

# Textbook of Primary Care Dermatology

David Buckley  
Paola Pasquali  
*Editors*



Springer

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Editors

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*This book is dedicated to my wife, Áine, who is my greatest supporter and greatest critique.*

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My mentors in GP training in Dublin including Prof Fergus O Kelly, Dr Manne Berber, Prof Bill Shannon and Prof Andrew Murphy all instilled in me a love of general practice.

I spent 18 months working in hospital-based dermatology in Dublin after qualifying in medicine and received excellent teaching from some of the top Irish dermatologists at the time including Prof Sarah Rogers, Prof Frank Powell and Dr Sean O’Loughlin. My 2 years working in a rural mission hospital in “the bush” in Northern Kenya allowed me a wonderful opportunity to improve my surgical skills and knowledge of tropical medicine that was mostly learnt from my Kenyan doctor and nurse colleagues to whom I am eternally grateful.

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25 May 2020

David Buckley

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## About the Editors

**David Buckley** is a principal in general practice and GP trainer who has developed a special interest and expertise in dermatology and skin surgery. He obtained his basic medical degree from the National University of Ireland (Dublin) in 1982. He joined the Dublin Vocational Training Scheme in General Practice in 1983 and spent the next 3 years specialising in general practice which included 6 months in the dermatology department of the Mater Hospital, Dublin. After this, he spent 6 months training at the Dublin Skin and Cancer Hospital, Hume Street, Dublin. Dr Buckley then spent 2 years working as the chief medical officer in a large mission hospital in the north of Kenya with APSO (the Irish development agency) where he perfected his skills in surgery and tropical medicine. On return to Ireland in 1989, Dr Buckley established a new general practice combined with a primary care dermatology practice in his hometown of Tralee. Having begun on his own, the practice has since grown to include five doctors, three nurses, a clinical assistant and seven administration staff. He completed a year-long Diploma in Practical Dermatology in the University of Wales College of Medicine in 1990. He established the *Solas Dermatology and Laser Clinic* 2000 and changed the name to the *Kerry Skin Clinic* in 2018. Dr Buckley is a founding member of the Primary Care Dermatology Society of Ireland (1996) and the Primary Care Surgical Society (2012). He has been a member of the European Academy of Dermatology and Venereology since 1993. He has published eight scientific papers on community-based dermatology and cryosurgery in peer-reviewed journals, and this is his second book on primary care dermatology. He has also contributed chapters on two international textbooks on cryosurgery. He is an honorary lecturer in dermatology for the Irish College of General Practitioners and was awarded a fellowship of the Royal College of General Practitioners (London) in 2018 for his contribution to medicine.

**Paola Pasquali** is a dermatologist with a specialty in non-melanoma skin cancer treatment with special emphasis on cryosurgery, non-invasive imaging techniques and teledermatology. She is a Springer editor for *Skin Cancer: A Practical Approach*, *Cryosurgery: A Practical Manual and Photography in Clinical Medicine*. She is Past President of the International Society of Teledermatology and chairman of AAD International Affairs Committee and member of EADV School.

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**Part I**

**Overview**



# Dermatology in Primary Care

1

David Buckley

## Key Points

- At least 15% of GP (general practitioner) consultations involve dermatology problems.
- Dermatology in primary can be very different from that seen in hospital dermatology departments.
- Most dermatology textbooks are written by hospital based doctors and tend to include rare and dramatic skin complaints that are not normally managed by GP's.
- GPs can manage their patients holistically, dealing not only with the physical problems, but also the psychological and social aspects of their skin problems.
- Give realistic expectations as to how long it will take for a treatment to work.

## 1.1 Introduction

Skin, hair and nail problems are very common in the community. Studies have estimated that the overall proportion of the population with any form of skin disease was 55%, with 22.5% considered worthy of medical care (that is, moderate or severe) by a member of the primary care team such as a general practitioner, nurse practitioner,

public health nurse or community pharmacist [1]. Skin diseases account for 12–23% of all symptoms based requests for advice from community pharmacists [2]. At least 15% of GP consultations involve dermatology problems [3, 4].

There are more diagnoses in dermatology (>2000) than in any other speciality in medicine. Despite this, there are only ten common dermatology problems that make up 80% of skin problems seen in general practice [5, 6] (Table 1.1). This list will change depending on the population studied. There are many elements that need to be accounted for like ethnicity, availability to diagnostics and treatment services among others [7].

Most GPs should be able to confidently diagnose and manage most patients with mild to moderate forms of these 10 problems. In general, when faced with an unusual rash or lesion it is more likely to be an unusual presentation of a common problem, rather than a rare dermatology diagnosis.

## 1.2 The Patient's Perspective

The dermatology problem may be presented as the primary reason for the patient attending or may be part of a list of problems the patient brings to the GP. There are even situations where a patient may be embarrassed or reluctant to discuss their skin problems with their GP with the mistaken belief that the problem is trivial or that

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**Table 1.1** The 10 most common dermatological problems that make up 80% of skin problems seen in general practice

• Eczema
• Psoriasis
• Acne
• Urticaria
• Rosacea
• Skin infections (Bacterial, viral, fungal + parasitic)
• Wound care including leg ulcer
• Skin tumors (benign + malignant)
• Lichen planus
• Drug rashes

they should not be wasting their GP's time. The patient may tag a significant dermatology problem onto the end a busy consultation dealing with other unrelated problems. It is good practice to establish all the reasons the patient is attending at the start of the consultation ("are there any other problems") so the doctor can decide which problems are a priority and which may have to be left to another visit if there is not enough time.

It takes time to properly examine the skin and a GP should have it in order to diagnose and propose solutions to the patient. Conditions like acne or psoriasis can be quick to diagnose but explaining the aetiology and the management strategy to the patient is time consuming.

Writing a quick prescription without having time to deal with the patient's concerns, lifestyle changes and any non-prescription items is often doomed to fail.

For chronic skin conditions, patient empowerment is a must. People should know and understand their condition and learn how to live with it, knowing how to contact their GP should their condition get worse or if there is an emergency.

### 1.3 Websites and Apps

We live in a world with a lot of information available. This accessibility can be good because it empowers the patient on his/her own skin condition; it can have the drawback of giving incorrect information or the worse scenario of a skin condition. GPs can help in reducing fear and anxiety

associated with skin problems by guiding the patient to a good website that will explain the diagnosis and treatment in simplistic terms (see Chap. 67 on useful patient resources and websites).

### 1.4 Patient Information Leaflets

As treatment plans can be complicated, and the patient will only remember a small amount of what you tell them, patient information leaflets (PILs) explaining the treatments are very useful (Chap. 66).

### 1.5 The GP Perspective

GPs have the rare opportunity to see skin diseases in their early stages. This is why the description of certain diseases found in classical textbooks are not always how they present in GP's clinical practice. For example nodular BCCs (basal cell carcinoma) do not always present as the classical textbook description of a pearly white ulcerated nodule with raised rolled edges.

Many GPs, non-dermatologist hospital practitioners, pharmacists and nurses struggle with dermatology problems because of the lack of proper training in this area both at the undergraduate and postgraduate level. There is a lack of a simplified and accessible knowledge addressed to these professionals who are the first at attending a population with skin conditions.

Managing basic concepts and simple skills will allow non dermatologist to deal correctly with a large number of skin problems. The need for properly trained GP's becomes more imperative as the number of patients with skin problems increase (the population is living longer; elderly tend to have more skin diseases) and the number of dermatologists stays stagnant (even reduced by the outflow into cosmetic medicine).

A properly trained GP will not refer patients with relatively simple skin problems to dermatology OPD (outpatient department). This will

reduce overcrowding, long waiting times and lack of time for the dermatologists to get involved in training in dermatology in primary care. Teledermatology will further help GP's in getting advice on cases where there is no certainty.

The diagnosis and treatment of most dermatology problems rarely requires complicated or expensive imaging modalities such as CT scans or operating theatres with general anaesthesia. Treatment usually involves simple topical or oral treatments and many lumps and bumps can easily be excised or removed with basic surgical skills that are well within the scope of many GPs once the correct diagnosis is confirmed.

But simplicity comes from experience. Even treating a viral wart requires proper training and equipment.



**Fig. 1.1** Atopic eczema and psoriasis overlap in a 16 year old

## 1.6 Primary Care Dermatology

Dermatology in primary care can be very different from that seen in hospital dermatology departments [8]. In primary care diseases are often seen at an early stage when the clinical signs are vague and ill-defined. Patients may have overlap of more than one skin problem (e.g. acne and rosacea or psoriasis and atopic eczema) (Fig. 1.1). The clinical features may be altered by the patient's own interventions (self medications, scratching, etc.). Others patients have chronic skin problems that are unresponsive or only partially controlled with hospital treatments.

It is important to realise that while the skin specialist knows more about skin diseases, the GP has the advantage of knowing more about the patient ! GPs are ideally suited to manage patients with simple straight forward skin diseases as they can manage the patient holistically, dealing not only with the physical problems but also the psychological and social aspects of their skin problems. It is vital that skin problems are not dismissed as trivial or unimportant by the doctor. It is important to show empathy and understanding of the distress that skin problem can cause to

the patient. Primary care is probably the most appropriate place for chronic disease management and this is true for many common mild to moderate chronic dermatology problems. GPs should try to empower patients with chronic skin problems to manage at least some parts of their skin problem themselves. Nowadays, apps can help patients to manage their treatments, get advice on changing moles, etc.

Some skin problems may involve other organ systems (atopic children may have asthma and allergic rhinitis as well as eczema) and the GP can manage all aspects of the illness rather than just the skin component. Also many skin conditions can have associated underlying pathology (e.g. diabetes, arthritis, depression, etc.) and GPs can manage the skin diseases holistically, dealing with all the underlying ailments as well as the skin problem.

Nurses, pharmacists and GPs are ideally placed to promote skin wellbeing by applying health promotion and disease prevention strategies appropriately, including sun protection, occupational health advice and hand care.

This book will hopefully make dermatological knowledge for the most common skin conditions accessible and practical in a simplified manner.

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# History Taking and Examination

2

David Buckley

## Key Points

- It is important to assess the patients' **ideas** and **concerns** about their complaint and their **expectations** regarding treatment.
- The distribution of a rash may help clinch the clinical diagnosis.
- When examining scaly or scabby lesions it is advisable to remove the scale or scab to reveal what is underneath.
- In dermatology, taking the history from the patient may be carried out during or after the examination in order to save time and be more focused with questioning.
- Absolute recognition of a rash or a lesion on the initial consultation is desirable although not essential once the doctor can rule out more serious pathology such as a melanoma or a serious skin infection.

## 2.1 Introduction

The vast majority of skin problems in primary care can be diagnosed by taking a careful history and carrying out a thorough physical examination. A full body skin examination including the scalp, nails, genitalia and peri-anal skin may

need to be carried out looking for clues to the diagnosis or signs of skin conditions including cancer or pre cancer on other parts of the body.

## 2.2 History Taking

Diagnosis in dermatology, like most other specialities, involves careful history taking (Table 2.1) and a thorough physical examination. In dermatol-

**Table 2.1** History taking in dermatology

- |   |
|---|
| • History of the presenting complaint                             |
| – Onset, duration, periodicity                                    |
| – Site of onset, spread, distribution                             |
| – Aggravating or relieving factors                                |
| • Previous history and family history                             |
| – Skin problems   |
| – General medical problems  |
| • Medications   |
| – Oral or topical   |
| – Prescribed and non-prescribed                                   |
| • Allergies   |
| – To drugs  |
| – To food, clothing, footwear, jewellery, toiletries or cosmetics |
| • Occupational  |
| – Current and past  |
| • Sports and hobbies  |
| • Animal contacts   |
| – Including birds, fish and rodents                               |
| • Human contacts  |
| • Foreign travel  |
| • Recent stress   |
| • Alcohol intake  |

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ogy, taking the history from the patient may be carried out during or after the examination in order to save time and be more focused with questioning. It is important to elicit the timing of the rash, where it first appeared, how it spread and its response to various over the counter or prescribed treatments. Sometimes the patient may have no rash on the day they visit the doctor and the history may be the only way to try to make the diagnosis. A recurrent vesicular eruption that always comes up in the same area and heals without scarring is almost certainly due to herpes simplex (“Cold Sores”) but could be due to a fixed drug eruption which is very rare. A hive or nettle sting like itchy rash that can come and go on various parts of the body and does not stay in any one area for more than 12–24 hours is almost certainly due to urticaria. Symptoms such as itch (=allergy), pain (=infection), oozing or bleeding is also important to document. Even when the diagnosis is obvious (e.g. acne or psoriasis) a careful history is still important to identify any precipitating or aggravating factors and to assess the patients’ **ideas** and **concerns** about their complaint and their **expectations** regarding treatment (“ICE”). Sometimes mild disease, (e.g. acne,) may have to be treated more aggressively if the condition is having a severe impact on the patient’s quality of life. Questionnaires such as the “Dermatology Life Quality Index” can help identify how the skin problem is affecting the patient’s quality of life [1]. It is also important to note that sometimes patients have an obvious skin condition (like acne) but their motive for consultation is a different problem. Wait for the patient to tell you what is bothering him/her before jumping to conclusions.

A dietary history is sometimes necessary as excess sugar, fats, caffeine or alcohol may precipitate or aggravate certain skin problems. A dietary history may also reveal the possibility of an underlying food allergy or a dietary deficiency (e.g. calcium, iron, folic acid, etc.). A drug history (oral or topical, prescribed or over the counter) is also important particularly when faced with an unusual rash. Occupations, hobbies, pets, foreign travel, contact with others with similar symptoms, underlying medical problems (e.g. diabetes, SLE, etc.) and family history may also be relevant.

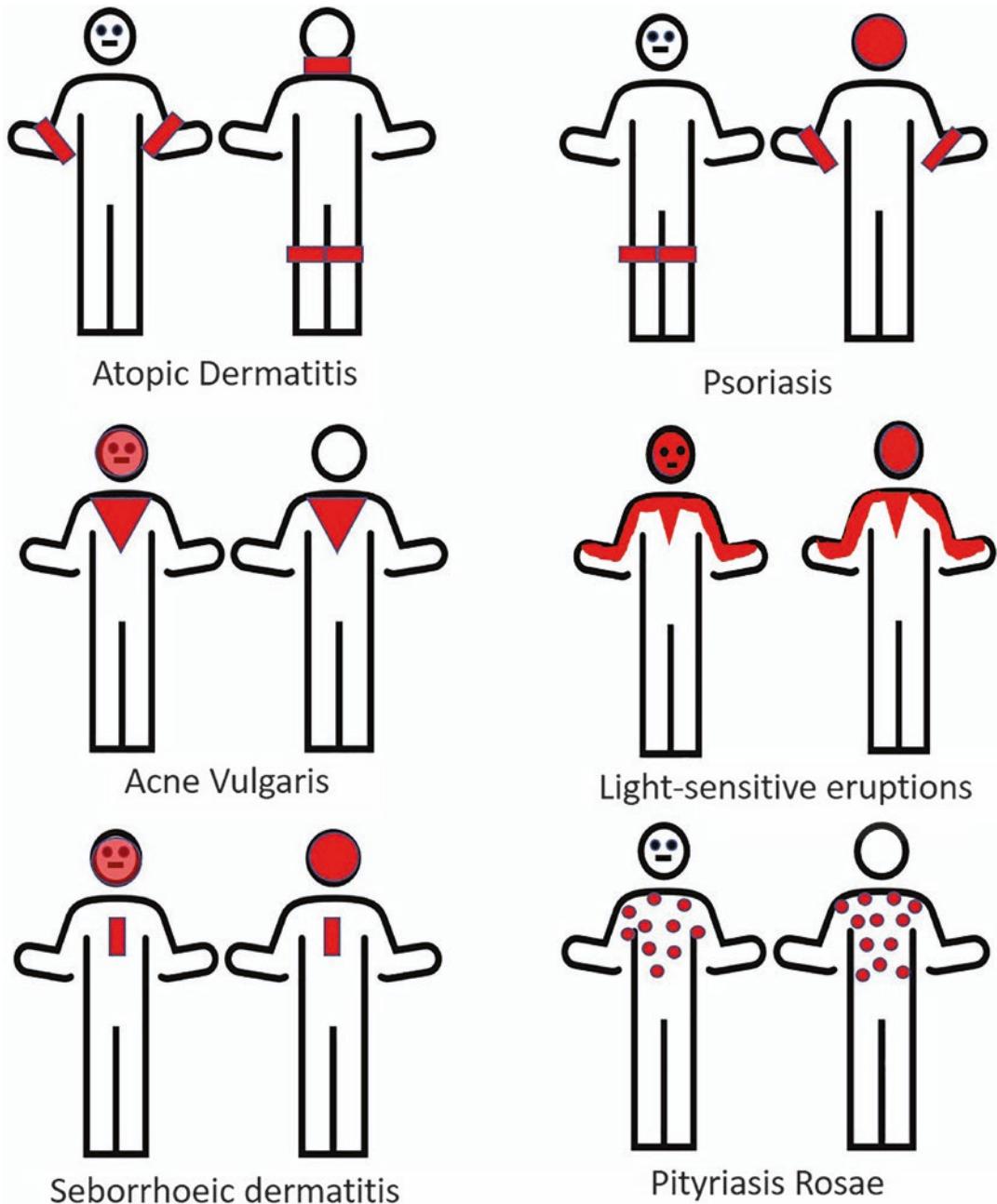
## 2.3 Physical Examination

A thorough physical examination is important when dealing with difficult to diagnose rashes as important clues to the diagnosis may be found in hidden areas, such as the scalp, the groin or between the toes or in the nails. Good light (daylight if possible), magnification and a warm room are helpful. Make up should be removed when examining the face. A complete head to toe examination is necessary especially when the diagnosis is not clear as the distribution of a rash may help clinch the clinical diagnosis.

When examining a rash, first study the form of the individual lesions, then the pattern of the lesions on the body and their spatial relation to each other. Many rashes can look similar when you examine the individual lesions but they often have distinctive patterns which will help with the diagnosis (e.g.: psoriasis usually occurs on the backs of the elbows and front of the knees while eczema usually has the opposite pattern) (Fig. 2.1). A unilateral red scaly rash is more likely to be fungal. A persistent isolated scaly rash may be due to fungal infection, neurodermatitis (lichen simplex chronicus) or a skin cancer or pre-cancer such as actinic keratosis, Bowens disease or a superficial spreading basal cell carcinoma. Photosensitive reactions will be found in sun exposed areas.

A full skin examination is also necessary when dealing with patients with skin cancer or suspicious moles. In addition, by examining all the skin you may also find other significant skin, hair or nail pathology that the patient was not aware of. All patients should be offered a chaperone if examining intimate areas and this should be recorded in the notes, even if the patient declines.

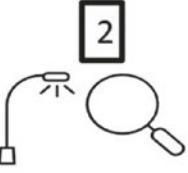
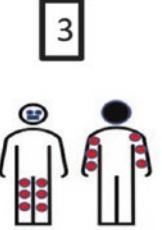
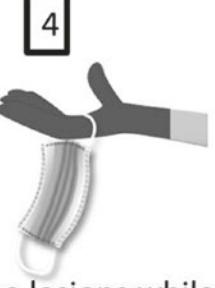
When examining all the skin, it is not necessary to ask the patient to strip completely. Get the patient to take off their top clothing (including their bra if necessary) and examine that area first. Then ask them to put back on their top clothes before removing their lower clothes (including their underwear if necessary). By retaining at least half of their clothing they will feel less exposed and vulnerable. Great care and sensitiv-



**Fig. 2.1** Distribution of various common rashes

ity is required when examining children who can be very easily embarrassed. Also, young adults may want a chaperone before examining intimate areas. Very often, explaining why you need to examine these areas can alleviate fears and embarrassment. A magnifying light is extremely

useful when examining and treating small lesions. Dermoscopy (skin microscopy) is invaluable when diagnosing suspicious pigmented lesions and can also be useful in diagnosing some non-pigmented lesions, scabies and scalp problems (Fig. 2.2).

 <p>Undress the patient (provide robe/chaperone when needed)</p>	 <p>You need good light, magnifying lens and dermoscope at hand</p>
 <p>Take a long view to understand where lesions are located</p>	 <p>Touch the lesions while protecting yourself</p>
 <p>Do not forget to examine the scalp and areas between fingers</p>	 <p>Do not forget to examine "hidden" areas</p>

**Fig. 2.2** How to examine the skin

When examining scaly or scabby lesions it is advisable to remove the scale or scab to reveal what is underneath (e.g. a nodule, superficial ulceration, an erythematous macular area or perhaps normal skin) which will help in making the diagnosis (Figs. 2.3 and 2.4). Palpating a lesion (e.g. the characteristic pebbly feel of a dermatofibroma), stretching the skin around the lesion to

delineate the borders (see Fig. 45.20; Chap. 45) or feeling a rash, (e.g. the typical feel of keratosis pilaris, psoriasis or actinic keratosis) can often help in the diagnosis and also reassure the patient that it is not contagious. If the lesion is bleeding or weeping, gloves must be worn. Careful hand washing or using hand sanitizers before and after examining all patients is important, especially



**Fig. 2.3** SCC (Squamous cell carcinoma) on the tip of the ear which is more obvious after removing the scab



**Fig. 2.4** BCC L upper cheek

when palpating the skin. Do this in front of the patient so they can observe the importance placed on hygiene. All suspicious lesions should be measured (largest and opposite diameter and sometimes the amount of elevation) in millimetres and an accurate description of the location of the lesion should be made. A surface anatomy mapper that describes a location on the body can be very useful (e.g. <http://anatomymapper.com>). Photos are also an effective way to record the size and location of a lesion and observe changes over time and useful when referring a patient as they may speed up the referral process if the receiving doctor realises how severe the rash or lesion is. A ruler may need to be included in the photo to give the lesion's perspective.

The severity of a skin problem can be scored as mild, moderate or severe. As this three point scoring system can be limited, add two more levels ("mild to moderate" and "moderate to severe"). It is surprising how often the doctor and patient agree on the level of severity of their skin

problem. If the patient is complaining of a "severe" skin problem but the doctor is only seeing mild disease, it should alert you to the possibility of underlying psychological problems (e.g. body dysmorphic syndrome). On follow up, after initiating treatment, it is helpful to ask the patient if their skin condition is the "same, better or worse" than their last visit. Sometimes it is useful to score the improvement (e.g. 25%, 50%, 75% or 90% better).

Becoming skilful in dermatology depends on having a good knowledge of the natural history of common skin diseases and an accurate visual memory of previous cases. Discussing cases with colleagues within the practice may help, as they may recall seeing a similar case previously. Published and "on line" dermatology atlases can be a very useful way of learning the appearance of common rashes and lesions. Artificial intelligence (AI) is already available to aid diagnosis using algorithms and photos in dermoscopy and will become more available in the future [2].

Absolute recognition of a rash or a lesion is desirable, although not essential, on the initial consultation once the doctor can rule out more serious pathology such as a melanoma or a serious skin infection. Reviewing the patient after a few weeks may be helpful as the classical, clinical signs of a disease may become more obvious with time. A good atlas of dermatology or a good website with lots of pictures is a significant help when struggling with a diagnosis and when trying to explain the nature of a problem to a patient (see Chap. 67 = useful resources + bibliography). "**Google images**" is a very quick way to find pictures of various skin rashes or lesions to compare to what the patient has or to explain to the patient what the rash or lesion can look like on other patients. However, "Google images" are not always correctly filtered according to diagnosis and the photos may scare some patients. The patient should be encouraged to photograph their rash or lesion over time to monitor the progress and they may help clinch the diagnosis in cases where the rash is not present or florid on the day and time the patient presents as in urticaria.

## 2.4 Conclusion

Taking a proper history in dermatology not only helps make the diagnosis but will also illicit how the patient feels about their skin problem and what they expect from treatment. A thorough physical examination of the skin may help make a diagnosis and not infrequently can show up other dermatology problems that the patient may not have been aware of.

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# Investigations and Treatment in Primary Care Dermatology

3

David Buckley

## Key Points

- Photos of the rash or lesion, which the patient may have taken themselves on their phone or home camera, can often help in making a diagnosis or assess severity.
- Skin scrapings for fungal stain and culture are sometimes necessary to rule out a fungal infection before starting a potent topical steroid in a vague, non-specific, asymmetrical rash.
- Biopsying vague, non-specific rashes is often unhelpful unless looking for specific conditions that have characteristic histological features such as lupus or lichen planus.
- Non-prescription or over the counter products can often be as or more important than prescription items when treating skin disease.

## 3.1 Introduction

If a careful history and a thorough physical examination does not make a confident clinical diagnosis the doctor may need to carry out various investigations such as blood analysis, skin scrapings, nail clippings or a skin biopsy to help

make the diagnosis. All potentially malignant skin conditions should have a biopsy to confirm the diagnosis histologically. It should be made clear to patients when treating skin conditions whether we are trying to cure the condition or merely control the symptoms and improve the appearance of the skin.

## 3.2 Investigations

Most skin problems can be diagnosed clinically with a detailed history and a thorough physical examination using good light, magnification and a dermoscope if available. Special investigations may sometimes be necessary to confirm a clinical diagnosis or to rule out more serious pathology.

Dermoscopy is a very useful tool for diagnosing not only suspicious pigmented lesions but also a number of common benign and malignant lesions that have characteristic dermoscopy features such as melanoma, BCC, seborrhoeic keratosis and scabies [1].

Skin scrapings for fungal stain and culture are sometimes necessary to rule out a fungal infection before starting a potent topical steroid in a vague, non-specific, asymmetrical rash (Fig. 3.1). With a little practice and experience a GP should be able to examine skin under a Wood's lamp and scrapings under a microscope to reveal fungal elements or scabies (mites or eggs). Special haematological, biochemical, histological or micro-

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**Fig. 3.1** Skin scraping to isolate the fungus in Tinea manuum

biological tests can be performed by taking specimens in the surgery and sending them to the local hospital. Biopsying vague, non-specific rashes is often unhelpful unless you are looking for a specific diagnosis that has characteristic histological features such as lupus or dermatitis herpetiformis. It is best to send your histology specimens to a pathologist who has a special interest in dermato-histopathology and with as much clinical details as possible. The pathologist will need to know the age of the patient (date of birth), the location of the biopsy and the type of biopsy (punch, shave, incision, excision, etc.) and a reasonable working and differential diagnosis. If you cannot do this, it might be better to refer the patient to a more knowledgeable colleague rather than taking a biopsy. Pathologists dislike getting a specimen with sparse clinical details such as “itchy rash” (see Chap. 57). The response will probably be “non specific chronic inflammation”.

Other special investigations may be indicated, such as biopsies for immunofluorescence to assess blistering diseases or a jejuna biopsy for coeliac disease, but patients are usually referred for these tests.

Allergy testing such as IgE and RAST test, skin prick testing, skin patch testing or exclusion diets are sometimes necessary to identify underlying allergies that may or may not be already suspected from the history. Allergy testing may also be necessary to exclude allergies such as food allergies that the patient or parents may falsely believe are responsible for their skin prob-

lem as a result of spurious “allergy tests” carried out in health food shops or by alternative practitioners [2] (see Chap. 21).

### 3.3 Treatment Approach

When treating dermatology patients it is very important to explain whether you are aiming to cure or simply control their problem. Give realistic expectations as to how long it will take for the treatment to work as, unlike other branches of medicine, with dermatology problems, everything is on the surface for the patient to see and monitor themselves. For example, psoriasis usually takes 6–12 weeks to improve and acne can take 3–6 months to clear. By not giving this information from the outset, the patient may give up their treatment too early or shop around for another opinion.

Follow up is also important to monitor the success of the treatments and encourage the patient to continue with them. At follow up, it is helpful to try to quantify the improvement (if any) by a global assessment by the doctor and the patient (e.g. 25%, 50%, 75% or 90% improved). Photography can be very valuable. Take a good standardized picture previously consented by the patient anytime you feel it will help to monitor a skin condition. Some patients may over estimate the response to treatment or fail to admit that it is getting worse in an attempt to please the doctor. Asking the patient if the condition is “the same, better or worse” is more open ended and more likely to get an accurate response. If the patient is not responding to treatments it is important to check compliance and review the original diagnosis. My personal “rule of three” is that if a patient comes back three times with the same problem, it means that it is not improving. If this is the case, refer the patient on to another colleague with more experience in that area for their opinion; otherwise the patient may lose confidence and they will probably default on follow up and go elsewhere. In general, patients tend to appreciate when their doctors ask for a second opinion. It shows humility and capacity for team work.

It is important to reassure patients that their rash is not infectious, disfiguring or cancerous (when it is not), as this will often be their biggest fear. It is also important to confidently inform the patient that you can treat their condition (either cure or improve the visual appearance of the condition) or at least reassure the patient that you can refer them to someone else who can help them.

When treating patients, have a plan of action with a list of differential diagnoses and treatment options and record them in the original consultation. Should the patient not improve on follow up, then review to your original notes to establish what was thought on the first visit. A busy practitioner will not remember what they were thinking a month previously. My dad used to say: “Bad ink is better than a good memory”, write it down!

Some dermatology problems may be temporarily improved on the day the patient visits you. Always ask the patient is the problem “average, good or a bad” on the day they present. Photos of the rash or lesion, which the patient may have taken themselves on their phone or home camera, can often help in making a diagnosis or assess severity. The clinical signs may be much different on a follow up visit so do not expect to always have to make a definitive diagnosis on the first visit.

Many dermatological disorders are treated by topical agents. It is important that the doctor instructs the patient carefully on how to use these preparations, particularly potentially irritating creams and topical steroids (see Chaps. 62–64). The doctor should demonstrate exactly how much is to be applied, how often to apply it, in which way should it be applied (e.g. rub downwards) and on which part of the body the topical treatment should be applied. Otherwise, patients may under or over use their treatment, resulting in poor response or side effects.

An old saying in dermatology is “if it is wet, dry it and if it is dry, wet it”. This is a little over simplistic but still has some merits. “Dry” skin is usually itchy and scaly and will almost always benefit from a good, safe, greasy moisturiser and avoidance of soaps and other irritants. Skin that is “wet” or weepy is usually very inflamed and may

be infected. It will usually benefit from dampening down the inflammation with a simple cooling cream and/or a topical steroid cream and treating any infections with an appropriate topical or oral antibiotic.

While prescription medications are often necessary when treating dermatology patients, the non-prescription or over the counter items can often be as important or more important, (e.g. moisturisers, soap substitutes, gloves, acne washes, non-comedogenic make-ups, etc.). Show the patient or parents what these products look like by having various tubes and pots in your clinic. It is helpful to demonstrate (either you or your assistant) how to apply the various products and give them estimation as to how long a tube or tube should last.

Life style modification may also be necessary in order to alleviate the problem or prevent relapse (e.g. weight loss, alcohol reduction or avoidance, dietary restrictions, etc.). Patients may also need psychological help to manage scratching, squeezing or picking their rash or lesion. Some dermatology patients can have deep psychological problems such as body dysmorphic disorder, delusional infestation or dermatitis artefacta that may need psychiatric assessment (see Chap. 53).

### 3.4 Conclusion

Investigations in dermatology may be necessary to make a diagnosis or to reassure the patient that there is nothing more sinister underlying their skin complaint. Treatment does not always require prescription medication or skin surgery. Sometimes, simple over the counter treatment, lifestyle advice and reassurance may be all that is required.

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# Structure and Function of the Skin

4

David Buckley

## Key Points

- The skin is made up of three layers—the outer epidermis, the middle dermis and the deep subcutaneous fat layer.
- If the physical, microbiological or chemical barrier function of the skin is compromised, it can lead to various local cutaneous or general systemic diseases.
- Diseased skin, especially in exposed parts of the body, can lead to low self esteem, withdrawn behaviour and even unemployment.

## 4.1 Introduction

It is important to understand the structure and function of the skin as this will lead to a better understanding of how to diagnose various skin conditions and how to manage them. Treatments work better if we not only try to improve the appearance of the skin but also restore the function of the skin that may have been compromised by the skin problem.

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## 4.2 Structure and Function of the Skin

It is important to know the structure and function of the skin to understand how it can be affected in disease processes [1].

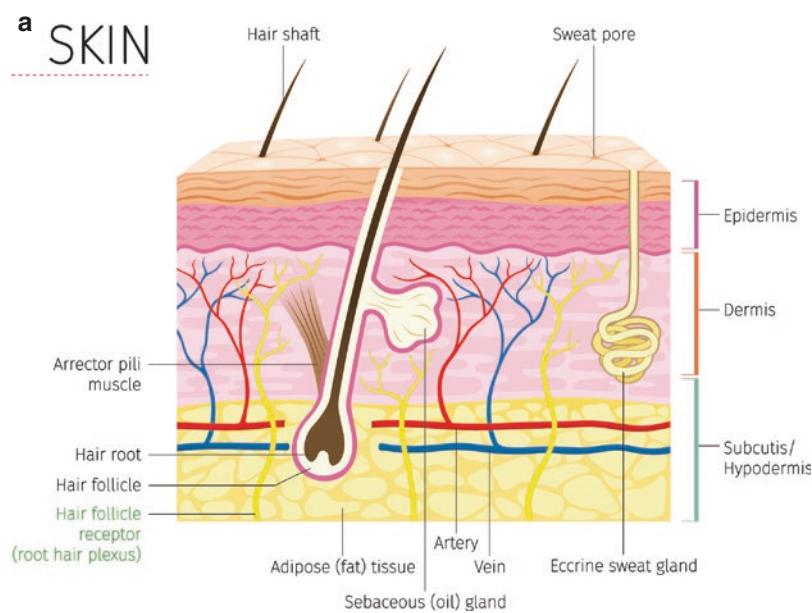
The skin is the largest and most visible organ in the body. Without it, life would not be possible. A burn of >40% of the body surface area can often be fatal.

The skin consists of three layers (Fig. 4.1a, b). The **epidermis** is made up of a complex matrix of cells called keratinocytes, which produce a protein called keratin. The keratinocyte cells are stacked like a stone wall with keratin in between each cell like mortar in a stone wall. If the mortar is defective or absent, it will affect the skin barrier function. Recent studies have shown that many children with atopic eczema have a mutation in the gene which encodes filaggrin which is a key structural protein in the outermost layer of the epidermis and which binds keratinocytes together. This is why these children have a defective skin barrier and are susceptible to infections, allergens and irritants.

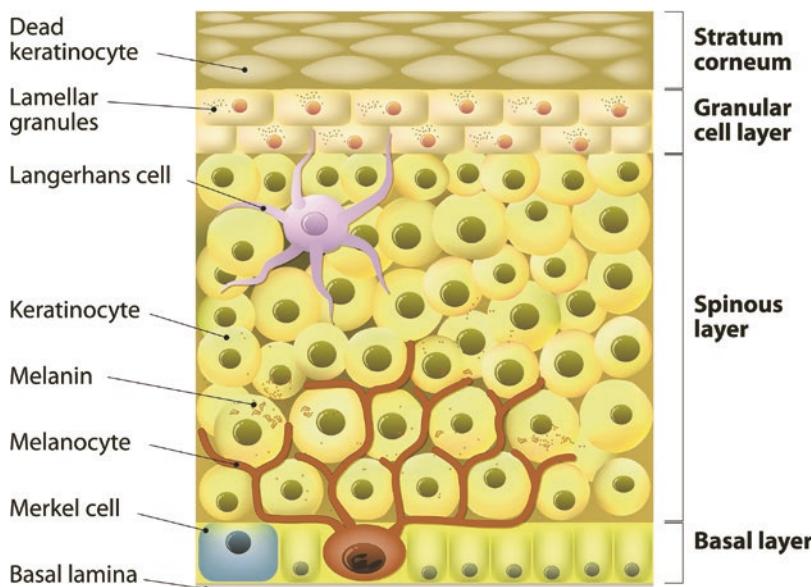
The epidermis is constantly being replaced by new cells dividing at the basal layer. The old cells gradually die and fill up with hard keratin. As each cell dies it moves up towards the surface of the skin, to be shed or worn away. This production of cells at the base of the epidermis is

**Fig. 4.1 (a)** Structure of the skin. SKIN© [matoomi]123RF.COM Image ID: 85121693. Media Type: Vector. [https://www.123rf.com/photo\\_85121693\\_human-anatomy-skin-and-hair-diagram-complexion-physiology-system-medical-healthy-beauty-cosmetic-mak.html?vti=njjzii1h3b](https://www.123rf.com/photo_85121693_human-anatomy-skin-and-hair-diagram-complexion-physiology-system-medical-healthy-beauty-cosmetic-mak.html?vti=njjzii1h3b)

**bngxxosc-1-1** Copyright matoommi@123RF.com  
**(b)** Epidermal layers of the skin. EPIDERMIS© [designua]123RF.COM Image ID: . 35866489. Media Type: Vector. [https://www.123rf.com/photo\\_35866489\\_cell-in-the-epidermis-layers-of-epidermis-structure-of-the-human-skin-.html?vti=n1xflmnybo3764190e-1-12](https://www.123rf.com/photo_35866489_cell-in-the-epidermis-layers-of-epidermis-structure-of-the-human-skin-.html?vti=n1xflmnybo3764190e-1-12). Copyright designua@123RF.com

**b**

## EPIDERMIS



carefully balanced with the loss of cells at the surface of the skin.

If the rate of cell replacement is altered, a skin problem develops. For example, in psoriasis there is an abnormal build-up of cells being produced and pushed up from the base of the epidermis resulting in thick scaly skin.

The basal layer of the epidermis contains pigment producing cells called melanocytes. The epidermis is separated from the dermis by the basement membrane.

The **dermis** contains the pilo-sebaceous units which consist of the hair follicle, oil (sebaceous) producing gland and the arrector pili muscle,

sweat (eccrine) and scent (apocrine) producing glands. Nerve endings and blood vessels are also found in the dermis. The dermis contains collagen and elastin which support the skin and give it suppleness and elasticity. With age these gradually deteriorate, causing the skin to sag and wrinkle. This process is accelerated by excessive exposure to ultra violet light. Despite claims by many cosmetic companies, there is little evidence that cosmetic creams or lotions can penetrate into the dermis and counteract the effects of ageing.

The **subcutaneous fat** provides cushioning, thermal insulation and energy storage. It also has important function in hormone production.

### 4.3 Hair and Nails

Hair and nails are formed from dead keratin, so various commercial preparations advertised to strengthen or rebuild hair or nails are of questionable value. Plucking or shaving facial hair does not make it grow any faster, darker or thicker.

### 4.4 Function of the Skin

The skin has a number of vital functions (Table 4.1). Health care professionals often underestimate the importance of the skin, hair and nails in socio-sexual communication. This function is often enhanced by cosmetics, jewellery and perfumes. Diseased skin, especially in exposed parts of the body, can lead to low self esteem, withdrawn behaviour and even unemployment. In addition, chronic pain or itch can lead to insomnia, depression and suicide. If the

**Table 4.1** Summary of the most important functions of the skin

- |   |
|---|
| 1. <b>Barrier</b> —physical, thermal, antimicrobial, chemical and radiation barrier                                     |
| 2. <b>Regulates body temperature</b> —cools us when too hot and heats us when cold                                      |
| 3. <b>Fluid balance</b> —prevents loss of essential body fluids and excretes salt and other toxic substances with sweat |
| 4. <b>Mechanical support</b>  |
| 5. <b>Immunological function</b> mediated by Langerhans cells—protects against microbes and allergens                   |
| 6. <b>Communication</b> —sensory organ for touch, heat, cold and emotional sensations                                   |
| 7. <b>Metabolic</b> —vitamin D synthesis, steroid synthesis and storage of energy in the fat layer                      |
| 8. <b>Psycho-sexual</b> function  |

physical, microbiological or chemical barrier function of the skin is compromised, it can lead to various local cutaneous or general systemic diseases.

### 4.5 Conclusion

The skin is the largest organ in the body in surface area and weight. It consists of three layers: the epidermis and the dermis and the subcutaneous fatty tissue. It has three main functions: protection, regulation and sensation. Wounds, rashes and tumours can affect varying levels of the skin from superficial to deep and can affect various functions of the skin.

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# Terminology in Dermatology

5

David Buckley

## Key Points

- The name of many dermatological conditions are based on the Greek or Latin words to describe the physical appearance of the rash.
- Many conditions in dermatology can have more than one name to describe the same thing.
- Many rashes look similar when examined close up but they can have a predictable distribution which can help in the diagnosis.

## 5.1 Introduction

Dermatology, like any other specialist area such as computers or economics, has its own unique language and terminology. The name of many dermatological conditions are based on the Greek or Latin words to describe the physical appearance of the rash. Knowing the meaning of the words can make it easier to understand the underlying problem and remember the name [1]. For example, the term, **lichen planus**, was derived from the Greek word “**leichen**” meaning “tree moss” and the Latin word **planus** meaning “flat.” Lichen planus is a chronic inflammatory dermatosis of unknown origin that causes purple or vio-

let papules and polygonal plaques that are shiny, flat-topped and firm on palpation. It can occur anywhere on the skin, mucous membranes, scalp or nails but it often starts on the anterior wrists, ankles and lower back in adults.

The word “**pityriasis**” was used by the physician Hippocrates in ancient Greece to describe the scruffy appearance of the skin that looked like it was covered by the fine bran of grain called “pityron”. “**Versicolour**” comes from the Latin word “*versus*”, or “*vertere*”, which means *to turn* or change color. **Pityriasis versicolor** is a common skin complaint in which fine, flaky, discoloured patches appear mainly on the chest and back mostly in young adults. It can cause hypo or hyperpigmentation that can vary according the seasons and the amount of ultraviolet light on the skin—hence the name = versicolor.

To add to the confusion, many conditions in dermatology can have more than one name to describe the same thing. For example, a seborrhoeic keratosis is also known as a seborrhoeic wart or a basal cell papilloma. Actinic keratosis is also known as solar keratosis. Some conditions in dermatology may have a medical name which is often based on Latin or Greek terms but they may also have a common lay male name. Examples include dandruff (pityriasis capitis), ringworm (tinea corporis) or mole (melanocytic nevus).

Descriptive terms are based on the colour, shape or texture of a lesion or rash. The spatial relationship of rashes or lesions are also important

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to describe, as various skin diseases have a characteristic distribution (isolated, clustered, satellite lesions, dermatomal, etc.). Many rashes look similar when examined close up but they can have a predictable pattern which can help in the diagnosis (e.g. psoriasis usually affects the backs of the elbows and front of the knees whereas atopic dermatitis usually has the opposite distribution).

