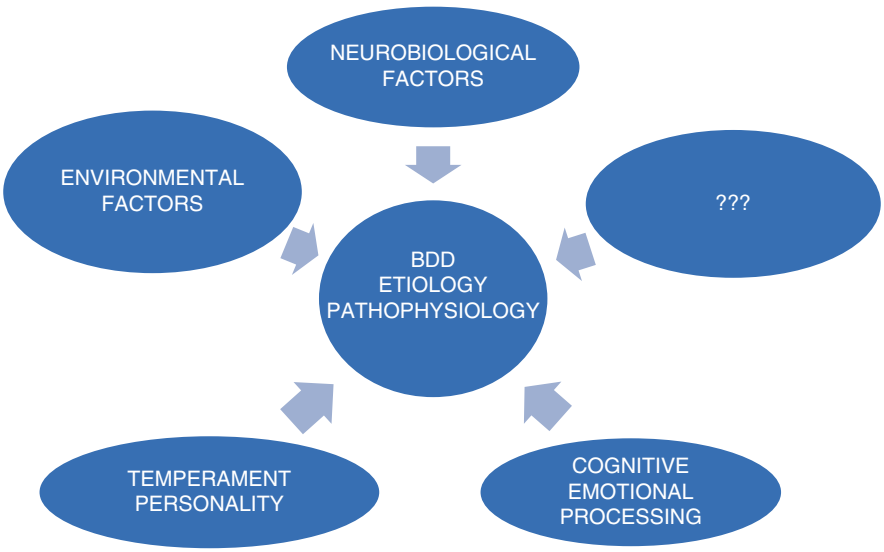


Fig. 14.1 Factors influencing the etio-pathophysiology of BDD

visuospatial information processing that contribute to the disorder's pathological deviation (McCurdy-McKinnon and Feusner 2017).

There is also evidence for a genetic component in the pathogenesis of BDD. A study analyzing the genetic influence among patients with OCD and BDD ascertained that the GABA (A)-gamma-2 1(A) allele was associated with BDD (Phillips 2017; Monzani et al. 2012).

Recent research suggested that environmental factors also play an important role among patients with BDD and may contribute to both, the development and maintenance of the disorder. Earlier experiences like abuse, bullying, maltreatment, together with current concerns about perfect physical appearance and the presence of certain personality traits, may all be important (Neziroglu and Barile 2017). We have personal clinical experience with a patient who developed BDD and suicidal ideation after she witnessed sexual abuse (Dimitrov et al. 2016).

Studies analyzing Cognitive and Emotional Processing in Body Dysmorphic Disorder patients have ascertained abnormalities in regards to information processing, emotion recognition, emotion deficits and selective processing of appearance-related information, as well as dysfunction of beliefs about one's own appearance (Buhlmann and Hartmann 2017).

Neuroimaging research has developed a working neurobiological model of BDD pathophysiology, Neuroimaging studies confirmed changes among patients with BDD. Functional magnetic resonance imaging studies demonstrated differences between patients with BDD and control subjects in several regions within the lateral-temporal-parietal cortices upon exposure to low spatial frequency images (Hong et al. 2018).

Key Clinical Features

The fundamental issue with BDD is that the patient is obsessively and distressingly preoccupied with a real (often objectively trivial) or an imagined defect in his/her appearance. The main body areas of patient concern are the face and facial features, skin, but also breasts, genitals and buttocks. Patients can present signs of this disorder at any age, but in most patients the symptoms begin in adolescence and even childhood. Most patients with BDD spend considerable amounts of time in self-reflective, time-consuming and unproductive rumination. Ritualistic behaviours such as mirror checking are common, as are camouflage, covering 'defects' and ideas of reference (some patients believe that others have noticed their 'defect' and are acting on that knowledge). Affected individuals often need constant reassurance from others, but still continue repeatedly to seek dermatologic or cosmetic referral for correction of the 'defect' (Phillips et al. 2006).

Co-morbidities such as social avoidance, depression, anxiety, poor quality of life and suicidal ideation are common with a lifetime prevalence of 24–28% for suicide attempts (Helwick 2011; Veale et al. 1996a; Phillips et al. 2005; Phillips and Diaz 1997). In an observational study of 200 people with BDD, followed up for almost 5

years, the rate of completed suicide was 22–36 times higher than the general population (Helwick 2011).

Violent behavior toward practitioners can also become a possibility. For example, 2% of BDD patients threaten their practitioners and surgeons physically and at least two cosmetic surgeons have been murdered by patients with BDD (Crerand et al. 2006).

According to one survey, 12% of plastic surgeons said that they had been threatened physically by a dissatisfied BDD patient (Sarwer 2002).

The key clinical features are briefly presented in Box 14.2.

Box 14.2 Key Clinical Features

Preoccupied with an imagined or real objectively trivial defect in his/her appearance
Considerable amounts of time in self-reflective, time-consuming and unproductive rumination
Ritualistic behaviours
Ideas of reference
Need constant reassurance
Seek dermatologic/cosmetic/aesthetic referral for correction of the 'defect'
Social avoidance, depression, anxiety
Poor quality of life
Suicidality
Violent behavior toward practitioners

It is of paramount importance, especially for those practicing in various surgical and non-surgical aesthetic services, to recognize patients with BDD for the reasons presented in Box 14.3.

Box 14.3 Reasons for Recognizing Patients with BDD

1. The prime pathology is psychological rather than physical (Helwick 2011).
2. Psychosocial co-morbidities and suicidal ideation are common (Phillips et al. 2005).
3. Patients with BDD are rarely satisfied with the results of their aesthetic procedures (Veale et al. 1996a; Crerand et al. 2006).
4. Patients quite often become litigious after 'failure' to resolve their 'defect' (Francis 2012).
5. Special attention should be paid to the problem with informed consent in BDD patients undergoing plastic/aesthetic surgery/dermatological procedures. The question that practitioners should address is: **do the patients with BDD have full capacity to give a truly informed consent for cosmetic procedures? (Millard and Millard 2010).**

Techniques for Addressing the Diagnosis of BDD with Patients

Once the diagnosis of BDD has been established, sympathetically discussing this with the patient is crucial, however, it is important to still acknowledge that there is a visible difference in their appearance (if there really is one). Dismissing the concern, trying to reassure the patient that they look fine, or telling them that they should not worry is usually ineffective. Do not argue about the diagnosis; listen carefully and with sympathy to the patient's story but allow enough time for discussion. One technique is to ask the patient to allocate a severity score for their 'defect', (this is usually 10 out of 10 for most patients), and then compare that with your own assessment of the severity of the 'defect' (which can be considerably less than the patient's numeric severity assessment). A discussion about the 'gap' between the patient's and the practitioner's assessment can then be a way to open the discussion about the diagnosis of BDD.

Assessment and Use of Screening Instruments

Recognition of the condition might be achieved with proper screening by well-established and validated instruments in the form of questionnaires.

There are a number of tools available from various organizations that are presented in Table 14.2.

In busy clinical practices, the following questionnaire can be a quick and helpful tool to help you gauge whether a patient may be suffering from BDD: Ahmed (2019). See Table 14.3.

Body Dysmorphic Disorder should be suspected if the patient answers Yes to Question 1; if the answer to Question 2 is (b) or (c); answers Yes to Question 3 and Yes to any part of Question 4.

The following is a more detailed screening questionnaire for BDD patients with skin concerns (Baldock and Veale 2014). See Table 14.4.

Table 14.2 Body Dysmorphic Disorder screening instruments

Body Dysmorphic Disorder screening instrument	References
The Cosmetic Procedure Screening Questionnaire (COPS)	Veale et al. (2011)
The Body Dysmorphic Disorder Questionnaire (BDDQ)	Body Dysmorphic Disorder Foundation (n.d.)
The Yale-Brown Obsessive-Compulsive Scale Modified for Body Dysmorphic Disorder (BDD-YBOCS)	Phillips et al. (1997)
Body Dysmorphic Disorder, NICE Guidance	National Collaborating Centre for Mental Health (2006)
Body Dysmorphic Disorder, Five Questions Psychiatric Evaluation for Cosmetic Procedure by Veale	Veale (2001)

Table 14.3 Screening questions for BDD

# Questions	Answer
1. Are you worried about how you look? (Yes/No); if you are, do you think about your appearance problems a lot and wish you could think about them less?	Yes/No
2. How much time per day, on average, do you spend thinking about how you look?	(a) Less than 1 h a day (b) 1–3 h a day (c) More than 3 h a day
3. Is your main concern with how you look that you are not thin enough or that you might become too fat?	Yes/No
4. How has this problem with how you look affected your life?	
(a) Has it often upset you a lot?	Yes/No
(b) Has it often gotten in the way of doing things with friends, your family or dating?	Yes/No
(c) Has it caused you any problems with school or work?	Yes/No
(d) Are there things you avoid because of how you look?	Yes/No

Table 14.4 Screening questions for BDD with skin concerns

Do you currently think a lot about your skin?
On an average day, how many hours do you spend thinking about your skin? Please add up all the time that your feature is on your mind and make your best estimate.
Do you feel your skin is ugly or very unattractive?
How noticeable do you think your skin is?
Does your skin currently cause you a lot of distress?
How many times a day do you usually check your skin, either in a mirror or by feeling it with your fingers?
How often do you feel anxious about your skin in social situations? Does it lead to you avoiding social situations?
Has your skin had an effect on dating or on existing relationship?
Has your skin interfered with your ability to work or study, or your role as a homemaker?

Differential Diagnosis

The correct diagnosis is crucial for the outcome of the treatment. If the disorder is misdiagnosed, the patient will receive inadequate care resulting in no improvement, progress of the condition and disappointment from the health provider and the health system in general.

Conditions, commonly mistakenly diagnosed as BDD are presented in Box 14.4 (Phillips n.d.).

Box 14.4 Common Misdiagnoses of BDD
BDD is commonly misdiagnosed as one of the following disorders:

- *Obsessive-Compulsive Disorder*
- *Social anxiety disorder (social phobia)*
- *Major depressive disorder*
- *Trichotillomania (hair-pulling disorder)*
- *Excoriation (skin-picking disorder)*
- *Agoraphobia*
- *Generalized anxiety disorder*
- *Schizophrenia and schizoaffective disorder*
- *Olfactory reference syndrome*
- *Eating disorder*
- *Dysmorphic concern*

Referral

Referral to a mental healthcare specialist or a psycho-dermatology clinic may be necessary for the management of BDD. The role of a dermatologist, surgeon or practitioner is to prepare the patient for potential psychiatric help. Without necessary preparation, the patient will usually refuse to seek psychiatric treatment and may continue their journey with other doctors. Discussing the distress caused by their concerns may help patients to understand the need for mental health referral. The patient should be informed that this is a recognized problem and there is a successful treatment, however, some may not be ready during the first consultation to accept that idea. Do not force the patients; allow them enough time; keep a good professional relationship and ask if they would like to come again. Referral to local or regional psycho-dermatology clinics may be easily accepted by the patient as they may feel more comfortable to be seen in a dermatology clinic by a dermatologist and psychiatrist. Some patients may not want other people to know that they need psychiatric help and may feel ashamed to be seen in a psychiatric department. One helpful approach, in the absence of psycho-dermatology clinic, might be offering the patient a telephone consultation with a psychiatrist during the dermatology consultation (Dimitrov and Elsabbahy 2013).

Practice Point
The patient should be informed that this is a recognized problem and there is successful treatment, however some may not be ready during the first consultation to accept that idea.

Treatment

The treatments of choice in BDD are cognitive behavioural therapy (CBT) and serotonin specific reuptake inhibitor (SSRI) medication.

Cognitive behavioural therapy (CBT) is an evidence-based psychotherapeutic method for BDD. Self-focused attention can be reduced, as can rumination, the need for reassurance, social avoidance and other symptoms. It facilitates patients' true understanding of their problem and aids the development of helpful coping strategies (Singh and Veale 2019).

The National Institute for Health and Clinical Excellence (UK) guidelines recommend CBT for patients with BDD, suggesting a protocol with16–24 sessions (National Collaborating Centre for Mental Health 2006).

Four small randomized controlled trials of CBT have demonstrated its efficacy (Rabiei et al. 2012; Rosen et al. 1995; Veale et al. 1996b; Wilhelm et al. 2014).

A study comparing CBT for BDD with anxiety management found that CBT was significantly superior, not only in reducing symptom severities, but also improving both quality of life and level of insight (Veale et al. 2014).

An internet-based CBT programme (BDD-NET) was found to be superior to internet supportive therapy (Enander et al. 2016).

The biggest BDD therapy study, and also the first to compare Cognitive Behavioural Therapy versus Supportive Psychotherapy for adults with Body Dysmorphic Disorder, analyzed the efficacy and posttreatment effects of both therapeutic methods. More consistent improvements in symptom severity and quality of life were found in those treated with CBT (Wilhelm et al. 2019).

Medical research has proven the benefit of serotonin reuptake inhibitors as well. Three randomized controlled trials have proven the benefit of SSRIs for patients with BDD. Fluoxetine was found to be significantly more effective than a placebo in improving the symptoms of BDD sufferers (Phillips et al. 2002).

Clomipramine was found to be more efficacious than the non-SSRI antidepressant desipramine for BDD symptoms, depressive symptoms and functional disability (Hollander et al. 1999).

Another study analyzed the effect of switching to a placebo or continued on escitalopram for a further 6 months, with BDD patients who had already responded to escitalopram (Phillips et al. 2016). The researchers ascertained that the time to relapse was longer and the rates of relapse were less for those who continued on escitalopram (18% versus 40%).

An open-label trial of fluvoxamine, citalopram and escitalopram demonstrated that these medications improved BDD as well as the associated symptoms in 63%–83% of patients (Perugi et al. 1996; Phillips et al. 1998; Phillips and Najjar 2003; Phillips 2006).

There is no established guideline in regards to the dosage of SSRI in BDD patients. The clinical experts recommended higher doses in comparison to those in depression. Some patients may require a dose above the maximum licenced one (Phillips 2004).

Both cognitive behavioural therapy (CBT) and serotonin reuptake inhibitor (SSRI) medications have been proven in their efficacy for the treatment of BDD. Whether one of them is better than the other is not known, since no randomized controlled studies have directly compared them (Singh and Veale 2019).

Despite its prevalence and recent development in medical science, BDD still remains under-recognized and under-diagnosed. Proper education of health care providers in all specialties and levels of the various medical services might help the sufferers to be identified and referred accordingly. Early recognition is of paramount importance to prevent further progress of the disease and to improve the quality of life of the patients and their families.

Patients with severe problems should have continuing access to multidisciplinary teams with specialist expertise in BDD.

References

Ahmed I. Body Dysmorphic Disorder. Medscape. (Updated 2019). http://emedicine.medscape.com/article/291182-overview

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.

Baldock E, Veale D. Body dysmorphic disorder. In: Bewley A, Taylor R, Reichenberg J, Magid M. Practical psychodermatology. London: Wiley; 2014. pp. 127–134.

Body Dysmorphic Disorder Foundation. Questionnaires. http://bddfoundation.org/helping-you/questionnaires/

Buhlmann U, Hartmann AS. Cognitive and emotional processing in body dysmorphic disorder. In: Phillips KA, editor. Body dysmorphic disorder. Advances in research and clinical practice. Oxford: Oxford University Press; 2017. https://doi.org/10.1093/med/9780190254131.003.0022.

Crerand CE, Franklin ME, Sarwer DB. Body dysmorphic disorder and cosmetic surgery. Plastic Reconstr Surg. 2006;118:167e–80.

Dimitrov D, Elsabbahy M. The role of telephone psychiatric/psychology consultation in Psychodermatological Practice 15th Congress of the European Society of Dermatology and Psychiatry, Roskilde, June 2013.

Dimitrov D, Tanev T, Bewley A. Body dysmorphic disorder and suicidal ideation in a 15-year-old girl who witnessed sexual abuse.74th AAD Annual Meeting, Washington, DC, March 2016. J Am Acad Dermal. 2016;74:AB209.

Dyl J, Kittler J, Phillips KA, Hunt JI. Body dysmorphic disorder and other clinically significant body image concerns in adolescent psychiatric inpatients: prevalence and clinical characteristics. Child Psychiat Hum Dev. 2006;36:369–82.

Enander J, Andersson E, Mataix-Cols D, Lichtenstein L, Alström K, Andersson G, et al. Therapist guided internet based cognitive behavioural therapy for body dysmorphic disorder: single blind randomized controlled trial. BMJ. 2016;352:i241.

Francis TE. Informed consent in body dysmorphic disorder. Medscape Plastic Surgery. 2012. http://www.medscape.com/viewarticle/758800_1

Haas CF, Champion A, Secor D. Motivational factors for seeking cosmetic surgery: a synthesis of literature. Plast Surg Nurs. 2008;28:177–82.

Harth W, Gieler U, Kusnir D, Tausk A. Body dysmorphic disorder (Dysmorphophobia). In: Clinical management in psychodermatology. Berlin: Springer; 2009. p. 45–6.

Helwick C. Body dysmorphic disorder can be lethal. Medscape Medical News > Psychiatry. 2011. http://www.medscape.com/viewarticle/740015

Hollander E, Allen A, Kwon J, Aronowitz B, Schmeidler J, Wong C, et al. Clomipramine vs desipramine crossover trial in body dysmorphic disorder: selective efficacy of a serotonin reuptake inhibitor in imagined ugliness. Arch Gen Psychiatry. 1999;56:1033–9.

Hong K, Nezgovorova V, Hollander E. New perspectives in the treatment of body dysmorphic disorder. F1000 Res. 2018;7:361. https://doi.org/10.12688/f1000research.13700.1.
McCurdy-McKinnon D, Feusner JD. Neurobiology of body dysmorphic disorder: heritability/genetics, brain circuitry, and visual processing. In: Phillips KA, editor. Body dysmorphic disorder. Advances in research and clinical practice. Oxford: Oxford University Press; 2017. https://doi.org/10.1093/med/9780190254131.003.0020.
Millard LG, Millard J. Psychocutaneous disorder. In: Burns T, Breathnach S, Neil C, Griffiths C, editors. Rook's textbook of dermatology. 8th ed: Wiley-Blackwell; 2010. p. 64.17–21.
Monzani B, Rijsdijk F, Iervolino AC, et al. Evidence for a genetic overlap between body dysmorphic concerns and obsessive-compulsive symptoms in an adult female community twin sample. Am J Med Genet B Neuropsychiatr Genet. 2012;159B(4):376–82.
National Collaborating Centre for Mental Health: Obsessive Compulsive Disorder: Core Interventions in the Treatment of Obsessive Compulsive Disorder and Body Dysmorphic Disorder. National Clinical Practice Guideline Number 31. London, British Psychiatric Society and Royal College of Psychiatrists; 2006. https://www.nice.org.uk/guidance/cg31
Neziroglu F, Barile N. Environmental factors in body dysmorphic disorder. In: Phillips KA, editor. Body dysmorphic disorder. Advances in research and clinical practice. Oxford: Oxford University Press; 2017. https://doi.org/10.1093/med/9780190254131.003.0021.
Perugi G, Giannotti D, Di Vaio S, Frare F, Saettoni M, Cassano GB, et al. Fluvoxamine in the treatment of body dysmorphic disorder (dysmorphophobia). Int Clin Psychopharmacol. 1996;11:247–54.
Philips KA. The broken mirror: understanding and treating BDD. New York: Oxford University Press; 1996.
Phillips KA. Body dysmorphic disorder: recognizing and treating imagined ugliness. World Psychiatry. 2004;3:12–7.
Phillips KA. An open-label study of escitalopram in body dysmorphic disorder. Int Clin Psychopharmacol. 2006;21(3):177–9.
Phillips KA. Body dysmorphic disorder: Advances in research and clinical practice. Oxford: Oxford University Press; 2017.
Phillips KA. Diagnosing body dysmorphic disorder. International OCD Foundation. https://bdd.iocdf.org/professionals/diagnosis/
Phillips KA, Diaz SF. Gender differences in body dysmorphic disorder. J Nerv Ment Dis. 1997;185(9):570–7.
Phillips KA, Najjar F. An open-label study of citalopram in body dysmorphic disorder. J Clin Psychiatry. 2003;64:715–20.
Phillips KA, Hollander E, Rasmussen SA, et al. A severity rating scale for body dysmorphic disorder: development, reliability, and validity of a modified version of the Yale-Brown Obsessive Compulsive Scale. Psychopharmacol Bull. 1997;33:17–22.
Phillips KA, Dwight MM, McElroy SL. Efficacy and safety of fluvoxamine in body dysmorphic disorder. J Clin Psychiatry. 1998;59:165–71.
Phillips KA, Dufresne RG Jr, Wilkel CS, Vittorio CC. Rate of body dysmorphic disorder in dermatology patients. J Am Acad Dermatol. 2000;42:436–41.
Phillips KA, Albertini RS, Rasmussen SA. A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. Arch Gen Psychiatry. 2002;59(4):381–8.
Phillips KA, Coles ME, Menard W, Yen S, Fay C, Weisberg RB. Suicidal ideation and suicide attempts in body dysmorphic disorder. J Clin Psychiatry. 2005;66(6):717–25.
Phillips KA, Pagano ME, Menard W, et al. A 12-month follow-up study of the course of body dysmorphic disorder. Am J Psychiatry. 2006;163:907–12. https://doi.org/10.1176/ajp.2006.163.5.907.
Phillips KA, Keshaviah A, Dougherty DD, Stout RL, Menard W, Wilhelm S, et al. Pharmacotherapy relapse prevention in body dysmorphic disorder: a double-blind, placebo-controlled trial. Am J Psychiatry. 2016;173:887–95.
Rabiei M, Mulkens S, Kalantari M, Molavi H, Bahrami F. Metacognitive therapy for body dysmorphic disorder patients in Iran: acceptability and proof of concept. J Behav Ther Exp Psychiatry. 2012;43:724–9.

Ribeiro RVE. Prevalence of body dysmorphic disorder in plastic surgery and dermatology patients: a systematic review with meta-analysis. Aesth Plast Surg. 2017;41:964–70. https://doi.org/10.1007/s00266-017-0869-0.

Rosen JC, Reiter J, Orosan P. Cognitive-behavioral body image therapy for body dysmorphic disorder. J Consult Clin Psychol. 1995;63:263–9.

Sarwer DB. Awareness and identification of body dysmorphic disorder by aesthetic surgeons: results of a survey of American Society for Aesthetic Plastic Surgery members. Aesthet Surg J. 2002;22:531.

Singh AR, Veale D. Understanding and treating body dysmorphic disorder. Indian J Psychiatry. 2019;61(Suppl 1):S131–5. https://doi.org/10.4103/psychiatry.IndianJPsychiatry_528_18.

Veale D. Body dysmorphic disorder, five questions psychiatric evaluation for cosmetic procedure. Cambridge University Press, Advances in Psychiatric Treatment. 2001;7:125–132.

Veale D, Boocock A, Gournay K, Dryden W. Body dysmorphic disorder: a survey of fifty cases. Br J Psychiatry. 1996a;169:196–201.

Veale D, Gournay K, Dryden W, Boocock A, Shah F, Willson R, et al. Body dysmorphic disorder: a cognitive behavioural model and pilot randomised controlled trial. Behav Res Ther. 1996b;34:717–29.

Veale D, Ellison N, Werner TG, Dodhia R, Serafty M, Clarke A. Development of a Cosmetic Procedure Screening questionnaire (COPS) for body dysmorphic disorder. J Plast Reconstr Aesthet Surg. 2011;65:530–2.

Veale D, Anson M, Miles S, Pieta M, Costa A, Ellison N, et al. Efficacy of cognitive behaviour therapy versus anxiety management for body dysmorphic disorder: a randomized controlled trial. Psychother Psychosom. 2014;83:341–53.

Veale D, Gledhill LJ, Christodoulou P, Hodsoll J. Body dysmorphic disorder in different settings: a systematic review and estimated weighted prevalence. Body Image. 2016;18:168–86.

Wilhelm S, Phillips KA, Didie E, Buhlmann U, Greenberg JL, Fama JM, et al. Modular cognitive-behavioral therapy for body dysmorphic disorder: a randomized controlled trial. Behav Ther. 2014;45:314–27.

Wilhelm S, Phillips KA, Greenberg JL, et al. Efficacy and posttreatment effects of therapist-delivered cognitive behavioral therapy vs supportive psychotherapy for adults with body dysmorphic disorder: a randomized clinical trial. JAMA Psychiatry. 2019;76(4):363–73. https://doi.org/10.1001/jamapsychiatry.2018.4156.

Nodular Prurigo

15

Anthony Bewley and Richard Barlow

Definition

If one persistently scratches or rubs at one's skin, one will develop thickening (lichenification) and erosion, fissuring and excoriation of the skin. If one scratches or rubs at a specific or a group of specific areas, one will develop nodular prurigo. Nodular prurigo (NP) then is an itchy chronic skin disease characterised by localised lichenification and nodules anywhere (either localised or generalised) on the skin.

Localised forms of nodular prurigo may include
- Picker's nodules
- Lichen simplex chronicus
- Neurodermatitis circumscripta

A. Bewley (✉)
Barts Health NHS Trust, London, UK

Queen Mary University of London (QMUL), London, UK
e-mail: anthony.bewley@nhs.net

R. Barlow
University Hospital Coventry & Warwickshire, Coventry, West Midlands, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_15

Generalised forms of nodular prurigo
- Excoriated nodular prurigo
- Nodular prurigo
- Neurodermatitis (but this may not be universally accurate)

Demographics

Prurigo nodularis was first reported in 1909 by Hyde (1909).

It affects:

- All ages, but more commonly individuals aged 50–60
- Both sexes, but females more than males
- All ethnicities but may look different depending on the Hutchinson skin type
- Many people approximately 1:1000

It is commonly an extremely chronic condition and most patients have had their lesions for a mean duration of 6.4–8.7 years (Tanaka et al.1995; Dazzi et al.2011).

Clinical Features (Figs. 15.1 and 15.2)

Nodular prurigo is either localised (see above) or generalised.

Clinical features include

- Hyperpigmented nodules.
- Hyperpigmented papules.

Fig. 15.1 Multiple hyperkeratotic prurigo nodules with excoriated and slightly crusted central areas. Note how the older lesions have resolved as hyperpigmented and hypopigmented macules

Fig. 15.2 Extensive, symmetrically distributed prurigo nodules over the extensor aspects of the lower limbs, the most commonly affected site in patients

- Patches of lichenification with the above.
- Excoriation of the above.
- There may be a few or commonly very many nodules.
- The lesions are often symmetrically distributed on the limbs but can be on any part of the body.
- Extensor surfaces are also more commonly affected.
- Lesions are usually within the reach of the dominant hand.
- The 'butterfly sign' of mid back sparing is where the individual cannot reach that area of skin.
- Post-inflammatory hypopigmentation and scarring is common in darker skins.
- Crusting and weeping may indicate secondary infection (usually *Staphylococcus aureus*).

Aetiology (Fig. 15.3 and Table 15.1)

Nodular prurigo can be divided up into

1. Atopics with nodular prurigo
2. Non-atopics with nodular prurigo

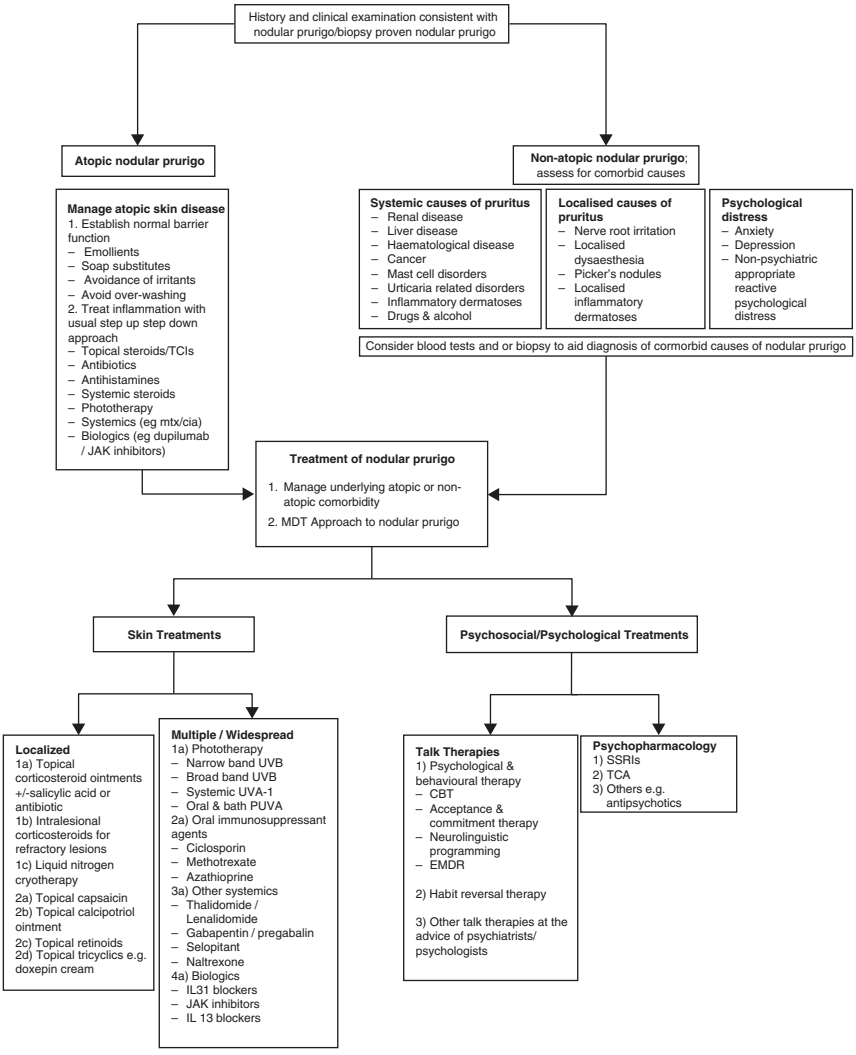


Fig. 15.3 Diagnosis and management of Nodular Prurigo

Table 15.1 Dermatological, systemic and psychological conditions reported in association with nodular purigo

Dermatological	Psychological
Atopic dermatitis	Anxiety
Stasis dermatitis	Depression
Allergic contact dermatitis	Delusional infestation
Cutaneous lymphoma	
Dermatitis herpetiformis	
Lichen planus	
Grover's disease	
Insect bite reaction	
Recurrent folliculitis	
Systemic	
Endocrine	Gastrointestinal
• Hyper/hypothyroidism	• Gluten sensitive enteropathy
• Diabetes mellitus	• Gastrointestinal malignancy
Haematological	• Obstructive biliary disease
• Iron deficiency anaemia	• Lactose intolerance
• Polycythaemiarubravera	• Sorbitol intolerance
• Lymphoma	• Alpha-1-anti-trypsin deficiency
• Leukaemia	Infections
Renal	• Human immunodeficiency virus (HIV)
• Chronic renal failure	• Hepatitis B
Neurological	• Hepatitis C
• Brachioradial pruritus	• Mycobacterium infection (*M. tuberculosis*, *M. aviumintracellulare*)
• Chronic pain syndrome	• *Helicobacter pylori* infection

But there is an overlap, and patients may have atopic dermatitis AND one of the non-atopic co-morbid causes of nodular prurigo, Fig. 15.3. Many conditions have been identified in association with nodular prurigo, Table 15.1

Non-atopic causes of nodular prurigo include:

- Generalised causes of pruritus
  - Renal disease
  - Liver disease
  - Haematological disease
  - Cancer
  - Psychological issues
  - Mast cell disorders
  - Urticaria related disorders
  - Non-atopic skin disease (other inflammatory dermatoses)
  - Drugs (pharma and recreational) and alcohol

- Localised causes of pruritus
  - Nerve root irritation (e.g. brachioradial pruritus)
  - Localised dysaesthesia (e.g. lichen simplex chronicus)
  - Picker's nodules
  - Localised inflammatory dermatoses
- Psychological distress
  - Anxiety
  - Depression
  - Anxiety and depression
  - Non-psychiatric appropriate reactive psychological distress (e.g. bereavement)
  - Others
- A combination of the above

Practice Point
Often patients with nodular prurigo have psycho-social co-morbidities which may be the primary cause of, or secondary to, the nodular prurigo. So identifying the psycho-social distress is crucial to the management of the disease.

Pathogenesis

The aetiology of nodular prurigo is thought to be secondary to repeated rubbing, scratching or picking of a localised area of the skin. This leads to skin changes including invasion of new cutaneous nerves containing a range of pro-inflammatory neuropeptides such as the calcitonin gene-related peptide, substance P and others. There is evidence that the neo-neuronal growth is associated with brain:skin interactions which may have a temporary calming effect. But the neo-neuronal growth is also associated with inflammatory changes in the skin which lead to the clinical features of nodular prurigo.

Differential Diagnosis

- Atopic skin disease
- Other inflammatory skin diseases
- Aetiologies above
- Immunobullous disease (e.g. nodular prurigo pemphigoides)
- Genuine infestations (e.g. scabies)
- Delusional infestation
- Cutaneous neoplasia which itches

Clinical Assessment

1. Careful dermatological history and examination
   (a) Check for history of atopic disease
   (b) Check for other features of atopy (e.g. Dennie-Morgan folds, white dermographism, etc.)
   (c) Check for pharmacological and recreational drugs
2. Careful psychological/psychiatric examination
3. Consider investigation based on history, examination and knowledge of the differential diagnoses

Investigations are best determined by the clinical picture. They may include:

- Full blood count
- Renal function test
- Liver function test
- Thyroid function test
- Recreational drug urinalysis
- Skin biopsy (rarely and only to exclude other diseases such as immunobullous or neoplastic disease)
- HIV serology
- Hepatitis serology
- Antinuclear antibodies
- Others directed by the clinical picture

Management of Nodular Prurigo (Fig. 15.3)

Practice Point

Often patients with nodular prurigo have psycho-social co-morbidities which may be the primary cause of, or secondary to, the nodular prurigo. So managing the skin disease and the psychological disease concomitantly is very important.

Practice Point
Treat any identifiable cause (e.g. any cause of pruritus) at the same time as treating the nodular prurigo.

A multi-disciplinary team (MDT) approach is more likely to achieve successful management of a patient with nodular prurigo than in a general dermatological or primary care clinic. MDT members may include dermatologists, psychiatrists, psychologists, paediatricians, primary care colleagues and nurses, but local structuring of an MDT may vary and be very successful depending on local resources.

Establishment of a Working Relationship with the Patient
Understanding the patient experience
Empowering the patient
Signposting to educational resources for patients:
www.bad.org.uk
www.skinsupport.org.uk
www.skinhealthinfo.org.uk
www.nodular-prurigo.org.uk
www.changingfaces.org.uk
www.eczema.org.uk
www.atopicskindisease.com

Treatment of the Skin
1. Establishment of the Normal Barrier Function of the Skin:
   - Emollients applied to the skin
   - Soap substitutes
   - Avoidance of irritants such as fragrances
   - Care about over-washing
2. Topical Treatment of Inflammation:
   Treatment of atopic dermatitis and nodular prurigo may include
   First line
   - Topical corticosteroid ointments (ointments are generally preferred by dermatologists to creams as they are unlikely to contain preservatives and irritants). Use under medical/nursing supervision, enough to cover the affected areas.

- Combination corticosteroid creams with salicylic acid (if scaly) or antibiotics (if infected)
- Intralesional corticosteroids under medical/nursing supervision for localised/refractory lesions

Second line

- Topical capsaicin cream may help with pruritus
- Topical calcipotriol ointment
- Topical retinoids
- Topical tricyclics such as doxepin cream

3. Systemic Treatments to Consider when Topicals Have Not Worked Sufficiently (Can Be Used in Combination with Topicals)

Phototherapy—Whole body or localised phototherapy with:

- Usually narrow band ultraviolet B (UVB)
- More rarely broad band UVB
- Systemic UVA-1
- Oral and bath PUVA

Oral ImmunosuppressantAgents (Use in Normal Anti-Inflammatory Doses)

- Ciclosporin
- Methotrexate
- Azathioprine
- Newer/Third Line Treatment Options eg. nemolizumab, dupilumab and JAK inhibitors are being prepared for use in patients with NP

Second and Third Line Systemic Agents

- Thalidomide/Lenalidomide has been reported. They are challenging drugs to use and are often expensive. They are reported to be helpful for a few recalcitrant patients. Gabapentin and pregabalin may help with the itch and the psycho-social co-morbidities, but are now counted as restricted use drugs in many countries
- Aprepitant has been used but may be limited by toxicity
- Serlopitant has also been used with some success
- Naltrexone (an opioid antagonist)
- Antipsychotics/antidepressants—In patients with associated psychiatric co-morbidities, psychiatric medications may be indicated. Olanzapine, an atypical antipsychotic, was used to treat five patients with concurrent depression and neuroticism with marked improvement of pruritus and lesional count [32].
- Others—The opioid antagonist naltrexone was reported to be effective in a small trial of 17 nodular prurigo patients, with nine patients having a 50% or greater reduction in pruritus intensity and lesion count. The onset of pruritus reduction also occurred rapidly within 2–8 days [33].

Treatment of the Psychological and Psycho-Social Co-morbidities

Talk Therapies

1. Psychological and behavioural therapy in nodular prurigo may be helpful in allowing patients to understand the psychological distress which is causing the nodular prurigo.
   Examples are:
   - Cognitive behavioural therapy (CBT)
   - Acceptance and commitment therapy (ACT)
   - Neurolinguistic programming (NLP)
   - Eye movement desensitization and reprocessing (EMDR)
2. Habit reversal therapy (HRT), which aims to modify patient scratching habits by introducing other competing responses to the itch, has been shown to be very helpful in the reversal of the itch/scratch cycle. HRT can be delivered by trained nurses.
3. Other talk therapies may be helpful at the advice of psychiatrists and psychologists.

Psychopharmacology Treatments

1. Selective serotonin reuptake inhibitors (SSRIs; often is higher doses, e.g. citalopram 40 mg or fluoxetine 60 mg) may reduce the compulsion to scratch. SSRIs are also antidepressant and anxiolytics drugs which may have independent anti-itch properties.
2. Tricyclic antidepressants (TCAs e.g. amitriptyline or doxepin). Often used in lower doses (e.g. amitriptyline 10 mg nocte) can be helpful as an antipruritic and can be sedative. Some reports indicate that sedation may not be always desirable in the longer term.
3. Other agents (e.g. antipsychotics) may be considered if a practitioner has the necessary skills and experience.

Practice Point
Talk therapies in combination with psychopharmacology are more likely to be successful than either in isolation.

Practice Point

Treat patients under supervision to the clearance of their disease.

It will probably be necessary to continue systemic immunosuppressants and antidepressants for a few months after the cessation of the disease.

Top up HRT may be helpful if symptoms recur.

Outlook

The prognosis is good when patients adhere to medication. Recurrences are common. The commonest cause of recurrence and treatment failure is poor adherence to agreed treatment plans. Treating the skin, the causes of the nodular prurigo and the psycho-social co-morbidities are more likely to lead to treatment success than any of the treatments in isolation.

Bibliography

Abadía Molina F, Burrows NP, Jones RR, et al. Increased peptides in nodular prurigo: a quantitative immunohistochemical analysis. Br J Dermatol. 1992;127:344–51.

Andersen TP, Fogh K. Thalidomide in 42 patients with prurigonodularis Hyde. Dermatology. 2011;223:107–12.

Berth-Jones J, Smith SG, Graham-Brown RA. Nodular prurigo responds to cyclosporin. Br J Dermatol. 1995;132:795–9.

Bruni E, Caccialanza M, Piccinno R. Phototherapy of generalized prurigonodularis. ClinExpDermatol. 2010;35:549–50.

Capoore HS, Rowland Payne CM, Goldin D. Does psychological intervention help chronic skin conditions? Postgrad Med J. 1998;74:662–4.

Dazzi C, Erma D, Piccinno R, Veraldi S, Caccialanza M. Psychological factors involved in prurigonodularis: a pilot study. J Dermatol Treat. 2011;22:211–4.

Gencoglan G, Inanir I, Gunduz K. Therapeutic hotline: treatment of prurigonodularis and lichen simplex chronicus with gabapentin. DermatolTher. 2010;23:194–8.

Grillo M, Long R, Long D. Habit reversal training for the itch-scratch cycle associated with pruritic skin conditions. DermatolNurs. 2007;19:243–8.

Hammes S, Hermann J, Roos S, Ockenfels HM. UVB 308-nm excimer light and bath PUVA: combination therapy is very effective in the treatment of prurigonodularis. J EurAcadDermatolVenereol. 2011;25:799–803.

Hyde JN. Prurigonodularis. In: Hyde JN, Montgomery FH, editors. A practical treatise on diseases of the skin for the use of students and practitioners. 8th ed. Philadelphia: Lea and Febiger; 1909. p. 174–5.

Lear JT, English JS, Smith AG. Nodularprurigo responsive to azathioprine. Br J Dermatol. 1996;134:1151.

Metze D, Reimann S, Beissert S, Luger T. Efficacy and safety of naltrexone, an oral opiate receptor antagonist, in the treatment of pruritus in internal and dermatological diseases. J Am AcadDermatol. 1999;41:533–9.

Saraceno R, Chiricozzi A, Nisticò SP, et al. An occlusive dressing containing betamethasone valerate 0.1% for the treatment of prurigonodularis. J Dermatol Treat. 2010;21:363–6.

Ständer S, Luger T, Metze D. Treatment of prurigonodularis with topical capsaicin. J Am AcadDermatol. 2001;44:471–8.

Ständer S, Siepmann D, Herrgott I, et al. Targeting the neurokinin receptor 1 with aprepitant: a novel antipruritic strategy. PLoS One. 2010;5:e10968.

Stoll DM, Fields JP, King LE Jr. Treatment of prurigonodularis: use of cryosurgery and intralesional steroids plus lidocaine. J DermatolSurgOncol. 1983;9:922–4.

Tanaka M, Aiba S, Matsumura N, Aoyama H, et al. Prurigonodularis consists of two distinct forms: early-onset atopic and late-onset non- atopic. Dermatology. 1995;190:269–76.

Trichotillomania (Hair Pulling Disorder)

16

Dmitry V. Romanov, Anna V. Michenko, Iulia Iu. Romanova, and Andrey N. Lvov

Definition

Trichotillomania (TTM) or hair pulling disorder (HPD) is defined as a recurrent pulling of one's own hair leading to significant hair loss, i.e. self-inflicted alopecia, traction alopecia or mechanical alopecia, accompanied by repeated unsuccessful attempts to decrease or stop the behaviour.

Etiology and Pathogenesis

TTM or HPD is referred to primary psychiatric conditions as there is no other medical state to explain hair loss.

Known etiological and pathogenetic factors include:

- Genetic predisposition: elevated rates and higher recurrences of hair pulling in first-degree relatives of probands with TTM and some candidate genes (HoxB8, SAPAP3, SliTrk5);

D. V. Romanov (✉)
Department of Psychiatry and Psychosomatics, I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia

Department of Boundary Conditions and Psychosomatic Disorders, Mental Health Research Centre, Moscow, Russia

Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

A. V. Michenko · I. Iu. Romanova · A. N. Lvov
Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_16

- MRI brain abnormalities in cortical and subcortical regions: right inferior frontal gyrus, amygdala, putamen, nucleus accumbens and caudate.
- The neuropsychological mechanisms: reducing/preventing anxiety/distress and/or getting pleasure/gratification providing positive reinforcement important for the conditioning of the pulling behaviour.
- The problem of cognitive control of the behaviour (motor inhibition): deficits of divided attention, response inhibition and working memory.

Practice Point
As for most other psychiatric disorders, there are no reliable biological markers to establish or support TTM diagnosis. Hair pulling is still diagnosed clinically.

Classification

Main Classification

In ICD-10 TTM (F63.2) was attributed to F63 *Habit and impulse disorders* along with Pathological gambling.

Recently, in DSM-5 TTM or HPD was reclassified to *Obsessive-Compulsive and Related Disorders* along with Obsessive-Compulsive Disorder per se, Body Dysmorphic Disorder, Hoarding Disorder and Excoriation (Skin-Picking) Disorder.

The similar approach has been accepted in ICD-11. TTM or HPD (6B25.0) is also attributed to *Obsessive-compulsive or related disorders* along with Obsessive-compulsive disorder per se.

Subtypes

Historical controversy in the attribution of TTM to either obsessive-compulsive or impulse-control pathology reflects the possibility of the existence of two subgroups, depending on the primary motivation in the act of pulling. Respectively, a ‘compulsive’ subgroup was designated to include ‘relief pullers’ (pulling primarily

to reduce anxiety), and an 'impulsive' subgroup was established to comprise 'pleasure/gratification pullers' (pulling primarily for 'reward'). However, there is a suggestion that the phenomenological difference between relief and pleasure/gratification pullers is not supported by other clinical features and associated neural dysfunction. As a result, in most cases, TTM fails to fit neatly into either category, in part because it may have distinct qualities of both categories. Depending on the input of any of two, a TTM individual may correspond to the point on the impulsive—compulsive spectrum.

Thus, there is another attempt to distinguish TTM subtypes including focused versus automatic pulling.

'Focused' pulling is performed with one's awareness in response to a negative emotional state (anxiety, dysphoria, anger, etc.), a set of pathological skin sensations (itch-like sensations, skin dysaesthesia, etc.), an intense thought or urge to pull, or in an attempt to establish symmetry or 'hair regularity'. Contrariwise, 'automatic' pulling refers to pulling occurring primarily out of one's awareness, e.g. as a kind of dissociative altered state of consciousness with the detachment of own motor acts. The problem in this dichotomy is the same as with impulsive versus compulsive pulling, i.e. in most cases, TTM fails to fit neatly into either category and a spectrum of awareness exists—more-focused pullers versus more-automatic pullers.

Practice Point
Most focused hair pullers share features of impulsive and compulsive hair pulling and occupy a point on the impulsive–compulsive continuum/spectrum, requiring approach differing as from impulse-control disorders, as from obsessive-compulsive disorders treatments. The same is relevant for focused and automatic pulling. As a result of automatic pulling and dissociation, some patients may strongly deny self-inflicted hair-pulling.

Clinical Presentations

General Symptoms

Most hair pullers do it with their own fingers; however, some also use instruments like tweezers. Patients may combine methods, fingers and tweezers. Episodically, some substitute finger-pulling on cosmetic significant areas (e.g. scalp) with tweezers used to pull on less visible sites (e.g. legs).

Most patients also reported diurnal variation in hair pulling, i.e. worse in the evening for the vast majority. Hair pulling usually occurs in privacy (in bathroom, bed, etc.) with the absence of any witnesses, except close relatives.

Dermatological Symptoms

Localisation Any hair-bearing region of the skin may be involved. Most frequently, the scalp, eyebrows and eyelashes are susceptible to hair pulling. Axillary, facial, pubic and peri-rectal regions are infrequently involved. The rarest localisations are arms, legs, chest and abdomen. Typically, localisation is frontoparietal and progress backward. As a result, 'tonsure trichotillomania' may be observed when a scalp exhibits a cropped appearance over the crown, extending to the frontal margin, but sparing the nape of the neck and lateral margins (see Fig. 16.1).

Hair Loss Pattern Severity of hair loss may vary from total depilation to minimal hair sparseness. Areas of complete hair loss or patches of alopecia are very common. However, there could be also a pattern resembling diffuse hair loss, i.e. areas of thinned hair density. This may lead to a widely distributed pattern such that hair loss may not be clearly visible. Either of the two patterns, patchy or diffuse, could predominate but the mixture of both is also possible (see Fig. 16.2).

Local Findings From a dermatological point of view, TTM corresponds to self-inflicted alopecia, traction alopecia or mechanical alopecia. The hair on the affected areas is shortened but remains of normal texture. Microscopically, the hair shaft remains normal. Along with decreased hair density, there are multiple broken hairs of different lengths. The trichoptilosis, i.e. a splitting of the shaft of the hair, giving it a feathery appearance, may be observed. Many of the short hairs may also have a

Fig. 16.1 Tonsure trichotillomania

Fig. 16.2 Diffuse hair loss pattern in TTM

tapered hypopigmented tip. If some hairs are totally removed, the findings are limited to anagen roots only. Persistent pulling is accompanied by the growth of new short anagen hairs.

Psychiatric Symptoms

Emotional States to Pull Hair In contrast to compulsions in OCD, there are no obsessions in TTM that may trigger motor acts. However, as the patient attempts to resist the urge to pull, there is associated anxiety, boredom, tension, irritability and dysphoria, which may resolve after hair pulling and lead to a sense of relief. But along with the reduction of negative emotional states, there may also be gratification and pleasure, when the hair is pulled out. Thus, many individuals with HPD experience feelings after hair pulling such as relief or gratification that maintains the vicious circle of pulling.

Skin Sensations to Pull Hair 'Itch-like' sensations or tingling in hair pulling areas may also precede and precipitate pulling. Skin sensations may be even more complex and get a kind of 'as if' property. For example, some patients compare selected hairs with 'needles' stuck in their skin like foreign objects or splinters. Consecutively, removal of the bothering hair results in a temporary relief and sensations alleviation or reduction.

Paradoxically, self-hair pulling in TTM is not usually accompanied by pain. However, if asked to compare, patients report that they feel pain if a hair is pulled out from an atypical area or the pulling is performed by another person, e.g. accidentally by their child.

Pulling Awareness There is a spectrum of conscious awareness to pull hair. Some patients report their focus on hair pulling from the very beginning ('focused pullers'), but some note that hair pulling always occurs without their full awareness ('automatic pullers'). When asked directly, some individuals may totally deny their hair pulling. However, most patients report a combination varying between full and incomplete awareness. For instance, there are episodes of hair pulling that start with incomplete awareness and then develop into full awareness.

Hair pulling often occurs while engaged in sedentary or contemplative activities, e.g. watching television, reading, writing or doing paperwork, driving, talking on the telephone, smoking, etc.

Pulling Associated Behaviours and Rituals Hair manipulation actions are typical for TTM. This activity involves 'pre-pulling', and 'after-pulling' behaviours.

- There may be a tactile and/or visual search for a particular kind of hair to pull (e.g. hairs with a specific texture or colour, somehow differing from the others). Respectively, coarse, rough, uneven or thick hairs, and darker, brighter or grey hairs are more likely to be extracted.
- There may be a specific manner of pulling, e.g. hair twisting or applying series of slight tractions, so that the hair comes out with intact root.
- There could be a visual examination or tactile manipulation with the hair after it has been pulled (e.g. rolling the hair between the fingers). Often patients report 'oral behaviours' associated with hair pulling, e.g. rubbing hair around the mouths after pulling it out, licking or eating hair, or biting and swallowing the hair bulb.
- After extraction, other hair-destroying rituals are possible. To get rid of pulled hairs, patients may roll them in a piece of paper and only then throw away or may burn them with a cigarette lighter.

TTM by Proxy Some patients report pulling hair from other persons, e.g. children, a spouse or partner/significant other.

Secondary Psychological Reactions to Hair Pulling There may be an immediate negative reaction to the failure of a proper hair pulling ritual performance (see the paragraph above). 'Improperly plugged out hair' causes a sense of incompleteness and leads to repeated pulling (maintaining a hair pulling circle) till the 'just right' feeling emerges.

Hair pulling flares also cause clinically significant distress, i.e. secondary psychological conditions or negative effects, including shame, embarrassment, sadness, guilt, irritability, and frustration. Such negative effects may also lead to avoidance of professional and educational activities, or other public/social situations (getting haircuts, swimming, dating, etc.) that may lead the disease signs to become evident to others. Thus, patients try to conceal or camouflage hair loss, e.g.

by using makeup, scarves, wigs or (paradoxically) total shaving of affected areas, for example, if located on the face or scalp in men.

In turn, these negative effects may contribute to emotional deregulation precipitating further hair pulling (see above) and maintain a hair pulling cycle.,

Associated Body-Focused Repetitive Behaviours In TTM, there are many other body-focused repetitive behaviours either current or past: nail biting, knuckle cracking, nose picking, thumb sucking, tongue chewing, cheek chewing, lip biting, head banging, body rocking; scab, skin and acne picking.

Other Psychiatric Comorbidities The highest lifetime prevalence values are reported for mood, anxiety, eating and substance use disorders.

Practice Point
Always ask about mood, as comorbid affective disorders (depression) may be associated with suicidal ideation and can be potentially life-threatening due to suicidal attempts.

Why is TTM So Debilitating?
Along with cosmetic consequences, functional impairment and psychological distress with avoidance and camouflage behaviour, TTM may be potentially life-threatening due to trichophagia (pulled hair eating). It may lead to surgical complications—trichobezoars which can result in gastrointestinal obstruction and require surgical intervention. In few cases, it presents with Rapunzel syndrome: gastric trichobezoar with a long tail extending into the small or even large bowel.

Practice Point
Always ask about what the patient does with pulled hairs to assess the risk of surgical complications.

Epidemiology

Prevalence Population lifetime prevalence of TTM, as measured mainly in college students, is about 0.6–1.2%. However, the frequency of hair pulling failing to meet full ICD TTM criteria ICD is higher, e.g. hair pulling resulting in visible hair loss involves 1.5% of males and 3.4% of females. In studies using less restrictive diagnostic criteria the values are therefore expectedly higher. For example, hair manipulation as a subtype of body-focused repetitive behaviours is observed in 13.3% of subjects.

Epidemiology Females suffer from TTM more frequently than males (10:1 to 4:1). In clinical samples, the predominance of females is more prominent (up to 15:1), possibly reflecting different treatment-seeking modalities (e.g. less problematic attitude to hair loss by males). Among children, TTM gender ratio tends to be about 1:1.

Age Distribution Young people are affected more frequently than adults. The mean age of TTM onset is between 9–15 years. However, some authors stress a bimodal distribution with two peaks.

Disease Duration and Course Although hair pulling may begin at any age, most often the onset is at a young age, i.e. late childhood or adolescence (the early teens—peak age of onset is 12–13 years). Many patients with illness onset in their teens become chronic pullers, and TTM persists for decades. In those subjects, the disease course may be chronic or episodic with remissions and relapses. Regarding concealing and dissimulation, the duration of untreated illness in some TTM chronic pullers may extend up to 22 years.

However, there are also episodic and transient cases, with a single hair pulling flare and no evidence of further recurrences. Particularly, it relates to early pulling in infants that resembles pathological habits like thumb sucking and nail biting.

Diagnostic Process

General Considerations

TTM diagnosis requires a holistic approach as dermatological (trichological), psychiatric and psychological signs and symptoms should be taken into consideration together.

Diagnosing Process

Precise dermatological examination of local status may provide several clues to TTM diagnosis as follows:

Non-scarring patchy or diffuse pattern alopecia

Hair in the pathological area partially saved with different lengths of the broken-off hair

The underlying skin is unaffected (no scaling, no inflammation)

Pull-test negative (in traction a hair may not be easily pulled out)

Investigations

In trichoscopy, there is simultaneous, chaotic coexistence of multiple hair shaft abnormalities. Hairs are broken at different lengths. Short hairs with trichoptilosis, i.e. split ends, may be observed. There are also coiled hairs and other hair distal ends changes (see Table 16.1). Exclamation mark hairs are also possible, however, are usually attributed to other types of alopecia (alopecia areata).

In some cases, there is a need for biopsy, especially in totally unaware patients, e.g. children who strongly deny hair pulling if parents are also totally unaware of pulling. This is particularly important, as there may be cases of hair pulling *by proxy*, when caregivers mutilate persons under their care.

The diagnostic process should be performed in a non-confrontational manner.

The first conversation should be focused on the clinical features of TTM, because it is crucial for the establishment of the TTM subtype and for trusting relationships. This may be a time consuming, but it is worth doing, as it plays an important role for diagnosis and further psychotherapeutic intervention (see Functional Analysis below).

Table 16.1 TTM Trichoscopy findings

1. Broken Hair with Different Length;
2. Hair Distal Ends Changes:
• *Trichoptilosis* (split ends of broken hairs).
• *Coiled hairs* (spiral shape hair shafts remnants fixed to the scalp as a result of hair shaft fracture and coiling of the remaining proximal part).
• *Hook hairs* ('partially coiled' distal parts of fractured hairs).
• *Tulip hairs* (short hairs with darker, tulip flower shaped ends).
• *Flame hairs* (semi-transparent, wavy and cone-shaped hair residues, which develop as a result of severe mechanical hair pulling and shredding).
• *V-signs* (V-sign is created when two or more hairs emerge from one follicular ostium, which is pulled simultaneously and break at the same length above scalp surface).
• *Hair powder* (When hair shafts are almost totally damaged by mechanical manipulation, only a sprinkled hair residue is visible. This finding is referred to as 'hair powder'.).
3. Follicular Pathology:
• *Black dots* supporting local trauma (small, round hair follicle openings seen on trichoscopy as spots of the corresponding colour, which are microhaemorrhages that corresponds to a follicular ostium stuffed with blood clot).

TTM is often triggered by a stressful event or when the patient is in persistent distress. It is important to identify acute stress (death of a partner/significant other, parental divorce, unemployment, etc.) or chronic stress (ongoing domestic violence, poor scholarly or job performance, long-lasting overwork, etc.). It is essential to fully consider the general situation of an individual's life circumstances, as there could be important signs of positive or negative reinforcement for hair pulling.

Some seemingly unaware pulling cases (due to dissimulation related to shame and embarrassment) may then emerge as aware and focused when patents confess self-induced mutilation after such a discussion. Therefore, it helps to distinguish more precisely aware/unaware pulling (focused/automatic). It is crucial for the further choice of psychotherapy, as further questions should be 'hair oriented' in unaware 'hair detached' cases ('How do you notice hair loss? What does provoke it'?), or 'patient oriented' in aware 'hair attached' cases ('What does provoke you to pull hair'?).

To maintain trusting relations, some open-ended questions ('What provokes pulling'?) might be combined with closed ones ('Is there itch/tension that provokes pulling'?). In such an approach, a patient feels that a physician is familiar with this condition and it improves rapport.

Practice Point
In patients totally unaware of their pulling and supported by their caregivers, it is essential to look for other sources of objective information (biopsy to document findings, trichoscopy with photographic evidence, third parties, etc.).

Differential Diagnosis (Table 16.2)

Other courses of hair loss should be excluded. Taking into consideration that both focal (patchy) and diffuse hair loss patterns are possible in TTM, the respective types of non-cicatricial alopecia should be ruled out, e.g. alopecia areata, telogen or anagen effluvium. Obviously, there should be no another trichological or dermatological conditions that can explain hair loss, e.g. tinea capitis.

Other neuropsychiatric causes of hair pulling or hair traumatisation should be excluded. There should be no other mental or neurological disorder accompanied with the hair self-injury focused on the scalp or other hair-bearing areas (skin picking disorder, body dysmorphic disorder, delusional disorder, e.g. delusional infestation, substance misuse, or neurological stereotyped movement disorders with hair-plucking in Tourette's syndrome, Parkinson's disease, catatonia, etc.).

Occasionally, TTM can overlap with other medical conditions, causing or contributing to the hair loss (e.g. telogen or anagen effluvium), but in these cases the degree and pattern of hair loss is clearly excessive in relation to its primary nature and progression. This could lead to the so-called *cum materia* situation or mixed/

Table 16.2 TTM differential diagnosis

Hair pathology to be excluded:
Alopecia areata
Tinea capitis
Scarring (cicatricial) alopecia
Androgenetic alopecia
Telogen effluvium
Loose anagen hair
Monilethrix
Dermatological conditions to be excluded:
Secondary syphilis
Lichen planopilaris
General medical conditions to be excluded:
Blood loss,
Malnutrition,
Chronic intoxication
Substance-induced conditions to be excluded
Medications-induced alopecia (telogen effluvium)
Chemotherapy-induced alopecia (anagen effluvium)

combined aetiology states, i.e. TTM amplification of hair loss related to another medical condition.

Treatment

General Considerations Optimal or gold standard treatment is still lacking. Psychiatric referral is recommended, although many psychiatrists are unfamiliar with the condition. Other service types may be preferable, like multidisciplinary psychodermatological clinics or consultation-liaison services. There is evidence for pharmacological treatment, as for psychotherapy. A combination of both may be more effective than either approach independently.

Pharmacotherapy (Table 16.3) There is no clear first-line agent or any medications approved by any regulatory agencies for TTM treatment at the time of writing.

Several RCTs show the effectiveness of the tricyclic antidepressant *clomipramine (up to 250 mg/day)*, the second generation antipsychotic *olanzapine (up to 20 mg/day),* and the glutamate modulator *N-acetylcysteine (up to 2400 mg/day)*. As for *N-acetylcysteine* (NAC), one RCT in adults demonstrated efficacy, whereas it failed to do so in children.