## 5.2 Descriptive Terms [1]

- **Lesion** = a single area of altered skin. It may be solitary or multiple, isolated or diffused.
- **Rash** = a widespread eruption of lesions.
- **Tumour** = a solid mass of the skin or subcutaneous tissues. A tumour can be benign or malignant.
- **Dermatitis** = inflammation of the skin; it is not a definitive diagnosis. There are many types of dermatitis. The term “dermatitis” and “**eczema**” mean the same thing.
- **Eruption** = a break out or becoming visible (e.g. drug eruption).
- **Exanthem** = another term for a rash (e.g. childhood viral exanthems).
- **Tinea** = the name of a group of diseases caused by a fungus.

## 5.3 Colour

- **Erythema/erythematous** = redness secondary to vasodilation which blanches on pressure.
- **Telangiectasia** = persistant, visibly, dilated blood vessels on the skin or mucosal surface (“broken veins”).
- **Erythroderma** = a skin condition which affects all or nearly all of the skin which is red all over (e.g. erythrodermic psoriasis).
- **Purpura** = bleeding into the skin. If they are small they are called **petechiae** (small <3 mm, red, purple or brown spots). Like a bruise, purpura does not blanch with pressure and is 3–10 mm in diameter. Palpable purpura is usually a sign of vasculitis.
- **Ecchymosis** = discoloration of the skin as a result of bleeding underneath = bruising >10 mm.

- **Pigmentation** = any shade of brown, black, grey or blue resulting from the presence of melanin at different depths in the skin.
- **Non-pigmented** = skin coloured, red, purple or white.
- **Hyperpigmentation** = excessive colour in the skin that causes it to be darker than the normal background skin.
- **Hypo-pigmentation** = loss of melanin causing the skin to be paler than the normal surrounding skin but not completely white.
- **Leukoderma** = white skin (e.g. vitiligo).
- **Alba** = comes from the Latin “albus” meaning white.
- **Leukonychia** = whiteness of the nails.

## 5.4 Shape or Configuration of Lesions

- **Annular** = lesion or rash in a circle or ring shaped such as ringworm or granuloma annulare (Fig. 5.1).
- **Discoid** = a disc or coin shaped circular lesion (it is also called nummular).
- **Linear** = in the shape of a straight line such as scratch marks or striae in pregnancy.
- **Polygonal** = varied, non-geometrical shape.
- **Polymorphic or multiform** = various different shapes.
- **Gyrate** = a rash that is whirling in a circle.
- **Serpiginous** = snake-like.
- **Poikiloderma** = a mixture of areas of hypopigmentation, hyperpigmentation, telangiectasias and atrophy (e.g. Poikiloderma of Civatte).



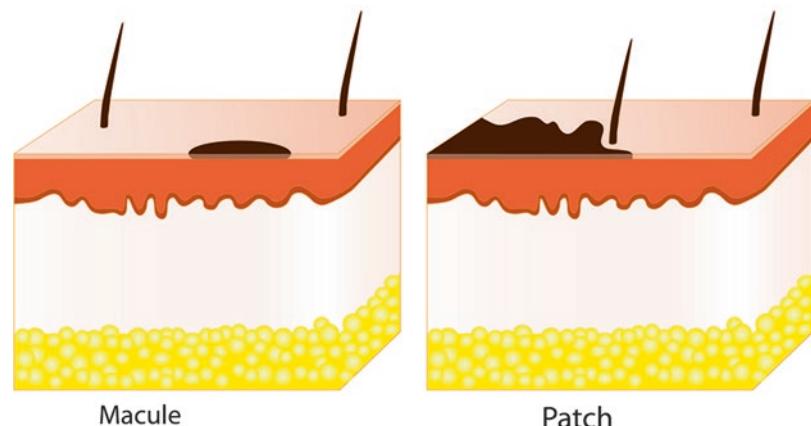
**Fig. 5.1** Anular rash of Granuloma annulare

- **Wheal (or weal)** = papule or plaque like with oedematous elevation caused by swelling in the dermis with a smooth skin surface (e.g. urticaria).
- **Flare** = erythema of the skin as a result of vasodilation often surrounding a wheal.
- **Target lesion** = a series of concentric rings like a dartboard (e.g. erythema multiforme) also known as **iris lesions**.
- **Reticular** = net-like lesions or rash (like the shape of a net curtain)

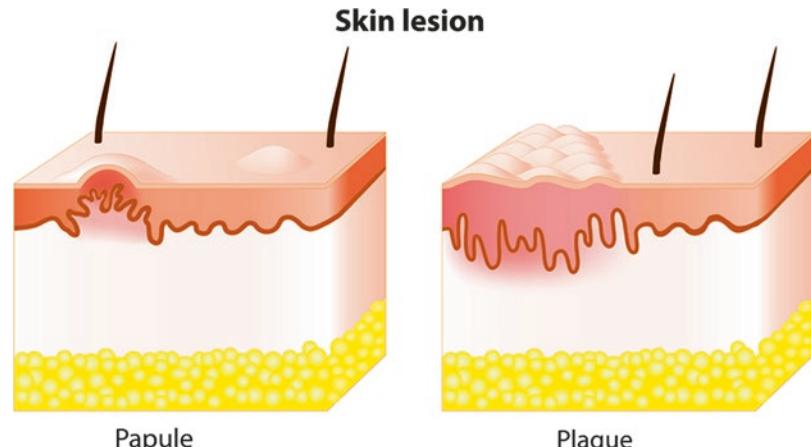
## 5.5 Texture or Morphology of Skin Lesion and Rashes

- **Macule** = flat discolouration less than 1 cm (e.g. flat mole) (Fig. 5.2).

**Fig. 5.2** Macule and patch. SKIN LESION© [designua]/123RF.COM Image ID 50902545. Media Type : Vector. [https://www.123rf.com/profile\\_designua?page=1&word=skin+lesion+&reverse\\_search\\_=mobile=&mediapopup=50902545](https://www.123rf.com/profile_designua?page=1&word=skin+lesion+&reverse_search_=mobile=&mediapopup=50902545)

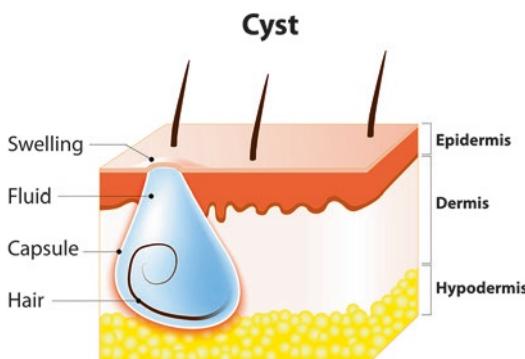
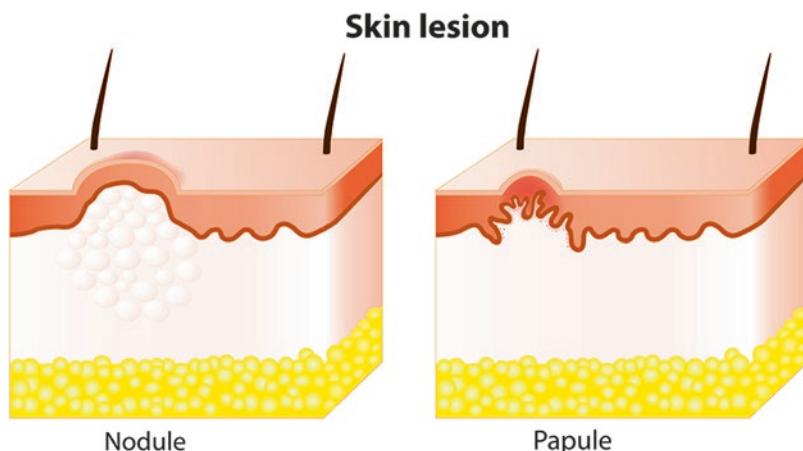


**Fig. 5.3** Papule and plaque. SKIN LESION© [designua]/123RF.COM Image ID 50902546. Media Type : Vector. [https://www.123rf.com/profile\\_designua?page=1&word=skin+lesion+&reverse\\_search\\_=mobile=&mediapopup=50902546](https://www.123rf.com/profile_designua?page=1&word=skin+lesion+&reverse_search_=mobile=&mediapopup=50902546)



- **Patch** = flat discolouration greater than 1 cm (e.g. lentigo maligna) (Fig. 5.2).
- **Papule** = palpable elevation less than 1 cm (e.g. acne spot) (Fig. 5.3).
- **Nodule or tumour** = a solid, palpable elevation greater than 1 cm (e.g. acne nodule) (Fig. 5.4).
- **Plaque** = a palpable lesion greater than 1 cm in diameter formed by the extension or coalescence of either papules or nodules (e.g. plaque psoriasis, granuloma annulare). Most plaques are elevated but a plaque can also be a thickened area without being visibly raised above the skin surface (Fig. 5.3).
- **Maculopapular** = a raised lesion or rash that is flat on the top (e.g. plaques of psoriasis).
- **Cyst** = epithelium lined cavity containing fluid or semi-solid material which may be

**Fig. 5.4** Nodule and papule. Note the volume difference. SKIN LESION© [designua]/123RF.COM Image ID 50902547. Media Type: Vector. [https://www.123rf.com/profile\\_designua?page=1&word=skin+lesion+&reverse\\_search\\_mobile=&mediapopup=50902547](https://www.123rf.com/profile_designua?page=1&word=skin+lesion+&reverse_search_mobile=&mediapopup=50902547)



**Fig. 5.5** Sebaceous cyst. SKIN LESION© [designua]/123RF.COM Image ID 50159201. Media Type: Vector. <https://www.123rf.com/portfolio/designua/10.html?mediapopup=50159201>

fluctuant (a fluid filled nodule such as in cystic acne) (Fig. 5.5).

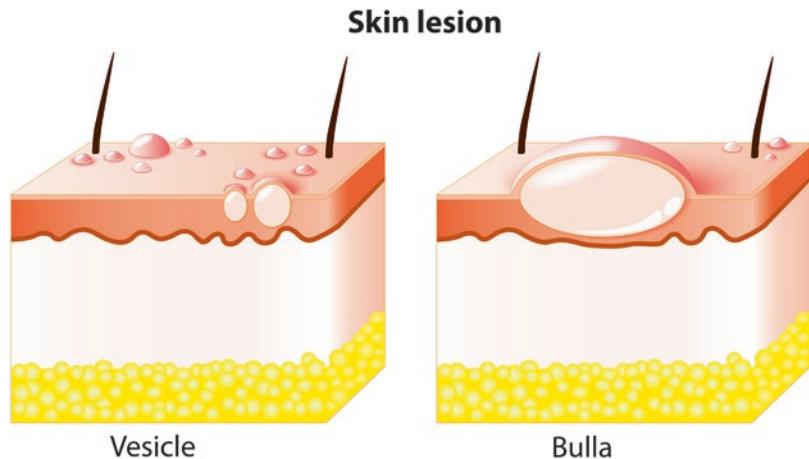
- **Abscess** = a puss filled cyst = usually infected.
- **Vesicle** = a papule containing fluid less than 5 mm (e.g. herpes simplex) (Fig. 5.6).
- **Bulla** = a large vesicle more than 5 mm in diameter (e.g. bullous pemphigoid) (Figs. 5.6 and 5.7).
- **Pustule** = a vesicle filled with pus (neutrophils) which may be yellow or white. This does not always imply infection (e.g. acne pustule).
- **Crust** = dried sebum, pus, or blood usually mixed with epithelial and sometimes bacterial debris (also called **eschar**).
- **Scale** = increased dead cells stuck together on

the skin surface (also called hyperkeratosis).

- **Desquamation** = skin shedding off in scales.
- **Psoriasiform** = large white or silver flakes like psoriasis.
- **Pityriasiform** = fine, powdery scale.
- **Morbilliform** = a rash that looks like the rash of measles (macular lesions that are red and are usually 2–10 mm in diameter but may be confluent in places).
- **Scarlatiniform** = looks like the rash of scarlet fever (numerous small red papules widely distributed in the skin)
- **Lichenoid (Lichen)** = scale tightly adherent to the skin surface like lichen on the rock at the seaside.
- **Lichenification** = caused by chronic rubbing, which results in thickened skin with increased skin markings and lichenoid scale (Fig. 5.8).
- **Keratotic (Keratosis, hyperkeratosis)** = horny scale with rough keratin (actinic keratosis).
- **Exfoliation** = skin peeling.
- **Maceration** = moist, peeling skin.
- **Dermatographism** is the ability to write on skin = scratching the skin surface creates a wheel flare type reaction (e.g. urticaria).
- **Keloid** = an exaggerated connective tissue response of injured skin that extends beyond the edge of the original wound.
- **Hypertrophic scar** = an exaggerated connective tissue response of cut or incised skin that does not extend beyond the edge of the original wound.

**Fig. 5.6** Vesicles and bulla.

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**Fig. 5.7** Bullae in a patient with bullous pemphigoid**Fig. 5.8** Lichenification of the low back

## 5.6 Feel, Form or Structure of a Lesion

**Papules** may be:

- **Dome shaped** = round on top like the dome of a mosque.
- **Filiform** = thread-like or small protrusions like a filiform wart.
- **Flat topped**
- **Pedunculated** = with a stalk.
- **Sessile** = without a stalk.
- **Umbilicated** = with a central depression (e.g. molluscum contagiosum).
- **Verrucous** = warty-like.

**Lesions or rashes** may be:

- **Depressed** = sunken under the skin.
- **Atrophic** = thinned out.
- **Hypertrophic** = thickened or raised up off the skin.

- **Soft, firm, hard, hot or cold.**
- **Fluctuant** = the movement within a swelling when it is examined by touch. It is a sign that the swelling contains fluid.
- **Sclerosis** = Localised hardening of skin.
- **Mobile or fixed.**

## 5.7 The Distribution of a Rash

Describes how rashes or lesions are scattered or spread throughout the skin (skin lesions may be isolated, solitary (single) or multiple. They can be localised or diffused.

- **Unilateral** = a rash or lesion that is predominantly on one side of the body.
- **Bilateral** = affects both sides of the body or specific region.
- **Symmetrical** = equal distribution on both sides of the body.

- **Truncal** = rash or lesions mainly confined to the trunk but not affecting the limbs, head or neck.
- **Flexural** = rash or lesion affecting the flexures (the bends or folds in skin such as the front of the elbows, back of the knees, around the neck in the axillae and groin creases).
- **Extensor** surfaces = the opposite side to the flexured surfaces.
- **Acral** = affecting the distal portions of the limbs, hands or feet.
- **Dermatomal** = a rash or lesion that runs along a dermal distribution (e.g. shingles).
- **Follicular** = lesions that arise out of hair follicles such as papules or pustules. They may be solitary or grouped into confluent plaques.
- **Herpetiformis** = groups of small vesicles like herpes simplex or herpes zoster.
- **Köebner phenomenon (Köebnerisation)** = refers to the tendency of a skin condition to affect areas that have been damaged due to injury such as scratching, laceration or burning. Common skin conditions that often demonstrate the Köebner phenomenon include psoriasis, lichen planus, vitiligo, warts and Darier disease (Fig. 5.9).
- **Fig.sensitivity** = Rashes or lesions that occur only on the exposed areas such as the face, the “V” area of the neck and the dorsum of the hands and lower legs.
- **Seborrhoeic** = a tendency towards oily skin (seborrhoea). The seborrhoeic area refers to those parts of the body that have a higher density of oil/sebum produced in glands such as the scalp, the eyebrows, the nasolabial folds, the post auricular area, over the sternum and the interscapular area of the back.



**Fig. 5.9** Körbner (Koebner) phenomenon in a patient with psoriasis

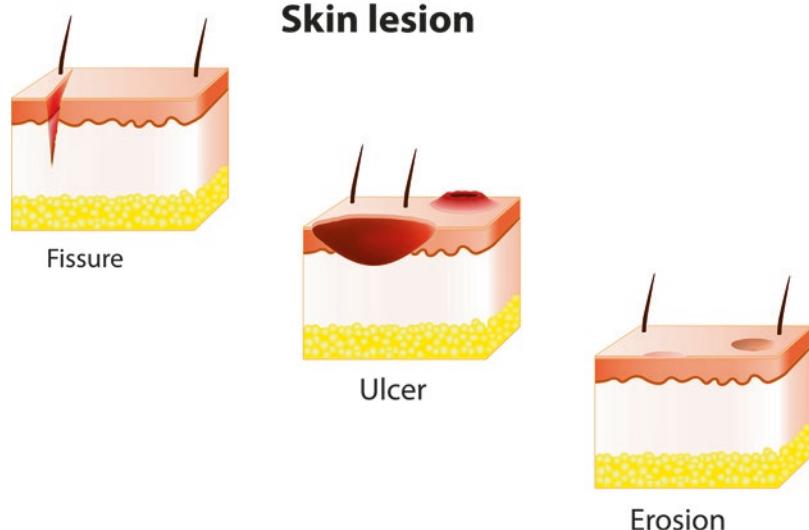
- **Discrete** = remains alone.
- **Clustered** = grouped together.
- **Confluent** = flowing together or merging
- **Guttate** = the Latin word for drops = i.e. looks like someone sprinkled the skin with drops.
- **Satellite** = a rash or lesion surrounded by numerous, smaller lesions or rashes located adjacent to the main lesion or rash, e.g. candidiasis.

## 5.8 Secondary Skin Changes

These are usually as a result of scratching, picking or infection:

- **Lichenification** = thickening and accentuation of the skin as a result of the chronic rubbing or scratching (e.g. lichen simplex chronicus) (Fig. 5.8).
- **Crusting** = arises as a result of plasma exuding through an eroded epidermis. Crust is usually yellow or brown and may ooze. Epidermal crusts may contain blood which can make them look more red, purple or black.
- **Dystrophy** = degeneration or abnormal morphology of the skin or nails.
- **Excoriation** = scratching which removes epidermis and causes bleeding or oozing. They are often linear.
- **Prurigo** = chronic skin disease with the eruption of pale, dome-shaped papules that itch severely and may be aggravated by picking and scratching. There can be many causes (e.g. purigo nodularis).
- **Erosion** = loss of the surface of the skin in the upper most layers causing a shallow, moist or crusty ulcer (Fig. 5.10).
- **Fissure** = a linear crack or break in the skin with abrupt side walls often due to excessive dryness (e.g. angular stomatitis, anal fissure) (Fig. 5.10).
- **Fungating** = a large tumour that erupts like a mushroom or fungus.
- **Granulation tissue** = formation of new capillaries and fibrous tissue in a healing wound that looks soft and red.

**Fig. 5.10** Fissure, ulcer, erosion. SKIN LESION© [designua]/123RF.COM Image ID 50902549. Media Type: Vector. [https://www.123rf.com/profile\\_designua?page=1&word=erosion&reverse\\_search\\_mobile=&media\\_popup=50902549](https://www.123rf.com/profile_designua?page=1&word=erosion&reverse_search_mobile=&media_popup=50902549)



- **Ulcer** = circumscribed loss of tissue. Ulcers may be superficial, deep or full thickness (Fig. 5.10). They may be covered or hidden by a dark coloured crust called **eschar**.
- **Scar** = permanent fibrotic changes that occur after healing of damaged to the dermis. Scars can be atrophic, hypertrophic, hypo or hyper-pigmented.
- **Granuloma** = this is an histological term. When a pathologist sees chronic inflammation and giant cells in the skin as a result of certain infections (e.g. tuberculosis, leprosy) or inflammatory skin diseases such as granuloma annulare or sarcoidosis.
- **Granulomatous Diseases** = those with the histological features of granuloma.
- **Nikolsky's sign** = is a skin finding in which the top layers of the skin slip away from the lower layers when slightly rubbed.

## 5.9 Nail Changes

- **Onychogryphosis** = thickening of the nail (not necessarily fungal in origin).
- **Nail dystrophy** = disruption of the nail surface.
- **Lamellar dystrophy** = splitting of the distal end of the nails in a horizontal plain (also known as onychoschizia).

- **Onycholysis** = lifting of the nail from the nail bed.
- **Pitting** = small indentations in the nail as if they were damaged with a sharp needle (e.g. psoriasis, alopecia areata, eczema).
- **Koilonychia** = spoon nails (e.g. with iron deficiency).
- **Clubbing** = increased curvature in both planes (e.g. lung cancer, valvular heart disease).
- **Subungual** = under the nail.
- **Periungual** = around the nail.
- **Pterygium** = a forward growth of the cuticle over the nail.

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## 5.10 Conclusion

There are many confusing and difficult terms used to describe lesions and rashes in dermatology. Many are derived from Greek or Latin. It is important to learn off these terms as they are the basic tools used to describe skin conditions and the descriptive terms are often used to make a named diagnosis.

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# Teledermatology

6

Paola Pasquali

## 6.1 Introduction

Teledermatology (TD) is a new branch of medicine. In TD, the face to face visit is substituted by the analyses of images of the patient's lesion or skin condition, the dermoscopic images and clinical relevant data which are all transferred via telecommunication technologies. Getting a second opinion can take minutes in comparison to costly transfer of the patient to the specialist's office or waiting for an appointment at a hospital with long waiting lists [1].

Many dermatology consultations are for benign conditions that can be managed by the FP as long as the diagnosis is confirmed. For malignant lesions, TD can assist in getting an urgent referral.

There are several reasons why many dermatology departments have long waiting lists:

1. Not sufficient numbers of dermatologists per 100,000 population
2. Mostly, because many benign skin conditions that do not require any medical treatment or could be treated effectively by the family physician (FP) are sent for consultation. These include skin tags, seborrheic keratosis and intradermal nevi.

Sometimes all that is needed is a second opinion to give reassurance on a diagnosis; in other cases, to make the diagnosis itself or even just to understand that the patient needs further and more specific examinations. Sometimes, TD helps in getting the FP to ask for certain lab or imaging tests before referring the patient, saving precious time for the patient.

## 6.2 What Can Be Sent? What Should Not Be Sent?

Some conditions are *more appropriate* for TD, such as:

- Single lesion
- Tumors suspected to be malignant; in this case, a clinical image needs to be accompanied by a dermoscopic image.
- Monitoring certain inflammatory conditions (like acne)

Some conditions are *less amenable* for TD:

- Multiple lesions (like in a multiple nevi patient)
- Hair diseases because they tend to be difficult to photograph. A dermoscopic image is required to make a correct diagnosis in inflammatory diseases of the scalp.

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### 6.2.1 Types of TD

**Primary Teledermatology** refers to direct communication between the patient and the health care professional (GP, nurse or dermatologist). It provides a direct service for initial diagnosis and referral [2].

**Secondary Teledermatology** refers to indirect communication between the patient and specialist. The patient (seeker) goes to a nurse/GP who then communicates with a specialist (provider) to receive advice. Other possible “intermediaries” are health insurance companies and healthcare institutions (nursing homes, emergency departments or pharmacies).

**Tertiary Teledermatology** refers to “second” opinion among specialists (dermatologist) and a dermatologist with a particular specialization. It is a specialist-to-specialist consult.

**Patient Assisted.** Patients communicate with a healthcare professional, usually for follow up or monitoring of skin conditions. It can be used for example for monitoring the response to treatment or for wound care.

**Direct to Consumer.** Patients initiates the care by accessing a healthcare provider through personal devices (Smartphone, laptop or tablet apps).

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## 6.3 Delivery Modalities

TD can be delivered in several modalities.

1. **Real-Time (RT) Video Consultation (Live Interactive):** employs live video conferencing.
2. **Asynchronous Store-and-Forward (SAF).**
3. **Hybrid:** Combines the above mentioned modalities.

Real-Time TD is similar to a face to face visit but at a distance using live video. The advantage is that the specialist can ask questions directly to the patient or to the physician. Real-Time TD is time consuming and requires all parties to be available at the same time. It requires a good internet connection from both sides.

The most common type of TD is “Store-and-Forward” (SAF). The TD is sent by means of clinical and/or dermoscopic images, lab results or others, and the opinion is given later at a convenient time by the specialist. Clarifying questions cannot be asked immediately but could always be asked later.

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## 6.4 Settings

The first TD was done to assist patients in rural or remote areas. With time, patients in large cities are also benefitting because transfer from one area to another in congested capitals can be time consuming and the difficulties to be seen promptly by a specialist are similar as for the patient in rural areas.

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## 6.5 A Good Photograph

If diagnosis are going to be made from a photograph, this needs to be of good quality [3].

A good medical photograph needs as a minimum:

1. To be taken with the same light. Flash light is a light that has the same temperature and can be adjusted in intensity (in most high end cameras). Otherwise, the skin colour will appear in the photograph in different tones depending on the light source. Light changes by the minute and every light source casts its own hue on the scene: tungsten and fluorescent bulbs, day or afternoon light, cloudy or sunny days. Getting the correct colour is also referred as white balance (where white looks white). If you are not going to use a flash, you will need to adjust the white balance settings on your camera or mobile phone if you want your patient’s skin to look natural.
2. Position: get the patient in the standard anatomical position. To get the right picture, the patient will need to pose and so does the photographer.
3. Backdrop. Probably the most relevant aspect in a medical image is to remove any distur-

bances from the background. Have a backdrop available (a piece of blue, green or black fabric) and put it behind the area you need to photograph. For point and shoot cameras, using a backdrop will help you get better focused images as the camera does not average the background focus with the skin lesion focus.

Remove all distractions from the patient (glasses, clothes, make-up). For a tumor, send a picture of the lesion, a dermoscopic image and also a medium view to understand the size of the lesion in relation to the area where it is located. Most important of all, get written consent to take the picture, explaining why the photograph is needed and in what context it will be used. Send only through secure connections. Most patients will agree to be photographed as long as they are treated with respect and the use of the image is clear and safe.

Include all the metadata/information necessary: age, sex, occupation, location of lesions, date of appearance, symptoms (itchy, painful, asymptomatic), chronic medication and medication used to treat the condition.

It has been shown that melanoma sent via TD has a thinner Breslow thickness and as such, a better prognosis [4].

Another advantage is the educational effect. The FP receives a diagnosis in hours or maximum a few days when the image (mental and also digital image!) is still “fresh” in his mind. The association of image and diagnosis is part of an educational process which occurs through pattern recognition.

## 6.6 Actors

The FP is not always the one making the consultation. Nurses and patients may send consultations to FP. For bedridden patients this is in ideal situation because many unnecessary transfers are avoided. For people with reduced mobility and the confined elderly, this represents an enormous advantage. Besides saving time and money, some elderly patients can get upset when moved out of their familiar environment.

## 6.7 Patient Empowerment

Patients are fortunately taking more and more control of their health and disease. In the past all decisions were exclusively taken by the doctors. Today, a shared decision based on an holistic view of the patient’s life, understanding their personal needs and focusing on what could be beneficial, will result in a closer patient-doctor relationship. TD helps with this as it incorporates the speed of new communication technologies and platforms that are becoming more user friendly like websites, apps, or shared electronic health records.

## 6.8 Conclusion

TD is a new way of practising medicine. The advantages of TD include saving unnecessary travelling and peace of mind for the patients. Nurses and doctors will get an immediate answer for a complicated health issues. Not all skin conditions are amenable for TD consultation but for those that are, the advantage for the patient and the doctor is enormous. The Covid-19 pandemic has shown so.

Artificial intelligence will help physicians and patients assess suspicious lesions and get second opinions faster and in a more precise way. The future is just beginning.

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**Part II**

**Adnexal Disease**



# A Stepwise Approach to the Management of Acne in Primary Care

7

David Buckley

## Key Points

- Acne is a chronic disease that can last months and years.
- Comedones and micro comedones are the hallmark of acne and it is important to treat comedones at all stages of acne.
- Acne originates in the pilosebaceous unit, occurring on areas of the body where these units are concentrated (face, neck, chest and back).
- Topical anti-comedone treatments are effective to treat acne and also to prevent relapse.
- Topical retinoids are a key class in acne management, having been shown to target micro-comedones, the precursor of all stages of acne lesions.
- Most cases of acne should be treated first line with a topical retinoid or retinoid-like agent and a topical antimicrobial agent such as benzoyl peroxide.
- Systemic therapy should be added to topical therapy in more severe cases.
- Oral antibiotics should generally be used for only 6–12 weeks and to a maximum of 6 months—otherwise resistance may develop.
- Oral antibiotics or oral hormone treatments should always be combined with suitable topical agents.

## What to Tell the Patient

- Many teenagers and young adults will get acne at some stage in their life.
- Acne occurs as a result of too much oil in the skin and blocked pores.
- Acne is not an infection and is not caused by poor hygiene.
- Everybody will outgrow their acne in time; however, it is hard to predict when exactly this will occur.
- There are safe, effective treatments for all degrees of severity of acne.
- Avoid picking skin lesions as it increases the risk of scarring (Fig. 7.1).
- Avoid oily products on the skin such as oil-bases foundations or oily moisturisers.
- Use oil free, non-comedogenic cosmetics if necessary.
- All acne treatments are slow; it usually takes 6–12 weeks to see a good response and can sometimes cases take 3–6 months or more to clear acne.
- Apply acne creams or gels all over the acne affected areas and not just onto the spots.
- Some acne treatments can dry and irritate the skin when started. Use sparingly initially and use alternate days if necessary for the first week or two of use. Avoid scrubbing in the topical medication as it increases irritation.

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- Too much refined sugars or too much dairy products like whey protein powders can aggravate acne.

## 7.1 Introduction

Acne vulgaris should be considered a chronic disease. Like many other chronic diseases such as asthma or rheumatoid arthritis, treatment should be aimed at both settling the acute symptoms (papules, pustules, nodules, cysts, etc.) and preventing relapse.

Acne is a very common, chronic, inflammatory skin condition. It occurs in 90% of teenagers and half of them continue to have acne as adults [1]. Acne is particularly cruel as it occurs on the worst part of the body (the face) at the worst time in a person's life (teens and young adult). 20% of young people have moderate to severe acne [2] (Fig. 7.2). Severe acne can have long lasting

physical and psychosocial effects [3, 4]. The reduction in quality of life has been estimated to be as great as that associated with epilepsy, asthma, diabetes, or arthritis [5]. Acne can be associated with low self-esteem, depression and anxiety. Bulling of acne patients is not uncommon. Many youngsters with acne feel rejected and develop low self esteem. More than 2 in 5 teenagers (44%) who have had acne have avoided having their photo taken on social media because of acne and 34% of teens with acne avoid video chatting [6]. Severe acne or picking acne can lead to permanent acne scars (Fig. 7.3a, b).

When treating acne, regardless of the severity, it is important to reassure the patient that acne can be controlled, although results may take 6–12 weeks and sometimes up to 6 months in more difficult cases. It is also important to explain that the treatments do not cure acne (except oral isotretinoin) and that they will need ongoing maintenance topical treatment when their acne has been brought under control so as to prevent relapse.



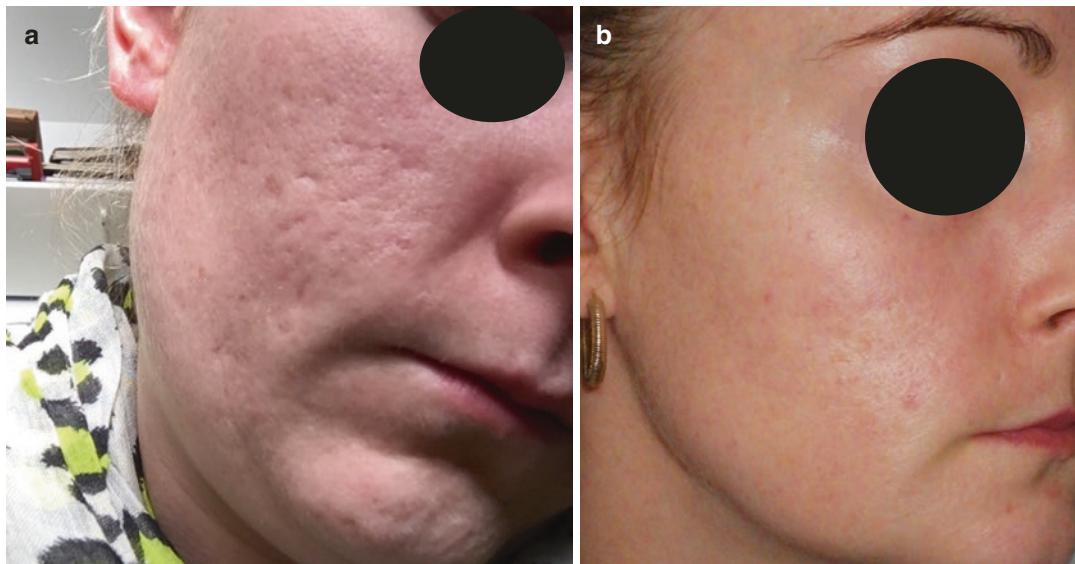
**Fig. 7.1** Picker's acne in a 17 year old



**Fig. 7.2** Nodulocystic acne pre-oral isotretinoin in a 19-year-old patient

## 7.2 Clinical Features and Diagnosis

**Acne** is a disease of the pilosebaceous units which are found in high numbers in the face, chest and back. The usual clinical features are oily skin with open and closed comedones (blackheads and whiteheads) (Fig. 7.4a, b) (Fig. 7.5). These features are known as non-inflammatory and may be all that is found in mild disease. Comedones may be large and obvious or tiny and only visible with a magnifying lense (micro-comedones). More moderate acne will have signs of inflammation with papules and pustules distribute on the face, neck, chest or back (Fig. 7.6). More severe acne may have nodules and cysts which may result in acne scarring (Table 7.1) (Fig. 7.7). When the sebaceous content of the overgrown sebaceous gland is exposed to air, it gets oxidized, blackens and it is called a blackhead. When the gland is closed by skin, the sebaceous content maintains its color and it is called a whitehead.



**Fig. 7.3** Permanent acne scars (a) Acne scars before microneedling; (b) same patient after 4 sessions of microneedling using the dermapen

When treating a patient with acne, it is important to know:

- When did it first appeared?
- Who has suffered from acne in the family and how severe was it?
- What medication has been used? What has worked and what has not?
- An assessment of the impact of the acne on the patient's quality of life. Mild acne in someone who is very upset with it may require more aggressive treatment.
- Understand the patients expectations
- Ask about their habits (likes to use make up, the use of sun or sunbeds, alcohol, diet, sports, body building, picking, etc.)

It is important to ask all questions and make visual contact with the adolescent patient. Parents have to be present but can sometimes take control and make decisions on the patient's behalf ("How should he use it? How do we apply the medication?). By talking directly to the patient, he/she will feel part of the decision process and be more likely to follow instructions. Do not treat adolescents like children.

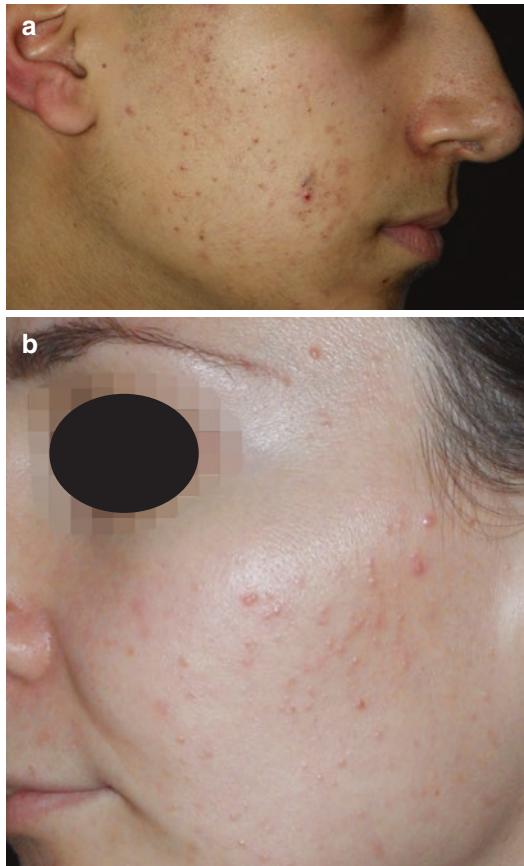
The non inflammatory and inflammatory features of acne may be found in certain patterns on the face, chest and back (e.g. T-zone, muzzle area, jaw line and neck, etc.). Some patients may only begin to develop features of acne in their 20s (adult onset acne) and some patients can have persistent acne into their 30s, 40s or 50s (Fig. 7.8). Acne is diagnosed clinically. There is no definitive diagnostic test.

### 7.3 Differential Diagnosis

Classical acne is easy to diagnose. Other conditions can cause papulopustular rashes on the face such as rosacea, peri-oral dermatitis, folliculitis, pseudofolliculitis barbae, a fungal infection, or pyoderma faciale (see Chap. 18). However they do not have the classical hallmark of acne which is oily skin (Fig. 7.9) and comedones (Table 7.2).

### 7.4 Pathophysiology

There are four main mechanisms in the aetiology of acne:



**Fig. 7.4** (a) Open comedones (blackheads) in a 15-year-old; (b) closed comedones in a 30-year-old female

- (a) Excessive production of sebum (under hormonal control)
- (b) Follicular plugging causing micro-comedones and comedones.
- (c) Overgrowth of micro-organisms especially *Cutibacterium acnes* (C acnes, formerly *Propionibacterium acnes*) which causes releases of inflammatory cytokines
- (d) Inflammation causes the pilosebaceous unit wall to ruptures resulting in an intense foreign body like reaction which leads to further development of inflammatory lesions (papules, pustules, nodules, cysts).

The problem with acne is that the end organ (the pilosebaceous unit) is very sensitive to the normal fluctuations of hormones that occur in adolescence and young adults leading to increased sebum production. This may be genetically determined. While it is clear hormones (especially androgens) play a major role in aetiology of acne, almost all patients will have normal hormonal levels in their blood. The only exception is patients with Polycystic Ovary Syndrome (PCOS).

Acne can be aggravated by various factors including hormonal (PCOS), greasy moisturisers or makeup (Fig. 7.10), drugs such as progesterone only pills and implants ("Implanon®") or the progesterone containing IUD ("Mirena®"), lithium,

**Fig. 7.5** Acne features.  
Blue = blackhead (open comedone), Yellow = Whitehead (closed comedone), Red = papule, Purple= pustule, Pink = excoriation from picking





**Fig. 7.6** Acne with comedones, papules and pustules in an 11-year-old female



**Fig. 7.7** Nodulocystic acne in a young adult pre-tetracycal + topical retinoid and benzoyl peroxide



**Fig. 7.8** Acne in a 44 -year-old woman with skin type V

B12, topical or oral steroids, stress, picking and lack of exposure to sunshine. Dietary factors may affect acne to a small extent, particularly excess dairy (skimmed milk, cheese and yogurt) or refined (processed) carbohydrates (sweets, cakes and fizzy drinks) [7]. Body builders may develop

acne or worsen their existing acne as a result of excess creatine and whey protein powders in their diet or from abusing testosterone or other anabolic steroids.



**Fig. 7.9** Oily acne in a 15-year-old patient prior to oral isotretinoin

**Table 7.2** Differential diagnosis of papulopustular rashes on the face and/or trunk

Acne vulgaris
Rosacea
Perioral dermatitis
Superficial (non gram negative) folliculitis
Gran negative folliculitis
Malassezia furfur folliculitis (usually on the trunk)
Tinea barbae
Chloracne (from occupational exposure to halogenated aromatic hydrocarbons)
Multiple milia
Pyoderma faciale (severe acneform eruption on the face in women)
Acneform drug eruptions
Adenoma sebaceum (angiofibromas- as seen in tuberous sclerosis)

## 7.5 Treatment

When faced with a patient with acne, some GPs immediately prescribe an oral antibiotic medication such as lymecycline or minocycline as it



**Fig. 7.10** Comedonal acne from vaseline in a 16 year old male

takes little or no time to explain to the patient how to use these medications. While oral antibiotics will reduce the inflammatory component of acne (papules and pustules) they do nothing for the basic underlying problem which is too much oil in the skin which leads to blocked pores and comedones (blackheads and white-heads). If the oiliness of the skin and the comedones are not treated then the patient will not respond adequately to treatment and will relapse quickly once the oral antibiotics are stopped or resistance to the antibiotic develops. In addition, there is concerns that the excessive use of antibiotics can adversely affect the patient's microbiome and may contribute to the development of antibiotic resistant bacteria.

It is important to understand the mode of action of various acne therapies (Table 7.3). Choose the most appropriate treatment for the particular type of acne and select logical combinations.

This step up, step down approach will hopefully simplify the management of acne and the success of treatment (Fig. 7.11).

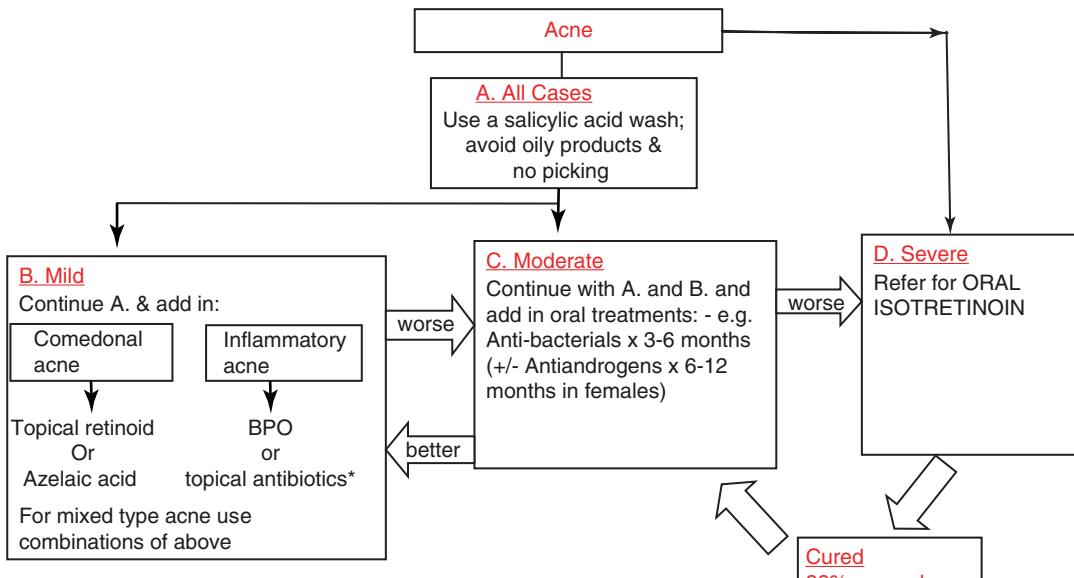
## 7.6 Topical Treatments

The simplest way to treat oily skin and comedones is with topical treatments (with the exception of oral isotretinoin, which we will discuss later). Topical anti-comedonal treatments are difficult to use, as most cause dryness and sometimes redness and soreness of the skin, particularly if they are used incorrectly.

**Table 7.3** Mode of action of various anti acne therapies

Mode of action	Salicylic acid wash	Benzoyl peroxide	Topical retinoids	Azelaic acid ("Skinoren®")	Topical antibiotics	Oral antibiotics	High dose oestrogens and/or anti-androgens	Oral isotretinoin
Decrease sebum production							++	++
Reduce follicular plugging	+		++	+			+	++
Reduce Cuti-bacterium acnes			++	+	++	++		++
Reduce inflammation		+	+	+	+	++		++

Adapted from Gollnick H, Cunliffe W, Berson D, et al. Management of acne: a report from a Global Alliance to Improve Outcomes in Acne. *J Am Acad Dermatol*. 2003 Jul;49(1 Suppl):S1–37



Topical retinoid = adapalene or isotretinoin  
BPO = benzoyl peroxide

Oral antibacterial agents = lymecycline, minocycline, erythromycin or trimethoprim.

Antiandrogens = "Dianette", OCP containing progestins with anti-androgenic properties (eg; drospirenone, chlormadinone acetate) or spironolactone.

\*Topical antibiotics should only be used for a maximum of 12 weeks

**Fig. 7.11** A stepwise approach to acne

The first step in the ladder in managing all patients with acne is to use a good anti-acne wash. While a bar of ordinary soap will definitely help, specific anti-acne washes containing salicylic acid are more effective but also more drying. Most patients with very oily skin will tolerate a wash containing **2% salicylic acid**. Those with more sensitive skin may only be able to tolerate a 0.5% salicylic acid wash.

Women should be advised not to over moisturise the acne affected areas and should only use oil free ("non-comedogenic") moisturisers and make-ups. All patients should be advised to never scratch, squeeze or pick their spots. They should be recommended to have a healthy balanced diet and take plenty of fresh air and exercise. Sun protecting products should preferably in gel and never in oils. Excess heat, humidity and sun-blocks can worsen acne ("tropical acne").

Patients with acne might have different type of lesions when examined but there tends to be one that predominates.

When patients have predominantly comedones (blackheads and whiteheads = non inflammatory acne) it is best to use **topical retinoids or retinoid like agents** combined with 2% salicylic acid wash (Fig. 7.4a, b). Topical retinoids, such as isotretinoin ("Isotrex®") or retinoid-like agents, such as adapalene ("Differin gel®") are primarily anti-comedonal. Patients should be instructed to put these agents over the affected areas of the face and neck and not just on the individual spots. The patient needs to be instructed to apply them sparingly, especially at the start of treatment, until their skin gets used to the preparations, for instance on alternate nights. It is important to tell the patient that these products can take months rather than weeks to clear comedones and to have a significant effect on acne. Topical retinoids should not be used in pregnancy. They have some skin lightening effects and may help if there is post-inflammatory hyperpigmentation (PIH) in darker skin types (Fig. 7.8). Topical retinoids not only help clear up acne

lesions, they can also be used long term (months or years if necessary) to prevent relapse.

When lesions are mostly inflammatory (papules and pustules), good results can be obtained by combining a salicylic acid wash with topical benzoyl peroxide 5% (BPO) (Fig. 7.6). Benzoyl peroxide is predominantly an anti-bacterial agent and is best used when there is mostly papules and pustules with not so many comedones. Benzoyl peroxide 5% is quite drying and has to be used sparingly, particularly at the start. New formulations such as "Acnecide 5%" are less irritating than the older formulations such as "Quinoderm®" or "Panoxil®". The 2.5% and 5% formulations are as effective as the 10% formulation with much less drying and irritation. Benzoyl peroxide is an over the counter medication, which makes it cheaper than many other topical acne preparations. It also comes in large tubes (60g) so it can be used on the chest and back, as well as on the face, if necessary. It is safe in pregnancy.

When using it on the neck or trunk you should warn patients that it can bleach coloured clothing, so advise them to use white shirts, sheets, pillow cases and towels. Patients (but especially parents or carers) will appreciate being given this piece of advice. Benzoyl peroxide is as effective as topical antibiotics without the problem of developing resistance so it can be used long term both to clear up the existing acne and to prevent acne relapsing once under control. It also reduces the carriage of antibiotic resistant micro-organisms and should be used in combination with oral or topical antibiotic acne therapies to improve their long term efficacy.

For most cases of mild to moderate acne, **combining benzoyl peroxide with a topical retinoid-like agent** can be very effective for both treating acne and preventing relapse. However, the combined effect of a salicylic acid wash, benzoyl peroxide in the morning and a topical retinoid at night can be very drying and irritating. These products should be started individually and sparingly and added in one week at a time. Combination products such as adapalene with benzoyl peroxide ("Epiduo®") are convenient and improve compliance since it only needs to be applied once a day. They are also more irritating

when used together and it might be more convenient to be used on alternate nights for the first week or two till the skin becomes accustomed to the products. A light weight, non-greasy, non-comedogenic moisturiser should be used in the mornings if there is excessive dryness. Some are commercially available and especially formulated for patient on oral acne treatment.

**Azelaic acid 20%** ("Skinorin®") is a useful alternative when patients cannot tolerate topical retinoids or topical BPO as it has some comedolytic, antibacterial and anti-inflammatory effects and is usually well tolerated. It also has some weak skin pigment lightening effects if there is any post inflammatory hyper-pigmentation. This effect makes it ideal for treating patients with darker skin types (Fitzpatrick type 4–6). It is safer in pregnancy than topical retinoids.

Limit the use of **topical antibiotics** to a maximum of 3 months, as resistance is almost inevitable after this length of time. All doctors should use as little topical or oral antibiotics as possible to prevent the emergence of antibacterial resistance organisms and fortunately we now have plenty of alternatives to antibiotics when treating acne of all stages. It may not be too far away before we are not allowed treat acne with antibiotics.

Unlike oral antibiotics for acne, topical antibiotics only have a weak anti-inflammatory effect. Also, it is important to realise that topical antibiotics have no effect on the oiliness of the skin or comedones. For this reason, it is probably better to combine topical antibiotics with a topical retinoid such as isotretinoin 0.05% + Erythromycin 2% (e.g. "Isotrexin®") or a benzoyl peroxide combined with 10 mg clindamycin such as "Duac®" for a maximum of 3 months. BPO used at the same time as a topical antibiotic many help reduce the development of resistance Once the topical antibiotic is stopped, a topical retinoid (together with benzoyl peroxide if there is any remaining inflammatory lesions) should be continued to prevent relapse. It is not good practice to use a topical and oral antibiotic together as this will encourage antibiotic resistance. Never use topical or oral antibiotics as a monotherapy. Always combine them with BPO and a topical retinoid if possible.

## 7.7 Systemic Treatments

While topical therapies will work very well for mild or mild to moderate acne, for more troublesome acne, a systemic treatment may have to be *added* to these topical agents or introduced at the same time [8]. Oral antibiotics, such as lymecycline 300 mg ("Tetralysal®") or doxycycline 100 mg daily should be used for at least 6–12 weeks and not more than 6 months as this will lead to resistance and loss of effect of the treatment. Oral antibiotics should always be used in conjunction with suitable topical agents such as a topical retinoid and topical benzoyl peroxide (Fig. 7.12a, b).

The oral antibiotics that are used in acne have powerful anti-inflammatory effects which are

probably as important as their antibiotic effect when treating acne [9]. There adsorption is *not* affected by food or milk. To avoid oesophageal irritation and ulceration, adequate fluids (water) should be taken with tetracyclines. Doxycycline can cause photosensitivity and is probably best avoided in the summer months.

Tetracyclines should be avoided in children less than 12 years old, in pregnancy and when breast feeding as they are associated with impaired bone growth, permanent discolouration of teeth and enamel hypoplasia in children (see section on infantile acne in Chap. 26). If the acne has not cleared after 6 months of oral antibiotics combined with good topical agents, then a different class of oral antibiotics could be tried such as doxycycline 100 mg daily or trimethoprim



**Fig. 7.12 (a, b)** Nodulocystic acne pre and post oral tetracyclines and topical adapalene +BPO combination in a 16 year old.

300 mg twice daily (these are off licence indications) [10] for 3–6 months. Trimethoprim can cause a severe generalized rash and all patients prescribed this drug for acne should be advised to stop it immediately if they develop a generalized rash. Erythromycin (500 mg BD for adults) for 3–6 months is useful if there is a risk of pregnancy and in children less than 12 years old although strains of C acnes resistant to erythromycin are becoming more common. Combining oral erythromycin with topical benzoyl peroxide may reduce the risk of developing resistant strains of C Acnes. Severe resistant cases not suitable for oral isotretinoin may respond to higher doses of oral antibiotics such as lymecycline 300 mg BD or doxycyclin 100 mg BD (off licence doses) combined with appropriate topical agents for 3–6 months [11]. Minocycline (“Minocin®”) is rarely used nowadays as it has more side effects than lymecycline or doxycycline (e.g. lupus and hepatitis) and is probably no more effective.

Large comedones can be easily removed by a comedone extractor. Large fluid filled acne cysts can persist for weeks or months and can leave permanent scars. They should be aspirated and injected with intralesional steroids which usually make them resolve within a week or two (Fig. 7.7). Most patients with this type of acne will end up on oral isotretinoin (“Roaccutane®”) sooner or later!

**Oral Isotretinoin** should be considered for patients with nodulocystic acne, acne conglobata (a severe form of nodulocystic acne with interconnecting **abscesses**, sinuses and scars), scarring acne or less severe acne not responding or relapsing after at least 6 months of a combination of topical and systemic treatments especially if the acne is having an adverse effect on the patients quality of life (see Chap. 8) (Fig. 7.13a, b).

For very inflamed acne, some dermatologists use topical steroids (preferably combined with topical antibiotic, like “Diprogenta®” a combination of betamethasone and gentamycin), for the



**Fig. 7.13** (a) Nodulocystic acne pre oral isotretinoin in a 17 year old, (b) Same patient after 6 months of oral isotretinoin

first 10–15 days of oral isotretinoin as inflammation could worsen at the start of a course of oral isotretinoin. It should be applied over the entire area. Sometimes even oral steroids for 7–10 days are used to reduce the expected worsening of inflamed lesions when starting oral isotretinoin.

“Diprogenta®” is also indicated in cysts or pustules that are appearing and can be felt (painful). In such cases, it is used only on the lesion and for a few days. Larger cysts and nodules may need to be aspirated and injected with steroid.

## 7.8 Acne in Women

All the above mentioned treatments are suitable for women. However, topical retinoids and oral therapies such as tetracyclines and trimethoprim should be avoided in pregnancy. Oral erythromycin and topical benzoyl peroxide are safe in pregnancy. If there are signs of an underlying hormonal imbalance (e.g. hirsutism, alopecia, obesity, infertility, menstrual problems, etc.) special investigations should be carried out (see Chap. 9).

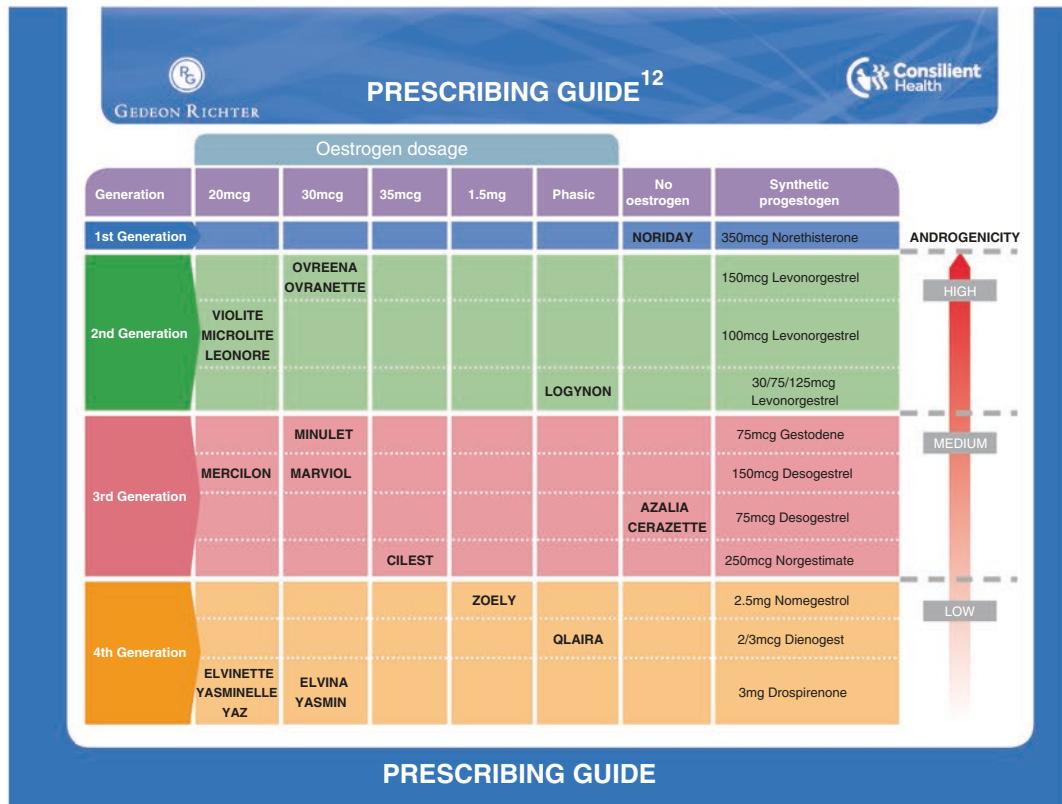
In younger, non smoking women, a combined pill containing 0.035 mg ethinylestradiol and the antiandrogen, 2.0 mg cyproterone acetate (“Dianette®”), can be helpful for acne, especially if she has other indications for the oral contraceptive pill, such as for contraception, menstrual problems, hirsutism or polycystic ovarian syndrome (PCOS). In addition, oral contraceptives are sometimes added to oral isotretinoin (which is teratogenic) to prevent an unwanted pregnancy. “Dianette®” is only licensed for moderate-to-severe acne which has failed to respond to alternative treatments such as topical therapies and oral antibiotics as mentioned above. “Dianette®” can only be used in women of reproductive age and should be stopped 3–4 cycles after the acne has cleared (usually after 6–12 months). At this stage a woman could be switched over to a conventional,

skin friendly combined oral contraceptive pill (COP) that has a high dose of oestrogen and a progestogen with low androgenic potency (e.g. drospirenone, dienogest, nomegestrol or levonorgestrel) for ongoing contraception or other indications (e.g. “Yasmin®”, “Qlaira®”, “Zoely®”, “Logynon®”, etc.) (Figs. 7.14 and 7.15).

Avoid 3rd generation OCP (e.g. “Marvion®”, “Minulet®”, “Cilest®”, etc.) as they have a twofold increased risk of venous thrombosis compared to second or fourth generation OCPs. Ultra low dose oestrogen pills with only 20 mcg of oestrogen (e.g. “Yasminelle®”, “Microlite®”, “Mercilon®”) should be avoided as they can make acne worse. Progesterone only contraceptives such as the mini pill, progesterone only implants (“Implanon®”) and the “Mirena®” IUD coil should be avoided in acne sufferers as they can aggravate acne.

“Dianette®” and other combined oral contraceptive pills should be avoided in heavy smokers and those with hypertension, hypercholesterolemia, obesity or a history of venous thromboembolism (VTE) (Table 7.4). All patients on these pills need to be advised about the possibility of VTE and be given the warning signs to look out for. These OCPs should be combined with benzoyl peroxide topically. They usually need to be continued for 6–12 months to be effective in acne (Fig. 7.16). Sometimes the OCP can be combined with oral antibiotics and good topical agents, although if the acne is bad enough to warrant this combination then the doctor should be considering oral isotretinoin.

Spironolactone is sometimes used for refractory acne in women especially if she is unsuitable or intolerant to oral contraceptive pills. It should be started at a small dose (25 mg/day) and gradually increased to an average of 100 mg/day. It can cause hyperkalemia, hypotension and menstrual abnormalities in some women. It is also teratogenic especially in the third trimester. It can be useful in women with PCOS.



**Fig. 7.14** Prescribing guide for the oral contraceptive pill in women with acne. Use a pill with high levels of oestrogen and low androgenicity (Table courtesy of Consilient Health)

## 7.9 Other Treatments

Other less common treatment options for acne include laser treatment (e.g. pulse dye laser), IPL (intense pulse light), chemical peels and photodynamic therapy.

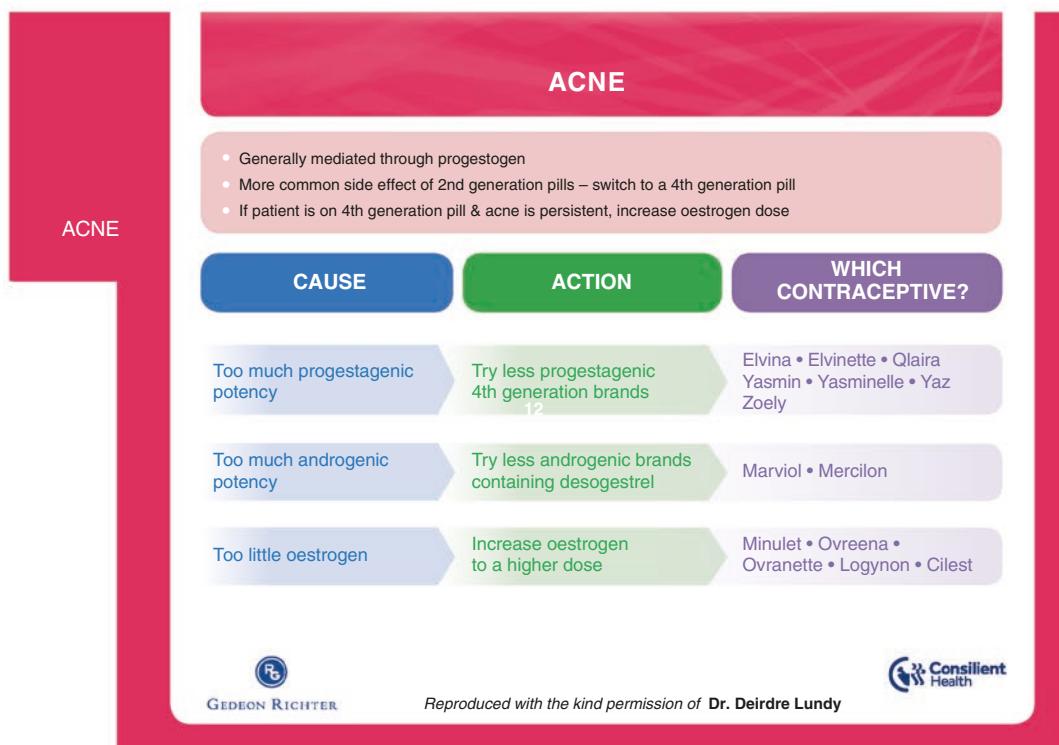
## 7.10 Maintenance Treatment

Regardless of which systemic agent is used for acne, it is important to remember to step down the treatment ladder to a topical retinoid maintenance treatment once the acne is under control to prevent relapse [8] (Fig. 7.11). Patients on oral therapies should be followed up every three months until the acne is under control to monitor response, check compliance and advise on main-

tenance treatment. It is also important to avoid oily makeup, creams and oily sun protecting products as these can provoke relapses.

## 7.11 Acne Scars

Prevention of scars by aggressive treatment of the acne before it has had a chance to cause extensive or severe scarring is the best approach but sometimes patients present too late and already have scarring. While there is no treatment that will totally eradicate all the scars, there are some safe effective treatments what will improve the appearance of the scars depending on the type and severity such as microneedling (Fig. 7.3a, b), chemical peels, laser resurfacing, intralesional steroids, cryosurgery, fillers, subcision and punching out deep pitted scars.



**Fig. 7.15** Prescribing guide for the oral contraceptive pill in women with acne

**Table 7.4** Relative and absolute contraindications to the oral contraceptive pill (OCP)

Age	>35 in a smoker >51 in non-smoker with no other risk factors
Weight	BMI >39 (WHO 4) BMI 30–39 (WHO 3) Note that measuring weight alone is not enough
Smoking	>40 per day (WHO 4) >15 per day WHO 3 at all ages!!
Hypertension	>160/100 WHO 4 always >140/90 or on treatment WHO 3 Previous pre-eclampsia WHO 2 but WHO 3 if smoker
Heart disease	Sometimes a contraindications. Check individual condition
High cholesterol	Always WHO 4 Family Hx in sibling or parent <45 is WHO 3 if no lipid profile is available
Migraine	>35 WHO 4 Aura or focal symptoms WHO 4 If another WHO 2 present e.g. if smokes <15/day then becomes WHO 4
Diabetes	Always at least WHO 3 WHO 4 if complications of diabetes present

**Table 7.4** (continued)

More rare problems	<ul style="list-style-type: none"> <li>• Known liver disease or abnormal LFT's</li> <li>• Epilepsy on some of the older anticonvulsants</li> <li>• Antiviral drugs</li> <li>• Immobility/Wheelchair bound</li> <li>• Sickle cell (sometimes WHO 3)</li> <li>• SLE (WHO 3 or 4)</li> <li>• Breast cancer (WHO 4)</li> <li>• Breast feeding (WHO 4)</li> <li>• History of VET or current VET (WHO 4)</li> <li>• Coronary artery disease or stroke</li> <li>• Factor V Leiden deficiency (WHO 4)</li> <li>• Liver cancer (WHO 4)</li> </ul>
WHO categories	<ul style="list-style-type: none"> <li>• Category 1: No restriction to use</li> <li>• Category 2: advantages of use of the method of contraception generally outweigh the risks</li> <li>• Category 3: risks generally outweigh advantages. Use not usually recommended</li> <li>• Category 4: use of the contraceptive method would result in unacceptable risk to health.</li> </ul>



**Fig. 7.16** Acne pre “Dianette®” with topical adapaline and benzoyl peroxide in an 18 year old woman

## 7.12 Rare Variants of Acne

**Gram negative folliculitis** can appear as a sudden eruption of pustules on the face and/or body in a patient already on long term oral or topical antibiotics such as tetracyclines (see Chap. 18).

**Pyoderma faciale** is a rare pustular eruption that occurs on the face in young women (Fig. 7.17). It usually starts suddenly and can be very severe with nodules or cysts and can leave scars. Unlike acne, there are usually no comedones or oily skin. It does not affect the trunk and does not occur in men. It usually responds to high dose anti-acne treatments such as tetracyclines, trimethoprim or isotretinoin. Large cysts may need to be drained and injected with interlesional steroids. Oral steroids may be necessary in severe inflammatory cases. Once cleared it usually does not relapse.

**Acne fulminans** is a very rare, severe form of nodulocystic acne where the inflammatory process is so severe there is usually systemic symptoms such as a low grade fever, flu-like symptoms and arthralgia. It causes bleeding, ulcerating nodules



**Fig. 7.17** Pyoderma faciale in a 22 year old woman

on the chest and back in teenagers or young adult males. It may be precipitated by anabolic steroids which are sometimes used by athletes and body-builders or at the initiation of oral isotretinoin. It usually has an abrupt onset and there may be systemic involvement with a raised white cell count, anaemia and a raised erythrocyte sedimentation rate (ESR). Urgent treatment is usually under specialist care and may involve oral steroids combined with high dose oral antiacne antibiotics such as lymecycline or trimethoprim initially. Most cases will require oral isotretinoin which should only be commenced after the initial inflammatory process has settled and it should be started as a very low dose. Resistant cases may respond to biologic agents such as tumour necrosis factor-alpha (TNF- $\alpha$ ) inhibitors (e.g.: infliximab).

**Acne conglobata** is another variant of nodular cystic acne where there are multiple abscesses and sinuses causing hypertrophic and atrophic scarring. There are cysts filled with smelly pus and multiple comedones. Acne conglobata is similar to hidradenitis suppurativa (HS) but in HS the boil-like lesions and scars are confined to the armpits, groins and under the breasts (see Chap. 11). The management of acne conglobata is similar to acne fulminans (see above).

### 7.13 Conclusion

Acne is a common condition of teenagers and young adults. Diagnosis is clinical and the management is straightforward [13]. No matter how mild or severe, acne is always treatable. This is usually with topical agents alone in mild acne

and a combination of topical and systemic treatment in more moderate or severe acne for 3–6 months. After clearing up the clinical signs of acne, the patient should be maintained on a long term topical treatment to prevent relapse for as long as they have a tendency towards acne. Severe resistant cases should respond to isotretinoin (“Roaccutane®”).

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# Oral Isotretinoin for Severe Acne

8

David Buckley

## Key Points

- Oral isotretinoin is extremely effective in treating severe forms of acne or resistant acne. It can offer a permanent cure.
- Approximately three quarters (72%) of patients who complete a course of oral isotretinoin will never get their acne back again.
- Oral isotretinoin is highly teratogenic and all women of child bearing age (even if they are not sexually active) should use effective contraception for a month before, during the treatment and a month after finishing the treatment.
- Oral isotretinoin may be associated with various psychiatric side effects such as depression, suicidal ideation and psychotic symptoms especially in young men. However, a causal relationship has not been established and the link between isotretinoin use and psychiatric events remains controversial.
- All patients taking oral isotretinoin should be encouraged to avoid alcohol and other psychoactive drugs while on this medication.

## What to Tell the Patient

- Despite its bad press, oral isotretinoin is safe and effective when used under careful medical

supervision by a doctor with experience in prescribing it.

- Try to resist the temptation to “Google” isotretinoin as there is allot of fake news stories about this drug, much of it fuelled by the legal profession and a small number of disgruntled patients. If more information is needed look up reliable unbiased sites such as [www.kerryskinclinic.ie](http://www.kerryskinclinic.ie), [www.aad.org](http://www.aad.org) or [www.bad.org.uk](http://www.bad.org.uk)
- There are some reports that oral isotretinoin may cause mood disorders such as depression especially in young men.
- If feeling down or low while taking oral isotretinoin, report this to the doctor prescribing this medication.
- The most common regret in patients who have completed a course of oral isotretinoin is that they did not take it sooner.
- There is plenty of evidence to show that bad acne itself can cause mood disorders or depression and oral isotretinoin can help alleviate these problems by clearing the acne.
- Everyone gets dry lips while taking oral isotretinoin and needs to carry a good lip balm at all times in their pocket or handbag and use it continuously.
- Oral isotretinoin is a photosensitizing medication so sun protection is essential.

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## 8.1 Introduction

Oral isotretinoin is a vitamin A derivative, which has been on the market for almost 30 years. It is an extremely effective treatment for severe nodulocystic acne, a condition where almost no other treatment will work (Fig. 8.1). It is also indicated for less severe acne, not responding to at least 6 months of appropriate oral and topical treatments (Table 8.1). Not only will it clear severe acne, it usually cures acne permanently in approximately three quarters (72%) of patients who complete a course [1].



**Fig. 8.1** Severe acne in a 17-year-old girl pre-oral isotretinoin

**Table 8.1** Indications for oral isotretinoin

- Severe nodulocystic acne
- Conglobate acne
- Acne fulminans
- Pyoderma faciale
- Acne unresponsive to standard oral and topical treatments especially if scarring
- Acne relapsing after repeated courses of oral and topical acne therapies
- Gram-negative folliculitis
- Acne associated with severe physiological upset

## 8.2 Mode of Action

Oral isotretinoin (“Roaccutane®”) works by shrinking the pilo-sebaceous unit (usually permanently) thus reducing the oiliness of the skin and comedones (blackheads + whiteheads). This leads to less bacterial overgrowth within the pilo-sebaceous unit. It also has a powerful anti-inflammatory effect on the acne. Because it works on all the levels in the pathogenesis of acne, it can be used as a monotherapy and no topical acne treatments are required.

The summary of product characteristics in Ireland states that, “*isotretinoin should only be prescribed by or under the supervision of physicians with experience in the use of systemic retinoids for the treatment of severe acne and a full understanding of the risks of isotretinoin therapy and monitoring requirements*”. This usually means that only doctors who have worked in a dermatology department for at least 6–12 months would have the experience required to prescribe it. Some GPs may have built up experience in prescribing isotretinoin over the years in practice. In New Zealand, registered GPs working in an appropriate field have been allowed prescribe isotretinoin since 2009 subject to Special Authority. From the years 2001–2012, 58% of prescriptions for isotretinoin in New Zealand originated in general practice [2].

## 8.3 Side Effects

“Roaccutane®” is highly teratogenic and so a hormonal contraceptive is compulsory for all women of child bearing age, even those who are not currently sexually active (young teenagers, nuns, etc.). Female patients need to sign a form stating they have been advised accordingly [3]. For those woman who are sexually active, two methods of contraception should be used for 1 month before, throughout and for at least 1 month after completing a course of oral isotretinoin (e.g. the oral contraceptive pill and condoms).

Oral isotretinoin may be associated with various psychiatric side effects such as depression, suicidal ideation and psychotic symptoms especially in young men. However, a causal relation-

ship has not been established and the link between isotretinoin use and psychiatric events remains controversial [4, 5].

It has to be considered that having acne may cause depression. Treating the acne (be it with oral isotretinoin or other less potent therapies) may improve the patient's mood, particularly if they have had severe acne.

It is best to tell all patients (and their parents if they are under the age of eighteen) that isotretinoin might cause mood disorders and depression and instruct them to contact their doctor immediately if they have any concerns or worries (Tables 8.2 and 8.3). It is best not to prescribe it in somebody who has a history of depression or if they are attending a counsellor for mental health issues. Advise all patients to keep alcohol to a minimum and in young patients try to get them to agree to avoid alcohol and other psycho-active drugs completely, while on this medication. Do not prescribe it around times of stress such as coming up to major exams, before a wedding or a major birthday. In high-end sport patients, the dose might need to be lower as it can cause fatigue and tiredness.

All patients develop a dryness of the lips, mucous membranes and the skin generally (Figs. 8.2 and 8.3). This is especially noted in atopic or dry skin patients with acne. This is not a side effect; this is how the drug works. This

**Table 8.2** Warning signs of the possible onset of depression

- Feeling sad or low
- Moody
- Anxiety
- Crying
- Irritability
- Low energy
- Loss of interest or pleasure in social or sporting activities
- Sleep disruption
- Change in weight or appetite
- School or work performance decreased
- Trouble concentrating
- Becoming violent or aggressive
- Suicidal thoughts

If you have some of these symptoms most days for more than 2 weeks please talk to your doctor

**Table 8.3** Patient health questionnaire-2: screening instrument for depression

Over the past 2 weeks, how often have you been bothered by any of the following problems?	Not at all	Several days	More than one-half the days	Nearly every day
Little interest or pleasure in doing things	0	1	2	3
Feeling down, depressed, or hopeless	0	1	2	3

Note: A negative response to both questions is considered a negative result for depression

Adapted from patient health questionnaire (PHQ) screeners. <http://www.phqscreeners.com>. Accessed September 6, 2011



**Fig. 8.2** Chelitis from oral isotretinoin

drug shrinks down the oil producing glands thus drying out the skin and mucous membranes. Frequent use of a lip balm usually controls the dry lips. If dryness is severe the dose may need to be reduced. The dryness of the mucous membranes usually settles after completing a course of isotretinoin.

Isotretinoin can have various predictable side effects such as epistaxis, myalgia, and alterations in serum lipid and transaminase concentrations [6] (Table 8.4). These side effects are usually reversible on reducing the dose or stopping the treatment. If skin dryness is not tolerable, non-comogenic moisturisers can be used for the acne areas while regular hydrating creams can be used on the rest of the body. Sun-protection needs to be in gel presentation because cream presentations tend to induce acne on acne prone patients.



**Fig. 8.3** Flare of eczema in patient on oral isotretinoin

**Table 8.4** Some of the more common or serious side effects of Roaccutane

- Dry lips/nose/eyes
- Eczema
- Fragile skin
- Rashes including erythema multiforme, Stevens-Johnson syndrome and toxic epidermal necrolysis
- Low back pain/arthralgia/myalgia
- Alopecia (reversible)
- Photosensitivity
- Difficulty with colour vision or night vision
- Drowsiness
- Calcinosis
- Teatrogenic
- Depression

## 8.4 Monitoring

All patients starting oral isotretinoin need fasting bloods (Full blood count, urea and electrolytes,

liver function tests, glucose and lipid profile) before starting their treatment; until recently, monthly testing was the standard. Today it is known that the evidence does not support such frequency and a test done every 3 months might be sufficient. Repeat these bloods again 3 months after finishing. In the majority of cases a transient increase in cholesterol and triglycerides is all that is observed, which reverts to its pre treatment levels after finishing the treatment. Women of child-bearing age also need a pregnancy test before starting oral isotretinoin, during the treatment and 5 weeks after completing a course of isotretinoin. The need for repeated medically supervised pregnancy tests every month should be determined according to local practice including consideration of the patient's sexual activity and recent menstrual history (abnormal menses, missed periods or amenorrhea). Where indicated, follow-up pregnancy tests should be performed on the day of the prescribing visit or in the 3 days prior to the visit to the prescriber.

Women can only be prescribed oral isotretinoin one month at a time (no repeat prescriptions) and have to fill their prescription within 7 days of it being issued. Oral isotretinoin is contraindicated in breast-feeding mothers.

It is vital to get all patients to sign a consent form informing them that it may cause mood disorders and/or depression and that they are to report to their doctor immediately if they have any mental health issues. All women should sign a consent form agreeing to avoid pregnancy for 1 month before, throughout and 1 month after completing the course of treatment. For children less than 18 years of age, a parent or legal guardian has to sign the consent form in the presence of the child.

Keep clinical photographs before and after completing the course of oral isotretinoin (Fig. 8.4a, b). It is also important to inform the patient's GP that they are starting this medication. Special programs are available on some computerized medical records to prompt the doctor or nurse to check and record the various recommended items at each visit so as not to forget anything.



**Fig. 8.4** (a) Nodulocystic acne pre oral isotretinoin in a 17-year-old patient. (b) Same patient 3 months after finishing a 6-month course of oral isotretinoin

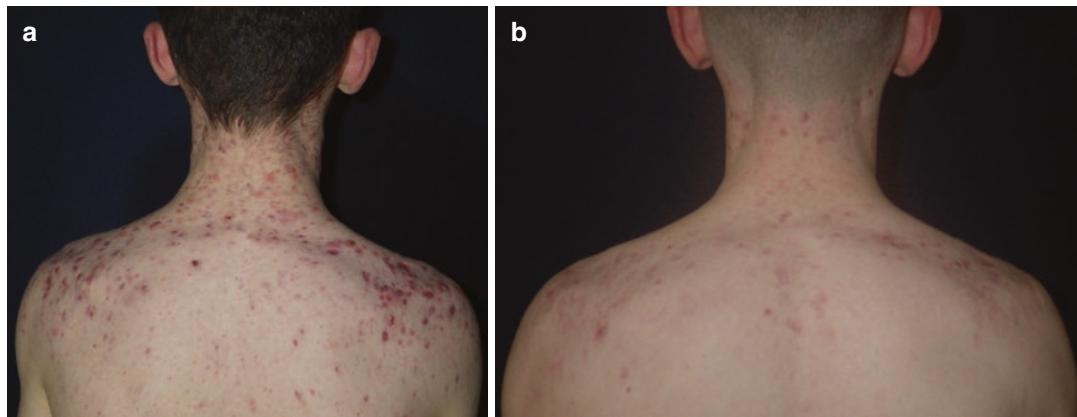
## 8.5 Dosage

The dose of oral isotretinoin is usually 0.5 mg/kg/day to be taken in one or two divided doses with food. If there is a poor response and not too many side effects after 1 or 2 months, the dose can be gradually increased to a maximum of 1 mg per kilogram per day. Skin types I and II are usually more sensitive. Oral isotretinoin is continued until all or almost all the spots have cleared up. The total dose is usually around 120 mg/kg. A course usually lasts 4–6 months but can take up to 9 months in some severe cases especially if there is extensive acne on the back and chest (Fig. 8.5a, b). Some dermatologists are experi-

menting with low dose isotretinoin over a longer period of time (20–30 mg × 3 times per week over 12–18 months) for less severe acne that might otherwise have to be treated with oral antibiotics. Low doses have the advantage of reducing side effects like dry lip, excess skin dryness and photosensitivity, thus improving patient's compliance.

## 8.6 Outcome

Oral isotretinoin clears acne and almost three out of every four of patients (72%) who complete a course are permanently cured [1, 7–9]. The others



**Fig. 8.5** (a) Severe acne in a 15-year-old boy before oral isotretinoin. (b) Same patient after 9 months of oral isotretinoin



**Fig. 8.6** Relapsing nodulocystic acne in a 22-year-old woman

may develop a relapse of their acne a few months or few years later. In general, patients are less tolerant to even mild acne after completing a course of oral isotretinoin which has resulted in a period of clear skin. When acne relapses after the course



**Fig. 8.7** Acne keloids in a 23 year-old-female. These lesions do not respond to isotretinoin treatment

of oral isotretinoin, it is usually less severe than the original acne and often responds to simple topical treatments or a combination of topical and standard oral therapies. Approximately one in five patients (22%) may develop more severe acne post oral isotretinoin that may require a second course [1] (Figs. 8.6 and 8.7).

Patients should avoid laser treatment, chemical peels and wax epilation during treatment and for at least 6 months after completing a course of oral isotretinoin.

## 8.7 Conclusion

Since pregnancy prevention and psychiatric assessment are the most crucial parts of managing patients on oral isotretinoin, a GP with a spe-

cial interest in dermatology and experience in psychiatry and family planning can be considered suitable to prescribe this drug. The threat of litigation is very high and so it should only be prescribed by physicians with experience with this drug [10].

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# Polycystic Ovarian Syndrome (PCOS)

9

David Buckley

## Key Points

- Polycystic ovarian syndrome (PCOS) occurs in women of child bearing age and causes excess androgen production.
- This can lead to skin, gynaecological, psychological and sometimes general health problems. Long term complications are type 2 diabetes and heart disease.
- It is not essential to demonstrate polycystic ovaries to make the diagnosis of PCOS.
- Treatments are targeted towards the primary gynecological cause and obesity reduction as well as to the secondary skin and metabolic problems.

## What to Tell the Patient

- PCOS is a treatable condition.
- Weight loss is important if the person is overweight.
- Oral contraceptives with a relatively high oestrogen and a progesterone can help most of the skin and gynaecological problems associated with PCOS.
- Do not smoke if you are prescribed the pill.

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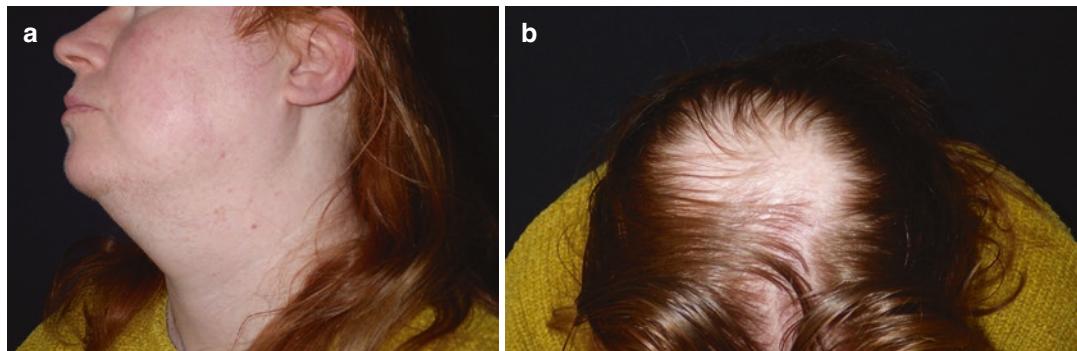
## 9.1 Introduction

PCOS is a multi-system disease that occurs in women after the menarche and before the menopause which causes excess androgen secretion. Polycystic ovaries can be seen with ultrasound in up to 20% of healthy women [1]. However, the majority of women with polycystic ovaries do not have features of polycystic ovary syndrome (PCOS) and do not require intervention. Prevalence figures vary depending on diagnostic criteria used, but PCOS is thought to affect 5–15% of women of reproductive age [2, 3]. It is probably more common than realised as many cases go undiagnosed or undetected.

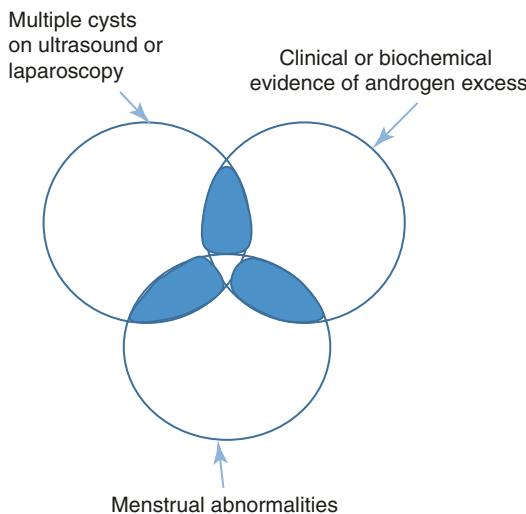
## 9.2 Clinical Features and Diagnosis

Women with PCOS may present with dermatology and gynaecology problems, weight gain, psychological symptoms [1] (mood swings, depression, anxiety, poor self-esteem) or sleep apnoea. PCOS signs and symptoms tend to be more severe in obese patients.

In dermatology, most patients with PCOS present with either acne, hirsutism or female pattern alopecia (androgenic alopecia) (Fig. 9.1a, b). Many of these women may also have weight problems, menstrual abnormalities, and fertility issues.



**Fig. 9.1** (a) Hirsutism in a 33 year-old-female with PCOS who is on spironolactone 100 mg daily. (b) Same female with male patterned hair loss and PCOS



**Fig. 9.2** Diagnosis of PCOC (2 out of 3)

#### Diagnosis of PCOS is based on the presence of any two of the following:

- Polycystic ovaries on ultrasound or laparoscopy.
- Menstrual irregularities (no periods or very occasional periods).
- Clinical and/or biochemical evidence of androgen excess.