SSRIs were previously considered promising in TTM, but in contrast to OCD they showed negative results in several small RCTs performed with fluoxetine (up to 80 mg/day) and sertraline (50–200 mg/day), which failed to prove efficacy compared to placebo. Likewise, fluvoxamine and citalopram failed to prove efficacy in open-label trials. Recent meta-analyses have shown that among serotonergic antidepressants, only clomipramine appears to be effective in TTM (see Table 16.3)

Table 16.3 Medication which may be used in TTM and potential side effects

Medication name	Starting dose	Target dose	Common side effects
Clomipramine	12.5 mg/d	100–250 mg/d	TCA and SSRI typical: dizziness, drowsiness, dry mouth, constipation, stomach upset, nausea, vomiting, changes in appetite/weight, blurred vision; avoid in adenoma, glaucoma
Olanzapine	2.5 mg at night	10–15 mg at night	Sedation, increased appetite, metabolic syndrome, avoid in diabetics
N-Acetylcysteine (NAC)	1200 mg/d	1200–2400 mg/d	Bloated feeling, flatulence

Negative results were obtained in Double-Blind, Placebo-Controlled trials for Milk Thistle, inositol (6–18 g/day), and naltrexone (50 mg/day).

Several small open-label trials demonstrated positive results for dronabinol, a cannabinoid agonist (dose ranging from 2.5–15 mg/day), the mood stabiliser *lithium carbonate*, the anticonvulsant *topiramate* (50–250 mg/day), and the antipsychotic *aripiprazole* in mean doses of 7.5 mg/day (±3.4 mg/day) and up to 15 mg/day.

Other case reports or case series support the use of chlorpromazine, pimozide, haloperidol (up to 2 mg/day), risperidone (up to 4 mg/day), and quetiapine (up to 100 mg/day) as monotherapy or as augmentation of a serotonin reuptake inhibitor. Although there are also case reports on some anticonvulsants, such as oxcarbazepine (up to 1200 mg/day), and non-serotonergic antidepressants, e.g. bupropion (up to 450 mg/day), antipsychotics appear highly promising agents requiring further research.

Psychotherapy

Habit Reversal Therapy (HRT) is considered one of the most effective psychotherapeutic treatments in TTM with the most empirical support. It has been proven to be effective in several RCTs and two meta-analyses. Among those, there are two RCTs comparing the effect of augmenting HRT with a pharmacological intervention. As a result, it was shown that HRT in combination with antidepressants resulted in better outcomes than HRT with placebo, reflecting a possible advantage of the combined treatment approach.

Other forms of psychotherapy have also been applied in TTM and showed some effect, also as an add-on to HRT to improve long-term gains and reduce the rate of recurrences and relapses. Among those modalities are ‘Acceptance and Commitment Therapy’ (ACT), ‘self-help decoupling’ with ‘self-help progressive muscle relaxation (PMR)’, ‘Dialectical Behaviour Therapy Enhanced Cognitive Behavioural Treatment’ (DBT/CBT), ‘metacognitive methods’ combined with habit reversal (MCT/HRT), and ‘exposure therapy’.

TTM in each patient has a unique pattern. Therefore, any approach should focus on an individual's specific and peculiar TTM pattern. To reach this goal, HRT procedures should be preceded by psychoeducation and functional analysis.

Psychoeducation

TTM diagnosis establishment should be followed by an explanation of its essence according to the biopsychosocial and behavioural approach to a patient. This should be undertaken in an accessible manner, taking into account the patient's age and educational level. The problem of pulling triggers should be discussed together with negative and positive reinforcement mechanisms. It is essential to explain to a patient that pulling may be unconsciously, semiconsciously or consciously used to reduce negative emotions, sensations, urges (negative reinforcement) and/or to achieve pleasure (positive reinforcement). There can be different degrees of awareness, and it can include compulsive/impulsive components. Comparison with other OCDs, habit and addictive behaviours may be rather illustrative.

Functional Analysis

Functional analysis is an assessment approach focused on the factors that trigger and reinforce pulling. It is based on a typical conditioning and cognitive-behavioural S-O-R-C scheme that is an acronym for **S**timulus-**O**rganism-**R**eaction-**C**onsequences.

Stimulus-Organism-Reaction-Consequences Scheme

Stimulus uncovering (external pulling triggers, e.g. environment or activities, etc.)

Organism conditions evaluation (internal pulling triggers, e.g. emotions, thoughts or sensations, etc.).

Reaction examination (pulling pattern peculiarities, e.g. fingers or tweezers used, in bathroom in front of the mirror or in bed on the touch, etc.).

Consequences analysis (assessment of reinforcement occurring immediately after pulling, as positive, as negative, e.g. achieving pleasant sensations/emotions or escaping unpleasant sensations/emotions).

Functional analysis helps to develop an individual plan for further habit reversal treatment that comprise (1) awareness training and self-monitoring, (2) stimulus control and (3) competing response procedures.

Habit Reversal Therapy (HRT) Major Steps

- **Awareness training and self-monitoring** (encouraging awareness of situations and triggers that can precede, initiate and maintain pulling)
- **Stimulus control** (removing or reducing the impact of pulling triggers in the environment)
- **Competing response training** (developing less conspicuous and harmless behaviours replacing pulling)

Awareness Training and Self-Monitoring

Patients keep a daily diary or a log of pulling episodes. Ideally, it should be completed immediately after pulling or as soon as possible. The standard record structure should be discussed with a patient beforehand and include an individual's basic pulling features. It may comprise the time of day, the duration of the pulling episode, how many hairs were pulled and pulling location, environment characteristics (bathroom, bed, etc.), physical states (e.g. fatigue, pain, etc.), emotions (e.g. anxiety, low mood, boredom, irritation, etc.), thoughts preceding hair pulling (e.g. hair symmetry, colour, texture, etc.), and thoughts and emotions following hair pulling (e.g. guilt, embarrassment, relief, pleasure). Patients may keep a diary in a most comfortable way (paper, mobile phone application, computer program).

In order to provide positive reinforcement and improve compliance, patient diaries or logs should be reviewed at each consultation to discuss achievements and difficulties in order to develop and correct methods applied at the next HRT steps (stimulus control and competing responses) and to focus on objectively achieved progress as it helps to monitor pulling changes throughout treatment.

Awareness training and self-monitoring may also include saving pulled hairs. This may prevent post-pulling manipulations, e.g. hair swallowing. But it is better to implement this procedure after the establishment of good rapport as it could be associated with considerable discomfort for the patient and lead to treatment refusal.

Patients with a predominance of automatic pulling may be advised to consider other awareness training approaches. 'Stop signs' or 'alarm signals' may be used to make an initiation of a pulling movement noticeable and aware (to put strong-smelling lotions or perfumes on hands and wrists, wear jangly bracelets, rings with small bells or bright nail polish) when hands are moving near the face and head. Visual reminders, such as bright stickers in the places of a high risk, e.g. on a mirror, could also improve awareness.

Self-monitoring should continue throughout the further treatment steps.

Stimulus Control (Table 16.4)

This step includes elimination, avoidance or correction of triggers for pulling behaviour. Pulling may be partly controlled by modification of certain circumstances that provoke it (e.g. modus vivendi in watching television, brushing teeth or doing makeup, reading or working on computer, etc.).

Pulling is often associated with behaviours that are part of a daily life and routine activities. Thus, total avoidance of these behaviours is often impossible. In such conditions, stimulus control may include an action modification, e.g. wearing gloves while engaging in triggering activities. (In some early TTM case reports, there were even more radical stimulus control methods suggested as highly effective, especially in infants, such as complete scalp haircut.)

It is important to distinguish between internal and external pulling triggers. It is often easier to control external stimuli, especially prominent in automatic pulling

Table 16.4 Stimulus control strategies in TTM

Trigger type	Trigger examples	Stimulus control possible behavioural strategy
Visual	Looking at hair in the mirror	Use dim light in the bathroom so that visual access is limited; remove mirrors (if possible), change a wall-type mirror to a hand-glass; limit time in front of a mirror (place a clock near a mirror to check time, use visual stop signals)
Tactile/ Proprioceptive	Sensations on fingertips	Wear gloves and/or change positions (i.e. do not rest head in hand) during high-risk activities
	Sensations on a hair-bearing skin region (itch, tingling, etc.)	Application of an itch-reducing lotion
Location	On the couch in front of the television	Avoid location, if possible (i.e. watch television in another room); modify position (watch short broadcasts, e.g. news, shows, on feet in a kitchen, long video on PC, etc.) and process (watch involved in other activities, e.g. cooking, etc.).
	In the bed before falling asleep	Go to bed when tired/sleepy only; if unable to fall asleep after 5 min, get up and return to bed when tired
Activity	Reading	Hold another object (e.g. pen, pencil,) in free hand or hold a book with both hands
	Driving	Keep both hands on the wheel; remove all instruments suitable for picking from the car

Modified from Grant J.E. et al. TTM, skin picking, and other body-focused repetitive behaviors; 2012; and Sarah H Morris, Hana F Zickgraf, Hilary E Dingfelder and Martin E Franklin. Habit reversal training in trichotillomania: guide for the clinician Expert Rev. Neurother. 13(9), 1069–1077 (2013)

(see Table 16.4). In these cases, the emphasis should be on awareness training with stimulus control. In focused pulling, which is usually more related to internal triggers, the three HRT steps strategy is worth to be modified to include emotion regulation issues, e.g. relaxation training and cognitive restructuring, acceptance-based strategies, and mindfulness.

Competing Responses

Competing responses include behavioural corrections or substitutes of hair pulling activities, applied until the urge episode passes. The latter may be replaced by manipulating with objects (e.g. stress balls, stretchy rubber toys) and other motor acts that make pulling difficult/impossible (e.g. clenching the fists, sitting on the hands, folding arms, putting hands in pockets, etc.). Ideally, this motor act should be (1) alternative, i.e. physically incompatible with the pulling; (2) universal, i.e. usable in almost any situation; (3) ordinary, i.e. unnoticeable to others, (4) acceptable to a patient.

Collaborative work with the patient helps to develop a list of strategies that can be applied flexibly across different situations in which pulling occurs in order to adjust competing responses to possible changes of TTM patterns during a process of treatment.

Prognosis

Prognosis becomes poorer as the age of onset approaches adulthood, i.e. adolescent to young adult sufferers usually have a more long-lasting form of the disorder and do not respond as well to treatment as children. Prognosis is poorer if there is no response to initial treatment, if focused pulling predominates (as it appears to worsen with age compared to automatic pulling), and if there are comorbid anxiety and depressive symptoms.

The prognosis is better when the disorder is diagnosed early and treatment begins early.

Bibliography

Ankad BS, Naidu MV, Beergouder SL, Sujana L. Trichoscopy in trichotillomania: a useful diagnostic tool. Int J Trichology. 2014;6(4):160–3. https://doi.org/10.4103/0974-7753.142856.

Duke DC, Keeley ML, Geffken GR, Stroch EA. Trichotillomania: a current review. Clin Psychol Rev. 2010;30:181–93.

França K, Jafferany M. Trichotillomania (hair pulling disorder): clinical characteristics, psychological interventions and emotional effects (psychiatry-theory, applications and treatments): Nova Science Pub Inc; 2017. p. 124.

Grant JE, Chamberlain SR. Trichotillomania. Am J Psychiatry. 2016;173(9):868–74. https://doi.org/10.1176/appi.ajp.2016.15111432.

Rothbart R, Amos T, Siegfried N, Ipser JC, Fineberg N, Chamberlain SR, et al. Pharmacotherapy for trichotillomania. Cochrane Database Syst Rev. 2013;(11):CD007662. https://doi.org/10.1002/14651858.CD007662.pub2.

Depression and Anxiety

17

Iyas Assalman

Common mental disorders such as anxiety disorder and depression, with lifetime prevalence of up to 20%, are frequently co-morbid with chronic skin disease. However, the relationship between the two is complicated. In dermatology the prevailing view is that skin disease causes distress, and likewise its treatment will reduce it. It is also true that mental disorders such as depression can increase the subjective distress and disability caused by chronic skin disease and can interfere with effective treatment adherence. Low mood and anxiety can contribute to flare ups in inflammatory skin disorders such as eczema and psoriasis.

Depression

Definition of the Disorder

The World Health Organization (WHO) defines depression as a common mental disorder, characterized by persistent sadness and a loss of interest in activities that the person normally enjoys, accompanied by an inability to carry out daily activities, for at least 2 weeks. In addition, people with depression normally have several of the following: a loss of energy; a change in appetite; sleeping more or less; anxiety; reduced concentration; indecisiveness; restlessness; feelings of worthlessness, guilt, or hopelessness; and thoughts of self-harm or suicide.

I. Assalman (✉)
East London Foundation Trust, London, UK

Queen Mary University of London, London, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_17

Classification

There are two main classification and diagnostic manuals to be considered when classifying mental health disorders (Table 17.1). These are the International Classification of Diseases 10th edition (ICD10), 11th Edition (ICD11) will come into effect on 1st January 2022, and the Statistical Manual of Mental Disorders, 5th Edition (DSM-5). The key depressive disorders are: Mild, Moderate, and Severe Depressive Episode, Recurrent Depressive Disorder and Dysthymia (Persistent Depressive Disorder).

Clinical Features and Presentation

The depressive mood is a pervasive state which is more likely to be experienced as unusual and atypical, associated with negative ideation like hopelessness and helplessness and may influence behaviour. Table 17.2 describes the DSM5 and ICD11

Table 17.1 Classification in ICD11 and DSM5

DSM5	ICD11
Disruptive mood dysregulation disorder	Single episode depressive disorder
Major depressive disorder, single and recurrent episodes	Recurrent depressive disorder
Persistent depressive disorder (dysthymia)	Dysthymic disorder
Premenstrual dysphoric disorder	Premenstrual dysphoric disorder
Substance/medication-induced depressive disorder	Mixed depressive and anxiety disorder
Depressive disorder due to another medical condition	Other specified depressive disorders
Other specified depressive disorder	Depressive disorders, unspecified
Unspecified depressive disorder	

Table 17.2 Depression symptoms as included in DSM5 and ICD11

DSM5
1. Depressed mood most of the day, nearly every day. 2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day. 3. Significant weight loss when not dieting or weight gain or decrease or increase in appetite nearly every day. 4. A slowing down of thought and a reduction of physical movement (observable by others, not merely subjective feelings of restlessness or being slowed down). 5. Fatigue or loss of energy nearly every day. 6. Feelings of worthlessness or excessive or inappropriate guilt nearly every day. 7. Diminished ability to think or concentrate, or indecisiveness, nearly every day. 8. Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
ICD11
A period of almost daily depressed mood or diminished interest in activities lasting at least 2 weeks accompanied by other symptoms such as: 1. Difficulty concentrating 2. Feelings of worthlessness or excessive or inappropriate guilt 3. Hopelessness 4. Recurrent thoughts of death or suicide 5. Changes in appetite or sleep 6. Psychomotor agitation or retardation 7. Reduced energy or fatigue

depression symptoms. However, negative beliefs such as loss of self-esteem and inappropriate guilt are specific core features of major depression. The duration of individual symptoms should be present most of the day and nearly every day during an episode.

Practice Point
The symptom which discriminates best between anxiety state and major depressive disorder is guilt.

The DSM-5 outlines the following criterion to make a diagnosis of depression. The individual must be experiencing five or more symptoms during the same 2-week period and at least one of the symptoms should be either (1) depressed mood or (2) loss of interest or pleasure. To receive a diagnosis of depression, these symptoms must cause the individual clinically significant distress or impairment in social, occupational, or other important areas of functioning. The symptoms must also not be a result of substance abuse or another medical condition.

In severe depressive episodes, psychotic symptoms like delusions and hallucinations can occur. Self-neglect may be present as a feature of depression and may impact on the adherence to treatment for depression and other medical illnesses like skin illnesses.

Some depressive symptoms are widely regarded as having special clinical significance and are here called 'somatic'. To qualify for the somatic syndrome, four of the symptoms described in Table 17.3 should be present.

Dysthymia is characterized by depressed mood for most of the day, for more days than not, as indicated by subjective accounts or observation by others, for at least 2 years. Presence while depressed of two or more of the symptoms in Table 17.4. During the 2 year period of the disturbance, the person has never been without symptoms from the above two criteria for more than 2 months at a time. Criteria for Major Depressive Disorder (MDD) may be continuously present for 2 years, in which case patients should be given co-morbid diagnoses of persistent

Table 17.3 Somatic symptoms

1. Marked loss of interest or pleasure in activities that are normally pleasurable
2. Lack of emotional reactions to events or activities that normally produce an emotional response
3. Waking in the morning 2 h or more before the usual time
4. Depression worse in the morning
5. Objective evidence of marked psychomotor retardation or agitation (remarked on or reported by other people)
6. Marked loss of appetite
7. Weight loss (5% or more of body weight in the past month)
8. Marked loss of libido

Table 17.4 Dysthymia symptoms as included in DSM5

1. Poor appetite or overeating
2. Insomnia or hypersomnia
3. Low energy or fatigue
4. Low self-esteem
5. Poor concentration or difficulty making decisions
6. Feelings of hopelessness

depressive disorder and MDD. To diagnose dysthymia there should be no manic episode, a mixed episode, or a hypomanic episode, the symptoms of dysthymia are not better explained by a psychotic disorder and are not due to the direct physiological effects of a substance (e.g. a drug of abuse or a medication) or a general medical condition, and the symptoms cause clinically significant distress or impairment in important areas of functioning.

Depression in older adults is under-reported and under-diagnosed. A variety of factors contribute to this. The presentation of depression in older adults is significantly different and the diagnostic criteria used for depression do not have the specific criteria for depression in older adults. Symptoms such as somatisation, hypochondriasis, psychomotor retardation, cognitive impairment and anxiety often mask the underlying depression leading to the diagnostic difficulties. With symptoms not conforming to the core criteria of depression as specified in the ICD-11, the presence of vague symptoms such as fatigue, insomnia, reduced appetite, etc. can be easily attributed to physical illness by patients and clinicians.

Practice Point
Biological symptoms, such as sleep, weight, and appetite are less helpful in the diagnosis of depression in physically ill patients, so it is vital to rely on depressive cognition such as guilt/rumination and negative thoughts.

Practice Point
Depressed mood, morning depression, and hopelessness are the key symptoms differentiating between medical patients with and without affective disorder.

The common differential diagnosis for depression in older adults is dementia, and depression often presents as apparent dementia (depressive pseudo-dementia). In depressive pseudo-dementia, the history of mood disturbance usually precedes cognitive symptoms; there is an unwillingness to answer questions rather than a failure of memory as in dementia patients. The deficits are often partial rather than

the global deficits seen in dementia. A trial of antidepressant therapy may sometimes be appropriate and helpful. Depression in older adults has a higher incidence in those with dementia and can coexist, and in this context make it more difficult to diagnose due to difficulties in communicating these symptoms.

Practice Point
The presence of dementia or mental retardation does not rule out the diagnosis of a treatable depressive episode, but communication difficulties are likely to make it necessary to rely more than usual for the diagnosis upon objectively observed somatic symptoms, such as psychomotor retardation, loss of appetite and weight, and sleep disturbance.

The indicators to look for in skin patients which may indicate depression are:

- Distress about skin disease being very severe.
- Mood change persistent (>2 weeks) and not responsive to the environment.
- Failure to adjust to the dermatology illness.
- Exaggerated perception of altered body image.
- Feeling ugly and disfigured which is out of proportion to the objective assessment.
- Difficulty adhering to treatments.
- Feeling overwhelmed.
- Physical function poorer than expected.
- Failure to continue/resume social and work roles.
- Recovery slower than expected, even after the skin improves.
- Non-dermatological presentations, e.g. burning sensations are frequently associated with depression.

Epidemiology and Aetiology

Prevalence of depression	The cumulative prevalence in those aged 18–32 may be over 40% Women to men ratio is at least 2:1
Depression is a common and debilitating disease	WHO in 2012 ranked depression as the fourth leading cause of disability worldwide
Risk factors	Genetic factors Gender Childhood experiences Personality Social environment Physical illness

Course and duration	Mean age of onset: 33 years (inpatients) 29.4 years (outpatient) The length of a depressive episode: 5.4 months Chronic depression is developed in 18–25% 43–52% of depressed outpatients became symptom-free between their episodes Unipolar depressives have twice the mortality risk of the general population

Diagnosis and Differential Diagnosis

DSM 5 and ICD 11 describes the criteria to diagnose depressive disorders with minor differences between the required numbers of symptoms (Table 17.1) and the duration of these symptoms.

In a busy clinic, using short questionnaires might be the most useful tool to identify patients with possible depressive disorder. The PHQ-2 (Patient Health Questionnaire) has been presented as an ultra-short screening questionnaire to detect depression in several healthcare settings. It consists of two questions on low mood and anhedonia, both questions being derived from the more accurate PHQ-9. The first question of the PHQ-2 enquires how often the patient has felt down, depressed, or hopeless over the past 2 weeks, with answers from 0 (not at all) to 3 (nearly every day). The second question enquires how often the patient has noted that he/she has lost pleasure or interest in doing things over the past 2 weeks, with the same possible answers. The cumulative score is added up, with a threshold score for considering the possibility of depression of three or higher. These patients should then be given more comprehensive diagnosis questionnaires, such as the PHQ-9 or (Hospital Anxiety and Depression Scale) HADS.

Patient Health Questionnaire (PHQ-9)

Over the last 2 weeks, how often have you been bothered by any of the following problems?
1. Little interest or pleasure in doing things?
2. Feeling down, depressed, or hopeless?
3. Trouble falling or staying asleep, or sleeping too much?
4. Feeling tired or having little energy?
5. Poor appetite or overeating?
6. Feeling bad about yourself—or that you are a failure or have let yourself or your family down?
7. Trouble concentrating on things, such as reading the newspaper or watching television?
8. Moving or speaking so slowly that other people could have noticed?Or the opposite—being so fidgety or restless that you have been moving around a lot more than usual?
9. Thoughts that you would be better off dead, or of hurting yourself in some way?

Hospital Anxiety and Depression Scale (HADS)

How you have been feeling in the past week?
1. I feel tense or 'wound up'.
2. I still enjoy the things I used to enjoy.
3. I get a sort of frightened feeling as if something awful is about to happen.
4. I can laugh and see the funny side of things.
5. Worrying thoughts go through my mind.
6. I feel cheerful.
7. I can sit at ease and feel relaxed.
8. I feel as if I am slowed down.
9. I get a sort of frightened feeling like 'butterflies' in the stomach.
10. I have lost interest in my appearance.
11. I feel restless as I have to be on the move.
12. I look forward with enjoyment to things.
13. I get sudden feelings of panic.
14. I can enjoy a good book or radio or TV program.

Psychiatric assessment and Mental State Examination (MSE) continue to be the standard for diagnosing depression and other psychiatric disorders, this is in addition to detailed medical history and physical health examination. Investigations like laboratory evaluation and imaging might also be needed.

Practice Point
All depressed patients must be questioned specifically about suicidal ideation and behaviour.

The differential diagnosis for depression includes a wide variety of medical and psychiatric disorders see Table 17.5. However, co-morbidity can also be a complicating factor when diagnosing depression. This also applies to psychiatric and medical illnesses. A careful psychiatric history helps to differentiate and clarify the diagnosis.

Treatment

The treatment for depression can be initiated and monitored in primary care as well as in some liaison clinics.

- The British Association for Psychotherapy (BAP) guidelines recommends using antidepressants as a first-line treatment for:

Table 17.5 Differential diagnosis for depression

Psychiatric	Medical
• Adjustment disorder with depressed mood • Attention deficit hyperactivity disorder • Bipolar disorder • Borderline personality disorder • Complicated grief • Delirium • Dementia • Schizophrenia and schizoaffective disorder	• Adrenal insufficiency • Huntington's disease • Hypercortisolism • Hypothyroidism • Mononucleosis • Multiple sclerosis • Obstructive sleep apnoea • Parkinson disease • Stroke • Systemic lupus erythematosus • Traumatic brain injury • Vitamin B12 insufficiency

  - Moderate and severe major depression in adults irrespective of environmental factors and depression symptom profile.
  - Depression of any severity that has persisted for 2 years or more.
- In the UK The National Institute for Health and Care Excellence (NICE) recommends the following:
- **For people with persistent subthreshold depressive symptoms or mild-to-moderate depression:**
  - Consider offering a low-intensity psychosocial intervention—In the UK this is usually accessed by referral or self-referral to IAPT (Improving Access to Psychological Therapies).
  - Consider group-based CBT for people who decline low-intensity psychosocial interventions.
  - Avoid the routine use of antidepressants, but consider this for people with:
    A history of moderate or severe depression.
    Subthreshold depressive symptoms that have persisted for a long period (typically at least 2 years).
    Subthreshold symptoms or mild depression that persists after other interventions.
    Mild depression that is complicating the care of a chronic physical health problem.
- **For people with moderate or severe depression:**
  - Offer an antidepressant and a high-intensity psychological intervention.
- **For people starting an antidepressant:**
  - Consider suicide risk and toxicity in overdose.
  - Explain that symptoms of anxiety may initially worsen.
  - Explain that antidepressants take time to work.
  - Explain that antidepressants should be continued for at least 6 months following remission of symptoms, as this greatly reduces the risk of relapse.

The choice between drug and non-drug treatments for depression should be informed by the evidence base, individual patient characteristics, patient choice and treatment availability.

Practice Point
Match choice of antidepressant drug to individual patient requirements as far as possible, taking into account likely short-term and long-term effects.

Practice Point
There is most evidence for selective serotonin reuptake inhibitors (SSRIs) which, together with other newer antidepressants, are first-line choices.

Antidepressants may be divided into the following classes:

- Monoamine uptake inhibitors
  - Tricyclic antidepressants (TCAs)
  - Selective serotonin reuptake inhibitors (SSRIs)
  - Noradrenaline reuptake inhibitors (NARIs)
  - Serotonin-noradrenaline reuptake inhibitors (SNRIs)
  - Noradrenaline-dopamine reuptake inhibitors (NRDIs)
- Monoamine oxidase (MAO) inhibitors
  - Non-selective monoamine oxidase inhibitors
  - Selective monoamine oxidase type A inhibitors
- Atypical antidepressants and other classes

SSRIs, SNRIs, and mirtazapine are commonly used in practice; these medications and their common side effects are listed in Tables 17.6, 17.7, 17.8, 17.9, and 17.10.

Antidepressants should usually be prescribed for 6 months to treat a patient with a single episode of unipolar depression.

Practice Point
Psychotropic medications have the risk of increasing QTc interval so there is a need for caution when using several drugs that can increase QTc interval.

Table 17.6 SSRIs and their dose ranges

SSRI	Adult daily dose range for depression (mg)
Citalopram	20–40
Escitalopram	10–20
Fluoxetine	20–60
Fluvoxamine	50–300
Paroxetine	20–50
Sertraline	25–200

Table 17.7 SSRI adverse effects

Symptom group	Effect
Gastrointestinal symptoms	Abdominal pain, constipation, diarrhoea, dyspepsia, nausea, vomiting, increased risk of bleeding
Sexual dysfunction	Anovulation, amenorrhoea, decreased libido and/or sexual arousal, galactorrhoea
Syndrome of inappropriate antidiuretic hormone secretion (SIADH)	Hyponatraemia manifesting as confusion, drowsiness, dizziness, seizures.

Table 17.8 Serotonin-noradrenaline reuptake inhibitors (SNRIs)

SNRI	Adult daily dose range for depression (mg)
Duloxetine	30–120
Venflaxine	75–375

Table 17.9 SNRI adverse effects

SNRI	Effect
Duloxetine	Constipation, decreased appetite, diarrhoea, dry mouth, nausea, vomiting, somnolence, headache, dizziness, insomnia, fatigue, sweating, erectile dysfunction
Venflaxine	Hypertension, headache, dizziness, nausea, dry mouth, constipation, abnormal ejaculation, impotence, insomnia, somnolence, nervousness, sweating

Table 17.10 Mirtazapine dose and adverse effects

Drug	Classification	Mechanism of action	Dose (mg)	Common adverse effects
Mirtazapine	Noradrenergic and specific serotonergic antidepressant (NaSSA)	Antagonizes the pre-synaptic alpha2 adrenoreceptor and serotonin receptor subtypes 5-HT2A, 5-HT2C, and 5-HT3 to increase central noradrenergic and serotonergic neurotransmission	15–45	Constipation, dizziness, drowsiness, dry mouth, increased appetite, somnolence, weight gain

Anxiety Disorders

Definition of the Disorder

Anxiety disorders include disorders that share features of excessive fear and anxiety and related behaviour disturbances. Fear is the emotional response to real or perceived imminent threat, whereas anxiety is the anticipation of future threats. Fear is associated with surge of autonomic arousal necessary for fight or flight, thoughts of immediate danger and escape behaviours, anxiety however is often associated with muscle tension and vigilance in preparation of future danger and cautious or avoidant behaviours.

Classification

There are several anxiety disorders in ICD11 and DSM-5, including generalized anxiety disorder, specific phobia, social anxiety disorder, separation anxiety disorder, agoraphobia, panic disorder, and selective mutism. The anxiety disorders differ from one another in the types of objects or situations that induce fear, anxiety, fear or avoidance behaviour and the associated cognitive ideation.

Clinical Features and Presentation

We will discuss generalized anxiety disorder (GAD), social anxiety disorder (SAD), agoraphobia, and panic disorder in this chapter. Individuals with GAD experience persistent anxiety and worry that is out of proportion to actual events or circumstances which involve minor or everyday matters. Often the focus of the worry shifts from one concern to another. The worries often interfere with concentration and performance. Individuals with social anxiety disorder fear situations involving potential evaluation by others and experience incapacitating levels of anxiety and a desire to escape or avoid situations. The most characteristic type of panic attack is the spontaneous 'out of the blue' episode of extreme anxiety. A significant number of people with panic attacks go on to develop fear and avoidance of situations associated with previous panic attacks. Some patients develop agoraphobic avoidance following their first attack. Tables 17.11, 17.12, 17.13, and 17.14 give the criteria required to give the diagnosis in different anxiety disorders.

Table 17.11 DSM-5 and ICD 11 criteria to make a diagnosis of GAD

DSM-5 Generalized Anxiety Disorder Criteria
A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance). B. The individual finds it difficult to control the worry. C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months): 1. Restlessness, feeling keyed up or on edge. 2. Being easily fatigued. 3. Difficulty concentrating or mind going blank. 4. Irritability. 5. Muscle tension. 6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep). D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. E. The disturbance is not attributable to the physiological effects of a substance (e.g. a drug of abuse, a medication) or another medical condition (e.g. hyperthyroidism). F. The disturbance is not better explained by another medical disorder.
ICD11 Generalized Anxiety Disorder Criteria
1. Marked symptoms of anxiety that persist for at least several months, for more days than not, manifested by: (a) Either general apprehension (i.e. 'free-floating anxiety') or (b) Excessive worry focused on multiple everyday events, most often concerning family, health, finances, and school or work 2. Together with additional symptoms such as: (a) Muscular tension or motor restlessness (b) Sympathetic autonomic over-activity (c) Subjective experience of nervousness (d) Difficulty maintaining concentration (e) Irritability (f) Sleep disturbance 3. The symptoms result in significant distress or significant impairment in personal, family, social, educational, occupational, or other important areas of functioning. 4. The symptoms are not a manifestation of another health condition and are not due to the effects of a substance or medication on the central nervous system.

Epidemiology and Aetiology

Prevalence of anxiety disorders	Up to 33.7% of the population are affected by an anxiety disorder during their lifetime. Women to men ratio is 2:1
Burden of the illness	It was estimated that in 2004, anxiety disorders cost in excess of 41 billion euros in the European Union. The work loss days for some anxiety disorders are higher than for common somatic disorders such as diabetes.
Age of onset and course:	Anxiety disorders start in childhood, adolescence, or early adulthood until they reach a peak in middle age, then tending to decrease again with older age. Anxiety disorders are chronic, i.e. patients may suffer from their disorder for years or decades.
Co-morbidity	There is a high overlap among the anxiety disorders and between anxiety disorders and other mental health disorders, respectively.

Table 17.12 DSM-5 and ICD11 criteria to make a diagnosis of social anxiety disorder

DSM-5 Social Anxiety Disorder Criteria
A. Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Examples include social interactions (e.g. having a conversation, meeting unfamiliar people), being observed (e.g. eating or drinking), and performing in front of others (e.g. giving a speech).
B. The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e. will be humiliating or embarrassing; will lead to rejection or offend others).
C. The social situations almost always provoke fear or anxiety.
D. The social situations are avoided or endured with intense fear or anxiety.
E. The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the socio-cultural context.
F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.
G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
H. The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g. a drug abuse, a medication) or another medical condition.
I. The fear, anxiety, or avoidance is not better explained by symptoms or another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.
J. If another medical condition (e.g. Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.
ICD11 Social Anxiety Disorder Criteria
1. Marked and excessive fear or anxiety that consistently occurs in one or more social situations such as: (a) Social interactions (e.g. having a conversation) (b) Being observed (e.g. eating or drinking) (c) Performing in front of others (e.g. giving a speech)
2. The individual is concerned that he or she will act in a way, or show anxiety symptoms, that will be negatively evaluated by others.
3. The social situations are consistently avoided or else endured with intense fear or anxiety.
4. The symptoms persist for at least several months and are sufficiently severe to result in significant distress or significant impairment in personal, family, social, educational, occupational, or other important areas of functioning.

Practice Point

The highest correlations among the anxiety disorders were found between seasonal affective disorder (SAD) and agoraphobia, between panic disorder and agoraphobia, and between specific phobia and agoraphobia.

Table 17.13 DSM-5 and ICD11 criteria to make a diagnosis of panic disorder

DSM-5 Panic Disorder Criteria
A. Recurrent unexpected panic attacks. A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four (or more) of the following symptoms occur: 1. Palpitations, pounding heart or accelerated heart rat 2. Sweating 3. Trembling or shaking 4. Sensations of shortness of breath or smothering 5. Feeling of choking 6. Chest pain or discomfort 7. Nausea or abdominal distress 8. Feeling dizzy, unsteady, light-headed, or faint 9. Chills or heat sensations 10. Paraesthesia (numbness or tingling sensations) 11. Derealization (feelings of unreality) or depersonalization (being detached from oneself) 12. Fear of losing control or 'going crazy' 13. Fear of dying
B. At least one of the attacks has been followed by 1 month (or more) of one or both of the following: 1. Persistent concern or worry about additional panic attacks or their consequences (e.g. losing control, having a heart attack, 'going crazy'). 2. A significant maladaptive change in behavior related to the attacks (e.g. behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).
C. The disturbance is not attributable to the physiological effects of a substance (e.g. a drug of abuse, a medication) or another medical condition (e.g. hyperthyroidism, cardiopulmonary disorders).
D. The disturbance is not better explained by another mental disorder (e.g. the panic attacks do not occur only in response to feared social situations, as in social anxiety disorder; in response to circumscribed phobic objects or situations, as in specific phobia; in response to obsessions, as in obsessive-compulsive disorder; in response to reminders of traumatic events, as in posttraumatic stress disorder; or in response to separation from attachment figures, as in separation anxiety disorder).
ICD11 Panic Disorder Criteria
1. Recurrent unexpected panic attacks that are not restricted to particular stimuli or situations.
2. Panic attacks are discrete episodes of intense fear or apprehension accompanied by the rapid and concurrent onset of several characteristic symptoms: (a) Palpitations (b) Increased heart rate (c) Sweating (d) Trembling (e) Shortness of breath (f) Chest pain (g) Dizziness (h) Light-headedness (i) Chills (j) Hot flushes (k) Fear of imminent death
2. In addition, panic disorder is characterized by persistent concern about the recurrence or significance of panic attacks, or behaviours intended to avoid their recurrence, that results in significant impairment in personal, family, social, educational, occupational, or other important areas of functioning.
3. The symptoms are not a manifestation of another health condition and are not due to the effects of a substance or medication on the central nervous system.

Table 17.14 DSM-5 and ICD 11 criteria to make a diagnosis of agoraphobia

DSM-5 Agoraphobia Criteria
A. Marked fear or anxiety about two (or more) of the following five situations: 1. Using public transportation such as automobiles, buses, trains, ships, or planes. 2. Being in open spaces such as parking lots, marketplaces, or bridges. 3. Being in enclosed places such as shops, theatres, or cinemas. 4. Standing in line or being in a crowd. 5. Being outside of the home alone.
B. The individual fears or avoids these situations because of thoughts that escape might be difficult, or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms such as fear of falling in the elderly or fear of incontinence.
C. The agoraphobic situations almost always provoke fear or anxiety.
D. The agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety.
E. The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situations and to the socio-cultural context.
F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.
G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
H. If another medical condition such as inflammatory bowel disease or Parkinson's disease is present, the fear, anxiety, or avoidance is clearly excessive.
I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, for example, the symptoms are not confined to specific phobia, situational type; do not involve only social situations as in social anxiety disorder, and are not related exclusively to obsessions as in obsessive-compulsive disorder, perceived effects of flaws in physical appearance as in body dysmorphic disorder, reminders of traumatic events as in posttraumatic stress disorder, or fear of separation as in separation anxiety disorder.
Note: Agoraphobia is diagnosed irrespective of the presence of panic disorder. If an individual's presentation meets the criteria for panic disorder and Agoraphobia, both diagnoses should be assigned.
ICD11 Agoraphobia Criteria
1. Marked and excessive fear or anxiety that occurs in response to multiple situations where escape might be difficult or help might not be available, such as: (a) Using public transportation (b) Being in crowds (c) Being outside the home alone (e.g. in shops, theatres, standing in line)
2. The individual is consistently anxious about these situations due to a fear of specific negative outcomes (e.g. panic attacks, other incapacitating or embarrassing physical symptoms).
3. The situations are actively avoided, entered only under specific circumstances such as in the presence of a trusted companion, or endured with intense fear or anxiety.
4. The symptoms persist for at least several months and are sufficiently severe to result in significant distress or significant impairment in personal, family, social, educational, occupational, or other important areas of functioning.

Practice Point

The correlation between GAD with major depression was particularly high, and high correlations were found between dysthymia and GAD or SAD.

Diagnosis and Differential Diagnosis

According to a WHO study, only approximately half of the cases of anxiety disorders have been recognized, and only one third of the affected patients were offered drug treatment. For many patients, it may take years until they are referred to a specialist. According to a survey among psychiatrists who were experienced in the treatment of anxiety disorders, 45% of patients suffered from symptoms of GAD for 2 years or more before they were correctly diagnosed with the disorder.

DSM 5 and ICD 11 describe the criteria to diagnose anxiety disorders which are described in detail in Tables 17.11, 17.12, 17.13, and 17.14. As mentioned before, using scales like the HADS is a useful instrument in diagnosing patients in a clinical setting. However, Psychiatric assessment and Mental State Examination (MSE) continue to be the standard for diagnosing anxiety disorders, this is in addition to detailed medical history and physical health examination. Investigations like laboratory evaluation and imaging might also be needed.

Practice Point
All patients with anxiety disorders must be questioned specifically about suicidal ideation and behaviour.

Anxiety disorders have one of the longest differential diagnosis lists of all psychiatric disorders. Anxiety is a nonspecific syndrome and can be due to a variety of medical or psychiatric syndromes (Table 17.15).

Treatment

When the anxiety disorder is confirmed and the co-morbidity is assessed, consider using antidepressants as a treatment option. Consider an SSRI for first-line treatment, as SSRIs are effective across the anxiety and related disorders, in both the short-term and long-term and are generally well tolerated (Table 17.6). However, it is important to remain familiar with the evidence base for other classes of medication, as many patients do not respond to or are intolerant of SSRI treatment but may respond to other classes of psychotropic drugs.

The efficacy of psychological and pharmacological approaches is broadly similar in the acute treatment of patients with anxiety disorders. Certain forms of psychotherapy, such as exposure therapy, cognitive therapy and cognitive behavioural therapy (CBT), have largely consistent evidence of efficacy in the treatment of anxiety disorders. In the UK these therapies are usually accessed by referral or self referral to IAPT (Improving Access to Psychological Therapies). Self-help approaches, such as the use of internet-based educational resources, are potentially beneficial in patients with mild anxiety and depressive symptoms. The Royal College of Psychiatrists website has useful patient information leaflets which can be downloaded giving information on antidepressant medication use and on different psychological therapies.

Practice Point
Remind patients that response to psychological treatment is not immediate and that a prolonged course is usually needed to maintain an initial treatment response.

Table 17.15 Differential diagnosis for anxiety disorders

Psychiatric	Medical
• Alcohol-related psychosis • Alcoholism • Amphetamine-related psychiatric disorders • Anorexia nervosa • Body dysmorphic disorder • Brief psychotic disorder • Bulimia nervosa • Cannabis-related disorders • Conversion disorders • Delirium • Delirium tremens (DTs) • Delusional disorder • Depression • Dissociative disorders • Dysthymic disorder • Factitious disorder imposed on self (Munchausen's syndrome) • Hallucinogen use • Inhalant-related psychiatric disorders • Injecting drug use • Insomnia • Malingering • Personality disorders • Phobic disorders • Schizoaffective disorder • Schizophrenia • Shared psychotic disorder • Somatic symptom disorders • Stimulant use	• Acute respiratory distress syndrome • Acute gastritis • Addison's disease • Adrenal crisis • Anaphylaxis • Androgen excess • Asthma • Atrial fibrillation • Atrial tachycardia • Caffeine-related psychiatric disorders • Cardiogenic shock • Chronic gastritis • Diabetic ketoacidosis (DKA) • Diffuse toxic goiter (Graves' disease) • Digitalis toxicity • Encephalopathy, dialysis • Epilepsy surgery • Esophageal motility disorders • Esophageal spasm • Euthyroid hyperthyroxinemia • Folic acid deficiency • Food poisoning • Geriatric sleep disorder • Goiter • Hepatic encephalopathy • Hypercalcemia • Hyperparathyroidism • Hyperprolactinemia • Hypertensive encephalopathy • Immediate hypersensitivity reactions • Irritable bowel syndrome (IBS) • Lyme disease • Meningitis • Multifocal atrial tachycardia • Obstructive sleep apnoea (OSA) • Premenstrual dysphoric disorder • Primary aldosteronism • Primary hypersomnia • Primary insomnia • Rehabilitation and fibromyalgia • Sleep-wake disorders • Syndrome of inappropriate antidiuretic hormone secretion (SIADH) • Thyroiditis, subacute • Tourette's syndrome • Type 1 diabetes mellitus • Undifferentiated connective-tissue disease • Unstable angina • Uremic encephalopathy

Trigeminal Trophic Syndrome

18

Jonathan Kentley and Alia Ahmed

Definition

Trigeminal trophic syndrome (TTS) is an uncommon cause of facial ulceration resulting from damage to the trigeminal nerve system, leading to self-mutilating behaviour and ulceration of the skin in the distribution of cranial nerve V. Recently, an atypical form of TTS has been recognised.

Aetiology and Classification

Trigeminal Trophic Syndrome

TTS is described as the triad of anaesthesia, paraesthesia and secondary facial ulceration as a result of self-mutilation. The intractable anaesthesia and paraesthesia encountered by the patient leads to repeated manipulation of the area with resultant tissue damage. The condition occurs secondary to an insult of the trigeminal nerve, or its associated central structures. The nasal ala is the most common site of ulceration, felt to be related to its location at the junction of sensory innervation by the ophthalmic and maxillary branches of the nerve (McVeigh et al. 2018).

The majority of historical cases were reported to be iatrogenic, specifically resulting from ablative therapy for the treatment of trigeminal neuralgia; although this is now less common with modern ablation techniques (Kurien et al. 2011).

J. Kentley
Chelsea and Westminster Hospital, London, UK

A. Ahmed (✉)
King Edward VII Hospital, Frimley Health Foundation Trust, Windsor, UK

Royal London Hospital, Barts Health NHS Trust, London, UK
e-mail: dr.alia.ahmed@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_18

Table 18.1 Causes of TTS

• Ablation of CN V or transection of the Gasserian ganglion
• Posterior circulation ischaemic stroke
• Post-craniofacial surgery
• Acoustic neuroma
• Post-encephalitis
• Syphilis
• Vertebral artery dissection
• Vertebrobasilar insufficiency
• Posterior fossa tumour (astrocytoma, meningioma, haemangioma)
• Syringobulbia
• Trauma
• Herpes zoster infection
• Amyloid deposition
• Leprosy
• Idiopathic

Ischemic damage of the posterior vascular territory, including posterior inferior cerebellar artery infarction and vertebral artery dissection, is another common precipitant of the condition (Sawada et al. 2014; Khan and Hachement 2019). In fact, TTS was first recognised by Adolf Wallenberg, most known for his clinical description of the lateral medullary syndrome, in 1901 (Wallenberg 1901). A number of other causes have been documented in the literature (see Table 18.1), and 5% of cases are described as being idiopathic (Slater 2006).

Atypical Trigeminal Trophic Syndrome

Recently, an atypical variant of TTS (ATTS) has been recognized. These patients present with severe, extensive facial ulceration that is responsive to treatment (Kentley et al. 2017; Gkini et al. 2019). Whilst no history of trigeminal nerve injury is identified, nerve dysfunction may be revealed following nerve conduction studies.

Clinical Presentation

Patients with TTS report distressing dysaesthesias, which may be described as burning, crawling, itching or tingling (Lane and Deliduka 2008). These altered sensations stimulate self-manipulation and due to impaired perception of pain and temperature in the area, repeated manipulation leads to trauma and ulceration (Sawada et al. 2014). An important consideration is that patients typically deny this self-mutilating behaviour, though it may be highlighted by those close to the patient

(Gkini et al. 2019). Patients may also report the sensation of a blocked nose due to impaired perception of nostril airflow (McVeigh et al. 2018).

Ulceration is most frequently seen unilaterally at the nasal ala, for reasons already discussed, and is more common on the right (Sawada et al. 2014). The characteristic evolution of the lesion is of a small crust that develops into a crescenteric or Y-shaped ulcer and may extend to involve the cheek and lip (Rashid and Khachemoune 2007). Ulcers may be single or multiple and are confined to one dermatome, usually the maxillary (V2), and particularly along the route of the infraorbital nerve (Garza 2008). The tip of the nose is spared, due to its innervation by a distal branch of the ophthalmic (V1) branch of the nerve (Curtis et al. 2012). Fibrosis of healing ulcers may cause facial distortion leading to ipsilateral ectropion or 'sneer-like facial expression' (Dicken 1997). Ulceration may also affect the forehead, eyelid, cornea or scalp. Whilst ulceration in the mandibular (V3) branch has been reported, this is uncommon (Sawada et al. 2014; Kavanagh et al. 1996).

In atypical trigeminal trophic ATTS, patients admit to manipulating their skin and describe a great sense of relief. They describe pain or abnormal sensation in the area which builds up until they pick at or manipulate the area which they describe as relieving the triggering pain or sensation. Lesions may be more widespread, affecting the face bilaterally or involving more than one dermatome (Kentley et al. 2017; Gkini et al. 2019) (Fig. 18.1).

Fig. 18.1 A patient with Trigeminal Trophic syndrome

Why Is TTS so Debilitating?

The chronic and severe facial ulceration that accompanies TTS can be psychosocially devastating for patients and lead to secondary depressive symptoms, which should be managed by psychiatric assessment (Gkini et al. 2019). The dysaesthesia suffered by patients can be extremely distressing and must be addressed effectively. Secondary bacterial or viral infection is common, and in extreme cases, tissue loss may extend to the facial sinuses leaving the patient at risk of osteomyelitis and meningoencephalitis (Ziccardi et al. 1996).

Epidemiology

Prevalence

TTS is uncommon, with 107 published articles on the topic up to 2017 (Khan and Khachemoune 2019). The latency period between trigeminal nerve damage and development of TTS is reported to be between 2 weeks and 30 years (median 1 year) (Sawada et al. 2014; Sadeghi et al. 2004). Only a small proportion of those with trigeminal insult will, however, develop the condition and it is theorised that those that do are predisposed to skin picking (Kurien et al. 2011). Psychiatric conditions such as Alzheimer's disease may provoke TTS after a long latency period due to disinhibition of skin picking behaviour (Setyadi et al., 2007). According to historical data, 18% of patients that underwent trigeminal ablation developed TTS (Rashid and Khachemoune 2007).

Epidemiology

Patients of all ages are affected. There have been reports in patients between 14 months and 94 years developing TTS, with a mean of 57 years. A female preponderance of 2.2:1 has been documented (presumably due to increased incidence of trigeminal neuralgia in female patients) (Sawada et al. 2014; Slater 2006).

Course and Prognosis

The course of the condition is chronic and progressive; once ulceration appears it is persistent and follows the cycle of a non-healing wound driving further self-injury. Lesions may persist for many years and without the reversal of habitual mutilating behaviour, they are unlikely to spontaneously resolve. TTS is often highly refractory to treatment, though ATTS has been reported to display a favourable response to pregabalin (Gkini et al. 2019).

Diagnosis

General Considerations

TTS is a clinical diagnosis, however, it is essential to rule out other causes of facial ulceration. Inciting injury to the trigeminal nerve may be readily apparent from a careful history, or further extensive investigation may be required, including neuroimaging and neurophysiological studies. In the case of ATTS, it may be the case that no cause can be identified.

Differential Diagnoses

The differential causes of facial ulceration are extensive and should be carefully considered before making the diagnosis (Rashid and Khachemoune 2007).

Differential Diagnosis of TTS
• Bacterial infection: including mycobacterial (tuberculosis, leprosy), treponemal
• Viral infection: herpes simplex and zoster
• Parasitic infection: leishmaniasis
• Fungal infection
• Vasculitis
• Malignancy: squamous and basal cell carcinoma, lymphoma, sarcoma
• Granulomatous disease
• Pyoderma gangrenosum
• Psychodermatological disease: dermatitis artefacta, acne excoriée, delusional infestation

Diagnostic Process

History and Examination

A careful history should be taken from the patient to identify a possible precipitant of TTS. Careful attention should be paid to other neurological symptoms, including those of raised intracranial pressure and hearing loss that may indicate the presence of a tumour involving the trigeminal nerve or its ganglia. Enquiry regarding previous craniofacial surgery or facial rash should be made. The physician should ask about other psychological co-morbidities. Patients may benefit from psychiatric review to rule out conditions such as dermatitis artefacta and neurotic excoriation or obsessive-compulsive disorder. A full skin and neurological examination should be performed on all patients.

Investigation

- Skin swabs for microbiology (including mycobacterial and fungal) and viral PCR.
- Blood tests.

  - Full blood count, renal function, liver function, bone profile, inflammatory markers, haematinics, thyroid function, HIV, treponemal serology, autoimmune screen including anti-nuclear and anti-neutrophil cytoplasmic antibodies.
- Skin biopsy to rule out neoplastic and vasculitic conditions
  - Typical histological findings in TTS are of ulceration with adjacent lichenification and scarring with a mixed inflammatory infiltrate (Khan and Khachemoune 2019).
- Consider MRI and electrophysiological studies when the history reveals no cause, to confirm damage to the trigeminal nerve.

Treatment

Patients should be managed in a multidisciplinary setting with dermatologists, psychiatrists, psychologics, neurologists and, where necessary, plastic, maxillofacial and ophthalmic surgeons. Pain services may also need to be involved in case management.

Psychological Interventions

Patients with insight into their condition have been reported to have better outcomes (Brewer et al. 2016). Explaining the self-induced nature of the condition and instituting behavioural modifications such as habit reversal therefore play a key role in the management of the condition (McVeigh et al. 2018; Setyadi et al. 2007). Simple interventions such as trimming the fingernails and wearing scratch mittens may be beneficial (Sawada et al. 2014).

Wound Management

Simple hydrocolloid dressings play a role in wound management and also create an occlusive barrier from self-manipulation. However, the wound may reoccur once dressings are removed. Several studies have reported successful wound heading with thermoplastic dressings, which also play a role in addressing the patient's somatosensory symptoms (Brewer et al. 2016; Preston et al. 2006; Swan et al. 2009) Negative pressure therapy has also been reported as beneficial (Fredeking and Silverman 2008). Once wounds are healed, it is essential to address the underlying dysaesthesia to prevent recurrence.

Pharmacological Management

There are no randomised-controlled trials in the treatment of TTS, nor published guidelines. Gabapentin has the greatest body of evidence in the treatment of TTS; tiagabine and oxcarbazepine are reported as therapeutic options when the use of gabapentin is limited by side effects (Sadeghi et al. 2004). Carbamazepine and the use of SSRIs have been shown to be of benefit (Sawada et al. 2014; Gkini et al. 2019; Pedicelli et al. 2009; Fruhauf et al. 2008). The addition of topical tacrolimus ointment may use as an adjunctive therapy alongside gabapentin (Nakamizo et al. 2010). ATTS is described as being responsive to treatment with pregabalin (Gkini et al. 2019).

First-line therapeutic options for TTS	
Medication	Dose
Gabapentin[a]	Initially 300 mg 1–3 times daily Increased in steps of 300 mg every 2–3 days in 3 divided doses to maximum 3.6 g per day
Carbamazepine[b]	Initially 100 mg daily Increased slowly to 600–800 mg daily in 2 divided doses
Citalopram	20–40 mg daily

[a]Prolonged—release formulations of carbamazepine such as Tegretol® and Carbagen® SR are preferred

[b]Gabapentin and carbamazepine dose should be titrated to clinical response

Surgical Management

Plastic surgery has been considered a last resort and the recurrence of ulceration is common post-procedure (Tollefson et al. 2004). Rotational skin flaps from areas outside the affected dermatome, however, may be considered in refractory cases (McVeigh et al. 2018).

Transcutaneous electrical stimulation, autologous epidermal cell transplant and radiotherapy have all been trialled in refractory cases with varying degrees of success (Khan and Khachemoune 2019; Schwerdtner et al. 2005; Westerhof and Bos 1983).

Prognosis

TTS is a chronic condition that may be highly refractory to treatment. Where some studies have reported complete healing, follow-up times have been short, and patients frequently relapse (Sawada et al. 2014). As trigeminal nerve damage will

inevitably persist, patients must be followed up. Pharmacological treatment must be continued long-term alongside behavioural modifications and wound management to allow sustained healing of ulceration.

References

Brewer JD, Sciallis GF, Hanson JL. The treatment of trigeminal trophic syndrome with a thermoplastic dressing. Dermatol Surg. 2016;42:438–40.

Curtis AR, Oaklander AL, Johnson A, Yosipovitch G. Trigeminal trophic syndrome from stroke: an under-recognized central neuropathic itch syndrome. Am J Clin Dermatol. 2012;13:125–8.

Dicken CH. Trigeminal trophic syndrome. Mayo Clin Proc. 1997;72:543–5.

Fredeking AE, Silverman RA. Successful treatment of trigeminal trophic syndrome in a 6-year-old boy with negative pressure wound therapy. Arch Dermatol. 2008;144:984–6.

Fruhauf J, Schaider H, Massone C, Kerl H, Mullegger RR. Carbamazepine as the only effective treatment in a 52-year-old man with trigeminal trophic syndrome. Mayo Clin Proc. 2008;83:502–4.

Garza I. The trigeminal trophic syndrome: an unusual cause of face pain, dysaesthesias, anaesthesia and skin/soft tissue lesions. Cephalalgia. 2008;28:980–5.

Gkini MA, Ahmed A, Aguilar-Duran S, et al. Atypical variant of trigeminal trophic syndrome successfully treated with pregabalin: a case report series. Clin Exp Dermatol. 2019;44:225–8.

Kavanagh GM, Tidman MJ, McLaren KM, Goldberg A, Benton EC. The trigeminal trophic syndrome: an under-recognized complication. Clin Exp Dermatol. 1996;21:299–301.

Kentley J, Marshall C, Gkini MA, Taylor R, Bewley A. Atypical trigeminal trophic syndrome: an unusual cause of facial ulceration. Acta Derm Venereol. 2017;97:971–2.

Khan AU, Khachemoune A. Trigeminal trophic syndrome: an updated review. Int J Dermatol. 2019;58:530–7.

Kurien AM, Damian DL, Moloney FJ. Trigeminal trophic syndrome treated with thermoplastic occlusion. Australas J Dermatol. 2011;52:e1–4.

Lane JE, Deliduka S. Self-induced nasal ulceration from trigeminal trophic syndrome. Cutis. 2008;81:419–20.

McVeigh KA, Adams M, Harrad R, Ford R. Periocular manifestations of trigeminal trophic syndrome: a case series and literature review. Orbit. 2018;37:32–5.

Nakamizo S, Miyachi Y, Kabashima K. Treatment of neuropathic itch possibly due to trigeminal trophic syndrome with 0.1% topical tacrolimus and gabapentin. Acta Derm Venereol. 2010;90:654–5.

Pedicelli C, Paradisi A, Fazio M, Angelo C, Fazio R, Paradisi M. Trigeminal neurotrophic ulceration in Wallenberg's syndrome. Int J Dermatol. 2009;48:443–5.

Preston PW, Orpin SD, Tucker WF, Zaki I. Successful use of a thermoplastic dressing in two cases of the trigeminal trophic syndrome. Clin Exp Dermatol. 2006;31:525–7.

Rashid RM, Khachemoune A. Trigeminal trophic syndrome. J Eur Acad Dermatol Venereol. 2007;21:725–31.

Sadeghi P, Papay FA, Vidimos AT. Trigeminal trophic syndrome--report of four cases and review of the literature. Dermatol Surg. 2004;30:807–12; discussion 12

Sawada T, Asai J, Nomiyama T, Masuda K, Takenaka H, Katoh N. Trigeminal trophic syndrome: report of a case and review of the published work. J Dermatol. 2014;41:525–8.

Schwerdtner O, Damaskos T, Kage A, Weitzel-Kage D, Klein M. Autologous epidermal cells can induce wound closure of neurotrophic ulceration caused by trigeminal trophic syndrome. Int J Oral Maxillofac Surg. 2005;34:443–5.

Setyadi HG, Cohen PR, Schulze KE, et al. Trigeminal trophic syndrome. South Med J. 2007;100:43–8.

Slater R. Trigeminal trophic syndrome. Int J Dermatol. 2006;45:865–6.
Swan MC, Downie IP, Horlock N. Management of trigeminal trophic syndrome. Plast Reconstr Surg. 2009;123:1124–6; author reply 6
Tollefson TT, Kriet JD, Wang TD, Cook TA. Self-induced nasal ulceration. Arch Facial Plast Surg. 2004;6:162–6.
Wallenberg A. Klinische Beitrage zur Diagnostik akuter Herderkrankungen des Verlangerten Marks und der Bruke. Dt Z Nervenheeilk. 1901;19:227–31.
Westerhof W, Bos JD. Trigeminal trophic syndrome: a successful treatment with transcutaneous electrical stimulation. Br J Dermatol. 1983;108:601–4.
Ziccardi VB, Rosenthal MS, Ochs MW. Trigeminal trophic syndrome: a case of maxillofacial self-mutilation. J Oral Maxillofac Surg. 1996;54:347–50.

Skin Picking Disorders

19

Anthony Bewley

Key Features

- Most prevalent in middle aged women (30–50 years).
- Thought to be a part of the Obsessive-Compulsive Spectrum Disorders.
- May commence with inflammatory dermatoses.
- Or may commence in skin de novo as a manifestation of underlying 'stress'.
- May be done in a dissociated state where the patient is not aware of the habit of picking.
- Intense desire to pick/rub or scratch real or imagined lesions.
- Sites affected are usually easily accessible such as the face, upper back, extensors of arms and legs, genitalia and buttocks.
- Anxiety and depression are strongly associated comorbidities.
- Also associated with eating disorders and substance/alcohol abuse.
- Treatment is with management of the skin together with management of the OCD component and any associated comorbidities.

Background

Skin picking disorder may be broadly divided into compulsive and impulsive forms. Most skin picking research concentrates on the compulsive form, which includes trichotillomania and acne excoriee. Compulsive behaviour is defined as repeated

A. Bewley (✉)
Barts Health NHS Trust, London, UK

Queen Mary University of London, London, UK
e-mail: anthony.bewley@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_19

behaviour with an obsessive ideation component that recurs on a repeated basis and leads to psychosocial comorbidities, social impairment and potentially self-harm. Any attempts to curb the urge can lead to increased psychological tension, triggering the compulsive act.

Compulsive skin picking syndromes are repetitive, sometimes ritualistic behaviours, occurring at regular intervals during the day for a sustained period of time. Patients feel the urge to act and find relief in the activity. Attempts to control the urge cause tension to rise. Although carrying out the skin picking behaviour is not denied, the patient does not always spontaneously admit it. The behaviour is generally related to the relief of tension and can sometimes be pleasurable. There is often an initial reward in terms of reduction of tension or pleasure in the picking behaviour followed by intense feelings of shame, self-disgust and guilt. Most patients experience urges to pick the skin, which are reported as intrusive. Even if there is an underlying skin disease, the symptoms cannot account for the severity of the lesions. Patients may claim an underlying itch, which can complicate the diagnosis.

Impulsive behaviour, in dermatology, consists of isolated or recurrent acts of uncontrolled drives to manipulate the skin, sometimes without an obsessive component, and rapid but short relief. Conscious awareness of the behaviour can vary but patients can engage in this type of self-injury in dissociative states where they may not have full awareness or recollection of the behaviour afterwards. Other factors such as substance abuse, risk-taking behaviour and eating disorders may also be found. Impulsive forms of skin damage disorder can be called ‘non-suicidal self-injury’ where there is no conscious suicidal intent. This may involve biting, cutting, scratching, hitting and burning. However, moderate to severe forms can be associated with suicidal ideation and suicidal attempts. The impulsive form of the condition may not fall within the realm of obsessive-compulsive disorder (OCD) and may occur in the context of abnormal personality development, e.g. emotionally unstable personality disorder.