(Rotterdam criteria): [3, 4] (see Fig. 9.2)

It is not essential to have cysts visible on ultrasound. Neither is a hormone analysis essential for diagnosis (Table 9.1). For instance, a woman with acne and/or hirsutism with oligomenorrhoea or amenorrhoea may be diagnosed as having PCOS. In this situation, neither an ultrasound nor

**Table 9.1** Investigations for women suspected of having PCOS<sup>a</sup>

Full blood count, urea and electrolytes, liver function tests
FSH/LH (LH elevated >10 IU/L, LH:FSH ratio increased (>2), with FSH normal)
Oestradiol
Sex hormone binding globulin (SHBG- reduced)
Free Testosterone: (raised >2.5 nmol/l). If total testosterone is >5 nmol/L, exclude androgen-secreting tumours and congenital adrenal hyperplasia
Random Blood Sugar
HbA1C
Thyroid function tests (TFT's)
Lipid Profile
B12, folate, ferritin
Prolactin
Ultrasound of the ovaries and the adrenals (the presence of cysts does not prove PCOS and cysts do not have to be present to make the diagnosis of PCOS)
Dihydroepiandrosterone sulphate (DHEAS) (for those with severe or rapidly progressive hyperandrogenism)
17-hydroxyprogesterone

<sup>a</sup>Bloods best taken during the first week after menstruation when not on any hormone treatment

hormonal analysis needs to be done to confirm the diagnosis.

An elevation of free testosterone in combination with a low sex hormone binding globulin (SHBG) is the most sensitive way to establish the presence of hyperandrogenism. If the total testosterone is normal (in the absence of the oral contraceptive pill), the diagnosis of PCOS is effectively ruled out. The patient needs to be off the OCP for at least 1 month before testing.

**Table 9.2** Differential diagnosis for PCOS

Cushing's disease
Later onset congenital adrenal hyperplasia
Androgen secreting tumours (ovary or adrenal)
Ovarian hyperthecosis

**Table 9.3** Clues that there may be a more serious cause of androgen excess

Abrupt onset of symptoms
More severe disease
Rapid progression
Older age of onset
Very high serum androgens (testosterone of >5 nmol/l)
Free testosterone level more than double the normal
Signs of virilisation (deep voice, cliteromegaly)

### 9.3 Differential Diagnosis

Most women with clinical and/or biochemical evidence of androgen excess and menstrual irregularities will have PCOS. However, other more sinister and serious factors can be responsible (Table 9.2).

Some of the signs and symptoms of PCOS can be mimicked by **androgen secreting tumours**. This should be investigated if there is rapid onset of severe acne or hirsutism especially if these problems start in women over the age of 35 (Table 9.3). Deepening of the voice, cliteromegaly, total testosterone of >5 nmol/l or free testosterone level more than double the normal might also give clues to an underlying androgen secreting tumour of the ovary or adrenal gland. Measurement of dehydroepiandrosterone sulphate (DHEAS) should be included for those with severe or rapidly progressive hyperandrogenism to screen for a primary adrenal source, as DHEAS is a marker for adrenal hyperandrogenism. Raised 17 OH progesterone suggests late onset congenital adrenal hyperplasia.

### 9.4 Pathophysiology

Polycystic ovaries are thought to develop when ovaries are stimulated to produce excessive amounts of male hormones, particularly testoster-

one. This stimulation is caused by excess LH produced by the anterior pituitary in response to increased gonadotrophin-releasing hormone (GnRH) or through high levels of insulin caused by insulin resistance. High insulin levels also suppress hepatic production of sex hormone-binding globulin (SHBG) leading to higher levels of free circulating androgens, further adding to the hyperandrogenaemia. The underlying endocrine disturbance can exist in the absence of polycystic ovaries. Androgen levels may not correlate with clinical presentation and serum androgen levels may be normal [5].

### 9.5 Treatment

The first line treatment for all forms of PCOS is diet and exercise to reduce weight in the overweight. Specific treatments should be tailored to the patient's major presenting complaints:

**Acne** can persist into the thirties or forties in women with PCOS and often responds poorly to oral antibiotic and topical therapies. Acne in PCOS may require long term treatment to prevent relapse (see Chap. 7—acne in women).

If a woman with PCOS and acne is overweight even a 5% drop in weight can help. Topical treatments such as topical retinoid, benzoyl peroxide or azelaic acid gel may help but most women with troublesome acne in PCOS will also need systemic treatment. While they may get some response from oral antibiotics, best results are obtained with hormonal treatments such as an oral contraceptive pill (OCP) with relatively high oestrogen dose and a progesterone with low androgenic action (e.g.: "Yasmin®" or "Cilest®").

"Dianette®" is a popular choice in women with PCOS, as it has a strong anti-androgen (cyproterone acetate) and 35 mcg of oestrogen (0.035 mg ethinylestradiol and 2.0 mg cyproterone acetate) which can be very effective. Results can be slow and can take 6 or 12 months before improvements are seen. Relapse is common when the treatment is stopped. "Dianette®" can only be used in women of reproductive age and should be avoided in smokers and those with hypertension, hypercholesterolaemia, obesity or a history of

venous thromboembolism (VTE) (see Chap. 7, Table 7.6). All patients on Dianette® need to be warned about the possibility of VTE and be given the warning signs to look out for.

For women who cannot go on an OCP, non hormonal anti-androgens such as spironolactone combined with topical anti-acne treatments may help. Spironolactone acts as an anti-androgen and can help acne, androgenic alopecia and hirsutism. It is teratogenic so effective contraception should be used in women of child bearing age on this drug. It is usually started at a small dose (25 mg/day) and gradually increased to a maximum of 200 mg/day if required and tolerated. It can cause hyperkalemia and the Summary of Product Characteristics recommends that electrolytes should be monitored regularly. Recent research in JAMA suggests that monitoring electrolytes in healthy, young females is not necessary [6].

Some women with more resistant or severe acne may require oral isotretinoin but the relapse rate is higher in women with PCOS (see Chap. 8).

**Hirsutism** is defined as excessive terminal hair (long, coarse and pigmented) that grows in a male pattern (beard area, lower abdomen and chest) and is more common in women with PCOS (Fig. 9.1a). While traditional methods such as bleaching, plucking, shaving or waxing may help and are safe, many women request a more specific treatment such as the oral contraceptive pill. “Yasmen®” (ethinyl oestradiol 30 + drospirenone) and “Yasminelle®” (ethinyl oestradiol 20 + drospirenone) arrest progression but do not reverse hirsutism. “Dianette®” (ethinyl oestradiol 35 + cyproterone 2 mg) on the other hand gives substantial reduction of hirsutism.

Relapse is almost invariable when stopped, therefore these treatments may need to be taken over a prolonged period. Eflornithine 11.5% cream twice a day (“Vaniqa®”) can slow down hair growth but can be slow to work. If there is no improvement after 2 or 3 months the treatment should be stopped. Laser or IPL hair removal can be helpful in women with dark hair and light skin but the success rate is limited in women with PCOS. Laser hair removal should be done after hormonal treatment, on the residual hair and not

in patients that have not controlled their excess androgen levels (see Chap. 40).

**Female pattern alopecia** (diffuse, non-scarring hair loss that presents with prominent thinning of frontal, central, parietal scalp hair) or sometimes **male pattern (androgenic) alopecia** can occur in approximately 22% of women with PCOS [7] (Fig. 9.1b). “Dianette®” or an OCP with low androgenicity are less effective against alopecia than against acne and hirsutism, but will give some improvement of alopecia in 30% of patients. Topical minoxidil (“Regaine®”) gives medium regrowth in 15% of patients. Spironolactone or hair transplant may also have to be considered (see Chap. 40).

PCOS is linked with the **metabolic syndrome and hyperinsulinaemia**. 10% will go on to develop diabetes at some stage, particularly if they are overweight or if there is a positive family history of diabetes. There is also an increased incidence of abnormal lipid profile in women with PCOS. Annual fasting blood sugar and lipids should be tested, especially in the overweight or those with a family history suggesting increased risk of cardiovascular disease.

If there is **oligomenorrhoea or amenorrhoea**, women should consider the oral contraceptive pill or the “Mirena®” IUD to give endometrial protection and reduce the risk of endometrial cancer in later life.

**Fertility issues**, if they arise, are best dealt with by a gynaecologist. Clomiphene can induce ovulation in 75–80% of women but there is a risk of multiple pregnancies. Weight loss and metformin may also improve the chances of conceiving [8].

## 9.6 Conclusion

PCOS is a common and debilitating condition which mainly affects young women. Early diagnosis and correction of any weight problems are key to successful management. Specific treatments can be targeted towards the primary presenting features such as, dermatology (acne, hirsutism, and alopecia), gynaecology (menstrual irregularities or fertility issues) or metabolic dysfunction.

*Patient information resource:* “Verity” is a self-help group for women with polycystic ovary syndrome (PCOS). See their website at: [www.verity-pcos.org.uk](http://www.verity-pcos.org.uk).

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# Rosacea

10

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## Key Points

- Rosacea is an inflammatory disease most commonly found in fair (type 1 and 2) skin.
- Most patients have papules, pustules and telangiectasia (broken veins) but *no* comedones.
- All patients with rosacea should protect their face from ultra violet light.
- Topical steroids aggravate rosacea.

## What to Tell the Patient

- There are safe, effective treatments for rosacea.
- Rosacea often goes through phases of relapse and recurrence.
- It is vital to protect the facial skin from natural and artificial ultraviolet light by the careful use for a SPF 30 or greater and a broad rimmed hat when outdoors.
- Mild cases will respond to topical treatments.
- More troublesome cases may need topical and tablet treatments.
- It usually takes 6–12 weeks to get a good response from rosacea treatments.
- If you are left with a lot of redness after a course of rosacea treatment, you may benefit from laser treatment for your broken veins.

## 10.1 Introduction

Rosacea is also known as “the curse of the Celts”. It is most commonly seen in type 1 and type 2 skins and affects up to 13.9% of the Irish population. There is a positive family history in 30% of patients. It is 2–3 times more common in females. It is sometimes referred to as “acne rosacea” although it is a different disease to acne.

## 10.2 Clinical Features and Diagnosis

It usually presents with multiple small papules and pustules (pimples) on the face with a red background due to telangiectasia (Figs. 10.1 and 10.2). The rash, which is confined to the face, is usually symmetrical affecting the convex areas of the centre of the face (cheeks, nose, forehead or chin). Some cases can be unilateral. Eye involvement (usually blepharitis) can occur in more than 50% of patients and can be the presenting feature in up to 20% of patients with rosacea (Fig. 10.3a, b). Rosacea is diagnosed clinically as there is not definitive biochemical or histological diagnostic features (Tables 10.1 and 10.2).

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**Fig. 10.1** Typical rosacea



**Fig. 10.2** Severe rosacea



**Fig. 10.3** (a) Rosacea of the nose and eyes. (b) Blepharitis associated with rosacea

### 10.3 Differential Diagnosis

Rosacea can be confused with other papular-pustular rashes on the face (acne, peri oral dermatitis, folliculitis, drug eruptions) or conditions that cause a red face such as seborrhoeic dermatitis, psoriasis, telangiectasia, keratosis pilaris, lupus,

cellulitis, steroid damage, photosensitivity, or dermatomyositis (see Chap. 17 on the red face).

## 10.4 Pathophysiology

The hair follicle mite (*demodex folliculorum*), which is a normal inhabitant on the facial skin, is often found in excessive amounts in the skin in patients with rosacea but their role in the pathophysiology of rosacea is unclear. Treatments that reduce the amount of mites on the skin (e.g. ivermectin or metronidazol) seem to help with rosacea.

Rosacea is an inflammatory process [1]. If there is redness (telangiectasia) but no papules or pustules then it may not be rosacea, just simply broken veins (Heliodermatitis). Rosacea can be distinguished from acne by the redness of the skin and

the absence of comedones (blackheads and whiteheads) (Fig. 10.4). Rosacea usually occurs in an older age group than those with acne (over 30 years old) but some unfortunate individuals can grow out of acne and into rosacea. At a certain stage some of these patients might have features of both conditions (“red acne”). Fortunately, many of the treatments for acne can also help rosacea (azelaic acid gel or oral tetracyclines). Rosacea can occasionally be seen in teenagers.

Unlike acne, rosacea is usually made worse by ultraviolet light, so strict sun avoidance is important. Patients with rosacea may also flush or blush easily and should avoid anything that causes blushing (e.g. stress, excess heat, strenuous exercise). Topical steroids (TS) (even weak ones) should be strictly avoided in rosacea. While they may give a temporary improvement at the beginning of treatment, the condition worsens if TS are continued long term. Rosacea can flare up badly if the TS is stopped suddenly without topical or oral treatments as outlined below.

There is not good evidence to implicate food as a cause or aggravating factor in rosacea. However, it may help if patients avoid foods or drinks that make them flush or blush (e.g. spicy foods, caffeine, alcohol). They should also avoid excessive heat such as saunas or sitting close to a hot fire. Smoking and alcohol consumption make rosacea more severe. Green based cosmetic camouflage may help hide the redness. The Red Cross and the UK based charity, Changing Faces, can help with cosmetic camouflage. ([www.changing-faces.org.uk](http://www.changing-faces.org.uk)).

**Table 10.1** Guidelines for the diagnosis of rosacea

Presence of one or more of the following primary features:
Flushing (transient erythema)
Permanent erythema
Papules and pustules
Telangiectasia
May include one or more of the following secondary features:
Burning or stinging
Plaques
Dry appearance
Oedema
Ocular manifestations: (Blepharitis, styes, chalazia, and corneal damage)
Phymatous changes

**Table 10.2** Subtypes of rosacea

Erythematotelangiectatic	Flushing and persistent central facial erythema with or without telangiectasia.
Papulopustular	Persistent central facial erythema with transient, central facial papules or pustules or both.
Phymatous	Thickening skin, irregular surface nodularities and enlargement. May occur on the nose, chin, forehead, cheeks, or ears.
Ocular	Foreign body sensation in the eye, burning or stinging, dryness, itching, ocular photosensitivity, blurred vision, telangiectasia of the sclera or other parts of the eye, or periorbital edema.
Steroid rosacea	Severe papules, pustules and telangiectasia which are aggravated by sudden withdrawal of the topical steroid



**Fig. 10.4** Rosacea with no comedones

## 10.5 Topical Treatments

Rosacea is a chronic disease with periods of relapses and remissions which can continue for many years. It is usually worse in pregnancy. Treatment of rosacea is usually either with topical or oral medication or both. Most mild cases will respond to topical metronidazole or azelaic acid 15% gel for 6–12 weeks. Topical ivermectin 1% cream (“Soolantra®”, an antiparasitic agent) is considered more effective than metronidazole and only has to be applied once a day for 6–12 weeks. With topical ivermectin there are no concerns about antibiotic resistance. It should be applied all over the face and not just on the red areas. If facial flushing is a problem, “Mervaso Gel®” (brimonidine tartrate) may help but rebound flushing can occur in up to 20%.

## 10.6 Systemic Treatments

More severe cases may need the addition of an oral antibiotic which has a strong anti-inflammatory action such as a lymecycline or doxycycline. Lymecycline is normally prescribed in the same dose as acne for 1–3 months (300 mg daily). Doxycycline 100 mg daily can cause photosensitivity so all patients should be warned about this unusual side effect. Low dose doxycycline (40 mg

daily) in a modified slow release capsule (“Efracea®”) can be effective and safe in milder forms of rosacea and does not promote bacterial resistance. Oral minocycline (100 mg OD), oral metronidazole or oral ivermectin may help in more severe cases. Oral erythromycin 500 mg BD may be helpful in severe flare-ups in pregnancy. Severe, resistant cases of rosacea might require low dose isotretinoin (0.3 mg/kg/day) or laser treatment. Sometimes 10 or 20 mg of oral isotretinoin three times per week might be sufficient to control rosacea.

Topical and oral medications mentioned above for rosacea will help primarily with the inflammatory features of rosacea (papules and pustules). However some patients may be left with persistent redness and telangiectases after clearing their papules and pustules. The redness may respond to cooling creams kept in the fridge, brimonidine gel (“Mirvaso®”), cosmetic camouflage or laser treatment. Some cases of flushing may respond to beta blockers such as propranolol or carvedilol.

Eye symptoms such as blepharitis conjunctivitis, and irregularity of the eyelid margins may respond to eye lubricants or metronidazole gel applied to the eyelids. Meibomian gland dysfunction causing chalazion or chronic staphylococcal infection that can cause hordeolum (stye) may also occur with ocular rosacea. Some patients may have decreased visual acuity caused by corneal complications (punctate keratitis, corneal infiltrates/ulcers, or marginal keratitis).

These more serious eye problems may need the assessment of an ophthalmologist and treatment with cyclosporine eye drops, oral antibiotics as mentioned above or low dose isotretinoin. Washing the eyelids with diluted Tea Tree oil and applying topical metronidazole or ivermectin carefully to the eyelids may also help. Topical steroid eye ointments or drops should be avoided.

Some patients with rosacea may develop rhinophyma which causes thickening of the skin on the nose with enlargement of the sebaceous glands (Fig. 10.5). This can lead to a red bulbous



**Fig. 10.5** Rhynophyma and rosacea

nose which can be unsightly and the patient may be mistakenly accused of being a heavy drinker. Thickening of the skin can also rarely affect the chin (gnathophyma), the ear (otophyma) or the forehead (metophyma). It is much more common in males and in some cases may need surgical treatment to correct the deformity.

## 10.7 Conclusion

With careful treatment using topical rosacea agents and/or systemic agents, most flare ups of rosacea can be settled within 6–12 weeks. Photo protection with a factor 30 SFP or greater and a broad brimmed hat should help prevent relapses.

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# Hidradenitis Suppurativa

11

David Buckley

## Key Points

- Hidradenitis Suppurativa (HS) is a chronic inflammatory disease resulting in papules, pustules and sometimes cysts and abscesses most commonly found in the axillae, groin, perianal and under the breasts in young adults.
- It is three times more common in females.
- There are many possible causes including genetic, hormonal, autoimmune, infective and inflammatory factors.
- Almost all cases of HS, regardless of the severity, can now be treated with appropriate topical, systemic and/or surgical treatments.

## What to Tell the Patient

- HS can lead to embarrassment, anxiety and even depressions especially in severe cases.
- HS can be painful and can affect your quality of life causing difficulty with work, sports, sex and leisure activities.
- Fortunately HS is a treatable condition.
- Treatments can be slow and it can take 3–6 months to see a significant improvement.
- Most patients will require a combination of topical and tablet treatments for at least 6 months.

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- Severe cases may require injection treatments with the newer biological agents and perhaps surgical treatments.

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## 11.1 Introduction

Hidradenitis Suppurativa (HS) is a chronic inflammatory disease affecting the apocrine gland-bearing skin. It usually presents with recurrent nodules and abscesses (“boils”) which result in sinus tract formation and scarring. Papules and pustules can occur. It typically affects the axillae, the groin, the perianal area or under the breasts in young adults. As it can resemble acne (although not in a classical acne distribution), it is sometimes referred to as “acne inversa”. However, unlike acne, HS can be a much more deep-seated chronic, inflammatory condition that progresses onto nodules and cysts with recurrent abscesses, sinuses and hypertrophic or atrophic scars (Figs. 11.1 and 11.2).

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## 11.2 Clinical Features and Diagnosis

The diagnosis of hidradenitis suppurativa is clinical (Table 11.1). Most patients present with bilateral, recurrent painful papules, pustules, nodules, cysts, sinuses or scars in the axillae, groin and sometimes under the breasts and perianal area.



**Fig. 11.1** Severe hidradenitis suppurativa in the axilla.  
Credit: Myriam Raquel González Oviedo



**Fig. 11.2** Severe hidradenitis suppurativa in the groin.  
Credit: Myriam Raquel González Oviedo

**Table 11.1** Diagnostic criteria of hidradenitis suppurativa (adopted by the Second International Conference on Hidradenitis suppurativa, March 5, 2009, San Francisco, CA US)

1. Typical lesions, i.e., deep-seated painful nodules: ‘blind boils’ in early lesions; abscesses, draining sinuses, bridged scars and ‘tombstone’ double-ended pseudo-comedones in secondary lesions
2. Typical topography, i.e., axillae, groins, perineal and perianal region, buttocks, infra and inter mammary folds
3. Chronicity and recurrences

All three criteria must be met for establishing the diagnosis

Milder cases may be diagnosed as simple boils but the recurrent nature and distribution of the sores should help make the diagnosis. Severe cases are more obvious with recurrent abscesses, sinuses and scars. Lesions persist for months

with pain and smelly discharge. Secondary infection can occur.

HS severity can be classified as in the Hurley stages (see Table 11.2). HS, especially stage 2 and 3, can have considerable effects on the patient’s quality of life because of chronic pain and smelly discharge which can lead to social isolation, low self-esteem, depression and even suicide [1].

### 11.3 Differential Diagnosis

Milder forms of HA may be confused with acne or boils. More severe cases can be confused with deep seated infections, tumours or cutaneous manifestations of inflammatory bowel diseases (Crohn’s). (see Table 11.3). Underlying factors such as diabetes or anaemia which can aggravate HS should be sought (Table 11.4) [2].

### 11.4 Pathophysiology

The etiology of HS is unclear. There can be genetic, hormonal, autoimmune, infective and inflammatory factors [3]. HS is three times more

**Table 11.2** Hurley stages for hidradenitis suppurativa

- Stage 1—solitary or multiple isolated abscesses without scarring or sinus tracks
- Stage 2—recurrent abscesses, single or multiple widely separated lesions with sinus track formation
- Stage 3—diffuse or broad involvement with multiple interconnecting sinus tracks and abscesses

**Table 11.3** Differential diagnosis of hidradenitis suppurativa

**Infections:**

Bacterial—Carbuncles, furuncles, abscesses, ischiorectal/perirectal abscesses, Bartholin’s duct abscess

Mycobacteria—TB

STI—Granuloma inguinale, lymphogranuloma venereum, syphilis

Deep fungi—Blastomycetes

**Tumors or cysts:**

Epidermoid cysts, Bartholin’s cysts, pilonidal sinus

**Miscellaneous:**

Crohn’s disease, anal or vulvovaginal fistulae

**Table 11.4** Investigation for patients with hidradenitis suppurativa

- Full blood count
- Urea and electrolytes, liver function test
- Random blood glucose and HbA1c
- B12, folate, ferritin
- Thyroid function test
- ESR and C-reactive protein (CRP)
- Coeliac antibody test
- Test for polycystic ovarian syndrome in females, especially if they show other features of PCOS such as acne, hirsutism, amenorrhoea or cysts in their ovaries

common in females and usually starts in the late teens or early twenties. More severe cases however are more common in men. Family history is positive in one-third of patients with hidradenitis suppurativa [4].

Although hidradenitis suppurativa appears to be a follicular disorder in the apocrine gland bearing skin the exact aetiology of HS are not fully known. The initial event appears to be occlusion of the hair follicle [5].

HS can persist for many years but is unusual after the menopause. It is more common in smokers and those who are overweight [5]. It can be associated with other dermatological conditions such as acne, psoriasis, polycystic ovarian syndrome and pyoderma gangrenosum [5]. It is more common in people with diabetes, inflammatory bowel disease (Crohns) and metabolic syndrome [4].

Complications of hidradenitis suppurativa include lymphoedema, fistula formation (into urethra, bladder, or rectum), anaemia, arthritis and rarely secondary amyloidosis. Squamous cell carcinoma can arise in long standing lesions particularly in the buttock area, and in male patients [5].

## 11.5 Treatment

All stages of HS may be helped by general lifestyle modification by such as losing weight and smoking cessation. Reducing friction and moisture in affected areas by wearing loose clothing and fragrance-free antiperspirants may help. Washing with an antiseptic such as chlorhexidine

("Hibiscrub®") or taking "Milton baths®", twice a week, can help prevent recurrent infections (see Chap. 66).

Stage 1 disease may respond to various topical antibacterial therapies such as topical clindamycin lotion 1% for a maximum of 12 weeks [5]. Acutely infected cysts, nodules or sinuses may respond to a short course of an oral antibiotic such as flucloxacillin 500 mg four times a day for 7–14 days. Abscesses may need to be surgically drained under local anaesthetic or general anaesthetic.

Stage 1 and 2 disease may require oral anti-inflammatory treatment with long courses of systemic anti-acne type antibiotics such as lymecycline, 300 mg daily or doxycycline, 100 mg daily for at least 4 months. As in acne, more severe disease may benefit from higher doses of these drugs such as lymecycline 300 mg twice a day or doxycycline, 100 mg twice a day for at least 3–6 months [6]. Females may respond to long courses of combined oral contraceptive with an anti-androgenic progesterone such as drospirenone (e.g. "Yasmin®") or cyproterone acetate ("Dianette®"). These combined oral contraceptive pills have a slightly higher incidence of thromboembolic events compared to the older second generation or the newer fourth generation pills. They should not be used long-term in smokers, patients who are grossly obese, patients with hypertension or a history of thromboembolic disease. Spironolactone may be a safer option in these high risk groups.

Acute flare-ups of nodules or cysts may respond to intralesional steroids. Short courses of systemic steroids (0.5–0.7 mg/kg/day) may help acute flare-ups in some patients with HS [5].

Chronic stage 2 and 3 disease may require a 12 week course of clindamycin, 300 mg BD combined with rifampicin, 300 mg BD [7]. Patients should be warned that rifampicin can discolour urine and tears to red. Rifampicin is a potent inducer of some of the cytochrome P450 enzymes and therefore can also influence the metabolism of other drugs such as anticonvulsants or the oral contraceptive pill [8]. The combination of clindamycin and rifampicin may cause pseudo-membranous colitis so a patient

should be warned to report to the doctor if they develop persistent diarrhoea on this treatment. Dapsone, oral retinoids such as acitretin or oral ciclosporin may be necessary in more persistent cases [5].

Severe stage 3 disease may require high dose TNF alpha inhibitors such as adalimumab (“Humira®”) given by weekly subcutaneous injections or infliximab (“Remicade®”) given by IV infusion every 2–6 weeks [5]. Ironically, these TNF alpha inhibitors can paradoxically induce new onset HS when used for conditions such as arthritis, psoriasis or inflammatory bowel disease [9].

Stage 2 and 3 disease may require surgical treatment such as incision and drainage, deroofing of nodules, abscesses or sinuses or radical excision of the entire affected area with skin grafting [10, 11].

## 11.6 Conclusion

Hidradenitis suppurativa is a disorder of the terminal follicular epithelium in the apocrine gland-bearing skin. This condition is a chronic disabling disorder mainly in young adults that frequently cause painful, smelly nodules, cysts and abscesses leading to sinus tracts and scarring. This can result in keloids, contractures, and immobility. Even mild disease can have a profound effect on the patient’s quality of life. Milder cases respond to oral treatments similar to those used for acne. More severe cases may require surgery, long term oral antibiotic combinations or some of the newer biological therapies such as the TNF alpha inhibitors.

## 11.7 Useful Information for Patients Is Available from the Following Sites

[irishskinfoundation.ie](http://irishskinfoundation.ie)  
[patient.co.uk](http://patient.co.uk)

Hidradenitis Suppurativa Foundation (UK):

<http://www.hs-foundation.org/>

The Hidradenitis Suppurativa Trust (USA):

[www.hstrust.org](http://www.hstrust.org)

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# Hyperhidrosis (Excessive Sweating)

12

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## Key Points

- Excessive sweating can be localized (e.g. hands or feet) or generalized, primary or secondary. Generalised hyperhidrosis is more likely as a result of an underlying condition.
- If the generalized sweating is of recent onset and/or associated with other symptoms such as fever, weight loss, fatigue or swollen glands, then a thorough search for an underlying cause should be made.
- Treatment of generalised hyperhidrosis is usually by dealing with the underlying cause and perhaps with systemic medicines.
- Localised hyperhidrosis is often idiopathic (of no known cause) and may be hereditary.
- Primary idiopathic focal hyperhidrosis usually stops during sleep.
- Focal hyperhidrosis often responds to local measures such as powerful antiperspirants, iontophoresis or botulinum toxin (Botox®).

## What to Tell the Patient

- Wearing appropriate clothing and pads may help. Staying calm and cool is also important.
- Special garments are available with a sweat-absorbent wicking interior that pulls sweat

away from the body and an ultra-thin, extremely breathable, sweat-repellent exterior to block sweat stains from showing. Open sandals or leather soled shoes can also help.

- Self help groups such as [www.sweathelp.org](http://www.sweathelp.org) or [www.hyperhidrosisuk.org](http://www.hyperhidrosisuk.org) are great resources for patients.

## 12.1 Introduction

Hyperhidrosis is a disease characterised by sweating (perspiration) in excess of the normal physiologic amount necessary to maintain body temperature. Primary or idiopathic hyperhidrosis and secondary hyperhidrosis are the two main categories. Patients can have excessive sweating either in a localized area (focal) or over the entire body (generalised). Primary disease is usually localized, affecting the soles, palms, and axillae in various combinations and with varying degrees of severity (Fig. 12.1). Secondary hyperhidrosis can be generalised or focal. In secondary hyperhidrosis the symptoms are due to one of a large number of medical conditions, including endocrine disorders, neurological problems, use of certain drugs, cancer, chronic infections, dermatologic syndromes, and conditions associated with excess catecholamine discharge (Table 12.1).

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**Fig. 12.1** Hyperhidrosis of the feet

**Table 12.1** Causes of generalized sweating

**Infections:** Acute viral or bacterial infections; chronic infections, e.g.: TB, malaria, brucellosis.

**Drugs:** Alcohol, cocaine, heroin, ciprofloxacin, acyclovir, esomeprazole, antidepressents, propranolol, etc.

**Endocrine:** Diabetes, hyperthyroidism, menopause, pregnancy, carcinoid syndrome, hyperpituitarism, pheochromocytoma, acromegaly.

**Neurological:** Stroke, spinal cord injury, gustatory after parotidectomy or primary gustatory, Parkinson's disease.

**Others:** Lymphoma and other myeloproliferative disorders, congestive cardiac failure, anxiety, obesity, rheumatoid arthritis.

## 12.2 Clinical Features and Diagnosis

Some patients can be very embarrassed with their excessive sweating which can ruin clothes, make writing or shaking hands difficult and can interfere with certain occupations or sports

(e.g.: golf, tennis, rock climbing). Bromohydrosis (smelly sweating) can often make the problem even more troublesome for the patient. Many are reluctant to seek medical help in the mistaken belief that nothing can be done. Idiopathic focal hyperhidrosis usually starts in childhood although many patients delay seeking medical help until they reach adult life. Between a third and half of the patients have a positive family history. Interestingly, idiopathic focal hyperhidrosis does not cause excessive sweating during sleep.

Hyperhidrosis is usually diagnosed clinically. If the presentation is characteristic of primary focal hyperhidrosis and there is no evidence of an underlying cause, no laboratory tests are required. If there is a suspicion of secondary hyperhidrosis due to some medical condition special tests and investigations may be required (Table 12.2).

There are various methods to quantify the amount of sweat being produced on the palms or axilla such as gravimetric measurement. This test is often utilized in clinical trials and is not part of routine clinical practice. After drying the surface, a pre-weighed filter paper is applied to the palm or axilla for a period of time measured by stopwatch. The paper is then weighed and the rate of sweat production is calculated in mg/min. >20 mg/min in men and >10 mg/min in women in the axilla is considered excessive.

**Table 12.2** Investigations for hyperhidrosis if an underlying medical cause is suspected

- Full blood count
- Blood film for malarial parasites if overseas travel
- ESR and/or CRP
- Renal function tests and electrolytes
- Liver function tests
- Fasting blood glucose or HbA1C
- Thyroid function tests
- Chest x-ray (may be useful to identify an intrathoracic neoplasm)
- HIV testing
- Urinalysis
- Hormonal studies in women in the perimenopausal age group (LH, FSH + oestrogen)

## 12.3 Differential Diagnosis

Excessive sweating may be focal or generalised, primary or secondary. Most cases of generalised sweating are due to an underlying cause (see Tables 12.1 and 12.3). If the generalised sweating is of recent onset and/or associated with other symptoms such as fever, weight loss, fatigue or swollen glands, then a thorough search for an underlying cause should be made (Table 12.2).

Focal hyperhidrosis is far more common and can affect up to 1% of the population (Table 12.4). It usually affects the hands, feet, axilla, face or scalp. Treatment depends on the location, the severity and the ability of the patient to cope.

## 12.4 Pathophysiology

Although the exact pathophysiology of primary hyperhidrosis is yet to be determined, there is much evidence for abnormalities in autonomic nervous system function. Since hyperhidrosis often begins in childhood and can be familial, the

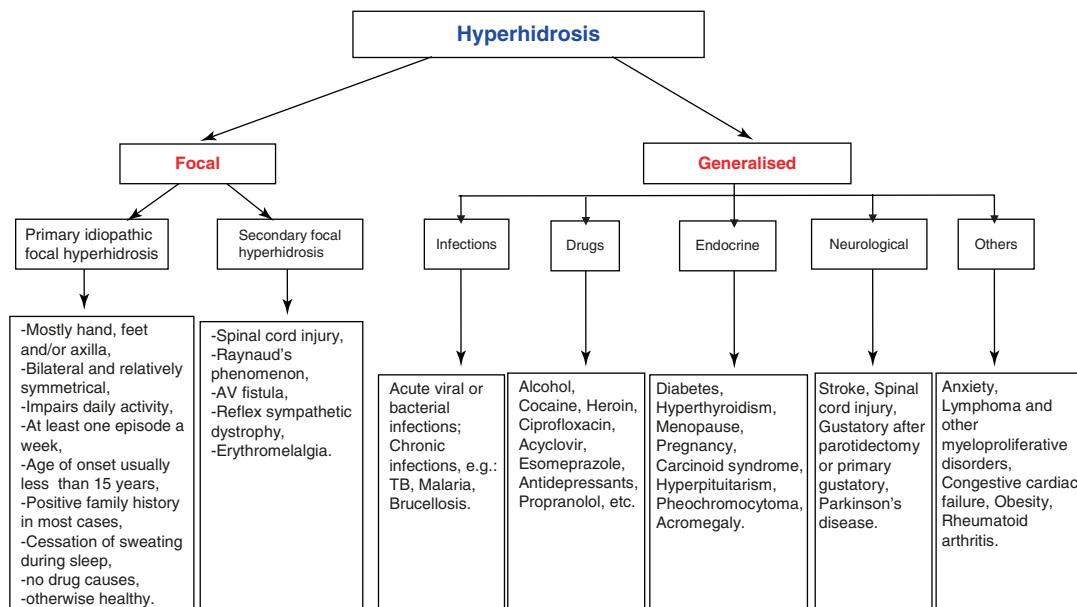
physiologic basis for this disorder may be genetically determined [1].

Sweat glands in patients with hyperhidrosis are not histopathologically different from those in normal patients, nor is there an increase in the number or size of glands. The condition is caused by hyper function of the sweat glands rather than hypertrophy [2]. Patients with primary hyperhidrosis have a higher-than-normal basal level of sweat production as well as an increased response to normal stimuli such as emotional or physical stress.

## 12.5 Treatment

Simple measures include frequent showers, loose clothing, wearing black or white shirts, *antiperspirants* and deodorants may help. Special garments are available with a sweat-absorbent wicking interior that pulls sweat away from the body and an ultra-thin, extremely breathable, sweat-repellent exterior to block sweat stains from showing. Patients should avoid triggers such as excessively hot rooms or spicy foods. Leather soled shoes or open sandals may help with sweaty

**Table 12.3** Hyperhidrosis assessment:



**Table 12.4** Criteria for diagnosing ideopathic focal hyperhidrosis

Focal, visible, excessive sweating for at least 6 months duration without any apparent cause and with at least 2 of the following
• Bilateral and relatively symmetrical
• Impairs daily activity
• At least one episode a week
• Age of onset less than 15 years
• Positive family history
• Cessation of sweating during sleep

feet. Adsorbent insoles and disposable axillary pads can also be extremely helpful. Self help groups can be very supportive for patients (See Patient information leaflet on Chap. 66).

Many patients may have already tried these simple methods. Assuming there is no underlying cause, the next step is to try a more potent antiperspirant such as 20% aluminium chloride ("Dricleor®", "Anhydrol Forte®" or "Sweat Stop®"). These usually come as a role-on or spray and can be used on the axilla, hands or feet. Aluminium chloride is a metal salt that physically blocks the ducts that drain the sweat glands. They come in various strengths. A 20% solution is the standard strength but lower concentrations may be necessary for the face and axilla and higher concentrations may be required for the hand and feet. Aluminium chloride can be quite irritating initially and should be applied overnight and washed off in the morning every second day for the first week or two. If there is a lot of irritation at the start of treatment a potent topical steroid may help if applied in the morning for the first week or two. It may take up to 2 weeks to see results with aluminium chloride. An ordinary antiperspirant/deodorant can be used in the morning during treatment.

If topical agents do not help, the next treatment to consider is *iontophoresis*. This is applied using shallow trays filled with water and a small electrical current from a battery is passed through the water. The exact mechanism of action is unclear but this method can significantly reduce focal sweating in 85% of patients. The hands and/

or feet can be placed in the trays for 20–30 min alternate nights initially but the frequency can be reduced after 2–3 weeks to twice a week if there is a response. "Idrostar®" has a direct pulsed current which is more suitable for children and those with sensitive skin and it also comes with axillary pads ([www.iontophoresis.info](http://www.iontophoresis.info)).

0.05% glycopyrrrolate solution can be added to water to enhance the response. Most patients buy their own unit for home use. For axillary hyperhidrosis small arm pads soaked in water and attached to the machine may help but the best results are obtained when iontophoresis is used for the hands and feet. It should not be used in patients with pacemakers, metal implants or in pregnancy.

For severe resistant cases of hyperhidrosis, *botulinum toxin* (e.g. "Botox®") can be very effective. It is given intradermally and blocks acetylcholine release and neurotransmission. It is most suitable for axillary hyperhidrosis and can cause 75–100% reduction in sweating for up to 6–9 months. It is less effective and more painful to use on the hands.

Generalised hyperhidrosis is best managed by dealing with the underlying cause (see Table 12.1). Oral anticholinergics, which are normally licensed for urinary frequency, can be very helpful in patients with generalised hyperhidrosis or patients with severe resistant forms of focal hyperhidrosis. They are contraindicated in patients with myasthenia gravis, pyloric stenosis, and ileus and they need to be used with caution in patients with narrowing angle glaucoma, gastro-oesophageal reflux, bladder outlet obstruction and heart failure. Probanteline bromide 15–30 mg three times a day, later increasing to 50 mg three times a day as required, may help. Oxybutynine 2.5 mg daily, gradually increasing to 2.5 or 5 mg twice a day is also useful. "Lyrinel XL®" 5 mg once daily is a new long acting oxybutynin anticholinergics which has a convenient once a day dose. Some patients may need to gradually increase the dose up to 30 mg daily. Dry mouth is a problem with all anticholinergics, especially at high doses [3].

Other drugs worth considering are beta blockers (e.g. propranolol 40 mg tid) or calcium channel blockers (e.g. diltiazem 60 mg tid). These may take 2 weeks before patients will see results.

Peri-menopausal sweating is best dealt with by hormone replacement therapy (HRT). For women who cannot or will not take HRT, clonidine, methyldopa or SSRI anti-depressants may help [4, 5].

*Endoscopic thoracic sympathectomy* was used in the past but it can cause compensatory hyperhidrosis in other areas of the body in up to 60% of cases and there have been some fatalities with this procedure which has now fallen out of favour.

*Laser sweat gland ablation* for axillary hyperhidrosis is coming on stream but its effectiveness is yet to be established.

*Facial hyperhidrosis* can be difficult to manage. Topical 0.5% glycopyrronium bromide cream (100 g) is expensive but may help. Weaker forms of aluminium chloride combined with Aloe Vera are available in a spray formulation which can be used on the face ("Sweat Stop®"). Oral anticholinergics can be used in severe cases of facial hyperhydrosis.

Some patients with foot hyperhidrosis may develop **pitted keratolysis**. This causes multiple small white smelly pits on the sole of the feet usually sparing the arch of the foot. It is usually caused by several bacterial species including *Kytococcus* and *Corynebacteria* species. Treatment is with a topical antibiotic such as fusidic acid ("Fucidin®") twice a day for 7–14 days. More severe cases may respond to oral antibiotics such as erythromycin for 7 days followed by measures outlined above to reduce sweating.

## 12.6 Conclusion

Excessive sweating is a common and embarrassing condition. A careful history, a thorough physical clinical examination and investigations may be necessary to distinguish between primary idiopathic hyperhidrosis and hyperhidrosis secondary to an underlying medical condition. Fortunately most cases of primary idiopathic hyperhidrosis can be managed with topical therapies, oral medications, botulinum toxin or special techniques such as iontophoresis.

### Useful Website

<http://www.sweathelp.org/> (USA site)  
[www.hyperhidrosisuk.org](http://www.hyperhidrosisuk.org) (UK site)

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## **Part III**

### **Papulosquamous and Eczematous Dermatoses**



# Atopic Eczema in Children

13

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## Key Points

- Atopic eczema (AE) is an extremely itchy condition. If there is little or no itch, reconsider the diagnosis.
- AE is a chronic disease that can often last years and may go through flare ups and remissions.
- AE is often associated with other atopic diseases in the patient or other first degree relatives (asthma, allergic rhinitis, allergic conjunctivitis).
- AE is a genetic condition which causes dry skin and a defective skin barrier which makes the patient prone to infections, irritants and allergens.
- The cornerstone of treatments is emollients which will restore the skin barrier, reverse the dryness of the skin and ease itch.
- Topical steroids are safe and effective once used within the guidelines and under careful medical supervision.
- Children with AE have very sensitive skin and should avoid soaps, detergents, perfumes and wool next to their skin
- Dry, sensitive skin is the basic underlying problem and liberal, frequent applications of a safe greasy moisturisers is the key to success
- Liberal applications of an emulsion-type moisturiser applied daily during the first 32 weeks of a baby's life may help prevent the development of atopic eczema in those children at risk because of their family history
- Most children with mild to moderate eczema do not require allergy testing or restrictive diets
- Topical steroids are safe when used under careful medical supervision
- Feeding children peanuts (in pureed form) early in childhood may prevent the onset of peanut allergy later in childhood

## What to Tell the Patient

- Atopic eczema is very common affecting approximately one in five children
- Most children with mild to moderate eczema will outgrow their skin condition in time

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## 13.1 Introduction

Atopic eczema (AE) is a common chronic, itchy inflammatory skin disorder that usually lasts months or years. It can affect up to 20–30% of children and 10% of adults and the incident of atopic eczema is still increasing [1]. Like all chronic diseases, (e.g. asthma, rheumatoid arthritis) it can go through phases of exacerbations and remissions, although more than 60% of cases of

childhood eczema will eventually resolve by adulthood. However, most adults who had atopic eczema in childhood will continue to have sensitive skin and may continue to have flare-ups of eczema if exposed to harsh chemicals, (e.g. perfumed soaps, bubble bath) or are in a high risk occupation for contact dermatitis (e.g. nurses, hairdressers, dairy farmers, plasterers etc.).