The DSM-5 classification has listed self-induced dermatoses under the diagnostic group termed as ‘Obsessive-Compulsive and related disorders’. Within this classification system, the impulsive form of the skin picking disorder is separate from the compulsive form.

Clinical Presentation

Lesions may arise from pre-existing skin problems like acne or urticated papules or they may be created de novo. The most common sites of involvement are the face and back, followed by the neck, scalp and ears. The ‘butterfly sign’ is a characteristic feature as the areas of sparing where the patient is unable to reach bear a resemblance to the shape of a butterfly. Most patients use their fingernails to pick or squeeze lesions. Many patients also use instruments such as tweezers and needles.

General Symptoms

Patients may complain of itch, burning, pain, oozing and bleeding. Patients may complain that the symptoms keep them awake at night and that is why they become so tired and sleep deprived. Some patients indicate that their symptoms are persistently in their thoughts and that it is very difficult to ignore the symptoms and the urge to pick at the skin. Other patients (quite commonly) say that they find that they are picking at their skin unconsciously and that when they realise that they have been picking (or a friend or relative points this out to them) they have already damaged their skin quite badly. This then leads to feelings of shame and a sense of loss of control over body functions.

Dermatological Signs

(see Figs. 19.1–19.2)

Fig. 19.1 Scarring from extensive skin picking

Fig. 19.2 Skin picking can resemble linear tears of the skin

Lesions may range in size from a few millimetres to several centimetres. Lesions can affect any part of the body, but are more common on facial and visible skin. Sometimes the part of the body which is affected can be relevant (e.g. breasts and genitals in patients who have been sexually abused). And sometimes the dominant handedness of the patient can be relevant (e.g. the left side of the body may be more extensively involved in right-handed individuals, and the middle of the back may be spared areas due to its relative inaccessibility). Morphologies vary from superficial erosions to deep ulceration and even alteration of facial and other skin structures. Scarring, post-inflammatory hypopigmentation or hyperpigmentation and all stages of the healing process may be seen. Damage to skin appendages with alopecia and loss of sweating may be a feature. Scarring can be extensive and patients may find that they have severely altered their skin and have a very clear visible difference.

Psychiatric symptoms and comorbidities include

- Anxiety
- Depression
- Suicidal ideation
- Other OCD spectrum disorders
- Body image disturbance and loss of self-esteem
- Body Dysmorphic Disorder
- Eating disorders
- Substance and alcohol abuse

Differential Diagnosis

Skin picking disorder is separate from non-pathological manipulations of the skin such as piercings or tattoos which carry cultural or socio-aesthetic values. Grooming behaviours can also lead to episodic or repetitive skin manipulations which are not pathological and may or may not lead to skin lesions. The distinction between non-pathological and pathological skin picking is not always clear and this is especially in the case with children or in the initial stages of syndromes such as trichotillomania.

Skin picking disorder should be distinguished from other psychiatric or medical conditions which would better explain why the patient would inflict damage on themselves. Psychiatric conditions such as autistic spectrum disorders, schizophrenia, Tourette's syndrome and chronic tic disorders can lead to self-inflicted skin lesions. Delusional infestations can lead to self-inflicted skin damage due to scratching and picking but it would not be considered skin picking disorder as there is a primary psychiatric condition and delusional belief which explain the symptoms. Certain inherited conditions such as Lesch–Nyhan or Prader–Willi syndromes can lead to self-mutilation due to their neurological and behavioural abnormalities. Lesions arising from these conditions should not be considered to be part of the spectrum of skin picking disorder.

Practice Point
Some patients may pick at areas of skin where there is dysaesthesia (e.g. the trigeminal trophic syndrome or post-stroke patients). Localised areas of skin picking may be due to a habit too (e.g. lichen simplex chronicus unilaterally on genital skin; or picker's nodules). Please see chapters on dysaesthesia and trigeminal trophic syndrome.

Practice Point
To differentiate skin picking from Dermatitis Artefacta (and DA related disorders): patients with skin picking will usually (and sometimes reluctantly) admit that they are picking at their skin. They may also indicate that there are some symptoms which lead to their picking habit (e.g. itch or relief when the skin is picked). Patients with DA most commonly do not admit that they are harming their skin in some way and find it difficult to know how the skin damage happened.

Medical Causes of Self-Excoriating Behaviour

- Urticaria
- Uraemia
- Cholestatic hepatitis
- Xerosis
- Cutaneous dysaesthesia
- Porphyria cutanea tarda
- Malignancies

Psychiatric Causes of Self-Excoriating Behaviour

- Depression
- Anxiety
- OCD
- Body dysmorphic disorder
- Borderline personality disorder
- Delusions of parasitosis
- Dermatitis artefacta
- Somatoform disorders such as hypochondriasis

Epidemiology

The true extent of this disorder is unknown as there are only a few studies as to its overall incidence. Adding to the difficulty is the lack of a coherent classification system and the use of various terminologies and descriptions in different studies. Various studies have demonstrated an overall prevalence of between 1.4% and 18% of adults. One study showed that about one in five children reported occasional skin picking with 1.9% regularly indulging in this behaviour and reporting stress regulation difficulties. In a review of 18 studies, the rate of self-inflicted skin lesions ranged from 0.03% to 9.4%. The term, self-inflicted skin lesion is slightly different from skin picking disorder as it encompasses other conditions such as factitious disorder, and it is important to be precise about the exact diagnosis as much as possible. The condition can present at any age but the peak ages of presentation are between 30 and 50 years. There is a distinct female preponderance with a sex ratio of up to 1:3.

Diagnostic Process

Figure 19.3 shows a helpful algorithmic approach to identify the different diagnoses of self inflicted skin disease.

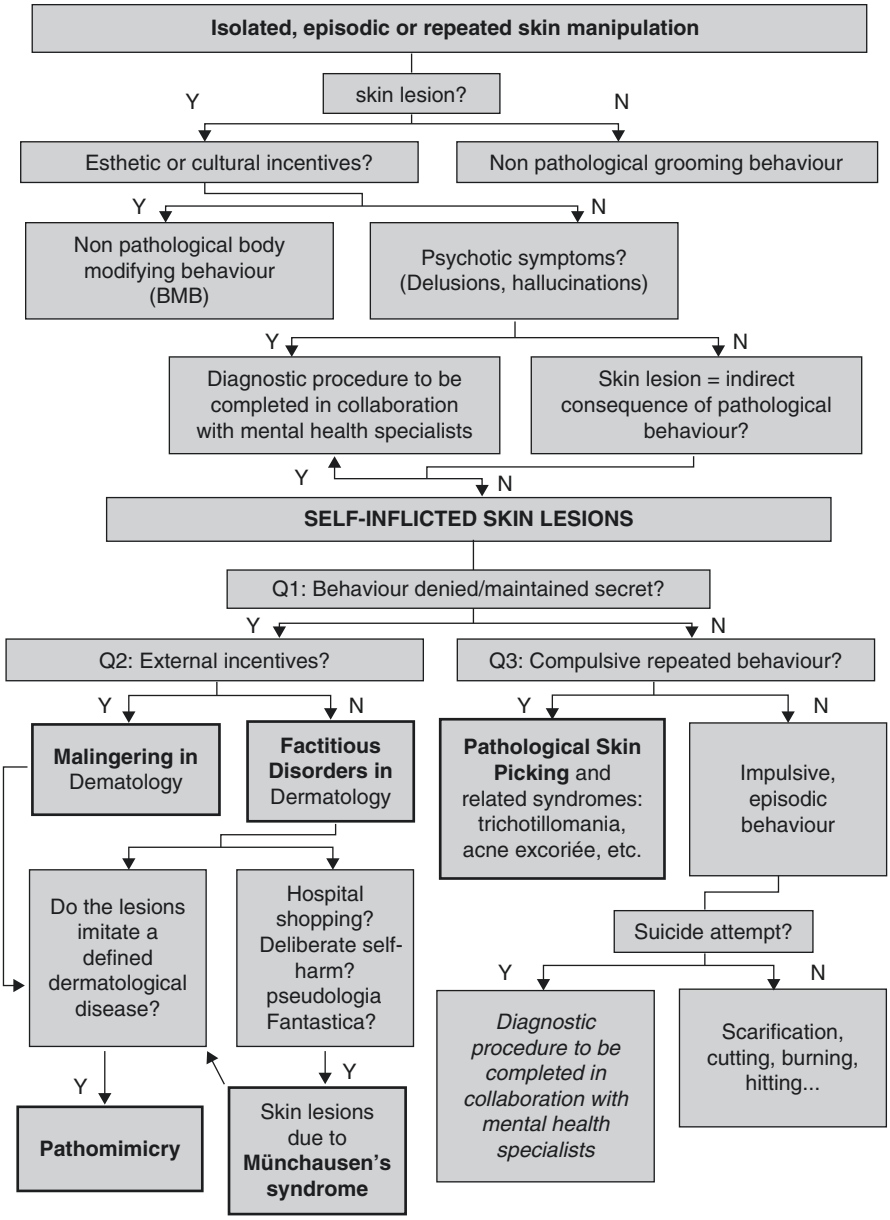


Fig. 19.3 European Society for Dermatology and Psychiatry classification of Skin picking Acta Derm Venereol. 2013 Jan;93(1):4–12

There are three questions that are helpful in correctly classifying abnormal behaviour leading to skin damage.

- Is the behaviour responsible for the somatic damage denied or kept 'secret' by the patient? A 'yes' answer points to a factitious disorder.
- If the answer to the first question is 'yes', are there any external incentives? A 'yes' answer indicates malingering, a 'no' answer points to factitious disorders.
- If the answer to the first question is 'no', is the behaviour responsible for the somatic damage compulsive or impulsive?

Investigations

Only where indicated by the clinical picture, and to exclude organic disease or as part of a pruritus investigation.

- **Blood tests** [Full Blood Count/Thyroid Function Test/Liver Function Test/Renal Function Test/Iron/Ferritin/Glucose], HIV serology and protein electrophoresis as clinically indicated.
- **Skin swabs** for microscopy and culture.
- **Skin biopsies with immunofluorescence** if needed.
- **Other tests** such as Chest X-ray/CT scans depending on the situation for suspected malignancies.

Treatment

Patients presenting with skin picking disorder should ideally be managed in a dedicated psycho-dermatology clinic, with the input of a dermatologist and psychiatrist with access to a psychologist and dermatology nurse specialist. A multidisciplinary approach is important as there may be different therapies treating both skin and mind. Assessment of psychosocial morbidities such as stressful life events and psychological trauma is important as these factors have been shown to have a direct impact on skin barrier function and immune responses. Simply managing the skin lesions does not deal with psychological suffering (Fig. 19.4). There are numerous scales which can be disease-specific such as the Y-BOCS assessment tool, or general quality of life tools such as DLQI and HAD that assess stress and the impact on the skin (see Chap. 30).

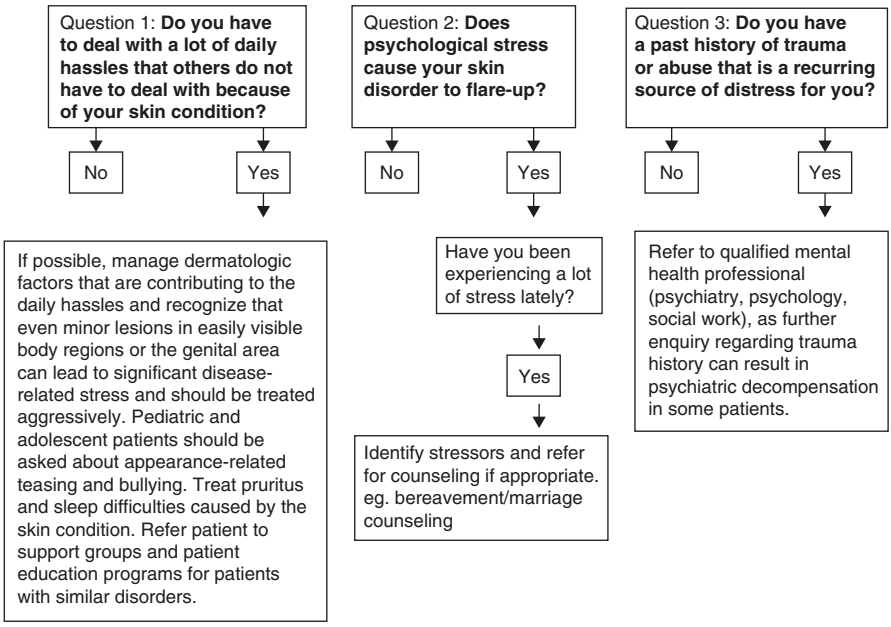


Fig. 19.4 An approach to the assessment and initial management of psychosocial stressors in a dermatology patient (Gupta MA, Levenson JL. The American Psychiatric Publishing textbook of psychosomatic medicine. 2nd edition 2011;667–90.)

Communication

Patients' lack of awareness of the psychological causes responsible for their symptoms can be a challenge in their management. A simplified discussion on stress-reactions in the skin and activation of skin nerves would be a good starting point in getting the patient to consider an holistic approach to their management. A clear statement about the complexity, rather than the difficulty, of the patients' case is recommended. It may also be helpful to explain that although the actual reason for their symptoms is not clearly understood, there are strategies that can be employed which can change the way the skin and brain process the signals it receives.

As with all psychodermatological conditions, patients should be dealt with in a non-confrontational manner. It is unlikely to be helpful for the patients to be told to simply stop the skin-damaging behaviour as they will often have already tried to resist the behaviour. Family members should be advised not to try to simply stop the behaviour unless the patient and the family have come to an agreement about this.

Practice Point
Treat the skin and the psychological disease/comorbidities concurrently
Treat the psychiatric/psychological component with non-pharmacological and pharmacological treatments concurrently where appropriate and acceptable to the patient (as the combination is likely to lead to greater treatment success)

Treatment of the Skin/Itch (See also Pruritus Chapter)

Topicals

Treatment is based on symptom severity. For example, if pruritus is an issue preparations of menthol containing emollients may be useful (e.g. in Menthol in Aqueous Cream) or 5% Doxepin cream. Cool compresses can be helpful to remove crusting and to soothe the skin.

Emollients (patient preference to be considered) with or without antiseptics can also be suggested to improve hydration, and thereby reduce the sensation of itch. A point to note here is that adopting a positive approach in dealing with the skin issues helps enormously as patients invariably become upset if the cutaneous component of their condition is overlooked.

Topical/intralesional steroids/tape to address the inflammatory component of existing lesions can be used as an adjunct for chronic or non-healing lesions. Combinations of antibiotics and glucocorticoids can also be applied in a tapering dose over days or weeks.

Phototherapy and Systemic Treatments

Phototherapy (TL01) for the whole body or localised, can be used with good results especially in cases where widespread itching is a feature. The mechanism of action is through the immunomodulatory and anti-inflammatory effects of phototherapy which leads to itch reduction and improvement of underlying dermatitis.

Antibiotics from the tetracycline group (Lymecycline or Doxycycline) have been tried with benefit where there may be a cutaneous super-infection. Tetracyclines may be preferred for their anti-inflammatory as well as antibiotic effects. Treatment courses tend to last for weeks or months depending on the response.

Conventional sedative antihistamines such as Hydroxyzine or Chlorpheniramine can help with itching. The antipruritic effects of Doxepin may be particularly beneficial for patients with associated depression and anxiety. It can be given in doses of between 10 and 20 mg in the elderly, and up to 75 mg in younger patients.

Treatment of the Psychological or Psychiatric Disease

Non-pharmacological Therapy (See also Chapter on Habit Reversal and Cognitive Behavioural Therapy)

Counselling can be beneficial in those patients who have psychosocial stressors that have precipitated, or are perpetuating their skin problem (e.g. bullying at school, marriage breakdown, bereavement). Cognitive behavioural therapy (CBT) can be very effective for patients with signs and symptoms of OCD, those with affective disease, and who are willing to engage with their psychologist. It involves tailored treatment according to the individual needs of the patient, but is often psychoeducation, thought re-training and cognitive restructuring which consists of helping patients change their habits [for example, through habit reversal training-see Chap. 29 on this] and by changing the perceptions of their appearance.

Other non-pharmacological therapies include Eye Movement Desensitisation And Reprocessing (EMDR), Mindfulness, hypnosis, relaxation techniques, acceptance and commitment therapy and bibliotherapy.

Cognitive behavioural therapy (CBT) is thought to be the most effective treatment for skin picking. There are a range of different talking therapies which may help patients with skin picking disorder, and the choice of which talking therapy is best can be made by the patient and clinician together. One form of CBT, schema therapy, has been used with obsessive-compulsive disorders, and small-group intensive CBT was found to improve obsessive-compulsive symptoms. CBT works by cognitive restructuring which consists of helping patients challenge the interactions of their thoughts, behaviour and feelings. Habit reversal techniques aimed at stopping the obsessive-compulsive disorder seem to have positive outcomes although evidence currently is limited. Cognitive interaction using a diary is another option for patients to become more conscious of their behaviour.

Two systemic meta-analyses have shown that combinations of talking therapies and psychopharmacology may be superior to each therapeutic line in isolation. But, as always in psychodermatology, the patient remains pivotal in the choice of therapeutics and patient-centred therapeutic approaches are likely to have better adherence and outcomes.

A–B–C Model of Habit Reversal for Skin Picking

A: Affect regulation
B: Behavioural addiction
C: Cognitive control

Commonly used psychological treatments in Skin Picking Disorder

Habit reversal
Relaxation techniques (including mindfulness)
Cognitive behavioural therapy
Acceptance and commitment therapy

Less commonly used psychological treatments used in SPD (possibly due to availability or cost)
Hypnosis
Psychodynamic therapy
Eye movement desensitisation and reprocessing (EMDR)

Pharmacological Therapy

Pharmacological Therapy
Commonly used medication
SSRIs, e.g. citalopram or fluoxetine often used in higher doses
SNRIs, e.g. duloxetine, venlafaxine
NaSSAs, e.g. Mirtazapine (watch for weight gain and sedation)
Tricyclics (e.g. doxepin)
Less commonly used
Antipsychotics, e.g. risperidone
N-acetyl Cysteine
Naltrexone
Anti-convulsants
Topiramate

The reader is referred to the chapter on psychopharmacology for more information on psychotropic medications. Obsessive-compulsive symptoms as seen in skin picking disorders have been associated with Serotonin mediated neural pathways. Antidepressants that selectively block serotonin uptake (SSRIs) can be of benefit in patients with this problem. Commonly used SSRIs include Citalopram, Sertraline and Fluoxetine. Mirtazapine, a noradrenergic and specific serotonergic antidepressant (NaSSA), is used primarily in the treatment of depression and has anxiolytic and sedative effects. Mirtazapine has a place on the therapeutic ladder where either the patient cannot tolerate SSRIs or where insomnia is a key feature. Tricyclic

antidepressants may be used (e.g. doxepin, a commonly used tricyclic in this condition, with its potent anti-histamine activity).

Less often used psycho-pharmacological medications such as N-acetyl cysteine (NAC) may be considered if other medications are not effective. Second and third-generation antipsychotics such as risperidone, olanzapine and aripiprazole are occasionally used for severe obsessive-compulsive disorder associated with self-inflicted skin lesions under specialist psychiatrist supervision. Psychiatrists should be involved in these cases. Anticonvulsive drugs such as lithium, carbamazepine, valproate and others are commonly used in bipolar disorder in psychiatry. These can be useful in certain conditions associated with self-inflicted skin lesions where the behaviour is triggered by rapid mood change, but the involvement of a psychiatrist would be required. Naltrexone, normally used in opioid toxicity, may be useful in skin picking disorder associated with severe pruritus. Benzodiazepines can be used very rarely for patients with anxiety but side effects and dependence mean that these drugs are often used only as a last resort or in special circumstances (usually around addiction issues). Topiramate has been used with anecdotal success.

Prognosis

The prognosis depends on a few factors. Predisposing factors, for example anxiety, depression and other psychiatric comorbidities may need to be addressed and dealt with. Further precipitants such as stressful life events may need to be addressed. The average duration of illness is reported to be around 5–8 years with relapses and remissions that parallel stressful situations.

Bibliography

Gieler U, Consoli SG, Tomás-Aragones L, Linder DM, Jemec GB, Poot F, Szepietowski JC, de Korte J, Taube KM, Lvov A, Consoli SM. Self-inflicted lesions in dermatology: terminology and classification--a position paper from the European Society for Dermatology and Psychiatry (ESDaP). Acta Derm Venereol. 2013;93(1):4–12.

Grant JE, Redden SA, Leppink EW, Odlaug BL, Chamberlain SR. Psychosocial dysfunction associated with skin picking disorder and trichotillomania. Psychiatry Res. 2016;239:68–71.

Gupta MA, Gupta AK. Self-induced dermatoses: a great imitator. Clin Dermatol. 2019;37(3):268–77.

Gupta MA, Vujcic B, Gupta AK. Dissociation and conversion symptoms in dermatology. Clin Dermatol. 2017;35(3):267–72.

Gardner–Diamond Syndrome

20

Anna V. Michenko, A. N. Lvov, Dmitry V. Romanov, and N. N. Potekaev

Definitions

Synonyms

Autoerythrocyte sensitization syndrome
Psychogenic purpura
Painful bruising syndrome
Painful blue spots

Gardner–Diamond syndrome (GDS) is an autoimmune disorder characterized by sensitization to phosphatidylserine of erythrocyte stroma often provoked or exacerbated by stressful events.

This syndrome is named after the American paediatrician Frank H. Gardner and the Russian-American paediatrician Louis Klein Diamond who first systematized the data about psychogenic purpura. However, the first description of this disorder dates back to 1927 when German psychiatrist F. Schindler described 16 patients

A. V. Michenko (✉) · A. N. Lvov
Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

D. V. Romanov
Department of Psychiatry and Psychosomatics, I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia

Department of Boundary Conditions and Psychosomatic Disorders, Mental Health Research Centre, Moscow, Russia

N. N. Potekaev
Director of Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_20

with similar skin haemorrhages. Soon after, another German psychiatrist, E. Jakobi, noted the association of psychogenic purpura with mental disorders (1929).

Epidemiology

GDS is an extremely rare disorder and probably underdiagnosed. Only about 185 cases have been described in the literature by now. These reports come from different countries: Japan, Germany, the United States, India, Turkey, Lebanon, Mexico, France, Norway, Czech, Spain, and Russia. Thus, GDS is considered to be a rare worldwide disorder.

Notably, this disorder is observed almost exceptionally in women, mainly younger than 30 years old. GDS in men, adolescents, and children is unlikely.

Aetiology and Potential Pathogenesis

Theories about the pathogenesis include:

- Originally Diamond suggested that skin lesions in patients treated by Gardner develop due to autosensitization of the patient to some components of his own blood. In 1955 they confirmed this suggestion by skin tests, and concluded that a causative agent is located in the erythrocyte stroma but not in the blood plasma and that it was not associated with haemoglobin.
- Ten years later, Groch et al. (1966) conducted a study to detect the specific substance in the erythrocyte stroma, involved in the development of GDS. They first revealed autosensitization to phosphoglyceride of red blood cells membrane called phosphatidylserine and suggested that it plays a pathogenetic role in GDS.
- Struneck et al. used indirect immunofluorescence and showed that more than 50% of erythrocyte phosphatidylserine of patients with GDS was redistributed on the outer surface of the cell membrane. They successfully induced such redistribution in an experiment after incubation of homologous erythrocytes from a healthy donor with blood plasma of patients, containing specific antibodies of the IgE class to cardiolipin and phosphatidylserine. Some role of immunologic disorders can also be suggested due to the successful treatment of GDS with plasmapheresis in comparison to placebo in one case report.
- Merlen suggested that disturbances in the tonus regulation of venous capillaries due to fluctuations in the kallikrein–kinin system may play an important role in the pathogenesis of GDS. Also, disorders of fibrin synthesis in the endothelium and formation of defective structures of capillary walls were detected as well as extravasation of erythrocytes, carrying sensitizing antibodies.
- Emotional factor may play a determinant role in the pathogenesis of this disease although the mechanisms mediating this association still need to be elucidated. For a long time, GDS was considered as a psychodermatosis, which develops in women with histrionic personality traits. Agle and Ratnoff noted marked emotional lability in patients with GDS and conversional symptoms, coinciding with mental stress. Besides that, some patients from their study suffered from mental disorders and received psychiatric treatment before the development of purpura.

- There are a few observational studies where typical GDS lesions were induced under hypnosis.

It is therefore unclear which mechanisms underlie stress influence on physiological processes in GDS and how it switches the immune system to the synthesis of autoantibodies to erythrocytes.

Although some authors observed gastrointestinal haemorrhages, haematuria, haemarthrosis, intermuscular haematoma and disorders of cerebral blood supply in patients with dermatological symptoms of GDS, the pathologic process is usually confined to skin lesions and not associated with disturbances in the blood coagulation system or abnormalities in vessel development. It was shown that these patients can receive surgical treatment without bleeding complications, although normally surgical interventions are contraindicated in persons with this condition.

There are only sporadic reports about comorbidity of GDS with thrombocytosis, defective thrombocyte aggregation, increase of activated partial thromboplastin time as a result of factor XII deficits, idiopathic thrombocytopenic purpura, and circulating fibrinolytic factor. GDS with haemorrhagic blisters was also reported in patient with alcohol dependence (the GDS features disappeared on cessation of alcohol use).

Clinical Presentation

Dermatologic Manifestations

The development of the disease is usually preceded by slight mechanical injuries, stress, surgical operations, or hard physical work. Sometimes, spontaneous development can be observed. In one case, a copper-containing intrauterine device (IUD) worsened the GDS symptoms. In some patients, the prodromal stage before GDS exacerbation includes general symptoms such as malaise and fatigue. But almost all patients note local burning and stinging sensations, sometimes itch of the skin, just before the development of typical lesions. A few minutes later specific induration of the corresponding skin area appear. The lesions become visible after 4–5 h, when painful oedematous plaques of pink to red in colour and 3–10 cm in diameter develop. The swelling may be severe (Fig. 20.1).

Gradually, the lesions become blueish yellow and, during the next 1–1.5 days, turn into ecchymoses. Erythema and swelling may be present for about 1 day or longer. After the regression of inflammatory infiltration, ecchymoses become less painful, change their colour (blue, greenish and then yellow) and entirely disappear within 7–10 days.

Although the lower limbs, especially on their ventral surfaces, and the trunk are the most often reported localizations of these lesions, they can appear on any other skin area, including the face. Multiple areas may be involved at the same time.

Mental Health Associations

In the majority of publications, mental health associations are considered as an important clinical sign of GDS; their development can be one of the diagnostic criteria of this disorder.

Fig. 20.1 Patient with Gardner–Diamond syndrome. The typical evolution of lesions can be seen: first lesions developed on breasts 2 days prior to the examination and became yellowish; on the left shoulder lesions appeared 1 day prior to the examination and still have a more bright red-bluish colour (photo courtesy of MD, PhD Mikhail Kochetkov)

Psychological Assessment

Many authors state that histrionic personality traits and tendencies to somatic reactions to emotional stimuli are commonly observed in these patients. Psychological examination, sometimes with psychological scales, may show emotional dysregulation.

Psychiatric Examination

According to recent reviews of patients with GDS, co-morbid symptoms of depression are most common (49% of patients), followed by anxiety (16%), personality disorder (4%), conversion disorder (4%), mixed anxiety and depressive disorder (4%), and bipolar disorder (2%). These results correspond to the results of Ratnoff's study who treated the biggest sample of 71 patients with GDS (predominantly women) in the University Hospitals of Cleveland. He also noted the predominance of depressive syndromes in this sample. Some patients also complained about sexual problems and demonstrated histrionic, explosive-dysphoric and obsessive-compulsive behaviour (the terminology for the personality traits was the terminology used at the time of the data publication).

Concomitant Disorders

The development of skin changes can be accompanied by several systemic disorders. Sometimes, the appearance of new skin lesions is associated with fever, arthralgias, myalgias, headaches, and dizziness. More than half of patients with GDS report gastrointestinal symptoms (epigastric pain, gastrointestinal haemorrhages, nausea, vomiting, diarrhoea), which develop simultaneously with the skin lesions. Some authors report haematuria, epistaxis, and menorrhagia. Glomerulonephritis was additionally diagnosed in one patient, and one had a lymphoid interstitial pneumonia. In one case, GDS with

angioimmunoblastic lymphadenopathy was described. Finally, in an original report from Gardner and Diamond, cerebrovascular disease was reported in two cases.

Diagnostic Process

The diagnosis of GDS is based on several clinical and laboratory criteria. An algorithm of examination includes (Table 20.1):

1. Detailed history.
2. Clinical dermatological examination.
3. Laboratory examination (blood count, coagulation parameters).
4. Test with intracutaneous injections of 1 ml of washed autoerythrocytes taken from patient.
5. Psychiatric and psychological examination.

Laboratory Examination

There are no specific laboratory changes in GDS. Haematological parameters are usually within normal ranges (including haemoglobin, haematocrit, platelet counts, peripheral smear, erythrocyte sedimentation rate, electrolytes, bleeding time, prothrombin, thrombin, partial thromboplastin time, factors of coagulation). All other laboratory signs of systemic disorders are usually absent.

The most reliable diagnostic test for GDS consists of an intracutaneous injection of 1 ml 80% suspension of washed erythrocytes obtained from the patient.

The test is positive if the typical for GDS inflammatory lesion develops within 24 h and then gradually progresses into ecchymosis (Fig. 20.2). The test should be made on skin areas the patient cannot access. In some modifications, it is possible to make this test with a suspension of washed autologous leucocytes, minimal quantity of heterologous or autologous DNA and with homologous erythrocytes of a healthy donor.

Table 20.1 Criteria of diagnosis of Gardner–Diamond syndrome

Diagnostic criteria	Comments
1. Typical history.	Episode of physical trauma or stressful event just before the manifestation of GDS, usually in women. A family history of bleeding disorders or platelet dysfunction should be considered.
2. Typical skin lesions.	Inflammatory infiltrated patches and plaques, which turn into painful ecchymoses within 24 h.
3. Parameters of blood count and blood coagulation system.	Usually within normal ranges.
4. Test with intracutaneous injections of 1 ml of autoerythrocytes.	Test is interpreted as positive when typical GDS lesions develop at the injection site in the following day.
5. Most patients with GDS have co-morbid mental health disease.	Depression, anxiety, conversion disorder, bipolar disorder, and personality disorder can be detected

Histopathology

Biopsies of the ecchymotic lesions reveal extravascular erythrocytes in the dermis, oedema, and non-specific lymphohistiocytic infiltration around the blood vessels in the middle and lower layers of the dermis. In macrophages, pigment deposition is observed, which stains positive for iron. In older lesions, subcutal oedema and haemorrhages can be observed. Leukocytoclastic changes in infiltrates or fibrinoid degeneration of vessels are not typical.

In most cases, the histopathological examination is not obligatory (Fig. 20.3).

Fig. 20.2 Positive intracutaneous test with own washed erythrocytes: typical ecchymotic lesions are seen on the left shoulder of the patient, whereas on the right shoulder there is no reaction at the place of saline injection (control site) (photo courtesy of MD, PhD Mikhail Kochetkov)

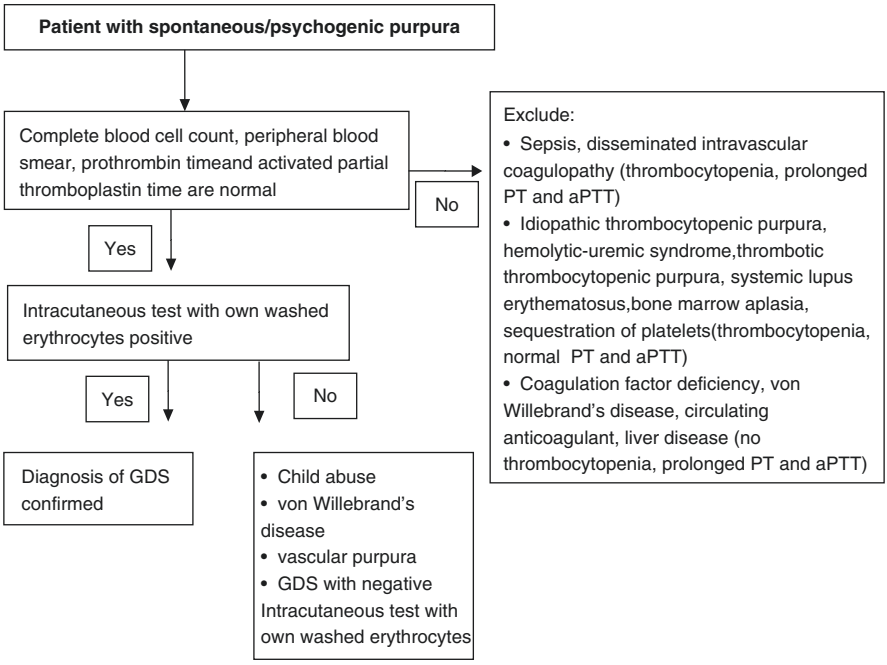


Fig. 20.3 Algorithm for diagnosis of GDS

Differential Diagnosis

Several conditions should be considered as differential diagnosis of GDS (Table 20.2) and should be excluded based on their typical clinical symptoms and laboratory parameters. In some cases, skin biopsy of lesional skin is justified.

Table 20.2 Differential diagnosis of Gardner–Diamond syndrome

Disorder	Distinctive features
Skin manifestations of coagulation disorders (disseminated intravascular coagulation, idiopathic thrombocytopenic purpura, etc.	Typical laboratory changes of blood parameters, history, familial predisposition in idiopathic thrombocytopenic purpura.
Anaphylactoid purpura (Henoch–Schönlein purpura)	This occurs predominantly in children, usually following an infection or medication. The diagnosis is made based on the presence of petechiae (without thrombocytopenia) or palpable purpura that predominantly affects the lower limbs plus at least one of the following four characteristics: abdominal pain; arthralgia or arthritis; renal involvement (proteinuria, red blood cell casts, or haematuria); proliferative glomerulonephritis or leukocytoclastic vasculitis with predominant deposition of IgA on histology.
Polymorphous dermal angiitis	Skin lesions usually have a smaller size and typical histological changes.
Angiitis nodosa	Systemic symptoms (fever, weight loss, myalgias, arthralgias), cutaneous involvement (nodules, livedo, neurologic manifestations, mononeuritis multiplex, peripheral neuropathy, central nervous system, cranial nerve palsy), gastrointestinal tract involvement, abdominal pain, nausea/vomiting, diarrhoea, hematochezia/melaena, hematemesis, oesophageal ulceration, gastroduodenal ulceration, colorectal ulceration, surgical abdomen/peritonitis), urologic and renal involvement (hypertension (recent onset), hematuria, proteinuria, orchitis/epididymitis), ophthalmologic manifestations, cardiac disease, respiratory tract disease pleuritis.
Spontaneous panniculitis (Pfeifer–Weber–Christian disease)	Skin lesions appear as long-existing profound subcutaneous nodes.
Ehlers–Danlos syndrome	In addition to skin ecchymoses, patients have other degenerative stigmas and malformations: excessive distensibility and fineness of skin, hypermobility of joints, muscle hypotonia, eye malformations, etc
Dermatitis artefacta, Münchhausen syndrome	History, clinical presentation (localization and dynamics of lesions) and inconsistent results of intracutaneous test 81–83. Münchhausen requires multiple hospitalizations with the aim to obtain intrusive investigations.
Physical trauma and abuse	History, clinical presentation.

The Course of the Disease

In the majority of patients, relapses and remissions of these lesions may last for many years. The remissions may be long-lasting. In some cases, clinical symptoms of GDS may persist and even worsen. Their severity may fluctuate considerably. The onset of new lesions is mostly seen after physical trauma or stress.

Treatment

Based on the high prevalence of mental disorders and stress reactivity in patients with GDS as well as the absence of coagulation disorders, psychotherapy and psychopharmacotherapy seem to be the most pathogenetically supported treatment modalities. A recent review of 45 patients with GDS reported a 100% success rate in patients receiving selective serotonin reuptake inhibitors (escitalopram, citalopram, and sertraline), and a 96% success rate for talking therapy, which included psychotherapy and reassurance therapy, and a 71% success rate in patients receiving tricyclic antidepressants (desipramine and amitriptyline). When patient management did not include the above-mentioned treatment modalities, the success rate was only 44%. Selective serotonin reuptake inhibitors (SSRIs) and corticosteroids (in combination) may be effective first-line treatments for GDS with proven efficacy in symptomatic relief. GDS refractory to initial treatment may require regular psychotherapy and titrated SSRI dosages to achieve long-term effects. Corticosteroids in isolation are often prescribed and improve symptoms to some extent, but they appear to be less effective than SSRIs. Most of other treatment modalities used in patients with GDS were not effective (Table 20.3).

Table 20.3 Other treatment modalities that were used in patients with GDS

Treatment modalities described in literature	Effectiveness
Cytostatic drugs, hormonal contraceptives, antibiotics, quinolones	Not effective
Busulfan (myelosan)	Effective in a patient with GDS, accompanied by thrombocytosis.
Promethazine	Was more helpful in relieving pain than tramadol
Beta-blockers	Not effective
Bioflavonoids and calcium entry blockers	Not effective
Hypno- and suggestive therapy, psychotherapy	Most effectively improved skin condition as well as mental disorders in young patients

Prognosis

Prognosis for life is favourable.

Practice Point
If you see purpura turning into ecchymoses without significant laboratory changes—ask the patient about stressful and emotionally important events and assess their influence on skin lesions.

Acne and Psychodermatology

21

Janet Angus

Introduction

Acne is estimated to affect 9.4% of the global population, making it the eighth most prevalent disease worldwide (Tan and Bhate 2015). It results in significant psychological problems, such as anxiety, depression, stigmatisation from peers, lower self-esteem, relationship difficulties, and higher unemployment rates (Koo 1995; Mulder et al. 2001). Severe psychological consequences such as depression, eating disorder, and body dysmorphic disorder are common among people with acne (Law et al. 2006).

The psychological distress can often significantly outweigh the physical impact of the disease. Acne is both very visible and inflammatory lesions leave the possibility of permanent scarring and consequent disfigurement (Fig. 21.1).

The psychological impact of acne can affect any age group and does not necessarily correlate with the disease severity (Yang et al. 2014). Patients should be routinely asked about the psychological impact of their disease to optimise treatments and outcomes.

It is important to make an assessment of the psychological impact of all patients with acne.

The severity of acne does not correlate with severity of psychological impact.

J. Angus (✉)
Bristol Royal Infirmary, Bristol, UK
e-mail: janet.angus@UHBw.nhs.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_21

Fig. 21.1 Nodulo-cystic acne can lead to significant scarring and disfigurement

Failure to address the psychological issues will result in poor outcome to treatment.

Prevalence of Psychological Impact Associated with Acne

Between 30 and 50% of adolescents with acne will experience psychological difficulties associated with their disease. This can vary from mild distress to extremely significant psychological impact with a risk of suicide (Goulden et al. 1999). Psychological abnormalities include self-reported depression and anxiety, embarrassment, social isolation, and psychosomatic symptoms including pain and discomfort.

Depression and anxiety has been found in 18% and 44% of acne patients, respectively (Kellett and Gawkrodger 1999). Six percent of acne patients in one study reported active suicidal ideation (Gupta and Gupta 1998). Patients with acne had greater impairment in mental health scores compared with those with asthma, epilepsy, diabetes, back pain, arthritis, or coronary artery disease. Suffering from acne is also associated with significantly higher levels of unemployment (Mallon et al. 1999).

Acne also occurs into adulthood with clinically significant acne in up to 12% of adult women and 3% of men. This persistent acne and consequent psychological impact often continues to middle age (Tanghetti et al. 2014). Older females with unremitting acne even if not severe, appear to be most negatively affected. Studies have shown a lack of self-confidence and poorer quality of life with depression and anxiety being common (Tan 2004; Stein and Hollander 1992).

The psychological impact of acne can frequently continue into adulthood.

Older women with persistent acne are severally affected.

Patients should be screened for suicide risk.

Pathogenesis of Psychological and Psychiatric Disorders in Acne

Acne commonly develops during adolescence, a vulnerable time of significant hormonal changes, development, and emotional instability.

There is increasing pressure on both young and old to conform to ideals of appearance and meet the socially perceived image of attractiveness and body image. Focus on self-image is exponentially increasing, in a digital world dominated by social media where photographs are shared and appraised continually.

Studies have found that teenagers and young adults make judgements about people's personality characteristic based on their skin. This judgement is a major contributor to the way adolescents deal with their acne and the psychological impact it has on them. In a study, adults and teenagers who viewed digitally altered photographs of teenagers with or without acne perceived the teenagers with acne as being shy, nerdy, stressed, lonely, boring, unkempt, unhealthy, introverted and rebellious, while adolescents with clear skin were perceived as being intelligent, happy, trustworthy, healthy, and creative (Ritvo et al. 2011).

Their relationships with their family and parents may also be affected, as parents worry about the impact acne will have on their child, and they may also lack education and knowledge about the causes and treatment, which may delay or prevent the teenager from seeking medical intervention (Dunn et al. 2011).

Acne and psychological distress can create a vicious cycle (see Fig. 21.2). Acne may cause psychosocial impact, which may result in a psychological disease such as anxiety and depression. This may result in less ability to cope with the disease and loss in faith in treatments or compliance with treatments. This worsens the skin condition.

Coping Skills that Become the Problem

Behaviours that are used to cope with stresses are sometimes referred to as safety behaviours. These are coping behaviours used to reduce anxiety and fear when threatened. These safety behaviours, although useful for reducing anxiety in the short term, sometimes become maladaptive and over the long term prolong anxiety and fear of non-threatening situations.

The common strategies used for coping with the impact of acne often can become the clinical presentations of psychological distress of acne and can become problem

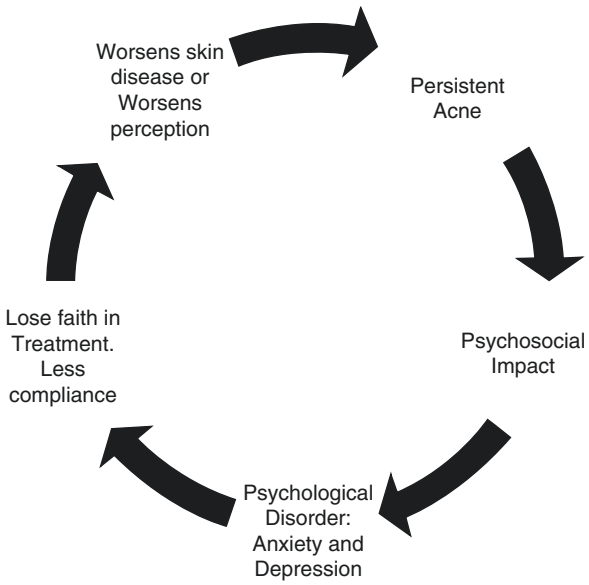


Fig. 21.2 Vicious cycle of acne and psychological impact

Table 21.1 Common safety behaviours in acne

Common safety behaviours	Behaviour	Initial benefit	Long-term issues
Avoidance	Failing to go to work or social events Avoiding relationships	Reduces anxiety	Lack of Social interaction Problems at Work Failure to form Relationships
Concealment	Excessive use of make-up	Alleviates fear of judgement	Makes acne worse Spending excessive time getting ready to leave house
Seeking reassurance	Asking partners or family about appearance	Provides reassurance	Causes relationship difficulties between family members Increases separation and isolation
Excessive checking	Repeatedly looking in reflective surfaces	Relieves anxiety	Increases preoccupation with skin

behaviours themselves (see Table 21.1). These would commonly be concealing the condition, avoidance of social interactions and failure to form relationships.

Clinical Presentations of Psychological Impact

Common psychological issues associated with acne may present with the features outlined in Table 21.2. Assessment should be made specifically of these areas in all acne patients. Behaviours are defined as problematic when they interfere with

Table 21.2 Common clinical presentations of psychological impact

Clinical presentations of psychological impact
Poor self-esteem and lack of confidence
A mismatch of severity of disease and impact
Social withdrawal
Excoriation disorder

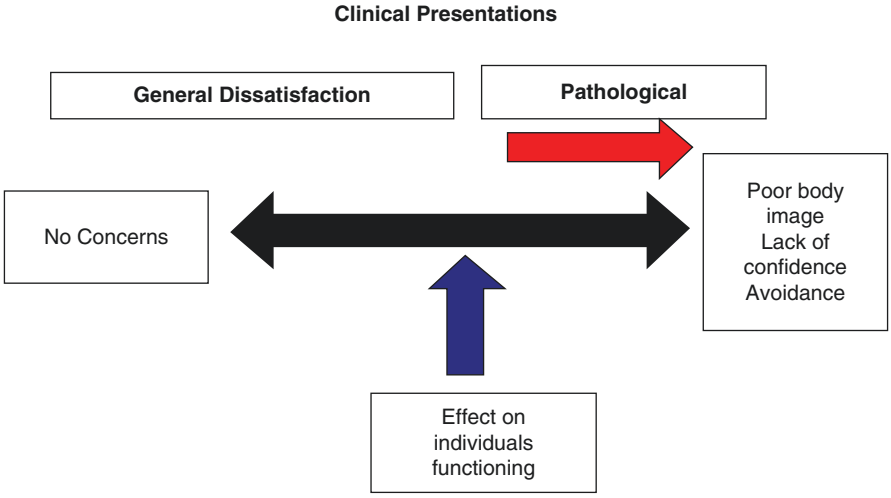


Fig. 21.3 Spectrum of severity in clinical presentations of psychological impact of acne

function. Behaviours and psychological issues lie on a spectrum of severity—see Fig. 21.3.

Poor Self-Esteem and Lack of Confidence

There is often poor-esteem and concerns with appearance. Signs of this are often about efforts to conceal disease. These might be for example:

- Growing their hair long
- Heavy use of makeup
- Refusal to remove make-up during consultations

A Mismatch of Severity of Disease and Impact

A common indicator of psychological difficulties is lack of a realistic appraisal of disease or type of treatment indicated. The severity of acne does not match with the

concern from the patients or the patient is visibly distressed. There may also be doubt about treatment success or insistence on the escalation of therapy, e.g. requesting isotretinoin in mild disease. Other features to note include:

- Tearful during consultations
- Significant parental concern
- Significant concern about scarring
- Seeking cosmetic or laser treatments

Social Withdrawal

At a time when teenagers are learning to form relationships, those with acne may lack the self-confidence to go out and make these bonds. They become shy and even reclusive. The main concern is a fear of negative appraisal by others. In extreme cases, social phobia can develop. These may present as:

- Retreating to their bedroom
- Avoidance of peers
- Avoidance of eye contact
- Failure to attend school or work or participate in sport
- Failure to form sexual relationships

Failure to Attend or Progress in Education and Work

- Missing school or sudden poorer academic performance
- Increasing sick days from work
- More likely to be unemployed
- Acne patients are less successful in job interviews due to lack of self-confidence

Excoriation Disorder

Excoriation disorder (ED), compulsive skin picking, dermatillomania or acne excoriee (Fig. 21.4) when seen in patients with acne has been categorised in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition (DSM-5) as an impulse control disorder. It is grouped with obsessive-compulsive and related disorders. It is an important sign to recognise and address as it is often an indicator of psychological distress (Lochner et al. 2012). Table 21.3 outlines the diagnostic criteria for Excoriation Disorder.

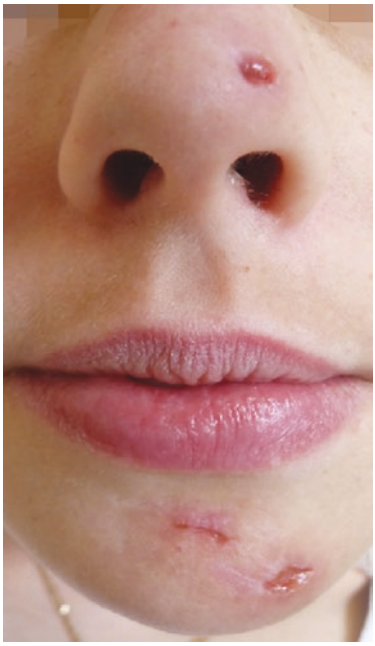


Fig. 21.4 Acne excoriee. Note picked areas on the face

Table 21.3 Diagnostic criteria for excoriation disorder

Diagnostic criteria include:
• Recurrent skin picking that results in skin lesions
• Repeated attempts to stop the behaviour
• The symptoms cause clinically significant distress or impairment
• The symptoms are not caused by a substance misuse or medical, or dermatological condition
• The symptoms are not better explained by another psychiatric disorder

ED is characterised by an inability to stop picking despite repeated efforts to do so and may lead to shame, anxiety, and depression (Odlaug and Grant 2011). It may occur at any age, but it generally has its onset in adolescence, typically coinciding with the onset of puberty (Flessner and Woods 2006). The picking may be preceded by a feeling of tension or anxiety that is relieved by the picking. There is an initial feeling of gratification and pleasure but this is rapidly followed by guilt. A range of behaviours or rituals may accompany the skin picking.

Individuals with ED often spend a significant amount of time on picking "binges" and camouflage. This can add up to several hours per day focusing on their skin and leads to them missing or being late for work, school, or social activities (Grant et al. 2012).

ED provokes shame and guilt both about the activity and their inability to control an obviously self-destructive behaviour.

Avoidance of situations or activities where skin lesions might be detected is common (Stein and Lochner 2017). Possible medical sequelae include

Table 21.4 Difference between excoriation disorder and dermatitis artefacta

Clinical presentations	Excoriation disorder	Dermatitis artefacta
Admits to picking	If gently asked easily admit to rubbing or picking	Deliberately conceals information from clinicians
Underlying psychiatric disease	Anxiety and depression	Often borderline personality disorder
Motivation	Associated shame and guilt about behaviour	May be seeking care/patient may not understand motivation

infections, lesions, scarring, and even serious physical disfigurement (Odlaug and Grant 2008). Individuals with ED may not commonly seek treatment for their condition; it is estimated that less than a fifth of patients seek treatment (Grant et al. 2012). Reasons for this are that the condition is accompanied by embarrassment, shame, or hopelessness. Those who do seek treatment more often present to a general practitioner or to a dermatologist rather than to psychological or psychiatric services.

Sometimes there is confusion with ED and Dermatitis artefacta (DA) and DA is diagnosed in error in cases of ED. Dermatitis artefacta (DA) is a factitious dermatological disorder in which skin lesions are self-induced to satisfy an unconscious psychological or emotional need. With DA there is a desire to manipulate and conceal the self-inflicted cause of the problem from health professionals. There is an association with more significant underlying psychiatric pathology. If asked sensitively, ED patients are usually happy to discuss their picking and there is not an association with complicated underlying psychiatric disorders. Table 21.4 outlines the differences between Excoriation Disorder and Dermatitis Artefacta.

Practice Points

Do not confuse the diagnosis of excoriation disorder and dermatitis artefacta. Excoriation disorder is a coping strategy for stress and anxiety.

Dermatitis artefacta (DA) is a factitious dermatological disorder in which skin lesions are self-induced to satisfy an unconscious psychological or emotional need.

With DA there is a desire to manipulate and conceal from health professionals and association with more significant underlying psychiatric pathology.

Assessment Tools

Psychological tools are very useful for the assessment of the psychological impact of the disease. They provide a structured assessment and are a helpful, non-judgemental way of quantifying anxiety and depression, monitoring disease and assessing suicide risk.

They are also useful in non-communicative teenagers. Moreover, these tools are easy to access and implement. They take around 5 min to complete.

Examples of such tools are as follows:

1. Hospital Anxiety and Depression scale (Zigmond and Snaith 1983). Developed in 1983, this is a 14-item scale in which seven items relate to anxiety and seven relate to depression.
2. The Patient Health Questionnaire (PHQ-9) (Kroenke et al. 2001) is a self-administered version of the PRIME-MD diagnostic instrument for common mental disorders. The PHQ-2 (Kroenke et al. 2003) is an abbreviated version that enquires about the frequency of depressed mood and anhedonia over the past 2 weeks.

   The PHQ-2 includes the first two items of the PHQ-9. The purpose of the PHQ-2 is to screen for depression in a "first-step" approach. Patients who screen positive should be further evaluated with the PHQ-9 to determine whether they meet criteria for a depressive disorder.
3. The Generalised Anxiety Disorder Assessment (GAD-7) (Spitzer et al. 2006) is a seven-item instrument that is used to measure or assess the severity of generalised anxiety disorder (GAD). Each item asks the individual to rate the severity of his or her symptoms over the past 2 weeks.
4. Cardiff Acne Disability Index (Motley and Finlay 1992). This five-item questionnaire is a shortened form of the Acne Disability Index (ADI), designed for use in teenagers and young adults.
5. The Acne-specific Quality of Life questionnaire (Acne-QoL) (Martin et al. 2001) is a 12-item health-related quality of life instrument developed for use in clinical trials to assess the impact of therapy on quality of life among persons 13–35 years of age with facial acne.

Identifying Psychiatric Conditions

Symptoms of Social Anxiety Disorder

Fear or anxiety specific to social settings.

Fear that they will display their anxiety and experience social rejection.

Social interaction will consistently provoke distress.

Social interactions are either avoided or reluctantly endured.

Fear and anxiety will be markedly disproportionate to the actual situation.

Persist for 6 months or longer and cause personal distress and impairment of functioning in one or more domains, such as interpersonal or occupational functioning.

Fear or anxiety cannot be attributed to a medical disorder, substance use, or adverse medication effects or other mental disorder.

Studies have found 15% of acne patients had clinically significant anxiety, and 6% had depression. Social anxiety appears particularly common. Identifying patients who fulfil diagnostic criteria for anxiety and depression is important, as early input from psychology and psychiatric colleagues may be crucial in successful management (Picardi et al. 2000).

Depression

The DSM-5 outlines the following criteria to make a diagnosis of depression:

The individual must be experiencing five or more symptoms during the same 2-week period and at least one of the symptoms should be either (1) depressed mood or (2) loss of interest or pleasure

Depressed mood most of the day, nearly every day.

Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.

Significant weight loss when not dieting or weight gain, or decrease or increase in appetite nearly every day.

A slowing down of thought and a reduction of physical movement (observable by others, not merely subjective feelings of restlessness or being slowed down).

Fatigue or loss of energy nearly every day.

Feelings of worthlessness or excessive or inappropriate guilt nearly every day.

Diminished ability to think or concentrate, or indecisiveness, nearly every day.

Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

To receive a diagnosis of depression, these symptoms must cause the individual clinically significant distress or impairment in social, occupational, or other important areas of functioning. The symptoms must also not be a result of substance abuse or another medical condition.

Body Dysmorphic Disorder

Individuals with Body Dysmorphic Disorder (BDD), also known as dysmorphophobia, are preoccupied with an imagined or slight defect in appearance; if a slight physical anomaly is present, the appearance concerns are excessive. To differentiate BDD from normal appearance concerns, the preoccupation must cause clinically significant distress or impairment in functioning, e.g. social or occupational interference.

BDD is classified as a somatoform disorder by the official psychiatric classification system, the Diagnostic and Statistical Manual of Mental Disorders (DSM). Dissatisfaction with treatment outcomes and professional interactions is common in patients suffering from BDD. Studies have found BDD is common in acne patients. The three most frequent compulsive behaviours in patients who screened positive for BDD were mirror checking (90.7%), camouflaging (79.1%) and using make-up (72.1%) (Marron et al. 2020) (Table 21.5).

Treating Underlying Psychological and Psychiatric Disease

The underlying principle is to treat both the acne and psychological or psychiatric consequence in conjunction. Psychological and psychiatric disease should be assessed and referred to psychology and psychiatric services if appropriate. Psychological, psychiatric and medical intervention (Fig. 21.5) can occur in conjunction and it is probably optimal if they can occur in parallel.

Appropriate treatment of the acne has been shown to improve the psychological impact. Longitudinal evaluation of psychometric outcomes has demonstrated that effective treatment of acne was accompanied by improvement in self-esteem, shame, embarrassment, body image, social assertiveness, and self-confidence. The majority of these patients were treated with oral isotretinoin (71%) (Klassen et al. 2000; Lasek and Chren 1998; Myhill et al. 1988).

Table 21.5 Screening tool for BDD (Phillips 2003)

Answering yes to all questions is a likely diagnosis of BDD
Do you currently think about a feature of your appearance you dislike?
If yes how many hours do you think about it?
Do you check in mirrors or reflective surfaces or touch your features a lot?
Do you compare your features often?
Does it cause you a lot of distress?
Do you avoid situations or people because of it?
Does it interfere with work, social life or relationships?

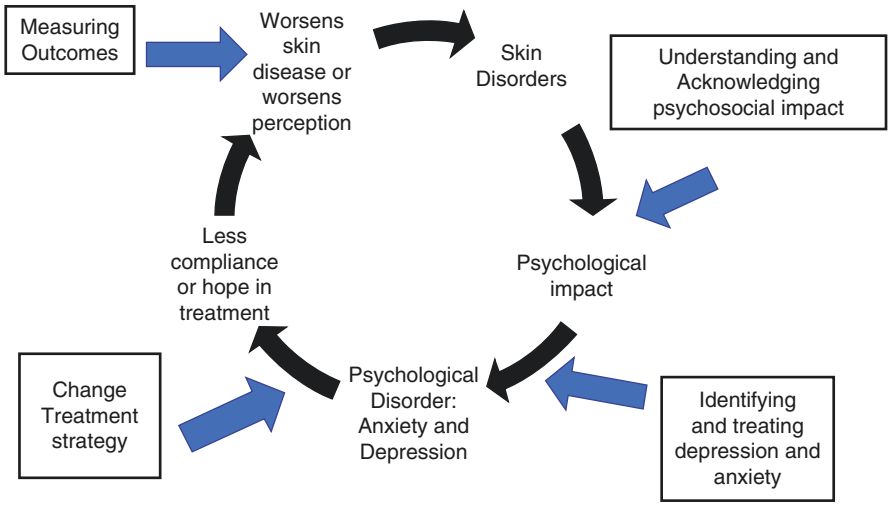


Fig. 21.5 Breaking the Cycle

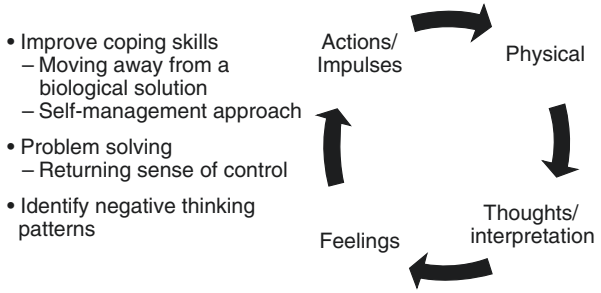


Fig. 21.6 CBT model

Psychological Interventions

Cognitive Behaviour Therapy (CBT)

CBT is a psychological intervention that focuses on how a person's thoughts, beliefs, feelings and behaviours affect their situation, see Fig. 21.6. CBT aims to identify unhelpful thinking patterns and behaviours that lead to psychological problems. Behavioural experiments allow an opportunity for people to test their belief systems in small graded individually designed challenges.

Habit Reversal Techniques for Excoriation Disorder

Habit Reversal is a simple form of intervention that involves several components, including:

- Building awareness of how much they are picking or unwanted behaviour
- Identifying and understanding the situations, places, activities, and urges that typically precipitate the behaviour
- Reducing cues that lead to the behaviour
- Developing a competing response that the person can use instead of the behaviour

Prescribing Antidepressants

It may be appropriate to prescribe antidepressants for anxiety and depression. Medications are often used in conjunction with cognitive behaviour therapy. The drugs of choice are the selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, paroxetine, sertraline and fluvoxamine. Some of these medications can also be helpful in compulsive skin picking, reducing impulse control disorders and BDD. Acetylcysteine has also been reported to be effective in ED.

Prescribing Isotretinoin

Management of patients with current or previous depression who require isotretinoin treatment for acne is a challenging area. A study published in 1983 asserted that oral isotretinoin could cause depressive symptoms (Hazen et al. 1983), and since then multiple publications have fuelled controversy on this subject. Current opinion favours the view that isotretinoin-induced mood disturbance is a rare, idiosyncratic reaction, not reliably related to the presence of pre-existing depression (Azoulay et al. 2008a; Bremner et al. 2012).

Most studies have not found an association between oral isotretinoin and depression, but rather have found a beneficial effect of reduced depressive symptoms with the treatment (Kaymak et al. 2006; Strahan and Raimer 2006; Marqueling and Zane 2007; Hahm et al. 2009; Gnanaraj et al. 2015; Singer et al. 2019; Li et al. 2019; Huang and Cheng 2017). Despite this, there may be reluctance to prescribe the drug especially in the context of the presence of psychological difficulties. However, delays in adequate treatment of acne may actually result in more risk of psychological harm.

Suicide and Isotretinoin

The critical question of whether or not the use of isotretinoin increases the risk of depression and suicidal ideation in individuals with acne, over and above the risk due to acne itself, has not been resolved. Various observational studies have yielded conflicting results (Wysowski et al. 2001; Bremner et al. 2012; Jick et al. 2000; Friedman et al. 2006; Azoulay et al. 2008b).