Atopic eczema in childhood can be considered a family disease. AE can severely affect the quality of life not only of the child but also the parents, who are left exhausted and frustrated in trying to manage their child's chronic itch. There is also a significant financial burden on the family and the state in managing children with atopic eczema.

## 13.2 Clinical Features and Diagnosis

Atopic eczema is a dry, itchy skin condition (see Table 13.1). If there is little or no itch then you should reconsider the diagnosis (see Table 13.2). Most of the clinical features of atopic eczema arise as a consequence of scratching itchy, dry skin. Scratching further compromises the skin barrier function. Therefore, all treatments should be aimed at breaking the itch-scratch cycle. This is primarily achieved by moisturising the skin with the appropriate moisturisers, avoiding soaps and other irritants and targeted therapies with topical calcineurin inhibitors and/or topical steroids.

**Table 13.1** Diagnostic criteria for atopic eczema

Must have:

An *itchy skin* condition (or report of scratching or rubbing in a child)

Plus three or more of the following:

- a. History of *itchiness* in skin creases such as folds of the elbows, behind the knees, fronts of ankles, or around neck (or cheeks in children under 4 years).
- b. History of asthma or hay fever (or *history of atopic disease* in a first degree relative in children under 4 years).
- c. General *dry skin* in the past year.
- d. Visible *flexural eczema* (or eczema affecting the cheeks or forehead and outer limbs in children under 4 years).
- e. Onset in the first 2 years of life (not always diagnostic in children under 4 years).

Most children (85%) with AE will develop the itchy rash in the 1st year of life. 95% of patients with AE will develop the rash before the age of 5 years old. There is usually a history of chronic or relapsing disease.

In babies, AE can cause a generalised rash (Figs. 13.1 and 13.2); as the child grows, it becomes primarily a flexural rash especially affecting the front of the elbows, the backs of the

**Table 13.2** Differential diagnosis for atopic eczema

Seborrhoeic dermatitis
Psoriasis
Scabies
Pityriasis Rosea
Tinea corporis
Numular (discoid) eczema
Conact allergic dermatitis
Drug rash
Ichthyosis
Molluscum dermatitis
Dermatitis herpetiformis
Ichthyosis



**Fig. 13.1** Atopic eczema (infected on face) in a 5-month-old child



**Fig. 13.2** Atopic eczema in a 7-month child which is infected

knees and the neck (Fig. 13.3). The skin is dry with mild erythema and evidence of scratching (e.g. excoriation, bleeding, scabs or crusts). More severe cases can have generalised dry red itchy skin affection most of the body, face and scalp (Fig. 13.4). Infraorbital folds (Dennie-Morgan folds) are common in patients with AE (25% of patients) and may occur as the eyelids are a site of predilection for AE causing typical extra lines or skin folds under the eyes. Paradoxically, the nappy area in children is often spared. There may be a number of reasons for this. The child may not be able to scratch the area because it is inaccessible. In addition, the occlusive effects of the nappy combined with the natural moisturizing effects of urea may help ease itch and prevent scratching.

The most obvious sign of AE in children is the intense scratching which is worse once the child is stripped down to their bare skin. The itch is so severe that distraction rarely stops the scratching.



**Fig. 13.3** Atopic eczema in dark skin with post-inflammatory hyperpigmentation in an 11-year-old boy



**Fig. 13.4** Atopic eczema in a 4-year-old child not responding to clobetasone butyrate ointment

One can recognize an atopic child by their continuous scratching.

Emotional factors can also affect atopic eczema. Children with chronic itch are often tired and irritable as a result of sleep disruption, not

only for the child but also for the parents. The children may scratch more if they are tired and irritable leading to a vicious cycle of itching and scratching. Older children may scratch their skin in an attempt to manipulate their parents so they can get their own way. Scratching can sometimes become habitual and can be helped by behavioural modification. Like all bad behaviour in childhood, it is best dealt with by praising good behaviour and ignoring bad behaviour as much as possible, especially when children are using their bad behaviour to get their own way. Teaching the child to rub or pinch the itchy skin rather than scratch it may help alleviate itch without damaging the skin.

### 13.3 Differential Diagnosis (Table 13.2)

Other common itchy skin conditions in childhood include scabies but this does not usually have the typical flexural rash. Scabies can mimic many conditions. Seborrhoeic dermatitis and psoriasis in children may look similar to AE but these conditions do not usually itch anything as bad as AE. Pityriasis rosea and discoid eczema may itch but their distribution and shape are usually characteristic. Contact irritant and allergic dermatitis are less common in children but there is sometimes an overlap between AE and the other forms of dermatitis (See Table 4 in Chap. 14). As children are on less chronic drugs than adult and the elderly, drug eruptions are less common and usually more obvious in children. Tinea infections can affect the flexures in children and the typical clinical signs may be altered by the inappropriate use of topical steroids (Tinea incognito). This may result in more severe, extensive tinea infections which can be confused with AE. Skin scrapings should be taken if there are any suspicions of a fungal infection.

Ictyosis can cause severe dryness of the skin but it is usually familial and unlike AE, there is normally little or no itch. Children with AE are more prone to infections including Molluscum contagiosum. Molluscum infection can cause an inflammatory reaction resulting in molluscum

dermatitis or a worsening of AE. Dermatitis herpetiformis (DH) can cause a generalised eczematous rash and intense itch. The distribution of DH is usually the opposite of AE (DH usually affects the backs of the elbows, the front of the knees and the scalp). DH may cause tiny vesicles but these are often scratched away before the doctor has time to see them (see Chap. 23).

### 13.4 Pathophysiology

The pathophysiology of atopic eczema remains incompletely understood with a complex interaction between genetic predisposition and environmental triggers. There is often a personal or family history of other IgE related atopic diseases such as asthma, allergic rhinitis or allergic conjunctivitis. Indeed, it has been argued that there is a causal link between eczema and a later onset of allergic rhinitis and asthma ("the atopic march"). About one in every three children with eczema develops asthma or allergic rhinitis during later childhood. Severe eczema, early onset eczema and persistent eczema increase the risks of developing asthma and rhinitis.

Recent studies have shown that many children with atopic eczema have a mutation in the gene which encodes filaggrin (FLG) which is a key structural protein in the outermost layer of the epidermis. This can lead to a defect in the skin barrier function making the child more susceptible to *irritants* (e.g. soaps, bubble baths and shampoos), *infections* (e.g. *staphylococcus aureus*, herpes simplex, molluscum contagiosum) and *allergens* (house dust mite, animal dander, pollen). A useful clinical marker that is found in some children and their families with the FLG mutation is *palmar hyperlinearity* (i.e. have extra lines on the palmer creases of the hands). In some children with atopic eczema there may also be a defect in the gut barrier function, especially in younger children with more severe atopic eczema, which can lead to allergic reactions to certain foods, such as milk, eggs, nuts, wheat, etc. Children may also be sensitized by the foods entering the system through a defective skin barrier.

### 13.5 Treatment

The management of AE is multi factorial and will require a combination of non-prescription and prescription items to tackle the various underlying factors including itch, dry sensitive skin, infections and allergies.

Because atopic eczema is a chronic disease, the doctor must have strategies for treating acute exacerbations and also for maintenance treatment between exacerbations. Parents need to understand that AE is a chronic condition and they will need to apply treatment depending on the extend of the rash and the severity of the disease at presentation (mild, moderate or severe).

The corner stone of treatment is "*The 3 E's*"

1. Emollients
2. Education of the child and parents on management of AE
3. Elimination of irritants, allergens and itch.

Advice should be given with regards to clothing, feeding, bedroom decor, entertainments like sun bathing and swimming pools. For instance, AD children should avoid sleeping in rooms with many books, rugs or any other dust attracting material (like curtains). Frequent vacuuming is recommended, washing of curtains, leave books out of the room and maintain correct level of humidity. Air allergens may be a trigger for an exacerbation of AD. Avoiding them can be useful for secondary prevention.

### 13.6 Skin Infection

Regardless of the severity of the presentation, all children with atopic eczema should be assessed for signs of clinical infection. Eczema is a dry, itchy skin condition. If the skin is crusty, weepy or sore then it is most likely infected, usually with *Staphylococcus aureus* (Figs. 13.5 and 13.6). *Staphylococcus aureus* is commensal in 10% of children who have no skin problems but is found in up to 90% of children with atopic eczema. If it penetrates intact skin as a result of a defective skin barrier function and or scratching, it can cause clinical infection that can resemble impetigo (impetiginised eczema). Once there is



**Fig. 13.5** Infected atopic eczema



**Fig. 13.6** Infected atopic eczema on the face

clinical infection no treatment will work until the infection is cleared. Keeping eczematous, weepy skin clean is an art: on the one hand, soaps worsen eczema; on the other hand, bacteria need to be removed by washing appropriately. Syndet soaps are great as they tend to protect the skin from further inflammation while eliminating bacteria.

Once infected, the child will usually require oral antibiotics such as flucloxacillin or clarithromycin for at least 7 days. Topical antibiotics such as fusidic acid (“Fucidin®” or “Fucidin HC®”) may also be required for a maximum of 7–14 days.

Even in the absence of clinical infection, *Staphylococcus aureus*, which commonly colonises the skin in children with atopic eczema, can secrete a superantigen, which can lead to release of inflammatory cytokines and worsening of the atopic dermatitis. Strategies to reduce the burden of *Staphylococcus aureus* in the skin such as with “Milton® (Bleach) baths” twice a week (1 ml/liter of bath water) have been recommended, particularly in children with severe atopic eczema or those with recurrent skin infections although there is not a lot of scientific evidence to support the benefits of bleach baths. “Milton Fluid®” is made of an aqueous solution of sodium hypochlorite and 16.5% sodium chloride and it is important to use the correct concentration in the bath.

Washing in a bath without soaps, bubble baths and other irritants may be as effective as bleach baths in removing bacteria from the surface of the skin [2]. See patient advice leaflet. (Chap. 66).

Children with atopic eczema are also susceptible to viral infections including the herpes simplex virus (HSV). Infections with the HSV can cause *eczema herpetiformis* which is a rare but potentially life threatening infection in children with atopic eczema. It is more common in young children with severe atopic eczema, particularly when it affects the face and neck. It presents as a cluster of vesicles or punched out ulcers which may become secondarily infected with crusting and weeping and may look like impetiginized eczema, which fails to respond to topical and oral antibiotics. Children with *eczema herpetiformis* usually require hospital admission and systemic treatment with antivirals.

### 13.7 Moisturisers

The ideal moisturiser should be greasy, cheap, available in large quantities and free from irritants such as sodium lauryl sulphate (SLS). Ointments are safer than creams as they contain fewer preservatives. It should be hypoallergenic, perfume and fragrance free. Greasy ointments are messy and sticky. Children sometimes dislike them but parents should persist as they are far more effective than light, creamy, cosmetically more acceptable moisturisers. Parents should be given a choice of a few moisturisers to try out to see which helps their child’s dry skin the best. A greasy moisturiser will remain on the skin much longer and usually only have to be applied twice a day except on the hands, where they should be applied more often as the moisturiser rub off or are washed off more frequently. A greasy moisturiser should always be rubbed downwards, especially on hairy parts of the body, as rubbing upwards can irritate the hair and cause folliculitis. Sufficient quantities of moisturisers need to be recommended and as a general rule the parents should be encouraged to use ten times more moisturisers than topical steroids or topical calcineurin inhibitors.

A recent Cochrane review showed that moisturisers prolong the time to flare, decrease the number of flares ups and reduce the amounts of topical corticosteroids needed to control eczema. It also showed that topical anti-inflammatory treatment in combination with moisturisers yields better results than without moisturisers [3, 4].

Another recent study assessed the benefits of an emulsion-type moisturiser applied daily during the first 32 weeks of life to 59 of 118 neonates at high risk for AD (based on having a parent or sibling with AD). It showed daily application of moisturiser during the first 32 weeks of life reduces the risk of AD/eczema in infants by 32% [5]. This is a very practical and useful tip for parents of children with atopic eczema to protect their newborn siblings. Emollient therapy in newborns with high risk for AD could be recommended for primary prevention.

Emollient bath additives have not been proven to help but are far safer than bubble baths and other perfumed bath additives. Moisturisers should be applied immediately after a bath to “seal in” the hydration from the bath water. Adding some powered oat into the bathtub can also help reduce itching. Pat-drying is better than rubbing. Short baths are recommended (less than 5 min). Soap substitutes can be used in those areas that require deeper cleaning such as axilla, genitals and feet. (see Chap. 62 on emollients).

### 13.8 Avoiding Irritants

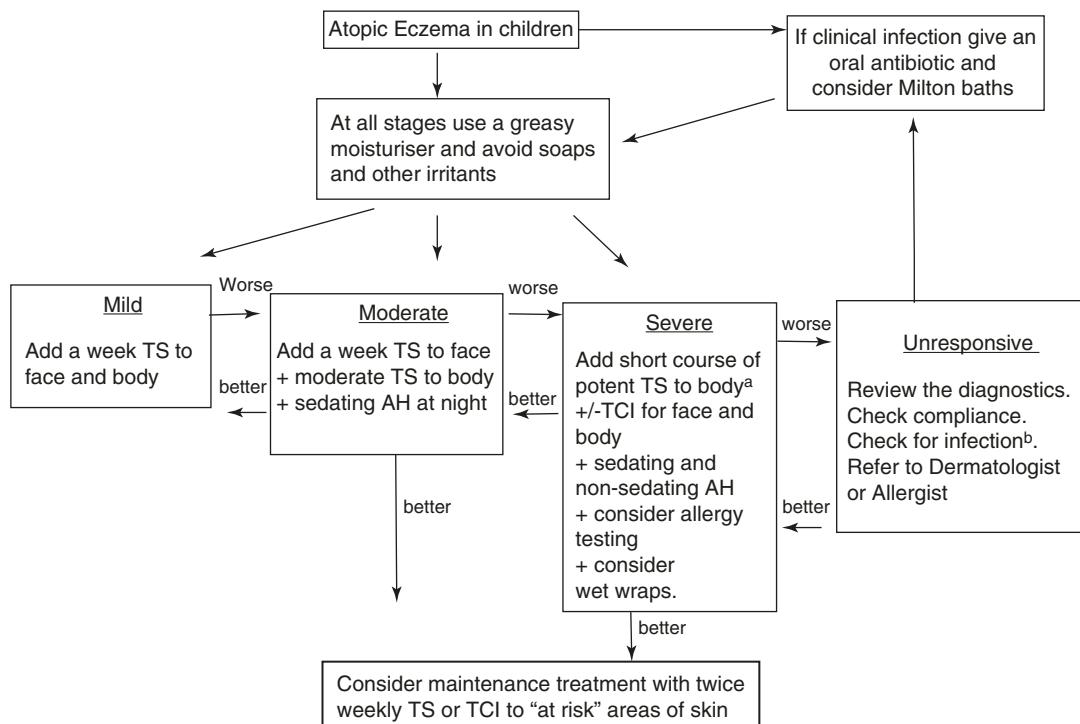
Children with atopic eczema have a defective skin barrier function and are far more susceptible to irritants such as soaps, shampoos, bubble baths, perfumes, fragrances, SLS or preservatives such as paraben. Soaps and shampoos contain

detergents such as SLS, which are designed to break down and remove oil; however, this is a very thing that is in short supply in the skin of children with atopic eczema. Parents should be advised to use suitable soap and shampoo substitutes such as “Elave®” wash, “Elave®” shampoo or “Aveeno®” wash. A cheap, safe alternative is aqueous cream which makes a nice soap substitute. Parents should be warned not to use aqueous cream as a leave-on moisturiser as it contains Sodium Lauryl Sulfate (SLS) which can damage delicate skin if left in contact with the skin for a prolonged period of time.

### 13.9 Stepwise Approach to Atopic Eczema (Tables 13.3 and 13.4)

All patients with atopic eczema, regardless of the severity, need plenty of moisturising and need to

**Table 13.3** Stepwise approach to atopic eczema in children

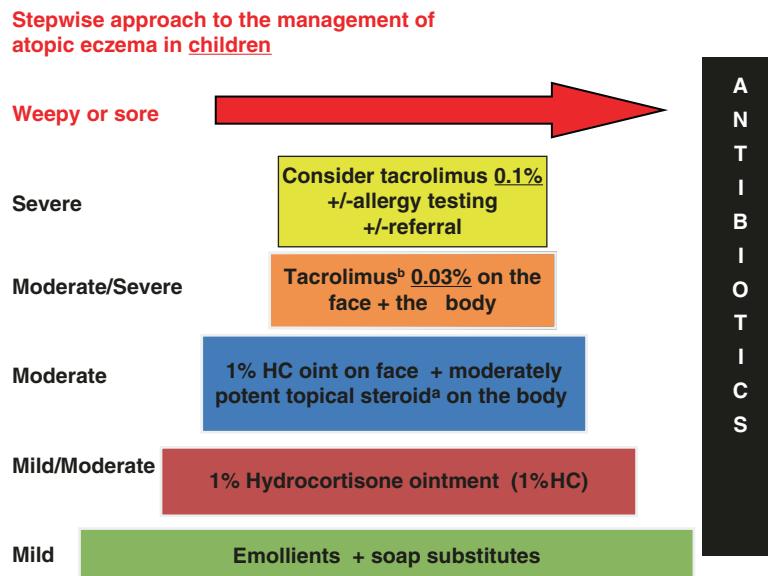


TS Topical steroid, TCI Topical calcineurin inhibitor (e.g. “Protopic”), AH Antihistamine

<sup>a</sup>Avoid using potent TS in children <3 years old

<sup>b</sup>Infection = e.g. staph aureus, herpes, scabies, fungal?

**Table 13.4** Stepwise approach to the management of atopic eczema in children



<sup>a</sup>Moderately potent topical steroid—e.g. = Clobetasone butyrate 0.5% = “Eumovate<sup>®</sup>”, “Ointment<sup>®</sup>”

<sup>b</sup>Tacrolimus = “Protopic 0.03%<sup>®</sup>” = Not licensed for children < 2 years of age. “Protopic 0.1%<sup>®</sup>” not licenced for children <12 years old.

avoid soaps and other irritants. If clinical infection is present it needs to be treated with an oral antibiotic. Chronic non-infected, itchy eczema require more targeted treatment to ease the itch, reduce scratching and strengthen the skin barrier function.

The choice of targeted therapies lies between topical calcineurin inhibitors (TCI) and topical steroids (TS). Targeted treatments should be tailored to the severity of the disease. In severe eczema, it is best to start with the most potent treatment recommended for a particular stage in the disease and then to step down the potency as the symptoms improve (Table 13.5).

when used properly under careful medical supervision, topical steroids are extremely safe and effective in managing atopic eczema in children. TS can be used daily to control an acute attack and can be used twice a week to the areas normally affected by eczema once cleared to prevent a relapse. Once daily applications of TS at bedtime is probably as effective as twice daily dosing.

Topical steroids come in four strengths (see Table 13.5). 1% Hydrocortisone is considered so safe it can be purchased without prescription and can be used on the face in children and adults. It is also effective for mild atopic eczema on the body. Ointment preparations are more effective and safer than cream formulation for dry, itchy eczema. 1% Hydrocortisone is often not strong enough for treatment of more moderate or severity atopic eczema. In this situation it is safe and better to use a moderate potency topical steroid in children on the body (but not on the face). Moderate potency topical steroids are highly unlikely to cause skin atrophy or adrenalin suppression from systemic absorption.

### 13.10 Topical Steroids (TS)

TS are very effective for treating acute exacerbations of atopic eczema where there is intense itch but no infection. Topical steroids have been on the market for over 60 years and have been first line of treatment for years. Parents should be advised (especially steroid phobic's) than

**Table 13.5** Topical steroid potency

Potency	Example	Trade names	Potency ratio
Super potent	Clobetasol propionate	Dermovate®	600
Potent	Betamethasone (as valerate) Betamethaone dipropionate Hydrocortisone butyrate Mometasone furoate	Betnovate® Diprosone® Locoid® Elocon®	100
Moderately potent	Hydrocortisone butyrate Alclometasone dipropionate	Eumovate® Modrasone®	25
Weak	Hydrocortisone 0.1–2.5%	1% Hydrocortisone Diaderm®	1

**Table 13.6** Maximum amount of topical steroids (TS) per month for chronic use<sup>a</sup>

Potency	Age adult	12 years	3 years	Infant < 12 months old
Mild	No max	No max	200 g <sup>c</sup>	100 g <sup>c</sup>
Moderate	200 g	100 g	60 g	30 g
Potent <sup>b</sup>	90 g	30 g	15 g. For acute use only	Avoid
Very potent	30–60 g	Avoid	Avoid	Avoid

(Greater than 2 month's duration) (Higher amounts can be used for a short period of time in acute flare ups)

<sup>a</sup>Adapted from: Position paper on diagnosis and treatment of AE. EADV (2005)19,286–295

<sup>b</sup>Four times this amount can be prescribed if using "Betnovate RD®"

<sup>c</sup>This is for demonstration purposes only. In practice it would be impractical to use this much mild topical steroid in a child. They probably need moderate potent TS rather than large quantity of mildly potent TS

Pharmacists often write "use sparingly" on the tube but this is ambiguous and unhelpful. The finger tip unit is an easy way to explain to parents how much steroid to use. This is the amount of ointment required to spread from the distal interphangeal joint crease to the tip of the nail in an adult's hand. This equates to 0.5 g of ointment and is enough to cover the area equal to 2 adult palms. Table 13.6 shows the safe amount of TS that can be used for chronic use in various age groups. Higher amounts can be used over short periods of time (1 or 2 weeks) for acute exacerbations.

Potent topical steroids should be avoided in children with atopic eczema in general practice; however, under specialist supervision, a short course (1–2 weeks) of a potent topical steroid can be very useful for severe exacerbations, just as a short course of high dose oral steroids is used in children with acute exacerbations of asthma. Unlike the lung, the skin is an easily accessible organ so topical treatments are usually safer and more effective than systemic steroids in atopic eczema.

Potent topical steroids should never be applied to the face and should only be used for short courses in children (1–2 weeks maximum) as prolonged use will lead to skin atrophy and possibly adrenalin suppression as a result of systemic absorption. They should also be avoided in the presence of obvious clinical infection. Very potent steroids should never be used in children (see Chap. 63). Oral steroid for atopic eczema is discouraged and reserved for special circumstances usually under specialist supervision [6].

### 13.11 Topical Calcineurin Inhibitors (TCI)

Topical immune-modulators have been on the market now for almost 15 years. They have the advantage of having the same anti-inflammatory effects as a potent topical steroid without the risks of skin atrophy or adrenalin suppression. They can be safely used on the face and body. In

comparison to topical steroids, they are expensive, not licensed for children under the age of two and can cause a transient irritation and apparent worsening of the eczema in the first week of use in 50% of patients. TCIs should not be used in the presence of obvious skin infection. TCIs are probably not as effective for moderate to severe exacerbations of atopic eczema compared to topical steroids. They may be better and safer for maintenance treatment and to prevent relapse as they appear to help restore the skin barrier function. "Protopic®" (tacrolimus) comes in two sizes (30 g and 60 g) and two strengths (0.03% and 0.1%). "Elidel®" (pimecrolimus) comes in two sizes (30 g and 60 g, sometimes in 100 g) and one strength (1%). They should be applied to the itchy areas, twice a day, for three weeks and then once a day or once or twice a week to prevent relapse. Paediatric dermatologist sometimes use "Protopic®" 0.03% in children under the age of two and may use the 0.1% strength in children over the age of 2 years old if they have severe, unresponsive atopic eczema (this is an off-licence indication), as the risks of side effects with TCIs are less than potent topical steroids in children (see Chap. 64). Patients using TCIs should protect their skin from UVL as there is a theoretical risk that they may predispose to skin cancer. In severe flares, it might be effective to improve the eczema with a topical steroid first and then change to a TCI as TCIs can sometimes cause transient itching and worsening of the eczema in the first week of use in up to 50% of patients.

## 13.12 Clothing

Children with AE should only wear cotton next to the skin and all labels should be removed from the inside of clothing. Cotton gloves or mittens at night might help reduce skin damage from scratching, however children soon learn how to pull them off. An alternative is a baby grow with the hands covered by long sleeves sealed at the

end or "Scratch sleeves™" which are mittens attached to a small cardigan which makes it almost impossible for the child to remove it on their own. If there are localised area that are particularly bad such as the wrists or ankles, bandaging with ichropaste or tubigrip applied to the area may protect it from scratching. Wet wraps can be helpful in children with severe generalised itch once there is no clinical infection. It is now much easier to use "Tubifast Garments®" which are cotton long sleeved vests and leggings. The parents have to be taught how to apply a greasy moisturiser all over at night. Then a moderately potent topical steroid is applied on top of the moisturiser to the badly affected areas. A damp garment is then fitted followed a second dry outer garment. This can be left on overnight and sometimes 24 h and repeated daily for up to 2 weeks. They appear to help via occluding the topical agents for increased penetration, decreasing water loss and providing a physical barrier against scratching.

Silver impregnated cotton clothing and silk clothing are also available and are supposed to reduce skin infections and colonisation by *staph aureus*, although hard evidence is not yet available for this. These garments last a certain number of washes. They can be used later to wear over moisturisers.

## 13.13 Antihistamines

Atopic eczema is not a histamine provoked disease. Therefore, the new non-sedating potent antihistamines (second generation) are rarely effective. There is no evidence that they improve pruritus in AD unless there is severe itching or concurrent urticaria. The older (first generation) sedating antihistamines can be helpful in relieving itch at night time in children with atopic eczema. Their anti-itch benefits are more related to their sedative rather than the antihistamine effects. Severe pruritus should be managed by specialists. (see Chap 50)

### 13.14 Allergies

Restricting the mother's diet during pregnancy does not affect the incidence or severity of AE. Breast feeding may reduce the incidence or severity of AE in children with a strong family history of atopic disease. If these children are bottle fed, using hypoallergic formulas (partially hydrolysed formula) may help [7].

The "Learning Early About Peanut" (LEAP Study), published in the *New England Journal of Medicine*, demonstrate that consumption of a peanut-containing snack by infants who are at high-risk for developing peanut allergy prevents the subsequent development of allergy. Of the children who avoided peanut, 17% developed peanut allergy by the age of 5 years. Remarkably, only 3% of the children who were randomised to eating the peanut snack developed allergy by age 5. Therefore, in high-risk infants, sustained consumption of peanut beginning in the first 11 months of life was highly effective in preventing the development of peanut allergy [8]. Allergists are now beginning to gradually reintroduce foods into the diet of children who have previously been found allergic. This should be done under expert supervision and never for foods that cause anaphylaxis. The early introduction of food with high allergenic potential (hen's egg, fish, milk, peanut) may prevent food allergy. Exclusive breast feeding is suggested for the first 4 months and subsequently different allergenic foods should be introduced one at a time to the diet, as first exposure through mouth mucosa generates tolerance while first exposure through the skin may generate sensitization.

Some children with AE may benefit from a diet low in colourings and preservatives. However, the role of allergy testing and allergen avoidance is controversial. Many children are placed on prolonged restricted diets by their parents or alternative practitioners without any clear evidence that it helps. This can lead to food fadism and an unbalanced diet. In younger children with severe atopic eczema it may be worth con-

sidering doing allergy testing. Allergy testing for *milk, egg, peanut, wheat and soya* should be considered in children aged <5 years, who have moderate-to-severe AD refractory to topical treatment and/or if there is immediate reaction after a specific food ingestion [9].

The three most common tests are blood tests for IgE and RAST (for foods and aeroallergens) a skin prick test and exclusion diets. The most common allergens found include house dust mite, animal dander, pollen and foods including milk, eggs, wheat, soya and nuts. The benefits of anti-house dust mite measures including new mattresses, mattress covers and no carpets are unclear. Exclusion diets should be carried out with the assistance of a dietician who can ensure the child is getting all the essential nutrients while on the exclusion diet. Exclusion of staples such as milk, wheat and eggs should only be for a limited period (e.g. 6–12 weeks) and the food should be reintroduced if there is no significant improvement in the atopic eczema during the exclusion diet. Even in children with proven food allergies such as milk or egg allergy, they often outgrow their allergies as they get older and so the food should be reintroduced gradually, one at a time, once a year, as their atopic eczema begins to improve. Children who develop an anaphylactic reaction to foods such as nuts or shellfish should not have a food challenge unless under specialist supervision in a hospital setting with full resuscitation facilities.

Dermatitis herpetiformis in children may resemble AE so in atypical cases a blood test for coeliac antibodies, a skin biopsy and a jejuna biopsy should be considered.

*Specific allergen immunotherapy* (SIT) is a treatment that may improve disease severity in people with atopic eczema (AE) by inducing immune tolerance to the relevant allergen by gradually exposing the patient to specific allergens such as house dust mite, grass pollen, or other inhalant allergens using various routes including subcutaneous, sublingual, intradermal, and oral drops.

A recent Cochrane review found limited evidence that SIT may be an effective treatment for people with AE, but overall, the quality of the evidence was low [10].

*Patch testing* may be required if there is a suspicion of contact allergic dermatitis to various products that are in contact with the skin including moisturisers and topical steroids but patches may be difficult to apply in a small child (see Chap. 21). A new *atopy patch test* has been developed which tests for a Type 4 (delayed T-lymphocyte hypersensitivity reaction) to common allergens such as house dust mite, grass pollen, cat dander, cow's milk, hen eggs, wheat and soy products. Compared the skin prick tests and IgE and RAST tests (which test for type 1, IgE mediated hypersensitivity reactions), the atopy patch test appears to be more specific but less sensitive [11].

### 13.15 Systemic Treatment

Children with severe unresponsive AE may have to be referred to a consultant dermatologist (Tables 13.7 and 13.8) for phototherapy or systemic treatment such as oral steroids, methotrexinate, cyclosporine or azathioprine.

### 13.16 Newer Biologic Agents for Severe AE

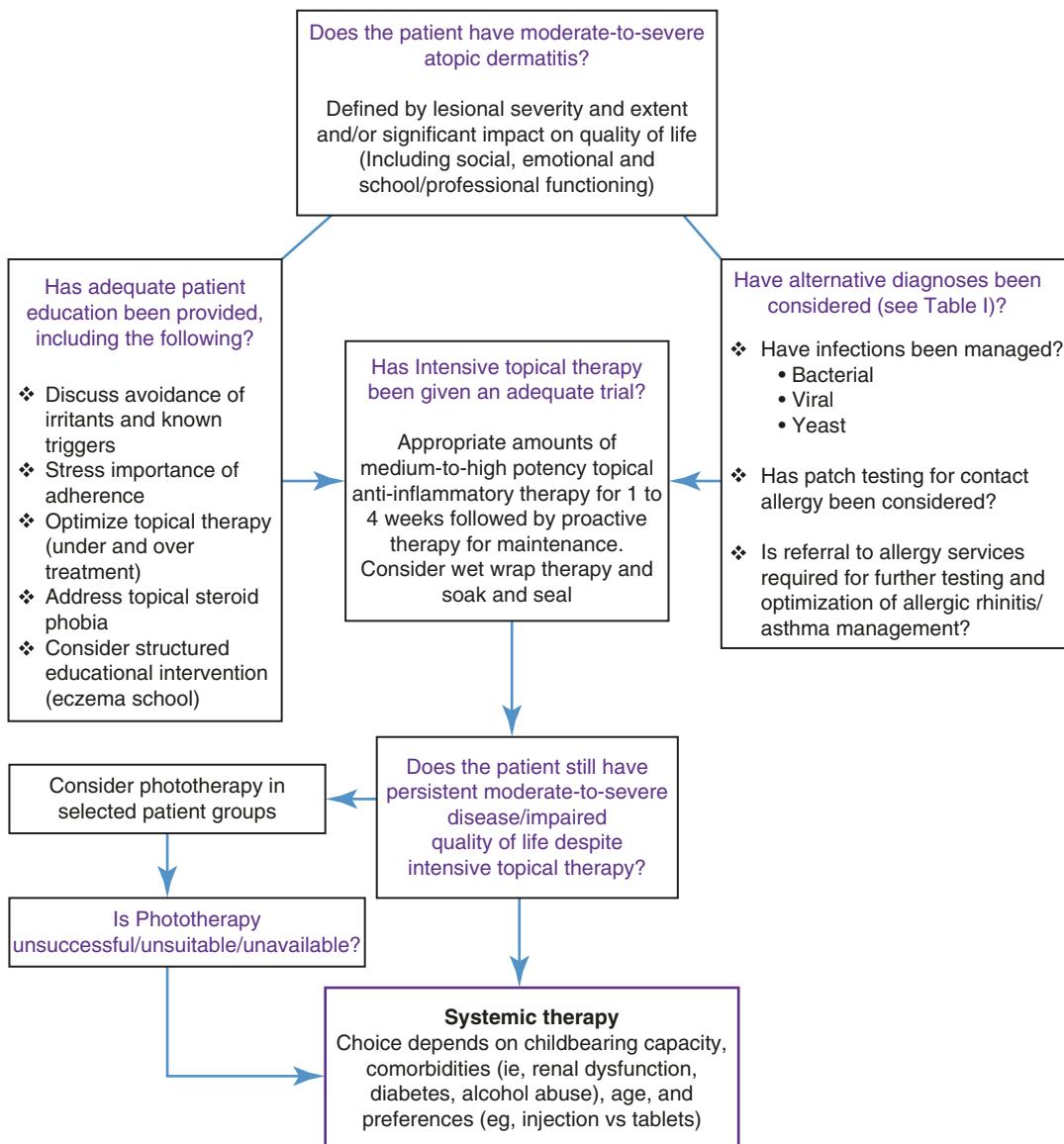
There is some very exciting work being carried out in this area and a number of new biologic agents and monoclonal antibodies will be coming

**Table 13.7** Recommendations for referral for a second medical opinion

- Severe infection when herpes simplex (eczema herpeticum) is suspected.
- The disease is severe and has not responded to appropriate therapy in primary care.
- The rash becomes infected with bacteria and treatment with an oral antibiotic plus a topical corticosteroid has failed.
- The rash is giving rise to severe social or psychological problems.
- The patient or parents are becoming exasperated with the disease or worried about side effects of treatment.
- Treatment requires the use of excessive amounts of potent topical corticosteroids.
- Management in primary care has not controlled the rash satisfactorily.
- The patient or family might benefit from additional advice on application of treatments (bandaging techniques).
- Contact dermatitis is suspected and confirmation requires patch-testing (this is rarely needed).
- Dietary factors are suspected and dietary control a possibility.
- The diagnosis is, or has become, uncertain.

on the market in the next few years to manage itch and inflammation in severe AE. These will act primarily as anti-inflammatory but are much more specific and targeted at how they work compared to some of the old systemic anti-inflammatory such as cyclosporine or methotrexate. It will be more difficult to get a license for these new biologic agents in children and they will probably be allot more expensive than the more conventional systemic anti-inflammatory drugs currently available (see Chap. 14 on eczema and dermatitis in adults).

**Table 13.8** Algorithm to decide when systemic immunomodulatory therapy is warranted in patients with atopic dermatitis



Ref: When does atopic dermatitis warrant systemic therapy? Recommendations from an expert panel of the International Eczema Council. Simpson, Eric L. et al. Journal of the American Academy of Dermatology, 2017 Oct; Volume 77, Issue 4, 623–633

### 13.17 Conclusion

Atopic eczema is a chronic, inflammatory, itchy skin disease that requires ongoing management with plenty of greasy moisturisers, avoidance of soaps and other irritants and targeted treatment with topical steroids or topical calcineurin inhibitors. Infective exacerbations need to be treated with systemic antibiotics and allergy testing with allergen avoidance may be required for more severe cases. While most cases of AE can be managed in general practice, severe, unresponsive cases should be referred to a consultant dermatologist as they may need treatment with phototherapy or systemic treatments with agents such as cyclosporine or azathioprim (Tables 13.7 and 13.8).

### Suggested Readings

[www.atopicskindisease.com](http://www.atopicskindisease.com) is a self-funded membership website for patients and practitioners. This site explains a combined approach to managing atopic eczema, combining optimal conventional topical treatment with the behaviour modification technique and habit reversal to eliminate habitual scratching.

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# Management of Eczema/ Dermatitis in Adults

14

David Buckley

## Key Points

- Dermatitis causes a reduced skin barrier function which leads to dry, itchy, scaly skin and a susceptibility to external aggravating factors such as irritants, allergens and infections.
- The majority of cases of contact dermatitis (80%) are caused by irritant contact dermatitis.
- The definitive treatment of contact dermatitis is the identification and avoidance of the underlying causes (irritants or allergens).
- The clinical and histological features of atopic eczema, contact allergic and irritant dermatitis may be indistinguishable.

## What to Tell the Patient

- The first step in the management of dermatitis is always to moisturise liberally with a safe, greasy, cheap moisturiser.
- Patients with dermatitis should avoid soaps and other irritants such as shampoos, shower gels, bubble baths, washing up liquids and detergents.
- When used under careful medical supervision, topical steroids are extremely safe and effective for treating dermatitis.

## 14.1 Introduction

The terms eczema and dermatitis are synonymous. Eczema is more commonly used when there are constitutional abnormalities (e.g. atopic eczema) whereas dermatitis usually implies some external causative factors (e.g. contact dermatitis or hand dermatitis). In this chapter we will use the term “dermatitis” only.

## 14.2 Clinical Features and Diagnosis

While in children the most common form of dermatitis is atopic dermatitis, in adults there are many other forms of dermatitis (Table 14.1). All share the common problem of reduced skin barrier function which leads to dry, itchy, scaly skin and a susceptibility to external aggravating factors such as irritants, allergens and infections. Scratching and picking further compromises the skin barrier function. Chronic scratching can lead to thick, leathery patches of the skin (lichenification).

It is not unusual to find patients with a current or past medical history of atopic eczema developing irritant and/or allergic contact dermatitis in adult life. Some patients may have features of both allergic and irritant dermatitis particularly when presenting with foot and hand eczema [1].

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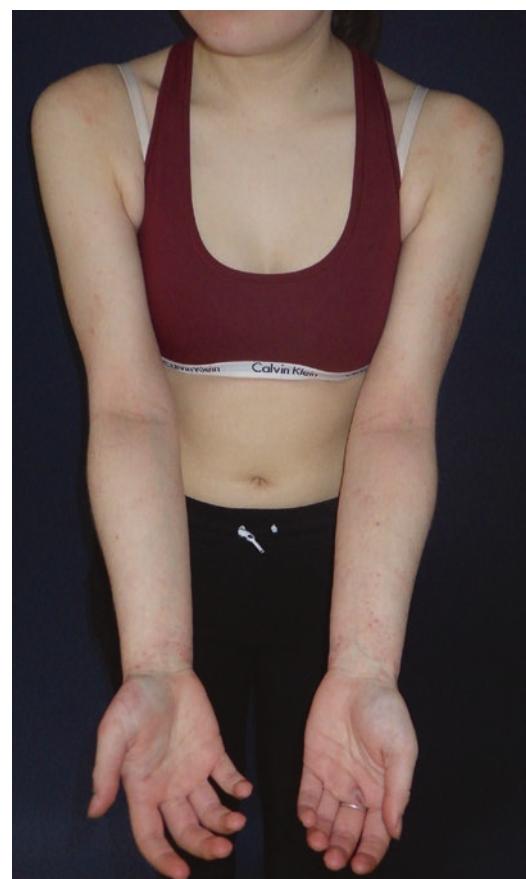
**Table 14.1** Some common types of dermatitis seen in adults

- Atopic dermatitis
- Irritant contact dermatitis
- Allergic contact dermatitis
- Asteatotic dermatitis (also called eczema craquele)
- Nummular dermatitis (also called discoid eczema)
- Seborrhoeic dermatitis
- Varicose eczema
- Otitis externa
- Hand dermatitis
- Pomphylix dermatitis
- Neurodermatitis (also called lichen simplex chronicus)



**Fig. 14.1** Contact irritant hand dermatitis

The majority of cases of contact dermatitis (80%) are caused by irritant contact dermatitis (Fig. 14.1) [2]. Contact dermatitis accounts for 70–90% of all occupational skin diseases [3]. High risk occupations include hairdressing, florists, beauticians, cooks, metal workers and dental assistants. Homemakers, kitchen workers, plasterers, health care workers and dairy farmers are also prone to irritant dermatitis. Atopic eczema (Fig. 14.2) and contact dermatitis are diagnosed clinically. Histology is usu-



**Fig. 14.2** Atopic eczema in a 17-year-old patient

ally nonspecific and will not always help distinguish between the various forms of dermatitis.

The clinical features of atopic eczema, contact allergic and irritant dermatitis may be indistinguishable and more detailed allergy testing may be required in severe resistant cases (see allergy testing below) (See Table 14.2).

Allergy testing such as skin prick testing, IgE and RAST testing, exclusion diets and skin patch testing (Table 14.3) may be necessary for more severe, refractory cases but should only be carried out by doctors who have experience in conducting these tests and interpreting the results (see Chap. 21 on allergic skin disorders). Food allergies are far less common in adults than in children. Patients who need to stay on a restricted diet should be seen by a dietician to ensure the diet is balanced and nutritious. Restricted diets should be reviewed annually.

Severe, resistant or frequently relapsing cases of dermatitis or cases requiring an allergy work up should be referred to a skin specialist for further evaluation (see Chap. 21 on Allergic Skin Disorders).