A study concluded that there was an increased risk of suicide attempts up to 6 months after the end of treatment with isotretinoin, but patients with a history of suicide attempts before treatment made fewer new attempts of suicide (Sundström

et al. 2010). This emphasised that patients with severe acne with a history of attempted suicide should not automatically be refused isotretinoin treatment. The authors also state that suicide risk was already rising prior to treatment and that the additional risk cannot, therefore, be attributed to isotretinoin use.

Subsequent studies (Singer et al. 2019) have demonstrated a lower risk of suicide attempt compared to the normal general population. There will, however, be a small subset of patients who have increased depression and suicidal thoughts while on isotretinoin. With this in mind, it is probably prudent to recommend closer monitoring of all patients with acne, who are identified as at high risk of depression.

References

Azoulay L, Blais L, Koren G, LeLorier J, Bérard A. Isotretinoin and the risk of depression in patients with acne vulgaris: a case-crossover study. J Clin Psychiatry. 2008a;69(4):526–32.

Azoulay L, Blais L, Koren G, et al. Isotretinoin and the risk of depression in patients with acne vulgaris: a case-crossover study. J Clin Psychiatry. 2008b;69:526–32.

Bremner JD, Shearer KD, McCaffery PJ. Retinoic acid and affective disorders: the evidence for an association. J Clin Psychiatry. 2012;73(1):37–50.

Dunn LK, O'Neill JL, Feldman SR. Acne in adolescents: quality of life, self-esteem, mood, and psychological disorders. Dermatol Online J. 2011;17:1.

Flessner CA, Woods DW. Phenomenological characteristics, social problems, and the economic impact associated with chronic skin picking. Behav Modif. 2006;30(6):944–63.

Friedman T, Wohl Y, Knobler HY, et al. Increased use of mental health services related to isotretinoin treatment: a 5-year analysis. Eur Neuropsychopharmacol. 2006;16:413–6.

Gnanaraj P, Karthikeyan S, Narasimhan M, Rajagopalan V. Decrease in "Hamilton rating scale for depression" following isotretinoin therapy in acne: an open-label prospective study. Indian J Dermatol. 2015;60(5):461–4.

Goulden V, Stables GI, Cunliffe WJ. Prevalence of facial acne in adults. J Am Acad Dermatol. 1999;41:577–80.

Grant JE, Odlaug BL, Chamberlain SR, Keuthen NJ, Lochner C, Stein DJ. Skin picking disorder. Am J Psychiatry. 2012;169(11):1143–9.

Gupta MA, Gupta AK. Depression and suicidal ideation in dermatology patients with acne, alopecia areata, atopic dermatitis and psoriasis. Br J Dermatol. 1998;139(5):846–50.

Hahm BJ, Min SU, Yoon MY, et al. Changes of psychiatric parameters and their relationships by oral isotretinoin in acne patients. J Dermatol. 2009;36(5):255–61.

Hazen PG, Carney JF, Walker AE, Stewart JJ. Depression–a side effect of 13-cis-retinoic acid therapy. J Am Acad Dermatol. 1983;9(2):278–9.

Huang YC, Cheng YC. Isotretinoin treatment for acne and risk of depression: a systematic review and meta-analysis. J Am Acad Dermatol. 2017;76(6):1068–1076.e9.

Jick SS, Kremers HM, Vasilakis-Scaramozza C. Isotretinoin use and risk of depression, psychotic symptoms, suicide, and attempted suicide. Arch Dermatol. 2000;136:123.

Kaymak Y, Kalay M, Ilter N, Taner E. Incidence of depression related to isotretinoin treatment in 100 acne vulgaris patients. Psychol Rep. 2006;99(3):897–906.

Kellett SC, Gawkrodger DJ. The psychological and emotional impact of acne and the effect of treatment with isotretinoin. Br J Dermatol. 1999;140(2):273–82.

Klassen AF, Newton JN, Mallon E. Measuring quality of life in people referred for specialist care of acne: comparing generic and disease-specific measures. J Am Acad Dermatol. 2000;43:229–33.

Koo J. The psychosocial impact of 2 Koo J. the psychosocial impact of acne: patients' perceptions. J Am Acad Dermatol. 1995;32:S26–30.

Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13.
Kroenke K, Spitzer RL, Williams JB. The patient health questionnaire-2: validity of a two-item depression screener. Med Care. 2003;41:1284–92.
Lasek RJ, Chren MM. Acne vulgaris and the quality of life of adult dermatology patients. Arch Dermatol. 1998;134(4):454–8.
Law PM, Chuh AAT, Lee A. Acne: its psychological consequences and management. HK Pract. 2006;28:1–5.
Li C, Chen J, Wang W, Ai M, Zhang Q, Kuang L. Use of isotretinoin and risk of depression in patients with acne: a systematic review and meta-analysis. BMJ Open. 2019;9:e021549.
Lochner C, Grant JE, Odlaug BL, Stein DJ. DSM-5 field survey: skin picking disorder. Ann Clin Psychiatry. 2012;24(4):300–4.
Mallon E, Newton JN, Klassen A, Stewart-Brown SL, Ryan TJ, Finlay AY. The quality of life in acne: a comparison with general medical conditions using generic questionnaires. Br J Dermatol. 1999;140(4):672–6.
Marqueling AL, Zane LT. Depression and suicidal behavior in acne patients treated with isotretinoin: a systematic review. Semin Cutan Med Surg. 2007;26(4):210–20.
Marron SE, Miranda-Sivelo A, Tomas-Aragones L, Rodriguez-Cerdeira C, Tribo-Boixaro MJ, Garcia-Bustinduy M, Gracia-Cazaña T, Ros-Abarca S, Roe-Crespo E, Diaz-Díaz RM, Brufau-Redondo C, Martinez-Gonzalez MC, Guerra-Tapia A, González-Guerra E, Puig L. Body dysmorphic disorder in patients with acne: a multicentre study. J Eur Acad Dermatol Venereol. 2020;34(2):370–6.
Martin AR, Lookingbill DP, Botek A, Light J, Thiboutot D, Girman CJ. Health-related quality of life among patients with facial acne—assessment of a new acne-specific questionnaire. Clin a Exp Dermato. 2001;26(5):380–5.
Motley RJ, Finlay AY. Practical use of a disability index in the routine management of acne. Clin Exp Dermatol. 1992;17:1–3.
Mulder MM, Sigurdsson V, Van Zuuren EJ, et al. Psychosocial impact of acne vulgaris. Evaluation of the relation between a change in clinical acne severity and psychosocial state. Dermatology. 2001;203:124–30.
Myhill JE, Leichtman SR, Burnett JW. Self-esteem and social assertiveness in patients receiving isotretinoin treatment for cystic acne. Cutis. 1988;41:171–3.
Odlaug BL, Grant JE. Clinical characteristics and medical complications of pathologic skin picking. Gen Hosp Psychiatry. 2008;30(1):61–6.
Odlaug BL, Grant JE. Phenomenology and epidemiology of pathological skin picking. In: Grant JE, Potenza MN, editors. The Oxford library of psychology: Oxford handbook of impulse control disorders. New York: Oxford University Press; 2011. p. 186–95.
Phillips KA. Body dysmorphic disorder: recognizing and treating imagined ugliness. World Psychiatry. 2003;3(1):12–7.
Picardi A, Abeni D, Melchi CF, et al. Psychiatric morbidity in dermatological outpatients: an issue to be recognized. Br J Dermatol. 2000;143:983–91.
Ritvo E, Del Rosso JQ, Stillman MA, La Riche C. Psychosocial judgements and perceptions of adolescents with acne vulgaris: a blinded, controlled comparison of adult and peer evaluations. Biopsychosoc Med. 2011;5:11.
Singer S, Tkachenko E, Sharma P, Barbieri J, Mostaghimi A. Psychiatric adverse events in patients taking isotretinoin as reported in a food and drug administration database from 1997 to 2017. JAMA Dermatol. 2019;155(10):1162–6.
Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166(10):1092–7.
Stein DJ, Hollander E. Dermatology and conditions related to obsessive-compulsive disorder. J Am Acad Dermatol. 1992;126:237–42.
Stein DJ, Lochner C. Obsessive-compulsive and related disorders. In: Sadock BJ, Sadock VA, Ruiz P, editors. Comprehensive textbook of psychiatry. Philadelphia, PA: Wolters Kluwer; 2017.

Strahan JE, Raimer S. Isotretinoin and the controversy of psychiatric adverse effects. Int J Dermatol. 2006;45(7):789–99.
Sundström A, Alfredsson L, Sjölin-Forsberg G, et al. Association of suicide attempts with acne and treatment with isotretinoin: retrospective Swedish cohort study. BMJ. 2010;341:c5812.
Tan JK. Psychosocial impact of acne vulgaris: evaluating the evidence. Skin Therapy Lett. 2004;29:1–3.
Tan J, Bhate K. A global perspective on the epidemiology of acne. Br J Dermatol. 2015;172:3–12.
Tanghetti EA, Kawata AK, Daniels SR, et al. Understanding the burden of adult female acne. J Clin Aesthet Dermatol. 2014;7:22–30.
Wysowski DK, Pitts M, Beitz J. An analysis of reports of depres- Sion and suicide in patients treated with isotretinoin. J Am Acad Dermatol. 2001;45:515–9.
Yang Y-C, et al. Female gender and acne disease are jointly and independently associated with the risk of major depression and suicide: a national population-based study. Biomed Res Int. 2014;2014:504279.
Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361–70.

Hair Disorders and Impact on Quality of Life

22

Maria-Angeliki Gkini and Victoria Joliffe

Introduction

Hair Loss

Alopecia, or hair loss, is defined as loss of hair from the scalp or other parts of the body. It is a common clinical complaint that constitutes a major source of distress for patients. Although the cause of hair loss in the majority of cases is easily diagnosed, sometimes diagnosis as well as treatment can be quite challenging.

The main subcategories of alopecia are:
(a) Non-scarring or non-cicatricial alopecias
(b) Scarring or cicatricial alopecias

In addition, many hair shaft disorders can produce hair shaft fragility, resulting in different patterns of hair loss.

The psychological impact of alopecia is significant. Hair is considered an essential part of overall identity: especially for women, for whom it often represents

M.-A. Gkini (✉)
Barts Health NHS Trust, London, UK
e-mail: margo.gkini@nhs.net

V. Joliffe
Barts Health NHS Trust, London, UK

Department of Dermatology, Queen Mary University London, London, UK

Department of Dermatology, Royal London Hospital, London, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_22

femininity and attractiveness. Men typically associate a full head of hair with youth and vigour. Therefore, patients with alopecia can present with low self-esteem and poor self-image, feelings of isolation, anxiety, depression, or even suicidal ideation. Psychological issues are more severe at the onset of symptoms and early recognition and management through a holistic approach is crucial.

Alopecia is defined as loss of hair from the scalp or the body. Hair loss has a significant psychological impact on patients, who may present with low self-esteem, poor self-image, depression, or even suicidal ideation. A holistic approach is crucial for the management of the hair disorder itself as well as the psychosocial co-morbidities.

Practice Point
Always ask your patients how they feel about their hair disease and its impact on their quality of life. Always ask for a potential triggering stressful life event during clinical history. At the moment, there is no standardised questionnaire to evaluate Quality of Life (QoL) in hair diseases.

Excess Hair and Hair in the Wrong Place

Other hair disorders include hirsutism and hypertrichosis. Hypertrichosis is excessive hair growth over and above the normal for the age, sex and race of an individual, in contrast to hirsutism, which is excess hair growth in women following a male distribution pattern. Hypertrichosis can develop all over the body or can be isolated to small patches and it can be congenital (present at birth) or acquired (arises later in life).

Classification and Aetiology

The main clinical classification of alopecias is based on the presence or loss of hair follicles and includes: (a) cicatricial and (b) non-cicatricial alopecias (Table 22.1). Another clinical classification is based on the extent of the disease and includes: (a) localised or focal and (b) generalised alopecias (Figs. 22.1a and 22.1b).

From the psychodermatology point of view, an updated classification of alopecias can be suggested based on the presence of primary or secondary psychosocial co-morbidities, and includes: (a) hair diseases that cause secondary psychosocial co-morbidities and (b) psychological and psychiatric diseases that can affect the scalp and hair (secondary alopecia) (Table 22.2).

Table 22.1 Commonest types of scarring and non-scarring alopecias

Commonest types of alopecia	
Non-scarring	Scarring
Androgenetic alopecia (AGA) Pattern hair loss (PHL)	Discoid Lupus erythematosus (DLE)
Alopecia areata (AA)	Lichen planopilaris (LPP)
Trichotillomania	Frontal fibrosing alopecia (FFA)
Telogen effluvium	Folliculitis decalvans
Anagen effluvium-chemotherapy induced	Dissecting cellulitis of the scalp
Traumatic	Central centrifugal cicatricial alopecia (CCCA)
Syphilitic	Chronic alopecia areata (AA)
Tinea capitis	Chronic androgenetic alopecia (PHL)
Drug-induced	Pseudopelade of Brocq
	Morphea
	Alopecia mucinosa
	Alopecia neoplastica

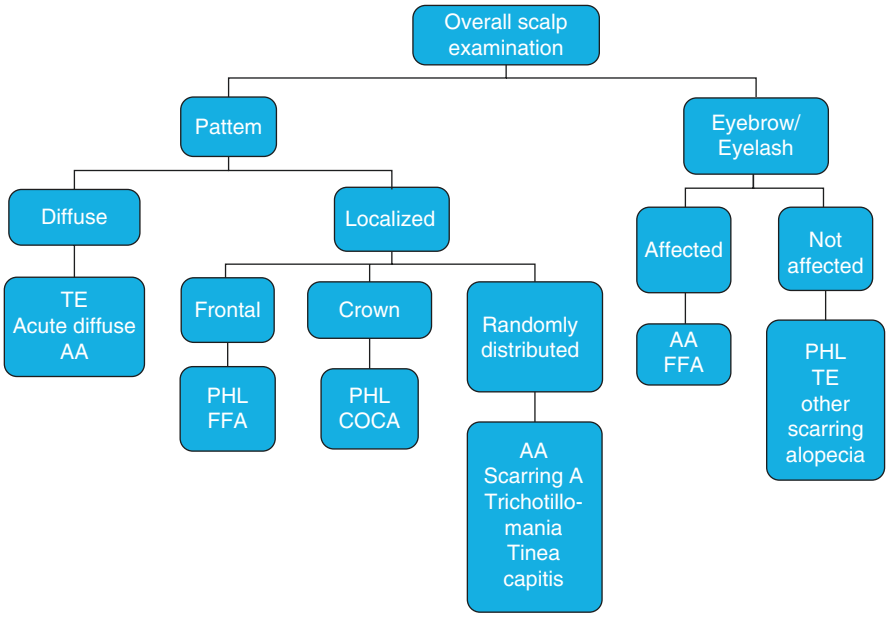


Fig. 22.1a Algorithmic approach on alopecias, based on pattern. Mubki T, Rudnicka L, Olszewska M, Shapiro J. Evaluation and diagnosis of the hair loss patient: part I. History and clinical examination. J Am Acad Dermatol. 2014 Sep;71(3):415.e1-415.e15. A, Alopecia; AA, alopecia areata; CCCA, central centrifugal cicatricial alopecia, scarring; DLE, discoid lupus erythematosus; FD, folliculitis decalvans; FFA, frontal fibrosing alopecia; LPP, lichen planopilaris; PHL, patterned hair loss; PPB, pseudopelade of Brocq; TE, telogen effluvium

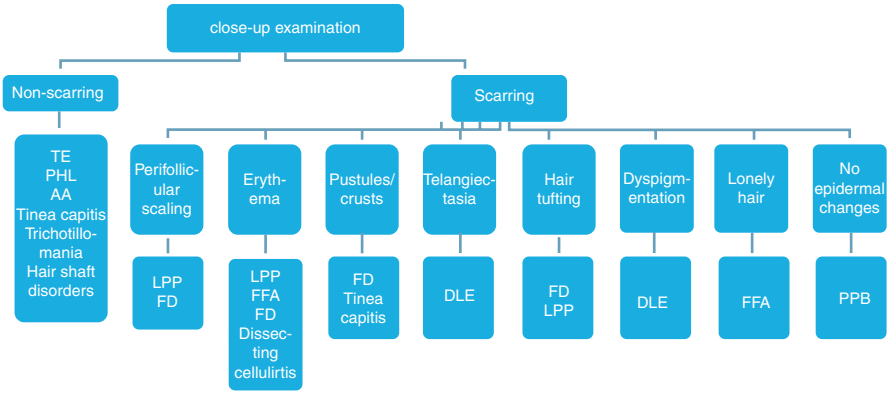


Fig. 22.1b Algorithmic approach on alopecias, based on the presence/absence of scarring. Mubki T, Rudnicka L, Olszewska M, Shapiro J. Evaluation and diagnosis of the hair loss patient: part I. History and clinical examination. J Am Acad Dermatol. 2014 Sep;71(3):415.e1-415.e15. A, Alopecia; AA, alopecia areata; CCCA, central centrifugal cicatrical alopecia, scarring; DLE, discoid lupus erythematosus; FD, folliculitis decalvans; FFA, frontal fibrosing alopecia; LPP, lichen planopilaris; PHL, patterned hair loss; PPB, pseudopelade of Brocq; TE, telogen effluvium

Table 22.2 Psychodermatological classification of alopecias

Dermatological hair disease with psychosocial co-morbidities	Psychological and psychiatric disease that may affect the scalp and hair
Alopecia areata	Trichotillomania
Androgenetic alopecia (males and females)	Body dysmorphic disorder
Telogen Effluvium	Post-traumatic stress disorder
Chemotherapy-induced anagen effluvium	Delusional infestation
Scarring alopecias	Depression
	Anxiety

Patho-aetiology

The pathophysiology of non-scarring alopecias is different depending on the type of alopecia. In alopecia areata, aetiology is unknown, but the most common hypothesis involves autoimmunity in the form of T cell-mediated pathway affecting the peribulbar area. In androgenetic alopecia, both genetic and hormonal androgens play a role in the pathogenesis. In telogen effluvium, the shedding of telogen hairs is under the influence of hormone or stress, but sometimes the trigger is not very clear. In tinea capitis, the dermatophytes infection is responsible for hair loss. In anagen effluvium, the shedding of anagen hairs is under the effect of chemotherapeutic agents.

Cicatricial (scarring) alopecia results from irreversible damage to epithelial stem cells located in the bulge region of the hair follicle, generally as a result of inflammatory mechanisms (e.g. in the context of autoimmune disease). This group of permanent hair loss disorders can be classified into distinct subgroups, characterised by the predominant perifollicular inflammatory cell type, such as lymphocytic, neutrophilic, mixed, or non-specific. Clinically, they are characterised by the loss of visible follicular ostia within the area of alopecia, and often accompanied by epidermal changes and signs of inflammation.

Alopecias can be classified based on: (a) the extent of the disease (localised and generalised) and (b) the presence/loss of follicular ostia (non-cicatricial and cicatricial). Other classifications are based on the duration of the lesions (congenital and acquired) or the cellular infiltrate for the cicatricial alopecias (lymphocytic, neutrophilic, mixed, non-specific).

From the psychodermatology point of view, alopecias can be classified as: (a) dermatological hair diseases with secondary psychosocial co-morbidities and (b) primary psychiatric or psychological diseases that may affect scalp and hair.

Clinical and Psychodermatological Approach on Patients with Hair Loss

Diagnostic algorithms (Figs. 22.1a and 22.1b) can be very useful in the clinical approach of alopecias. Clinical as well as trichoscopic features almost always provide us with a diagnosis and in few cases, a biopsy is needed.

Practice Point
Always make a diagnosis of the hair disorder, prior to treatment initiation. It is important to provide the patient with a management plan, explain the prognosis and manage their expectations.

Alopecias have a significant impact on patients' quality of life. Psychosocial co-morbidities are really common and it is crucial to spot them and offer a holistic approach to our patients. Stress and hair loss seem to part of a vicious circle, as stress can lead to hair loss and the opposite. Apart from stress, patients with hair disorders appear to have lower self-esteem, feel more isolated and develop more secondary psychiatric diseases, such as depression, compared to healthy sex- and age-matched controls. During the consultation it is important to ask direct and open

Table 22.3 Standardised screening questionnaires for assessment of depression, anxiety, suicidal ideation, body dysmorphic disorder and quality of life

Questionnaires	Condition
PHQ-9	9-item questionnaire for depression
GAD-7	7-item questionnaire for anxiety
HADS score	14-item questionnaire for depression and anxiety
Body dysmorphic disorder questionnaire	Body dysmorphic questionnaire
Short Form Health Survey (SF-36)	Self-assessment health questionnaire
DLQI	10-item questionnaire (not specific for hair disorders)
Alopecia Areata Quality of Life Index	Specific questionnaire for AA
Columbia Suicide-Severity Rating Scale	Assessment of suicidal ideation

questions, such as "Do you feel low because of your hair loss?" or "How do you feel about your hair loss?". In that way, patients feel that the physician is actually interested in their condition and they engage better, which results in a better therapeutic result and satisfied patients.

Standardised screening questionnaires are useful in assessing patients' mental state as well as their well-being (Table 22.3).

In the following paragraphs, we analyse in detail the main non-scarring and scarring forms of alopecia.

Practice Point
Ask your patients to fill in screening questionnaires, while they are in the waiting room. They feel that you are actually interested in them as a whole and you can have a very quick assessment of their mental health and well-being, even before seeing them.

Alopecia Areata

Alopecia areata (AA) is an autoimmune condition characterised by transient, non-scarring hair loss at its early stages. Scarring can occur in later stages. Alopecia areata affects nearly 2% of the general population at some point during their lifetime. It usually presents before the age of 20 years. Patients may have a personal or family history of other autoimmune diseases, such as Hashimoto's disease, diabetes type I, vitiligo. Hair loss can take many forms, ranging from loss in well-defined patches to diffuse or total hair loss, which can affect all hair-bearing sites and not only the scalp. Patchy alopecia affecting the scalp is the most common type, while 5% of patients with AA develop alopecia totalis. There are different forms of alopecia areata (Table 22.4). On trichoscopy, exclamation mark hairs may be present at the periphery of the patches, an indicator of active disease. Other key trichoscopic features of AA are yellow and/or black dots, broken hairs and short vellus hairs, which are a sign of early regrowth. In chronic cases of AA, follicular ostia may be lost.

Table 22.4 Types of alopecia areata (AA)

Types of alopecia areata (AA)	
Patchy AA	One or multiple separate or reticular patches of hair loss
Alopecia Totalis	Near-total or total hair loss affecting the scalp
Alopecia Universalis	Near-total or total hair loss on hair-bearing areas on body
Alopecia Incognita	Diffuse total hair loss and short miniaturised regrowing hair
Ophiasis	Hair loss in a band-like shape along the circumference of the head, more specifically along the border of the temporal and occipital bones.
Sisaipho	Extensive scalp alopecia, sparing its periphery
Marie Antoinette Syndrome	Acute episode of diffuse alopecia with very abrupt "overnight greying" of pigmented hair

Skin biopsies of AA, although rarely needed, show a lymphocytic infiltrate in and around the bulb or the lower part of the hair follicle in the anagen (hair growth) phase, like a swarm of bees. A breakdown of the immune privilege of the hair follicle is thought to be an important driver of AA. Genetic studies in patients and mouse models showed that alopecia areata is a complex, polygenic disease.

Psychodermatology in AA

It has long been postulated that psychological events may trigger episodes of AA in some individuals. The prototypic stress-associated neuropeptide, substance P, in organ culture models of human scalp hair, can induce loss of the hair follicle immune privilege, which is key in the pathogenesis of AA. Studies have shown that substance P is increased in early AA lesions as well as CD8 T cells that express the neurokinin-1 receptor, which mediates substance P action, supporting the "stress-related" theory. In case series there is usually a history of childhood and lifetime traumatic events. In many studies, stressful and life traumatic events (i.e. death of spouse, divorce, fire at work, etc.) may act as triggers that increase the incidence of disease episodes when the predisposing circumstances are present

However, in larger series, stress has been identified as a potential trigger in less than 10% of patients. Studies have emphasised the role of trait-anxiety, stress perception, and vulnerability as major factors of AA development. Therefore, personality characteristics, such as alexithymia and poor social support, may be of more importance in the development of AA in comparison to the frequency of stressful events.

Undoubtedly, AA has a significant impact on patients' feelings and well-being. The individual coping response to alopecia varies from the alopecia being an inconvenience that has little or no impact on leading a normal life, through to a life-changing experience that can have a devastating impact on psychological well-being with consequences that include clinically significant depression, loss of employment and social isolation. In a systematic review by Tucker et al., AA appeared to have a negative impact on self-esteem, and increased levels of depression, anxiety, phobic

reactions and paranoia. Females seem to be more commonly affected and the psychological impact is more severe. Age also plays a key role, as patients younger than 20 years old have more evidence of clinical depression compared to adults who present more with anxiety symptoms. Finally, in addition to medical treatments, (e.g. topical, intralesional or oral corticosteroids) in AA patients with depression, there may be a beneficial response of hair regrowth with the use of antidepressants.

Contributing factors to psychosocial disease include: (a) potential early onset of AA; (b) sudden onset of disease; (c) unpredictable course; (d) disease is profound and it is not easily "hidden"; (e) treatment options at the moment are limited; and (f) even positive response to treatment with hair regrowth can be followed by hair loss again.

For the more severe forms of AA, such as alopecia universalis and totalis, treatment armamentarium is limited, although Janus Kinase (JAK) inhibitors appear to be a promising option. The clinician has an important role in recognising the psychological impact of alopecia and in helping the patient to overcome and adapt to this issue. Some patients find camouflage extremely important and helpful. Some others will need professional support from a clinical psychologist or other practitioners skilled in managing disfigurement. Many benefit from contact with patient support organisations, for example the National Alopecia Areata Foundation (https://www.naaf.org/) and Alopecia UK (http://www.alopeciaonline.org.uk/). In children, alopecia areata can be particularly difficult to deal with. If a parent feels there is a considerable change in the needs of their child (withdrawn, low self-esteem, failing to achieve at school and/or change in behaviour) the child may need to be referred to a paediatric clinical psychologist, educational psychologist or social worker (https://www.naaf.org/alopecia-areata/living-with-alopecia-areata/alopecia-areata-in-children).

Alopecia areata (AA) is an autoimmune disorder causing a **significant impact on patients' quality of life. Psychiatric co-morbidities are very common** especially in more severe forms, such as alopecia totalis or universalis. There is an increasing need to recognise and treat them. Offer your patients all available treatment options for their hair, although limited, and encouraging them to take part in trials for new drugs and treat also the psychiatric concomitant disorders, offering a **holistic management plan.**

Practice Point
Apart from treatment for the AA and the psychosocial co-morbidities, offer your patients camouflage options as well as encouraging them to join patient support organisations.

Androgenetic Alopecia—Pattern Hair Loss (Males and Females)

Androgenetic alopecia (AGA) is the most frequent form of alopecia in men and women. It is characterised by progressive hair thinning and hair loss, usually in a pattern distribution. The onset may be at any age following puberty and its prevalence increases with age. By the age of 70, 80% of Caucasian men and up to 40% of women will have been affected by AGA. As hair is an important feature of image both in Western and developing societies, hair loss can cause significant psychological distress.

Androgenetic alopecia is a genetically determined hair disorder characterised by the effects of dihydrotestosterone (DHT) in the androgen-related areas of the scalp. DHT plays a key role in shortening the anagen phase of the hair cycle, from a usual duration of 3–6 years to just weeks or months. It further contributes to the miniaturisation of the follicles which results in the progressive production of fewer and finer hairs. AGA can also affect female patients. Women with AGA often have excessive levels of androgens as well as a genetic predisposition. These women also tend to suffer from acne, irregular menses and excessive facial and body hair. These symptoms are suggestive of polycystic ovarian syndrome (PCOS), although the majority of women with PCOS do not experience hair loss. Less often, congenital adrenal hyperplasia may be responsible. Females that are losing their hair with age are more likely to present with female pattern hair loss, in which hormone tests are normal. The distribution is also different compared to men, suggesting that a different pathogenetic mechanism may be implicated.

In both males and females with androgenetic alopecia, the transition from thick, pigmented terminal hairs to thinner and finally shorter non-pigmented vellus hairs in the involved areas is gradual. As the androgenetic alopecia progresses, more hairs are in the telogen phase, while the anagen phase has been shortened. Hence, there is a noticeable hair shedding.

Women with androgenetic alopecia generally lose hair diffusely over the crown and there is not a definite bald area. The frontal hairline is often preserved in contrast to male patients, who develop a gradual frontotemporal recession. The Hamilton–Norwood scale is used to assess the level of AGA in men and the Ludwig scale or the newer Olsen scale (with the characteristic Christmas tree pattern) in women.

The diagnosis is straightforward in the majority of cases. Clinically, thinning of hair is observed in the androgen-related areas. The distribution has been described above for both sexes. On trichoscopy, a hair shaft diameter variability of more than 20% is diagnostic. Other trichoscopic features include the presence of a single hair in the follicle, unlike normal unaffected follicles which bear up to four terminal hairs, white dots, the peripilar sign, a honeycomb pigmented pattern over the bald areas and/or the presence of empty follicles.

Main treatment options include: (A) for men: (a) topical minoxidil (foam or lotion), (b) finasteride, (c) dutasteride (off label), (d) low-level laser, (e) platelet-rich plasma (PRP) (off label) and (f) hair transplantation; (B) for women: (a) topical

minoxidil, (b) spironolactone, cyproterone acetate, flutamide and cimetidine can block the action of dihydrotestosterone on the scalp (off label), (c) platelet-rich plasma (PRP) (off label), (d) finasteride and dutasteride (off label and not in women of childbearing potential), (e) hair transplantation in selected cases. Wigs and cosmetic camouflage can be also very helpful in disguising the areas of AGA.

Androgenetic alopecia (AGA) or pattern hair loss (PHL) is a genetically determined hair disorder affecting both men and women, with its pathogenesis being mainly attributed to the increased sensitivity of hair follicles to the action of dihydrotestosterone (DHT). It is characterised by progressive hair loss with a different distribution between men and women, causing a significant impact on patients' quality of life.

Psychodermatology in AGA/PHL

Studies have shown that bald men are perceived as being older, less attractive and less confident than men with normal hair. Balding can preoccupy the sufferer and cause stress, lower body image satisfaction, lower self-esteem and even lower sex-drive, affecting their relationships. These effects are more marked in younger men, single men and those with an earlier hair loss onset and larger extent. A negative impact on QoL is also more pronounced in young men, although not restricted to this age group. A significant contributing factor to distress is the fact that there is treatment effect only when medication is used. If discontinued, any hair that has been maintained or regrown as a result of the medication will be lost, resulting in a need for indefinite use. And studies have shown that balding men report frequent peer teasing about their condition. In a non-clinical sample, such teasing was reported by 45% of men with modest hair loss and by 79% of men with more extensive baldness. In a study of men seeking treatment for AGA, 60% reported being teased. Men who have a successful treatment experience also seem to experience an improvement in their self-esteem and their perception of personal attractiveness.

The psychosocial impact of hair loss can be more severe for women, since there is little understanding or acceptance of the condition. Women may also have issues of low self-esteem and feeling unattractive. While some men may cope well, as they can cut their hair short and this limits the impact of balding, for women it is much harder to cover the bald areas or the areas with thinner hair. Furthermore, it is much harder to apply topical treatments on longer hair.

Female pattern hair loss has been shown to have a negative effect on daily life, lowering self-esteem and causing social co-morbidities. Studies comparing the impact of hair loss on men and women have shown that women suffer more emotional distress and make significantly more efforts to cope with hair loss, which can be attributed to the fact that hair loss in women is not seen as a normal age-related process, as in men. The impact of hair loss is not uniform and it can be affected by

the severity, whether the hair loss is noticeable by the social environment or not, and by age (a younger age of onset has a greater psychological impact). Finally, women may be more prone to a secondary psychiatric disorder due to their hair loss, such as depression, anxiety and body dysmorphic disorder. Nevertheless, compared to men, it is more socially acceptable and easier for women to use forms of camouflage to disguise their hair loss.

Androgenetic alopecia (AGA) or pattern hair loss (PHL) can cause stress, lower self-esteem, lower body image satisfaction, depression, anxiety, body dysmorphic disorder and even suicidal ideation in rare cases. Women seem to be more severely affected by psychosocial co-morbidities compared to men. The progressive nature of the disease, its permanent result as well as the lack of effective lasting treatment contribute to the psychosocial co-morbidities of patients with AGA.

Telogen Effluvium

Practice Point
The majority of AGA treatments are not on prescription and off licence. Nevertheless, offer all the licensed and off-label treatments to your patients, as majority of them are happy to try them and they are not put off by the cost or the lack of evidence-based data. Offer also hair transplant, if needed, as well as camouflage options.

Telogen effluvium (TE) (Fig. 22.2) is a common hair disorder characterised by a disturbance of the hair cycle where hair follicles become synchronised and enter the regression (catagen) and resting phase (telogen) together, resulting in sudden significant hair loss. When hairs reach the end of the telogen phase (after 2–3 months), they enter into exogen and the old hair is lost before the follicle re-enters the anagen growth phase.

Common triggers of telogen effluvium are presented in Table 22.5. No cause is found in around a third of people diagnosed with telogen effluvium.

Stress seems to play a key role. A sudden increase in hair shedding, the hallmark of TE, can definitely be very distressing to the patients. However, the short duration of the disease as well as the full recovery are significant factors that make patients feel better. At the molecular level, stress hormones, such as catecholamines alter hair growth by amending the release of various neuropeptides. On the other hand, the hair follicle itself can generate an abundance of stress mediators and thus may directly be involved in the modulation of stress responses at the local level, possibly as part of a "skin stress system".

The diagnosis is based on clinical history and examination. Trichoscopy has limited diagnostic value for TE. Frequent—but not specific—trichoscopic findings for

Fig. 22.2 Telogen effluvium. Any hair loss can be very distressing

Table 22.5 Causes of telogen effluvium

Common causes of telogen effluvium	
Pregnancy	Stressful or major life event
Labour	Marked weight loss
Stressful event	Extreme dieting
Recent surgical operation	Skin disorders affecting the scalp
Severe illness, with fever	Medication (new or already being used)
Seasonal	Withdrawal of hormonal treatment (including the pill)

TE include empty hair follicles, a high percentage of follicular units with only one hair and brown perifollicular discoloration (the peripilar sign). Multiple upright regrowing hairs may be observed in the regrowth phase of TE. No significant differences are observed in the trichoscopic findings between the frontal and occipital

Telogen effluvium (TE) is a reversible hair disorder characterised by sudden and significant hair loss. The role of stress is dual. It can be a key factor in the pathogenesis of the disease but also a result of the disease. Stress hormones as well as neuropeptides are involved in the molecular level.

Practice Point
Reassure your patients about the temporary nature of the disease. Explain that they will experience full hair regrowth. In some cases, topical steroid lotions or multivitamins can boost hair regrowth.

areas and can be a useful clue to this difference between TE and FPHL. However, clinicians should be aware of the frequent coexistence of these two conditions.

Treatment includes reassurance to the patients and no further action. Some physicians may prescribe topical steroids of high potency to boost hair regrowth or vitamin and amino acid supplements.

Anagen Effluvium

Anagen effluvium (AE) occurs after any impairment in the mitotic or metabolic activity of the hair follicle. AE or chemotherapy-induced alopecia is caused by exposure to chemotherapeutic agents such as antimetabolites, alkylating agents and mitotic inhibitors used to treat cancer. Other causes of AE include radiation therapy, endocrine diseases, alopecia areata, cicatricial diseases, trauma or pressure, but also pemphigus vulgaris.

Clinically, hair shafts are characterised by tapered fractures. Damage to the matrix results in narrowing of the hair shaft, which finally results in shaft fractures in the area of the narrowing. AE is temporary with hair regrowth typically occurring after 3–6 months of stopping chemotherapy. In some cases, hair regrows despite chemotherapy treatment while in other cases there may be changes in the hair colour and/or texture after regrowth. The diagnosis is based on clinical history mainly. Trichoscopic findings include black dots, monilethrix-like hairs and exclamation mark hairs.

Drug-induced anagen effluvium can be psychologically devastating to the patient. Patients have even refused life-saving or palliative treatments because they could not accept the temporary or prolonged baldness. There are cases where patients felt that hair loss had been more traumatic than the surgical treatment of the cancer itself. AE may also cause negative body image, lowered self-esteem and even anxiety and depression. One of the main reasons for this is that the alopecia serves as a reminder of the cancer to both the patient and the social environment. But there are patients who look more on the bright side and consider the alopecia as a sign of effective treatment. As AE is an inevitable consequence of treatment, patients' concerns and expectations should be managed before the onset of treatment.

When treating AE, patients should be reassured that hair loss is temporary, although some more permanent changes in colour or texture can rarely occur. Topical minoxidil can shorten the duration of alopecia and can be used, although it is not effective in the prevention of AE. There is a lot of controversy about the use of cooling caps and scalp cooling. The application of a pressure cuff on the scalp, when chemotherapy is infused causes local hypothermia and delays the anagen arrest. Nevertheless, hypothermia causes vasoconstriction and reduced blood flow. Therefore, chemotherapy may not reach all the malignant cells that can be present on the scalp and the cooling cap should not be used in patients with leukaemia, lymphoma and other hematologic malignancies. Finally, no evidence-based data

Anagen effluvium (AE) is a reversible hair disorder that is mainly caused by chemotherapy treatment. Hair loss is temporary and full hair regrowth normally happens after the end of the treatment. Patients need reassurance and should be offered camouflage options, such as wigs. Minoxidil can potentially shorten the duration of alopecia.

Practice Point
Reassure your patients about the temporary nature of the disease. Explain that they will experience full hair regrowth. Do not suggest the use of cooling caps and explain to your patient the potential risks so they can take an informed decision.

exist about the different forms of cancer, chemotherapy and the use of cooling caps and its use cannot be suggested. Wigs can serve as a potential camouflage option.

Scarring Hair Loss

Scarring alopecias are characterised by the destruction of the hair follicle and replacement by scar tissue. Inflammatory processes can target the bulge area, where the hair stem cells are found, which leads to the inability for hair regrowth. Moreover, trauma to the scalp or non-scarring alopecias, due to their chronicity, can cause scarring hair loss, without the follicle being the "target". Scarring alopecias can be primary or secondary. The commonest scarring alopecias are presented in Table 22.1. Another classification is based on the type of cell infiltrate, such as neutrophilic, lymphocytic or mixed.

The key point in scarring alopecias is the permanence of the hair loss, which can be devastating for patients. And symptoms, such as pain, pruritus or burning sensation can cause further distress to the patient. Therefore, it is really crucial to spot scarring alopecias at their early stage and treat them accordingly in order to prevent scar formation and loss of hair follicles with often aggressive systemic treatment.

Systemic treatment varies depending on the underlying cause. Few large, randomised, blinded, controlled studies are available. Most treatments are considered off label and may include hydroxychloroquine, dutasteride, oral prednisolone, intralesional triamcinolone, a combination of antibiotics such as clindamycin and rifampicin, doxycycline and more. Although hair transplants theoretically can offer cosmetically acceptable correction, it is important to inform the patient that there is risk of koebnerisation of the disease. Furthermore, a hair transplant attempt should never be performed when there is active disease and inflammation. As scarring alopecias are relatively rare, the psychological impact has not been studied extensively. In a study with 23 patients, 74% of the patients with cicatricial alopecia had

Scarring alopecias are a spectrum of different hair disorders that lead to the destruction of the hair follicle and the development of scar tissue. Underlying cause varies and early detection and aggressive systemic treatment are crucial. The permanent nature of the condition has a significant impact on the psychosocial well-being of patients. Where possible, psychodermatological advice should be offered during the consultation.

moderate to severe psychosocial impact attributed to their hair loss. Younger patients and patients with inactive disease experienced a more severe psychosocial impact. So, patients with cicatricial alopecia should be offered a holistic approach and the psychosocial co-morbidities should be spotted and addressed.

Psychiatric Disorders That Can Present with Hair Loss

Trichotillomania

Trichotillomania (Fig. 22.3) or hair-pulling disorder is characterised by the persistent and excessive pulling of one's own hair, resulting in alopecia and excoriations. Trichotillomania has been classified as an obsessive-compulsive and related disorder, according to the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)*.

Hair pulling can occur in any area of the body where hair grows. The commonest area affected is the scalp, followed by eyelashes and eyebrows. In right-handed patients, the right side is often affected and accordingly the left area for left-handed. Trichotillomania is quite common in early adolescence. Trichotillomania most commonly presents in children and teenagers with the prevalence being higher between 4 and 17 years. Clinically, there can be areas with undetectable thinning of hair, or there may be frank bald patches. Excoriations, erosions and bleeding may be present. In the majority of patients, psychosocial co-morbidities exist. Patients may experience distress, impairment in social, academic and/or relationship functioning.

Although there is a lot of research on trichotillomania by psychiatrists, the majority of the patients present to dermatologists. Therefore, it is really important for dermatologists to be able to recognise and treat such patients holistically.

The diagnosis can be challenging. From a dermatological perspective, shaving a circumscribed area weekly (the "hair growth window") can contribute to diagnosis. Trichoscopy can be also very helpful. Trichoscopic features include black dots, coiled or hook hair, shafts of varying lengths with fraying or split ends (trichoptilosis), flame hair, v-sign, follicular haemorrhages and tulip hair. The absence of exclamation mark hairs and yellow dots are also suggestive of trichotillomania, apart from the macroscopic clinical picture with unilateral and focal distribution. From a

Fig. 22.3 Trichotillosis. Note hair shafts of different lengths. Trichotilloscopy will help

Table 22.6 DSM criteria for trichotillomania

DSM 5 Criteria for Trichotillomania
A. Recurrent pulling out of one's hair resulting in noticeable hair loss.
B. An increasing sense of tension immediately before pulling out the hair or when attempting to resist the behaviour.
C. Pleasure, gratification or relief when pulling out the hair.
D. The disturbance is not better accounted for by another mental disorder and is not due to a general medical condition (e.g. a dermatologic condition).
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

psychiatric perspective, diagnostic criteria as set by the DSM can be useful (Table 22.6). The majority of patients deny their habit and try to hide it. Parents may struggle to acknowledge and accept the situation. Patients and relatives often feel shame and stigma because of the act of hair pulling.

Table 22.7 Management of trichotillomania

Try to understand *why* this pattern has developed early in the course of the condition.
Offer cognitive-behavioural therapy early.
Offer habit reversal technique.
Treat the hair loss with physical measures where appropriate (weaves/appliances, etc.).
Offer *N*-acetylcysteine (NAC).
If *N*-acetylcysteine (NAC) and behavioural treatment are ineffective, consider SSRIs, if appropriate, and other psychotropic treatment via expert advice.
Refer to a psychodermatology clinic.

In terms of treatment (Table 22.7), there are no large double-blind controlled trials. The mainstay of treatment includes behavioural therapies as well as pharmacologic treatment. Flessner et al. have suggested cognitive-behavioural therapy (CBT) as the most widely used behavioural treatment for trichotillomania compared to Bloch et al. who have suggested that habit reversal training is the most effective intervention for trichotillomania. Other behavioural treatments include self-monitoring, competing reaction training, relaxation training, hypnosis and elimination of a comorbid behaviour.

Pharmacological medication can be effective. Recommendations suggest that *N*-acetylcysteine (NAC) should be considered in all cases of trichotillomania. It has demonstrated efficacy and a favourable adverse effect profile. Selective serotonin reuptake inhibitors (SSRIs) have demonstrated a degree of effectiveness in selected patients and it is a good option if psychiatric co-morbidities exist. Nevertheless, the UK Medicines and Healthcare Products Regulatory Agency (MHRA) has issued an advisory stating that most SSRIs should not be used in patients younger than 18 years old, as the risk:benefit ratio is high. Only child and adult mental health service specialists should prescribe SSRIs in the under 18 age group, so referral to such services will be needed if use of SSRIs is being considered in a patient under age 18. SSRIs may be a safer choice than tricyclic antidepressants (TCAs) with less adverse events.

If hair pulling is associated with a specific activity, such as watching TV or studying, close monitoring by parents or partners is required.

Finally, joining a support group can be beneficial.

Delusional Infestation

Delusional infestation (DI) is defined as the perception held by the patient that they are infested with insects or inanimate particles, despite the contradicting evidence. Patients with DI often present to the hair clinic complaining of their scalp symptoms, such as hearing the mites on their scalp, or mites coming out of the hair root. Many patients bring hair material videos or photographs of the perceived infestation in an attempt to persuade the treating physicians (the specimen sign). In such cases, diagnosis is very easy from clinical history. A referral to a specialist psychodermatology clinic is suggested for a holistic approach to treatment.

Table 22.8 Screening questionnaire for BDD

Cosmetic procedure screening questionnaire (COPS) for Body Dysmorphic Disorder
1. How often do you **deliberately** check your feature(s)? **Not accidentally catch sight of it.** Please include looking at your feature in a mirror or other reflective surfaces like a shop window or looking at it directly or feeling it with your fingers.
2. To what extent do you feel your feature(s) are **currently** ugly, unattractive or "not right"?
3. To what extent does your feature(s) **currently** cause you a lot of distress?
4. How often does your feature(s) **currently** lead you to avoid situations or activities?
5. To what extent does your feature(s) **currently** preoccupy you? That is, you think about it a lot and it is hard to stop thinking about it?
6. If you have a partner, to what extent does your feature(s) currently have an effect on your relationship with an existing partner? (e.g. affectionate feelings, number of arguments, enjoying activities together). If you do not have a partner, to what extent does your feature(s) currently have an effect on dating or developing a relationship?
7. To what extent does your feature(s) currently interfere with your ability to work or study, or your role as a homemaker? (Please rate this even if you are not working or studying: we are interested in your ability to work or study.)
8. To what extent does your feature(s) currently interfere with your social life? (with other people, e.g. parties, pubs, clubs, outings, visits, home entertainment).
9. To what extent do you feel your appearance is the most important aspect of who you are?

Table 22.9 Practical guideline for management of BDD in the dermatology clinic

Practical guideline for management of BDD
1. Screen and assess for BDD.
2. If you believe the patient has BDD following screening and assessment, share the diagnosis with them.
3. Advise referral to a mental health practitioner for a full assessment and further advice.
4. Explain the rationale for referral to a mental health practitioner in terms of the significant risk of dissatisfaction with the procedure, and the likelihood that preoccupation and distress will remain or move to another body area.
5. Avoid arguing with the patient about whether his/her perceived "defect" is real or imagined.
6. Express lots of empathy about his/her distress and preoccupation.
7. Do not refer for a second opinion from another dermatologist; instead, discuss with a mental health practitioner and refer for a psychological or psychiatric assessment.
8. Try and liaise with the patient and mental health professional to allow dermatological procedures to be completed prior to starting psychological therapy, or to postpone them until after psychological therapy or after a trial of SSRI medication.
9. Consider co-working with a mental health professional, especially if there is damage from skin picking or if it helps to increase engagement.

Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) or body dysmorphia is considered more fully elsewhere in this volume. Patients with BDD often focus on the appearance of their hair. They may believe that they are shedding more hair and alopecia is inevitable. A diagnosis can be challenging. It is based on clinical history and screening questionnaires can provide additional help (Table 22.8).

Treatment can be challenging as the patients may lack insight and can be difficult to engage. In Table 22.9, there is a practical guideline approach for the management

Fig. 22.4 Scaling from scalp disease (e.g. psoriasis) can be very troubling, and is easily underestimated, for men and women. Scalp scaling on black clothes is very obvious

of BDD in the dermatology clinic. If concerned, you can refer to a psychodermatology clinic for a holistic approach.

Scalp Disease

Scalp disease (Fig. 22.4) can lead to scaling and redness of the scalp which in itself can be very debilitating. Underestimating the impact of scalp disease on hair styles/colour and choice of clothing is very common amongst health care professionals, and must be avoided. As usual in the management of patients with psychodermatological disease, it is important to manage the scalp disease (e.g. psoriasis, atopic dermatitis) at the same time as managing the psychosocial co-morbidities.

Appliances/Wigs, Camouflage and Support Groups

Wigs or hairpieces are commonly used to camouflage alopecia areas, mainly by women. Patients who choose to use a wig for hair loss seem to have a better quality of life and self-esteem, with less prevalence of depression and anxiety. Wigs are

Practice Points

- Hair loss is accompanied by significant psychosocial co-morbidities.
- Women and young patients are generally more affected, without excluding other categories of patients.
- It is crucial and it should be part of the routine clinical practice to screen patients with hair loss for depression, anxiety and body dysmorphic disorder.
- Patients should get a holistic approach to their hair problem, and not only dermatology treatments
- Camouflage can be extremely beneficial for a subset of patients.
- If unsure, offer to the patient a referral to a psychodermatology clinic.

very common among patients with AA, AGA and AE. Other options may include keratin filaments, tattoo and more.

Psychological support by experts as well as joining support groups can be beneficial.

Support groups include:

- National Alopecia Areata Foundation (http://www.naaf.org)
- Alopecia UK (http://www.alopeciaonline.org.uk)
- Cicatricial Alopecia Research

Bibliography

Bewley A, Taylor R, Reichenberg J, Magrid M, editors. Practical psychodermatology. 1st ed. London: Wiley Blackwall; 2014.

Hesketh P, et al. Chemotherapy induced alopecia: psychological impact and therapeutic approaches. Support Cancer Care. 2004;12:543–9.

Montgomery K, White C, Thompson A. A mixed methods survey of social anxiety, anxiety, depression and wig use in alopecia. BMJ Open. 2017;7(4):e015468.

Mubki T, Rudnicka L, Olszewska M, Shapiro J. Evaluation and diagnosis of the hair loss patient: part I. History and clinical examination. J Am Acad Dermatol. 2014a Sep;71(3):415.e1–415.e15.

Mubki T, Rudnicka L, Olszewska M, Shapiro J. Evaluation and diagnosis of the hair loss patient: part II. Trichoscopic and laboratory evaluations. J Am Acad Dermatol. 2014b Sep;71(3):431.e1–431.e11.

Rencz F, Gulacsi L, Pentek M, et al. Alopecia areata and health-related quality of life: a systematic review and meta-analysis. British J Dermatol. 2016;175(3):561–71.

Rudnicka L, Olszewska M, Rakowska A, editors. Atlas of trichoscopy. Dermoscopy in hair and scalp disease. London: Springer; 2012.

Psychogenic Pruritus

23

Ahmed Kazmi and Tabi Leslie

Definition

Psychogenic pruritus (also known as *psychogenic itch*, *somatoform pruritus*, *functional itch disorder*, *non-organic pruritus*, *psychosomatic pruritus* and *itch disorder associated with psychological factors*) will be an entity already well known to the reader. For some, it may be a 'heart-sink' diagnosis and consultation, and whilst we recognise, as with much of psychodermatology, psychogenic pruritus may be both a diagnostic and interpersonal challenge, it need not be a cause of apprehension for the treating physician.

The common definition of psychogenic pruritus is generally taken to mean itch which is not attributable to a dermatological or systemic cause. A more holistic definition would probably be that of the French psychodermatology group (FPDG), where psychogenic pruritus is described as 'an itch disorder where itch is at the centre of the symptomatology and where psychological factors play an evident role in the triggering, intensity, aggravation or persistence of the pruritus' (Misery et al. 2007).

Although elementary for some, it is crucial to be able to define 'itch', itch being an unpleasant sensation in the skin leading to the desire to scratch (Misery and Stander 2010). Other sensations than itch, can lead to the desire to scratch including paraesthesia, burning and pain—it is worth clarifying what exactly the patient means by 'itch', as the nature of the sensation may offer clues to the cause (Table 23.1).

It is also useful to think of itch in terms of the pathways involved (Potenzieri and Undem 2012). Not all itch is histamine mediated therefore antihistamines are not a universal strategy for pruritus management. Pharmacological treatment of the

A. Kazmi · T. Leslie (✉)
Royal London Hospital, London, UK
e-mail: ahmed.kazmi@nhs.net; tabi.leslie@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_23

Table 23.1 Itch characteristics and example diagnoses

Generalised itch	Eczema Xerosis Prebullous bullous pemphigoid Scabies Systemic itch, e.g. uraemia, bilirubinaemia, malignancy, iron deficiency, thyroid dysfunction Psychogenic pruritus
Facial itch	Eczema Seborrheic dermatitis Allergic contact dermatitis Chronic actinic dermatitis Systemic lupus erythematosus Psychogenic pruritus
Facial tingling/burning/electric shock	Herpes Zoster (VZV) Herpes Labialis (HSV) Post-herpetic neuralgia Trigeminal neuralgia Trigeminal trophic syndrome Atypical face pain Psychogenic pruritus
Tingling/Burning/electric shock in specific body region or dermatome	Erythromelalgia Peripheral neuropathy Radiculopathy, e.g. sciatica, meralgia paraesthetica, post-herpetic neuralgia, carpal tunnel syndrome, notalgia paraesthetica Conversion disorder Dysesthesia syndromes, e.g. vulvodynia, penodynia, scrotodynia and burning scrotum syndrome

patient is based on the suspected pathway involved. In reality, a mixture of approaches may be required, and the approach is often empirical.

Classification

IFSI proposes six aetiological categories of itch (Stander et al. 2007). These categories are very useful in approaching differential diagnoses for the itchy patient and rationalising subsequent therapy (Table 23.2).

Psychiatric classifications of psychogenic pruritus are less clear. Psychogenic pruritus is not mentioned specifically in the ICD10, pruritus, however, is mentioned along with several other conditions including dysphagia and bruxism. In this system, it is categorised as a *somatoform disorder* that falls within the broader group of 'neurotic disorders, stress-linked disorders and somatoform disorders' (World Health Organisation 2004). DSM IV does not specifically categorise or reference psychogenic pruritus either. In this system, it can be classified similarly as an 'undifferentiated somatoform disorder' or a conversion disorder (American Psychiatric Association 1994). DSM-5 has phased out the term somatoform disorder and

substituted it with 'somatic symptom disorders' (SSD) (American Psychiatric Association 2013). This term includes any somatic symptoms which are very distressing for the patient or cause significant functional impairment. In diagnosing an SSD, it is irrelevant whether the symptoms are medically explained or not.

Pathophysiology

The pathophysiology of itch is complex (Fig. 23.1). On a conceptual level, it is very helpful to think of itch either due to dermatological/systemic causes, psychogenic causes, or a mixture thereof (Buteau and Reichenberg 2018).

Table 23.2 IFSI categories of itch with examples

Dermatological	Eczema Psoriasis Lichen planus Scabies Insect bites Urticaria
Systemic	Chronic renal failure Hepatic disease Cholestasis HIV Hep C Iron deficiency Polycythaemia vera Thyroid dysfunction Lymphoma Solid organ tumours Drug induced, e.g. opiates, amphetamines
Neurological	Shingles Small fibre neuropathies Radiculopathies (notalgia paraesthetica, brachioradial pruritus) Trigeminal nerve lesion
Psychogenic	Psychogenic pruritus Delusional infestation
Mixed	Overlapping causes, e.g. eczema with significant depression/anxiety
Other	Undetermined cause

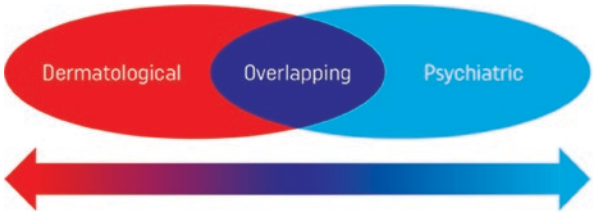


Fig. 23.1 Pathophysiology of itch is complex

Following on from this, psychogenic pruritus may be divided into three categories:

1. A primary dermatologic pruritic disease (e.g. urticaria and eczema) developing psychiatric sequelae.
2. A skin disease where the pruritic symptoms are exacerbated by stress.
3. A psychiatric disorder leading to the sensation of itch.

The best explanation of a totally psychogenic itch would be the 'contagious' itch one experiences when someone hears a story about head lice or scabies—here there has been no external activation or stimulus to skin and no systemic or dermatological cause, yet itch is experienced and scratching usually follows.

Scratching induces neuronal hyperplasia in the dermis, which further magnifies and perpetuates pruritus (Ikoma et al. 2006). Chronic and increasing scratching is common and likely a result of peripheral and central nerve sensitisation to itch. It is also well recognised that secondary changes induced in the skin from scratching such as lichenification and nodules, drive the sensation of itch.

Clinical Presentation

Psychogenic pruritus, as discussed so far, may exist as part of a spectrum, overlapping with itch of a dermatological or systemic cause, or existing solely as a manifestation of a psychological aetiology. As such, the clinical presentation can also vary. Some patients will present with no positive dermatological signs at all. Others may show evidence of sequelae of scratching or manipulating the skin-excoriations, scars, nodules, pigmentary changes, but without evidence of a primary dermopathy. Some may show evidence of current or previous dermatoses such as eczema, psoriasis, seborrheic dermatitis, but report a degree of itch which is disproportionate to what is expected or usually encountered.

It is worth noting if skin changes (Figs. 23.2, 23.3, and 23.4) due to scratching exist, these are usually in areas most accessible to the hands (lateral arms, face, flanks) and the upper-middle back is usually spared (butterfly sign). Excoriations can also vary in appearance, particularly if the patient has been using an object to scratch (may not look linear), or if secondary bacterial infection has developed (may give a red, eroded, crusted appearance). Ethnic skin is also more likely to develop pigmentary change and lichenification at an earlier stage.

In this selection of common secondary skin changes we can see linear excoriations from scratching, but the background skin is essentially normal.

There may be a corresponding negative life event that coincides with the itch as well as a long history of repeated medical presentations and attempts to elucidate a

Fig. 23.2 A patient with nodular prurigo and clear preservation of the interscapular skin showing normal skin (Butterfly sign)

cause (multiple biopsies, seeing several specialties at several hospitals, private sector consultations). There is often a concomitant psychiatric disorder including depression, anxiety, obsessive-compulsive disorder (OCD), psychosis and/or substance misuse, the diagnosis of which may already be established, or is yet to be elucidated during the dermatology consultation (Buteau and Reichenberg 2018).

Practice Point
When identifying a primary psychiatric disease, it is important to first assess patients for psychiatric safety. Screening for alcohol or drug abuse in the workup of a new patient is advisable.

Fig. 23.3 Thickened skin with exaggerated skin markings from excessive rubbing and scratching, referred to as lichenification

Fig. 23.4 Multiple chronic nodules and erosions from scratching and picking. These are usually most concentrated on the lower legs and upper outer arms

Why Is Psychogenic Pruritus so Debilitating?

Pruritus can be a major cause of suffering. A large multicentre study in Europe demonstrated dermatoses associated with itch were associated with a much higher negative impact on quality of life than non-pruritic conditions (Dalgard et al. 2015). There is an established positive associated with itch and increased levels of depression and anxiety (Lee et al. 2017).

It is important to understand that these symptoms can cause profound distress to the patient, heighten or generate psychiatric morbidity and become an obstacle to social interactions and functioning. Just as scratching itself can cause neuronal hyperplasia thus propagating the itch, the subsequent depression and anxiety and loss of relationship or work can also perpetuate the itch and heighten the perception of itch. The presence of pruritus is also positively correlated with other increased somatic symptoms such as pain (Misery and Stander 2010).

Epidemiology

A study of 2540 general public participants in Germany found a point prevalence of chronic pruritus of 13.5%, a 12-month prevalence of 16.4% and lifetime prevalence of 22.0% (Matterne et al. 2011). It is estimated that up to 70% of patients with no skin lesions have an underlying psychiatric disease (Ferm et al. 2010). A study focusing specifically on psychogenic pruritus, found that 6.5% of their general dermatology outpatient cohort carried a diagnosis of psychogenic pruritus (Weisshaar et al. 2012). This is in contrast to psychiatric inpatients, where studies have found rates of marked daily itching between 32 and 42%, as these patients may be experiencing psychogenic pruritus at the more severe end of the spectrum (Mazeh et al. 2008; Kretzmer et al. 2008).

Diagnostic Process

As with all aspects of medicine, a good history in this context is paramount. Give your patient ample opportunity to recount their narrative. Listen to cues about social stressors, flags to psychiatric problems and the impact the itch is having on the patient's life. It is also very useful to establish the patient's own ideas about the cause of their itch and what their expectation is of you.

Ascertain with clarity and precision what the patient means by 'itch' and if there are any other associated symptoms. As referenced earlier features such as pain, allodynia, paraesthesia, hyperaesthesia, hypoesthesia and electric shock sensations may suggest a neuropathic cause.

In the patient's medical history, ask if there are other related problems such as IBS, fibromyalgia or other medically unexplained symptoms. Approaches to raising these sensitive issues are shown in Table 23.3. Always take a drug history to ascertain if they are on any prescription, over the counter or illicit drugs, which are associated with itch.

Use the first appointment as an opportunity to review what investigations and treatments have already been completed. Many patients come to you already having had multiple dermatological reviews including skin biopsies, scrapings and phototherapy. Duplication is rarely of benefit.