### 14.3 Differential Diagnosis

Atopic dermatitis can be confused with many other forms of eczema (Tables 14.1 and 14.2) and skin disease. Psoriasis, seborrheic dermatitis, allergic contact and irritant dermatitis, nummular eczema (also called discoid eczema) (Figs. 14.3 and 14.4), neurotic excoriation and dermatitis herpetiformis can share similar clinical presentations. Patients with scabies can develop widespread, itchy, eczematous inflammation. Tinea infection, especially when the

classical clinical features are altered with the inappropriate use of topical steroids (*tinea incognito*) may resemble dermatitis. Cutaneous T-cell lymphoma such as mycosis fungoides can resemble atopic dermatitis but has characteristic histological features. Think of this possibility in patients in their fifth or sixth decade, with generalised relapsing dermatitis. Subacute and discoid lupus erythematosus may

**Table 14.3** Indications of skin patch testing include

- Acute or chronic dermatitis if a contact allergy is suspected
- Chronic eczema failing to respond to treatment
- Hand, foot face or eyelid dermatitis
- Chronic stasis (varicose) dermatitis
- Chronic or recurrent otitis externa
- Dermatitis in individuals involved in high risk occupations for contact dermatitis

**Table 14.2** How to distinguish the various forms of Eczema-Dermatitis

	Atopic eczema (AE) <sup>a</sup>	Allergic Contact Dermatitis (ACD) <sup>a</sup>	Irritant Contact Dermatitis (ICD) <sup>a</sup>
Age	– 20% children – 10% adults	Mostly adults	Mostly adults 80% of CD-irritant
Aetiology	Atopic disease, strong hereditary factors. Filaggrin gene defect → skin barrier defect	Type IV delayed hypersensitivity reaction to an allergen in contact with the skin	Not immunologically mediated. Irritation for detergents and other harsh chemicals
Onset after exposure	Not relevant	8–96 h	Minutes to hours
Distribution	Mainly flexural	At site of contact (e.g.: Earlobe, face, hands) may become more generalised	Mostly hands
Clues to diagnosis	Personal or family history atopy. Itchy +++	Reaction to cheap earrings (nickel). Itchy++. May blister. Relapses within 24–48 h after returning to work after holidays.	Work space involvement. High risk occupation: e.g. hairdressing, kitchen worker, homemaker, healthcare worker, cleaner. Slow to relapse after holidays
Diagnosis	Clinical. IgE + RAST Skin patch test. Atopy patch test	Patch test	Clinical
Treatment	Emollients (E), Soap substitutes (SS), Topical steroids (TS), Topical Immunomodulators (TIM)	Avoidance of allergens E, SS, TS, TIM	Avoidance of irritants and barriers (gloves) E, SS, TS, TIM

Ref: adapted from BMJ 2016;353:i3299

<sup>a</sup>There can be considerable overlap

also resemble dermatitis and these conditions are usually diagnosed histologically.

Patients with hand dermatitis should always have their feet examined as occasionally tinea pedis (athlete's foot) can provoke an “**id reaction**” (also called autosensitisation dermatitis or interface dermatitis) on the hands. This is an itchy rash with small vesicles similar to allergic dermatitis or pompholyx in the hands and is

caused as response to the primary infection (Fig. 14.5a, b). Treating the tinea pedis may clear the hand dermatitis. Contact allergic dermatitis from a leg ulcer dressings or inflammation from an infected leg ulcer may also cause a generalised eczematous reaction (disseminated secondary eczema). In general, a severe inflammatory process may cause a distant eczematous lesion.



**Fig. 14.3** Discoid eczema



**Fig. 14.4** Discoid eczema



**Fig. 14.5** (a) and (b). Pompholyx on the hands



#### 14.4 Pathophysiology

As in children, the pathophysiology of atopic eczema in adults remains incompletely understood with a complex interaction between genetic predisposition and environmental triggers. There is often a personal or family history of other IgE related atopic diseases such as asthma, allergic rhinitis or allergic conjunctivitis. Patients with a per-

sonal or family history of atopic eczema are more likely to suffer from allergic or irritant contact dermatitis as a result of a defective skin barrier.

The reaction to the various substances with which patients come in contact may be immunologically mediated (**allergic contact dermatitis**) (Fig. 14.6a, b, c) or merely due to a patient's sensitive skin reacting adversely to irritating substances such as soaps, detergents, or industrial



**Fig. 14.6** (a) Contact allergic dermatitis to cutting oils in the workplace. (b) Sofa dermatitis from contact allergic dermatitis to dimethyl fumarate in a cheap leather couch. (c) Allergic reaction to benzoyl peroxide



**Fig. 14.7** Neurodermatitis on the skin

oils (**irritant contact dermatitis**). Some patients may have features of both allergic and irritant contact dermatitis (Table 14.2).

Chronic scratching in one area of the skin with no obvious underlying cause can lead to **neurodermatitis (lichen simplex chronicus)**. It starts with an itch which leads to scratching which in turn causes more skin irritation and more itch. Eventually the patient gets into an “itch-scratch cycle” causing a patch of dry, thickened, scaly skin which is common in certain areas of the body such as the shins, the elbows, wrists and the back to the neck (Fig. 14.7). Treatment is by blocking the itch and stopping the scratching using emollients, potent topical steroids and sometimes by protecting the area with bandaging.

## 14.5 Treatment

The definitive treatment of contact dermatitis is the identification and avoidance of the underlying causes (irritants or allergens). Treatment can vary according to whether the eczema/dermatitis is

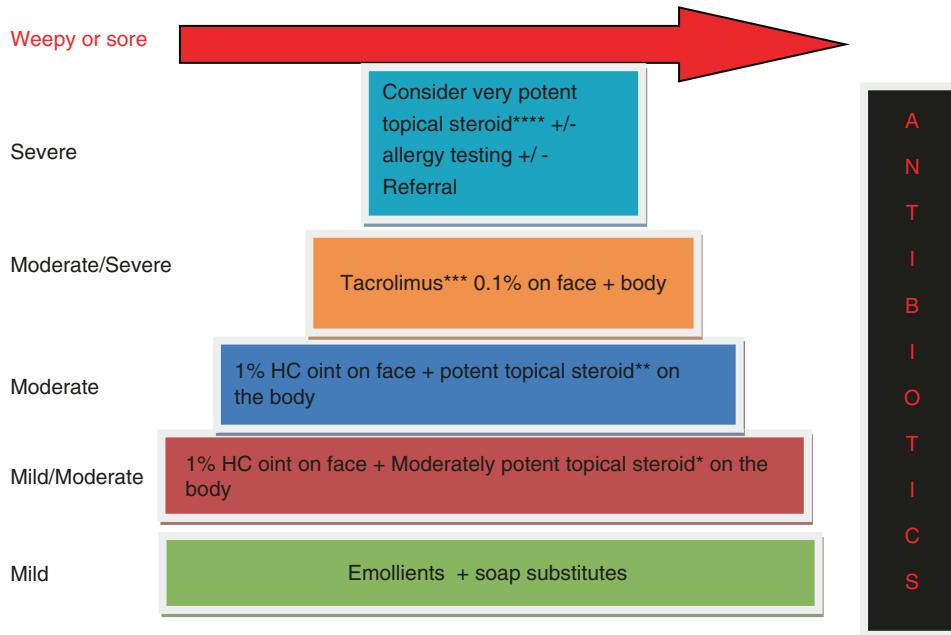
generalised or localised (see Chap. 36 on regional dermatology for localised eczema/dermatitis management).

The stepwise approach to the management of dermatitis in adults (Fig. 14.8) is similar to that in children (See Chap. 13—Atopic eczema in children). In adults, potent topical steroids like betamethasone (“Betnovate®”) and sometimes very potent topical steroids like clobetasol propionate (“Dermovate®”) can be used on the body (but not on the face).

## 14.6 Emollients

Topical steroids will do nothing for dry skin. The first step in the management of dermatitis is always to moisturise liberally with a safe, greasy, cheap moisturiser. The best moisturiser is the greasiest one that the patient is willing to use. Dermatitis sufferers should only use hypoallergic, oil based moisturisers that come in sufficiently large quantities and are relatively inexpensive as most of these moisturisers will have to be used in high quantities over a long period of time (sometimes up to 500 g per week for an adult). Ointments are the best on dry skin, next creams and lastly lotions which do not offer much as they evaporate quickly off the skin.

Emulsifying ointment is cheap, safe, effective and usually available on national health services. Some patients may not like it as they find it too thick or greasy. It is important to give patients a choice of different moisturisers, a greasy one such as Emulsifying ointment, Paraffin gel or “Epaderm®” ointment to be used at home and at night and a less greasy one such as “Aveeno Dermexa®” or “Epaderm®” cream to be used during the day and at work. Greasy moisturisers should be rubbed downwards, especially on hairy areas on the body, as rubbing up and down will irritate the skin and may cause folliculitis. Urea based moisturisers (eg, “Calmurid®” or “Eucerin®”) are less greasy, but can sting, if the skin is cracked or fissured (see Chap. 62 on emollients). It is helpful to have a few different types of emollients in the office to show the patient what they look like and their consistency.



**Fig. 14.8** Stepwise approach to the management of dermatitis/eczema in adults. \*Moderately potent topical steroid—e.g. = Clobetasone butyrate 0.5% (= “Eumovate®” ointment). \*\*Potent Topical Steroid—eg Betamethasone

valerate 0.1% (= “Betnovate®”). \*\*\*Tacrolimus (= “Protopic®”). \*\*\*\*Very potent topical steroids e.g. = Clobetasol Propionate 0.05% (= “Dermovate®”)

## 14.7 Soap Substitutes

Patients with dermatitis should avoid soaps and other irritants such as shampoos, shower gels, bubble baths, washing up liquids and detergents. Safe soap free washes, such as “Elave®” wash, Aveeno wash and “Elave®” Shampoo can be used instead. Bath oils such Oilatum® bath oil or Aveeno® bath treatment can be added to a lukewarm bath (27–32 °C) but the patient should be instructed not to spend more than 5 min in the bath. Rubbing in a greasy moisturiser should be done immediately after patting the skin dry once out of the bath: this will help seal in the bath moisturiser.

Adding an anti-bacterial agent to the bath, such as “Milton®” (sodium hypochlorite), can reduce bacterial load on the skin in patients with infected dermatitis and reduce exacerbations (see Chap. 66). People whose job involves getting

their hands wet a lot should wear gloves for all wet work, not only when their hands are very irritated but also as they improve, to prevent relapse. It is advisable to give patients with hand dermatitis written instructions on how to moisturise and avoid irritants since most patients will remember very little of what they are advised by the time they get home (See Hand Dermatitis patient information leaflet, Chap. 66).

Aqueous Cream and Silcocks Base are cheap and usually available on national health schemes but they are not suitable as moisturisers as they are not greasy enough and both contain sodium lauryl sulphate (SLS) which is a surfactant used in many cleansing products and detergents. Aqueous Cream and Silcocks Base and syndet may be used as cheap, safe soap substitutes.

Some patients can reduce itching by spraying on colloidal oatmeal 1%.

## 14.8 Topical Steroids

Topical steroids are the most effective way to alleviate itch and scratching. When used under careful medical supervision, they are extremely safe. Once daily topical steroids are usually sufficient and convenient for the patient. Start with a potent topical steroid and work down to a moderate potency topical steroid or reduce the frequency of application of the potent topical steroid as the dermatitis improves. This should be applied to the itchy areas at night and ointment based topical steroids are generally safer and more effective than creams. Emollients can be applied all over the affected areas 15 minutes after the topical steroid ointment has been applied to the irritated areas. An adult may use up to 100 g of a potent topical steroids per month if necessary.

The fingertip unit (FTU) is a useful way of explaining to a patient how much topical steroid to apply. A FTU is the amount of ointment expressed from a tube with a 5 mm nozzle and measured from the distal skin crease to the tip of the index finger. This equates to approximately 0.5 g of the ointment and is an adequate amount to cover an area equivalent to two adult palms.

It is rare have to use a very potent topical steroid in dermatitis in adults and if they are required they should usually only be for the first few weeks, until the eczema comes under control. It is important to reduce to a less potent topical steroid as the dermatitis improves (see Chap. 63 on topical steroids). “Haelan tape®”, which contains a moderately potent topical steroid impregnated into the tape, can be useful for fissures on the feet or hands. Wet wraps, as described in Chap. 13 for children, can also be used in adults and special wet wrap garments are available in adult sizes.

As the skin is such an accessible organ for topical therapies, it is rare to have to resort to oral steroids for the management of dermatitis in general practice (see Chap. 63).

## 14.9 Anti-histamines

Histamine is not primarily involved in the pathophysiology of dermatitis and so non-sedating antihistamines do not usually help. The older

sedating oral antihistamines may help at night to relieve itch by virtue of their sedating effect (see Chap. 50 on pruritis).

## 14.10 Topical Calcineurin Inhibitors

A topical calcineurin inhibitor such as tacrolimus (“Protopic® 0.1%”) is very useful for more severe eczema on delicate skin such as on the face, the axilla, the groin or the perianal skin, especially when the dermatitis in these areas is not responding to weak or moderately potent topical steroids (Fig. 14.9). Tacrolimus is the treatment of first choice for eyelid dermatitis (Fig. 14.10).

Tacrolimus is a large molecule and will not penetrate thick skin such as the soles of the feet or the palms of the hands (see Chap. 64 topical calcineurin inhibitors). It may cause a transient stinging or burning of the skin for the first few days of application. It often takes a week for benefits to be seen. If used on exposed skin, such as the face or hands, then a high factor, total UV block should also be used. “Protopic®” recently got a licence for maintenance treatment to prevent flare ups and to prolong flare free intervals by applying it twice weekly to the commonly affected areas but treatment should be reviewed after 12 months.



**Fig. 14.9** Eczema around the eyes and upper lip



**Fig. 14.10** Contact allergic dermatitis possibly to nickel



**Fig. 14.11** Pompholyx hands and feet. Potassium permanganate soaks can help dry up the rash

## 14.11 Antibacterials

Dermatitis is usually dry and itchy. Moderately to severe presentations might weep (skin oozing) and become sore suggesting it has become infected. If there are clinical signs of infection (weepy, crusty and painful instead of dry and itchy) it is important to give the patient a course of oral antibiotics, (usually flucloxicillin 500 mg, three times a day or erythromycin if they are penicillin allergic) for seven to fourteen days. Topical antibiotics (eg “Fucidin®”, “Fucidin H®” or “Fucibet®”) may be used for less severe infections but should only be used for a maximum of fourteen days and should not be repeated for a further three months, otherwise resistance will develop rapidly. **Potassium permanganate** soaks can be helpful for weepy infected hand or foot eczema (Fig. 14.11). “Permitabs® 400 mg tablets” are the most convenient way to make up the solution. One tablet diluted in 4 liters of water makes up a 1:10,000 solution (light pink) which is suitable for soaking hands, feet or legs for 10–15 min. Patients should be warned that the tablets are poisonous and should not be swallowed. Gloves should be worn when handling the tablets as they are corrosive. The solution should be made up fresh and it will stain the skin and nails a dark purple for a few weeks after use. A 1:1000 solution may be used as a wet soak on a piece of gauze that can be held against the skin for 20–30 min-

utes. A 1% solution (1 in 100) is used to treat fungal infections such as athlete’s foot. Some patients may be sensitive to PPM soaks and develop redness or itchiness in the areas soaked. If this happens the solution should be washed off immediately and not used in the future. In mild oozing, chamomile compress can be helpful.

## 14.12 Habit Reversal

Habit reversal (see [www.atopicskindisease.com](http://www.atopicskindisease.com)) is a self-funded membership website for patients and practitioners. This site explains a combined approach to managing atopic eczema, combining optimal conventional topical treatment with the behaviour modification technique and habit reversal to eliminate habitual scratching.

## 14.13 Systemic Treatments

Severe resistant cases of atopic eczema in adults may require phototherapy or systemic anti-inflammatory such as oral steroids, cyclosporin, methotrexate, azathioprine or systemic retinoids such as acitretin.

Alitretinoin (“Toctino®”) is an oral retinoid similar to “Roaccutane®” which is licensed for the treatment of chronic refractory hand eczema. It is not available in all countries. It should not be used in pregnancy, with hyperlipidaemia, uncontrolled hypothyroidism and hypervitaminosis A. The side effect profile is similar to

“Roaccutane®”. It is usually given for up to 6 months and the treatment can be repeated if the hand eczema recurs.

Dupilumab (“Dupixnet®”) is a new human monoclonal antibody that specifically inhibits IL-4 and IL-13 which are believed to be major drivers in the persistent underlying inflammation in AE. It constitutes one of the biggest therapeutic promises in the AD management and is now licensed to treat moderate to severe, resistant atopic eczema in adults in the US and Europe [2]. It is given by self-administered, prefilled subcutaneous injections every 2 weeks after an initial loading dose. The most common side effects are injection site reactions, eye and eyelid inflammation, allergic reactions and herpes simplex infections of the mouth or lips. It should not be used in pregnancy or when breast feeding and is not yet licensed in people less than 18 years old.

A number of other new biologic agents and monoclonal antibodies will be coming on the market in the next few years to manage itch and inflammation in severe AE. These are much more specific at how they work compared to some of the old systemic anti-inflammatories. These new biologic agents will be considerably more expensive than the more conventional systemic anti-inflammatory drugs currently available [4].

## 14.14 Conclusion

Dermatitis in adults can have many causes. Most forms of adult dermatitis will respond to moisturising, avoiding soaps and other irritants and topical treatment with a steroid or calcinurin inhibitor to relieve inflammation, itch and scratching. More resistant cases may need special investigations such as swabs and skin scrapings to look for underlying infections, skin biopsy to rule out other skin disease and allergy testing looking for allergens which may be causing or aggravating the dermatitis.

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# Management of Psoriasis in Primary Care

15

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## Key Points

- The exact cause of psoriasis is currently unknown. A combination of genetic, autoimmune, and environmental factors are likely to be involved.
- Psoriasis is diagnosed clinically as there is no definitive blood test; although histological features can be similar to other skin conditions, it has histological features that may confirm a diagnosis when in doubt.
- Psoriatic arthritis occurs in 15–25% of patients with psoriasis.
- Patients with psoriasis have a higher incidence of obesity, hypertension, hypercholesterolemia, diabetes, heart disease, depression, and the metabolic syndrome. They should be screened for these conditions.
- Although there is no “cure” for psoriasis there are many safe, effective treatments available.
- Regular application of a greasy moisturiser and avoiding soaps will help improve the appearance of the rash and will reduce scaling and itch.
- The first line treatment for adults with chronic, stable, plaque psoriasis on the body is usually a combination of calcipotriol (a Vitamin D analogue) and betamethasone (a potent topical steroid).
- Sunlight helps the majority of psoriasis patients but they must avoid burning. Sunbeds should be avoided. Alcohol in excess will usually make it worse.

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## 15.1 Introduction

### What to Tell the Patient

- Psoriasis is not infectious, contagious or cancerous.
- It is usually considered genetic in origin. In up to 50% of cases another family member will also have psoriasis. It might skip generations. Some people may have the gene but never get the rash.

Psoriasis is a chronic, relapsing, scaly, often itchy, immune-mediated, inflammatory skin condition which is usually easy to diagnose but can be difficult to manage. It affects approximately 2–3% of the population. It can occur at any age including in childhood but peak incidence is between 15 and 25 years, with a second, smaller peak between 50 and 60 years. It is equally common in males and females.

While most mild to moderate cases can be managed in general practice, patients with more resistant, severe or extensive psoriasis may have

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to be referred to a dermatologist for ultraviolet light, oral treatment or systemic therapy.

## 15.2 Clinical Features and Diagnosis

Psoriasis is diagnosed clinically as there is no definitive blood test and the histological features can be similar to other skin conditions. Psoriasis is considered mild in 60% of patients, moderate in 30% and severe in 10%. There are many different types of psoriasis (Table 15.1), the most common being **chronic plaque psoriasis** (Fig. 15.1). This causes well defined, red plaques that are covered with silvery scale. Plaques are distributed over characteristic body sites such as the extensor surfaces of the knees and elbows, the lower back and the scalp (Figs. 15.2 and 15.3). It can occur on any part of the body and itch may occur but is usually mild, unlike atopic eczema where the itch is severe. Nail changes are common and can be used as a clue to the diagnosis in atypical cases.

The most common **psoriatic nail** changes are onycholysis (lifting of the nail from the nail bed with subungual debris under the distal end of the nails) (Fig. 15.4), thickening and nail pitting (small dints on the surface of the nail plate as if they were scored with a pin) (Fig. 15.5). These occur in up to 50% of patients with psoriasis. Pitting can also be present in eczema and alopecia areata. Psoriatic nail changes can occasionally occur in isolation and it is sometimes necessary to send nail clippings to the lab for fungal stain and culture to distinguish psoriatic nails from tinea unguis.

**Table 15.1** Types of psoriasis

Chronic plaque psoriasis
Small plaque psoriasis
Guttate psoriasis
Nail psoriasis
Flexural psoriasis
Palmoplantar, pustular psoriasis (also known as Palmoplantar pustulosis)
Generalised pustular psoriasis
Erythrodermic psoriasis
Psoriatic arthritis



**Fig. 15.1** Chronic plaque psoriasis in a 44-year-old male



**Fig. 15.2** Chronic plaque psoriasis

**Flexural psoriasis** (also called inverse psoriasis) can occur in isolation or in combination with other types of psoriasis. When psoriasis occurs in the flexures it usually has little or no scale because of friction and moisture present in the flexures



**Fig. 15.3** Chronic plaque psoriasis



**Fig. 15.6** Flexural psoriasis



**Fig. 15.4** Onycholysis in psoriasis



**Fig. 15.5** Nail pitting in psoriasis

(axilla, groin, peri-anal area, between the buttocks, under the breasts and on the lower abdomen under folds of fat in obese patients). The rash in flexural psoriasis is usually deeply red and very well defined, with a sharp cut off between the involved and uninvolved skin (Fig. 15.6). This helps distinguish it from other scaly flexural rashes such as tinea, candidiasis or intertrigo. Skin scraping for fungal stain and culture and a



**Fig. 15.7** Guttate psoriasis

skin biopsy is sometimes required to make an accurate diagnosis of flexural psoriasis.

**Guttate psoriasis** is a usual presentation in younger patients (teens or early 20s) and presents with multiple small plaques of scaly skin scattered symmetrically throughout the body, usually sparing the face, scalp, flexures and nails (Fig. 15.7). Guttate psoriasis often occurs after a streptococcal sore throat but also may present for no apparent reason. Most cases will clear spontaneously within 6–12 weeks. Some patients may develop more attacks and approximately one



**Fig. 15.8** Small plaque psoriasis

third of patients may go on to develop chronic plaque psoriasis.

**Small plaque psoriasis**, as the name implies, is made up of multiple, small plaques of red, scaly, skin which are well defined and scattered symmetrically throughout the body (Fig. 15.8). It can look similar to guttate psoriasis but, unlike it, it does not clear spontaneously in 6–12 weeks.

**Palmar plantar pustular psoriasis (PPP)** is confined to the palms of the hands and/or the soles of the feet. It is now more commonly referred to as **palmoplantar pustulosis (PPP)** and may be a different disease than psoriasis. It is associated with psoriasis elsewhere in only 10–25% of cases. It mainly affects women in their 60s and 70s. It causes a red, well defined scaly rash with a sharp cut off between the involved and uninvolved skin. There are usually multiple small sterile pustules that are as a result of inflammation rather than infection (Fig. 15.9). This is one of the few skin conditions that is found more commonly in smokers. Stopping smoking may help clear the rash. It can rarely be associated with certain autoimmune diseases such as gluten sensitive enteropathy (celiac disease), thyroid disease and type 1 diabetes.

**Generalised pustular psoriasis and erythrodermic psoriasis** cause an extensive rash covering most of the body (Fig. 15.10). The patient is usually systemically unwell, with fever and flu-like symptoms. These conditions are considered



**Fig. 15.9** Palmar planter pustulosis in a 32 year old smoker



**Fig. 15.10** Psoriasis in a patient on beta blockers

a medical emergency and usually require hospital admission for treatment.

**Psoriatic arthritis (PsA)** is present in 15–25% of patients with psoriasis. The arthritis may start before (in 15% of PsA), during or after the skin manifestations appear. It usually starts in the fourth and fifth decades of life. Males and females are affected equally. PsA causes a sero-negative, inflammatory arthritis and if left untreated may result in joint destruction in a similar fashion to rheumatoid arthritis (RA). It may affect only one joint or a number of joints. In the hands, the distal interphalangeal joints are more commonly involved followed by the feet, knees and low back (spondylitis).

Some patients can develop a sausage shaped finger or toe (dactylitis) and psoriatic nail changes on the affected finger. This distribution and X-ray often helps distinguish PsA from RA and other forms of inflammatory arthritis. Male patients, those who are overweight, those with HLA-B-27 antigen and patients with polyarticular disease do

less well. Apart from arthritis and spondylitis, PsA can cause fatigue and be associated with inflammation in other organs, such as the eyes and lungs. Treatment of more severe cases will require disease modifying drugs such as methotrexate or the newer biological agents.

### 15.3 Differential Diagnosis

Chronic plaque psoriasis is usually fairly obvious from the classical clinical features of red, scaly, well defined plaques in the typical psoriasis distribution. Atypical cases or partially treated disease

may be more difficult to diagnose and psoriasis can be confused with seborrhoeic dermatitis, a fungal infection (tinea curis), contact allergic or irritant dermatitis, discoid eczema, pityriasis rosea, secondary syphilis or rare conditions such as the rash of HIV or mycosis fungoides.

### 15.4 Pathophysiology

The exact cause of psoriasis is currently unknown. A combination of genetic, autoimmune, and environmental factors are likely to be involved (Fig. 15.11). Thirty to 50% of patients with psoriasis have a family history of psoriasis.

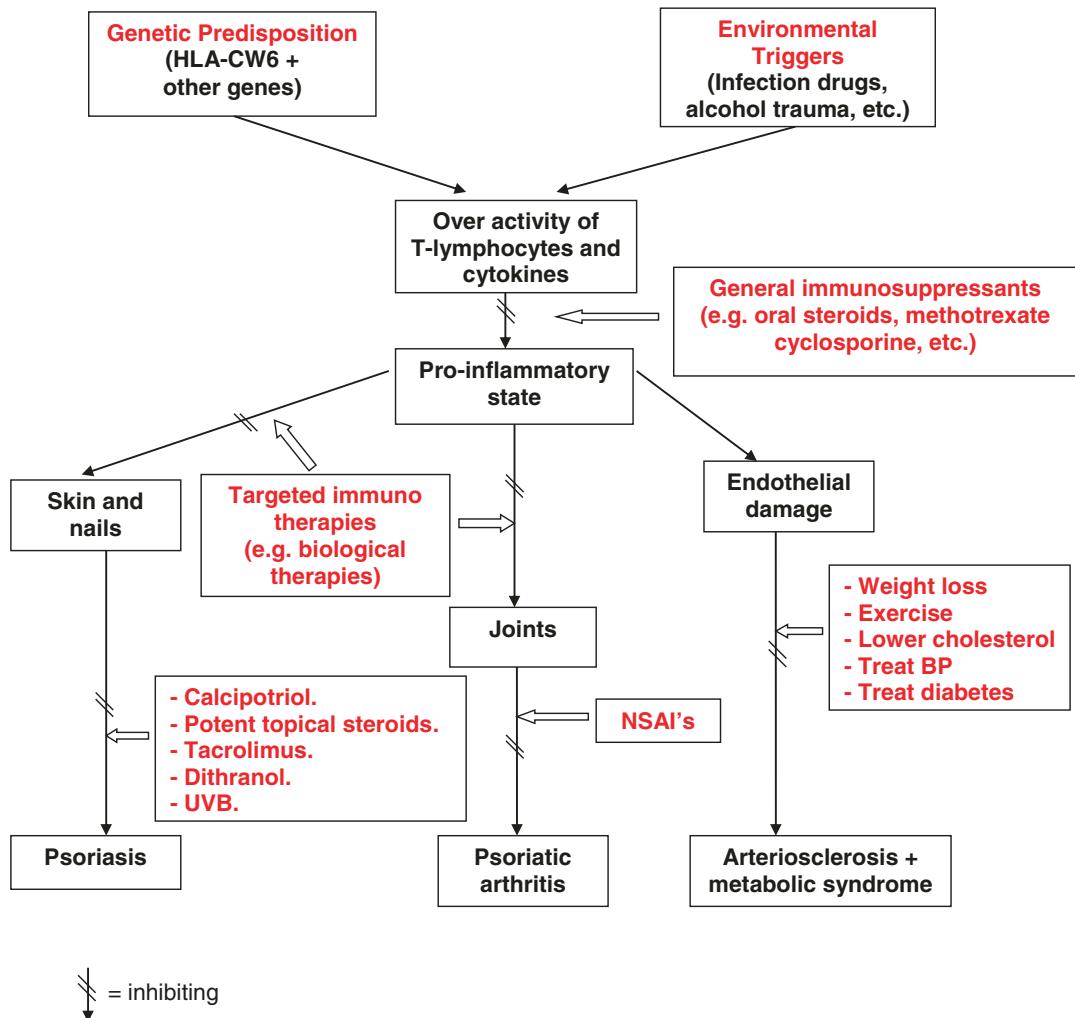


Fig. 15.11 Aetiology of Psoriasis

riasis will have a first degree relative with psoriasis. The risk of developing psoriasis is 20% for those with one parent with psoriasis and 75% if both parents have psoriasis.

Psoriasis is perpetrated by the body's own immune cells, particularly the T-lymphocytes and cytokines. For whatever reason, these cells, which are designed to defend the body from external noxious agents, attack healthy tissue and cause inflammatory changes in the skin, nails or joints in genetically predisposed patients. Our understanding of the molecular dynamics that drives psoriasis is still evolving, with a whole raft of cytokines messengers implicated in the immune dysfunction, especially the tumour necrosis factor alpha (TNFa) and some interleukins (IL-23 and IL-17) and cytokynes. Several of these cytokines have become targets for some of the newer novel biological therapies for psoriasis and PsA.

Overactive T-lymphocytes trigger an immune response that causes dilation of blood vessels and an increased production of healthy skin cells. This triggers an ongoing cycle in which new skin cells move to the outermost layer of skin too quickly—in days rather than weeks. Dead skin can not slough off quickly enough and build up in thick, scaly patches on the skin's surface. This rapid turnover of cells usually does not stop unless treatment interrupts the cycle.

There can be various environmental triggers including infection (*Streptococcal* throat infection or *pytirosorum* in the skin), smoking, alcohol in excess, stress, trauma, and acute withdrawal of potent topical or systemic steroids. Psoriasis is not caused by food allergy. Rare cases have been linked with coeliac diseases and it may be worth considering a gluten free diet in patients with positive coeliac antibodies [1]. If vitamin D levels are low it might be worth considering vitamin D supplements provided the patient is not also being prescribed topical calcipotriol (a vitamin D analogue that is found in “Dovonex®”, “Dovobet®”, “Enstilar®”). A healthy diet, rich in oily fish, green leafy vegetables, carrots, tomatoes and fresh fruit may help [2]. Recent studies have suggested that a Mediterranean diet may help psoriasis [3].

Certain prescription medication can precipitate or aggravate psoriasis or cause a psoriasisiform (psoriasis like) eruption and these may have to be stopped or substituted if the psoriasis proves difficult to control [4] (Table 15.2). Alcohol in excess is also a common cause of psoriasis flare-ups. Patients should be encouraged to avoid alcohol or keep it to an absolute minimum (less than 14 units a week). Sunlight can help most (90%) of patients with psoriasis provided they do not get sunburn which may worsen psoriasis as a result of the **Köbner phenomenon**. This response, first described by Heinrich Koebner in 1876 is also called the isomorphic response, or koebnerization. It refers to the formation of psoriatic lesions in uninvolved skin of psoriatic patients after cutaneous trauma. This might explain why psoriasis is so common on the elbows and knees. This isomorphic phenomenon can occur in other diseases such as warts, vitiligo, lichen planus, and Darier disease.

As psoriasis is a chronic inflammatory condition these patients are at increased risk of developing the **metabolic syndrome** (Table 15.3). The metabolic syndrome is a cluster of risk factors that increases the overall risk of cardiovascular disease and type 2 diabetes. The interaction between the various components of the metabolic syndrome contributes to the development of a pro-inflammatory state and a chronic, subclinical vascular

**Table 15.2** Drugs that may trigger or aggravate psoriasis or cause an psoriasisiform eruption [1]

- Alcohol in excess
- Anti-malarials (e.g. chloroquine and hydroxychloroquine)
- Lithium
- Beta Adrenergic Antagonists (e.g. Atenolol®)
- Angiotensin-converting enzyme inhibitors (ACE inhibitors)
- Sudden withdrawal of potent topical or systemic steroids
- Antibiotics (e.g. tetracycline)
- NSAIDs
- Interferon
- Terbinafine
- Benzodiazepines
- Nicotine may aggravate palmoplantar, pustular psoriasis (also known as palmoplantar pustulosis)

**Table 15.3** The metabolic syndrome<sup>a</sup>

To have the metabolic syndrome a patient must have three or more of these characteristics:

*Obesity: a waist size greater than 35 inches for women and 40 inches for men.* Certain genetic risk factors, such as having a family history of diabetes or being of Asian descent, lower the waist circumference limit: If you have one of these genetic risk factors, waist size limits are 31–35 inches for women and 37–39 inches for men.

*Abnormal blood cholesterol levels:* either elevated triglycerides (a type of fat in the blood) or low levels of HDL (the “good cholesterol”)

*Hypertension*

*Diabetes or insulin resistance*

<sup>a</sup>According to guidelines developed by the National Cholesterol Education Program (USA), with modifications by the American Heart Association

inflammation which results in atherosclerosis. The metabolic syndrome confers a five-fold increase in the risk of type 2 diabetes mellitus and two-fold the risk of developing cardiovascular disease over the next 5–10 years. Patients with the metabolic syndrome are at two to four-fold increased risk of stroke, a three to four-fold increased risk of myocardial infarction, and two-fold the risk of dying from such an event compared with those without the syndrome, regardless of a previous history of cardiovascular events [5]. First line treatment of the metabolic syndrome is life style modification including weight loss, a low sugar, low fat diet and more aerobic exercise. Further studies are needed to determine whether drugs such as TNF $\alpha$  inhibitors could also improve associated metabolic syndrome or cardiovascular risks.

**Depression** is more prevalent in people with psoriasis. Patients with severe psoriasis are three times more likely to suffer depression compared to controls [6]. Treatment of psoriasis may help the patient’s mood.

## 15.5 Topical Treatments

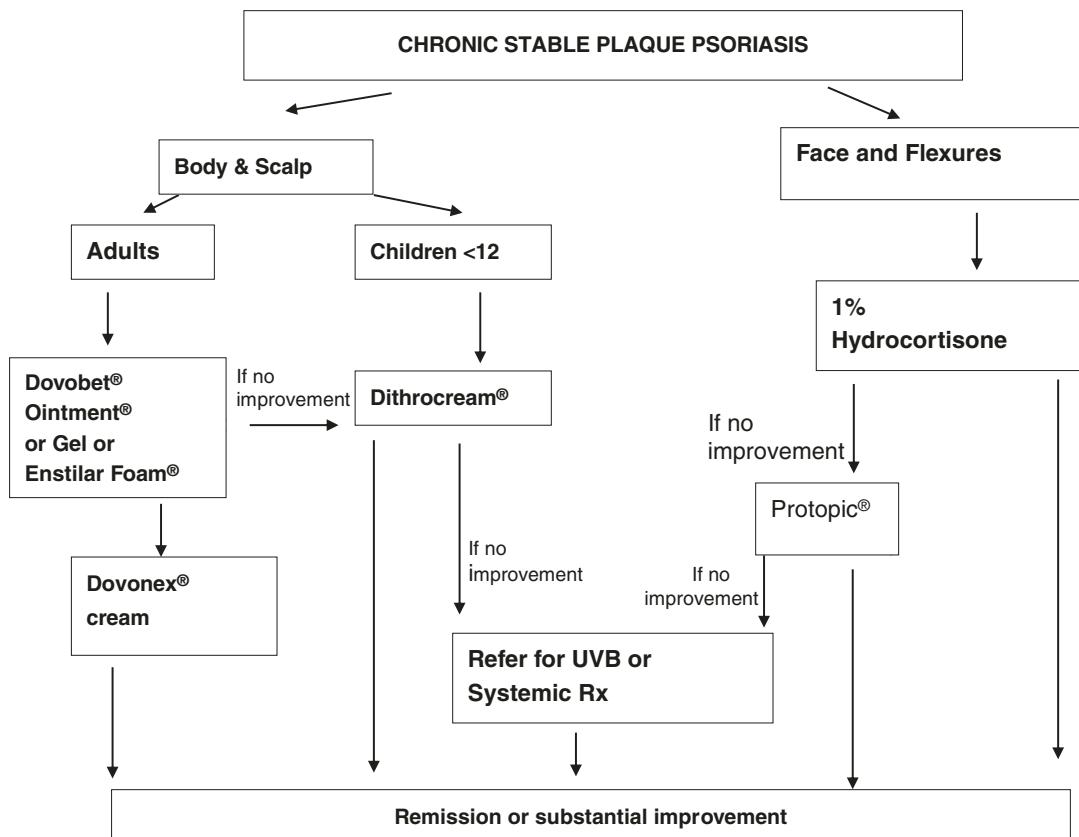
Not all cases of psoriasis require treatment. Many patients with localised psoriasis on their elbows, knees and or scalp can learn to live with their condition by covering it up with appropriate clothing or manage it with simple moisturisers. Others can

be very self-conscious even with limited disease, which may interfere with their quality of life and affect their work, social life, sex life or hobbies. For some, going to the swimming pool, a changing room or the beach can be a nightmare.

Fortunately, most patients with mild to moderate disease can now be managed safely and effectively in general practice. The first step in the management of psoriasis is making the patient understand that this is a chronic condition. They need to know they will have to incorporate routines of care into their daily life. The plaques need to be **moisturised** liberally with a safe, greasy moisturiser after baths or showers, such as Emulsifying ointment, “Epaderm ointment®” or Paraffin gel. This will reduce the silvery scale and make the psoriasis look and feel better. Moisturisers also aid penetration of more specific psoriasis treatments. A tar-based shampoo will help lift off the scales on the scalp and if there is co-existing dandruff, a good anti-dandruff shampoo such as “Nizoral®” or “Stieprox®” shampoo should be used two or three times a week. Very thick scalp scales can be removed with a tar and salicylic acid ointment such as “Cocois®”.

**Guttate psoriasis** is usually self limiting and should be treated symptomatically. Simple emollients may be sufficient in mild cases. If there is troublesome itch, a potent topical steroid may help on the body. Coal tar preparations such as “Exorex® 5% lotion” or urea containing creams may also help ease itch and reduce the scale. For more troublesome guttate psoriasis phototherapy can be very helpful.

The first line treatment for adults with chronic, stable, plaque psoriasis on the body is usually with a combination of **calcipotriol (a Vitamin D analogue) and betamethasone**, (a potent steroid, the same as is found in “Betnovate®”) (e.g. “Dovobet®” ointment or gel or “Enstilar Cutaneous Foam®”), (Fig. 15.12). The foam preparation is more cosmetically acceptable. In clinical trials, response rates were higher with “Enstilar®” foam than with an ointment or gel formulation of calcipotriol/betamethasone (“Dovobet®”) and were achieved earlier [7]. The gel formulation can be used on the scalp. The ointment preparation is greasier to use.



**Fig. 15.12** Psoriasis flow chart

The advantage of “Dovobet®” and “Enstilar Cutaneous Foam®” is that it is relatively quick to clear the psoriasis plaques and can be used in a convenient once a day application, which usually does not burn, sting or stain the skin. The disadvantages are that it is expensive and does not work on all patients with psoriasis. “Dovobet®” is not licensed for people under 18 years of age, although it can sometimes be used off licence in teenagers from the age of 12–18. The maximum dose in adults is 15 g a day, or 100 g a week for acute management of psoriasis in the first month of treatment and it should not be used on more than 30% of body surface area. It is applied once daily for 4 weeks and by this stage the silvery, scaly plaques should have faded out to a red, macular rash. If necessary, “Dovobet®” can be continued three times a week for at least another month. There is experience with repeated courses

of “Dovobet®” up to 52 weeks. If it is necessary to continue or restart treatment after 4 weeks, treatment should be continued after medical review and under regular medical supervision to ensure the patient does not develop any potent steroid side effects. Maintenance treatment, if required, can be continued with “Dovonex® cream” or “Silkis® ointment” (“Silkis®” contains calcitriol, a vitamin D analogue like calcipotrol) which can be used daily until the psoriasis is fully cleared or improved to an acceptable level.

“Enstilar Cutaneous Foam®” is applied to the affected area (not exceeding 30% of total body surface area) once daily for 4 weeks, with a maximum daily dose of 15 g (0.5 g covers the equivalent of an adult hand). One 60 g can should therefore last at least 4 days. 0.5 g corresponds to the amount administered from the can if the actuator is fully depressed for 2 seconds.

More recently these combinations of **calcipotriol and betamethasone** ("Dovobet®" or "Enstilar®") have obtained a licence for long-term use. It might be preferable to wean patients off these potent steroid combinations after 1–3 months and save it for relapses of psoriasis. Using potent steroids long term (greater than 3 months) may cause skin atrophy (which looks very like partially treated psoriasis) and possibly systemic absorption with adrenal suppression. A rebound flare of psoriasis can occur if these potent steroid combinations are stopped suddenly. "Dovobet®" and "Enstilar®" should never be applied to the face or flexures in adults and it is not suitable for children under the age of 12 years.

"Dovobet® gel" is useful for **scalp psoriasis** where it should be rubbed into the plaques and left on overnight. It can be removed in the morning by applying a shampoo to the gel on the dry scalp for a few minutes to soften the gel before wetting the hair and lathering up the shampoo. The gel will then easily wash out once the hair is rinsed. Daily hair washing can be tedious for some people. Applying "Dovobet® gel" to the scalp daily for the first week or two and then three times a week until the psoriasis has cleared may be more convenient (this usually takes 1–3 months).

For more resistant scalp psoriasis, "**Etrivex Shampoo®**" which contains a super potent topical steroid (clobetasol propionate which is also found in "Dermovate®") may help in adults, but treatment should be limited to 1 month and then weened down.

These complicated treatment regimes for applying "Dovobet gel®" or ointment to the body and scalp are difficult to explain to a patient during the course of routine general practice consultation. Written instructions are essential and follow-up monthly for the first few months is useful to encourage compliance and to monitor progress. Advice by a nurse trained in the use of these products is very useful in helping patients manage their psoriasis.

Patients who have only very small plaques of psoriasis in localised areas of the body may not want the expense of buying a large tube of "Dovobet®" or "Enstilar®". In these circumstances, it can be more cost effective to prescribe

"Dovonex®" in the morning and a potent topical steroid (e.g. "Betnovate® ointment") at night to all the plaques on the body for 1 month. The patient can then be weaned off the steroid ointment by using it three times a week in the second month and stopping it altogether in the third month of treatment while continuing with "Dovonex®" daily until the psoriasis is cleared or well controlled. "Dovonex®" cream can be used with a moderately potent® topical steroid (e.g. "Eumovate® Ointment") in children from 6 to 12 years old in a similar fashion.

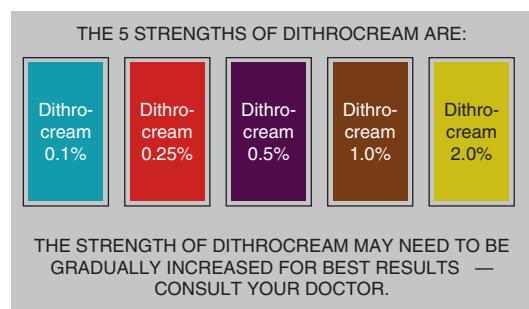
While "Dovobet®" and "Enstilar®" are clean and relatively simple to use, experience shows that they only work in approximately 60–70% of patients with chronic stable plaque psoriasis; besides, they are not licensed for children under the age of 18. In these patients, **dithranol** is extremely effective, although more messy and time consuming to use. Although not commonly prescribed nowadays, dithranol (also called anthralin) is one of the most effective preparations available for treating chronic plaque psoriasis. Dithranol has been used for more than 100 years in the treatment of psoriasis. It is a chemical of plant origin, taken from the bark of a South American tree. Its precise mode of action is still to be confirmed, although it has been shown to inhibit DNA replication, keratinocyte hyperproliferation, granulocyte function and, in addition, may exert an immunosuppressive effect. Free radicals, histamine, eicosanoids and platelet-activating factor have been shown to be involved in dithranol-induced dermatitis and the oxidation products of the drug are responsible for the staining.

The "Short Contact Treatment" using a preparation called "Dithrocream®" is the most convenient way to use dithranol for home treatment. "Dithrocream®" comes in five different strengths from 0.1% up to 2%. Patients should be instructed to start with the weakest strength and to apply it to the plaques on the body and scalp (not for the face or flexures) for 30 minutes daily for 1 week. It can be washed off in the shower but patients should be warned that it will stain everything, including clothing, towels and the skin. Each week, the strength should be increased until the psoriasis clears. If the skin gets red or sore (usu-

ally at the higher strengths) the treatment should be stopped for a few days and an emollient applied until the soreness settles. Then the treatment can be restarted but at the next strength down. The best way to tell when the psoriasis is cleared and when to stop “Dithrocream®” is to get the patient to rub their hand over the affected area. If it is smooth like the surrounding skin, although stained with dithranol, it can be stopped (Fig. 15.13a, b). If it is rough, they should continue short contact treatment with dithranol until smooth.

Once “Dithrocream” is stopped, the staining will fade spontaneously over the following few weeks, or this can be accelerated by applying a Tar based ointment such as “Coal Tar and Urea” or “Exorex Lotion®”, rubbing it downwards daily to the stained area (Fig. 15.14). Tar ointments are smelly, sticky

and not usually popular with the patients. When prescribing dithranol, written instructions are essential for the patient and a trained nurse can ensure good compliance and good success. When “Dithrocream®” is used properly it can clear up to



**Fig. 15.14** The various strengths of “Dithrocream®”



**Fig. 15.13** (a) Small plaque psoriasis before treatment (b) Same patient immediately after 4 weeks of dithranol treatment

80–90% of adults and children with mild to moderate, stable plaque psoriasis in approximately 6 weeks and can result in longer remissions than other treatments, such as “Dovobet®” and “Dovonex®”. It is however more time consuming and messy to apply and wash off [8].

For more troublesome chronic plaque psoriasis, **combining treatments** may be helpful. For example, dithranol and tar is a good combination, or “Dovobet®” and dithranol can be used simultaneously. Patients with severe psoriasis should be referred for ultraviolet light therapy or systemic treatments. The sun can help psoriasis in most patients and may augment the therapeutic response from topical treatment such as “Dovobet®/Dovonex®” or dithranol. The national health services could consider paying for a cheap 2 week package holiday to the sun for psoriasis patients, which might work out cheaper than 6 weeks phototherapy!

Psoriasis on the **face and flexures** is usually less thick and scaly than on other parts of the body and will often respond to 1% hydrocortisone ointment (Figs. 15.15 and 15.16). If there are any signs of co-existing seborrhoeic dermatitis, then

1% hydrocortisone combined with an imidazole anti-fungal like “Daktacort®”, “Canesten HC®” should help. For more resistant psoriasis on the face or flexures, tacrolimus (“Protopic®”) can be extremely safe and effective, although it is not licensed for this indication. It can cause a transient redness and soreness of the skin during the first week of treatment in 50% of patients and it is important to warn patients of this possibility.

**Nail** psoriasis is very difficult to treat. Using a potent topical steroid gel or lotion (e.g. “Betnovate Scalp Application®” or “Dovobet Jel®”), which can be flooded under the distal end of the nail, may help some cases. If the nail changes are severe and the patient is demanding treatment, systemic treatments are the most successful way to manage this problem (see below).

**Palmoplantar pustulosis** (PPP) may not be a true form of psoriasis but it may respond to a potent topical steroid or a steroid/calcipotrol combination (Fig. 15.17). More severe cases may require a super potent topical steroid such as clobetasol propionate which is also found in “Dermovate®”. Patients with PPP should moisturise liberally, avoid soaps and other irritants,



**Fig. 15.15** Psoriasis of the face (left image) and on face, neck and thorax (right image)



**Fig. 15.16** (a) Psoriasis on a child's face (b) Psoriasis on the same child's leg

keep their hands dry as much as possible by the careful use of cotton and rubber gloves and avoid smoking.

## 15.6 Systemic treatments

More **severe or resistant cases** of psoriasis may need referral to a dermatologist for ultraviolet light therapy (narrowband UVB or PUVA) or **systemic treatment** with drugs such as methotrexate, retinoids (acitretin), fumaric acid esters or cyclosporine.

**Apremilast ("Otezla®")** is an oral PDE4 inhibitor, indicated for the treatment of moderate to severe chronic plaque psoriasis in adult patients who failed to respond to or who have a contrain-

dication to, or are intolerant to other systemic therapies including cyclosporine, methotrexate or psoralen and ultraviolet-A light (PUVA). It should be initiated by specialists experienced in the diagnosis and treatment of psoriasis or psoriatic arthritis. The dose has to be titrated up gradually from 10 mg OD up to 30 mg BD over a week. During pivotal trials the greatest improvement was observed within the first 24 weeks of treatment. Side effects of apremilast in psoriasis clinical studies included nausea and vomiting (in up to 30% of patients), depression, weight loss, diarrhoea, upper respiratory tract infection and headache. It is not licensed for children under the age of 18 years and cannot be used in pregnancy.

If patients do not respond to these treatments they may be eligible for the newer, more expen-



**Fig. 15.17** Plantar psoriasis on a patient with palmoplantar psoriasis

sive **biological treatments** such as TNF-alpha antagonist. These are large molecules and most are given by subcutaneous injections at home or IV infusions in hospital every few weeks. Their introduction has revolutionised the management of severe psoriasis in recent years.

**Biological therapy** (also known as targeted immune modulators) has developed at a remarkable rate with indications for a range of diseases within gastroenterology, rheumatology, dermatology, oncology and ophthalmology. Biologic agents are a set of engineered proteins that possess pharmacologic activity and can be extracted from animal tissue or, much more commonly, synthesised in large quantities through recombinant DNA techniques. Biologic molecules (antibodies, fusion proteins or recombinant cytokines) can be designed to either mimic the actions of normal human proteins or to interact with circulating proteins or cellular receptors to modify the immune responses in psoriasis [9].

Biologics used to treat psoriasis include adalimumab (“Humira®”), etanercept (“Enbrel®”), infliximab (“Remicade®”), secukinumab

(“Cosentyx®”), and ustekinumab (“Stelara®”). Because they are very expensive and may cause immunosuppression, these biological therapies are restricted to hospital use only and are used only in severe, extensive, resistant psoriasis and in psoriatic arthritis.

## 15.7 Conclusion

Psoriasis can run an unpredictable course. Some patients can get one, self limiting break out of the rash which may never recur again. Others can have chronic or relapsing flare-ups of their psoriasis all their life. Psoriasis may be very limited and mild in some patients, yet extensive and severe in others. Although there is as yet no cure for psoriasis, almost all patients can be managed by simple topical treatments at home while some require hospital based treatment such as UVL or systemic therapies. The improved understanding of the pathophysiology of psoriasis has led to the development of a number of targeted biological treatments which are extremely effective. Many of these new therapies are very expensive and can have unpredictable side effects, especially in relation to their immunosuppressive effects.

It is important to reassure patients that psoriasis is not contagious, infectious or cancerous but, most of all, that is a chronic condition.

Patients with psoriasis have a higher incidence of obesity, hypertension, hypercholesterolaemia, diabetes, heart disease, depression, and the metabolic syndrome and they should be screened for these conditions. Care should be taken on the choice of medications for these conditions as some may precipitate or aggravate psoriasis.

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# Seborrhoeic Dermatitis (SD)

16

David Buckley

## Key Points

- Infantile seborrhoeic dermatitis is sometimes confused with atopic eczema but unlike atopic eczema, there is usually little or no itch—Seborrhoeic dermatitis is usually associated with cradle cap in children and dandruff in adults.
- It is thought that seborrhoeic dermatitis sufferers may develop an excessive inflammatory response to the commensal yeast, *Malassezia*, which is found in excessive numbers in patients with seborrhoeic dermatitis.
- Anti-yeast shampoos, creams and tablets often help in seborrhoeic dermatitis.

## What to Tell the Patient

- Seborrhoeic dermatitis may be aggravated by stress, fatigue, depression, diabetes, some medications, excess alcohol or excess sugar in the diet.
- Anti-dandruff shampoos usually helps on the scalp and face.
- It usually responds to a weak topical steroid (1% hydrocortisone) on the face and flexures. Frequent, recurrent and resistant cases may need topical calcineurin inhibitors such as tacrolimus (“Protopic®”).

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## 16.1 Introduction

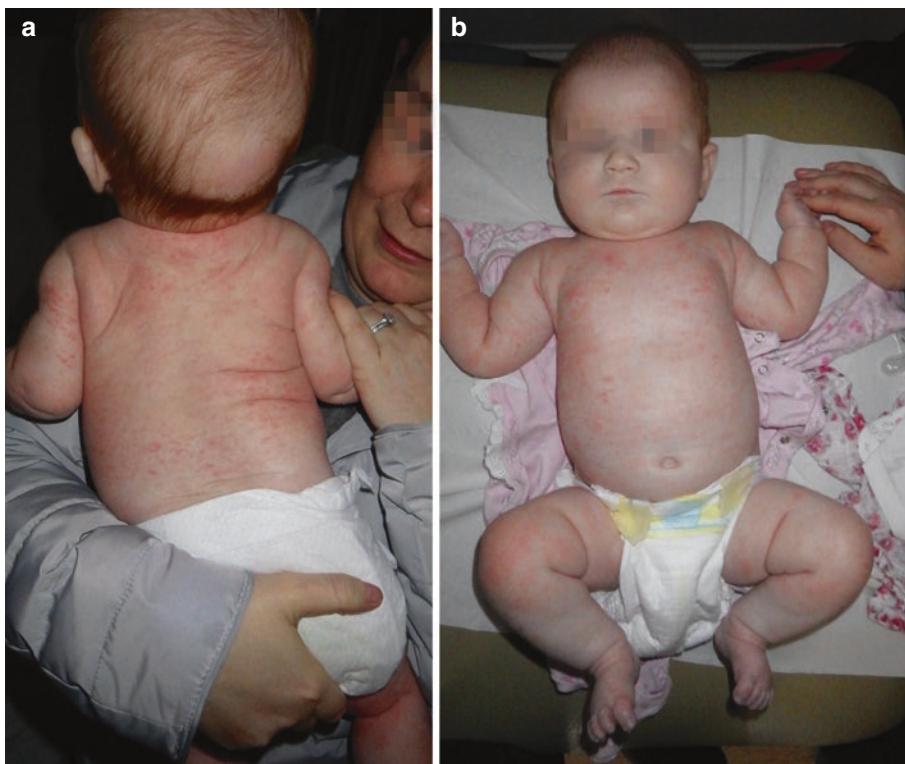
Seborrhoeic dermatitis (SD) (also known as “seborrhoeic eczema or seborrhoeic dermatitis”) is a very common skin condition in infants and adults. While it is not dangerous in any way, it can be uncomfortable and unsightly, leading to distress and embarrassment for the sufferer [1]. The name seborrhoeic derives from the fact that the rash is distributed in the greasy (sebaceous) areas like the face, scalp and centre of chest and back where there is a high concentration of sebaceous glands.

## 16.2 Clinical Features and Diagnosis

There are two distinctive forms of seborrhoeic dermatitis: infantile and adult seborrhoeic dermatitis.

### 16.2.1 Infantile Seborrhoeic Dermatitis

This usually presents as a non-itchy, red, scaly rash in infants less than 12 months old. It is sometimes confused with atopic eczema but unlike it, there is usually little or no itch. The rash usually presents as cradle cap in an infant. It can then spread onto the face and trunk. The rash is mainly distributed in flexures, especially the neck and axillae. It can also affect the trunk in a similar



**Fig. 16.1 (a, b)** Infantile seborrhoeic dermatitis

distribution to atopic eczema (Fig. 16.1a, b). There can sometimes be a rash over the sternum and there may be associated napkin dermatitis.

Treatment of infantile seborrhoeic dermatitis is with emollients and soap substitutes. If the rash is itchy or unsightly, 1% hydrocortisone ointment is safe and helpful on the face and body. As the condition is not itchy and resolves spontaneously within the first year of life, simple reassurance for the parents may be all that is required.

Troublesome cradle cap can be treated with a salicylic acid shampoo (e.g. "Capasal®") which can be applied to the scaly areas for a few minutes and then rinsed out with water. It can be used daily until the scales become loosened and can be easily combed off.

### 16.2.2 Adult Seborrhoeic Dermatitis

This usually begins in teens or twenties and may be associated with a dry, itchy scalp (dandruff = pity-

riasis capitis) with flaking but little or no erythema. When there is more inflammation, there will be more erythema and diffuse fine scaling but no thick scales. If there is deep erythema with a sharp cut off between the involved and uninvolved skin, spreading beyond the hair line and thick scaling, it is more likely to be due to psoriasis.

Most patients with seborrhoeic dermatitis will have an erythematous, slightly scaly rash in a characteristic distribution of the face and body. The rash usually affects the nasolabial folds, the eyebrows and the moustache or beard area of men who have facial hair (Fig. 16.2). The rash may also develop between the eyebrows, behind the ears and on the eyelids (blepharitis). There may be an associated dermatitis in the ear canal (otitis externa). More severe cases may have a mild, non-itchy, erythematous rash over the sternum and between the scapula on the back (Figs. 16.3 and 16.4). Some cases may have an eczematous rash in the axillae, groin, penis and perianal skin.



**Fig. 16.2** Seborrhoeic dermatitis in an adult's face. A more severe case on the left; a milder case on the right



**Fig. 16.3** Seborrhoeic dermatitis on face and chest in an adult male

Diagnosis of seborrhoeic dermatitis is usually clinical. Skin scrapings and biopsies are normally not necessary and may be unhelpful in making the diagnosis.



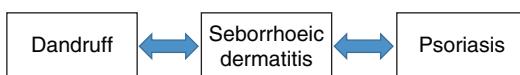
**Fig. 16.4** Seborrhoeic dermatitis in the central part of the chest

latter is usually responds itchy and is associated with other atopic diseases such as asthma, allergic rhinitis and allergic conjunctivitis. SD can also be confused with psoriasis and indeed sometimes coexists with psoriasis (sebopsoriasis) (Fig. 16.5). It can sometimes be difficult to judge where seborrhoeic dermatitis ends and psoriasis begins although psoriasis is usually more red and scaly and has a very sharp cut off between the involved and uninvolved skin. There may also be nail changes or evidence of psoriatic arthritis which would favour a diagnosis of psoriasis. Seborrhoeic dermatitis on the trunk may resemble pityriasis versicolor.

Other conditions that cause a red face such as perioral dermatitis, rosacea, and lupus erythematosus may be confused with seborrhoeic dermatitis. Contact allergic or irritant dermatitis or a drug eruption may present like seborrhoeic dermatitis. Scalp SD and dandruff are of a con-

### 16.3 Differential Diagnosis

Seborrhoeic dermatitis (SD) can be confused with atopic eczema in children and adults. The



**Fig. 16.5** The spectrum of disease in SD

**Table 16.1** Differentiated Diagnosis for seborrhoeic dermatitis

<b>Scalp</b>	<ul style="list-style-type: none"> <li>- Psoriasis</li> <li>- Tinea capitis</li> <li>- Dermatitis (irritant or allergic or atopic)</li> <li>- Pityriasis amantacea</li> </ul>
<b>Face</b>	<ul style="list-style-type: none"> <li>- Psoriasis</li> <li>- Steroid rosacea</li> <li>- Lupus (DLE/SLE)</li> <li>- Perioral dermatitis</li> <li>- Dermatitis (irritant or allergic or atopic)</li> </ul>
<b>Body</b>	<ul style="list-style-type: none"> <li>- Psoriasis</li> <li>- Atopic eczema</li> <li>- Pityriasis versicolour</li> </ul>
<b>Groin</b>	<ul style="list-style-type: none"> <li>- Psoriasis</li> <li>- Pruritis ani/vulva</li> <li>- Candidiasis</li> <li>- Intertrigo</li> <li>- Tinea curis</li> <li>- Dermatitis (irritant or allergic or atopic)</li> <li>- Erythrasma</li> <li>- Lichen planus</li> <li>- Lichen sclerosus</li> </ul>

tinuous spectrum of the same disease that affects the seborrhoeic areas of the body. Dandruff is itchy, restricted to the scalp and it shows no visible inflammation. SD is itchy, flaking or scaling with inflammation. The latter can be confused with pityriasis amantacea or tinea capitis. Flexural seborrhoeic dermatitis may look like intertrigo, erythrasma or flexural psoriasis (Table 16.1).

## 16.4 Pathophysiology

The aetiology of SD is not fully understood but there is a strong genetic predisposition in most cases and there is often a positive family history of SD, dandruff or psoriasis (Fig. 16.5). It is thought that sufferers may develop an excessive inflammatory response to the commensal yeast, malassezia, which is found in excessive numbers

on sufferers. Malassezia refers to a group of yeasts of the genus Malassezia, which has several different species. This group of yeasts are also implicated in malassezia folliculitis and pityriasis versicolour.

SD may belong to a spectrum of diseases with dandruff being the mildest manifestation and psoriasis being the most severe (see Fig. 16.5). Some cases may progress onto psoriasis and there is sometimes a family history of psoriasis in patients with SD and vice versa. Some patients have features of both SD and psoriasis (sebopsoriasis). SD may be aggravated by stress, fatigue, depression, Parkinson's disease, epilepsy, excess alcohol or excess sugar in the diet. Certain medications such as buspirone, chlorpromazine, cimetidine, griseofulvin, haloperidol, lithium, interferon alfa and methyldopa can all aggravate it. Underlying illnesses such as diabetes or immune-suppression (e.g. HIV, chemotherapy, systemic steroids) may precipitate or aggravate this condition. Most cases improve in the summer. Many patients go through periods of exacerbation and remissions. It is much less common in the elderly and almost never affects the balding scalp.

## 16.5 Treatment of adult SD

Treatment of SD is symptomatic. A dry, itchy scalp usually responds to a twice or three times weekly anti-dandruff shampoo such as zinc pyrithione, selenium sulphide, ketoconazole ("Nizoral® shampoo") or ciclopiroxolamine ("Stieprox® shampoo"). Bringing the suds of these shampoos down onto the affected areas of the face and/or body and leaving it soak into the scalp and other areas may help clear the face and body rash as well as the dry, itchy scalp (Fig. 16.6). Some patients may benefit from coal tar or a salicylic acid shampoo (e.g. "Capasal®"). If there is excessive build-up of scale, a salicylic acid ointment (e.g. "Cocois®") left soak in for an hour and then washed out with one of the above mentioned shampoos may help. This product



**Fig. 16.6** Mild SD of the face



**Fig. 16.7** Severe SD of the face

should be avoided in children less than 6 years old as excessive absorption may cause salicylate toxicity.

More persistent scalp SD may respond to potent topical steroids in scalp lotion like betamethasone (“Betnovate<sup>®</sup>”) scalp application or mousse (“Bettamousse<sup>®</sup>”) which can be applied on alternate days until the condition is under control. Very potent steroid shampoos such as clorotetasol (“Etrivex shampoo<sup>®</sup>”) may help in severe scalp SD but it should be washed out after 15 minutes and should only be used for a maximum of 1 month.

A weak topical steroid such as 1% hydrocortisone ointment is safe and effective for SD affecting the face and ears. Some patients may respond to topical ketoconazole cream. Mixing 1% hydrocortisone with an imidazole antifungal such as “Daktakort<sup>®</sup>” or “Canestan HC<sup>®</sup>” can also be safe

and effective for face and ears. More troublesome, resistant cases may respond to topical tacrolimus 0.1% ointment (“Protopic<sup>®</sup>”) or pimecrolimus but this is an off-licence indication [2] (Fig. 16.7).

Azelaic acid (“Skinorin<sup>®</sup>”) has antifungal as well as antikeratinizing, and anti-inflammatory activity. Azelaic acid has been shown to be helpful in SD with concomitant rosacea or acne [3].

When SB affects the flexures, groin, genitalia or perianal skin it should be treated the same as outlined above for face and ears SD. On the body it usually responds to emollients, avoidance of soaps and other irritants and a moderately potent or potent topical steroid.

Oral anti yeast medications are occasionally required for more severe, resistant cases [2]. Itraconazole (“Sporanox<sup>®</sup>”) 200 mg daily for a

week and then 200 mg daily for two consecutive days once a month for 6–12 months may help in chronic relapsing cases. Patients on oral isotretinoin for acne who also have SD may find their rash tends to improve on this drug.

## 16.6 Conclusion

Seborrhoeic dermatitis (SD) is a common, scaly skin condition that usually occurs on the face, scalp and chest. It may be associated with underlying illness such as diabetes or HIV, although the majority of patients are quite healthy. Most patients will have coexisting dry scaly scalp. Treatment is usually with an anti-

dandruff shampoo for the scalp and 1% hydrocortisone for the face. More resistant cases may need topical calcineurin inhibitors such as tacrolimus (“Protopic®”).

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# The Red Face

17

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## Key Points

- A red face can be the result of a physiological process such as flushing, because of ageing, particularly photo ageing or due to a wide range of dermatological conditions.
- Telangiectasia (broken or thread veins) are probably the most common cause of permanent red face.
- Flushing/Blushing can cause embarrassment, anxiety and even social phobia.
- In rosacea there are no comedones and the skin is usually not greasy.

## What to Tell the Patient

- Be careful to avoid the use of moderately potent or potent topical steroids on the face long term, especially in children and on the eyelids, as they will invariably lead to skin thinning and redness of the face.
- Most conditions causing a red face will be aggravated by too much ultraviolet light. Wear a high SPF sun-block and a broad brimmed hat when outdoors.
- Make-up and cosmetics can help hide redness on the face and are usually safe to use.

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## 17.1 Introduction

Many patients present to their doctor with a red face. It can be an embarrassing and sometimes uncomfortable problem which can often lead to anxiety and social phobia. Management will depend on the underlying cause. Some patients may have more than one problem contributing to the redness on their face. Most patients will be helped by cosmetic camouflage and photoprotection.

## 17.2 Telangiectasia (Broken or Thread Veins)

This is probably the most common cause of permanent red face. It is made up of multiple small, permanent blood vessels (telangiectasia) varying from 1 to 3 mm in diameter usually affecting the nose, upper cheek and the chin (Fig. 17.1). They mostly occur in people with type 1 and type 2 skin. They are usually caused by excessive ultraviolet light (UVL) as a result of an outdoor occupation, outdoor sports, outdoor hobbies, foreign travel or tanning. Although they usually occur in isolation, there may be other signs of photo damage such as solar lentigo, solar elastosis, actinic keratosis or perhaps even non-melanoma skin cancer. Strict avoidance of UVL on the face (SPF 50, a broad rimmed hat and Vitamin D supplements) will help prevent the problem getting worse.



**Fig. 17.1** Red face. Note the numerous telangiectasia on the face

Brimonidine cream (“Mirvaso®”) is a topical vasoconstrictor which works within 30 min and can last up to 12 hours. It can give temporary relief of redness particularly for special occasions. 10–15% of patients using this cream can experience a rebound flare of their redness once the effects of the cream wear off after approximately 12 hours. Patients should be encouraged to try this cream on a small test area for the first few applications before applying it all over the face. Cosmetic camouflage will also help. The only permanent solution is treatment with a vascular laser such as a pulsed dye or an Nd:YAG laser system. These lasers are also useful for removing spider naevi.

### 17.3 Flushing/Blushing

Flushing/Blushing is a physiological reaction that usually causes sudden, temporary redness on the face and it sometimes can affect the neck, the upper chest and back. It can be triggered by various factors including emotional stress, embarrassment, exercise, heat, alcohol, spicy foods, hormonal factors, febrile illness and certain drugs (Table 17.1). Some patients may benefit from a diet low in histamines and other vasoactive amines (Table 17.2). Management involves avoiding the triggering factors if possible, cosmetic camouflage, and using cooling creams such as “Silcocks Base®” or “Aveeno with Menthol®”

**Table 17.1** Some causes of flushing

Heat
Exercise
Febrile illness
Embarrassment
Menopause
Panic disorder
Hyperthyroidism
Diabetes insipidus
Carcinoma of the pancreas
Carcinoid tumours
Phaeochromocytoma
Brain tumours and spinal cord lesions
Frey’s syndrome (flushing when eating)
Foods—e.g., Spicy foods, monosodium glutamate, allergic reactions
Drugs:
• Nitrates
• Alcohol
• Calcium-channel blockers
• Selective serotonin reuptake inhibitors (SSRIs)
• Levodopa
• Selective oestrogen receptor modulators (SERMS) such as raloxifene and tamoxifen
• Anti-androgens such as cyproterone, spironolactone, bicalutamide, 5-alpha-reductase inhibitors
• Danazol
• Gonadotropin releasing hormone superagonist ( <a href="#">GnRH agonist</a> ). e.g. Goserelin (“Zoladex®”)

**Table 17.2** Foods and drinks that are particularly high in vasoactive amines include

• Wine, beer, cider, champagne
• Coffee, cocoa, chocolate
• Fermented soya products including miso and tempeh
• Blue cheeses, parmesan cheese, camembert, emmental, old gouda, cheddar and other hard cheeses, fresh and hard sheep and goat cheeses
• Cured meat especially pork products e.g. sausages and other processed meats (ham, salami, pepperoni, bacon)
• Fresh or canned tuna, sardines, mackerel, salmon, herring, processed fish products e.g. fish pastes, smoked or dried pickled fish
• Tomatoes, pickled cabbage (sauerkraut), broad beans, aubergine, spinach
• Peanuts, tree nuts
• Oranges, tangerines, bananas, pineapple, grapes, strawberries

which can be kept in the fridge. Flushing/Blushing can cause embarrassment, anxiety and even social phobia. Some patients can be helped by psychotherapy such as cognitive behaviour therapy. Beta blockers or clonidine (50–75 mcg BD) may also help. HRT can be very helpful with menopausal flushing. “Mirvaso®” can be useful especially for special occasions but rebound flushing can be a problem in up to 20% of patients. Selective serotonin reuptake inhibitors (SSRIs) or venlafaxine may help some patients. Gabapentin which is usually used as an anticonvulsant may also be effective. Some cases respond to laser treatment.

#### 17.4 Rosacea

As the name implies, rosacea usually presents with redness of the face from a combination of flushing and telangiectasia and is mostly found in people with fair skin (Celts). It is an inflammatory process and there are usually small papules and pustules confined to the face. However, unlike acne, in rosacea there are no comedones and the skin is usually not greasy (Fig. 17.2). Rosacea usually occurs in adults over the age of 30 years old but can occasionally occur in younger people. Occasionally, a patient can present with features of both rosacea and acne (“red acne”). Eyelid involvement is common in rosacea with blepharitis being the most common complaint (see Chap. 10).

#### 17.5 Seborrhoeic Dermatitis (SD) and Psoriasis

Seborrhoeic dermatitis and psoriasis can cause a red, scaly rash on the face. There are often clues to the diagnosis elsewhere such as a dry scaly scalp, a rash over the sternum or the typical signs of psoriasis on the elbows, knees and nails. Seborrhoeic dermatitis and psoriasis are usually diagnosed clinically. The rash on the face usually has a characteristic distribution affecting the naso-labial folds, the eyebrows, the external auditory canal, the moustache and beard area in



**Fig. 17.2** Rosacea in a 49 year old

men who have left their beard or moustache grow. The rash can sometimes affect the eyelids (blepharitis), the groin or peri-anal area. SD and psoriasis, when they affect the face, are often chronic or relapsing and treatment is usually with an anti yeast shampoo and a weak topical steroid or topical calcineurin inhibitor [1] (e.g. “Protopic®”) (see Chaps. 14, 15 and 64).

#### 17.6 Steroid Damage

Moderately potent or potent topical steroids should never be used on the face long term as they will invariably lead to skin thinning and redness of the skin (Fig. 17.3). This can occur more rapidly in patients with rosacea who often get a rebound flare-up of their rosacea once the potent steroid is stopped. In this situation it may be kinder to the patient to wean them from a potent steroid down to a moderately potent steroid for the first 2 weeks followed by a weak topical steroid such as 1% hydrocortisone for a further 2 weeks before stop-



**Fig. 17.3** Steroid rosacea in an 11-year-old child using hydrocortisone butyrate cream on the face. (“Locoid®”)

ping the steroid completely. At the same time the patient should be treated with topical and oral rosacea treatments (see Chap. 10).

Steroid damage is more likely to occur in children and on the eyelid in adults where the skin is extremely thin. Even prolonged use of weak topical steroids can sometimes cause steroid damage to the eyelids. Tacrolimus is a safer alternative for eczema/dermatitis on the face or the eyelids, particularly in children.

Short courses of moderately potent or potent topical steroids can occasionally be used on the face in adults for a maximum of 5 days for severe flare-ups of eczema/dermatitis in the absence of infection or rosacea. The only other exception is that potent or very potent topical steroids may be required to treat lupus affecting the face but they should only be applied directly to the plaques and not to the surrounding uninvolved skin.

## 17.7 Cellulitis and Erysipelas

Bacterial infections of the skin can occur on any part of the body including the face. Cellulitis is a deep-seated infection of the skin that may be associated with systemic flu-like symptoms and a fever. It has all the usual hallmarks of infection with redness, heat, tenderness and swelling. The rash usually spreads outwards from its origins which is usually a small scrape or cut in the skin which allows the organism (which is often a commensal) into deeper layers of the skin. Cellulitis is usually caused by *Streptococcus pyogenes* (2/3 of cases) or *Staphylococcus aureus* (1/3 of cases). Treatment is normally with flucloxacillin, either orally or intravenously for 10–14 days. With more severe cellulitis, benzoyl penicillin (penicillin g) or oral penicillin (penicillin v) may need to be given in addition to flucloxacillin.

Erysipelas (also known as St Anthony’s fire) is a similar infection to cellulitis but usually occurs on a more superficial plane in the skin and so there is a more obvious firm, red, raised, well defined border than in cellulitis. However, it can be difficult to distinguish erysipelas from cellulitis clinically. Erysipelas is almost always caused by *Streptococcus pyogenes* which is usually sensitive to oral or intravenous penicillin. Patients with cellulitis or erysipelas may have underlying risk factors which predispose them to infections such as diabetes or immunosuppression. There is often an underlying skin problem that may result in small cracks or breaks in the skin that allow a portal of entry for the organism (eczema/dermatitis, tinea infection, etc.) (see Chap. 30).

## 17.8 Lupus Erythematosus

Discoid Lupus Erythematosus (DLE) and Systemic Lupus Erythematosus (SLE) can cause a characteristic red butterfly rash across the upper cheeks and nose in most cases. (Fig. 17.4) (See Chap. 51). Skin biopsy and blood tests for an anti-nuclear factor may be required to confirm the diagnosis. Most patients respond to topical or oral steroids.



**Fig. 17.4** Discoid lupus erythematosus

### 17.9 Eczema/Dermatitis

Atopic eczema and contact dermatitis (irritant or allergic) may result in a red face. It usually presents with a red, scaly, itchy, eczematous rash. Underlying aggravating factors such as irritation or allergies should be considered. Patients may need allergy testing (IGE and RAST, skin prick test, skin patch test) for severe resistant cases. Treatment involves avoiding irritants and allergens, moisturisers and usually a weak topical steroid or a topical calcineurin inhibitor (see Chap. 13, 14).

### 17.10 Keratosis Pilaris

This is a common condition that often presents in children and young adults. It is autosomal dominant and normally runs in families. It is more common in families with atopic dermatitis. It is caused by a disorder of keratinisation in the upper

part of the hair follicle resulting in follicular plugging. These children have multiple follicular keratotic papules 1–2 mm in diameter which feels rough like sandpaper or “goose bumps”. They usually appear on the outer aspects of the upper arms, the lateral thighs and sometimes on the lateral cheeks. The rash usually has an erythematous base and has a symmetrical distribution. It is usually asymptomatic but causes problems due to cosmetic appearance. It normally resolves in adolescence or early adult life. Exfoliating with sponges and washes can help. Moisturisers with urea, salicylic acid or alphahydroxy acid are sometimes required. More severe cases may respond to topical retinoids [2].

### 17.11 Sunburn and Photosensitive Rashes on Face

Sunburn is usually obvious from the history with redness, burning or soreness of the skin after light exposure. However, certain conditions and drugs can cause a photosensitivity rash which can cause redness of the face and other exposed area with minimal UVL (see Chap. 49).

### 17.12 Dermatomyositis

This is a very rare condition that normally occurs in the elderly where the patient usually presents with a reddish purple (heliotrope) rash on the face, sometimes with periorbital oedema. They also can have papules on the backs of the fingers and hands (Gottron's papules). There may be telangiectasia around the cuticles which are usually thickened and ragged. There is usually associated proximal muscle weakness (myositis). Approximately one third of cases can have an underlying malignancy, particularly in the elderly and this can present before, during or after the rash and muscle weakness. The most common cancers found are lung, breast and female genital tract cancers. The mortality with dermatomyositis is 25% (see Chap. 51).

### 17.13 Conclusion

Rosacea is one of the most common causes of a red face especially in people of Celtic descent. However, many other common conditions can present as a red face including seborrhoeic dermatitis, solar damage, telangiectasia, flushing, acne, dermatitis, psoriasis, skin infections and lupus.

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# Papulo-Pustular Rashes on the Face

18

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## Key Points

- While acne is by far the most common cause of papulo-pustular rash on the face, there are many other causes.
- Close examination with magnifying lenses and a good light will help in the diagnosis.
- A careful history and a thorough physical examination will diagnose most papulo-pustular rashes on the face.
- Biopsies and blood tests are usually not necessary.

## What to Tell the Patient

- Most papulo-pustular rashes on the face can be cleared with a few weeks of either topical or oral therapies.
- Topical steroids should be avoided in all papulo-pustular rashes on the face.
- Many conditions that cause a red face can be aggravated by excess ultraviolet light.
- Cosmetics can camouflage redness of the face and are usually safe on most skin conditions.

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## 18.1 Introduction

Papules (pimples) and pustules (spots) are common on the face. By far the most common cause is *acne* especially in teenagers and young adults. The cardinal sign of acne are open and closed comedones (blackheads and whiteheads) which are usually associated with oily skin, although sometimes these can be difficult to see unless the skin is examined closely with good light and magnification (micro-comedones). Acne can also occur on the chest and back (see Chaps. 7 and 8).

## 18.2 Rosacea

The next most common papulo-pustular rash on the face is rosacea. This is usually associated with redness from broken veins and flushing. There are no comedones and the rash is usually confined to the face in adults. It is far more common in people with Celtic ancestry. Too much UVL and steroids aggravate rosacea (See Chap. 10).

## 18.3 Perioral Dermatitis (Periorificial Dermatitis)

The name is a misnomer as the rash is usually made up of multiple minute papules that coalesce to form a red rash around the mouth (perioral). It

is more like acne than dermatitis and often responds to acne therapies such as topical or oral antibiotics. Unlike perioral eczema, with perioral dermatitis there is normally a rim of uninvolved skin between the rash and the vermillion border of the lip (Fig. 18.1). The rash can be asymmetrical and can occur on only one side of the upper lip or chin or any combination of these areas (Fig. 18.2). More severe cases can spread all the way around the mouth. Some patients can have a micro-papular rash below and lateral to the lower eyelid on one or both sides (periocular dermatitis) (Fig. 18.3). This can occur together with perioral dermatitis or in isolation. Hence the name *periorificial dermatitis*.

Perioral dermatitis almost always occurs in young adult women but can also occur in young girls. There is often a history of using topical steroids or inhaled steroids with a mask. These need to be stopped with the risk of a rebound flare and worsening of the rash in the first 2 weeks; the



**Fig. 18.1** Periorificial dermatitis in a 51-year-old woman



**Fig. 18.2** Perioral dermatitis in a 42-year-old woman who was putting betamethasone valerate on her face



**Fig. 18.3** Periocular dermatitis (around the eyelids) in a female who was putting betamethasone valerate on her face

patient needs to be warned about this possibility from the outset. They can either stop the steroid immediately (cold turkey) or wean themselves from a potent to a moderately potent and then a mildly potent topical steroid over the course of a few weeks. Alternatively, the frequency of application of the topical steroid can be reduced gradually over 2 weeks.

Treatment is usually with oral antibiotics that have a strong anti inflammatory effect such as lymecycline, doxycycline or a erythromycin for 1–2 months in a dose similar to the one used for acne. Milder cases may respond to topical antibiotics such as erythromycin, clindamycin or metronidazole gel. Other topical agents that have been tried include azelaic acid (“Skinorin®”) or topical calcineurin inhibitors (tacrolimus).

## 18.4 Steroid Rosacea

Using potent topical steroids on the face over a number of weeks or months can lead to steroid damage (thinning and redness of the skin) and sometimes steroid rosacea. This can look and behave like classical rosacea but normally clears up once the steroid is stopped. Steroids are probably best stopped gradually as sudden withdrawal will result in a severe flare of the steroid rosacea. As the steroid is gradually reduced in potency and frequency of application over a few weeks, topical and oral rosacea therapies should be commenced and continued until all papules and pustules are cleared (see Chap. 10).

## 18.5 Folliculitis

This can cause papules and pustules affecting the hair follicles most commonly on the beard and moustache area in men (*Sycosis barbae*). Its distribution on the hairy parts of the face only and the lack of redness and comedones should help confirm the diagnosis. Swabs should be taken from a freshly ruptured pustule for culture and sensitivity. The most common organism is *Staphylococcus aureus* which is normally responsive to topical antibiotics such as fusidic acid 2% or oral flucloxacillin for 1–2 weeks. There may be underlying predisposing factors such as diabetes, topical or oral steroids or immunosuppression. Growing a beard may help prevent relapse. Chronic folliculitis may need longer courses of oral and topical antibiotics, combined with topical antiseptic washes (e.g. “Hibiscrub®”) or baths (e.g. “Milton®” baths). If nasal carriage of *Staph aureus* is found, it may need to be eradicated with topical nasal antibiotics for a few weeks. Using a topical antibiotic after shaving may help.

## 18.6 Gram Negative Folliculitis

This is a relatively rare complication of a prolonged course of oral antibiotics for acne or rosacea such as doxycycline or minocycline. It usually presents as a sudden flare of pustules and occasionally cysts on the cheek, chin and paranasal areas during a course of oral acne therapy. It is thought to be caused by tetracyclines or other antibiotics altering the normal flora on the skin, allowing gram negative bacteria to proliferate. Diagnosis is by isolating gram negative organisms on a swab. Various organisms can be identified including *E. coli*, *Klebsiella pneumonia*, *Proteus*, *Pseudomonas*, etc. Treatment usually requires an antibiotic which is effective against gram negative organisms such as amoxicilline or trimethoprim but culture and sensitivity should help identify the most appropriate antibiotic. More severe cases may need treatment with oral isotretinoin.

## 18.7 Pseudofolliculitis Barbae (Razor Bumps, Shaving Rash)

This normally occurs on the side of the neck but can sometimes extend up onto the chin and jaw line in adult males. It is caused by hairs growing out of the skin at an acute angle and then curling back into the skin causing a foreign body type reaction. It is best managed by growing a beard or laser hair removal (See Chap. 36 under beard rashes). For men that require to shave, using a beard trimmer set at 0.5–1 mm, shaving in the direction of the hair, using a single blade razor and shaving after a warm shower to soften the beard may help.

## 18.8 Tinea Barbae

Fungal infections on the face are rare but can occur on the beard area in farmers and if left untreated or if inadvertently treated with oral or potent topical steroids, can become more inflammatory and pustular. It is usually asymmetrical and can be diagnosed by taking skin scrapings or plucking hairs and sending them for fungal stain and culture. They are usually caused by *T. verrucosum* (from cattle) or *T. mentagrophytes* (from horses). Treatment is usually with a 4–6 week course of an oral anti-fungal such as terbinafine or itraconazole.

Another fungal condition is folliculitis due to *Pityrosporum ovale*. This type of folliculitis produces chronic, red and itchy pustules on the areas commonly affected by pytiriasis versicolor: back and chest, neck, shoulders, upper arms and face. It usually responds to topical or oral anti yeast medication (see Chap. 31).

## 18.9 Drug Induced Acneiform Eruptions

The sudden onset of an acne like eruption on the face and upper trunk or worsening of existing acne on the introduction of a new drug should raise the possibility of a drug induced acneiform

**Table 18.1** Drugs that may cause acneiform eruptions or aggravate existing acne

Corticosteroids
Androgenic hormones
Oestrogen (e.g. a low dose oestrogen combined pill may aggravate acne)
Progesterone (e.g. = Progesterone only pill, "Implanon®", "Mirena IUD®")
Lithium
Danazol
Phenobarbitone
Phenytoin
Haloperidol
Isoniazid
Rifampicin
Disulfiram
Vitamin B2, B6 or B12
EGF receptor antagonists (a chemotherapy drug)

eruption. There are usually no comedones. Table 18.1 lists the most commonly offending drugs. The rash usually responds to withdrawal of the drug and treating the rash with topical anti acne treatments such as benzoyl peroxides and for more severe cases adding in an oral anti acne treatment such as lymecycline.

Rare causes of papulo-pustular looking eruptions on the face include molluscum contagiosum, Darier disease and the facial angiofibromas of tuberous sclerosis. Although these conditions do not cause typical papules and pustules, they

can sometimes resemble them. They are usually removed surgically if required for cosmetic reasons.