Table 23.3 Example questions to help explore itch

Please tell me a bit more about your symptoms
Can you describe the sensation to me?
Do you have any thoughts on what may be causing the itch?
Can you recall anything significant occurring at the start of the itch?
What impact are your symptoms having on you?
Do you think anything going on in your life might be aggravating your itch?
I find many patients may develop itch because of stress, or may become very low in mood due to the itch, do you think this may apply to you?

Perform a thorough examination of the skin looking for primary and secondary lesions, performing a mental state examination is paramount and you may find using depression and anxiety screening tools the easiest way of doing this. PHQ9, PHQ 15 and HADS are some commonly used, validated scales you may wish to integrate into your practice. A selection of scales is outlined in Table 23.4.

If there are any investigations you feel necessary to perform it is best to do these before giving the diagnosis of psychogenic pruritus, but it is preferable to plant the seed early, that not all itch arises from the skin. It is important to highlight to the patient at an early stage also, that just as itch is a sensation noted in the skin, it is processed and modulated by central processes and so treatments can include dermatological, neuromodulatory and psychiatric interventions.

It is important also to note that although we would wish to ensure no dermatologic or systemic cause for the pruritus is missed, psychogenic pruritus is not a diagnosis of exclusion or an idiopathic phenomenon. There are important positive features that will also aid diagnosis and seeing it as a specific entity will make it more likely to be diagnosed and treated effectively. The FPDG proposed criteria (Misery et al. 2007) are shown in Table 23.5.

Table 23.4 Assessment scales helpful in psychogenic pruritus

Impact on life	DLQI
Depression and anxiety scoring	PHQ-9 PHQ-15 HADS GAD-7 GDS (Geriatric Depression Scale) K10 Beck Depression Inventory DSSS (Depression and Somatic Symptoms Scale)
Itch scoring	5-D itch scale 12-Item pruritus severity scale Visual Analogue scale

Table 23.5 FPDG criteria for psychogenic pruritus

3 compulsory criteria • Localised or generalised pruritus without primary skin lesion • Duration of itch more than 6 weeks • No somatic cause
3/7 optional criteria • Chronological relationship between pruritus and one or several life events, which could have psychological repercussions • Variations in intensity with stress • Nocturnal variations • Predominance during rest or inaction • Associated psychological disorder • Pruritus that can be improved with psychotropic drugs • Pruritus that can be improved with psychotherapies

Treatment

Treatment needs to be sensitive and holistic. Basic skincare measures such as soap substitute use and emollient use will help reduce xerosis and promote a good skin barrier thus optimising skin health generally. There is also often an expectation from the patient, for the dermatologist to prescribe something topical and prescribing emollients often builds rapport and acts as a natural transition to discussing psychotropic medication. If the clinician has taken the time to explain the multifactorial cause of itch, it is much easier to explain the rationale for the inclusion of measures such as psychotropic mediation and psychological therapies in the management plan.

All patients are likely to benefit from relaxation strategies and stress reduction measures (Yosipovitch and Samuel 2008). Habit reversal therapy can also be useful, particularly if the individual has skin picking, excoriating or hair pulling in addition to scratching. Cognitive Behavioural Therapy (CBT) has been demonstrated to be useful in the context of pruritus with OCD (Schut et al. 2016) (Table 23.6).

Practice Point
Where patients are advised to seek CBT, it is important to tell them to obtain a therapist or psychiatrist who specifically performs CBT, since it is different from classical therapy.

Table 23.6 Non-pharmacological treatments for psychogenic pruritus

Bland emollients and soap substitutes
Emollients containing special antipruritic agents, e.g. menthol (1%), urea (5%-10%), glycerol (20%) and camphor (2%)
Topical crotamiton (10%)
Topical anaesthetics, e.g. pramoxine (1%) and polidocanol (2%-10%)
Topical capsaicin (0.025%-0.075%)
Topical tricyclics, e.g. doxepin cream (5%)
Lifestyle modification: Avoiding over-heating, avoiding irritating products or fabrics, addressing obvious social stressors which may be perpetuating itch
Relaxation techniques: meditation and yoga (self-directed or classes), relaxation apps, autogenic training
Wet wraps
Bathing—maximum 20 minutes, lukewarm and can use an emollient bath additive
Psychotherapeutic interventions: Mindfulness bases therapies, CBT, habit reversal therapy (self-directed with books, apps, online materials, or via therapist)
Educational training programmes for itch/scratch cycle
Phototherapy

Where depression, anxiety or OCD have been demonstrated, an SSRI or SSRI-like drug is indicated (Steinhoff et al. 2011). This can be augmented if necessary, with an antiepileptic or antipsychotic. Antiepileptics are particularly useful if there are also overlapping neuropathic features (Otto et al. 2019) and antipsychotics may be helpful where there are some features of delusional thinking such as infestations. You may find it helpful to remind patients that 'antipsychotics', like many medications, are also used for indications other than just mental health disorders. Literature has shown that antipsychotics can act directly on histaminergic mechanisms and this may be part of how they help with psychogenic pruritus (Gupta et al. 2018).

Table 23.7 summarises some of the most commonly used medications for psychogenic pruritus, including some dosing instructions and general considerations.

The choice of pharmacological options available can seem overwhelming, particularly if this drug group is not one you are most accustomed to prescribing. Although more data exists with the typical antipsychotic pimozidine, in the context of psychogenic pruritus, in view of the better side effect profile we would

Table 23.7 Common pharmacological measures in psychogenic pruritus

Doxepin	10 mg nocte up to 100 mg nocte	Dry mouth Constipation Sexual dysfunction Decreased seizure threshold	Antipruritic but also helps improve mood and sleep
Sertraline	50 mg mane up to 200 mg mane	Sexual dysfunction GI upset	Good for mixed anxiety/depression
Paroxetine	10 mg mane up to 40 mg mane	Sexual dysfunction GI upset	Helpful for depression, anxiety and especially OCD Taper off slowly
Mirtazapine	15 mg nocte up to 45 mg nocte	Dry mouth Weight gain	Helpful for depression and with sleep and appetite Avoid in closed angle glaucoma
Pimozidine	1 mg mane up to 6 mg mane	Extrapyramidal side effects Dyskinesias QT prolongation	Acts directly on histamine receptors Helps with psychotic symptoms and depression
Olanzapine	5 mg mane up to 10 mg mane	Weight gain Sedation	Acts directly on histamine receptors Helps with psychotic symptoms and depression
Aripiprazole	5 mg mane up to 25 mg mane	Insomnia Restlessness Akathisia	Acts directly on histamine receptors Helps with psychotic symptoms and depression
Gabapentin	100 mg tds up to 600 mg tds	Sedation	Good if coexisting depression, anxiety and OCD
Pregabalin	50 mg bd up to 150 mg bd	Sedation Ataxia Dizziness	Good if coexisting depression, anxiety and OCD

Table 23.8 Psychogenic pruritus pharmacological ladder

	First line	Second line	Third line
Psychogenic pruritus with anxiety/depression/OCD features	SSRI + psychological support (consider co-management with GP/psychologist/psychiatrist)	SSRI + either antipsychotic or antiepileptic	SSRI + antipsychotic + anti-epileptic + tertiary referral
Psychogenic pruritus with delusional or psychotic features	Antipsychotic + psychological support (consider co-management with a psychiatrist)	Antipsychotic + either SSRI or antiepileptic	SSRI + Antipsychotic + anti-epileptic + tertiary referral
Psychogenic pruritus with overlapping neuropathic/dysaesthesia features	Antiepileptic + psychological support (consider co-management with pain team/neurologist)	Antiepileptic + either antipsychotic or SSRI	SSRI + antipsychotic + anti-epileptic + tertiary referral

recommend first line use of an atypical antipsychotic such as aripiprazole is. Pregabalin is thought to be generally better tolerated and more efficacious than gabapentin (Table 23.8).

Practice Point
The general psychiatric context will inform the selection of psychopharmacologic drugs, as well as the psychiatric symptoms that are identified. It can be helpful to point out that some medications bind the histamine receptor, therefore putting focus on the patient's symptoms of itch.

The dermatologist is likely to need to manage the patient in association with other health care professionals. Mild-to-moderate psychiatric illness existing with the pruritus may require psychologist and GP input. Psychotic illness or severe depression or anxiety, particularly where significant suicidal ideation or intent is present, is likely to also require the expertise and review of a psychiatrist. When screening for depression, thoughts of harm to self or others should always be assessed and where positive, immediate advice from on call psychiatry colleagues (or law enforcement if applicable) should be sought. Which psychological and psychiatric services are available to you will depend greatly on local service provision but their importance should not be overlooked.

Reassure the patient that they are not responsible for their itch and it is not 'in their head' or being made up. Give a comprehensive treatment plan where the patient understands clearly the indication and rationale behind each component of the plan. Book a follow-up and set reasonable expectations of treatment, disease control may be possible rather than cure and a dermatological cause may never be found.

Figure 23.5 shows a proposed algorithm summarising the management of the itchy patient.

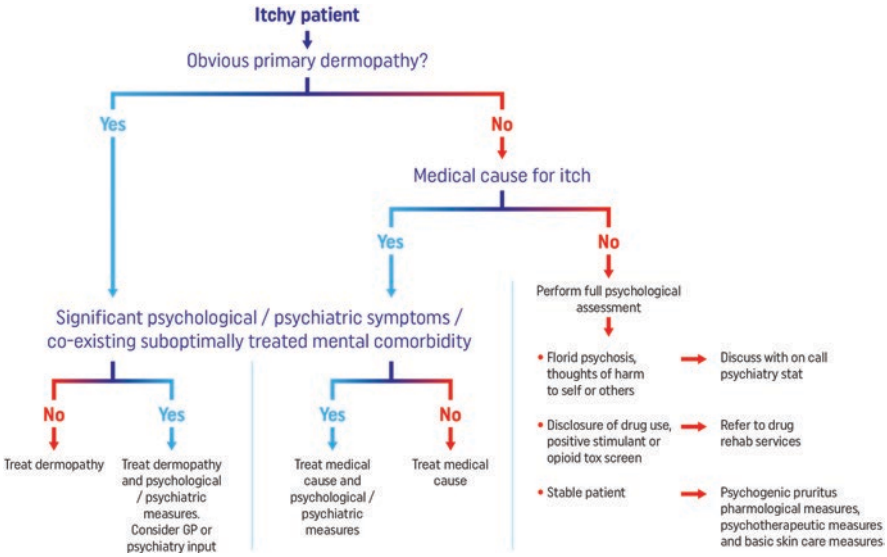


Fig. 23.5 Algorithm for the basic triage and management of itch

Some patients may never be able to accept a psychogenic cause for their pruritus or engage with suggested therapies, despite achieving good doctor–patient rapport and a sensitive and adequate explanation. In these cases, it may be appropriate for the dermatologist and patient to amicably part ways, with the offer of review in the future, should the patient wish to re-consider psychiatric therapies.

Practice Point
Normalizing the idea of psychiatric care is worthwhile, in cases where the patient will ideally obtain help from a psychologist or psychiatrist. Completion of a standard psychiatric screening tool may help provide assurance to the patient that referral is appropriate.

Prognosis

The authors were unable to find significant data on the prognosis of psychogenic pruritus. It is our experience, however, that with the use of psychotropic medication and psychological therapies, the patient experiences significant improvements in the symptom severity and impact of the complaint. Relapse is common.

Bibliography

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Arlington: American Psychiatric Publishing; 1994.
American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington: American Psychiatric Publishing; 2013.
Buteau A, Reichenberg J. Psychogenic pruritus and its management. Dermatol Clin. 2018;36:309–14.
Dalgard FJ, et al. The psychological burden of skin diseases: a cross-sectional multicentre study among dermatological out-patients in 13 European countries. J Invest Dermatol. 2015;135:984–91.
Ferm I, Sterner M, Wallengren J. Somatic and psychiatric comorbidity in patients with chronic pruritus. Acta Derm Venereol. 2010;90:395–400.
Gupta MA, Vujcic B, Pur DR, Gupta AK. Use of antipsychotic drugs in dermatology. Clin Dermatol. 2018;36(6):765–73.
Ikoma A, et al. The neurobiology of itch. Nat Rev. 2006;7:535–47.
Kretzmer GE, et al. Idiopathic pruritus in psychiatric inpatients: an explorative study. Gen Hosp Psychiatry. 2008;30(4):344.
Lee HG, et al. Psychiatric disorders and pruritus. Clin Dermatol. 2017;35:273–80.
Matterne U, et al. Prevalence, correlates and characteristics of chronic pruritus: a population-based cross-sectional study. Acta Derm Venereol. 2011;91(6):674–9.
Mazeh D, et al. Itching in the psychiatric ward. Aca Derm Venereol. 2008;88:128–31.
Misery L, Stander S. Pruritus. London: Springer; 2010.
Misery L, et al. Functional itch disorder or psychogenic pruritus: suggested diagnosis criteria from the French psychodermatology group. Acta Derm Venereol. 2007;87:341–4.
Otto J, Forstenpointner J, Binder A, Baron R. Pharmacotherapy of chronic neuropathic pain. Internist (Berl). 2019;60(7):711–23.
Potenzieri C, Undem B. Basic mechanisms of itch. Clin Exp Allergy. 2012;42(1):8–19.
Schut C, et al. Psychologic interventions in the treatment of chronic itch. Acta Derm Venereol. 2016;96:157–61.
Stander S, et al. Clinical classification of itch: a position paper of the international forum of the study of itch. Acta Derm Venereol. 2007;87:291–4.
Steinhoff M, et al. Pruritus management algorithms and experimental therapies. Semin Cutan Med Surg. 2011;30(2):127–37.
Weisshaar E, et al. European guidelines on chronic pruritus. Acta Derm Venerol. 2012;92:563–81.
World Health Organisation. International statistical classification of diseases and health related problems ICD-10. Geneva: World Health Organisation; 2004.
Yosipovitch G, Samuel L. Neuropathic and psychogenic itch. Dermatol Ther. 2008;21:32–41.

Part III

Concepts

Personality Disorders in Dermatology

24

Dmitry V. Romanov

Definitions

Personality disorder (PD) is defined as an extreme, maladaptive manifestation of individual traits presented as an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture. It is rigid, pervasive and inflexible, starts in adolescence or early adulthood, and leads to distress or impairment. PD tends to be more or less stable over life-time and mediates associated reactions (often stereotyped and maladaptive) on real-life circumstances. ICD-10 describes personality disorder as "a severe disturbance in the personality and behavioural tendencies of the individual, not directly resulting from disease, damage or other insult to the brain, or from another psychiatric disorder. They are nearly always associated with considerable personal distress and social disruption".

Historical names for PD:
(a) "Monomanie raisonnante" by Esquirol [1838]
(b) "Moral insanity" by Prichard [1837]
(c) ''Psychopathy" by Koch [1888]
(d) "Psychopathic personality" by Kraepelin [1904]
(e) "Accentuated Personality" by Leonhard [1972]

D. V. Romanov (✉)
Department of Psychiatry and Psychosomatics, I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia

Department of Boundary Conditions and Psychosomatic Disorders, Mental Health Research Centre, Moscow, Russia

Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_24

Classification

Overview

In ICD-10, PDs are coded as F60 (*Specific personality disorders*) and placed among F60-F69 *Disorders of adult personality and behaviour.* PDs are coded as 301 in DSM-5 and as 6D10 in ICD-11. Most classifications follow a categorical approach to PDs and are comprised of a set of specific subtypes. Comparison of PD subtypes in ICD-10 and in DSM-5 are listed in Table 24.1.

In DSM-5, PDs are subdivided into three clusters (Table 24.2). Cluster A (odd or eccentric) includes paranoid, schizoid, and schizotypal PDs. Cluster B (dramatic, emotional, or erratic) includes antisocial, borderline, histrionic, and narcissistic PDs. Cluster C (anxious or fearful) includes avoidant, dependent, and obsessive-compulsive PDs. The problem of such a categorical approach is that considerable overlap exists between the clusters and specific PDs.

The conceptualisation of PDs in ICD-11 is contrasting to ICD-10 and DSM as it departs from the traditional categorical approach and integrates dimensional and psychological ones based on a factor model of a general personality structure. First proposed, a PD model based on a factor analysis included six-factor dimensions: neuroticism, introversion, antagonism, conscientiousness, openness, and oddity. Later it was modified to include factors of negative emotionality, introversion, antagonism, compulsivity, disinhibition, and schizotypy. Then it was revised to a five-dimensional model: emotional dysregulation, detachment, antagonism, disinhibition, and peculiarity. In ICD-11, PDs are classified in accordance with a dimensional approach and primarily subdivided by severity: mild, moderate, or severe personality disorder to define the effect on functioning (Along with Personality

Table 24.1 PDs subtypes in ICD-10 and in DSM-5

ICD-10 PDs (F60)	DSM-5 PDs
Paranoid personality disorder (F60.0)	Paranoid personality disorder (301.0)
Schizoid personality disorder (F60.1)	Schizoid personality disorder (301.20)
Schizotypal disorder (F21) is attributed to F2 section *Schizophrenia, schizotypal, and delusional disorders*	Schizotypal personality disorder (301.22) is included in the PDs section
Dissocial personality disorder (F60.2)	Antisocial personality disorder (301.7)
Emotionally unstable personality disorder (F60.3) Impulsive type (.30) Borderline type (.31)	Borderline personality disorder (301.83)
Histrionic personality disorder (F60.4)	Histrionic personality disorder (301.50)
–	Narcissistic personality disorder (301.81)
Anankastic personality disorder (F60.5)	Obsessive-compulsive personality disorder (301.4)
Anxious [avoidant] personality disorder (F60.6)	Avoidant personality disorder (301.82)
Dependent personality disorder (F60.7)	Dependent personality disorder (301.6)

Table 24.2 Brief descriptions of PD subtypes in ICD-10 and DSM-5

PD subtype	Main pattern
Paranoid PD (PPD)*	A pattern of distrust and suspiciousness such that others' motives are interpreted as malevolent.
Schizoid PD (SPD)*	A pattern of detachment from social relationships and a restricted range of emotional expression.
Schizotypal PD (STPD)*	A pattern of acute discomfort in close relationships, cognitive or perceptual distortions, and eccentricities of behaviour.
Antisocial (ASPD) or dissocial PD**	A pattern of disregard for, and violation of, the rights of others.
Borderline PD (BPD)** or emotionally unstable PD, borderline type	A pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity.
Emotionally unstable PD, impulsive type	A pattern of impulsivity and tendency to act without consideration of the consequences.
Histrionic PD (HPD)**	A pattern of excessive emotionality and attention-seeking.
Narcissistic PD (NPD)**	A pattern of grandiosity, need for admiration, and lack of empathy. Arrogance is also considered as the central trait of NPD.
Avoidant PD (APD)***	A pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation.
Dependent PD (DPD)***	A pattern of submissive and clinging behaviour related to an excessive need to be taken care of.
Obsessive-compulsive PD (OCPD)*** or anankastic PD	A pattern of preoccupation with orderliness, perfectionism, and control.

Adapted from World Health Organization and American Psychiatric Association (*-DSM-5 cluster A; **- DSM-5 cluster B, *** - DSM-5 cluster C)

Disorders, in ICD-11 Personality Difficulty is a subclinical category.). This subdivision should be used with domains (factor analysis based) defined as prominent personality traits or patterns. The later describes the characteristics of the individual's personality that are most prominent, contribute to personality disturbance, and are continuous with normal personality characteristics in individuals who do not have Personality Disorder or Personality Difficulty. As a result, there are six prominent personality traits or patterns that may coincide in a given individual (Table 24.3).

As a result, PD description in an individual patient according to ICD-11 may look like this: *Severe Personality Disorder, Borderline Pattern, and prominent traits of Negative Affectivity, Disinhibition, and Dissociality;* or *Mild Personality Disorder, and prominent traits of Negative Affectivity and Detachment*.

Practice Point
The ICD-11 nomenclature for Personality Disorders focuses on the impairment of self and interpersonal personality functioning, which may be classified according to the degree of severity. It considers personality traits to be dimensional concepts, with personality disorder exemplifying the severe end.

Table 24.3 Prominent personality traits or patterns for ICD-11

Personality trait or pattern	Core manifestations (not all of which may be present in a given individual at a given time)
Negative affectivity	Tendency to experience a broad range of negative emotions with a frequency and intensity out of proportion to the situation; emotional lability and poor emotion regulation; negativistic attitudes; low self-esteem and self-confidence; and mistrustfulness.
Detachment	Tendency to maintain interpersonal distance (social detachment, i.e. avoidance of social interactions, lack of friendships, and avoidance of intimacy) and emotional distance (emotional detachment, i.e. reserve, aloofness, and limited emotional expression and experience).
Dissociality	Disregard for the rights and feelings of others, encompassing both self-centeredness (e.g. sense of entitlement, expectation of others' admiration, positive or negative attention-seeking behaviours, concern with one's own needs, desires and comfort and not those of others) and lack of empathy (i.e. indifference to whether one's actions inconvenience hurt others, which may include being deceptive, manipulative, and exploitative of others, being mean and physically aggressive, callousness in response to others' suffering, and ruthlessness in obtaining one's goals).
Disinhibition	Tendency to act rashly based on immediate external or internal stimuli (i.e. sensations, emotions, thoughts), without consideration of potential negative consequences. Main traits include impulsivity, distractibility, irresponsibility, recklessness, and lack of planning.
Anankastia	Narrow focus on one's rigid standard of perfection and of right and wrong, and on controlling one's own and others' behaviour and controlling situations to ensure conformity to these standards. Main traits include: perfectionism (e.g. concern with social rules, obligations, and norms of right and wrong, scrupulous attention to detail, rigid, systematic, day-to-day routines, hyper-scheduling and planfulness, emphasis on organisation, orderliness, and neatness), and emotional and behavioural constraint (e.g. rigid control over emotional expression, stubbornness and inflexibility, risk-avoidance, perseveration, and deliberativeness).
Borderline	Pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity. Main traits include: frantic efforts to avoid real or imagined abandonment; a pattern of unstable and intense interpersonal relationships; identity disturbance, manifested in markedly and persistently unstable self-image or sense of self; a tendency to act rashly in states of high negative affect, leading to potentially self-damaging behaviours (Figs. 24.1 and 24.2); recurrent episodes of self-harm; emotional instability due to marked reactivity of mood; chronic feelings of emptiness; inappropriate intense anger or difficulty controlling anger; transient dissociative symptoms or psychotic-like features in situations of high affective arousal.

Adapted from World Health Organization

For the purpose of this chapter, we focus on the terminology from ICD-10, as this is still most commonly in use in day-to-day practice at the time of publication (2021).

Aetiology and Pathology

Environmental Factors

There is substantial evidence of environmental factors that contribute to PDs. Most authors consider attachment problems to be a main aspect in the development of PD, which could be understood as the best possible adjustment to the environment at the time. It has been clearly shown that important risk factors for developing PD include childhood abuse (sexual, physical, or emotional), or neglect experienced over the course of early and childhood development (and some other social factors considered particular for specific PDs).

Genetic Factors

Like personality itself, PD is a result of the interaction of biological and psychosocial factors over the course of individual development. Genetic factors contribute significantly to an individual's personality. A role of heredity has also been proven for PDs in twin studies, particularly for personality traits associated with borderline PD, such as affective instability, identity problems, negative relationships, and self-harm. Twin studies have also supported heritability for narcissistic personality traits.

Biological Aspects

Some significant differences between PDs and non-PDs controls have been obtained in neuropsychological and biological studies including biochemistry, neuroendocrine, metabolic, and neuroimaging evidences. There are also biological data for some specific PDs and personality domains, particularly borderline, antisocial and schizotypal.

In PDs of cluster A, disturbances in cognitive organisation and information processing are supposed to contribute to the detachment, desynchrony with the environment, and cognitive/perceptional distortions. For instance, disruption of white matter cohesion in the temporal and cingulate regions has been demonstrated in schizotypal PD.

In PDs of cluster B, particularly borderline PD, there is an affective instability that is considered to be mediated by excessive limbic reactivity in gabaminergic/glutamatergic/cholinergic circuits, resulting in an increased sensitivity or reactivity to environmental emotional stimuli. For borderline PD, there is also evidence for changes in serotonergic, dopaminergic, adrenergic, and opioid systems. Striatal volumes tend to be enlarged in subjects with symptoms of cluster B personality disorder. In borderline PD, structural imaging studies also showed decreased volumes of both the orbitofrontal cortex and anterior cingulate grey matter. In antisocial PD (as well as in borderline PD), there is a low threshold for impulsive aggression that is supposed to be related to excessive amygdala reactivity, reduced prefrontal

inhibition, and diminished serotonergic facilitation of prefrontal controls. Antisocial PD shows changes in the ratios of dopamine to serotonin in CSF and cortisol to testosterone in serum.

In PDs of cluster C, there is a low threshold for anxiety that may contribute to the avoidant, dependent, and compulsive behaviours, but biological data for underlying mechanism is scarce.

It is important to note that no causality should be implied from these findings. The observed differences to normal controls may be a consequence of the brain's modulation over time whilst a particular personality develops.

Clinical Presentation

General considerations PDs are characterised by long-term traits patterns influencing cognition, emotions, and behaviour that can be described by the following domains:

- Problems in functioning of aspects of the self (e.g. identity, self-worth, accuracy of self-view, self-direction).
- Interpersonal dysfunction (e.g. ability to develop and maintain close and mutually satisfying relationships, ability to understand others' perspectives and to manage conflict in relationships).
- Disturbance in patterns of cognition, emotional experience, emotional expression, and behaviours that are maladaptive (e.g. inflexible or poorly regulated emotions) and is manifest across a range of personal and social situations (i.e. is not limited to specific relationships or social roles).
- The patterns of behaviour characterising the disturbance are not developmentally appropriate and cannot be explained primarily by social or cultural factors, including socio-political conflict.
- The disturbance is associated with substantial distress or significant impairment in personal, family, social, educational, occupational or other important areas of functioning.

Psychiatric symptoms
PDs are characterised by personality traits or patterns but not psychiatric symptoms; however, there is high comorbidity with depression, anxiety, adjustment and addiction disorders.

Systemic symptoms
Patients with PDs typically do not have general symptoms; however, the later may result from comorbid physical or psychiatric conditions, which are common in PD.

Dermatological symptoms
PDs with poor impulse control and emotional dysregulation may be associated with self-harm behaviours including skin damage leading to artificial lesions (Figs. 24.1 and 24.2). It is important to distinguish non-suicidal self-damage behaviour (e.g. in borderline and histrionic PD) from consequences of suicidal attempts.

Practice Point
Pay attention to any potential self-harm behaviours. If concomitant depressive symptoms are evident, do not hesitate to ask about suicidal ideas and intents.

Practice Point
Do not hesitate to perform thorough skin examination to look for signs of self-mutilations, characteristic for several PDs, that may result from suicidal or as self-harming behaviour: skin cuts, scars, pigmentations, cigarette burns, etc.

Fig. 24.1 Self-inflicted scar lesions of a forearm in a patient with borderline PD as a presentation of emotional dysregulation (in attempt "to dull emotional pain by physical one")

Fig. 24.2 Self-inflicted scar lesions and "cavities/tracks" of a forearm in a patient with dissocial and borderline personality traits, and comorbid heroin addiction

Personality Disorders and Skin

Principles

PDs are typically present prior to the onset of any dermatological or general medical condition, as well as prior to other mental disorders. As a result, they comprise pre-morbid characteristics of a person that may influence and modify clinical presentations of the aforementioned diseases that PD subjects may encounter during their lifespan. PDs are proved to be associated with primary psycho-dermatologic conditions (e.g. skin picking disorder, trichotillomania, body dysmorphic disorder etc.), secondary psychiatric disorders and psychological disturbances or reactions due to skin diseases (e.g. depressive and/or anxious symptoms), and dermatoses per se. This is a so-called comorbidity, a phenomenon very common for PDs. According to available data, the most diagnosed PDs in dermatological outpatients are obsessive-compulsive followed by avoidant, borderline and dependent PDs.

PDs and Associated Primary Psycho-dermatologic Conditions

Obsessive-compulsive PD (OCPD) or anankastic PD is associated with skin picking disorder that is classified in DSM-5 in the obsessive-compulsive spectrum. This association may be explained by OCPD domains as stereotyped and inflexible thoughts and behaviours that may maintain compulsive acts focused on skin. Orderliness and perfectionism, as a feature of OCPD, may also maintain skin picking if there is a sense of "skin irregularity" and an urge to "restore its right planes" that may become important triggers for excoriative acts in some cases of skin picking disorder.

Emotionally unstable PD (EUPD)—or Borderline PD (BPD) in DSM—is associated with body dysmorphic disorder (BDD), dermatitis artefacta, trichotillomania, and skin picking disorder. This association may be explained by the attribution of the abovementioned conditions to the compulsivity–impulsivity spectrum that share impulsivity dimension with borderline PD. Impulsivity and emotional dysregulation, typical for EUPD, may support the mechanism of seeking temporary relief from negative emotional states via self-mutilation of own skin or its appendages that is observed in many cases of trichotillomania, skin picking disorder and dermatitis artefacta. Identity disturbances, typical for EUPD, may also include distortions in body image as important facets of BDD. Thus, disturbed body image is considered by some authors to be a common psychopathological feature for both disorders, EUPD and BDD.

Histrionic and narcissistic PDs (HPD and NPD) are associated with BDD and prevalent in subjects seeking cosmetic procedures and cosmetic surgery. Narcissistic PD is observed in 25% of cosmetic surgery patients. Attention seeking, overvalued attitude to self-perfection and excessive preoccupation with attractive appearance, including skin, nails, and hair, are typical for those PDs.

Avoidant and dependent PDs (APD and DPD) are characterised by anxiety personality traits that are greatly related to certain psychological anxious reactions or psychiatric symptoms of adjustment disorders (F43 in ICD-10), triggered by the stress of skin diseases in dermatological patients (see below).

PDs Mediated Reactions in Dermatoses

In dermatology, patients with different PDs tend to react with a set of particular psychological or psychiatric phenomena that are triggered by specific dermatoses and mediated by personality dimensions and PDs. Some secondary psychological reactions in skin diseases are associated with particular personality dimensions. Thus, there is a complex multidimensional structure with three basic patterns: (1) personality or PD, (2) dermatosis, and (3) psychological reaction or psychiatric (e.g. adjustment) disorder. The latter are secondary to dermatoses but greatly influenced by personality. Thus, these secondary psychological conditions or psychiatric disorders may be markers of some particular personality dimensions and PDs. This may be illustrated by a range of reactions due to the disfiguring effect of some dermatoses with visible localisation on the face, scalp, and hands (e.g. acne, alopecia, and psoriasis). These reactions may include sociophobic phenomena, overvalued ideas, and ideas of reference depending not just on skin presentations, but mainly on particular personality mediated patterns which influence the reaction to stress of objective or subjective disfigurement.

Sociophobic Reactions

Sociophobic reactions include obsessive fears of exhibiting skin defects in people and fears of causing a negative assessment of one's own appearance by others. A cosmetic defect is perceived by patients in terms of the opinions of others about it,

which leads to the manifestation/exacerbation of anxiety in situations involving social interaction (social phobia). In fact, the discrepancy of one's own ideas about the proper appearance of the skin/hair/nails with social standards ("skin should look properly, like in everyone else") becomes the denotation of anxiety. As a result, patients try to avoid social situations and/or "camouflage" their defects. Such reactions are observed in several categorical PDs: histrionic, schizoid, avoidant (APD), and anankastic (OCPD). Those PDs exhibit a set of dimensions causing social anxiety: high importance of social approval, a tendency to imitate and follow fashion, high interpersonal sensitivity with fear of being dishonoured by others, a tendency to anxious fears, and high conformity ("external perfectionism").

Overvalued Reactions

Overvalued reactions ("Dorian Gray Syndrome") include actualization of the overvalued idea of preserving "eternal" youth of the skin/hair, endangered by lesions in dermatoses or by the natural process of ageing (wrinkles, hair loss, etc.). Patients do not just exhibit "camouflage" methods (see sociophobic reactions above), but are also engaged in self-destruction: scratching and picking acne lesions ("acne excoriee"), pulling out gray hairs ("pseudo-trichotillomania"), etc. Paradoxically, such overvalued struggle against primary "skin defects" (real and exaggerated or imaginable) perceived as disfigurement typically worsens or causes cosmetic defects leading to irreversible consequences. Such reactions are observed in several categorical PDs prone to overvalued ideas: paranoid, narcissistic, schizoid, and anankastic PDs. Those PDs exhibit a set of dimensions causing an overvalued idea of struggle against the disfigurement: high importance of beauty and the perfection of one's own appearance is crucial for self-esteem and self-judgement ("skin must look perfect so that I can feel perfect"). This "internal perfectionism" is contrasting with fearful expectations for the external appraisal of those prone to sociophobic reactions ("external perfectionism"—see above).

Ideas of Reference Reactions

Reactions with ideas of references include transient delusional ideas of reference caused by alleged skin/hair disfigurement visible by others. In contrast to sociophobic reactions (see above), patients not just fear that they cause a negative assessment of their appearance by others, but they are sure that others notice their ugliness due to their pathological skin/hair condition. Patients interpret other people's behaviours in a delusional way as a consequence of negative attitudes towards them caused by other people's alleged negative appraisal of a skin defect. Patients wrongly assume that others generalise alleged negative attitudes about their alleged skin defect to judge their character and personality in a negative way. They may think that "others think that my skin condition is due to my general neglect of my appearance and/or health". Patients wrongly assume that other people take special notice of their real or perceived skin/hair disfigurement. However, they do not just pay attention to it but "react" and "behave" correspondingly. As a result, patients interpret other people's normal behaviour in a delusional way: "strangers stare at my disfigurement", "they talk about it behind my back", "and they mock me". Such

reactions differ from delusional disorder of somatic type, which is typically chronic and persistent (see chapter 26 "Delusional Disorders in Dermatology") rather than having a transient course. Ideas of reference fade after improvement/remission/cure of a skin/hair condition and may stereotypically exacerbate after the subsequent skin/hair condition flares up again. Such reactions are observed in several categorical PDs prone to transient psychotic phenomena such as emotionally unstable and schizotypal PDs. Those PDs exhibit a dimension of psychoticism that causes transient psychotic symptoms including ideas of reference.

Practice Point
To communicate with PD patients adequately, it is important to understand the nature of reactions closely related to PDs and their relationship with personality traits and dimensions.

PDs as a Predisposition to Dermatoses

There is also some limited evidence that PDs may predispose people to primary dermatological symptoms. For instance, it has been shown that borderline PD accumulates in patients with chronic dermatoses, including psoriasis, urticaria, and prurigo nodularis (approximate prevalence rate in EUPD: about 13–17.5%). However, the causality is very unclear.

Why PDs Are so Debilitating

Influence on doctor–patient relations In dermatological settings, patients with PDs are often perceived by medical providers as "difficult" due to psychological and interpersonal relationships problems influencing efficacy of communication and management (particularly important in emotionally unstable, narcissistic and antisocial PD).

Diagnostic difficulties Some PD patient's cognitive styles (e.g. schizotypal or histrionic) may influence the way patients present their subjective complaints. For instance, itch sensations may be presented with a variety of additional characteristics, influenced by oddity and/or fantasy proneness, like "burning" as if "hot water was placed on the skin"; "crawling" or "stinging" as if "insects move" or "bite" respectively (the so-called formication phenomenon), etc. As a result, it may require differentiation from some severe dermatoses and psychodermatological conditions like delusional infestation.

Influence on compliance PD-related illness anxiety (e.g. avoidant, histrionic and schizoid patients) causes health concerns and may influence adherence to medica-

tions. Such patients may experience fear to take some medications (e.g. topical or systemic corticosteroids) in recommended doses and regimens, due to the fear of perceived or real side effects (e.g. steroid phobia).

Influence on duration of untreated period of dermatoses PD-related social anxiety and detachment (e.g. in avoidant and schizoid/schizotypal PD) may lead to delay in seeking medical/dermatological diagnosis and treatment, as it requires social interactions with medical staff. As a result, it may lead to the worsening of a medical condition and prognosis.

High rate of psychiatric comorbidity Depression, anxiety, substance abuse and addictive behaviours are highly prevalent in PDs and constitute a substantial comorbidity that may differ in specific PDs. For instance, antisocial and emotionally unstable PD patients show significant comorbidity with substance abuse and general use of primary care services. Dependent PD is a basis for depression in response to interpersonal loss. Patients with narcissistic PD may respond with depression to perceived insults to their self-esteem.

High rate of physical comorbidity Despite the increased use of health services, patients with PDs have poorer physical health outcomes, as the aforementioned debilitating factors influence not only skin diseases but other medical conditions as well.

Practice Point
If there is a concern about PD in a patient, it is important to check for other factors influencing general health and well-being: psychiatric comorbidity (addictive disorders, depression, anxiety etc.) and concomitant general medical conditions.

Practice Point
Comorbid PDs complicate treatments of medical illnesses including der*matological conditions.*

Epidemiology

Prevalence in the Population

In the US National Comorbidity Survey Replication study, the population prevalence of PDs is 9.1% (5.7% for Cluster A, 1.5% for Cluster B, and 6.0% for Cluster C). This is a high estimate compared to European studies and may indicate the difficulty making dichotomous diagnoses in dimensional disorders. The PD prevalence is higher in medical settings.

PD in Dermatology

Data about PDs prevalence in dermatology are scarce; however, there is evidence that PDs are more prevalent in the dermatology settings than in general population. One study found that approximately 15% of dermatology outpatients met criteria for a PD, while in healthy controls it was only 5%. Data are mainly limited to particular PDs in some specific dermatoses. For instance, the prevalence of EUPD in dermatological inpatients with chronic dermatoses is estimated to be 13.4%. Prevalence of specific PDs in psoriasis reaches 35%. In psoriasis, there is a high prevalence of personalities defined by certain personality dimensions: combination of social inhibition and negative affectivity patterns were found in 38.7%. In a chronic idiopathic urticaria setting, one reported prevalence rate of PDs was 44.9%, with most patients having a common obsessive-compulsive PD (30.3%).

Epidemiology

There is a gender difference between certain PDs in their prevalence. Dependent, histrionic, and emotionally unstable PDs are more prevalent in females. Approximately 75% of EUPD patients are female. Antisocial, narcissistic, and obsessive-compulsive PDs are more prevalent in males.

Course and Duration

According to its own definition, PD patterns are characterised by being stable and of long duration and their onset can be traced back at least to childhood, adolescence or early adulthood. However, there are some peculiarities and exceptions for certain PDs regarding their long-term course. For instance, EUPD begins in adolescence and is not necessarily a lifelong disorder, as some patients demonstrate a loss of typological characteristics of EUPD during their life span and many patients may retain only residual symptoms later in life. Some PDs may manifest earlier in childhood. For instance, antisocial PD is the only PD known to have childhood antecedents called “conduct disorder”. Approximately 40% of the children with conduct disorder grow up to meet criteria for ASPD.

Diagnostic Process

General Considerations

Diagnosing PDs is a challenging problem. Usually, it requires additional time and resources not always available for a dermatologist or even for a liaison psychiatrist in a dermatological setting. A correct PD diagnosis requires information about the

patient's behaviours and reactions over time, as it is necessary to find out how PD patients react not only to a skin disease but to a wide range of other life situations and circumstances. Patients may lack insight into their personality or its presentations. In many PDs, patients self-reporting of personality patterns is therefore not enough, but "objective" data from close relatives or other informants are required. Thus, in many cases, a correct diagnosis may require a follow-up that is difficult to arrange in dermatological or other medical settings.

Practice Point
To establish effective communication with patients with PD, keep in mind key PD traits that influence interpersonal relationships, including those with doctors.

Diagnosing Process

PD patients often do not complain about their personality problems and dysfunctional traits, as most of them are perceived ego-syntonic, i.e. they are seen as part of the self and therefore not typically considered to be pathological. Thus, if suspected, PDs require active diagnostic search to prove the preliminary diagnosis. There are some external and behavioural clues to suspect a PD and to start an active search. Some of them may be evident from the form of communication with a physician and in the patient's relationship to a dermatological illness.

For instance, patients with EUPD may have terrifying fantasies about their skin disease and often feel either completely well or very ill. This dichotomous thinking ("black or white thinking") contributes to emotional and behavioural instability typical for this type of PD. Patients may make complaints if they feel abandoned or disappointed by their clinician.

In contrast, patients with histrionic or narcissistic PD may have unrealistic expectations when it comes to their appearance, influence of dermatoses on their appearance and the results of cosmetic procedures. Patients with histrionic PD may also react with anger and dysphoria if they feel that they do not get enough attention from a physician. In HPD, an initially idealised clinician–patient relationship may easily turn into dissatisfaction that leads to abrupt breaks and hopping to another provider, as it does in their personal lives.

Patients with obsessive-compulsive PD often experience extreme anxiety of losing control over their skin disease and emotions that result from their medical illness. This can lead to a focus on knowledge of their medical condition and exploration into excessive details as a behavioural strategy for controlling health-related anxiety and fears. As a result, they may ask excessive and repetitive questions and appear as rather exhausting to the clinician.

Further precise diagnosis often requires interaction with psychological and psychiatric services. Such environments may provide opportunities for a detailed examination and further treatment (see below).

To assess PDs or screen for a possible PD, there are a number of questionnaires, as well as structured and semi-structured interviews. Some psychometric instruments used to measure PDs are listed in Table 24.4.

Table 24.4 Psychometric instruments to measure all the subtypes of categorical PDs

The ICD-10 International personality disorder examination (IPDE)
Structured clinical interview for DSM-5 personality disorders (SCID-5-PD)
Personality Disorder Interview (PDI-IV)
Iowa Personality Disorder Screen (IPDS)
Standardised Assessment of Personality: Abbreviated Scale (SAPAS)
Schedule for Non-adaptive and Adaptive Personality (SNAP)
The Personality Diagnostic Questionnaire (PDQ)
Personality Beliefs Questionnaire (PBQ)
Minnesota multiphasic personality inventory (MMPI)

Practice Point
Some psychometrical instruments may be helpful in a formal assessment of PD; however, they are time-consuming, require special training and are not typically used for the diagnosis in routine clinical practice.

Differential Diagnosis

Differential diagnosis of PD should always start with screening for other primary psychiatric conditions such as mood disorders, anxiety, substance abuse disorders, ADHD, and some psychotic conditions. A diagnosis of PD requires differentiation from "normal" personality traits, i.e. subjects that do not fit diagnostic criteria for a PD but rather have strong personality traits as indicated in the new ICD-11 criteria of PD as a dimensional disorder. It is also important to distinguish PDs from personality changes caused by neurological or general medical conditions, such as traumatic brain injury, stroke, epilepsy, or endocrine disorders.

The main clue here is that PDs typically precede the onset of major psychiatric disorders or other medical conditions, develop in childhood or adolescence, and exhibit a relatively stable and stereotypic pattern of behaviour.

Differential diagnosis for PDs

PD subtype	Conditions to differ from and key features
Paranoid PD (PPD)	Requires differentiation from schizophrenia, delusional disorder (persecutory type), a depressive disorder, or bipolar disorder with psychotic features. Paranoid PD differs from delusional disorder in a lack of delusional thinking (system) and delusional behaviour. Paranoid PD differs from schizophrenia in a lack of negative symptoms and prominent positive phenomena (e.g. delusions and hallucinations). It differs from affective disorders in stability as recurrent depression and bipolar disorder with psychotic features exhibit psychotic symptoms only in affective episodes.

PD subtype	Conditions to differ from and key features
Schizoid PD (SPD)	Requires differentiation from schizophrenia. Requires differentiation from schizotypal and avoidant PD. Schizoid PD shares the feature of social withdrawal with STPD but may be distinguished from it by the absence of social anxiety and cognitive-perceptual aberrations. In contrast to STPD, schizoid PD may represent sub-syndromal negative symptoms (social and physical anhedonia) that are the source of social withdrawal in SPD, while STPD might represent sub-syndromal positive symptoms resembling those in schizophrenia (e.g. cognitive-perceptual aberrations and magical thinking). SPD differs from avoidant PD, but they share the feature of social withdrawal and social anxiety. Social anxiety does not diminish with familiarity, while in avoidant PD social withdrawal and anxiety are associated only with the initiation of a relationship and then fade.
Schizotypal PD (STPD)	Requires differentiation from schizophrenia, especially its prodromal (or residual) phase. Can be distinguished from schizophrenia due to age of onset (since childhood) and stability as there are no signs of a recent deterioration in functioning. In contrast to a prodromal phase of schizophrenia, STPD is not followed by an active psychotic episode. Requires differentiation from other PDs, particularly schizoid and avoidant.
Antisocial PD (ASPD)	Requires differentiation from a substance dependence disorder. However, as there is an overlap, both diagnoses are possible in a single person. Can be distinguished from a substance dependence disorder due to a conduct disorder present prior to the age of 15 years and prior to an addiction. Requires differentiation from other PDs, particularly narcissistic.
Emotionally unstable PD (EUPD)	Requires differentiation from a number of psychiatric disorders, e.g. affective, anxious, dissociative, addictive, and some psychotic (e.g. acute transient) disorders. However, as there is an overlap, and several diagnoses are possible in a single person. Can be distinguished from other major psychiatric disorders due to an onset in adolescence and prior to other primary psychiatric conditions. Along with impulsivity and anger flares, EUPD may be distinguished due to a "stable in its instability" pattern of intermittent transient affective and anxious symptoms, dissociative experiences, and highly stress-related delusional ideation. Mood changes are usually rapid in onset and not clearly defined as episodic-like in bipolar affective disorder. Chronic dysthymia and emotional numbness are common. Impulsivity can appear similar to ADHD, but other ADHD features such as poor attention and persistent hyperactivity are usually lacking.
Histrionic PD (HPD)	Requires differentiation from other personality disorders, particularly emotionally unstable, antisocial, and narcissistic. HPD may share with EUPD attention-seeking, manipulative behaviour, and rapidly shifting emotions, but in contrast to EUPD, it lacks self-destructiveness, angry disruptions in close relationships, and chronic feelings of emptiness and identity disturbance. HPD may share with ASPD impulsivity, excitement seeking, recklessness, seduction, and manipulative behaviour, but in contrast to ASPD it tends to be more exaggerated in own emotions, lacks engagement in antisocial behaviours, and manipulation to gain profit, power, or some other material gratification (HPD patients typically manipulate to gain nurturance and care). HPD may share with NPD a tendency to seek attention from others. In contrast to NPD, histrionic patients do not want praise for their perceived superiority but maybe ready to be viewed as fragile or dependent if this helps to get attention.

PD subtype	Conditions to differ from and key features
Narcissistic PD (NPD)	Requires differentiation from antisocial PD. NPD patients are typically more grandiose but not so exploitative and are not involved in recurrent antisocial activities, as ASPD patients. The willingness to exploit others in NPD patients is more passive, serving to enhance self-image by attaining praise or power. In contrast, ASPD patients' exploitation of others is more likely to be consciously and actively related to materialistic or sexual gain.
Avoidant PD (APD)	Requires differentiation from the social anxiety disorder. Requires differentiation from schizotypal and schizoid PD (see above).
Dependent PD (DPD)	Requires differentiation from other medical conditions with excessively dependent behaviour, e.g. agoraphobia, schizophrenia, dementia, severe injuries etc. Dependent PD differs from those acquired conditions in the presence of dependent traits since late childhood or adolescence. Dependent PD requires differentiation from culturally determined behaviour patterns as there is a great difference in the degree of importance attached to deferent behaviour, politeness, and passivity in different cultural groups.
Obsessive-compulsive PD (OCPD) or anankastic PD	Requires differentiation from obsessive-compulsive disorder (OCD) and hoarding. OCPD differs from obsessive-compulsive disorder by the presence of true obsessions and compulsions in OCD; however, there may be comorbidity and both diagnoses are possible. OCPD differs from hoarding disorder by lack of extreme apartment "warehousing" (e.g. accumulated stacks of worthless objects present a fire hazard and making it difficult for others to walk through the house). With narcissistic PD, OCPD may share a commitment to perfectionism and believe that others cannot do things as well, but NPD patients are more likely to believe that they have achieved perfection, whereas those with OCPD are usually self-critical. OCPD may share an apparent tendency to value formality and social detachment with schizoid PD, but in OCPD this stems from discomfort with emotions and excessive devotion to work, in contrast to schizoid PD where it is a fundamental lack of capacity for intimacy.

Treatment

General Considerations

Treatment of PDs is a difficult task as there is often no or only partial insight into PD presentations. Patients often do not complain about dysfunctional traits directly. Thus, involvement in treatment may be a challenging problem in some PDs (e.g. in antisocial, paranoid, and schizoid). PDs are defined by well-established and rigid behaviour patterns that can be integral to a patient's self-image and structure of personality. Thus, it is considered that it is highly unrealistic to treat a PD in a purely non-psychiatric setting such as a dermatology clinic. For dermatology and other specialities who deal with a high prevalence of PD in their patients, it is

Practice Point
PDs require differentiation from primary psychiatric disorders and some medical/neurological conditions that may require involvement of psychiatric services trained in PD assessment and management.

recommended to focus on optimising the working relationship with a patient with PD in order to provide the necessary dermatologic care to the patient's satisfaction. In a second step, and if possible, the clinician can endeavour to involve the patient in joint care including psychiatric and psychological modalities or try to refer to psychotherapeutic treatment. To be effective in PD management, treatment often requires a close interaction between the patient's general health provider, dermatologist, psychiatrist, psychologist, social worker, nurse etc. The "psy-specialists", involved in the interaction, ought to have a special knowledge and skills in PDs management as it is a rather specific mental health issue. Despite the abovementioned difficulties in PD management, there is strong evidence for its effectiveness and for clinically and socially meaningful changes in response to psychotherapeutic and pharmacologic treatments.

Practice Point
PD management ideally requires joint care from a multispecialty team to address the multidimensional issues related to the complex nature and multiple comorbidities seen in PD.

Non-pharmacological Interventions

The main treatment approach in PDs is psychotherapy whereas pharmacotherapy plays an adjunctive role (see below). As it is a complex and specific issue, it will be addressed briefly. Specific psychotherapy techniques, either in individual or group settings are effective. Different approaches are effective in PD treatment, such as psycho-dynamically based psychotherapy (mentalization-based treatment), behavioural therapy-derived dialectic behavioural therapy (DBT), comprehensive validation therapy (CVT), and a fusion of cognitive and psychodynamic therapy called cognitive analytic therapy (CAT).

A number of other promising psychotherapies are under investigation: interpersonal therapy, cognitive therapy, systems training for emotional predictability and problem-solving (STEPPS), transference focused psychotherapy (TFP), schema therapy etc. There is also value of psychoeducation primarily focused on insight of PD.

It should be noted that EUPD remains the most studied in regard of psychotherapy. To get acquainted with basic psychotherapeutic principles, various psychiatric organisations around the world have published guidelines for treatment.

Pharmacological Treatment

Psychotropic medications are widely used in PDs; however, no medication is officially approved for PD treatment by any national or international authority. Thus, medications use in PDs is generally off-label. Some reviews have shown modest improvements with antipsychotics for some PDs. Importantly, medications may target comorbid psychiatric conditions (affective, anxiety disorders etc.), transient psychotic phenomena or some specific "traits-symptoms" like negative and unstable affect or impulsivity in EUPD, or cognitive and perceptual distortions in schizotypal PD.

In comorbid psychiatric conditions, it is recommended to use psychiatric medication according to indication and accepted guidelines, as PDs can negatively influence outcomes in psychiatric disorders. When using psychostimulants for ADHD, especially in emotionally unstable or antisocial PD as there is a potential risk of addiction, so a cautious increase in doses and regular monitoring is recommended.

Regarding transient psychotic phenomena, particularly in EUPD and schizotypal PDs, it is recommended to use antipsychotics doses lower than those used in schizophrenia or other primary psychoses, and that there should be a titration from minimal dosages.

Regarding specific "traits-symptoms" or component traits of PDs, there are specific treatment targets (best developed for EUPD but also evident for STPD) that include negative and unstable affect, impulsivity, aggressive behaviours, and others. As of 2021, it has been shown that some antipsychotics are moderately effective for traits such as antagonism, negative affectivity, disinhibition, psychotic symptoms, and interpersonal dysfunction. However, overall, data is scarce and sometimes inconclusive. Antiepileptic medications (valproate, topiramate, lamotrigine) may also influence antagonism, negative affectivity, and disinhibition. There is often a need for slow dose titration, particularly for lamotrigine in dermatology, as it can cause severe dermatological side effects.

Discussing the Diagnosis and Management with a Patient

The discussion style should be based on PD subtype, predominant dysfunctional traits, and PDs related reactions to skin problems.

For instance, as patients with EUPD suffer from an exaggerated fear of abandonment and rejection of any kind, denial of requested treatments or perceived inadequate treatment of their dermatological condition can be felt as a sign of abandonment and cause intense anger, leading to the potential conflict with the provider. Thus, when approaching patients with EUPD it is important not to minimise (verbally/

Practice Point
PD treatment depends greatly on severity, PD subtype, main dysfunctional traits, psychiatric and medical comorbidity resulting in a set of possible options: psychotherapy alone or, a combination of psychotherapy and medications.

behaviourally) or undertreat the presenting dermatologic condition, which may appear minor to the dermatologist or purely psychiatric in nature, but maybe of a high/exaggerated subjective value for the EUPD patient at that moment.

In patients with OCPD who may be feeling a loss of control because of their skin condition leading to anxiety and other negative emotions, the discussion of the dermatological diagnosis and treatment should be structured and professional, with a very scientific or detailed "lecture-like" explanation. Written information, specific resources including websites or journal articles for accurate information about the patient's condition, along with specific plans for diagnosis and treatment may be helpful here.

Practice Point
In PDs, physicians' communication style requires adjustment to the patient's individual traits.

Prognosis

Existing Prognosis Data

In the past, the prognosis for PD was considered poor due to the persistent and inflexible nature of PDs. However, it has been shown in recent follow-up studies that this is not quite the case and the prognosis is variable. For instance, it has been reported that about 50% of initially diagnosed EUPD patients did to meet diagnostic criteria after 7 years of follow-up, and about one-third of patients with emotionally unstable personality disorder had recovered by their follow-up evaluation. It has been shown that the severity of pathology at the initial assessment primarily predicts the level of borderline psychopathology at follow-up.

Duration of Treatment

As PDs are long-standing, treatment is typically long term and may require months or years.

Bibliography

Dorozhenok IY, Matyushenko EN, Olisova OY. Dysmorphophobia in dermatological practice. Russian J Skin Venerial Diseases. 2014;1:42–7. (In Rus). https://doi.org/10.17816/dv36845.

Nakamura M, Koo J. Personality disorders and the "difficult" dermatology patient: maximizing patient satisfaction. Clin Dermatol. 2017;35(3):312–8. https://doi.org/10.1016/j.clindermatol.2017.01.009.

Napoleon A. The presentation of personalities in plastic surgery. Ann Plast Surg. 1993;31:193–208.

Smulevich AB. Personality disorders. Trajectory in mental and somatic diseases. Moscow: Medical Informational Agency; 2012. (In Rus).

Young JQ. Personality disorders. In: Feldman MD, Christensen JF, Satterfield JM, editors. Behavioral medicine: a guide for clinical practice. 4th ed. New York: McGraw Hill; 2014.

Dysaesthetic Disorders

25

J. M. R. Goulding

Dysaesthesia refers to unpleasant skin sensations, described variably by patients as burning, stinging, painful, pruritic, prickling, or lancinating. This may present anywhere on the skin from the scalp to the soles and can lead to significant distress and functional limitations. This chapter provides an overview of the spectrum of cases which may present and a practical guide for approaching such problems.

Differential Diagnosis

As in all psychodermatology, the first responsibility of the treating clinician is to actively consider, and if necessary exclude, identifiable organic disease. For those presenting with dysaesthetic skin symptoms, the obvious broad category which springs to mind is that of the neuropathies. Depending on the body site affected, a range of conditions may be implicated (see Table 25.1). A detailed exposition of this subject is clearly beyond the scope of this chapter but suffice to say, if there is sufficient suspicion, onward referral to a neurologist is recommended.

Localised Neuropathic Disorders

There are a handful of well-recognised localised neuropathic disorders that are encountered not infrequently in dermatology clinics. Three of these are summarised below.

J. M. R. Goulding (✉)
Dermatology Department, Solihull Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK
e-mail: jon.goulding@uhb.nhs.uk

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_25

Table 25.1 Neurological differential diagnosis for dysaesthetic disorders

Sensory mononeuropathies, e.g. meralgia paraesthetica, trigeminal neuralgia, pudendal neuralgia
Polyneuropathies, e.g. due to diabetes mellitus, vitamin B12 deficiency, alcohol excess, drug-induced
CNS disorders, e.g. multiple sclerosis, HIV or other infections, cerebrovascular disease
Erythromelalgia
Complex regional pain syndrome

Meralgia paraesthetica presents with unilateral numbness or dysaesthesia over the anterolateral thigh. This arises as a consequence of localised compression of the lateral cutaneous nerve of the thigh, on account of anatomical variation, obesity, or injury. Men are affected more commonly than women. Treatment, if required, includes topical agents such as capsaicin cream, local nerve blocks, low-dose amitriptyline or gabapentin, and in resistant cases, surgical decompression (Harney and Patijn 2007).

Notalgia paraesthetica refers to a unilateral patch of dysaesthetic skin in the region just below the scapula, lateral to the spinal column. There is uncertainty regarding its aetiology, but it is assumed to reflect damage to, or impingement of, spinal nerves supplying the skin in this area, as a consequence of cervical or thoracic musculoskeletal disease. Mild hyperpigmentation is often noted at affected sites, occurring as a result of repetitive scratching or rubbing. Rippled macular hyperpigmentation suggests the presence of secondary cutaneous amyloid deposits. Treatment may best be directed towards any underlying musculoskeletal issues, such as with physiotherapy or transcutaneous electrical nerve stimulation (TENS). Topical therapies may help in addition, such as with menthol in aqueous cream, capsaicin or doxepin creams, or lidocaine gel or plasters. Again, neuropathic pain medications such as amitriptyline, gabapentin, or pregabalin could be considered in more severe cases (Hylwa et al. 2014).

Brachioradial pruritus describes the occurrence of intensely pruritic or dysaesthetic skin over one or both upper arms and/or forearms. Symptoms are typically described as severe and debilitating, relieved with the application of ice-packs. Clinically there is little to see other than secondary skin manifestations of scratching or rubbing. This condition is thought to reflect a neuropathy of C fibre nerves in the skin (De Ridder et al. 2010). Cervical radiculopathy may be implicated, and imaging should be considered to exclude degenerative disc disease, a cervical spinal tumour, or cervical rib. Investigation and management are probably best coordinated by a neurologist, to whom such patients should be referred.

Trigeminal Syndromes

Special mention should be made of a set of disorders involving damage to the trigeminal nerve.

Table 25.2 Trigeminal trophic syndrome: possible causes (Hylwa et al. 2014)

Post-surgical, e.g. ablation for trigeminal neuralgia
Vascular, e.g. cerebrovascular accident, aneurysm
Trauma
Multiple sclerosis
Malignancy, e.g. glioma, schwannoma, meningioma
Infection, e.g. intra-cranial abscess, syphilis, post-encephalitis
Peripheral trigeminal neuropathy, e.g. due to leprosy, amyloid, diabetes mellitus

Trigeminal neuralgia is characterised by intense paroxysms of unilateral pain affecting tissues in the distribution of one or more branches of the trigeminal nerve. There are a number of potential underlying causes and differential diagnoses which necessitates careful evaluation and management by a neurologist.

Trigeminal trophic syndrome is more likely to present to a dermatologist since the clinical features may first raise suspicion for skin cancer. The typical presentation is that of a unilateral arcuate or crescentic eroded or ulcerated area on the nasal ala. Any part of the skin innervated by the trigeminal nerve may be affected, however, with chronic scratching causing deep tissue destruction, driven by localised dysaesthesia or anaesthesia and recalcitrant pruritus. This clinical picture implies some form of damage to the trigeminal nerve anywhere along its course, hence the need for a careful history and thorough neurological investigation (see Table 25.2) (Hylwa et al. 2014). In addition, a skin biopsy may be taken to exclude the possibility of neoplasia. Factitious disorders such as dermatitis artefacta and skin picking disorder should certainly be borne in mind for this presentation, and a detailed biopsychosocial assessment may help to differentiate such entities. Treatment depends upon the cause, and often requires a multidisciplinary approach, with patient education key to success (Hylwa et al. 2014). Oral neuropathic pain medication may help, and surgical techniques are occasionally employed (Preston et al. 2006).

Atypical trigeminal trophic syndrome is a recently described entity in which there is a demonstrable trigeminal nerve abnormality upon neurophysiological testing, but atypical clinical features, such as with bilateral skin changes, or those affecting more than one dermatome (Gkini et al. 2019). Pregabalin is said to be particularly effective in such cases, to reduce abnormal skin sensations, and on account of its anxiolytic properties (Gkini et al. 2019). Referral to a clinical psychologist may also help to manage associated stress, anxiety, and depression, as well as to teach techniques to reduce skin manipulation (Gkini et al. 2019).

Regional Dysaesthesia

While rare in their own right, the commonest category of dysaesthetic disorder encountered in dermatology, particularly in specialist clinics, is that which involves an entire anatomical region, such as the genitals, scalp, face, or oral mucosa. Patients with such presentations are complex and require lengthy appointments to elicit the

full extent of their problems, as outlined later in the suggested approach to take. Conducting a holistic, biopsychosocial assessment is key to making progress.

Dysaesthetic penoscrotodynia (DPSD) (Fig. 25.1) may affect any part of the male genitalia, including adjacent skin in the groins, thighs, perineum, and perianal area. Patients often report redness or other perceived visible abnormalities, though clinical examination is normal (Markos 2011). As with all disorders of this type, organic disease should be considered and excluded if necessary (see Table 25.3).

DPSD shares many overlapping features with (burning) red scrotum syndrome, and it is likely that these terms refer to the same condition. The scant existing literature subscribes to the view that DPSD reflects a neuropathic pain disorder, in spite of a lack of supporting evidence. This is reflected in the range of topical (e.g. lidocaine gel) and systemic (e.g. low-dose amitriptyline) treatments commonly employed, which in my practical experience are rarely beneficial.

The invariable presence of psychopathology when such patients are actively screened, and the remarkable response to psychodermatological treatment in those who accept it, suggests that DPSD is a functional somatic symptom disorder (Anyasodor et al. 2016). A strikingly intense and anxious persona is usually projected by affected patients, who describe an unremitting focus on their symptoms, frequent checks on the physical state of their skin, and relentless online searching for information on cause and cure. Such behaviours only serve to exacerbate anxiety, and may lead to relationship dysfunction and increasing social isolation. Anecdotally there seems to be a high incidence of stressful trigger events closely preceding the onset of symptoms, with regret or shame over sexual infidelity

Fig. 25.1 Penoscrotodynia. Chronic rubbing of the penile shaft skin has led to mild lichenification

Table 25.3 Differential diagnosis for DPSD

Pudendal neuralgia
Chronic epididymitis
Chronic prostatitis
Testicular cancer
Sexually transmitted infections, e.g. Chlamydia

Table 25.4 Differential diagnosis for vulvodynia

Infective, e.g. recurrent candidiasis or herpes simplex
Inflammatory, e.g. lichen sclerosus, lichen planus
Neurological, e.g. pudendal neuralgia, post-herpetic neuralgia
Neoplastic, e.g. extra-mammary Paget's disease, squamous cell carcinoma
Vulvo-vaginal atrophy

sometimes admitted by patients. Treatment with selective serotonin reuptake inhibitors (SSRIs, e.g. sertraline or citalopram) and/or psychological talking therapy is typically highly rewarding.

Vulvodynia is the female equivalent of DPSD and has been subject to more extensive research. Referring to vulval pain of at least 3 months' duration, it is again diagnosable only after excluding identifiable physical causes, though it may co-exist with a specific disorder (see Table 25.4). Different variants are recognised, including localised and generalised, provoked and spontaneous vulvodynia (Bornstein et al. 2016).

There is some evidence that specific physical factors may play a role in the pathogenesis of this condition, particularly related to the perception and processing of painful stimuli, which may lead to "central sensitisation" (Zhang et al. 2011). It is well-recognised that vulvodynia is frequently comorbid with a range of other chronic pain disorders such as irritable bowel syndrome and fibromyalgia (Reed et al. 2012). Furthermore, the high prevalence of psychosocial distress in such patients has led to calls for vulvodynia to be treated as a somatoform disorder, analogous to DPSD (Lynch 2008). A multidisciplinary management approach is ideal, providing an individualised mix of skin-directed treatment alongside physiotherapy, pharmacological therapy (e.g. pregabalin, sertraline, citalopram), and psychological talking therapy.

Scalp dysaesthesia is commonly encountered in specialist hair clinics, and patients share many of the characteristics described above, with heightened awareness of physical sensations, hypervigilance, high levels of anxiety and depression, and consequent functional disability. Patients frequently count shed hairs, conduct physical checks on their scalp throughout the day, and report erythema, inflammation, and burning discomfort out of proportion with the clinical appearance.

Burning red face syndrome (Fig. 25.2) is synonymous with terms such as neurogenic rosacea and facial erythrodysaesthesia and patients present in general

Fig. 25.2 Burning red face

Table 25.5 Differential diagnosis for burning mouth syndrome (Bruce et al. 2014)

Infective, e.g. recurrent candidiasis or herpes simplex
Inflammatory, e.g. recurrent aphthosis, lichen planus, immunobullous disease
Dental, e.g. poor dentition, oral prostheses, allergic contact stomatitis
Neoplastic, e.g. squamous cell carcinoma
Drug-induced
Xerostomia, e.g. Sjogren's syndrome
Nutritional deficiency, e.g. folate, vitamin B12, iron

dermatology clinics. Chronic pain conditions and psychiatric, including other somatoform, disorders frequently coexist, again mandating a thorough biopsychosocial approach to assessment and subsequent management (Affleck and Stewart 2016).

Burning mouth syndrome refers to recalcitrant oral mucosal discomfort in the absence of an identifiable physical disorder. Again, alternative explanations must be sought (see Table 25.5) (Bruce et al. 2014), though not at the expense of timely and meaningful action to address the significant background patient distress and functional limitations.

Approach to the Patient with Dysaesthesia

Patients with a straightforward localised neuropathic disorder can usually be helped satisfactorily within a standard outpatient dermatology appointment. Those with a more complex multifactorial presentation, and especially those with a regional dysaesthetic syndrome, need and deserve a comprehensive holistic assessment. There is no substitute for providing sufficient time in such consultations, either allowing the clinic to run late on the first presentation, or rebooking into a longer appointment slot

at a later date. This allows the patient to tell their story completely in an unhurried fashion, and the clinician to demonstrate empathy, to defuse hostility born of previous unsatisfactory consultations, and to build trust and rapport. Investing time and energy in this manner yields huge dividends, both at the time and in subsequent meetings.

History

A full dermatological and general medical history is required to exclude the possibility of organic physical disease. Always ask about alcohol intake and recreational drug use. A sexual history is helpful in cases of DPSD and vulvodynia. A basic psychiatric assessment is crucial, including current mood and suicidal ideation, the impact of symptoms on daily functioning, and past personal and family psychiatric history. Beyond this, a sensitive but thorough exploration of the patient's social history is illuminating, including potential stressors at work, at home, and within relationships. It is often rewarding to explore what was happening in the patient's life just before the onset of symptoms. Do ask specifically about the patient's ideas regarding causation, as well as fears and expectations. This may yield invaluable insights that help guide further management.

Examination

A comprehensive skin examination should be conducted, including the scalp, nails, and oral cavity. Not only does this permit subtleties to be spotted which might lead to a physical diagnosis, but it also demonstrates openly to the patient that their physical complaint is being taken seriously. A meticulous genital examination is very important in cases of DPSD and vulvodynia. An abridged mental state examination can be carried out while the history is being taken or during physical examination.

Investigations

Validated indices screening for anxiety and depression should be administered to every patient presenting with a regional dysaesthetic syndrome. The GAD-7 (Spitzer et al. 2006) and PHQ-9 (Kroenke et al. 2001) scales are particularly recommended, since these are used widely in primary care and to gain access to local psychological services. Patients never take issue with completing such instruments, particularly if introduced casually with the explanation that it forms part of the standard clinic assessment for everyone.

Beyond this, it may be that no further investigations are deemed necessary. Any that are requested should be chosen judiciously, kept to a minimum, and only where clinically indicated. Their rationale should be explained to the patient, including the ramifications in the event of a normal result.

Management

An empathic acknowledgement of the genuine and distressing physical symptoms experienced by the patient is of paramount importance to maintain trust. It is appropriate to provide reassurance that no worrisome cause has been found thus far, but also to indicate a willingness on the part of the clinician to keep an open mind going forward. The same can be politely requested of the patient, so as to allow both parties to embrace all possible explanations for the symptom complex.

It is surprising how often patients are willing to accept a psychological construct for their condition at the first consultation, even to be referred there and then for psychological therapy or psychiatric assessment. This of course assumes that sufficient time has been invested by the clinician, and the above structure followed carefully. Patients often come to the same realisation themselves if they have struggled with their symptoms for long enough, and exhausted other possibilities.

If, however, there is great reluctance or unwillingness to accept a psychological explanation, on account of limited or absent patient insight, the clinician must back off and regroup. In this situation, it is best to seek common ground by focusing on the negative impact of symptoms on the patient's ability to function day to day. Offering physical or skin-directed treatment alongside psychosocial help and support is usually well-received. It may take several consultations before patients feel able to acknowledge a psychological dimension to their situation, but this will be time well spent to preserve and nurture the doctor–patient relationship.

Providing basic psychoeducation at every opportunity is helpful (see Table 25.6 for suggested phrases).

There is often a tendency for patients to focus relentlessly on aetiology, but it is better to pivot towards a pragmatic treatment plan if possible. Regular communication with local GPs is strongly encouraged, and it may be that they are best placed to initiate psychotropic medication such as SSRIs, to facilitate close follow-up. There should be a low threshold for referral to psychiatry if there is severe mood disturbance, suicidal ideation, or a significant personality disorder. Referral to an integrated multidisciplinary psychodermatology clinic is ideal if available locally.

Table 25.6 Basic psychoeducation: stock phrases

"This is a common condition; we've seen this many times before"
"This is a distressing problem, but it is not dangerous"
"I believe you about your symptoms, but we may need to find a different explanation for them"
"We may not be able to explain exactly why it has happened, but we can help you get better"
"Distracting yourself when you get the symptoms may make things more manageable"
"It would help for you not to check your skin/search online so often"
"Get out of the house and keep active—this will help take your mind off things"

References

Affleck A, Stewart M. Burning red face syndrome: a heterogeneous group of facial erythrodysaesthesias. Clin Exp Dermatol. 2016;41:430–44.

Anyasodor MC, Taylor RE, Bewley A, Goulding JMR. Dysaesthetic penoscrotodynia may be a somatoform disorder: results from a two-centre retrospective case series. Clin Exp Dermatol. 2016;41:474–9.

Bornstein J, Goldstein AT, Stockdale CK, et al. 2015 ISSVD, ISSWSH and IPPS consensus terminology and classification of persistent vulvar pain and vulvodynia. Obstet Gynecol. 2016;127(4):745–51.

Bruce A, Torgerson RR, Wriston CC, Gonzalez Santiago TM. Burning mouth syndrome. In: Bewley A, Taylor RE, Reichenberg JS, Magid M, editors. Practical psychodermatology. Chichester: Wiley-Blackwell; 2014.

De Ridder D, Hans G, Pals P, Menovsky T. A C-fiber-mediated neuropathic brachioradial pruritus. J Neurosurg. 2010;113(1):118–21.

Gkini MA, Ahmed A, Aguilar-Duran S, et al. Atypical variant of trigeminal trophic syndrome successfully treated with pregabalin: a case report series. Clin Exp Dermatol. 2019;44(2):225–8.

Harney D, Patijn J. Meralgia paresthetica: diagnosis and management strategies. Pain Med. 2007;8(8):669–77.

Hylwa SA, Davis MDP, Pittelkow MR. Dysesthetic syndromes. In: Bewley A, Taylor RE, Reichenberg JS, Magid M, editors. Practical psychodermatology. Chichester: Wiley-Blackwell; 2014.

Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16:606–13.

Lynch PJ. Vulvodynia as a somatoform disorder. J Reprod Med. 2008;53(6):390–6.

Markos AR. Dysaesthetic penoscrotodynia: nomenclature, classification, diagnosis and treatment. Int J STD AIDS. 2011;22:483–7.

Preston PW, Orpin SD, Tucker WF, Zaki I. Successful use of a thermoplastic dressing in two cases of the trigeminal trophic syndrome. Clin Exp Dermatol. 2006;31(4):525–7.

Reed BD, Harlow SD, Sen A, et al. Relationship between vulvodynia and chronic comorbid pain conditions. Obstet Gynecol. 2012;120:145–51.

Spitzer RL, Kroenke K, Williams JW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166:1092–7.

Zhang Z, Zolnoun DA, Francisco EM, et al. Altered central sensitization in subgroups of women with vulvodynia. Clin J Pain. 2011;27(9):755–63.

Delusional Disorders in Dermatology

26

Dmitry V. Romanov and Peter Lepping

Definitions

Delusional disorder (DD) is defined by the presence of one or more delusions that are false beliefs of different content. The latter may include persecutory, erotomanic, grandiose, jealous, and somatically focused erroneous ideas. In contrast to schizophrenic delusions, there are no other characteristic symptoms of schizophrenia (e.g. persistent auditory hallucinations, disorganized thinking, negative symptoms). Various forms of perceptual disturbances (e.g. hallucinations, illusions, misidentifications, etc.) thematically related to the delusion may be present. Apart from actions and attitudes directly related to the delusion or delusional system, other mental functions (affect, cognition, speech, behaviour, etc.) are typically unaffected.

Classification In ICD-10, DD is coded as F22, attributed to *Persistent delusional disorders,* and placed in F2 class *Schizophrenia, schizotypal and delusional disorders.* In DSM-5, DD is coded as 297.1 and attributed to *Schizophrenia Spectrum and Other*

D. V. Romanov (✉)
Department of Psychiatry and Psychosomatics, I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia

Department of Boundary Conditions and Psychosomatic Disorders, Mental Health Research Centre, Moscow, Russia

Department of Clinical Dermatovenereology and Cosmetology, Moscow Scientific and Practical Center for Dermatovenereology and Cosmetology of the Moscow Department of Health, Moscow, Russia

P. Lepping
Wrexham Maelor Hospital Liaison Service (BCULHB), Wales, Wrexham, UK

Centre for Mental Health and Society, Bangor University, Wales, Bangor, UK

Mysore Medical College and Research Institute, Karnataka, India

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_26

Psychotic Disorders. The similar conceptualization is provided in ICD-11. DD is coded as 6A24 and placed among *Schizophrenia or other primary psychotic disorders.*

In both ICD-10 and DSM-5, DD is subdivided into several types according to delusional content.

DD types:

Type	The central theme of the delusion
Erotomanic	The individual's belief that they are the object of another person's love or sexual desire
Grandiose	The individual's belief that they possess a great talent or have made an important discovery which is underestimated and associated with an inflated sense of worth, power, knowledge, or identity.
Jealous	The individual's belief in their spouse or lover's infidelity.
Persecutory	The individual's belief that they are being intentionally harmed or is going to be intentionally harmed by other(s) who may conspire against, cheat, spy on, follow, poison, maliciously malign, harass, or obstruct etc.
Somatic	The individual's belief that something is wrong with bodily functions and/or appearance, or there are pathological sensations.

There are also mixed or unspecified types of DD, when there are several delusional themes but no one predominates or the dominant delusional belief cannot be clearly determined, respectively

Some delusions in DD of somatic type may have content related to skin, and as DD patients lack awareness or insight, i.e. strongly believe in an erroneous idea and deny mental illness, they present to a dermatologist rather than to a psychiatrist.

There are several somatic DD subtypes that dermatologists may encounter:

- Delusional infestation—DI (see Chap. 12): the belief that there is an infestation of insects or other parasites (or even inanimate objects) on or in the skin
- Delusions of internal infestation: the belief that there is an internal parasite that may migrate outside from the viscera through the skin (see Chap. 12)
- Delusional body dysmorphic disorder—DBDD (delusional dysmorphism): the belief that certain parts of the body, e.g. skin itself or its appendages, are abnormal, misshapen, or ugly
- Delusional olfactory reference syndrome—DORS: the belief that the individual emits a foul odour (cacosmia), e.g. perceiving skin as an origin of such a smell

2. *Historical names for DD:*
   (a) "Paranoia" by Heinroth [1818], Kahlbaum [1863] and Kraepelin [1899, 1921]
   (b) "Intellectual monomania" by Esquirol [1838]
   (c) "Chronic mania" by Griesinger [1845]
   (d) "Paranoid disorder" in DSM-III
   (e) "Monosymptomatic hypochondrical psychosis" by Munro (for DD of somatic type)

- Illness delusion or delusion of disease (hypochondriacal delusion) (AIDS-delusions, syphilis delusions, etc): the belief that the individual has an undiagnosed somatic illness or medical condition, e.g. sexually transmitted infection

1. *Aetiology and Pathology: see Chapter about delusional infestation*

Clinical Presentation

General considerations DD is characterized by a delusional system or a patient's specific explanatory model. The system includes a set of well organized and persistent ideas around a particular theme. Such delusional ideas are interconnected with each other in a "logical" structure with a set of "evidences" and explanations ("logical reasoning"). As there is no insight or awareness, such delusional ideas may be vigorously defended. Delusions occur in clear consciousness and not due to an underlying physical illness or to a psychiatric disorder other than delusional disorder.

Typically, delusional ideas lead to corresponding delusional behaviour. However, delusions in DD are relatively isolated (encapsulated) and coincide with otherwise intact non-delusional mental functioning, behaviour outside the delusional topic often remains normal and may mask episodes of delusional behaviour; or there are just some delusional acts incorporated into otherwise non-psychotic behaviours (partial psychosis). Esquirol (1818) stated that aside from these areas relevant to their "delusional systems", patients "think, reason, and act, like other men".

Delusional Disorders in Dermatology (Hypochondriacal or Somatic Delusions Related to Skin)

In *Delusional Body dysmorphic disorder* (DBDD), there is a delusional belief in an imagined defect in one's appearance. Patients also may have a minor or non-discernible defect that is real but rigidly and inflexibly believe they are deformed. It should be noted that there is also a non-delusional body dysmorphic disorder (BDD, see Chap. 14 about body dysmorphic disorder) not classified as a psychotic condition but classed as a somatoform disorder in ICD-10 or as an obsessive-compulsive (OCD) and related disorder in DSM-5 and ICD-11. However, this just means that appearance preoccupation may be a key feature of anxious, obsessive, overvalued, or delusional syndromes with a continuum of insight from full to absent. Here we discuss the latter.

The important feature of DBDD to distinguish from non-psychotic BDD is a lack of insight. However, sometimes it is hard to establish, as there are also OCD disorders with low insight and overvalued ideas. Thus, a dimensional approach may be recommended as there is a spectrum of dysmorphic concerns with a range of severity.

The Delusional system in dermatological DBDD includes a central belief about skin ugliness visible to others. Patients usually complain of a variety of skin blemishes that they may call "acne", "pimples", "nodules", "vascular spiders", etc. Cosmetic complaints may also include inappropriate skin colour (dyschromia) or irregular texture. The key psychotic features of the delusional system in DBDD are fixed delusional beliefs about appearance and delusional ideas of reference (referential thinking). Patients believe that other people take special notice of the supposed defect, and do not just pay attention to it but "react" and "behave" correspondingly: stare at it, talk about it behind patient's back, laugh at or even mock them.

Delusional behaviour in dermatological DBDD comprises seeking care from cosmetic dermatologists and surgeons to get rid of a defect. Due to lack of insight, patients believe that only a cosmetic procedure or an operation may change the situation. As a result, the patients' medical history may be full of clinically unnecessary dermatological treatments, cosmetic and surgical interventions or even self-mutilations. The paradox is that the results of such procedures may lead to complications that objectively are disfiguring. However, often those are not perceived by patients as a cosmetic problem or are perceived as less problematic than the primary complaint. Due to referential thinking, delusional behaviour may also lead to social isolation (avoidance of situations that may cause others to pay attention to their appearance) and camouflaging of the perceived defect (make-up, special clothes, glasses, etc.).

In *Delusional Olfactory reference syndrome (DORS),* there is a delusional belief that one emits a malodorous smell. As in DBDD (see above), DORS also has its non-delusional equivalent not classified as a psychotic condition but attributed to obsessive-compulsive or related disorders in DSM-5 and ICD-11. Thus, preoccupation with offensive odour may also encompass a range of insight.

The delusional system in dermatological DORS includes central beliefs about the individual's own skin as a definite source of an unpleasant smell. The entire skin may be "involved" or there could be some specific sites, mainly the groin, armpits, or feet. Patients may complain that their sweat has changed its physiological properties and produces such an odour or that the entire body metabolism has altered to produce the smell of the skin. Delusional beliefs about mucosa, i.e. oral (delusional halitosis) or vaginal odour, may also occur.

As in DBDD, there are ideas of reference corresponding to offensive body odour. In DORS, they focus on the impact of an "odour" on others (i.e. the conviction that people are taking notice, judging, or talking about the odour, especially in confined spaces, e.g. in trains, buses, elevators etc). Patients may perceive others' behaviours as caused by odour impact (delusional misinterpretation), including "evidence" of others' desire to stop any social interaction as soon as possible. Patients "notice" others' gestures indicating reaction to the odour, and insist others try to escape and avoid them in situations of any social interaction (e.g. those close to a patient "wrinkle their noses and move away", "touch their nose", "open a window", etc.).

Delusional behaviour includes attempts to camouflage, alter, or prevent the perceived odour.

Patients may wash excessively, change clothes with more than usual frequency, or overuse perfumes and deodorants. Referential thinking leads to avoidance of social situations, sitting at a distance from others, minimizing movement in an attempt "not to spread the odour" etc.

In *Illness delusions or delusions of disease*, there is a delusional belief that one has a specific illness. Delusions of disease typically include transmitted infections, especially venereal diseases (syphilis, gonorrhoea, in last decades especially HIV, etc.).

The Delusional system may comprise the conviction that one is infected. Typically, patients describe in detail how and when they caught the precise pathogen. Usually, they are at low risk of STDs and may cite a single sexual contact, they believe is the cause, that took place decades ago or describe some exotic ways of disease transmission. They appeal to medical literature describing signs and symptoms of the infection to "prove" the diagnosis. Patients often provide delusional explanations of negative medical and laboratory findings that fail to prove infection, e.g. "the smears were taken in periods of latent infection", "the procedure of blood sampling was performed improperly", etc.

Delusional behaviour includes "medical odyssey" as a search for a doctor able to confirm their delusional beliefs. As a result, they demand and often get multiple retests. Another option is a struggle against the infection. They repeatedly take courses of antibiotics or antiviral medications with no or minimal effect. Patients also try to prevent others from becoming infected. They may not just avoid sexual contacts but develop a system of preventive measures including disinfection of household articles and evading any physical contacts with relatives. Patients may even abandon their family/house and move to a separate apartment ("delusional migration").

Dermatological symptoms

Patients with DD of somatic type in dermatology usually complain of changes in their skin. However, it is important to distinguish subjective complaints (skin irregularity, skin odour etc.) and objective self-inflicted lesions as a result of delusional behaviour or underlying minimal dermatological conditions (e.g. mild acne). In DBDD, patients' skin may exhibit a picture of the consequences of multiple cosmetic procedures (scars, pigmentation, etc) and self-inflicted lesions performed in an attempt to improve skin appearance but in fact making it worse. In DORS, the skin is usually intact but in some cases may exhibit consequences of excessive cleansing in attempts to get rid of an odour (skin dryness, irritation, or contact dermatitis), similar to that seen in excessive washing in OCD. In illness delusions or delusions of disease, skin is typically unaffected but in cases with delusional behaviour, focused on genitalia disinfection procedures, corresponding local changes may occur (e.g. dermatitis due to overuse of local antiseptics). Thus, at first glance, skin lesions in DDs of somatic type in dermatology may look very similar to those observed in dermatitis artefacta and skin picking disorder (see corresponding chapters), but the underlying precise psychiatric pattern of self-mutilation is quite different and should be recognized.

Systemic symptoms
Patients with DBDD typically do not have general symptoms and complaints are limited to the perceived deformity. Patients with DORS and illness delusions or delusions of disease may complain of fatigue, dizziness, or malaise depending on the exact delusional system. In DORS, they may think that the skin odour is a result of a general metabolic disturbance of the whole body and as a result other systemic symptoms occur. In illness delusions or delusions of disease, they may focus on the idea of a total organism involvement in the infectious process and express any somatic complaints depending on "damage" to the preferred organ (heart, brain, intestines, etc).

Environmental Presentations
In DBDD and DORS, environmental presentations are rare. But patients with infectious illness delusions or delusions of disease may complain of infections of their immediate environment. In such cases, the delusional behaviour may be very similar to delusional infestation (see Chap. 12). They regularly change bedding, clothes, or furniture, disinfect their apartment and repetitively ask relevant services for decontamination. They may believe that relatives are also infected and make them take courses of antibiotics and antiviral medications or even some toxic non-conventional substances (e.g. veterinarian or industrial).

Practice Point
Pay attention to any delusional behaviours involving self-management as the later may have toxic systemic effects. Activities driven by delusional beliefs and focused on others may also be potentially dangerous for close relatives (be aware of safeguarding issues if it involves children in their care) or medical staff.

Why DD of Somatic Type Is so Debilitating

Self-referential thinking Ideas of reference may lead to severe interpersonal problems and social isolation. DBDD and DORS patients may give up their studies or jobs due to perceived "negative attitudes" to imagined disfigurement or odour "expressed by" classmates or colleagues. Some patients with infectious illness delusions or delusions of disease may exclude any personal relations being convinced that a pathogen is able to transmit easily during routine domestic activities.

Self-damage Some DBDD patients, being disappointed with the results of cosmetic procedures applied by professionals, may try to "fix" their defect or disfigurement themselves with tweezers or chemicals. As a result, real skin disfigurement appears or complications develop (scarring, secondary infection, bleeding). Some DORS patients overuse cleansers to release their skin from the odour, that then causes further skin irritations. DORS patients may ingest inappropriate substances to get rid of the odour if they believe the source is inside the body due to metabolic changes. Patients with illness delusions may overdose antibiotics or other medications to "cure" their infection.

Threat to those around Patients with DBDD may be a threat to cosmetologists, being dissatisfied with the results of their defect or disfigurement correction. As a result, they may not just threaten revenge but also commit physical violence. Patients with illness delusions, who become convinced that their relatives are also infected, may make them go in for inappropriate treatments with a high chance of adverse effects and negative health or life consequences. Some DD patients may also have litigious ideas related to inappropriate cosmetic or medical procedures done. They can place a heavy burden on medicolegal services with multiple claims to the authorities. It may be necessary to consider safeguarding procedures where patients who have care of children apply potentially damaging 'treatments' to their children because of their delusional beliefs that the child is similarly affected.

High rate of psychiatric comorbidity Many patients with DD (nearly 50%) also have depressive symptoms, and comorbid anxiety is also frequent. This may lead to a higher risk of suicidality.

Practice Point
If depressive symptoms are evident, do not hesitate to ask about suicidal ideas and intent.

Epidemiology and Aetiology

Prevalence Epidemiological data about DD are very sparse, as individuals with isolated delusions are often able to function unnoticed in the community and they rarely present themselves to mental health services. The DD lifetime prevalence is considered to be about 0.2%. In the community, point prevalence is 0.03%. Among psychiatric inpatients, it is estimated to be about 2–4%. In some special settings, DD of different types may be accumulated (DBDD in cosmetology, DI in parasitology and dermatology, etc). In prison, point prevalence of DD is reported to be as high as 0.24%, and the most common type of DD content there was persecutory (63.6% of cases), followed by mixed (18.2%), grandiose (14.5%), and somatic (3.6%). In psychiatric settings, the main delusion topic in DD is also persecutory (60.5%) followed by somatic (27.9%), delusional jealousy (7%), and erotomania

(4.7%). There is a lack of data about the prevalence of DD in dermatological settings, except DD of somatic type presented as delusional infestation (see Chap. 12).

Epidemiology In DD, the sex ratio is almost equal (1:1). In general, men are younger than women. The mean age at onset is between 35 and 55 years, 33.8 years in males and 46.4 years in females. DD of jealous type is considered to be more common in males than in females whereas DD of somatic type with delusional infestation and DBDD are more frequent in females (see also DI chapter).

Course and duration of illness DD onset may be acute or subacute, but is typically insidious. Although the severity of the delusion may fluctuate in the disease course, in most cases it is unremitting and chronic. Accordingly, DD is called *persistent delusional disorder* in ICD-10. The mean duration of illness, as published, ranges from a minimum of 3 months to a maximum of 37 years.

Diagnostic Process

General considerations As in other delusional conditions, in DD it is futile to persuade or get into an argument with patients. The thing that is of real value and importance is to establish a trusting relationship with a patient and his/her relatives (if available); however, this may be time-consuming and requires some patience and flexibility. It is beneficial to communicate in a non-confrontational manner with an engaged and interested expression trying to elucidate as much detail of the delusional system and behaviour as possible. Some closed questions about particular thoughts and behaviours typical for DD (see above) may improve this trust. This helps to show that a doctor is aware of the problem and does not consider it as "fake".

There is also an ethical and in some countries the legislative problem of feedback to patients about their condition. It seems that there is no universal approach. There is always a dilemma in deciding whether to announce the DD diagnosis, as this may lead to a loss of trust in the therapeutic relationship, lack of adherence to treatment and potentially subsequently a worse prognosis. Alternatively, a gradual approach may be taken, e.g. referring to the necessity of additional examinations, including psychiatric evaluation. Some general consideration that "an involvement of the nervous system is possible and it should be excluded" may be sufficient at the initial stage of patients' management. Anyway, the approach provided by a dermatologist (or other medical doctors) and a psychiatrist may differ in those circumstances. For example, a consultation-liaison psychiatrist may focus on comorbid psychiatric conditions (e.g. depression, anxiety, insomnia, etc.) related to delusional beliefs.

Diagnostic process The diagnosis of DD is made clinically. No relevant biological markers are available in routine clinical practice. Thus, first of all, it is important to exclude other organic causes for the patient's complaints (e.g. underlying skin disease, e.g. acne, in DBDD; metabolic diseases in DORs or infections in illness delu-

sions). However, even if such causes do really exist, this does not fully exclude DD. There are many DD cases triggered and maintained by some minor medical conditions or disfigurement. This may be illustrated in the joke by Joseph Heller: "Just because you are paranoid doesn't mean they aren't after you". Thus, the reality or falsity of the belief is not a main diagnostic criteria. It is more important to evaluate the patient's explanatory model that may reveal a delusional system. So, this is not a question of true or false, but how patients selectively choose and interpret facts which support their beliefs and interpret away facts that do not.

Differential diagnosis It includes other neuropsychiatric conditions and somatic illnesses. The symptoms are not due to another disorder or disease attributed to mental, behavioural, or neurodevelopmental disorders that could present with delusions, like schizophrenia, schizoaffective disorder, delirium, dementia, psychotic disorder due to another medical condition and substance/medication-induced psychotic disorder, depressive and bipolar disorders, obsessive-compulsive and related disorders (Table 26.1).

Table 26.1 Differential diagnosis for DD and other psychiatric conditions

Psychiatric diagnoses to be excluded	Features that differ from DD
Schizophrenia	Can be distinguished from DD due to other characteristic symptoms of schizophrenia, including disorders of thinking (e.g. disorganization), perception (e.g. hallucinations), self-experience (e.g. the experience that one's feelings, impulses, thoughts, or behaviour are under the control of an external force), cognition (e.g. impaired attention, verbal memory, and social cognition), volition (e.g. loss of motivation), affect (e.g. blunted emotional expression), and behaviour (e.g. behaviour that appears bizarre or purposeless, unpredictable, or inappropriate emotional responses that interfere with the organization of behaviour).
Depression, bipolar disorders, and schizoaffective disorder	Can be distinguished from DD by the temporal relationship between affective symptoms and delusions that may occur exclusively during mood episodes, e.g. in depressive or bipolar disorder with psychotic features, i.e. if the psychotic symptoms are better explained by an affective episode (depression, mania, or mixed episode).
Delirium or psychotic disorder due to another medical condition	Can be distinguished from DD due to medical history, sudden onset, physical examination, paraclinical or laboratory tests that reveal underlying medical condition with potential physiological influence on the brain. There is a temporal relationship between the decompensation of medical condition and psychosis (they begin and end at the same time), and effective management of the medical condition often reduces the severity of psychotic symptoms.
Substance/ medication-induced psychotic disorder	Can be distinguished from DD by the chronological relationship of substance use to the onset and remission of the delusional beliefs; often transient and single delusional ideas with no signs of developed delusional system; often associated with hallucinations. History of substance use and the nature of the substance being used, supplied with laboratory tests, such as a urine drug screen or a blood alcohol level, may distinguish condition.

(continued)

Table 26.1 (continued)

Psychiatric diagnoses to be excluded	Features that differ from DD
Dementia	Can be distinguished from DD due to other characteristic symptoms of dementia, including gradual, significant cognitive decline from a previous level of performance (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition)
Obsessive-compulsive disorder	Can be distinguished from DD due to preserved insight into the excessiveness and unreasonableness of obsessions and/or compulsions (except cases with poor or no insight) that are intrusive, experienced as distressing and "ego-dystonic" with no signs of delusional system development. In OCD, the patient has intrusive thoughts or imagery, identifies them as senseless, resists them, but resistance causes anxiety.

To finish the discussion of differential diagnoses of other DD of somatic type in dermatology, this is a list of medical conditions to be excluded in DORS:

- Dermatological (hyperhidrosis)
- Genito-urinary (rectal abscess, fistulae)
- Metabolic (trimethylaminuria)
- Otolaryngeal (halitosis)
- Dental (abscess)
- Neurological (temporal lobe epilepsy, arteriovenous malformations, Parkinson's disease)

Treatment

General considerations According to most data, the medications of choice in DD, as in other disorders with prominent delusions, are antipsychotics. The key problem of management in DD is compliance as there is no insight or awareness of a psychiatric origin of symptoms. As a result, it is difficult both to initiate appropriate treatment and maintain long-term adherence. However, there is data that DD may have a good prognosis if appropriate adherence can be achieved.

Pharmacological treatment

The only available RCT meta-analysis in the Cochrane Database (2015) states there is a paucity of high-quality randomized trials on DD, and there is currently insufficient evidence to make evidence-based recommendations for treatments of any type for people with DD. The limited data that were found could not be generalized to the population of people with DD. Thus, it is suggested that until further evidence is found, it seems reasonable to offer treatments which have efficacy in other psychotic disorders. This means that antipsychotics are medications of choice. Olanzapine, risperidone, quetiapine, aripiprazole, ziprasidone, amisulpride, sulpiride and haloperidol are medications most frequently used for

DD. However, there could be a need for augmentation with other psychotropic medications: antidepressants (if depressive symptoms are prominent) and hypnotics (in insomnia cases).

Regarding problems with adherence, long-acting injectable antipsychotics (depot antipsychotics) may be beneficial. Recent studies show that DD patients treated with such long-lasting medications show higher attendance rates for outpatient appointments and a lower rate of prescriptions for other psychotropic drugs. Thus, these drugs are considered as a promising option in the treatment of DD.

The general approach to the pharmacological treatment in DD does not differ greatly from that followed in patients with delusional infestation (see Chap. 12).

Non-pharmacological interventions Data about the efficacy of psychosocial interventions in DD are limited. There is a single RCT, considered to be of a high quality, that compared cognitive-behavioural therapy (CBT) and attention placebo control (APC). It showed a significant posttreatment change in several belief dimensions for both APC and CBT, e.g. the posttreatment decrease in the "strength of belief" parameters was 40% in CBT and 28% in APC. The most significant change in both groups lay in an increased ability to control actions and communications related to the belief. CBT also improved outcomes on depression and self-esteem. However, neither CBT nor ACP succeeded overall in reducing the strength of conviction to zero. Thus, from a practice point of view, psychotherapy, particularly CBT, may be considered as a good adjunct to antipsychotic treatment. Such an approach has been shown to be beneficial in several less good quality trials, most of them compared CBT with standard psychiatric care or treatment as usual. Regarding targets of CBT in delusional beliefs, there is a suggestion that it should be focused not primarily on belief reassurance, but on "allowing the patient a free rein to talk, gradually making the link between external stressors, emotion, and beliefs, leading to a joint exploration of alternative explanations for certain beliefs", i.e. trying to modify the valence of beliefs (that is, the emotion attached to the beliefs) rather than their content.

Discussing the diagnosis and management with a patient Patients with DD are convinced that their beliefs are true. So, as mentioned above, there is no value in arguing with them. However, very often they are open to discussion of their delusional ideas if they feel they have encountered an interested listener. Thus, a non-confrontational and empathic approach is a key not just for making the diagnosis, but also for discussing the diagnosis with the patient. Regarding the patient's ability to understand the diagnosis, it may be primarily announced in general as a "disorder of the nervous system", "generally underinvestigated", "rather rare but empirically proved to be effectively treated by neuroleptics" (see Chap. 12).

Practice Point
As engagement and adherence are the challenging issues in DD management, psychosocial interventions, performed in a non-confrontational manner, should be primarily focused on establishing a positive therapeutic relationship.

Practice Point
As DD is attributed to a primary psychotic disorder and high-quality treatment evidence is limited, it is suggested to use antipsychotics as first-line medications.

Prognosis

Existing prognosis data Primarily, DD has been considered difficult to manage due to high rates of treatment resistance and relatively poor prognosis. A lack of adherence to medication is considered as one of the most common factors associated with a poor response. However, recent retrospective analyses, case series and several trials suggest that DD has a "moderate", "acceptable", and even "good prognosis" if treated adequately. There are even some benign cases with a fast response and full remissions. Among predictors of better response are non-prominent hallucinations consistent with the content of delusions.

Bibliography

González-Rodríguez A, Molina-Andreu O, Imaz Gurrutxaga ML, Catalan Campos R, Bernardo Arroyo M. A descriptive retrospective study of the treatment and outpatient service use in a clinical group of delusional disorder patients. Rev Psiquiatr Salud Ment. 2014;7(2):64–71.

Joseph SM, Siddiqui W. Delusional disorder. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020.

Kulkarni KR. Clinical presentation and course of persistent delusional disorder: data from a tertiary care center in India. Prim Care Companion CNS Disord. 2016;18(1) https://doi.org/10.4088/PCC.15m01883.

Munro A. Delusional disorder: paranoia and related illnesess. Cambridge: Cambridge University Press; 1999. p. 261.

Skelton M, Khokhar WA, Thacker SP. Treatments for delusional disorder. Schizophr Bull. 2015;41(5):1010–2. https://doi.org/10.1093/schbul/sbv080. Epub 2015 Jul 23.

Psychodermatology in Children

27

Susannah Baron, Rukshana Ali, and Benjamin Baig

Paediatric Dermatology is a rewarding and challenging speciality, as it involves managing a child/young person with a skin problem within the context of their parents/carers/family circumstances and psycho-social situation.

It is important to remember that all of these factors can vary in the same child between consultations and also between children suffering from the same condition, e.g. eczema. Mild eczema can have a large psycho-social impact on one child, whilst another child can cope very well despite having severe eczema. A holistic approach with every child and their family is necessary in order to properly assess and address their individual needs.

In this chapter, please read parent/carer/family interchangeably for whoever attends the consultation with the child. An important practice point is to find out early in the consultation what the relationship between the child and accompanying adult is: put this question to the child (if old enough) thus engaging the child at an early stage. Try not to make any assumptions about who is the accompanying adult. Some of the top tips for approaching paediatric patients with psychodermatological disease are in Table 27.1.

As in other areas of psychodermatology, conditions can be classified:

1. Skin conditions that result in psychological morbidity
2. Skin conditions exacerbated by stress/psychosocial factors

S. Baron (✉) · R. Ali
St John's Institute of Dermatology, Guy's and St Thomas' NHS Foundation Trust, London, UK
e-mail: Susannah.Baron@gstt.nhs.uk

B. Baig
Institute of Psychiatry, Psychology and Neuroscience, Kings College London, London, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_27

Table 27.1 Top tips for your consultation

Observe	Non-verbal	Verbal
Watch behaviour/dynamics carefully as you call child/family from the waiting room	Engage with child (age-dependent) before parent so they know the consultation is about them	Ask about a toy/blanket/book/computer game they have brought with them to aid engagement
Watch dynamics as child and family settle into the room	Any clues? • Does the child look happy/miserable/comfortable/clingy/exploring the room • Do the parents look agitated/angry/anxious/unhappy/upset • If very young engage/talk whilst down at their eye level/whilst playing/on parents lap	• Try and talk to the child prior to their parents (age-dependent) • Ask/establish who is with the child • Ask what brought them to the clinic today/how you can help? • Ask the child/parent what they are hoping to get from the consultation (very useful to establish parental expectation early on so you can tailor your consultation accordingly)
• Always introduce/ask permission if you have others in the room with you • Ask permission of child/parent before examining • Only examine a child with a parent/carer present • Children often become embarrassed so let them get undressed behind a curtain with their parent before you come in • Be sensitive to child being embarrassed particularly if opposite sex health professional(s) in room • Examine in parts, e.g. top half first/then redress and bottom half next • Examine young children on parent's lap	• Look at behaviour whilst examining • Are they scratching incessantly as soon as they are undressed? • Do you recognise the physical signs, e.g. eczema or are they atypical and suspicious? • Is the child withdrawn/tearful/anxious/upset? • Does the child have a "laissez faire" attitude/look proud of their lesions? • Is the parental behaviour appropriate?	• Chat whilst examining • Ask questions of child/parent to clarify your history taking • If atypical lesions exist and you suspect dermatitis artefacta/USL examine carefully/observe if lesions are only in areas the child can reach and ask about each lesion/when it came/how long it has been there/what was going on in child's life at the time? • Ask consent from child/parent for medical photographs: atypical lesions/useful for monitoring treatment response or progression, e.g. vitiligo/alopecia

3. Skin conditions due to psychological/psychiatric disturbance/neurodevelopmental disorders

However, within paediatric dermatology, the impact of visible skin disease can be different, dependent on the child's age, and can be particularly challenging for adolescents who may be under many social pressures, not least from social media.

It is also important to consider the possibility of neurodevelopmental disorders such as autism as these may be undiagnosed and can cause sensory differences that lead to particular behaviours, for example, skin picking.

The impact on the child of particular parental mental health issues/beliefs/behaviour needs to be considered and be explored sensitively. This may take time and building rapport over several clinic visits is helpful.

Skin Conditions That Result in Psychological Morbidity

These may be skin conditions that have a great physical effect, e.g. eczema and psoriasis and/or those that are visible and can lead to low self-esteem, e.g. vitiligo and alopecia (Fig. 27.1). There is often an overlap between the physical symptoms and the resulting psychological impact, leading to significant anxiety and depression (Fig. 27.2).

The Children's Dermatology Life Quality Index (CDLQI) *https://www.cardiff.ac.uk/medicine/resources/quality-of-life-questionnaires/childrens-dermatology-life-quality-index*) is useful to assess the impact of the skin condition on a child's life. However, this will not pick up the psychological effects, e.g. anxiety/depression/low self-esteem. Therefore, it is helpful to add a follow-up question, e.g.:

- Q1. How itchy, scratchy, painful has your skin been?
  - How does that make you feel?

It can be helpful to use validated questionnaires to further explore psychological impact (see later in the chapter).

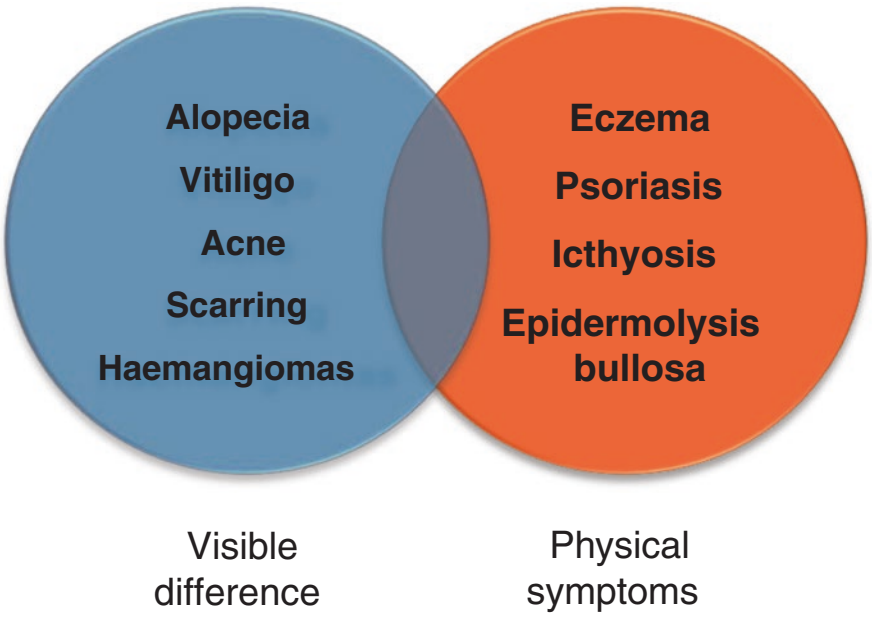


Fig. 27.1 Venn diagram showing the overlap of physical symptoms and visible difference caused by having skin disease in childhood which can lead to psychological impact

Fig. 27.2 Cross-sectional formulation used in cognitive behavioural therapy (CBT) illustrating how skin disease in childhood can cause long-term effects on a child's sense of self and behaviour

Skin Conditions That Are Exacerbated by Stress/ Psychosocial Factors

Stress may flare many skin diseases and in children, stress includes illnesses, change in home/family and school environments, exams, school pressures, e.g. bullying. This can lead to a vicious circle, e.g. a flare-up of eczema will cause increased itching, scratching and sleep loss for the child and their family. This can result in increased child and parental stress if the child/parent is then unable to attend school/work, thus contributing to a vicious cycle of anxiety over lost school work, fear of return to school due to visible skin changes, difficulty in concentrating due to poor sleep pattern and parental stress and anxiety if difficult to return to work.

Skin Conditions Due to Psychological/Psychiatric Disturbance/ Neurodevelopmental Disorders

Attention Deficit and Hyperactivity Disorder (ADHD)

An association between atopic eczema in children and attention deficit (hyperactivity) disorder has been identified. Children with more severe eczema and greater sleep loss have higher odds of having ADD/ADHD and this may be undiagnosed, so be aware of this and suggest a referral for assessment if appropriate.

Trichotillomania/Compulsive Hair Pulling

Children are more likely to pull hair out from their scalp than from other parts of the body but eyebrows and eyelashes are other common sites. In younger children, hair pulling can be unconscious and then become a self-soothing habit. Some children find that pulling hair makes them feel good and is a tension relief. Children can develop rituals around their pulled hair and some may eat it.

Girls are more commonly affected than boys and trichotillomania is more common in teenagers than younger children. Trichotillomania is also more likely to develop in children who have obsessive-compulsive disorder (OCD), or in those have a first degree relative with OCD. Hair pulling can develop in children with anxiety.

As with other psychodermatological conditions, it is important to explore psychosocial factors at the time when the behaviour began/was triggered (Table 27.2).

Treatments

- Reassure the child and family that this behaviour is often used as a coping strategy similar to nail-biting. Discuss that it can be due to emotional distress.
- Explore psycho-social factors (Table 27.2).
- In young children who use hair pulling as a self-soothing mechanism, use distraction methods/introduce a different behaviour instead, e.g. fidget spinner/stress ball, playing with other objects instead of their hair.
- Encourage child to wear a bandana/beanie/hat so access to hair more difficult.
- Cognitive Behaviour Therapy (CBT) can be very helpful, in particular, habit reversal therapy (see later in the chapter).

Table 27.2 Psycho-social discussion areas to include in your consultation

When did the behaviour start?
Age of onset?
Any family stressors, e.g. divorce, moving house?
Any change in school?
Any bullying or friendship issues?
Any exam pressures?
Are there any triggers?
Does it get worse before/during any stressors, e.g. exams?
Is there a place/time it usually happens?
Is it unconscious, i.e. children will pull in front of others?
Is it focused and a hidden activity?
Is there a Personal/FHx?
Are they a worrier?
Is there any personal history of anxiety/in a family member?
Any other OCD rituals/tendencies in child/family member?
Are there times when the behaviour is better?
Does behaviour improve/flare in the school holidays?
Does behaviour lessen if the child involved in enjoyable activities?

- For focused hair pulling associated with significant emotional distress, further evaluation with a psychologist, to explore background and triggers may be needed together with tailored CBT and medication, particularly if there is significant anxiety/depression.

Self-Harm

Self-harm is deliberate injury, typically as a manifestation of psychological distress or psychiatric disorder. "Cutting" of the skin is a frequent presentation of self-harm and is most common in adolescent girls. Cutting typically presents with linear cuts on forearms or other areas of the body, which may leave scars. Cutting may offer a temporary release of anger or tension and can enable feeling in control of difficult feelings, but it can become a habit. Although young people may hide the marks or feel ashamed, they would not present as unexplained skin disease and for this reason, cutting generally does not primarily present to dermatologists. However, self-harm can be an expression of severe emotional distress so it is important to ask about suicidal thoughts (see later in the chapter) and refer to the appropriate mental health team accordingly.

Dermatitis Artefacta/Unexplained Skin Lesions in Children

Dermatitis artefacta (DA) are unexplained skin lesions (USL) presenting in children, often in response to emotional distress and/or psychological need. Unlike deliberate self-harm, the behaviour causing these skin lesions is often denied by the child. Although previous definitions state that DA is performed "deliberately and secretly", this is not always true. In children, this behaviour may occur whilst in a dissociated state (a disruption in all senses, including those in the skin) or subconscious conversion (conversion of mental stress into physical symptoms).

Children themselves may not admit to causing the skin lesions and it is not, on the whole, important that they do. Parents may be extremely worried, and usually do not consider/accept that their child is damaging their skin. Be aware that parents can become angry if this is/has been suggested by health care professionals.

In childhood USL, it is essential to exclude these from skin damage performed by others (abuse or fabricated or induced illness) and from other organic skin diseases. It is important to sensitively explore the current mental health of the child. There may or may not be secondary gain and it is crucial to build up a safe and trusting environment in which to work with the child and their family.

It is common for paediatricians, without full dermatology training, to over-investigate and medicalise children with USL, e.g. with multiple blood tests and skin biopsies. These are generally not necessary but it is always important to treat the child's skin as part of your holistic approach. USL can present with/before/after other unexplained medical symptoms (UMS) in children eg. headaches, fatigue, non-epileptic

seizures, stomach problems, musculo-skeletal pain, so do ask about these symptoms and discuss with the other health professionals involved in the child's care.

Health professionals can confuse USL with self-harm lesions, e.g. cutting but these look very different, fulfill a different emotional need and this behaviour is rarely denied by the child/young person.

If a diagnosis of USL is suspected, early referral to your Paediatric Dermatology team is recommended in order that the child and family can be treated in an MDT, ideally with involvement of a psychologist.

Clues in History:

- Parent/child says lesions just appear (usually overnight or when in school).
- No idea how they happened—"hollow history".
- Parent usually very worried and child can have "laissez faire" (not bothered) attitude/child may seem proud of lesions.
- Lesions may have begun at stressful times, e.g. transition to secondary school, exams.
- Previous medically unexplained symptoms, e.g. headaches, abdominal pain.
- Known history of mental health problems, for example support for anxiety or depression.
- Contact with an illness role model, e.g. parent.
- Previous secondary gain with illness, e.g. more time with parent/off school.

- *Do not try and "catch the child out".*
- *Do not initially confront the child and parent, but just gently take a history from both.*
- *In time, having built a supportive therapeutic relationship, encourage an open and honest discussion about the behaviour and need for psychological support.*

Clues in Examination:

- USL is atypical and only in areas that the child can reach.
- If lesions are in areas where the child cannot reach, this is a warning sign for possible abuse.
- Lesions are atypical and vary in shape/size and their morphology is dependent on how they have been made:
  - Aerosols held close to the skin can produce blisters and erosions/can get trickle marks making them linear.
  - Suction blisters by child.
  - Burns made by chemicals/lighters/cigarettes/inhalers.
  - Cuts/bruises/purpura made by finger nails/ligatures/sharp and blunt objects.
  - Young children may draw on their skin in pen, nail varnish.
  - Glue can be used on skin and lips.
- Lesions can be in different stages from acute to healed, e.g. blistered, eroded, scabbed, post-inflammatory change (Figs. 27.3, 27.4, 27.5 and 27.6).

Fig. 27.3 Multiple ecchymotic lesions on arm, all of a similar size and shape, in a child with USL

Fig. 27.4 Multiple lesions on the backs of both hands all of similar size and shape in different stages of healing

Fig. 27.5 Linear excoriations all of the same size and shape

Fig. 27.6 Multiple healing areas on lower face showing post-inflammatory erythema

Investigations

Unless an organic skin disease is suspected, keep investigations to a minimum:

- Skin swab if possible infection.
- Desquamation can be peeled off and sent for analysis to show glue/chemicals.

Try to avoid a skin biopsy, as this will leave a scar and medicalise and reinforce the behaviour and support a belief in an underlying cause.

Aetiology

The important question is not *how* the skin lesions are appearing but *why* (Table 27.3). USL in children has a spectrum of underlying causes from simple experimentation to a physical defence/self-mutilation due to underlying physical/emotional/sexual abuse. USL may be the best or only way of expressing this need in some children and maybe done to some degree subconsciously. The prognosis, therefore, is related to the underlying psychological/emotional need, the child's resilience and their current psycho-social environment.

Treatment

- Recognise that children with USL are likely to have an emotional or psychological need and establish a supportive and therapeutic space to address this.
- Avoid direct confrontation with the child/parents. You do not need to "catch the child out".
- If the child appears to fall within the experimental/recreational category (above) initiate a discussion, conveying non-verbally that you know what is happening, maintaining your eye contact with the child, using statements such as:

Table 27.3 Simple classification of DA/USL in children and young people. *Reproduced (with minor adaptations) by kind permission of Dr Celia Moss (2014)*

Nature of DA/USL	Mental state	Emotional need/ secondary gain	Can gain support/ help from	Prognosis
Experimental	Normal	Satisfies curiosity	None required	Good
Recreational	Normal	Peer/social acceptance/gets a buzz	School, youth group	Good
Coping strategy	Withdrawn/ anxious/ worried	Situational avoidance/time/ sympathy/way of expressing emotional distress	School counselling, GP counselling, referral to CAMHS if threshold reached, psychologist if available	Recurrent/chronic depending on situation and resilience of child
Physical defence	Abuse	Defers abuser, cry for help	Local child protection services, psychology, social services	Chronic but treatable if help available
Mutilating	Self-loathing	Self-punishment/way of expressing emotional distress	Psychology, Psychiatry, MDT	Serious with risk of suicide

I have seen this many times before in children your age and it gets better.

Stress can often come out in the skin as the mind and the skin are closely interlinked.

Your skin is like the tentacles of your brain and skin can sometimes show if you are struggling or unhappy.

Sometimes, when we are stressed, we damage our skin without realising, perhaps when we are asleep.

- This can provide an exit strategy for a child without "losing face".
- Children and young people may welcome the association of stress or unhappiness showing in the skin and therefore open to psychological support.
- Parents will often be aware that stress and skin can be linked, and thus will often welcome psychological support for their child.
- Remember the child has been brought to your clinic for a skin condition so always provide treatment for the skin.
- Prescribe antimicrobial washes in the bath/shower and a moisturiser to "soothe/settle" the skin.
- If USL is used as a coping strategy (Table 27.3) and there is significant anxiety/depression assess using tools discussed later in the chapter. If there is significant school avoidance or other psycho-social impacts it can be helpful to liaise with the school to see if they can offer counselling and support.
- If you have a liaison psychologist/local paediatric psychologist, referral can be very helpful.
- If you suspect more serious underlying issues, e.g. forms of abuse, self-mutilation, a more extended history, taken from the child alone if possible, is indicated.
- Assess suicide risk if appropriate and refer immediately to the crisis team.
- The Internet is accessible with a great deal of medical information, so be honest yet non-judgemental in verbal and written communications (one reason why the term USL is preferable to DA).

The following are helpful techniques in making a full assessment:

- Taking the child out of the room to be weighed/measured allowing you to talk to the parents or child individually.
- Seeing the child first with parents and then asking to see the child alone (making sure you have another health care professional in the room).
- Reassure the child that your conversation is confidential, only to be disclosed if they/someone else is at risk of serious harm.
- Using open-ended questions.
- If there is a psychologist in the clinic/department introducing them to the family with a view to an immediate/urgent consultation.

- Taking advice from or referring to your local child protection team/social services/crisis team (if suicidal).
- Referring to paediatric liaison psychiatry team via local referral pathway.
- Telephoning child's GP to discuss if there are known concerns.
- Following up with GP, child protection, social services in case of subsequent DNA appointments.

Summary

USL in children presents to dermatologists and paediatricians. Unlike in adults it usually has a good prognosis, dependent on the underlying psychological/emotional cause, the acceptance of psychological support, if appropriate, and the support and insight of the parents.

It is essential to make your consultation a safe and non-judgemental environment, building trust between child, parents and clinician. Sensitively exploring the underlying and possible associated psycho-social triggers in a holistic way, is the key to success.

If USL does not resolve after 1–2 visits an MDT referral for more specialist intervention, e.g. clinical psychologist, psychiatrist, psychodermatology clinic is advised and the child/family will be more likely to accept this if it is local and within the department/hospital.

Skin Picking and Self-Injury

Children with neurodevelopmental disorders, e.g. autism and learning difficulties may present with skin picking, biting, rubbing, or other skin self-injury which can be difficult to manage. This can be due to an altered skin sensation, which makes it an enjoyable sensation for the child to explore/self-stimulate or as a method of self-soothing.

If a child is not known to have a neurodevelopmental diagnosis then screening questionnaires, e.g. Strengths and Difficulties questionnaire (see below) can be helpful. If concerned, refer to local community paediatric service for further assessment.

Skin picking/rubbing/scratching can be used as a coping strategy and this often worsens in situations that the child finds stressful.

It is important to take a careful history and examination and discuss psychological interventions as appropriate. If the areas are limited, occluding with a moisturiser under occlusive dressing or paste bandaging for 1–2 weeks can be very helpful in conjunction with habit reversal and a reward chart. Encourage the child by discussing how they can choose their own distraction toy, e.g. a fidget spinner, stress ball. Children often enjoy making their own reward charts, choosing their stickers and rewards, e.g. going to the cinema.

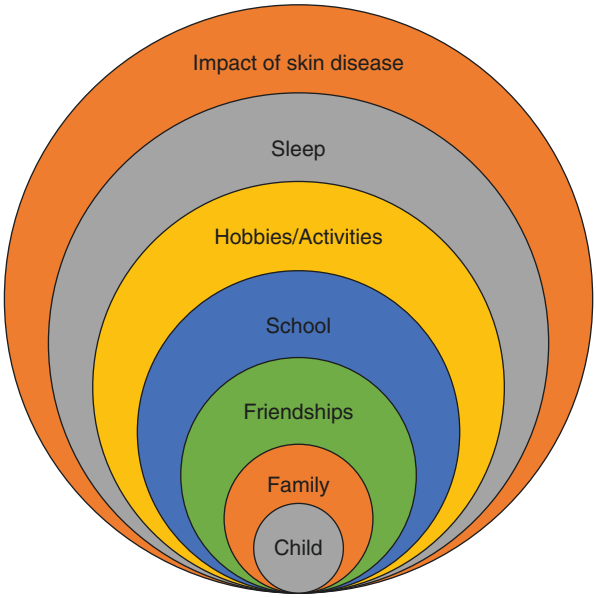


Fig. 27.7 The impact of skin disease on the varying aspects of a child's life

Psychological Assessment in Paediatric Dermatology

A psychological assessment enables clinical psychologists to understand what challenges the child is experiencing and how they make sense of their situation.

A comprehensive psychological assessment should consider the impact of the skin disease on varying aspects of the child's life, including how the family are affected, as well as how the child is coping in their everyday life. Figure 27.7 highlights the differing areas that should be considered when seeing a child with psychological distress and the reciprocal relationship each aspect has with the child.

A comprehensive psychological assessment conducted by a clinical psychologist should examine these areas in more depth including thoughts, beliefs and feelings around each. It is important to remember that children are not autonomous and exist within a wider system, so considering the perspectives of parents and school may be beneficial. Examples of the types of questions a psychologist may ask are highlighted in Table 27.4.

Standardised Measures

In addition to a clinical interview, standardised measures may be beneficial in examining the psychological impact of skin disease. These can then help to explore mood issues within a clinical setting. Useful measures include the Revised Children's Anxiety and Depression Scale (RCADS), Paediatric Index of Emotional Distress (PI-ED), and the Mood and Feelings Questionnaire (MFQ). Some tests may incur a cost for administration so please refer to the publisher's website.

Table 27.4 Areas to include in a paediatric psychology assessment

Views around the problem
• What is the main issue they wish to focus on? (Does this fit in with the referral?)
• What's been the journey of the problem; how long has it been going on for? (A timeline of events can be helpful)
• What impact does the skin disease have in their life? (Also think about impact on family, friends, school and hobbies etc)
• How do other people react to the skin disease?
• When is the skin disease most/least bothersome?
• What thoughts and feelings does the child (or family) have about the skin disease?
Ways of coping
• What things have they tried to do to cope with the issue?
• What's been helpful/unhelpful?
• Who notices when the child is struggling with their skin condition? What do they do?
• What needs to change to in order to help the child/family?
• Any current or previous experiences of psychological therapy?
• Any other professionals involved (e.g. social work)?
Family history and early experiences
• Gain information around pregnancy, early development and milestones (if applicable)
• Genogram of the family
Educational history
• Which school does the child attend and what year are they in?
• What have been the experiences at school?
• Is the child meeting their academic attainments?
• Have there been any experiences of bullying or teasing?
• What is the impact of skin disease on school and learning?
• What support is available in school? Is it helpful?
Hobbies and friendships
• What things does the child like to do in their spare time?
• What do their friends think about their skin disease?
• Does their skin disease ever get in the way of activities (e.g. swimming)?
Beliefs around skin disease and treatment
• What thoughts or beliefs does the child/family have about the skin disease?
• How have the family managed with the condition and treatment?
• Have there been any side effects from treatment?
• Examine adherence to treatment; how many times are the medication/creams given? Who manages them? How do they remember to take the treatment? How often do they forget?
• Explore beliefs around treatment
• Experiences of health teams, hospitals and doctors
• Any signs of procedural anxiety (e.g. needle phobia)? Current or in the past?
Hopes and expectations
• What are their goals from psychological therapy?
• What do they expect?
• What needs to happen to achieve that goal or expectation?
Initial plan for psychological intervention
• How many sessions?

A comprehensive list of psychological outcome measures can be found on the Child Outcomes Research Consortium (CORC): https://www.corc.uk.net/outcome-experience-measures/

A psychological screening measure should not, however, replace a comprehensive assessment by a trained clinical psychologist.

It is also helpful to obtain quality of life measures such as the Children's Dermatology Quality of Life Index (CDQLI) as it can be the case that despite a child having a good quality of life, their mood may be poor, which is indicative of other factors being present in a child's life.

Psychological Risk Assessment

As an integral part of a psychological assessment, it is imperative to evaluate risk. Sometimes children with skin disease will say that they no longer wish to live or their parents will highlight concerns around their risk. It may even be that the outcome measures highlight that a child is experiencing significantly low mood.

When exploring any potential risk issues, you should ensure that the child is able to have timely access to specialist mental health support either through an in-house psychologist, access to liaison psychiatry or Child and Adolescent Mental Health Service (CAMHS).

An open question like the one in Table 27.5 can create space for a conversation about any potential risk.

Table 27.5 Psychological risk assessment questions

"When children struggle with skin disease, they may think about 'not wanting to be here'. Have you ever had thoughts about this?"
If the answer is yes or there is some ambivalence, the follow up questions below may be helpful:
Do these thoughts come and go or are they intense?
Has the young person ever made any plans to harm themselves? (e.g. collecting sharpener blades, stockpiling medication).
Have there been any previous attempts at harming themselves?
What would stop them from acting on these thoughts? What are their protective factors?
Have they ever spoken to anyone else about these thoughts?
Does the young person require immediate action to keep safe?—If 'Yes', you should speak immediately to your hospital's liaison psychiatry team or if the young person is known to CAM HS alert their local CAMHS team.

Referral to Child and Adolescent Mental Health Service (CAMHS)

Due to the lack of psychology provision within paediatric dermatology teams, it can be difficult to access appropriate mental health in a timely manner. Access to a CAMHS team is dependent on the area in which the child's GP is. Searching CAMHS services using the GP's postcode will find the nearest service for the child.

Most CAMHS services accept referrals from health professionals or the GP; however, some have specific referral forms that need to be completed or a Single Point of Access (SPA), where the referral is discussed with a mental health professional on duty. Contacting the CAMHS number on the website will enable the professional to find out the best way of referring to that service.

When referring to CAMHS, include scores of standardised measures, as well as your current concerns, recent stressors and with any risk the child is presenting.

Children and their families should always be made aware of where to access support out of hours, e.g. GP, A&E, or ChildLine.

Other Areas of Support

Due to the variability in CAMHS service delivery across the UK, it can also be helpful to signpost the child and family to local support groups for the skin disease.

In terms of mental health support, children and their families can also access charities such as ChildLine (https://www.childline.org.uk/ or 0800 1111) or Young Minds (https://youngminds.org.uk/).

Psychological Interventions for Paediatric Skin Disease

There are various ways a clinical psychologist can support a child or family with managing their skin disease.

Most commonly, Habit Reversal Therapy is used to support the child who struggles with feeling itchy and scratching. The main components of habit reversal include optimising emollients and topical steroids use, increasing awareness of frequency of scratching and then replacing the scratching behaviour using techniques e.g. fist clenching, pinching of the skin or using a finger to place pressure on the itchy skin. The protocol has been adapted for younger children as well (8 years old and below). The full protocol can be found on: https://www.atopicskindisease.com/

Other psychological therapies applied in paediatric dermatology include Cognitive Behavioural Therapy (CBT), Acceptance Commitment Therapy (ACT) and Mindfulness. These can be applied individually or in groups.

CBT has a good evidence base for a range of psychological problems. CBT involves helping the child to examine unhelpful thoughts and beliefs and see how they impact on physical symptoms, emotions, and behaviours. In CBT unhelpful thoughts and beliefs are challenged by examining the "evidence for" and "evidence

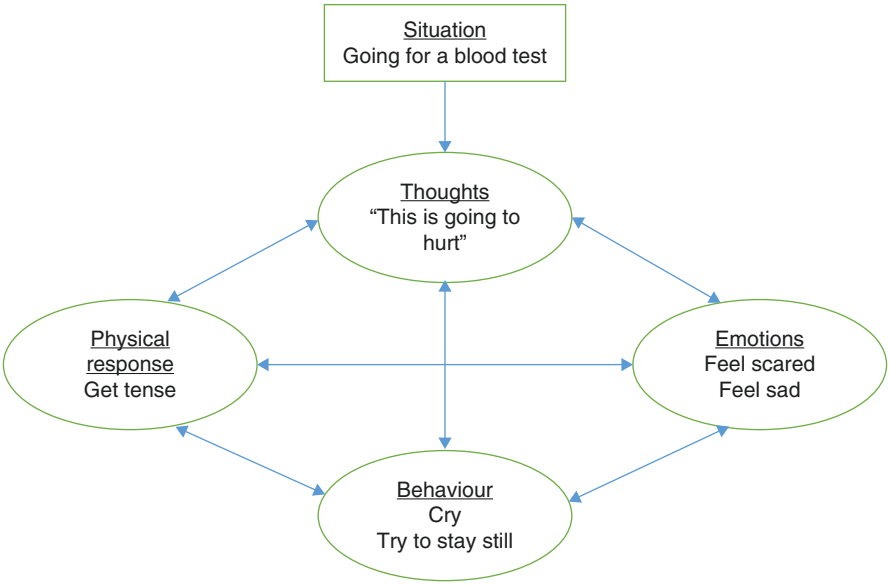


Fig. 27.8 Example of CBT cycle

against" the thought. Education is also provided explaining the role of physical responses and emotions (e.g. "fight or flight") and how to manage them using relaxation or mindfulness. The child is also introduced to the idea that it is the interpretation of a situation that results in certain thoughts or feelings and behaviours. Drawing out the cycle for children can show the links between the various areas and how addressing one area, can help another. An example of CBT and the vicious cycle is highlighted in Fig. 27.8.

A full explanation on CBT and ways to access CBT can be found on https://www.babcp.com.

In ACT, the way a child responds to their skin disease is linked to things that give the child meaning in the life (values). Values can be identified by asking the child what is important to them. The main aim of ACT is to support the child to stop struggling against their thoughts and feelings and teach them skills to manage these (e.g. using mindfulness). A further aspect of ACT is to ensure that actions are consistent with their values and to recognise when behaviours take the child away from their values. ACT has been found to be helpful in supporting people with long-term health conditions.

Mindfulness can be used in both CBT and ACT or used a stand-alone psychological intervention. Mindfulness is a way to bring your attention to the present moment. It is a way in which children can let their thoughts or feelings go past without engaging in them (e.g. like watching them float by on bubbles). There are various ways of applying mindfulness including mindfulness meditation, mindful eating, and mindful colouring. There are many available smartphone apps on mindfulness as well as examples of mindfulness on the Internet.

It is important to ensure that the psychological therapy also includes the parents or carers, as they can support interventions outside of the therapy room.

Psychopharmacology in Paediatric Dermatology

There is a good evidence base for the pharmacological treatment of mental health conditions in children. Medications may be used both instead of and alongside psychological treatments. Use of medications can be shown to significantly improve quality of life, treat conditions such as anxiety and depression and reduce the risk of self-harming and suicide. In this section, we will consider the use of pharmacology for common comorbid mental health conditions. The prescribing of psychotropic medication in children and adolescents should be restricted to specialists in Child and Adolescent Mental Health. Dermatologists treating children whom they think would benefit from psychotropic medication should refer their patient to Child and Adolescent Mental Health Services (CAMHS).

Depression

NICE guidelines for the management of depression in children and adolescents recommend the use of antidepressant medication for cases of moderate to severe depression. The best evidence supports the use of Selective Serotonin Reuptake Inhibitors (SSRIs); Fluoxetine (commencing 10–20 mg) or Sertraline (commencing 25–50 mg). Some evidence also exists for use of Citalopram (commencing 10–20 mg). The strongest evidence supports combined CBT and antidepressant for treatment for moderate to severe depression. The main cautions for use of these medications include: a history of bipolar disorder, haemophilia, epilepsy, and glaucoma. Dermatological side effects are very rarely seen in the commencement of an SSRI. Patients should be warned that they may experience heightened anxiety, suicidal thoughts, and agitation in the first 2 weeks of commencing an SSRI but that these effects will subside. An SSRI treatment, when there is a good response, should continue for up to 6 months.

Anxiety

Children may suffer from a number of anxiety conditions including separation anxiety, social anxiety, and generalised anxiety disorder. The primary treatments for these will include CBT and family therapy. In children who do not respond to these considerations, consider the use of an SSRI medication. As above, the same doses, side effects and treatment length can be considered.

Psychosis

Ekbom's syndrome is a psychotic illness which may be seen in dermatology clinics. Patients under 18 may show early evidence of psychotic disorders. Good evidence exists which supports the treatment of psychotic conditions in children and adolescents. The main pharmacological action is dopamine receptor blockade or partial agonism. The main agents commonly prescribed for psychosis in young people can be seen below:

- *Risperidone*—commence dose from 0.5 mg and increase to 6–8 mg
- *Aripiprazole*—commence 2 mg and increase to 5 mg then 10–15 mg
- *Olanzapine*—commence 2.5 mg and increase to 20 mg

The main side effects of antipsychotic medications include extrapyramidal side effects such as dystonia, parkinsonism, akathisia, and tardive dyskinesia. Particular concern exists around metabolic side effects such as weight gain and development of Type 2 diabetes. Arrhythmias may also be side effects. It is suggested that patients on these medications have baseline blood tests and six-monthly measurements of blood glucose, ECG, and weight. Up to 5% of patients taking antipsychotics have been shown to develop a cutaneous rash—exanthematous eruptions, skin pigmentary changes, photosensitivity, urticaria, and pruritus are the most common.

Attention Deficit Disorder (ADD)

Recent evidence has shown an increased rate of ADD in children with atopy, including eczema and asthma. While a paediatric dermatologist may not have the primary role of diagnosing this condition, the more frequent presence of the condition and challenges in managing comorbidity may be important. Patients with ADD are likely (and effectively) to be treated with psychostimulants (Methylphenidate and Dexamphetamine)—side effects include urticaria, exfoliative dermatitis, scalp hair loss, erythema multiforme rash, and Atomoxetine—side effects include pruritus and urticaria in 2%.

Acknowledgment We would like to thank Dr Tess McPherson for her contributions to this chapter.

Bibliography

Clinical guideline [CG28]. Depression in children and young people: identification and management. 2005 September. Last updated: September 2017.

Mohandas P, Ravenscroft JC, Bewley A. Dermatitis aretefacta in childhood and adolescence: a spectrum of disease. Italian J Dermatol Venereol. 2018;153:1–10.

Moss C. Dermatitis artefacta in children and adolescents. Paediatr Child Health. 2014;25(2):84–9.

Warnock JK, Morris DW. Adverse cutaneous reactions to antipsychotics. Am J Clin Dermatol. 2002;3(9):629–36.

Habit Reversal Therapy

28

Reena Shah

Background

In Atopic Dermatitis (AD), the hallmark features are the sensation of itch and the behaviour of scratching. AD is perpetuated by chronic scratching and can flare with stress through psycho-neuro-immunological mechanisms. Habit reversal therapy (HrT) was originally developed to help people with AD. A useful manual and website have been developed to help clinicians and patients (www.atopicskindisease.com) and a full overview of the therapy has been described previously (Bridgett 2014). HrT has been used successfully by all clinicians (dermatologists, nurses and psychologists) to decrease the frequency of scratching in patients with itch-related dermatoses.

Hrt is based on the theory of behaviour modification, which is an approach to change a negative behaviour or to modify an emotional behaviour, to develop and strengthen the new behaviour and to be able to maintain this new behaviour over time. The aim of the therapy is to reduce scratching (therefore the damage to the skin) and to let the skin heal. A positive outcome can consequently reduce feelings of stress, that have been caused by living with the condition. Certain additions to the therapy, such as stress management strategies can also be useful. Further studies demonstrated that those who received medical treatment in addition to HrT, when compared to patients who received medical treatment alone, had greater improvement in their skin status and a greater reduction of scratching behaviour (Noren 1995; Noren and Melin 1989). HrT was originally developed for 1:1 work, however, through clinical experience, facilitating HrT to groups of patients has been successful. Furthermore, offering the therapy to patients with acne excoriee and dermatillomania has also shown positive outcomes.

The model that underpins the therapy (see Fig. 28.1) highlights the relationships between eczema, dry skin, the sensation of itch and the behaviour of scratching. As

R. Shah (✉)
Central and North West London NHS Foundation Trust, University of Hertfordshire, Hatfield, UK

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_28

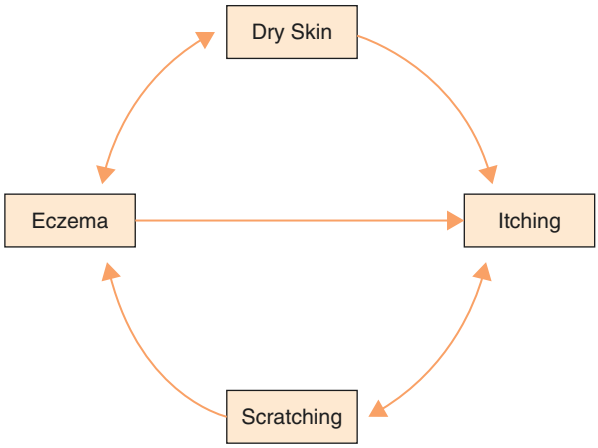


Fig. 28.1 Itch–scratch cycle

one scratches, this exacerbates the sensation of itch and maintains the eczema. Breaking this vicious cycle is key to the process of therapy. The therapy helps the patient to enhance their knowledge of eczema, improve their adherence of the prescribed skin regimen (i.e. emollients and steroids) and to stop scratching; together called the 'Combined Approach'.

Unfortunately, there are a number of vicious cycles that perpetuate eczema and stress. For example, worsening eczema can cause further stress for the patient, who then tend to scratch more and further worsen the dermatitis (through habitual and emotional scratching). Stress may be lessened with cognitive behavioural methods. However, it has been shown that integrating methods such as offering HrT with stress management techniques is effective in helping the individual with the sensation of itch as well as the high level of stress (Shah and Bewley 2014).

Motivation and Success; What Influences Therapy?

Before HrT is discussed with the patient, a thorough assessment is required. It is useful to ascertain the patient's level of motivation, whether any adaptations are required to meet the patient's needs and to understand and explore the biopsychosocial factors that could affect the therapy process. At both stages, pre- and post-therapy, measures of change should also be used; such as the Dermatology Life Quality Index (DLQI, Finlay and Khan, 1994), Severity Scoring of Atopic Dermatitis (SCORAD), rate of scratching scores used at every session and appropriate psychological assessments (such as the Patient Health Questionnaire (PHQ-9, Kroenke et al. 2001) and a brief measure for general anxiety disorder (GAD-7, Spitzer et al. 2006) or the Hospital Anxiety and Depression Scale (HADS, Zigmond and Snaith 1983).

There are two types of scratching: habitual and emotional scratching. Emotional scratching is when unconsciously one scratches to relieve psychological distress rather than to relieve an itch. A thorough assessment can establish whether the patient exhibits one or both types of scratching and can determine which treatment is most effective.

HrT has been shown to be very successful for habitual scratching, but for emotional scratching other strategies are required to work on the psychological distress or stress.

Motivation is a key factor for behaviour change and positive treatment outcomes. Approximately 80% of patients who finish HrT are successful in stopping scratching and letting their skin heal which has a positive effect on their mental well-being. However, 20% of patients with poor treatment outcomes were found to have low motivation to change and poor adherence to treatment. Therefore, thoroughly assessing motivation to change and commitment to treatment is essential. Various factors can explain this: patients need to be motivated to change and have the mental space and time to do so. Motivation to change needs to be established by discussing the pros and cons of change and exploring their commitment to change and the process of change.

Other factors that affect the success of therapy are the fear of using steroids or conducting a psychological therapy at home, due to the stigma of steroid use and mental health. HrT is time-consuming and therefore needs the dedication to complete the therapy and to maintain the positive outcomes. Given the known link between psychological distress, stress and flare-ups, being able to manage psychological difficulties can also affect the level of success of the therapy. Understanding and managing the patients' expectations of therapy and of the therapist is helpful to enhance the success of therapy. Exploring what patients expect as the outcome and what they want clinicians to do is key, as well as the patients understanding the amount of work that is required during the therapy process.

During therapy, there are many factors that can influence the process of therapy and hence the outcome. The patient's attitude towards their skin condition, the level of acceptance of it, the idea of having to manage the condition rather than wanting to cure it and whether the patient understands the role of the skin condition all play a part. In some cases, the skin condition is the centre to the person's identity. It is useful to explore whether the patient has considered how their life might be different without the skin condition. The patient's attitude towards the therapist will also affect the therapy process, it is important to explore and manage these expectations.

It is helpful to establish and explore lifestyle factors as these can influence therapy outcomes. For example, high-stress jobs, active social lives, family and social situations, poor support networks and relationship problems can be consuming and leave little time for a demanding treatment. Understanding what a patient can realistically commit to can help to make relevant adaptations to treatment, therefore increasing positive outcomes.

Increasing Adherence

Adherence to topical medications is poor (Patel et al. 2017) with rates being as low as 32% over 8 weeks in AD (Krejci-Manwaring et al. 2007). This was examined using electronic monitors in the containers; however, the patient/parent perspective was not recorded. Helping patients to increase adherence is key to successfully managing the eczema. 'Anchoring', a psychological technique can help encourage patients to use creams and take medications. Lewis and Feldman (2017) has included this into a Pyramid model of using different approaches to increasing patient adherence (Fig. 28.2).

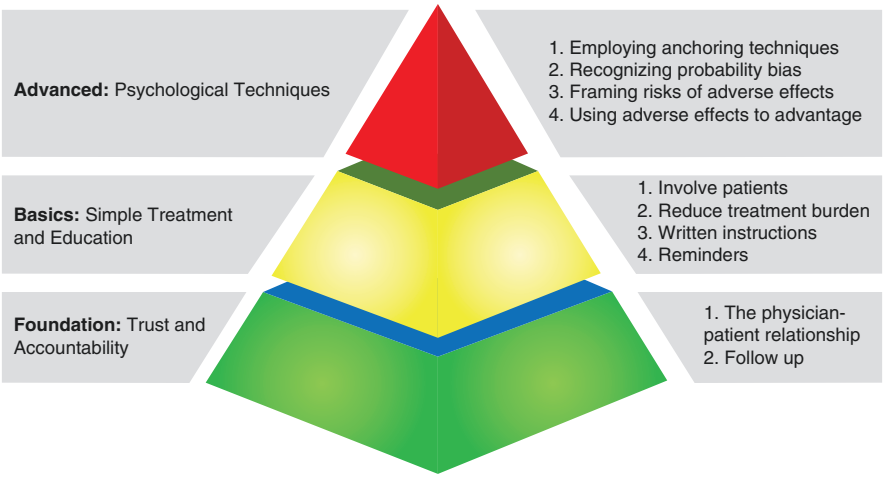


Fig. 28.2 Approaches to patient adherence (Lewis and Feldman 2017)

The anchoring effect is a cognitive bias; it is the tendency to rely heavily on the initial piece of information offered. During decision-making, anchoring occurs when individuals use this information to make subsequent judgements, hence patients making judgments about treatments. Therefore, when patients are given information about creams and medications, how the details are discussed can affect adherence. Many patients with AD express concerns about using standard treatments (especially steroid creams) due to potential side effects and many predominately focus on the rare adverse events. It can be helpful to re-frame these discussions about side effects. Lewis and Feldman (2017) explains this in detail and suggests to replace the phase '1 in 1000 patients get problem X' with the much more reassuring '999 of 1000 do not experience problem X'. In some cases, patients discontinue creams due to stinging or burning. However, reframing this by explaining that stinging is a sign that the cream is working can increase adherence.

Suggested Adaptations to Increase Adherence and Positive Treatment Outcomes

As explained above, often it can be helpful to adapt an average assessment for HrT and include a number of specific questions. During the process of therapy, one can use various skills and approaches to increase the success of the outcome. Below is a list of potential factors to consider and/or ask the patient which have been drawn from practice-based evidence.

During the assessment:

- Patient's expectations of therapy and self: level of acceptance of the skin condition; what does the patient realistically hope to achieve?

- Level of motivation—readiness for change.
- What are the triggers for scratching? Ascertain details of habitual versus emotional scratching.
- Lifestyle issues.
- Underlying issues, such as sexual abuse, trauma, identity factors.
- Relationships; social and personal networks. Those with stronger support networks correlate with better treatment outcomes. Exploring how new support networks can be accessed can be helpful.
- Their experience and severity of psychological distress and the impact on the skin condition/s. Highlighting the mind and skin link.
- If doing Hrt groups, then to assess the patient's personality as one is required to manage different personalities and complex dynamics in group work.

In therapy:

- Psycho-education about habit formation (12 weeks of consistency is required to change/stop habit); providing a range of accessible information on different creams and steroids.
- Collaboratively develop a skincare regimen in detail which is reviewed in each session.
- Provide basic written information for the patient to take away to support advice given at assessment. It can be useful to review and reinforce basic information in sessions:
  - For example how to use the cream/steroids
  - Certain basic strategies—such as having a cool bedroom, wearing 100% cotton clothes/bed sheets and using soap substitute
- Reinforcing that the process of finding the right skin care regimen (of emollients and steroids) and helpful strategies is 'trial and error' approach, as what may work for one person may not work for another person.
- Offering other strategies to the client, e.g:
  - Applying moisturiser when itchy and decanting the cream into small tubs in order to carry around with them; having cream in different areas in the house, in the car and/or in the office.
  - Using post-it notes for reminders of routine to increase adherence at times of stress or when busy.
  - Providing a range of distraction and stress management strategies, e.g. using a stress ball, doodling, knitting, in order to keep their hands busy.
  - Offer specific stress management techniques: relaxation and mindfulness strategies. For example, if pinching and clenching fists is not enough then additional deep breathing may help to distract from the itch. This is particularly important to those with emotional scratching.
- Explore type of strategies according to trigger and time—e.g. if scratching occurs when watching TV have distraction activities prepared and to hand (i.e. knitting, doodling).

- Giving the patient a minimum of 20–30 minutes per consultation to increase a positive outcome:
  - Discuss the positives and negatives of progress
  - Understanding why they may have had a bad week/scratched more and reviewing this and changing/adapting strategies if required
- Developing a relapse prevention plan towards the end of therapy. Reviewing what their triggers are, what has been helpful and unhelpful.
- Throughout sessions encourage the discussion of helpful strategies and establish and explore the patient's own strategies/ideas, as often when they come up with the strategy, they are more likely to do it.
- If facilitating group therapy, then to consider group dynamics.

Conclusion

HrT can be facilitated by non-psychologists, i.e. nurses, dermatologists and general practitioners. Depending on the individual or the group and the outcome of the assessment, clinicians can increase adherence, motivation and success in therapy by considering various adaptations as discussed. It is also key to assess and problem-solve difficulties as they arise and to consistently review the content and outcome of each session.

References

Bridgett C. Habit reversal therapy: a behavioural approach to atopic eczema and other skin conditions. In: Bewley A, Taylor R, editors. Practical psychodermatology. Chichester: Wiley; 2014.

Finlay AY, Khan GK. Dermatology Life Quality Index (DLQI): a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994;19:210–6.

Krejci-Manwaring J, Tusa MG, Carroll C, Camacho F, Kaur M, Carr D, et al. Stealth monitoring of adherence to topical medication: adherence is very poor in children with atopic dermatitis. J Am Acad Dermatol. 2007;56(2):211–6.

Kroenke K, Spintzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13.

Lewis JD, Feldman SR. Practical ways to improve patient adherence. Columbia, SC: Create Space Independent Publishing Platform; 2017.

Noren P. Habit reversal: a turning point in the treatment of atopic dermatitis. Clin Exp Dermatol. 1995;20:2–5.

Noren P, Melin L. The effect of combined topical steroids and habit-reversal treatment in patients with atopic dermatitis. Br J Dermatol. 1989;121:359–66.

Patel NU, D'Ambra V, Feldman SR. Increasing adherence with topical agents for atopic dermatitis. Am J Clin Dermatol. 2017;18:323–32.

Severity Scoring of Atopic Dermatitis. The SCORAD index. Consensus report of the European Task Force on Atopic Dermatitis. Dermatology. 1993;186(1):23–31.

Shah R, Bewley A. The importance of integrated psychological interventions and dedicated psychologists in dermatology. Clin Exp Dermatol. 2014;39:428–30.

Spitzer RL, Kroenke K, Williams JB, et al. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166:1092–7.

Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67(6):361–70.

Quality of Life

29

Kirsty E. Smith and Alia Ahmed

Introduction

The World Health Organization (WHO) defines the quality of life (QoL) as '*an individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns. It is a broad ranging concept affected in a complex way by the person's physical health, psychological state, personal beliefs, social relationships and their relationship to salient features of their environment*' (https://www.who.int/health-info/survey/whoqol-qualityoflife/en/). QoL in dermatology is becoming increasingly important, especially as the severity of the impact is now used as a criterion for access to specialised medications (e.g. high-cost drugs, biologics). In addition, QoL is a useful parameter to measure during treatment to monitor success.

Dermatological disease is associated with a high psychosocial burden, which inevitably impacts QoL. Outcome measures for QoL in dermatology patients should ideally consider the condition, age of the patient and psychosocial impact (including impact on families). There are several validated tools available for measuring QoL, both generic (i.e. across specialties) and disease/condition-specific in dermatology. It remains unclear which instruments are preferred. Standardising the use of outcome measures is important to allow comparisons between studies on QoL in dermatology.

Psychological/psychiatric co-morbidities are diagnoses that occur alongside the primary diagnosis for which the patient is referred (e.g. anxiety, low mood, depression), and can also impact the quality of life for patients, their families/carers.

K. E. Smith
King Edward VII Hospital, Frimley Health NHS Foundation Trust, Frimley, UK

A. Ahmed (✉)
King Edward VII Hospital, Frimley Health Foundation Trust, Windsor, UK

Royal London Hospital, Barts Health NHS Trust, London, UK
e-mail: dr.alia.ahmed@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_29

QoL in dermatology is assessed using generic, dermatology-specific and/or disease-specific measures. Table 29.1 lists commonly used generic QOL measures. Generic measures assess QoL for any condition, they are useful to compare QoL outcomes for people across specialties (e.g. impact on QoL of psoriasis versus asthma). Table 29.2 lists commonly used dermatology-specific QOL measures. Measures specific to dermatology (or dermatology-specific measures) can be used across any dermatological condition and allow comparisons to made between them; however, they do not consider disease-specific issues that are important to patients (Prinsen et al. 2013). The most widely used dermatology-specific QoL tool is the Dermatology Life Quality Index (DLQI) (Finlay and Khan 1994); other measures are the Dermatology Quality of Life Scales (DQOLS) (Morgan et al. 1997), Dermatology-Specific Quality of Life Instrument (Anderson and Rajagopalan 1997), Skindex-29 (Chren et al. 1997a) and 17 (Nijsten et al. 2006). Table 29.3 lists commonly used disease-specific Quality of Life measures. Disease-specific tools measure the impact on QoL for specific conditions in dermatology (e.g. psoriasis, vitiligo, atopic dermatitis) and take into account disease-specific characteristics.

There is a lack of consensus on the use of QoL measures in dermatology (Prinsen et al. 2013; Chren et al. 1997b). When selecting an instrument consider the following:

- What are you trying to measure?
- Does the instrument take disease-specific characteristics into account?
- Is it patient or physician-dependent?
- Will the patient be able to complete it (language barrier, literacy)?
- Is it burdensome to complete in an outpatient setting (consider asking the patient to complete prior to appointment, or afterwards)?
- The recommended strategy is to combine a generic dermatology and disease-specific measure (Both et al. 2007)

1. *Considering the psychosocial impact of dermatological disease*

Dermatological disease can affect many aspects of a patient's life and their self-perception. Although often interrelated and overlapping, psychosocial impacts to consider include:

- Feelings of embarrassment
- Decreased confidence and self-esteem
- Fears of stigma or rejection
- Social anxiety or social withdrawal
- Ethnic and cultural issues
- Secondary psychiatric co-morbidities, e.g. depression or anxiety
- Physical functioning
- Sleep disturbance
- Restrictions on family responsibilities

Table 29.1 Generic HRQoL instruments

QoL measure	Brief description	Total number of items	Domains assessed	Completion time
EuroQoL 5-Dimension (EQ-5D) (Group E 1990)	• One of the most commonly used HRQoL measurements in population health studies, clinical practice and clinical research • Two sections: a descriptive system with three levels to self-rate (no problems, some problems and severe problems) and a visual analogue scale for respondents to report their overall health status (worst to best health imaginable) • The first section is coded into a five-digit number: 11111 (no problems in all dimensions) and 33333 (severe problems in all dimensions) The numerals have no arithmetic properties • 243 different health states from the coded scoring possible, but ceiling effects present and lack sensitivity for changes with minor morbidity. • 171 languages versions available	Two parts: five items and a visual analogue scale	Mobility Self-care Usual activities Pain/discomfort Anxiety/depression	1 min
Medical Outcomes Study 36-item Short Form Health Survey (SF-36) (Brazier et al. 1992)	• Designed for and frequently used in epidemiological and clinical research. • Eight scaled scores which are coded, summed and translated onto a scale of 0–100 (worst and best health, respectively) • Includes a question on a subjectively perceived change in health and one on an impression of positive health "full of life" • 50+ language versions available and extensive testing for cultural equivalence • Two shorter versions: SF-6 and SF-12 are available	36 items	Physical functioning Social functioning Role limitations due to physical problems Role limitations due to emotional problems General health Vitality Mental health Bodily pain	5–10 min

(continued)

Table 29.1 (continued)

Nottingham Health Profile (NHP) (Hunt et al. 1980)	• Simple questionnaire format with subjective binary item responses ("yes/no") allowing quick self-administration but reduced sensitivity to minor impairments and unable to track deteriorations or improvements in individual items • Sleep included but the social domain is underestimated • Results presented as a profile rather than an overall score • Optional second part on particular life areas including occupation, housework, family life and hobbies	38 items	Energy level Physical mobility Sleep Emotional reaction Pain Social isolation	5–10 min
Sickness Impact Profile (SIP) (Bergner et al. 1981)	• Prioritises the objectively measurable impact of illness on daily activities and behaviours. Less focus on the mental aspects and subjective components of diseases e.g. pain scoring • Clear focus on disability so most suited to use in patients with mobility impairments (e.g. psoriatic arthropathy.) A ceiling effect is present in general population samples and patients with mild disabilities and is less responsive to mild changes • Items are weighted based on the level of dysfunction. The scores are converted onto a 0–100 scale can be calculated for each separate domain, group or as an overall score	136 items	*Physical dimension:* Ambulation Mobility Body care and movement *Psychosocial dimension:* Social interaction Communication Emotional behaviour Alertness behaviour *Independent Categories:* Sleep and rest Eating Work Home management Recreation and pastime	20–30 min

Table 29.1 (continued)

World Health Organisation Quality of Life assessment (WHOQOL-100) (WHOQOL 1998)	• Assesses overall QOL rather than being restricted to HRQoL • The 100 items (each to be rated on a five-point scale) included creates a significant time burden for the respondent but the ease of the scoring algorithm keeps the administrative burden relatively low • Prospectively designed across 15 health centres worldwide. Good discriminant validity, reliability and responsiveness demonstrated in UK and USA populations • A shorter version (WHOQOL-26) combines one item from each of the WHOQOL-100's 24 facets with two benchmark items for overall QoL and general health	100 items	Physical Psychological Level of independence Social relationships Environment Spirituality	30 min

Glossary: *QoL* quality of life, *HRQoL* health-related quality of life

Table 29.2 Dermatology-specific HRQoL instruments

QoL measure	Brief description	Total number of items	Domains assessed	Completion time
Dermatology Life Quality Index (DLQI) (Finlay and Khan 1994)	• First and most commonly used dermatology-specific instrument • Uses a four-point Likert scale for each of the 10 items. A composite score (0–30) is calculated by summing the score of each question. The higher the score, the more the QoL is impaired • Multiple versions are available including translations and an illustrated family and children's version. Scoring may be affected by nationality	10	Symptoms and feelings Daily activities Leisure Work and school Personal relationships Treatment	5 min

(continued)

Table 29.2 (continued)

Skindex-29 (Chren et al. 1997c)	• Assesses the frequency of each item over the previous 4 weeks (never, rarely, sometimes, often, all the time) Designed to assess changes over time and includes item on adverse effects of treatment • Responses for each domain are averaged and transformed into a linear scale of 100, (0 = no effect and 100 = all the time.) A single composite score can be calculated but its validity is unclear • Multiple other versions available including the SkinDex-teen (for adolescents)	29	Symptoms Emotions Functioning	5–10 min
Skindex-16 (Chren et al. 2001)	• A modified one-page version of the Skindex-29 which measures bother rather than frequency of the respondent's experiences • Responses are given on a three-point scale	17	Psychosocial Symptoms	2 min
Dermatology Quality of Life Scales (DQoLS) (Morgan et al. 1997)	• Developed in a single UK outpatient dermatology department to assess the impact of dermatological conditions on patients' psychosocial states and everyday activities • Has not been used frequently in cross-sectional studies	41	Dermatological symptoms Physical activities Psychosocial state	5–10 min

Table 29.2 (continued)

Dermatology-Specific Quality of Life Instrument (DSQL) (Anderson and Rajagopalan 1997)	• Developed from the SF-36, with a focus on acne and contact dermatitis. Not typically used for other conditions. • Easy to use format. The first set of questions use a score of intensity or satisfaction of 1–10. The second set uses a five-point ordinal score assessing frequency over the past month. A final score is summed from adding all the items	52	Dermatologic symptoms Physical activities Psychosocial state	10–15 min
Children Dermatology Life Quality Index (CDLQI) (Lewis-Jones and Finlay 1995)	• A short illustrated questionnaire designed for children (4–16 years) • Contains 10 questions (each on a four-point Likert scale) covering the impact of their skin disease and its treatment on their everyday activities and psychosocial state • An overall score is calculated by summing the score of each question. This is on a linear scale from 0 to 30, the higher the score the greater the impact on their QoL • The Teenager QoL index (T-QoL), an 18 item equivalent is available for teenagers (12–19 years)	10	Symptoms Feelings Leisure time School and holidays Relationships Sleep Treatment impact	2–3 min
Family Dermatology Life Quality Index (FDLQI) (Basra et al. 2007)	• Used to assess the secondary impact of a child's skin condition on the QOL of adult family members. Can also be used for the partner of an adult patient • Uses 10 items, each on a four-point Likert scale • An overall score of 0–30 can be calculated by summing the responses. The higher the score the greater the impact on family members	10	Physical well-being Emotional distress Relationships Household responsibilities Leisure time and hobbies Finance Ability to work/study	2–3 min

Table 29.3 Disease-specific HRQoL instruments

QoL measure	Brief description	Total number of items	Domains assessed	Completion time
Psoriasis Disability Index (PDI) (Lewis and Finlay 2005)	• A subjective questionnaire to be completed by the patient using a 4 week recall period to quantify the level of handicap experienced by patients with psoriasis. It has been translated into at least 16 languages and has been used in published research in 20 countries. Recognised by NICE • 15 questions each with four options (not at all, a little, a lot, very much) scored on a scale of 0–3. Total score is creating by summing the scores of each item. The higher the overall score the more the QoL is impaired	15	Daily activities Work or school or alternative questions Personal relationships Leisure Treatment	3–5 min
Psoriasis Index of Quality of Life (PSORIQoL) (McKenna et al. 2003)	• Specifically designed to measure QoL in psoriasis. Shown to be a practical, reliable and valid instrument for measuring the impact of psoriasis on QoL and recognised for use by NICE. • Consists of 25 questions in a true/not true format. Ever positive response is score 1 point and the individuals points of summed into a final score (maximum 25) with higher scores indicating worse QoL	25	Self-consciousness Problems with socialising Physical contact and intimacy Limitations on personal freedom Impaired relaxation and sleep Emotional stability	3–5 min
Quality of Life Index for Atopic dermatitis (QoLIAD) (Whalley et al. 2004)	• 25 item questionnaire commonly used to measure the impact of atopic dermatitis on a patient's QoL. Available in several languages • Binary responses in yes or no format for each question with each answer recorded as "yes" scoring 1. The higher the final summed score the worse the QoL	25		2 min

Table 29.3 (continued)

Infants' Dermatitis Quality of Life Index (Lewis-Jones et al. 2001)	• Designed for use in infants with atopic dermatitis below the age of 4 years. Is be completed by the child's parent or regular carer • The Infants' Dermatitis Quality of Life Index is calculated by summing the score of each question creating a total from 0 to 30. The higher the score, the more the QoL is impaired	10	Symptoms Daily life Activity limitations Emotions	2 min
Acne-Specific Quality of Life Questionnaire (AcneQoL) (Gupta et al. 1998)	• Developed and validated for use in clinical trials. Confirmed to be responsive, internally consistent, and valid • Patient-completed questionnaire with a 1-week recall period composed of 19 items in four subscales. The responses of each item are summed to yield four overall domain scores. A higher score represents a higher quality of life	19	Self-perception Role-emotional Role-social Symptoms	3–5 min
RosaQOL (Nicholson et al. 2007)	• Developed for acne rosacea to be specific for subjective disease burden related to rosacea and sensitive to changes in the disease over time • Reponses to each item score on a scale from 1 (never) to 5 (always). The higher the overall score, the worse the HRQoL	23	Emotions Functioning Symptoms	3–5 min

(continued)

Table 29.3 (continued)

Melasma Quality of Life Scale (MELASQOL) (Balkrishnan et al. 2003)	• Developed from other questionnaires to prioritise the emotional and psychosocial aspects of melasma (in female patients) with higher discriminatory value compare to other general scoring instruments • Uses a seven-point Likert scale ranging from 0 (not bothered at all) to 7 (bothered all the time). The answers are summed together to provide an overall score, with higher scores representing a poorer quality of life	10	Emotional well-being Social life Recreation and leisure	2–3 min
VitiQol (Lilly et al. 2013)	• Developed to assess the impact of vitiligo on the patient's QoL • Uses a seven-point Likert score ranging from 0 (never) to 6 (all the time) for the first 15 items to assess frequency. With scores that the patient reports for each item added to yield a total score. The final item is a seven-point Likert score which asks the patient to self-rate the severity of their vitiligo	16	Participation limitation Stigma Behaviour	2–3 min
Chronic Urticaria Quality of Life Questionnaire (CU-QOL) (Baiardini et al. 2005)	• A validated tool developed to detect the impact of chronic urticaria on subjective well-being and QoL • An easy to use format with each item being scored on a five-point Likert scale. The scores are then summed to create an overall score	23	Physical symptoms Impact on life activities Sleep problems Embarrassment Limits	5 min

- Limitations on recreational activities, leisure time or holidays
- Financial implications, e.g. reduced ability to work or occupational restrictions, cost of treatments

2. *Why ask about QoL?*

Specific consideration of QoL can improve patient care and service delivery in a number of ways. These are summarised below:

Reason	Examples
To inform clinical decision making and the consultation	• Improving shared decision making • Setting appropriate treatment aims • Guiding dose adjustments and use of clinical guidelines • Informing referral or discharge decisions
To improve communication between the patient and clinician	• QoL scoring systems provide a quick method for the clinician to see the subjective impact of the skin condition on different dimensions of the patient's QoL (some of which may not frequently be asked about) • High or low scores can then prompt further discussion of these areas.
For awareness of skin disease burden	• Prompts the patient to consider areas of their life which may be impacted that are not often asked about • For the clinician to gain an understanding of the patient's experience and skin-related QoL—which may often not correlate with skin lesion burden
For clinical service development	• For audit and quality improvement purposes • To inform clinical guideline development • For education purposes through improved understanding of the impact of skin disease

3. *How to start asking patients about their QoL*

Validated generic or dermatology-specific QoL questionnaires provide accurate tools to record, track and compare perceived QoL.

However, useful screening questions to open up a discussion about the patient's QoL include:

- Do you find that your skin condition affects your quality of life?
- It is common for skin conditions to have an impact on mood, is this something that you have experienced?
- Is there anything that you would like to do but are unable to or find difficult to do because of your skin condition?
- Do you find that your skin condition or its treatments interfere with your daily activities, responsibilities or ability to work?

- Do you find that your skin condition interferes with your family or social life? Your ability to build or maintain relationships?
- Does your skin condition interfere or stop you from doing activities that you find fun or fulfilling?

4. *So your patient reports a poor QOL?*

When helping a patient with a poor QoL, identified either through conversation or by using a QoL measurement tool, there are a number of questions we recommend considering.

Consider:

- Is their skin disease a key negative driver behind their poor-quality of life or are there any other contributing factors?
- Has a QoL score been recorded previously? What is the trend and what may be contributing to any change?
- Are there other related co-morbidities? Consideration of anxiety and depression is required.
  - The Hospital Anxiety and Depression Scale (HADS) can be used as a valid screening tool.
  - Could treatments being used, e.g. steroids or retinoids, be contributing to a recent change in mood?
- Is the patient at risk of suicidal behaviour?
  - Patients should be asked directly about this. There is no evidence that asking these questions increases suicide risk.
  - If yes, a risk assessment should be completed and the patient referred to an appropriate team as per local guidelines and their risk assessment. These teams may include: their GP, liaison psychiatry or a local crisis team
- What is the patient's view on how they can improve their QoL?

Take action to improve QoL

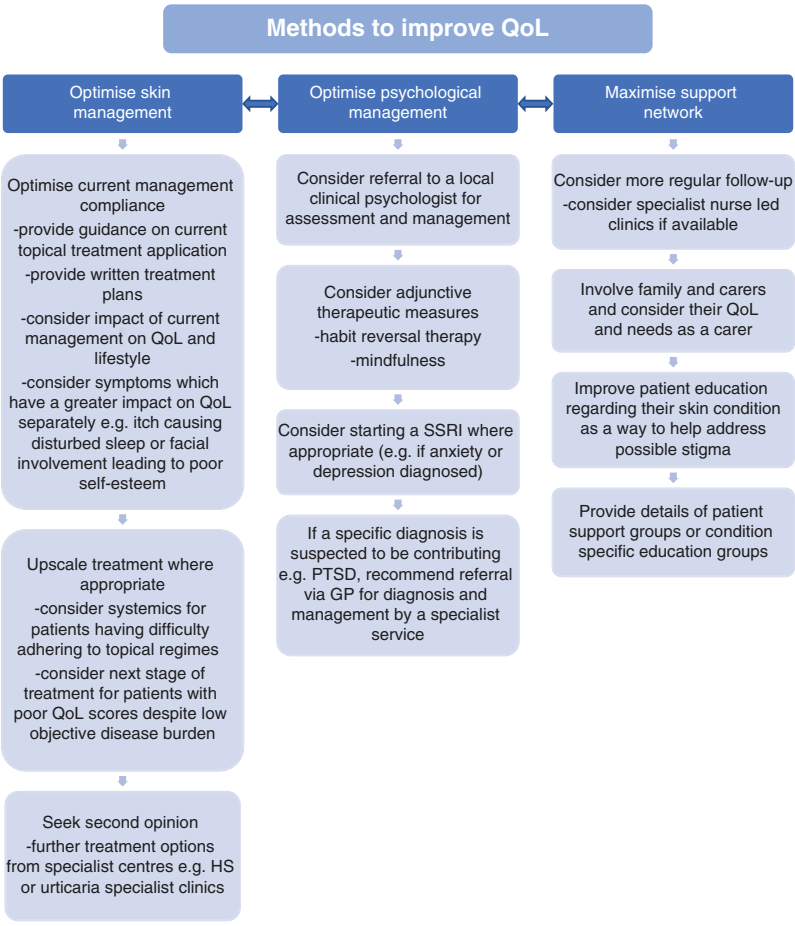
There is a bi-directional relationship between physical and mental health. Optimise disease management, bearing in mind potential negative implications of the treatment itself, e.g. time requirements which could lead to further limitations of social and work activities. It is important to include the patient in this discussion.

Alongside this, consider the patient's psychosocial well-being. This may involve referral to other clinical teams such as a clinical psychologist for assessment and to start target treatments (e.g. cognitive behavioural therapy or habit reversal therapy). You should also consider starting the patient on an SSRI if the patient has significant anxiety or depression. Starting an SSRI requires monitoring and may have associated risks. It may be appropriate to start treatment in conjunction with the patient's family doctor who may also be able to refer patients for evidence-based psychological therapies in the community such as Talking Therapies or the IAPT (Improving Access to Psychological Therapies) programme.

5. *How to improve QoL in dermatology patients*

Practice Point
Completed QoL scores should prompt a dialogue during the consultation where specific QoL domains which are rated particularly adversely or have had a marked difference since the previous score are discussed to determine contributing factors and prioritise a management plan. Recording the total score alone misses the opportunity for valuable insight and may reduce patient motivation to complete another score if nothing clearly happens.

When aiming to improve patients' QoL, there are three main aspects of management which are listed below. These should be managed simultaneously. The flow chart does not need to be followed in order, it instead provides a list of options to be considered and moved between.



6. *QoL of family members and caregivers*

It is important to also consider the impact of the patient's dermatological disease on their caregiver's QoL and family function. This is relevant for both adult and paediatric patients who rely on family members or other carers for either repeated emotional or physical (e.g. help with treatment administration) support.

Caring for a family member with skin disease can be very time consuming, which can have negative impacts on personal relationships, psychosocial functioning and cause sleep disturbance. An individual's ability to work may be restricted due to care commitments and they may need to take unplanned leave to care for a sick child.

A number of validated carer QoL scales exist. Commonly used generic scales include The Adult Carer Quality of Life Questionnaire (AC-QOL). The Family Dermatology Life Quality Index (FDLQI) has been created to acknowledge the specific needs of carers supporting a patient with dermatological disease, and the potential impact of the dermatological disease on family function. Carer QoL scales are also being developed for specific conditions, such as the Dermatitis Family Impact Scale. Further details of these tools are documented in the QoL tool summary tables.

Practice Point

Anxiety and depression were found to have a 36% prevalence in caregivers of children with either atopic dermatitis or psoriasis in a recent study. Both of these can reduce the ability of the caregiver to support the patient, and prevention or treatment of these may help skin disease management.

Use of a validated caregiver QoL scale for consideration of the burden on caregivers is important to identify negative impacts on different domains (psychosocial, relationships, financial.) Poor caregiver QoL scores may require consideration of more intensive treatment or additional support for the caregiver.

7. *Role of QoL assessment in access to high-cost treatments*

QoL measurements also have an impact on the availability of treatment options and management decisions.

Many national guidelines require minimum DLQI scores before certain treatments are available. These include:

NICE Guidelines including DLQI scores as criteria for funding		
Drug	Condition	Criteria
Adalimumab, Etanercept	Plaque psoriasis	• DLQI > 10 for commencing treatment • 5 point reduction in DLQI at 16w (adalimumab) and 12w (etanercept)
Infliximab	Plaque psoriasis	• DLQI > 18 for commencing treatment • 5 point reduction in DLQI at 10w

NICE Guidelines including DLQI scores as criteria for funding		
Drug	Condition	Criteria
Il-17a inhibitors e.g. Ixekizumab	Plaque psoriasis	• DLQI > 10 for commencing treatment • 5 point reduction in DLQI at 12w
Apremilast	Plaque psoriasis	• DLQI > 10 for commencing treatment • 5 point reduction in DLQI at 12w
Dupilumab	Atopic dermatitis	• 4 point reduction in DLQI at 16w
Alitretinoin	Chronic hand eczema	• DLQI > 15 for commencing treatment

8. *Interpretation of calculated DLQI scores*

How to interpret DLQI scores	
0–1	No effect at all on the patient's life
2–5	Small effect on patient's life
6–10	Moderate effect on patient's life
11–20	Very large effect on patient's life
21–30	Extremely large effect on patient's life

9. *Tools available to measure QOL*

Commonly used outcome measures for QoL are summarised in Tables 29.1–29.3.

Practice Point

Use both a generic QoL questionnaire as well as a condition-specific QoL questionnaire when considering QoL. Generic scales allow comparisons with other conditions and may be required for treatment guidelines, whereas condition-specific scales may allow more precise consideration of all dimensions of the patient's life that may be affected as a result of their specific skin condition.

References

Anderson RT, Rajagopalan R. Development and validation of a quality of life instrument for cutaneous diseases. J Am Acad Dermatol. 1997;37(1):41–50. https://doi.org/10.1016/s0190-9622(97)70210-x.

Baiardini I, Pasquali M, Braido F, Fumagalli F, Guerra L, Compalati E, Braga M, Lombardi C, Fassio O, Canonica GW. A new tool to evaluate the impact of chronic urticaria on quality of life: chronic urticaria quality of life questionnaire (CU-QoL). Allergy. 2005;60(8):1073–8. https://doi.org/10.1111/j.1398-9995.2005.00833.x.

Balkrishnan R, McMichael AJ, Camacho FT, Saltzberg F, Housman TS, Grummer S, Feldman SR, Chren MM. Development and validation of a health-related quality of life instrument for women with melasma. Br J Dermatol. 2003;149(3):572–7. https://doi.org/10.1046/j.1365-2133.2003.05419.x.

Basra MK, Sue-Ho R, Finlay AY. The Family Dermatology Life Quality Index: measuring the secondary impact of skin disease. Br J Dermatol. 2007;156(3):528–38. https://doi.org/10.1111/j.1365-2133.2006.07617.x.

Bergner M, Bobbitt RA, Carter WB, Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. Med Care. 1981;19(8):787–805. https://doi.org/10.1097/00005650-198108000-00001.

Both H, Essink-Bot M-L, Busschbach J, Nijsten T. Critical review of generic and dermatology-specific health-related quality of life instruments. J Invest Dermatol. 2007;127(12):2726–39.

Brazier JE, Harper R, Jones NM, O'Cathain A, Thomas KJ, Usherwood T, Westlake L. Validating the SF-36 health survey questionnaire: new outcome measure for primary care. BMJ. 1992;305(6846):160–4. https://doi.org/10.1136/bmj.305.6846.160.

Chren M-M, Lasek RJ, Flocke SA, Zyzanski SJ. Improved discriminative and evaluative capability of a refined version of Skindex, a quality-of-life instrument for patients with skin diseases. Arch Dermatol. 1997a;133(11):1433–40.

Chren MM, Lasek RJ, Quinn LM, Covinsky KE. Convergent and discriminant validity of a generic and a disease-specific instrument to measure quality of life in patients with skin disease. J Invest Dermatol. 1997b;108(1):103–7. https://doi.org/10.1111/1523-1747.ep12285650.

Chren M-M, Lasek RJ, Quinn LM, Covinsky KE. Convergent and discriminant validity of a generic and a disease-specific instrument to measure quality of life in patients with skin disease. J Invest Dermatol. 1997c;108(1):103–7.

Chren MM, Lasek RJ, Sahay AP, Sands LP. Measurement properties of Skindex-16: a brief quality-of-life measure for patients with skin diseases. J Cutan Med Surg. 2001;5(2):105–10. https://doi.org/10.1177/120347540100500202.

Finlay AY, Khan G. Dermatology Life Quality Index (DLQI)—a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994;19(3):210–6.

Group E. EuroQol–a new facility for the measurement of health-related quality of life. Health Policy. 1990;16(3):199–208.

Gupta MA, Johnson AM, Gupta AK. The development of an Acne Quality of Life scale: reliability, validity, and relation to subjective acne severity in mild to moderate acne vulgaris. Acta Derm Venereol. 1998;78(6):451–6.

Hunt SM, McKenna SP, McEwen J, Backett EM, Williams J, Papp E. A quantitative approach to perceived health status: a validation study. J Epidemiol Community Health. 1980;34(4):281–6. https://doi.org/10.1136/jech.34.4.281.

Lewis VJ, Finlay AY. Two decades experience of the Psoriasis Disability Index. Dermatology. 2005;210(4):261–8. https://doi.org/10.1159/000084748.

Lewis-Jones MS, Finlay AY. The Children's Dermatology Life Quality Index (CDLQI): initial validation and practical use. Br J Dermatol. 1995;132(6):942–9. https://doi.org/10.1111/j.1365-2133.1995.tb16953.x.

Lewis-Jones MS, Finlay AY, Dykes PJ. The Infants' Dermatitis Quality of Life Index. Br J Dermatol. 2001;144(1):104–10. https://doi.org/10.1046/j.1365-2133.2001.03960.x.

Lilly E, Lu PD, Borovicka JH, Victorson D, Kwasny MJ, West DP, Kundu RV. Development and validation of a vitiligo-specific quality-of-life instrument (VitiQoL). J Am Acad Dermatol. 2013;69(1):e11–8. https://doi.org/10.1016/j.jaad.2012.01.038.

McKenna SP, Cook SA, Whalley D, Doward LC, Richards HL, Griffiths CE, Van Assche D. Development of the PSORIQoL, a psoriasis-specific measure of quality of life designed for use in clinical practice and trials. Br J Dermatol. 2003;149(2):323–31. https://doi.org/10.1046/j.1365-2133.2003.05492.x.

Morgan M, McCreedy R, Simpson J, Hay RJ. Dermatology quality of life scales–a measure of the impact of skin diseases. Br J Dermatol. 1997;136(2):202–6.

Nicholson K, Abramova L, Chren MM, Yeung J, Chon SY, Chen SC. A pilot quality-of-life instrument for acne rosacea. J Am Acad Dermatol. 2007;57(2):213–21. https://doi.org/10.1016/j.jaad.2007.01.048.

Nijsten TE, Sampogna F, Chren M-M, Abeni DD. Testing and reducing skindex-29 using Rasch analysis: Skindex-17. J Invest Dermatol. 2006;126(6):1244–50.

Prinsen C, De Korte J, Augustin M, Sampogna F, Salek S, Basra M, Holm E, Nijsten T, EToQo L. Measurement of health-related quality of life in dermatological research and practice: outcome of the EADV Taskforce on Quality of Life. J Eur Acad Dermatol Venereol. 2013;27(10):1195–203.

The World Health Organization Quality of Life Assessment (WHOQOL). Development and general psychometric properties. Soc Sci Med. 1998;46(12):1569–85. https://doi.org/10.1016/s0277-9536(98)00009-4.

Whalley D, McKenna SP, Dewar AL, Erdman RA, Kohlmann T, Niero M, Cook SA, Crickx B, Herdman MJ, Frech F, Van Assche D. A new instrument for assessing quality of life in atopic dermatitis: international development of the Quality of Life Index for Atopic Dermatitis (QoLIAD). Br J Dermatol. 2004;150(2):274–83. https://doi.org/10.1111/j.1365-2133.2004.05783.x.

Complementary and Alternative Medicine and Psychodermatology

30

CAM Approach to Dermatology Focuses on the Whole Person, Including Psyche and Environment

Alex Laird

Introduction

Complementary and alternative medicine (CAM) takes an integrative and systems approach to dermatology. CAM modalities, such as nutritional therapy, acupuncture, Chinese herbal medicine and phytotherapy/western herbal medicine, consider the person as a physical, psychological and social whole and part of their environment, rather than focusing on the body or the disease alone. We now recognise that the mind and body are one, connected by multiple communication pathways, including those from the gut microbiome to the brain. The question for this patient-centred approach is: how is this unique individual functioning with this condition, in what environment, and how can their functioning be improved?

As a result, and as a principle, CAM considers the psychological aspect of any disease when assessing the whole person's functioning. Emotional or mental factors, however minor, may influence all physical conditions. In turn, these are determined to a great extent by the gut microbiome and diet as well as by other powerful factors such as economic status and relationships. The overall aim of CAM is to rebuild the patient's healthy functioning for the long term and coach them to take more control of their health.

CAM and Psychodermatology

CAM's holistic approach has much to offer psychodermatology, as well as to primary psychiatric and primary dermatological disorders. It aims to address the complex patho-aetiology of a condition in a specific way, given the multiple causes of disease from the interplay between a unique individual and their environment.

A. Laird (✉)
Barts Health NHS Trust, London, UK
e-mail: alex.laird@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_30

In primary psychiatric disorders, CAM treatment may be geared towards the patient's emotional state and nervous system, whereas in primary dermatological conditions it would also address both the nervous system and more organic pathophysiology. In both, encouraging patients to take an active role in their healthcare, however small, can be therapeutic. Clinicians in standard health services may have limited time to give lifestyle or dietary advice, so CAM practitioners may play a valuable role in both hospital and GP surgeries.

Disease Seen in Terms of Function

CAM's approach to disease also tends to view illness as the body/mind signalling an attempt to readjust to excess stressors. In this approach, illness is seen as the body's likely response to force us to stop and consider what changes we need to make to our diet, lifestyle, emotional state and environment. Instead of a pathology to be cured, it is more about better managing the body–mind interaction. This means first reducing the stressors and upstream causes, and then supporting function by improving the basics of circadian rhythm, diet, sleep, exercise, emotional well-being and the interconnected systems including digestion, circulation, liver metabolism, immune responses, the brain–gut and hormonal functions. As well as giving medicine or treatment, educating the patient in self-care is an important aspect of CAM.

Meaning of the Disease to the Patient

Psychologically, the disease itself may have important and fundamental meaning to the patient. As CAM clinicians, especially in psychodermatology, our job is to identify this as part of the multiple aetiology behind a person's condition. Our treatment of a patient aims to reflect and work with many aspects of their individuality and complexity.

Expanding the Patient's Sense of Self

Chronic skin disease often confines us as patients, narrowing our sense of self and confidence, our lifestyle, physical activity and social lives. A vital part of the CAM clinician's role is to expand the patient's sense of self, to recognise their many internal resources and recover their agency. Self-care is seen to be at the heart of resilience. At the very least, this will help the patient to manage their disease and live better with it.

Purpose of Treatment

By focusing not only on symptoms but also on underlying causes, the aims are:

- To improve the patient's various body functions

- To build the patient's resilience to disease
- To enable the patient's greater control over their condition

The treatment may take longer as a result of this approach, which requires the patient's understanding and cooperation.

Herbal Medicine and Phytotherapy

Western medical herbalism or phytotherapy, Chinese herbal medicine, Ayurveda and Unani Tibb are among the most widely practised forms of herbal medicine. Herbal skin products make up one of the four biggest natural products sold over the counter and herbal medicine has a growing global market share.

Western herbal medicine (phytotherapy, as it is known in Europe) will be used here as an example of CAM in psychodermatology alongside conventional medicine, as a medical herbalist clinic has operated within Whipps Cross University Hospital Dermatology Outpatients in London, UK, since 2000. Medical herbalists/phytotherapists are trained in rational, evidence-based natural medicine, underpinned by 3-4 years of biomedicine, nutrition, pharmacology, phytochemistry and clinical training. Scientific knowledge and research evidence is used to inform and refine valuable traditional herbal wisdom, clinical experience and patient opinion.

In professional herbal practice, there is no one herb for one physical or psychological condition. Several herbs are chosen for their pharmacological actions that together target multiple underlying factors. Each herb has at least two or three actions that improve specific cell, tissue or organ function. One person's eczema may be more anxiety-related than another's where gut dysbiosis predominates, so may be given bitter hops (calming and acting on skin/liver function), liquorice (anti-inflammatory, adrenal cortisol-sparing) and ashwagandha (adaptogenic to stress). A patient with rosacea accompanied by anxiety and hypertension may receive a mix including hawthorn (its cyanogenic glycosides calm and support myocardial function) and lime blossom (flavonoid-rich with relaxant and vascular support).

Herbal Pharmacology

Plants produce secondary metabolites for defence and communication, known as phytonutrients or phytochemicals. Phytonutrients are pharmacologically active compounds and are in all plant foods but especially concentrated in herbs and spices. They include anti-inflammatory, antimicrobial, immune-stimulating polyphenols (e.g. flavonoids, lignans), alkaloids, anthraquinones, cardiac glycosides and essential oils, which are particularly antimicrobial. Having co-evolved with plants, we have depended on their phytonutrients over millennia as the crucial medicinal part of our food. Whole food plants and herbs, if their phytonutrient content has not been degraded through breeding, chemical sprays, fertilisers and equally degraded

soils, will produce a diverse array of these vital chemicals. Most phytonutrients are stored in the plant's skin, pith and seeds, where their functions are most needed.

Efficacy and Safety of Plant Medicine

In an individualised prescription, herbs are selected for the evidence base of their pharmacology, traditional and modern clinical use showing how they address specific physiological functions and pathways. A particular part of the plant is used, be it root, leaf, flower or other parts, for its known therapeutic effects. Its safety profile and potential drug interactions are taken into account. Ensuring the authenticity of species and plant parts, along with a therapeutic dosage, is essential for efficacy.

Consultation and Diagnosis

Typically, patients have a 1-hour first consultation followed by several half-hour follow-ups. The first consultation with the patient includes:

- Explaining simply how herbal medicine/phytotherapy works, a collaboration between clinician and patient to include active self-care where possible, the patient's own wishes and expectations of treatment and the possible time-scale for treatment and likely outcome.
- A comprehensive medical history, current medical diagnoses, drug and health supplements, the state of each body system, a physical examination, pulse rate and blood pressure readings.
- Their basic daily functioning:
  - Sleep: time to bed, night waking and causes, quality, wake time
  - Digestion: appetite, bloating, wind, food irritants, bowel habit
  - Psychological state, including work/life balance, relationships
  - Exercise: what and how often
  - Daylight: how much and when—both for vitamin D and for circadian entrainment
  - Diet: meal times, specific food types and plant family intake, drinks, portion size
  - Smoking, alcohol, drug intake, other
- The patient's own treatment priorities—what matters most to them.
- A self-reported symptom outcome measure (an adapted MYMOP/Measure Your Medical Outcome Profile) is found to be a therapeutic tool for both patient and practitioner at Whipps Cross Hospital.

This comprehensive consultation allows the medical herbalist to build on the dermatological and/or other medical diagnoses to identify and address possible pathophysiology contributing to them:

- Inflammatory responses, local and systemic, including those arising from disrupted functions below
- Blood sugar/insulin responses
- Liver metabolism—phase I (creating reactive compounds) and II (conjugating/modifying reactive compounds for excretion)
- Circulation (micro and macro)
- HPA hypothalamic pituitary adrenal axis and hormonal function
- Immune responses (innate and adaptive including Th1/Th2, Th1:Treg
- Digestive functions (gut microbiome, gut–brain axis and neuroendocrine)
- Circadian rhythm

Other relevant investigations or referrals to medical colleagues may be made to clarify any comorbidity and its causes.

Treatment Plan

1. Apply basic self-care to reduce stressors on the body and nudge homeostasis.
2. Apply herbal medicine to improve key physical and psychological functions and address specific pathophysiology.

Basic Self-Care

The first step in the treatment of almost all conditions is to address diet, sleep, circadian rhythm and lifestyle. These are fundamental to supporting immune function and reducing systemic inflammation. Any concurrent medicine will then be more effective. We train our master and peripheral body clocks by matching our habits to the natural peaks of hormones, enzymes and neurotransmitters with daylight exposure, eating, sleep, rest and movement. This synchronicity reduces stress on the body and nudges optimal function to build resilience.

Diet Eating a diverse diet of unprocessed whole food and in time with our body clocks ensures balanced blood sugar and insulin levels. This is of fundamental importance in:

(a) How we deal with stress/anxiety by supporting adrenal response and moderating the effect of adrenaline
(b) Reducing inflammatory responses induced from excess hormones (oestrogen) and other inflammatory mediators driven by insulin and fat
(c) Addressing the health of the gut microbiome and brain–gut axis

In addition, fundamental to appropriate anti-inflammatory responses and nerve function is sufficient healthy fat intake. The brain and nerves are 60% fat, and the

body has to be provided with essential fatty acids, including omega 3 alpha-linolenic, EPA and DHA and omega 6 linoleic acids from the diet to produce inflammatory and anti-inflammatory reactions.

Diet diary and eating pattern Patients are asked to write a week's detailed food diary with timings. This facilitates step-by-step food changes and builds the patient's awareness and agency around when and what they eat. We explain simple principles and how foods affect the body/mind and give recipes rather than a specific, limiting diet. The key is to widen out the diet, inspire the patient with understanding to start with simple changes. Eating a wide range of foods is central to our design so that our many metabolic pathways are provided with the substrate needed to moderate inflammatory and immune responses.

See Tables 30.1, 30.2 and 30.3 for a summary.

The Gut–Brain–Skin Axis

There is symbiosis between our gut microbes and systemic immunity. Beneficial microbial species are fed by fibre-rich, deeply coloured whole foods to produce SCFAs/short-chain fatty acids, particularly butyrate, which suppress immune responses by inhibiting inflammatory cells' proliferation, migration, adhesion, and cytokine production. They are involved in the regulation of hair follicle stem cell differentiation and wound healing. Gut dysbiosis with disturbed tight junctions in the intestine's epithelial barrier can allow proteins, bacteria and intestinal microbes to enter the bloodstream, accumulate in the skin and disrupt its homeostasis. The gut microbiome also appears to influence the skin microbiome and its defence mechanisms, by producing strongly antimicrobial skin SCFAs. Oral supplementation of gut microbes, i.e. probiotics, may be helpful, but it is still not fully understood which species are needed in a specific patient.

Sleep: Self-Care and Herbal Medicine

Sleep is crucial to our circadian rhythm to allow rest and repair of genes, gut villi, brain cells and liver along with all other tissues and functions and reduce inflammation (Table 30.2).

Example of a Herbal Prescription for Patient with Eczema and Anxiety

This could be delivered as an alcohol-based tincture containing several herbs, or as dried herbs to be infused or decocted in a tea, as tablets, capsules or powder (Table 30.4).

Table 30.1 Anti-inflammatory, hormone- and immune-modulating diet

Guidelines (recommendations)	Foods and therapeutic actions
• Mostly wholefood plants complete with their edible skins, pith, seeds • Most in daytime, ideally within 10 h—eat either substantial fibre-rich wholefood breakfast or lunch as main meals • Mostly: small portions early evening meal to allow gut repair overnight • Wide variety of plant foods, some raw • Rainbow colours especially dark	• Fibre-rich wholefood > for gut microbiome (immunity, neurotransmitter, vitamins), bowel function, raise sex hormone-binding globulin/SHBG • Legumes (beans/lentils) 1 portion/day > protein, probiotic fibre, raise SHBG, bind ER to moderate inflammatory oestrogen levels • Oily fish high in omega 3 fatty acids—sardines, mackerel, salmon, pilchards, sprats—three times/week > provide skin EFAs essential for cell barrier, signalling, local and systemic anti-inflammatory responses • Green leafy veg 1–2 portions/daily > for minerals, phytonutrients, fibre, probiotics • Nuts mixed, especially walnuts and Brazil nuts (selenium-rich) • Mixed berries for anti-inflammatory polyphenol pigments and vitamin C • Seeds especially linseed, pumpkin (zinc-rich), sunflower—best ground to access oil—for unoxidised omega 6 and other EFAs for skin/nerve and immune function • Avocados, nuts, seeds and their cold-pressed oils for vital • Polyphenol-rich pigmented foods e.g. carrots, beetroot, squash, turnip, pumpkin and their oil/fibre-rich seeds • Water or green/herb tea intake >1–1.5 L/day • Add 1–2 tbs chopped parsley or coriander daily to a meal to increase micro and phytonutrients
• Avoid known triggers—alcohol (flavonoid-rich red wine may be less inflammatory than white), spicy and sugary foods/drinks that promote inflammation	• If diet is poor, consider supplementation: omega 3 fatty acids, vitamin A, vitamin C, zinc, selenium, etc., probiotics
• Avoid or reduce refined carbohydrates; our bodies, not machines, are designed to process them slowly in order to release glucose gradually into the bloodstream and spare insulin	• Substitute with filling, nutritious wholefood containing protein e.g. beans/lentils, wholegrains/seeds (short-grain/wild/basmati/red/black/brown rice, quinoa, millet)
• Remove possible food irritants one at a time for 3 weeks then reintroduce to see if symptoms return over 2–3 days. Common allergens or inflammatory triggers to gut villi, initiating immune responses are: cow's milk, eggs, soy, wheat gluten, nuts, shellfish	• Ensure full nutrition with wholegrains/seeds (short-grain/wild/basmati/red/black/brown rice, quinoa, millet and beans/lentils) provide more protein and other nutrients than refined wheat bread, cakes and pastry NB if patient not used to beans/lentils, start with small portions and increase gradually to allow gut microbial species that consume these foods to multiply and so avoid increased flatulence

Table 30.2 Guidelines and herbal medicines

Guidelines	Herbal medicine	Herbal therapeutics and pharmacology—examples
Reinforce circadian rhythm: • Set mostly regular bedtime, ideally well before 11 pm to reinforce body clock's cortisol fall and melatonin rise, though this varies whether patient is a lark or an owl • Aim for 8-h sleep window with 7+ hours sleep and minimal latency • Wind down pre-bedtime, e.g. hot bath to relax and lower core body temperature for sleep • Reduce stimulating TV/screen time and use night-mode to cut out blue light	**Herbs** general relaxants, sedatives, anxiolytics: valerian, hops, passionflower, skullcap, lavender, chamomile	Valerian *Valeriana officinalis* sesequiterpenes and other compounds are sedative by binding GABA to inhibit its breakdown similar to a benzodiazepine effect Hops *Humulus lupulus:* sedative through GABA modulation Chamomile *Matricaria chamomilla* Benzodiazepine agonist, Ca channel inhibitor, MAO inhibitor, blocks noradrenaline uptake Passionflower *Passiflora incarnata* benzodiazepine partial agonist
• Address sleep disrupters, e.g. menopausal hot flushes, anxiety, disrupted circadian rhythm, indigestion, light bedroom, improve sleep hygiene	**Herbs** for cause of sleep disruption: *Menopausal flushes* black cohosh, lady's mantle, astragalus, hops, zizyphus *Indigestion* eat less and earlier and bitter herbs, e.g. gentian, antispasmodics fennel, ginger; anti-inflammatory chamomile; heartburn: slippery elm powder, liquorice *Disrupted circadian rhythm* herbal adaptogens to reinforce rhythm: astragalus, Siberian ginseng, ashwagandha *Anxiety* herbal anxiolytics (as above)	Black cohosh root *Cimcifuga racemosa* (saponins, glycosides, salicylic acid) calming, reduces hot flushes likely via synergistic cAMP, serotonin, GABA and dopamine modulation Astragalus root *Astragalus membranaceous* (saponins, polysaccharides) Gentian root *Gentiana lutea* (bitter glycosides, alkaloids) stimulates bitter TAS2 receptors to increase absorption, delay gastric emptying, manage hyperglycaemia Slippery elm bark powder *Ulmus fulva* its mucopolysaccharides coat gut endothelium and feed gut microbiota as prebiotic Ashwagandha *Withania somnifera* (alkaloids, lactones, saponins) adaptogen, anti-inflammatory, nervine sedative

Table 30.3 Herbal treatment of physical and psychological aspects of skin disease

Condition	Treatment	Pharmacology
Depression and fatigue	**Self-care** Regular sleep/wake time, plenty of daylight. **Diet** as above for gut microbiome to provide SCFA energy and neurotransmitters, EFAs for nerve function, balance sugar levels for mood, lower inflammatory mediators **Herbs** may include, e.g. St. John's wort *H. perforatum,* skullcap, rhodiola, ashwagandha, Siberian ginseng, rosemary	St. John's wort *Hypericum perforatum* (Fig. 30.1) **NB Significant, potentially fatal drug-drug interactions are possible**
Stress/anxiety	**Self-care** and **diet** as above **Herbs** may include lime blossom, skullcap, hops, valerian, hawthorn	*Crataegus spp.* hawthorn

Table 30.4 Example of a herbal prescription for patient with eczema and anxiety

Functions/conditions	Herb types by action	Herbs
Inflammation	Skin/systemic anti-inflammatories	Turmeric (Fig. 30.2) *Curcuma longa,* Baical skullcap *Scutellaria baicalensis*
GI secretions	Digestive stimulants or antispasmodics	Gentian *Gentiana lutea,* wild yam *Dioscorea villosa*
Sleep	Sedatives/relaxants	Valerian *Valeriana officinalis,* Passionflower *Passiflora incarnata*
Anxiety	Anxiolytics	Skullcap *Scutellaria lateriflora,* St. John's wort *Hypericum perforatum*
Micro/macrocirculation	Circulatories	Capsicum *Capsicum minimum* (Fig. 30.2), ginkgo *Ginkgo biloba,* cocoa *Theobroma cacao*
Anxiety, stress	Nervine and psychotherapeutic herbs as above	Herbs as above

Fig. 30.1 St. John's wort, hawthorn, herbal tincture and infused oil

Turmeric

Capsicum infused oil and cream

Fig. 30.2 Turmeric and capsicum infused oil and cream

Topical treatment
Creams or ointments will be given as required with tissue healing herbs such as marigold, circulatories such as capsicum and anti-inflammatories such as liquorice or turmeric.

Other Examples

Rosacea
Hippocrates saw food as medicine. A red face meant that blood was too acidic, and alkaline-forming foods or "cold foods" were given. This has now proved to be good practice, as evidence supports a link between rosacea and gut dysbiosis and microbiome balance and metabolism.

Quality of food, herb supplements and registered herbal medicines
Caution: Most of the herbs bought over the counter are classified as food products, as long as they have no medicinal claims. They are not to be recommended as there is poor quality control for food products and surveys have shown that the vast majority are either adulterated or have no active markers. Some countries use registers to identify OTC herbal medicines appropriate to recommend, such as the UK's MHRA's THR (Traditional Herbal Registration) registration https://www.gov.uk/government/publications/herbal-medicinesgranted-a-traditional-herbal-registration-thr/herbal-medicines-granted-a-traditional-herbal-registration. Medical herbalists, however, have access to practitioner-only herbal medicines from trusted suppliers with GMP or higher manufacturing standards.

Bibliography

Bone K, Mills S. Principles and practice of phytotherapy: modern herbal medicine. 2nd ed. London: Churchill Livingstone; 2012.
http://www.herbalist.org.uk
http://www.thecpp.uk

http://www.nimh.org.uk.
Royal College of Psychiatrists Complementary & alternative medicines: Herbal remedies. https://www.rcpsych.ac.uk/mental-health/treatments-and-wellbeing/complementary-and-alternative-medicines?searchTerms=herb
Salem I, Ramser A, Isham N, Ghannoum MA. The Gut Microbiome as a Major Regulator of the Gut-Skin Axis. Frontiers in Microbiology 2018;9.
Spinella M. The psychopharmacology of herbal medicine. Cambridge, MA: MIT Press; 2001.

Hypnosis and Relaxation Techniques

31

Tanyo Tanev

Hypnosis

Chronology of Important Figures in Hypnosis

The information below is largely based on Heap and Aravinds' (2002) book. Hypnosis-like practices have been observed through human history both in the occult and mainstream religions. However, a clear starting point of modern hypnosis is in the eighteenth century with Franz Anton Mesmer (1734–1815) and his theory of the existence of "animal magnetism", a universal force possessed by all living things. His work was further developed by one of his students, Marquis de Puységur (1751–1825), who added the definition of "artificial somnambulism", explaining this state and why some people were amnesic afterward. Abbé de Faria (1756–1819) showed that some people (10–20% of the population) are highly suggestible and found this by asking people to sit on a chair and focus on the concept of sleep. James Esdaile (1808–1859) began experimenting with mesmerism as an anaesthetic and is recorded to have performed hundreds of surgeries with only the use of mesmerism. James Braid (1795–1860) shifted focus on the concept of sleep and theorised that hypnosis was caused by visual fatigue, which marks the beginning of the *physiological state explanation*. Jean Martin Charcot (1835–1893) insisted that hypnosis was an abnormal state of the mind found in the mentally ill. His opponent at the time, Hippolyte Bernheim (1837–1919), saw hypnosis as a form of intensified suggestibility. Pierre Janet (1859–1977) argued that hypnosis produces a division of consciousness and eliminates conscious control of certain behaviour. Hypnosis went into a decline from the early 1900s to around the 1950s, mostly due to behaviourism and other more observable and evidence-based methods.

T. Tanev (✉)
NHS Trust, The Royal London Hospital, London, UK
e-mail: tanyo.tanev@nhs.net

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_31

Hypnosis then saw an increase in popularity as it was integrated into more modern approaches such as cognitive-behavioural therapy. Various hypnosis-based therapies today are an integration of hypnosis and one or more other psychological therapy.

Current Hypnotherapies

There are various therapeutic approaches that include hypnosis currently available (Table 31.1). To name a few: hypno-analysis, Eriksonian hypnotherapy, past-life regression hypnotherapy, cognitive-behavioural hypnotherapy. The effectiveness of each of those therapies varies and most have been used at one point in time to test their effectiveness in dermatological conditions. Shenefelt (2000) found that many dermatological disorders including acne excoriée, alopecia areata, atopic dermatitis, congenital ichthyosiform erythroderma, dyshidrotic dermatitis, erythromelalgia, furuncles,

Table 31.1 Armamentarium of cognitive-behavioural hypnotherapy

1. Self-Hypnosis Training
2. Relaxation Skills Training/Breathing Exercises
3. Hypnotic & Autosuggestion Skills Training
4. Therapy Recordings (Self-Hypnosis CDs, etc.)
5. Alert Hypnosis
6. Hypnotic Desensitisation Therapy (Systematic Desensitisation/Regression Desensitisation)
7. Aversion Therapy/Covert Sensitisation
8. Assertiveness Skills Training (Conditioned Reflex Therapy)
9. Direct Therapeutic Suggestions & Positive Goal Imagery
10. Mental Rehearsal of Coping Skills
11. Imaginal Exposure Therapy & Response Prevention
12. Thought-Stopping & Thought-Substitution (Habit Reversal)
13. Mental Rehearsal of Positive Cognitions
14. Mindfulness Training (Body Scan) & Thought-Spotting (Gestalt Therapy)
15. Self-Monitoring (Thought Forms, etc.)
16. Socratic Questioning & Verbal Disputation (of Thinking Errors & Negative Cognitions)
17. Shaping Behaviour by Positive Reinforcement of Successive Approximations (Coping to Mastery)
18. Ego-Strengthening/Self-Efficacy Suggestions & Imagery
19. Structured Client Assessment & Evaluation
20. Psycho-education/Education in the Therapeutic Model
21. Tension Control/Progressive Muscle Relaxation
22. Biofeedback Training
23. Cue-Controlled Emotions (“Anchoring”)
24. Cognitive Mood Induction/Rational Emotive Imagery
25. Resilience & Relapse Prevention Training
26. Linguistic Training (General Semantics)
27. Acting “as if”/Role-Taking
28. Covert Role-Modelling Imagery
29. Collapsed Coping Statements/Symbol Suggestion
30. Role-Play/Behavioural Psychodrama (in Autosuggestion Training)

glossodynia, herpes simplex, hyperhidrosis, ichthyosis vulgaris, lichen planus, neurodermatitis, nummular dermatitis, postherpetic neuralgia, pruritus, psoriasis, rosacea, trichotillomania, urticaria, verruca vulgaris, and vitiligo can be treated with hypnosis. Further studies found that hypnosis can be beneficial not only for particular conditions but also as a helping tool for patients to better cope with certain dermatological procedures (Shenefelt 2003). Some of the latest research shows the integration of various evidence-based approaches such as cognitive-behavioural therapy with mindfulness and meditation techniques to help produce focalisation and specific improvements in skin disorders through psycho-neuro-endocrine-immunologic mechanisms.

Practice Point
In modern times hypnosis is usually used in combination with psychotherapeutic approaches.

What Is Cognitive-Behavioural Hypnosis?

Hypnosis takes advantage of various naturally occurring psychological and physiological states to evoke positive emotions and rehearse behaviour change. Most people can be hypnotised, exceptions are people who do not wish to be hypnotised or subjects with cognitive or developmental conditions that prevent them from being hypnotised. When people experience hypnosis, they are in a state of increased suggestibility and respond particularly to positive suggestions. Hypnosis is not a state of sleep, as most people report being aware of everything that happens. Also, hypnosis is not mind control as subjects cannot be made to do anything against their will. This is not a one-sided process but a collaboration between a therapist and client much like it is in other talking therapies. Cognitive therapies are generally defined as those therapies which emphasise the role of "cognitive mediation" in learning theory. Cognitive mediation is the theory that the client's current patterns of thinking, especially beliefs and conceptualisations, determine how they will respond to specific stimuli or cues. Therapies that emphasise the role of (classical and operant) conditioning principles in learning theory are generally deemed behavioural in orientation. Cognitive-behavioural hypnosis (CBH) utilises those therapies and combines them in an evidence-based approach.

Clinical Application in Psychodermatology?

It is important to consider the diagnosis of the patient in a psychodermatology clinic before selecting a treatment modality. For a basic understanding, patients can be broken into two groups: those with a primary psychiatric and secondary dermatological condition, and those with a primary dermatological and secondary psychological condition. Then healthcare professionals can be separated into two groups: staff with no formal talking therapy training and staff with formal talking therapy training. The first group of staff will be able to administer basic hypnosis

interventions to patients relating to relaxation and attention. Those interventions can be administered to all patients to reduce their anxiety and facilitate trust with the multidisciplinary team. More complex interventions such as cognitive-behavioural hypnotherapy will require a qualified professional such as a psychiatrist or psychologist that has completed training in the particular talking therapy that can be paired with hypnosis. The results of an 18-study meta-analysis showed that cognitive-behavioural therapy paired with hypnosis substantially enhanced treatment outcomes in a sample of patients across conditions treated by psychologists. Additionally, hypnotherapy is very brief compared to other talking therapies with patients showing improvements only after a few sessions.

Practice Point
Patients can be broken into two groups: those with a primary psychiatric and secondary dermatological condition and those with a primary dermatological and secondary psychological condition.

Using Basic Hypnosis?

The following script can be administered by virtually any member of a psychodermatology team (Table 31.2). There is no need for formal training, however, having

Table 31.2 Basic hypnosis relaxation script

Basic hypnosis relaxation script
[Preparation] You are about to begin a very simple process of hypnosis that has been carefully designed to help you relax… very deeply …Hypnosis itself is not relaxation…it is more about focusing your attention, your imagination, and expectations… However, relaxation is one of the simplest and most useful things you can do with hypnosis… People get out of hypnosis more or less what they put into it… So, think of this as a personal experiment, an opportunity to show yourself how deeply you can relax when you really put your heart and soul into the present task… Here you will learn more about relaxation and how to get the most out of it… Remember there are many factors that can influence how well you relax on a particular occasion so make a note that individual experiences may vary…
[Posture] Now…Settle down and relax… let your eyes close… Take a moment to make yourself comfortable, have your feet side-by-side flat on the floor and your hands resting on your lap… It helps to adopt a fairly neutral and balanced position… Become aware of the sensations in your body such as your heartbeat and your breathing…. Now ... Adjust your position to make sure you are completely at ease… Deep relaxation is accompanied by a pleasant feeling of contentment and peace of mind, as if you are smiling to yourself inside… With a little practice these responses will become more and more powerful and automatic… For over a hundred years, hypnotists have associated relaxation with eye-closure and the use of the trigger word, 'sleep'…. but those are just signals to relax… Hypnosis is not sleep…You may feel fully aware or partially asleep in hypnosis, either way that is fine… For the time being, imagine that any sounds you hear or sensations you feel simply help you to go deeper into hypnotic relaxation…

Table 31.2 (continued)

Basic hypnosis relaxation script
[Induction] Now listen to the following suggestions in turn, allowing yourself to accept them completely and utterly… Imagine the feelings as powerfully as you can… Beginning now… My whole body is becoming relaxed and comfortable. My whole body is becoming relaxed and comfortable. My whole body is now relaxed and comfortable. My legs are becoming relaxed and comfortable. My legs are becoming relaxed and comfortable. My legs now feel relaxed and comfortable… My arms are becoming relaxed and comfortable. My arms are becoming relaxed and comfortable. My arms now feel relaxed and comfortable… My arms and legs are becoming relaxed and comfortable. My arms and legs are becoming relaxed and comfortable. My neck and shoulders are becoming limp and relaxed. My neck and shoulders are becoming limp and relaxed. My neck and shoulders now feel limp and relaxed… My facial expression is becoming peaceful and relaxed. My facial expression is becoming peaceful and relaxed. My facial expression is now peaceful and relaxed… My whole body is becoming relaxed and comfortable. My whole body is becoming relaxed and comfortable. My whole body now feels relaxed and comfortable… My breathing is becoming gentle and soothing. My breathing is becoming gentle and soothing. My breathing now feels gentle and soothing… My heart is becoming peaceful and contented. My heart is becoming peaceful and contented. My heart now feels peaceful and content… My mind is becoming peaceful and contented. My mind is becoming peaceful and contented. My mind now feels peaceful and contented… Everything is becoming peaceful and relaxed. Everything is becoming peaceful and relaxed. Everything is now is peaceful and relaxed… Good… Now just rest and imagine going much deeper into hypnotic relaxation….
[Breathing] Good… Now do nothing… Just relax your body… Relax your mind… Focus on your breathing... particularly on the out-breath… With each exhalation of breath, just imagine that you are relaxing over and over again, deeper and deeper, and deeper… If it helps, repeat the word 'Peace' or another calming word to yourself, with each release of breath… Now I'm just going to allow you to continue relaxing in silence, contemplating each exhalation of breath, and repeating the word 'PEACE', or whichever word you choose to focus upon to keep letting go deeper into relaxation of the body and mind… Focusing on the breathing and the use of that word… Beginning now…[PAUSE] Good... Now just allow your mind to gradually become silent…
[Emerging] In a moment, I am going to begin counting from one up to five. With each number I count, you are emerging from a wonderful experience of deep, therapeutic relaxation. Beginning now... on the count of one, from the tips of your toes, all the way up to the top of your head, you feel calm, relaxed, and confident. You feel good. On the count of two, as you begin to breathe more deeply, with every breath you now take, imagine you are breathing more calm, relaxation, and confidence into your body. On the count of three your mind is calm, relaxed, and confident; your thinking is powerful, positive, solution-focused, and self-aware. On the count of four, your eyes beam with the calm, relaxation, and confidence that you now project to other people. Your eyes want to move, they want to flutter and open, and look around the room. Now on the count of five opening your eyes, as you take a deep breath and stretch, begin moving your arms and your legs. Now taking those positive feelings forward with you… into action, into the future, and into your daily life.

some basic therapeutic and helping skills is a benefit. The major point to remember is to give plenty of time and not to rush through the script. This script is not intended as psychotherapy and should not be used as such. Instead, its purpose is to introduce patients to a simple but effective relaxation technique that they can use by themselves. To make it easier for patients to use it in the future, the first use of the script can be recorded by the physician so the client can use it at later times or use it as a template for a recording of their own.

Relaxation Techniques

What Is Relaxation?

Relaxation can mean several things: For some people, relaxation is simply when they are asleep. For the current context, however, we look at relaxation as an activity scheduled by the individual, during which stressors are reduced or completely eliminated. There are various techniques that can promote relaxation, certain activities can also help individuals relax such as when listening to music or gardening.

When to Use Relaxation Techniques in Psychodermatology?

Relaxation techniques are useful tools in psychodermatology as they are easy to teach to patients and can then create benefits for a long time with little effort. Additionally, their costs are minimal. Most patients attending psychodermatology clinics have various reasons to be stressed. Firstly, the nature of their condition, for example, a patient with acne excoriée and resulting anxiety can have various levels of muscle tension. In that case, progressive muscle relaxation can be a good choice as a technique. A body dysmorphic disorder patient that has no visible changes on their skin can benefit from mental imagery techniques where they can relax by visualising themselves in a calming environment. Patients do not need to apply a relaxation technique directly for a particular condition - some might have difficulties in other aspects of their life or be frustrated with the care they have received so far. In those situations, relaxation techniques can be used to help them cope with stress and improve their overall quality of life. The process of instructing patients in a relaxation technique can seem trivial, but it is important in strengthening the trust between patients and the multidisciplinary team. Additionally, patients will be able to see the effects almost immediately, which can boost their confidence and strengthen their resolution to keep with other treatments given by their caregivers.

Practice Point
Relaxation techniques can have benefits for a variety of reasons in patients with psychodermatological problems.

Relaxation techniques
Progressive muscle relaxation
Diaphragmatic breathing
Meditation
Visualisation
Mindfulness
Yoga
Meditation
Exercise
Music

The Relaxation Response

The 'Relaxation Response' is a term coined by the cardiologist Dr. Herbert Benson. He describes the relaxation response as the opposite of the 'fight-or-flight' stress-response that naturally occurs in the body. Therefore, the relaxation response is a person's ability to encourage their body to release chemicals and brain signals that make the muscles and organs slow down and boost blood flow to the brain, entering a relaxed state. Since psychodermatology patients experience significant levels of stress, this technique can prove a promising tool in their care. Patients can be instructed in the technique and then are advised to apply it daily for 10–20 min in the morning. Dr. Benson's research points out that even minimal practice can have positive effects on the individual.

The relaxation response
1. Sit quietly in a comfortable position.
2. Close your eyes.
3. Deeply relax all your muscles, beginning at your feet and progressing up to your face.
Keep them relaxed.
4. Breathe through your nose. Become aware of your breathing.
As you breathe out, say the word, 'one'*, silently to yourself.
For example, breathe in ... out, 'one',—in .. out, 'one', etc.
Breathe easily and naturally.
5. Continue for 10–20 min.
You may open your eyes to check the time, but do not use an alarm.
When you finish, sit quietly for several minutes, at first with your eyes closed and later with your eyes opened.
Do not stand up for a few minutes.

The relaxation response
6. Do not worry about whether you are successful in achieving a deep level of relaxation.
Maintain a passive attitude and permit relaxation to occur at its own pace.
When distracting thoughts occur, try to ignore them by not dwelling upon them and return to repeating ‘one’.
With practice, the response should come with little effort.

Bibliography

Heap M and Aravind KK. Hartland’s medical and dental hypnosis. London: Churchill Livingston. 2002.

Shenefelt PD. Hypnosis in dermatology. Archives of dermatology. 2000;136(3):393–99.

Shenefelt PD. Biofeedback, cognitive-behavioral methods, and hypnosis in dermatology: is it all in your mind? Dermatologic Therapy. 2003;16(2):114–22.

Substance Abuse

32

Ilknur K. Altunay

Introduction

Substance abuse is defined as the harmful or hazardous use of psychoactive substances. This includes legal substances like alcohol and nicotine as well as illicit drugs such as heroin, marijuana, cocaine, etc. It also includes some prescription medicines and over-the-counter drugs, although there is no universally accepted definition. Excessive use of any substance causes a dysfunction of the reward system in the brain by making it highly active, thus both substance and behaviours become a major focus of the individual's life to the exclusion of other usual activities. Therefore, substance abuse may develop into dependence or addiction, which is characterized by tolerance and withdrawal symptoms. Dysfunctional reward circuitry leads to characteristic biological, psychological, social, and mental manifestations. The new revision of DSM-5 abandons the term "dependence," and substitutes it with the term "use disorder" which incorporates all physiological, behavioural, and cognitive elements for diagnosis and treatment. Although, "substance use disorder" (SUD) simply refers to heavy use of any substance over time, it should also be kept in mind that SUD is an illness associated with signs of dependency (Table 32.1).

SUD is a growing health problem and a major public health concern all over the world, because of the destructive effects for both the person and society. Today, substance abuse is a well-known risk factor for morbidity, disability, and premature mortality from adolescence to older ages. Although the prevalence of SUD peaks in adulthood, initiation of substance use most frequently occurs in adolescence. Older ages are at higher risk for poor health outcomes, and SUD can be the underlying reason of many serious disorders involving acute and/or chronic physical and

I. K. Altunay (✉)
Department of Dermatology and Venereology Clinic, Psychodermatology Liasion Unit, University of Health Sciences, Şişli Hamidiye Etfal Training and Research Hospital, Istanbul, Turkey

A. Bewley et al. (eds.), *Psychodermatology in Clinical Practice*,
https://doi.org/10.1007/978-3-030-54307-5_32

Table 32.1 Signs of dependency

1.	Craving for the substance
2.	Development of tolerance
3.	Use in larger amounts or for longer periods than intended
4.	Persistent desire or unsuccessful efforts to cut down
5.	Significant time is spent obtaining the substance or recovering from its effects
6.	Social, occupational, and recreational pursuits are given up or reduced because of substance use
7.	Use is continued despite knowledge of substance-related harm (physical or psychological)

psychiatric diseases. The risk of poor general and mental health outcomes such as hepatitis, cirrhosis of the liver, septicaemia, cardiovascular problems, endocarditis, subdural haematoma, infections, cancer, psychosis, and depression increases with age. Therefore, substance abusers have a substantially higher risk of death than the general population, in particularly older ages.

Practice Point
SUD is associated with significant comorbidities and poor health outcomes.

Mucocutaneous adverse effects are common and variable. They are mostly related to the type of substance used and its way of administration. Although there is no diagnostic skin finding for substance use, it can be said that any symptom or sign on the skin may indicate potential substance use after the exclusion of other aetiological factors. Some symptoms occur acutely and temporarily during the use of the substance (intoxication). When the effects of the substance are removed, the symptoms disappear, as in the case of conjunctivitis due to cannabis use or piloerection due to heroin use. Some skin findings are more frequent because of the long-term and repeated use of a substance. Moreover, some exhibit specific properties due to the administration route. For example, track marks/skin tracks are linked to the use of intravenous heroin and characterized by venous damage and thrombosis followed by scarring of the veins and hyperpigmentation of the skin in time. Skin popping scars are irregularly shaped, atrophic, hyper- or hypopigmented depressive and punctate lesions (Fig. 32.1a, b). Other non-specific dermatological manifestations of SUD include bacterial and mycotic skin infections, self-harm manifestations made by blade or razor, skin popping, punctures, ulcerations, necroses, and haematomas, foreign body granulomas, prurigo nodules, excoriations, shooting tattoos, burn scars, etc. (Fig. 32.2). Among substance users, tattoos may be representative symbols of different psychological dynamics and emotions such as anger, aggression, need for affection, or need of belonging to a group. They can include figures or letters of violence, death, and love. They may be applied both to hide scar tissue and mark the spot where injections can easily be made in the case of an emergency. Images are sometimes truly special markers among persons using drugs (Fig. 32.3a–f). In addition,

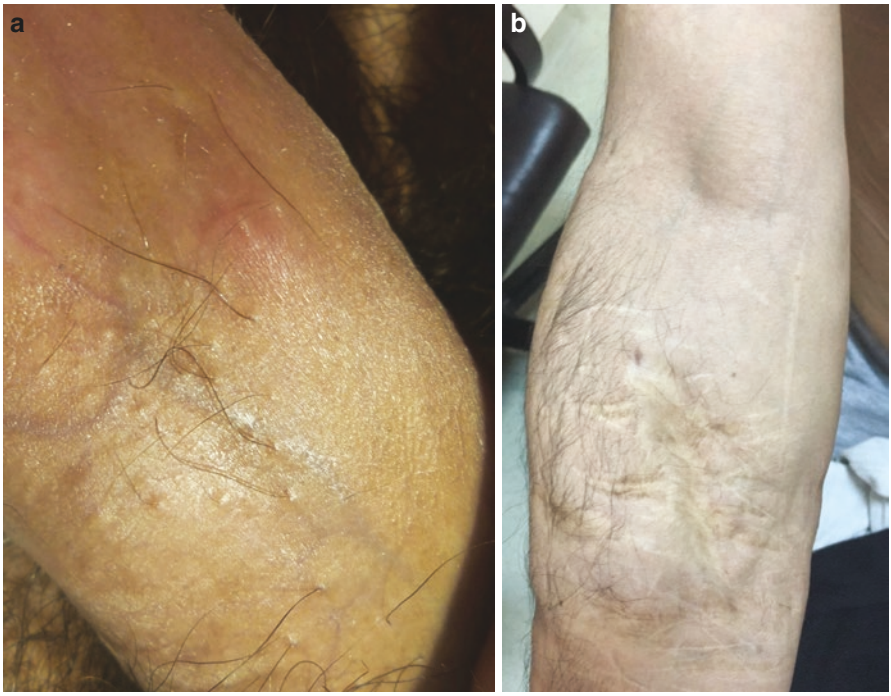


Fig. 32.1 (**a, b**) Skin tracks and skin poppings: linear and indurated lesions and irregular scars along the veins

generalized skin alterations such as acneiform and allergic skin lesions, acute generalized exanthemic pustulosis (AGEP), and different types of tattoos may be seen.

Patients may avoid to declare their substance abuse even in medical settings because of the pejorative perception of SUD in society. On the other hand, some patients may not be aware of being drug dependent and do not mention substance use in their history for that reason. An internal disease like hepatitis or vasculitis, originating from substance abuse, may easily be overlooked by examiners because there are many potential underlying reasons and differential diagnosis is not simple. The skin often presents the earliest changes and clues in substance abuse and therefore dermatologists play a pivotal role in the recognition, early intervention, and treatment of SUD patients. Even if dermatologists cannot necessarily facilitate the primary treatment of these patients, they are a part of a multidisciplinary, team-based approach. After exclusion of other causes, dermatologists should consider the possibility of SUD. When suspecting substance abuse, one should investigate the patient's medical and psychiatric history, their social environment, and their family history. Evaluation of the patient's mental state is of vital importance besides a complete physical and neurological examination. Laboratory tests include urine samples and blood tests (liver function in

Fig. 32.2 Self-harm scars by razor and self-induced tattoo

particular). They are not generally necessary to confirm the diagnosis but can be used to monitor progress, abstinence, or deterioration. Generally, urine screening immunoassays are used as cheap and rapid screening tests; but patients can easily manipulate the sample, and many substances cross-react with other drugs. Blood, saliva, sweat, and hair investigations may infrequently be required to confirm the diagnosis.

Fig. 32.3 (**a**, **b**, **c**, **d**, **e**, **f**) Tattoos may have symbolic or cryptic meanings in abusers

Treatment options can change from simple counselling to hospital-based detoxification treatments. Psychological interventions and rehabilitation programs are important for both underlying reasons leading to substance abuse and associated mental illnesses. The treatment used is based on the type and severity of substance abuse.

Substances: Clinical and Cutaneous Findings, Diagnosis and Treatment

Alcohol

Alcohol is one of the most frequently abused substances. Physiological effects of alcohol on the cardiovascular system, cellular immunity, and homeostasis are closely related to its cutaneous manifestations. Alterations in the skin, associated with the negative effects on these systems occur both de novo or as exacerbations of a preexisting disease. Although numerous skin manifestations are described due to alcohol abuse, most of them are non-specific.

Clinical and Cutaneous Findings

The gastrointestinal system is one of the most affected systems by alcohol abuse besides the cardiovascular system. The liver is directly affected by alcohol abuse and toxicity, resulting in fatty liver, alcoholic hepatitis, and cirrhosis. Especially skin findings such as jaundice, pruritus, hyperpigmentation, and nail changes such as red lunula, koilonychia, or clubbed nails, may be consequences of alcoholic liver disease. Heavy alcohol consumption is also a potential risk for acute and chronic pancreatitis. Purpura on the flank (Cullen's sign), subcutaneous extravasation of haemorrhagic peritoneal fluid (Grey Turner's sign), and subcutaneous fat necrosis, characterized by painful subcutaneous nodules and plaques, may occur secondary to pancreatitis. Additionally, oral mucosa involvement, malabsorption signs associated with the deficiency of water-soluble vitamins B and C, pellagra, and porphyria cutanea tarda are well-recognized cutaneous findings, resulting from gastrointestinal system involvement in alcohol patients. Glossodynia, angular cheilitis, atrophic glossitis, seborrheic dermatitis-like facial eruption, and hyperpigmentation of the hands and feet are typical signs of vitamin B deficiency. Scurvy presents itself with petechiae, follicular hyperkeratosis with corkscrew hairs, and swollen blueish gums. Cardiomyopathy, hypertension, and cerebrovascular hemorrhages may be seen. Regarding vascular changes, one should bear in mind that most of them can emerge secondary to underlying liver disease but they are non-specific for liver disease. Spider angiomas, palmar erythema, caput medusae, plethoral face, flushing, and unilateral nevoid telangiectasia are common (Figs. 32.4 and 32.5). Infections, skin cancers, and impaired wound healing are at an increased rate because of immunosuppression becoming more common.

Exacerbation of preexisting diseases has been described especially for inflammatory dermatoses including psoriasis, rosacea, discoid eczema, seborrheic dermatitis, and hidradenitis suppurativa. All these diseases have a psychological component and a negative impact on quality of life, with depression and anxiety as common secondary features. Alcohol is well known to be a direct inducer of depression. It also increases anxiety and binds at the same GABA receptors that as many commonly used anxiolytics, reducing their efficacy. Although alcohol is known to

Fig. 32.4 Alcohol-induced palmar erythema

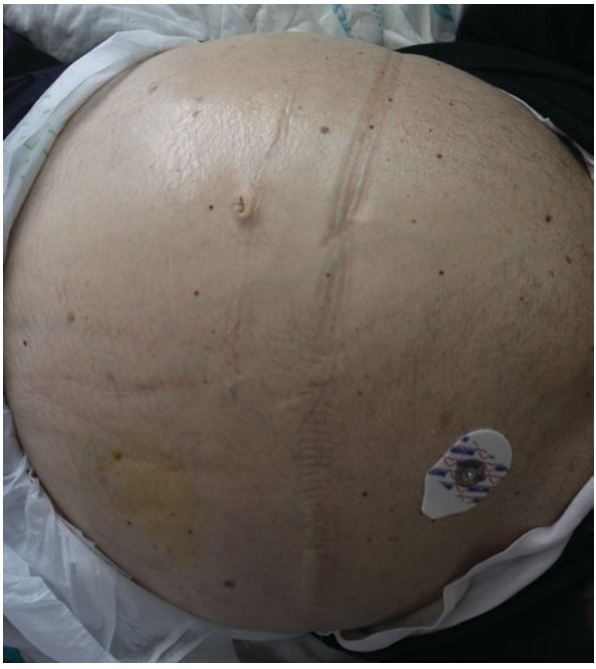


Fig. 32.5 Alcohol-induced cirrhosis and mild caput medusa due to alcohol abuse

induce proinflammatory cytokines, lymphocyte activation, and keratinocyte proliferation (thus activating the inflammatory cascade), it remains to be clarified whether alcohol abuse is a trigger or a secondary outcome of psychological factors based on chronic inflammatory disease. It seems likely that there is a bidirectional relationship between them.

Practice Point
There is a strong association between alcohol abuse, depression and anxiety, and exaggerated dermatological symptoms.

Psoriasis is one of the best-studied diseases regarding the relationship between stress and alcohol misuse of patients. Patients with psoriasis are well known to have a high prevalence of harmful drinking. While chronic alcohol exposure causes inflammatory cell responses to increase and thus an immune dysregulation by TNF-alfa production, there is some evidence that on psychological measurements, low scores for positivity and elevated scores for impulsivity in psoriasis patients may lead to alcohol abuse. Additionally, these patients often exhibit destructive mechanisms of coping such as drinking alcohol, smoking, and overeating. Therefore, the chicken and egg dilemma continues in psoriasis, too.

Diagnosis

The identification of patients drinking alcohol in a harmful manner is vital, not least in psoriasis patients, because the presence of comorbidities such as fatty liver, obesity, and cardiovascular disorders as well as treatment responses are considerably affected by alcohol abuse. However, the detection of such patients may not be easy due to misleading self-reporting with patients underestimating their alcohol intake. Therefore, alcohol screening questionnaires like AUDIT are recommended for all patients with moderate to severe psoriasis or other dermatological patients when harmful alcohol use is suspected. They incorporate questions about how often people drink, how often they drink heavily, whether their drinking is causing harm or injuries, if they ever feel guilty about their drinking, and whether they have tried to cut down.

Blood alcohol levels, breathalyzer test results, urine drug screens, and less commonly hair and saliva analysis can be used to assess patients for possible alcohol use. Alcohol levels at specific times can be estimated as the rate of metabolism is well known, as long as an alcohol blood level can be established within hours of intoxication. An alcohol (and drug) screen may be useful in evaluating an adolescent with school problems, or in occupational or traffic accidents, domestic violence, or other trauma situations as well.

Treatment

Since withdrawal symptoms may be life-threatening, an inpatient detoxification program in a hospital or treatment facility is often necessary to stabilize the person. Generally speaking, therapies with Chlordiazepoxide or other benzodiazepines are

targeted at acute withdrawal symptoms (commonly up to 5 days). Chronic relapsing disorder characterized by compulsive heavy alcohol drinking requires different strategies than acute detoxification. Treatment with either drugs such as Disulfiram, Naltrexone, Nalmefene, and psychotropics, or cognitive-behavioral therapies are elementary in alcohol dependency treatment programs. In addition, more novel therapies like Gabapentin and Pregabalin are suggested. Patients with chronic alcohol use may develop alcoholic hallucinosis with specific visual and auditory hallucinations. The mainstay treatment for such hallucinations is abstinence, as other pharmacological strategies are of limited efficacy.

Practice Point
Think of alcohol or drug use in cases of unexplained injuries, violence, and accidents.

Opioids

Opioids include heroin and legal narcotic analgesics such as morphine, codeine, Methadone, Tramadol, Fentanyl, etc. Essentially, these substances, except for heroin, are prescribed to ease moderate or severe pain in medicine. They are either opiates, opioid derivatives, or they get metabolized into opioids. Nevertheless, misuse and abuse are the most risky side effects of these drugs. Heroin is three times more potent than morphine and also has the fastest onset of action among opioids. Although it is used via intravenous injection, there is a recent tendency to sniff, snort, or smoke it.

Practice Point
Remember that commonly prescribed medication like co-codamol contains opiate metabolites.

Clinical and Cutaneous Findings

Overdose with prescription or illicit opioids is an important problem. During long-term use, cardiac and pulmonary disorders can occur. As parenteral drug users commonly use and share dirty needles, this is a sign of morbidity. Mortality can result from blood-borne viral, bacterial, or fungal infections. Skin and soft tissue infections consisting of abscesses, cellulitis, necrotizing fasciitis or myositis, or disseminated candidiasis are reported complications linked to intravenous injections, besides viral hepatitis and HIV infections. Injection anthrax by bacillus anthracis may rarely occur. Alternative injection techniques known as skin popping (intradermal or subcutaneous), booting (drawing blood into the syringe before injection), and speedballs (heroin) can also promote infections. Skin popping is considered the main risk factor for botulism. Skin popping leaves round, atrophic, and partly hyperpigmented scars while repeated non-sterile intravenous injections and

inflammation of veins lead to hyperpigmentation and scarring, which is called skin tracks or track marks. Heroin addicts can have immediate and generalized pruritus due to histamine liberation. Dry mouth, dry skin, rhinorrhea, and increased secretion of tears have been described. Sometimes pseudoacanthosis nigricans and hidradenitis suppurativa may be encountered in heroin addicts.

Diagnosis

The presence of opiates (derivatives of the opium poppy) or their metabolites in the blood, urine, hair, or saliva can be detected by inexpensive immunoassay screening tests in the blood up to 3–12 h, in urine up to 1–3 days, in hair up to 7–90 days, and in saliva up to 3–24 h. Gas chromatography/mass spectrometry is a more expensive laboratory test that is available for the detection of synthetic opioids, not usually included in screening immunoassays.

Treatment

Opioid agonists and antagonists are used for abstinence initiation, withdrawal treatment, and relapse prevention. Agonists include Methadone and Buprenorphine, antagonists are Naloxone and Naltrexone. α-2 adrenergic agonists (Lofexidine, Clonidine), Tramadol, N-methyl aspartate receptor antagonists (Memantine, Ketamine, Dextromethorphan, etc.), and corticotropin-releasing hormone antagonists are under investigation for the treatment of opiate dependency.

Stimulants

Amphetamine and cocaine are well-known examples of this substance group.

Cocaine

Cocaine is known under various names including coke, blow, coca, nose candy, snow, and flake. It has two forms as base and salt in illegal practices and is used by inhalation or intravenously. When inhaled intranasally, the toxic vapors are rapidly absorbed into the bloodstream. When injected intravenously, it goes directly to the bloodstream which increases the intensity of its effects.

Clinical and Cutaneous Findings

General health problems by stimulants are similar to other illicit substances. Cardiac and pulmonary problems, stroke, convulsion, seizure, headache, and mood disorders are possible side effects. The most frequent and suggestive manifestations of cocaine abuse are mucocutaneous complications as a result of nasal administration. Cocaine-induced destructive lesions of the midline face are the most common among them. Snorted cocaine also leads to corneal sensitivity, neutrophilic keratitis, and corneal damage. Oral mucosa involvement is frequently seen. Cocaine-induced gingivitis-periodontitis, necrotizing sialometaplasia, thermal burn or cheilitis, acute mucositis, lip swelling, mucosal erosive lesions, and even malignant transformation of oral mucosa epithelium have been reported. The free based form of cocaine is called “crack.” This form is more intense and more addictive. Palmar and digital

hyperkeratosis from holding the hot crack, and loss of the lateral eyebrow via the hot steam rising from the crack pipe may be seen in cocaine abusers. Administration via injection may result in skin and soft tissue infections including erysipelas, abscesses, necrotizing fasciitis, bacteremia, and sepsis.

Vasoconstriction is a secondary effect of α-adrenergic stimulation of cocaine and might cause Raynaud's phenomenon, digital gangrene, and distal ulcers. Purpuric rash may be seen in different skin areas. Pyoderma gangrenosum (PG) is a well-known skin manifestation particularly cocaine is adulterated with levamisole. Although lesions of PG are similar to those of classical PG, they can be generalized and refractory to treatment.

In addition, isolated case reports of severe mucocutaneous side effects consisting of Stevens–Johnson syndrome, acute generalized exanthematous pustulosis, angioedema-urticaria, pemphigus vegetans, and vulgaris, Churg–Strauss vasculitis, and Henoch–Schönlein vasculitis exist.

Psychiatric comorbidity is frequent in cocaine abuse. Cocaine intoxication can lead to acute paranoia, agitation, and aggression. Depression and anxiety are common medium-term effects. It is also common for cocaine users to have a period of intense low mood after acute use, which increases the risk of addiction because more use of cocaine appears to alleviate low mood, at least temporarily. Psychiatric disorders co-occurring with cocaine dependence are associated with a poor prognosis because they are associated with a worse response to treatments. A psychodermatological disease that can be directly caused by stimulant intoxication is delusional infestation. If the predominant symptom is a crawling sensation underneath the skin after the consumption of stimulants, it used to be called *cocaine bugs*. Illicit substances, particularly stimulants, are major trigger factors for delusional infestation.

Practice Point
Harmful stimulant use should always be considered if patients present with delusional infestation.

Diagnosis

Cocaine abuse may not be easy to diagnose clinically, although there are numerous and characteristic skin findings that are not considered pathognomonic. The best way is the toxicological screening of urine and blood specimens. It is the drug least likely to have false-positive results in testing procedures. Gas chromatography/mass spectrometry can isolate benzoylecgonine and cocaine. While benzoylecgonine can be detected up to 3 days after cocaine administration, cocaine and its metabolites are cleared rapidly from the blood. Furthermore, a new technique is the analysis of hair samples by electron microscopy. Ribbon-like hair fibers and balloon-like enlargement of hair shafts may suggest cocaine abuse.

Treatment

Treatment is often not fully effective. However, a number of drugs such as Modafinil, Ondansetron, NAC, Atomoxetine, and Topiramate have been investigated and appear

to be promising. Haloperidol or other antipsychotics can be used in cases of acute intoxication to reduce symptoms of paranoia, agitation, or delusional infestation.

Amphetamine and Methamphetamine

The main basis of what is widely known as "ecstasy" or "XTC" is MDMA (3,4-methylenedioxy-N-methylamphetamine). It is a sympathomimetic amphetamine similar to serotonin, acting both as a stimulant and as psychedelic associated with intense pleasure and energy, especially tactile stimulation. Serotonin syndrome and hyponatraemia are well-known possible toxic effects of MDMA. Nausea, muscle cramping, fever, headache, and jaw pain are likely to occur. Acute myocardial infarction, aortic dissection, paranoia, and serotonin syndrome have been reported as short-term side effects. Memory impairment and psychiatric disorders may occur in the long run, especially depression, anxiety, and occasionally mania. There are few cutaneous signs of ecstasy.

Clinical and Cutaneous Findings

The most common cutaneous findings are *ecstasy pimples*, clinically similar to perioral dermatitis. Multifocal oral ulcerations seen particularly in young patients should be a warning. Guttate psoriasis after the ingestion of ecstasy has been also reported.

Another variety of stimulants is Methamphetamine, which blocks the reuptake of monoamine transporters like cocaine, but also stimulates dopamine release and has a longer duration of action. Methamphetamine is used for the treatment of attention deficit and hyperactivity disorder (ADHD), and its illicit use requires much higher doses than those prescribed medically. As a powerful stimulant, Methamphetamine, even in small doses, can increase wakefulness and physical activity and decrease appetite. Methamphetamine can also cause a variety of cardiovascular problems, including rapid heart rate, irregular heartbeat, and increased blood pressure. Hyperthermia (elevated body temperature) and convulsions may occur with methamphetamine overdose. The acute administration of large amounts of the substance can lead to a severe psychosis called "MA psychosis." Long-term use can cause irreversible neuronal damage and neuropsychiatric manifestations. Dermatological problems are associated with oral mucosa problems, including xerostomia, and severe dental problems like dental caries and decay, which are called *meth mouth*. Cutaneous side effects include xerosis, pruritus, intense body odor, hyperhidrosis, and premature aging. Repetitive skin picking causing disfiguring ulcers and premature aging on the face, acne excoriee, and lichenoid drug eruptions have been reported.

Diagnosis

Urine, saliva, and blood screening tests are used besides physical, dermatological, and psychiatric clinical evaluations.

Treatment

There are currently no medications that counteract the specific effects of methamphetamine or that prolong abstinence from and reduce the misuse of methamphetamine by an individual addicted to the drug. Modafinil, bupropion, rivastigmine,

and lobeline are being evaluated as treatment options. Behavioural treatments and interventions to engage in treatment and maintaining abstinence currently appear to be the best treatment options.

Cannabis and Cannabinoids

Cannabis is obtained from the dried buds and flowers of the plant *Cannabis sativa*. The substance is the most frequently self-administered illicit drug and known as marijuana, pot, herb, weed, ganja, and Mary Jane commercially. Cannabinoids are compounds unique to cannabis and over 100 different cannabinoids have been identified with different psychoactive effects, from psychosis to anxiolytic properties. Synthetic cannabinoids are psychoactive substances with an agonistic effect on cannabinoid receptors, which are similar to the active metabolite of cannabis. There are cannabinoid receptors in the skin, too. Products containing synthetic cannabinoids are called differently in various countries, such as *spice* in Europe, *K2* in the USA, and *Bonsai* or *Jamaica* in Turkey. They are usually smoked as a cigarette or via pipe or bong.

Clinical and Cutaneous Findings

Common systemic complications are associated with the cardiovascular system and psychiatric manifestations. Acute intoxication and withdrawal symptoms may rarely necessitate hospitalization due to cardiac or respiratory side effects or seizure activity. Chronic cannabis abuse can cause cannabis arteritis as a subtype of thromboangiitis obliterans, peripheral arterial disease, Raynaud's syndrome, and digital gangrene. Cannabis arteritis can be differentiated from atherosclerotic arteritis by doppler ultrasound. Cannabis hyperemesis syndrome is one of the complications of chronic cannabis use. Erythema abigne linked to hyperemesis and compulsive hot showers for symptomatic benefits have been reported. Oral cancers, oral stomatitis, and candidiasis, cannabinoid allergy from mild urticarial reactions and pruritus to severe angioedema, AGEP, and hair shaft anomalies have been well documented with cannabis abuse. Acne varieties (a. vulgaris, a. keloidalis, a. excoriee), blade scars, and different and multiple tattoos are common.

Psychiatric complications of long-term cannabis use are lethargy and avolition, paranoia, hallucinations, agitation, depression, and loss of motivation in long-term and heavy use. Cannabis use can trigger psychotic episodes in people with comorbid schizophrenia.

Diagnosis

Cannabinoids can be detected in the urine for as many as 21 days after use; however, 1–5 days is the normal urine-positive period. Enzyme immunoassay or radioimmunoassay is the primary method for urinalysis detection. These methods are also beneficial for confirmation of abstinence. Moreover, blood samples and saliva testing, a newer technology for detection, may be used. Hair analysis is not a sensitive enough tool to detect cannabinoids.

Treatment

Although there are some investigations concerning novel medications affecting cannabis use disorder, inpatient and outpatient rehabilitation and behavioural therapies are the best options at present. Psychosocial interventions are beneficial in reducing the frequency of use and severity of dependence in abusers. Interestingly, the medical use of cannabis has been increasing to treat a variety of diseases and conditions including dermatologic diseases.

Hallucinogens

Gamma hydroxybutyrate (GHB, liquid ecstasy, liquid G, fantasy, party drug) and lysergic acid diethylamide (LSD) are conventional examples of this group. GHB is easily manufactured and available on the street as a powder or more often, a clear, odorless liquid sold in mini shampoo bottles. Misinformation on the internet deemphasizes the potentially lethal effects of GHB toxicity and boosts its popularity as a bodybuilding or club drug. Symptoms are dose-related with higher doses causing more severe respiratory and CNS depression. Bradycardia, hypotension, apnoea, vomiting, and hypothermia may also occur.

LSD is a serotonergic hallucinogen. Auditory and visual perceptual alterations are typical. Sympathomimetic effects are described with LSD, for example moderately increased blood pressure, heart rate, body temperature, and pupil size. Acute adverse effects include difficulty concentrating, headaches, dizziness, loss of appetite, dry mouth, nausea, imbalance, and feeling exhausted. However, it has been suggested that LSD within a therapeutic setting may be beneficial for patients with anxiety associated with severe illness, depression, or addiction via its action on the serotonin neurotransmitter system. Currently, these options lack sufficient evidence and are not in common use.

Diagnosis

GHB toxicity is a clinical diagnosis. Blood or urine testing for GHB is not routinely available in the hospital setting. Diagnostic confirmation via gas chromatography and mass spectrometry is possible but may take up to 7–14 days for results. A urine drug screen may aid in identifying or excluding coingestants. As with any potential intoxicated patient, a finger stick glucose, blood alcohol level, acetaminophen level, and salicylate level should be obtained.

Practice Point
Always consider that the patient may have ingested multiple substances at the same time.

LSD is ingested in quite small amounts. How long LSD can be detected in the body is variable. Routine forensic methods for confirmatory and quantitative testing for LSD employ high-performance thin-layer chromatography (HPTLC) and

Table 32.2 Common cutaneous side effects of different substances

Substance	Cutaneous side effects/signs
Alcohol	Plethoral face, flushing, jaundice, pruritus, palmar erythema, nail changes, hyperpigmentation of hands and feet, subcutaneous painful nodules, angular cheilitis, atrophic glossitis, the presence of exacerbated inflammatory dermatosis (psoriasis, acne rosacea, eczema)
Opioids	Track marks, skin poppings, skin infections (abscess, cellulitis, necrotizing fasciitis, etc.), pruritus, dry mouth, dry skin, rhinorrhea
Cocaine	Destructive lesions of midline face, oral mucosal disorders, petechial—purpuric rash, acral ulcerations, palmar-digital hyperkeratosis, delusional parasitosis
MDMA-Methamphetamine	Xerosis, pruritus, intense body odor, hyperhidrosis, pimples, dental decays, oral mucosal lesions, skin picking, acne excoriee
Cannabis-Cannabinoids	Oral cancers, oral stomatitis and candidiasis, cannabinoid allergy (mild urticarial reactions, pruritus, severe angioedema), AGEP, hair shaft abnormalities, blade scars, acne varieties, different and multiple tattoos
Hallucinogens	Dry mouth

different forms of gas chromatography/mass spectrometry (GC/MS) with detection limits set to approximately 0.4 μg/L. The practical (forensic) detection limits are as low as 0.1 and 0.25 ng/mL for LSD and N-desmethyl-LSD, respectively.

Treatment

LSD is physiologically well tolerated and psychological reactions can be controlled in a medically supervised setting, but complications may easily result from uncontrolled use. Acute LSD toxicity which can emerge with coma, hyperthermia, and bleeding may require appropriate supportive medical treatment.

Substance use disorder has devastating physical, dermatological, and mental consequences. Collaborative teamwork is needed in management and prevention. While different medical specialties including general practitioners, physicians, psychiatrists, and neurologists involved in the diagnosis and management of this global epidemic, dermatologists need to take a more active role than ever. There are numerous and various skin alterations, which need to be clarified for other aetiologies in these patients (summarized in Table 32.2). Not only physical and psychiatric problems, which can be difficult to identify in substance use, dermatological findings can help to recognize SUD patients in an early, reversible stage of SUD. Cutaneous stigmata caused by substances may help diagnose a covert or unreported substance abuse case. Dermatologists should be familiar with these cutaneous alterations and signs.