## 18.10 Pyoderma Faciale

This is a severe papulo-pustular eruption on the face that usually occurs in young women. Although it can resemble severe acne or severe rosacea, it appears to be a separate condition. It begins abruptly, there are little or no comedones, it is confined to the face and there are no eye symptoms. It usually causes nodules and cysts which are painful and can leave permanent scars. Swabs are usually sterile. It usually responds to high dose anti-acne therapy such as lymecycline or trimethoprim for 3–6 months. More severe cases may require oral isotretinoin. Large cysts may have to be aspirated and injected with intra-lesional steroids (see Chap. 7).

## 18.11 Conclusions

Papules and pustules do not always imply infection. Many pustular rashes on the face are due to inflammatory conditions (acne, rosacea, peri-orificial dermatitis, etc.) rather than the infectious skin conditions (folliculitis, carbuncles, fungal disease, etc.).



# Lichen Planus (LP)

19

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## Key Points

- Lichen Planus (LP) is considered an inflammatory, T-cell mediated, autoimmune disease of unknown origin.
- LP usually presents as red, purple or violet, scaly, itchy papules and plaques of various shapes and sizes. It can affect the skin, scalp, nails and/or mucous membranes (mouth, conjunctiva, penis, vagina or peri-anal skin).
- LP commonly presents in adults on the flexural surfaces of the wrists and ankles and often affects the low back.
- LP is usually diagnosed histologically by a skin biopsy. There are no underlying haematological markers.
- Treatment of LP is usually with potent topical or oral steroids.

## What to Tell the Patient

- LP can be itchy and unsightly but it is not infectious, contagious or cancerous.
- The cause is unknown but it is thought to be an autoimmune disease.
- LP usually responds to topical or oral steroids and most cases will burn themselves out eventually.

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## 19.1 Introduction

LP is an unusual rash in general practice. It can present in a number of ways and can affect the skin, hair, nails and/or mucous membranes. It occurs in 0.1–4% of the general population, most often in peri-menopausal women [1].

## 19.2 Clinical Features and Diagnosis

The classical clinical presentation of LP is red, purple or violet, scaly papules and plaques of various shapes (polygonal, linear or annular) and size (from a few millimetres up to many centimetres) (Fig. 19.1). There is usually intense itch but in some mild cases there may be little or no itch (Table 19.1) [1]. The plaques are usually shiny, scaly, flat-topped and firm on palpation, and they most commonly present in adults on the flexural surfaces of the wrists and ankles and often affect the low back (Figs. 19.2 and 19.3). There may be a reticular (netlike) network of fine white lines over the rash called Wickham striae. Some cases can be hypertrophic and occasionally blistering occurs. As the rash clears, either spontaneously or with treatment, it leaves greyish-brown post-



**Fig. 19.1** Lichen planus with no itch



**Fig. 19.2** Lichen planus in a patient who also has type 2 Diabetes. She had no itch

**Table 19.1** The Six P's of Lichen Planus

<b>Planar</b> (flat-topped)
<b>Purple</b>
<b>Polymorphic</b> (various different shapes)
<b>Pruritic</b> (itchy)
<b>Papules</b> (palpable elevation less than 1 cm)
<b>Plaques</b> (a palpable flat lesion greater than 1 cm)

inflammatory macules that may take months to fade.

It can become more generalised and can also affect the scalp, nails and mucous membranes. In the mouth it can cause a fine, white, “lace curtain” type reticular network affecting the inner cheeks (Wickham striae) (Figs. 19.4 and 19.5). Although asymptomatic, it is useful as a clue to the diagnosis.

LP can rarely cause gingivitis, cheilitis and painful erosions of the oral mucosa and tongue which can make eating very difficult (“eruptive LP”) (Fig. 19.6). LP of the mouth can occur in isolation without involvement of any other site. LP can also affect the conjunctivae, lacrimal glands, eyelids, peri anal mucosa, the vagina and the penis, causing an itchy and at sometimes a painful rash. Severe LP in the peri-anal and genital skin can lead to erosion (**eruptive lichen planus**), scarring and adhesions.

When LP affects the scalp, it can cause a scarring alopecia (**Lichen planopilaris**), which when established, is permanent, so early diagnosis and treatment in this area is vital. LP of the nails can cause a number of classical changes which can affect only one or two nails or all of



**Fig. 19.3** Lichen planus that was itchy

the nails. The most common changes are thinning of the nails which may become grooved (Fig. 19.7) and ridged, onycholysis (lifting of the distal nail from the nail bed) and destroyed or elongation of the cuticle (pterygium formation) (Fig. 19.8). Sometimes a nail may stop growing altogether and be replaced by thickened, scarred skin.

Dermoscopy can reveal small pinpoint vessels associated with whitish striations with an erythematous background. In the alopecic scalp areas, perifollicular whitish-gray scaling is associated with erythema, tree-like vessels, loss of follicular openings in addition to follicular plugging. Histologically, LP is quite characteristic and direct immunofluorescence study may reveal globular deposits of immunoglobulin M (IgM) and complement mixed with apoptotic keratinocytes. It has no underlying haematological markers.



**Fig. 19.4** Wickham striae in a patient who also had LP on the arms



**Fig. 19.6** Erosive lichen planus in the mouth in a 16-year-old patient. He also had nails LP



**Fig. 19.5** Oral LP



**Fig. 19.7** Lichen planus of the nails causing a linear ridge. This patient also had oral LP

### 19.3 Differential Diagnosis

Clinically, LP can be confused with other scaly, itchy dermatoses such as scabies, atopic dermatitis, lichen simplex chronicus (neurodermatitis), pityriasis rosea, prurigo nodularis, granuloma annulare and psoriasis. When it affects the scalp it can be confused with other causes of scarring alopecia such as lupus, frontal fibrosing alopecia, tinea or a superficial skin cancer such as superficial BCC. LP affecting the mouth has to be distinguished from other painful, erosive diseases affecting the mouth such as a drug eruption, contact allergic dermatitis, Behcet diseases or herpes stomatitis. LP affecting the peri-anal and genital areas can be confused with other itchy or painful conditions in these areas including pruritus ani and vulvae, seborrhoeic dermatitis, flexural psoriasis, candidiasis, intertrigo, tinea curis and lichen sclerosis. LP affecting the nails may be confused with psoriasis and nail clippings may have to be sent for fungal stain and culture to rule out a fungal nail infection.

### 19.4 Pathophysiology

The pathophysiology of LP is poorly understood. It is considered an inflammatory, T-cell mediated, autoimmune disease of unknown origin. There is usually no obvious trigger, although sometimes drugs may be implicated (lichenoid drug eruption = e.g. gold, captopril) (Fig. 19.9). Quinine and thiazide diuretics can cause a photosensitive lichenoid drug eruption. Some studies have found a statistically significant association between hepatitis C virus (HCV) infection and lichen planus, [2] although there is no known explanation for this association. Patients with LP should be screened for Hepatitis C.

LP can display the isomorphic response (Koebnerisation) whereby the rash appears in areas where the skin has been traumatised (e.g. scratched or burnt). LP can appear at any age, but most cases occur between 30 and 60 years of age. Most cases last a few months but oral LP can last for years. Relapse is not uncommon. There are



**Fig. 19.8** Lichen planus of the nails. Pterygium with scarring of the cuticle and dystrophy of nail



**Fig. 19.9** Lichenoid drug eruption

usually no underlying systemic problems. Longstanding erosive lichen planus can cause squamous cell carcinoma of the mouth, vulva or penis. This should be suspected if there is an enlarging nodule or a painful ulcer. Oral cancer is more common in smokers, and in those who carry oncogenic forms of the human papilloma (wart) virus (HPV).

## 19.5 Treatment

Treatment of LP is usually with topical or oral steroids. For a generalised and extensive first presentation, a short cycle of oral corticosteroids over 3 to 6 weeks can reduce the duration and sometimes the possibility of recurrence. Milder cases affecting the skin should respond to a potent topical steroid. More resistant cases affect-

ing the body may require a super potent topical steroid. Scalp or nail involvement may respond to interlesional steroid injections. LP affecting the face and mucous membranes should be treated with a weak topical steroid (e.g. 1% Hydrocortisone) or a topical calcineurin inhibitor (e.g. “Protopic®”), although TCIs are not licensed for this condition. Steroid oral mouth washes may help oral LP. Some patients may require a 3–6 weeks course of oral steroids starting at 30–80 mg/day, followed by a gradual tapering of the dose over the next 4–6 weeks until finished [3]. Resistant LP, especially if causing severe symptoms or permanent destruction of the scalp bearing skin or nails, should be referred to a dermatologist for more potent systemic treatments such as methotrexate, an oral retinoid such as acitretin, or hydroxychloroquine. Some cases respond to phototherapy (UVB or PUVA).

## 19.6 Conclusion

LP can cause a mild, itchy rash that can be easily diagnosed by clinical, dermoscopic evaluation and confirmed with a simple skin biopsy and managed safely with potent topical steroids. When severe, inflammation of the skin and/or mucous membranes is present it can lead to serious disruption in the patient’s quality of life and perhaps permanent scarring of the scalp, conjunctiva, genitalia or nails. Severe cases may require systemic treatment with oral steroids or referral to a dermatologist for methotrexate, acitretin, hydroxychloroquine or phototherapy.

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## **Part IV**

### **Urticaria, Erythema and Vesiculobullous Disease**



# Urticaria

20

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## Key Points

- Urticaria is a predominately IgE mediated reaction with a release of vasoactive agents, particularly histamine, that causes a generalised, itchy rash.
- Acute urticaria last less than 6 weeks and is often triggered by an upper respiratory tract infection or a new drug (e.g. aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), antibiotic) in children and adults.
- Chronic urticaria last more than 6 weeks and the most common identifiable cause is drugs. However, in up to 50% of cases no cause can be found. Most of these cases are considered autoimmune in origin.

- If you have urticaria, do not take any drugs with aspirin or NSAIDs (Nonsteroidal anti-inflammatory drugs).
- Urticaria may cause swelling of the lips but this is not usually life threatening.

## 20.1 Introduction

Urticaria is a common, distressing, itchy skin condition which can be either acute or chronic. It is more common in people with a personal or family history of atopy or allergies. There are many different forms and numerous possible triggers, but fortunately most cases will respond to the new generation oral anti histamines.

## What to Tell the Patient

- While the itch and rash in urticaria is distressing, most cases will respond to one of the new generation, non sedating oral anti histamines.
- Sometimes the new generation oral anti histamines have to be used at higher than the standard dose.
- Please let your doctor know if you have started any new prescribed or over the counter drug (oral, topical, patches, implants, IUD, etc.) in the few weeks or months prior to getting your rash.

## 20.2 Clinical Features and Diagnosis

The rash in urticaria usually lasts less than 24 hours in any one part of the body and clears without leaving a trace behind. Some patients can have daily symptoms with the rash coming and going on various parts of the body in an asymmetrical fashion. The classical features of urticaria are the “wheal and flare”; a nettle or hive like itchy rash as a result of dermal oedema. The wheals are well-defined lesions with a smooth surface and no scaling. They may be red or white in colour surrounded by a red or white

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flare. They can vary from a few millimetres up to many centimetres in diameter. Sometimes the patient may have no rash on presentation but the classical description of a transient, itchy nettle or hive type rash that fades completely within 24 hours is usually sufficient to make the diagnosis (Table 20.1). A photo of the rash can be very helpful at making the diagnosis (Fig. 20.1).

Many patients with urticaria display **dermatographism**, a rash provoked by scratching on the skin (“writing on the skin”) (Fig. 20.2). This can be a useful sign if the patient has no rash on the day they come to the doctor. Some poursa-

**Table 20.1** History Taking in urticaria

- Is there a typical wheal-flare reaction with individual lesions lasting less than 24 hours?
- Is the rash going on for more than 6 weeks?
- Are there any physical provoking factors?
- Drug history (including over-the-counter medications)?
- Food triggers?
- Family history of angio-oedema?
- Is there arthralgia or myalgia (autoimmune?)



**Fig. 20.1** Acute Urticaria



**Fig. 20.2** Dermatographism

tients with urticaria may also develop swelling of the lips and mucous membranes (**angio-oedema**). This can be quite upsetting for the patient as it looks like they got a punch in the face, but rarely causes problems such as respiratory distress.

Urticaria can occur at any age but the peak age in adults is between the ages of twenty and forty. It is slightly more common in females. Acute urticaria is much more common in children and young adults and is frequently the result of a viral infection, NSAIDs or after eating too much fruit or coloured sweets.

Classical generalised urticaria with the typical wheal-flare type rash can be divided into **acute urticaria**, which last *less than 6 weeks* and chronic urticaria which lasts more than 6 weeks but can often last months or years. Acute urticaria can be provoked by simple viral infections such as an upper respiratory tract infection (URTI) or tonsillitis. Other common causes, particularly in children, are medications, both prescribed and over-the-counter medication (e.g.; aspirin or NSAIDs). Fortunately, acute urticaria responds quickly to the new generation, non-sedating, oral antihistamines either in syrup or tablet form. The older, first generation, sedating antihistamines such as chlorphenamine maleate 4 mg (“Piriton”®) or promethazine (“Phenegran”®) can be useful when given at night if the itch is keeping the patient awake, by virtue of the sedating effects.

**Chronic urticaria** can be more difficult to manage. Some patients can have classical chronic urticaria for months or years, where a careful history will not identify a trigger. This condition can

be very distressing for the patient as they can have chronic itch on a daily basis and sometimes unpredictable disfiguring swelling of the lips. These patients may have an underlying allergic or autoimmune condition and require a detailed workup (Table 20.2) especially if they have arthralgia or myalgia with the rash.

**Cholinergic urticaria** commonly occurs when the body heats up, such as after exercise. This can make going to the gym or running very uncomfortable or perhaps impossible for some patients. Taking an antihistamine before sports may help, although this particular type of urticaria is often unresponsive to antihistamines.

Some patients develop **pressure urticaria** especially on the soles of their feet, the palms of their hands or on their buttocks, knees or elbows. This can make manual labour or even walking difficult. Hot or cold water can provoke an urticarial reaction in some patients and going for a shower or a swim can be a problem.

C1 esterase inhibitor and complement levels (C3 and C4) should be checked if the patient has chronic angio-oedema *without* urticaria or if there is a family history of angio-oedema (hereditary angioedema). If the C4 is low but the C1 esterase inhibitor level is normal then a functional assay of C1 esterase inhibitor needs to be done. For this to be accurate the blood sample has to arrive fresh and processed quickly in the lab and is best done in specialised centres.

**Table 20.2** Investigations to Consider for Chronic Idiopathic Urticaria

- Full blood count, erythrocyte sedimentation rate, C reactive protein
- Urea and electrolytes, liver function tests
- Thyroid function tests including T3, T4 and thyroid antibodies
- ANF and auto antibody screen
- Coeliac antibodies
- IgE and RAST (food screen)hepatitis B and C screening
- MSU
- CXR

*Consider the following in more chronic, refractory cases:*

- H. Pylori C.urea breath test
- Stool for O + P
- Skin biopsy if vasculitis suspected
- C1 esterase inhibitor + compliment levels (C3 + C4) if chronic angio-oedema without urticaria or a family history of angio-oedema

Fortunately, **C1 esterase inhibitor deficiency**, which is an autosomal dominant inherited disorder, is extremely rare but death may occur from laryngeal obstruction. Hereditary angioedema does not respond to antihistamines, adrenaline or corticosteroids. Treatment of acute attacks usually requires C1-esterase inhibitor (human or recombinant) replacement therapy by infusion. Other treatment options include icatibant, a bradykinin B2 receptor blocker, or ecallantide, a novel kallikrein inhibitor. Patients in an acute attack may need management in ICU with airway management by intubation or tracheostomy and iv fluids. Anabolic steroids (e.g., danazol) or tranexamic acid may help prevent attacks.

## 20.3 Differential Diagnosis

Urticaria is caused by sub epidermal inflammation and oedema but unlike eczema/dermatitis or scabies, there is no dryness, scaling, fissuring or scarring. Medical conditions that can cause generalised pruritus such as diabetes mellitus, chronic renal insufficiency, primary biliary cirrhosis, or other nonurticarial dermatologic disorders may be confused with urticaria [1, 2]. These conditions are not usually associated with a rash but there may be secondary features from scratching such as excoriation and scabbing.

The subepidermal autoimmune blistering diseases such as bullous pemphigoid, pemphigoid gestationis (pemphigoid associated with pregnancy), or linear IgA bullous dermatosis may initially present with urticarial lesions many days or weeks before blisters appear.

Insect bites may cause a papular eruption (**papular urticaria**) like hives in some individuals, especially children. Unlike true urticaria, the papules last days rather than hours and some of the papules may have an identifiable bite or puncture mark in the middle. Papules are most commonly seen on the exposed skin such as the legs, the arms and the face. Bed bugs (human flea, *Pulex irritans*) may get under night clothes and cause a papular urticaria on any part of the body. Sedating oral antihistamines, topical steroids, insect repellent sprays, house cleaning proce-

dures and insecticides should help to avoid further bites.

Angio-oedema has to be distinguished from **anaphylaxis** where the patient develops a sudden catastrophic, generalised, allergic reaction to a variety of substances such as drugs or food where they develop a rash, tachycardia, flushing, hypotension and severe respiratory distress (Table 20.3). This is a medical emergency and the patient should be treated with intramuscular adrenaline, oxygen, intravenous fluids and hospital transfer. Thankfully, patients with angio-oedema rarely develop anaphylaxis.

In some patients the wheal-flare reaction can last greater than 24 hours in any one area of the body and can sometimes leave some bruising. This may be a sign of **urticular vasculitis** usually requiring referral to a skin specialist for skin biopsy and further management (see Chap. 54). Most cases are of unknown cause although it may occasionally be provoked by infections, drugs or an underlying systemic disease such as SLE or a malignancy. A skin biopsy is diagnostic. Antihistamines are unhelpful but topical steroids, systemic steroids or dapsone may provide relief.

**Urticaria pigmentosa** (mastocytoma) is primarily a disease of childhood in which multiple, reddish-brown hyperpigmented maculopapular lesions are found, mainly on the trunk and limbs and may stay for many months or years (Figs. 20.3 and 20.4). Most cases resolve spontaneously. A skin biopsy is diagnostic and shows tightly packed aggregates of mast cells. The lesions are

not particularly itchy but mild friction or trauma will cause them to urticate due to histamine release (Darier's sign). Rare adult cases should be screened for systemic involvement or leukemic transformation.

**Erythema multiforme** (EM) may be confused with urticaria. EM usually presents with pathognomonic target or iris lesions with raised, edematous "bulls eye" type papules which usually begin on the extremities and often involve one or more mucous membranes. It is often precipitated by viral infections (e.g. herpes simplex) or drugs. The target lesions usually last up to 7 days and then fade spontaneously. Attacks are sometimes recurrent (e.g. with every attack of "cold sores"). EM is considered to be a type IV hypersensitivity reaction associated with certain infections, medications, and other various triggers. (see Chap. 22)

## 20.4 Pathophysiology

Urticaria is an IgE mediated reaction from the release of vaso active agents, particularly histamine, that causes a generalised itchy rash. In up to 50% of patients with chronic urticaria no trigger can be found. Most of these are likely to be due to an auto immune reaction. Positive antibodies, especially to anti thyroid antibodies, are sometimes found and may be a marker for auto immune diseases.

**Table 20.3** How to distinguish urticaria and angio-oedema from anaphylaxis

	Urticaria + angio-oedema	Anaphylaxis
Onset	Gradual (minutes to hours)	Delayed (5–30 min)
Skin	Wheal and flare + lip swelling	Flushed, swelling urticaria
Respiratory	Normal	Wheeze
Cardiovascular	Normal	Tachycardia
Neurological	Normal although there may be some anxiety if there is lip swelling	Weak, faint or loss of consciousness. Little response to lying flat.
Treatment	Non sedating antihistamines (may have to give higher doses)	Lie flat, head down, legs up. IV fluids—Crystallloid <i>IM adrenaline 1:1000</i> – adult = 0.5 ml – child 6–12 yo = 0.3 ml – child <6 yo = 0.15 ml



**Fig. 20.3** Urticaria pigmentosa in a child



**Fig. 20.4** Urticaria pigmentosa in an adult

Drugs are the most common known trigger. Simple over-the-counter medication such as aspirin and non-steroidal anti-inflammatories are the most common drug causes. A careful drug history including asking about over-the-counter medications such as vitamins, tonics, cough bottles, painkillers, laxatives and herbal medicine should be taken. Almost all drugs can cause urticaria but Table 20.4 lists the most common offenders. The urticarial rash may occur immediately on starting the drug or may develop months or years after being on the offending drug (e.g. ACE inhibitors or the combined oral contraceptive pill). There are many types of chronic urticaria that can be provoked by various physical and chemical factors (Table 20.5). Taking a careful history should help to identify the physical causes in most cases.

## 20.5 Treatment

Most patients with chronic urticaria respond to the new generation, non-sedating oral antihistamines but may require daily doses and sometimes in much higher doses than is normally prescribed (up to four times the standard daily dose given BD). Alternatively, two different non-sedating

**Table 20.4** Some of the more common drugs that can provoke urticaria

- Aspirin
- NSAIDs
- ACE inhibitors
- Oral contraceptive pill
- Opiates including codeine
- Statins
- Radio-contrast media

**Table 20.5** Types of Physical Urticaria

Dermatographism	“Writing on the skin”
Delayed pressure urticaria	E.g. Palms and soles
Cholinergic urticaria	Exercise induced
Heat/cold urticaria	Not always responsive to antihistamines
Aquagenic urticaria	Water provoked
Solar urticaria	UVR provoked
Contact urticaria	E.g. Nettles, rubber, etc.

antihistamines may be given every 12 hours. Very high dose antihistamines should be avoided in patients with cardiac disease or arrhythmias. Trying different types of non-sedating antihistamines in rotation may be helpful, as some patients will respond better to one particular type than another. If the patient is being kept awake at night with itch, one of the old fashioned, oral sedating antihistamine (e.g. Promethazine Hydrochloride 25 mg × one or two) can be added in at bedtime. Doxepin is a potent H<sub>1</sub> (and H<sub>2</sub> receptor) receptor antagonist and is effective in some patients with chronic urticaria at 10 mg three times daily or 25 mg at night [3]. Patients should be warned about the possibility that sedating antihistamines and doxepin may interfere with driving or operating dangerous machinery if excessive drowsiness occurs.

Some patients respond to mast cell stabilisers such as Montelukast, 10 mg at night. Topical and oral steroids are rarely necessary for acute simple urticaria as it usually responds well to oral antihistamines, which are a lot safer for long-term use. A short course of oral steroids combined with a non sedating antihistamine can be helpful in settling an acute attack of angio-oedema associate with urticaria, especially if the reaction was to occur at critical times such as before a wedding, on holidays or before an interview. Adrenaline auto injectors are rarely required for recurrent severe angioedema with upper airways obstruction.

Most cases of chronic urticaria will burn themselves out in time. Patients may require daily doses of antihistamines to keep them comfortable until this happens, which can sometimes take months or years. If an underlying autoimmune condition is suspected, consider referring the patient to an immunologist if the rash is difficult to control with conventional therapies.

Most patients with chronic urticaria will be convinced that they must have an underlying food allergy and this is often reinforced by pseudo-allergy testing, carried out by alternative practitioners. In practice, food allergies rarely cause urticaria and when it does happen, it can

usually be identified by careful dietary history. In chronic resistant idiopathic urticaria it may be worth doing some food allergy tests such as IgE and RAST to foods and coeliac antibody test, if only to reassure the patient that they do not have an underlying food allergy. Sometimes it is worth trying the patient on a low salicylate diet, especially if they give a history of allergy to aspirin. Others may respond to a diet free of colorings and preservatives or a low histamine diet [4] (Table 20.6). These restrictive diets should be continued for only 6 weeks and if there is no response in that time, the patient should be put back on the normal diet.

Some patients may have urticaria associated with *H. pylori* in their stomach. If chronic idiopathic urticaria is difficult to control it may be worth considering doing a C urea breath test or a gastroscopy for *H. pylori*. If found, the triple therapy will eradicate the *H. pylori* and may help reducing the urticaria.

Antifibrinolytic agents such as tranexamic acid ("Cyklokapron®") or androgenic steroids such as danazol may be helpful in resistant chronic idiopathic urticaria. High dose vitamin D supplementation regardless of the patients vitamin D status has been reported to be helpful in

**Table 20.6** Low histamine diet: foods and drinks that are particularly high in vasoactive amines

- Wine, beer, cider, champagne
- Coffee, cocoa, chocolate
- Fermented soya products including miso and tempeh
- Blue cheeses, parmesan cheese, camembert, emmental, old gouda, cheddar and other hard cheeses, fresh and hard sheep and goat cheeses
- Cured meat especially pork products e.g. sausages and other processed meats (ham, salami, pepperoni, bacon)
- Fresh or canned tuna, sardines, mackerel, salmon, herring
- Processed fish products, e.g. fish pastes
- Smoked or dried pickled fish
- Tomatoes, pickled cabbage (sauerkraut), broad beans, aubergine, spinach
- Peanuts, tree nuts
- Oranges, tangerines, bananas, pineapple, grapes, strawberries

some cases [5]. Dapsone has been shown to be useful as a second line treatment in some cases of chronic spontaneous urticaria [6]. Recent research has shown that omalizumab (“Xolair”®), an anti-IgE monoclonal antibody that targets IgE and affects mast cells and basophilic function which is licensed for severe asthma, will help some people with chronic resistant idiopathic urticaria [7] (Table 20.6).

## 20.6 Conclusion

Urticaria causes in an itchy, hive like, fleeting rash that stays in one area for a few minutes to a few hours, then fades completely only to arise in another area of the body a few hours or a few days later. The rash can recur for days, months or years. The cause of the rash is often not clear. Sometimes there are obvious triggers (e.g. drugs or infections) but in many cases no cause can be found and most cases of chronic idiopathic urticaria are probably auto-immune in origin. Some people also develop swelling of the lips, tongue or other areas (angio oedema). The symptoms of urticaria can often be eased with new generation, non sedating antihistamine tablets but in some cases double or quadruple the normal dose is required to control the rash [8].

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# Allergic Skin Disorders

21

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## Key points

- A careful history and a thorough physical examination will help identify the allergen in many cases.
- Allergy testing may be required to confirm what is suspected from the history, to identify hidden allergens or to exclude a false diagnosis of a specific allergy by alternative practitioners.
- Allergy tests performed by doctors are not 100% sensitive or specific. False positives and false negatives can occur.

## What to tell the patient

- Mild to moderate atopic eczema may not be allergic in origin.
- Young children with severe, unresponsive, atopic eczema may benefit from an allergy work up to see if there are any significant and avoidable underlying allergies responsible for their itch.
- Food allergies are unusual in adults and rare in the elderly. Drug allergies are far more common in the elderly.
- Allergy test carried out by alternative practitioners are usually neither accurate nor reliable.

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## 21.1 Introduction

There is a number of skin diseases that are known to have an allergic component in their aetiology, including urticaria, atopic eczema, anaphylaxis, allergic contact dermatitis, dermatitis herpetiformis and fixed drug eruptions. A careful history and a thorough physical examination will help identify the allergen in many cases. Allergy testing may be required to confirm what is suspected from the history or to identify hidden allergens. Validated allergy testing by a doctor may also be necessary to overrule false positive results of dubious allergy testing by alternative practitioners.

Food allergies are more common in children, especially if they have an atopic tendency. They are unusual in adults and rare in the elderly. On the other hand, drug allergies are far more common in the elderly, primarily because they are on a lot more drugs than children or young adults.

## 21.2 Urticaria

Ordinary acute urticaria (see Chap. 20) usually presents as intensely itchy, raised, pink papules like hives or nettle stings which may coalesce into large erythematous plaques, sometimes in an annular pattern (Fig. 21.1). The diagnostic feature is that the rash comes up within a few hours and will resolve without a trace within 24 hours. An irregular blanched wheal appears, surrounded



**Fig. 21.1** Acute urticarial after flucloxacillin

by an area of redness known as the wheal-and-flare reaction. The itch and rash may move from one part of the body to another, sometimes causing daily symptoms that may go on for months or years (chronic idiopathic urticaria). The acute forms of urticaria may be associated with subcutaneous oedema, involving swelling of the eyes, lips, larynx, respiratory or gastrointestinal tract (angioedema).

Urticaria may be provoked by allergic reactions such as a type I or a type III immunological response or non allergic factors which may be triggered by vasoactive or histamine releasing substances. It occurs more often in patients who have a personal or family history of atopic disease. Acute urticaria in children is usually triggered by infections (e.g. upper respiratory tract infection) or drugs (e.g. non-steroidal anti-inflammatory drugs). Occasionally general medical conditions such as chronic sinusitis, UTIs, chronic candidiasis, worm infestation, *H. pylori* GI infection, hepatitis, collagen vascular disease or internal malignancies, may be responsible for provoking an urticarial reaction.

Chronic urticaria (daily symptoms for more than six weeks) can be more troublesome and resistant to treatment. Persistent cases are worth investigating although in many cases no identifiable underlying cause may be found and these cases may be autoimmune in origin. The most

**Table 21.1** Investigations to consider for chronic idiopathic urticaria

- Full blood count, and erythrocyte sedimentation rate
- Urea and electrolytes and liver function tests
- Thyroid Function tests including T3, T4 and thyroid antibodies
- Antinuclear antibodies and auto antibody screen
- Coeliac antibodies
- IgE and RAST (food screen)
- *H. pylori* C. urea breath test
- Midstream urine for culture
- Stool for ova and parasites
- Skin biopsy if vasculitis suspected
- Chest Xray

common identifiable cause of chronic urticaria in adults is drugs. Asprin, NSAIDs, ACE inhibitors and the oral contraceptive pill are the most common offenders. Table 21.1 shows some common investigations of chronic urticaria. Some patients may benefit from being referred for an allergy work-up. A low salicylate diet should be considered, particularly if there is a history of allergy to aspirin. Others may respond to a colouring and preservative free diet or a diet free of histamine releasing foods. These restrictive diets should be abandoned after six weeks if there is no significant improvement (see Chap. 20).

### 21.3 Eczema/Dermatitis

The clinical features of atopic eczema, contact allergic and contact irritant dermatitis may be indistinguishable (see Table 21.2) and more detailed allergy testing such as a patch test may be required in severe resistant cases (see allergy testing below).

The reaction to the various substances with which patients come in contact may be immunologically mediated (allergic contact dermatitis) or merely due to a patient's sensitive skin reacting adversely to irritating substances such as soaps, detergents, or industrial oils (irritant contact dermatitis) (Fig. 21.2). Some patients may have features of both allergic and irritant contact dermatitis. The definitive treatment of contact

**Table 21.2** How to distinguish the various forms of Eczema/Dermatitis

	Atopic eczema (AE) <sup>a</sup>	Allergic Contact Dermatitis (ACD) <sup>a</sup>	Irritant Contact Dermatitis (ICD) <sup>a</sup>
Age	• 20% children • 10% adults	Mostly adults	Mostly adults 80% of CD—irritant
Aetiology	Atopic disease, strong hereditary factors. Filaggrin gene defect → skin barrier defect	Type IV delayed hypersensitivity reaction to an allergen in contact with the skin	Not immunologically mediated. Irritation for detergents and other harsh chemicals
Onset after exposure	Not relevant	8–96 hours	Minutes to hours
Distribution	Mainly flexural	At site of contact (e.g. earlobe, face, hands) may become more generalised	Mostly hands
Clues to diagnosis	Personal or family history atopy. Itchy +++	Reaction to nickel containing earrings	High risk occupation: e.g. hairdressing, kitchen worker, homemaker, healthcare worker, cleaner
Diagnosis	Clinical. IgE + RAST Skin patch test	Patch test	Clinical
Treatment	Emollients (E), Soap substitutes (SS), Topical steroids (TS), Topical Immunomodulators (TIM)	Avoidance of allergens, use emollients (E), Soap substitutes (SS), Topical steroids (TS), Topical Immunomodulators (TIM)	Avoidance of irritants and use barriers (gloves) Emollients (E), Soap substitutes (SS), Topical steroids (TS), Topical Immunomodulators (TIM)

<sup>a</sup>There can be considerable overlap

Ref: adapted from Rashid RS, Shim TN. Contact dermatitis. BMJ. 2016 Jun 30;353:i3299. doi: 10.1136/bmj.i3299. PMID: 27364956.

dermatitis is the identification and avoidance of the underlying causes (irritants or allergens) (Fig. 21.3).

Children with severe unresponsive atopic eczema may benefit from an allergy work up (e.g. IgE + RAST blood test, skin prick test, patch testing or exclusion diets followed by food challenge, etc.) to see if there are any significant and avoidable underlying allergies contributing to their symptoms. Children continuing on a restricted diet should be assessed by a dietitian and reviewed annually as kids sometimes “grow out” of their allergies (see Chaps. 13 and 14).

specific IgE antibodies on mast cells and basophils (type 1 hypersensitivity reaction), triggering the rapid release of histamine and the other vasoactive substances. This cause capillary leakage, mucosal oedema, wheeze, rash and shock. Anaphylactic reactions can vary in severity and rate of progression—they may progress rapidly (over a few minutes) or occur in a biphasic manner. The reaction may be delayed by a few hours and may persist for more than 24 hours.

Anaphylaxis causing severe laryngeal oedema and hypotension is very rare but may cause respiratory distress and should be treated as an emergency with immediate intramuscular adrenaline followed by hydrocortisone and antihistamines. All patients should be transferred to hospital for monitoring for 24 hours.

Patients with a history of anaphylaxis should have a self administered pre loaded adrenaline pen injector (e.g. “Anapen®”, “EpiPen®”) available at home, work and school for emergencies.

## 21.4 Anaphylaxis

This is an acute allergic reaction to an antigen (e.g. a bee sting, drugs, foods, etc.) to which the body has become hypersensitive. An anaphylactic reaction occurs when an allergen reacts with



**Fig. 21.2** Contact Allergic Dermatitis in a dairy farmer with a rash on neck and forearms

They should also wear a medical emergency identification bracelet or chain.

Anaphylaxis should be differentiated from urticaria with angioedema, hereditary angioedema, severe asthmatic attack and a panic attack, which all require different treatments (see Chap. 20).

**Dermatitis herpetiformis** (DH) (see Chap. 23) is a rare, itchy, vesicular eruption which occurs as a result of an abnormal immunological reaction to the gliadin fraction of gluten found in wheat, rye and barley. Eighty percent of patients with dermatitis herpetiformis also have gluten enteropathy which is associated with **coeliac disease**. The rash in DH has a characteristic distribution which may be the only clue to the diagnosis because the small vesicles may be hard to find as they are often scratched away soon after appearing because of intense itch. Histology of a vesicle or fresh rash may show features of DH (subepidermal blisters with neutrophils and eosinophils



**Fig. 21.3** Contact Allergic Dermatitis to "Dettol®" antiseptic

concentrated in the dermal papillae). Direct immunofluorescence reveals IgA immunoglobulin in dermal papillae of the involved skin but this test is only available in specialist labs and special transport medium is necessary to transfer the biopsy specimen.

The patient may or may not have gastrointestinal symptoms. There may be other associated autoimmune conditions (e.g. Type 1 diabetes, vitiligo, alopecia areata, urticaria or thyroid disease). Blood should be checked for coeliac antibodies: immunoglobulin A anti-tissue transglutaminase (IgA tTGA) is the initial screening test and if it is elevated it is confirmed by IgA antiendomysial antibodies (anti EMA). Measurement of serum IgA level is an appropriate next step if IgA based testing is negative and there is a strong clinical suspicion of celiac disease. Where an IgA deficiency exists then IgG based tests should be performed. The definitive test is a duodenal or jejunal biopsy which usually shows subtotal villous atrophy. False negative test results may occur if testing is performed

while the patient is on a gluten free diet. In cases where there are still doubts regarding the diagnosis after serology and histology, HLA-DQ2/DQ8 genotyping should be considered. Such testing has a high negative predictive value meaning those who test negative are very unlikely to develop the disease. Some patients have negative coeliac serology and normal duodenal mucosa but have resolution of symptoms on adherence to a gluten free diet. This is known as “non coeliac gluten sensitivity” (NCGS) [1].

Most cases of DH respond to a gluten free diet which should be initiated and monitored by a dietician. Patients should be encouraged to join their national celiac society where they can get useful tips and advice about diet, where to buy gluten free foods and how to reclaim some of the costs from the national health services or the revenue.

## 21.5 Fixed Drug Eruptions

Fixed drug eruptions occur at the same site(s) each time the offending drug is administered. The lesion is usually solitary, sharply demarcated, reddish brown in colour, circular or oval in shape and occurs within hours of taking the drug. Lesions most commonly occur on the trunk and proximal limbs or on the glans penis. Blistering may occasionally occur. Lesions usually fade within a few days but may leave some post inflammatory pigmentation. The most commonly implicated drugs are phenolphthalein which is contained in many laxatives, sulphonamides, tetracyclines and chlordiazepoxide (“Librium®”). Colouring agents in foods and medications may occasionally be responsible.

## 21.6 Latex Allergy

Natural Rubber Latex (NLR) is sourced from the sap of the *Hevea brasiliensis* tree which is commonly found in South America, Africa and South East Asia. Harvesting latex for rubber tyres became popular in the early 1900's and led to many human rights violations at the time that was

highlighted by Sir Roger Casement, an Irish patriot. Rubber use exploded in the 1980's with the awareness of HIV and hepatitis B transmission, especially in the healthcare profession. Rubber is now very commonly used in medical devices such as examination gloves and catheters and common everyday household items such as household rubber gloves, balloons, condoms and children's soothers (Tables 21.3 and 21.4).

With the increase use of latex came a significant increase in the incidence of latex allergy, especially in healthcare professionals and patients exposed to a lot of latex medical devices such as those with spina bifida.

**Table 21.3** Everyday items that contain latex

- Rubber balloons
- Rubber bands
- Elastic in clothing
- Erasers
- Foam rubber latex mattresses
- Condoms
- Contraceptive caps
- Baby teats/soothers
- Hot water bottles
- Swimming caps and goggles
- Washing up gloves
- Stress balls
- Sports equipment such as hand grips and gym mats
- Tyres

**Table 21.4** Medical equipment that may contain latex

- Examination and surgical gloves
- Oral and nasal airways
- Endotracheal tubes
- Intravenous tubing
- Surgical masks
- Rubber aprons
- Catheters
- Injection ports
- Wound drains
- Dental dams
- Anaesthesia masks
- Syringes
- Stethoscopes
- Tourniquets

There are two actual mechanisms of latex allergy, Type 1 and type 4 allergic reactions:

**Type 1—immediate IgE mediated anaphylactic type reaction** to proteins in the latex. These type 1 reactions are rare but potentially fatal, especially if latex proteins become airborne by binding with starch in powdered surgical gloves. If a person who is allergic to latex proteins inhales the powder, they may develop a severe allergic anaphylactic type reaction especially if they have a history of poorly controlled asthma. Balloons are another source of airborne latex allergens. People who are at risk of these types of reactions need to take great care to avoid latex products, should carry an in-date self-administered adrenaline pen for emergencies and wear a Medi-Alert bracelet. These type 1 reactions are usually diagnosed by an IgE and RAST test to latex protein, a skin prick test or by challenging tests under careful medical supervision.

Symptoms of Type 1 latex allergic reactions usually come on quite rapidly (within minutes) and may cause hives or an urticarial type rash anywhere on the body. More serious symptoms include swelling of the lips, tongue or throat, difficulty breathing, severe asthma, abdominal pain, nausea and vomiting. Extreme cases could cause collapse due to an anaphylactic shock.

Some patients with type 1 latex reactions may develop cross reactivity to certain foods such as kiwis, avocados, bananas and chestnuts. These foods may cause oral symptoms initially such as tingling in the mouth. Patients may progress to more severe allergic reactions to these foods if repeatedly exposed. Patients with a Type 1 allergic reaction to latex should be warned about the possibility of cross reactivity to these foods and avoid them if they cause any symptoms [2].

**Type 4—delayed hyper-sensitivity reactions** to chemicals used in the latex manufacturing process. This usually causes a contact allergic dermatitis between 6 and 48 hours after exposure on the affected skin. These Type 4 reactions are much more common and fortunately less dangerous than the Type 1 reactions. Type 4 reactions can be diagnosed by skin patch test to latex. Strict avoidance of latex containing products is the best way to manage this problem. Nitrile gloves and hypo-allergic condoms are usually safe. If a rash

develops it usually responds to potent topical steroids on the body or 1% hydrocortisone on the face and genitalia. The Latex Allergy Support Group in the UK offer free impartial advice on the commonly used alternatives to latex containing products ([www.lasg.org.uk](http://www.lasg.org.uk)).

## 21.7 Allergy Testing

The most useful allergy ‘test’ is a detailed history, using questionnaires when necessary, as this will give a good indication of whether a rash is allergic in origin and may give some pointers towards the possible etiologic factors. Specific allergic tests may be required to confirm what one suspects from the allergy history or in severe resistant cases where there is a strong suspicion of an underlying abnormal immunological reaction. Allergy testing is more likely to show significant results in children and young adults and less likely in the elderly.

A skin **patch test** is an extremely useful investigation for patients suffering from difficult eczema or dermatitis when one suspects a possible allergy to various products that come in contact with the skin, including cosmetics, toiletries, clothing, jewelry, medicated creams and ointments (Tables 21.5 and 21.6) (Fig. 21.4). Patch tests help identify allergens causing a Type 4, delayed, cell mediated T-lymphocyte reaction. A standard batch of 36 common contact sensitizers (European Standard Series) are placed under patches on the patient’s back for 48 hours and then removed to observe any reactions immediately after removing the patches and two days later. Patch test kits are expensive and results can be difficult to interpret so patients are best referred to colleagues with experience in this technique. TRUE TEST® is made in Denmark and is distributed by [www.diagenics.co.uk](http://www.diagenics.co.uk) is a

**Table 21.5** Indications for skin patch testing include

- Acute or chronic dermatitis if a contact allergy is suspected
- Chronic eczema failing to respond to treatment
- Hand or foot eczema
- Chronic stasis (varicose) eczema
- Chronic or recurrent otitis externa

**Table 21.6** Common allergens tested in the European standard T.R.U.E. Test

1. Nickel sulphate
2. Wool alcohols
3. Neomycin sulphate
4. Potassium dichromate
5. Caine mix
6. Fragrance mix
7. Colophony
8. Paraben mix
9. Negative control
10. Balsam of Peru
11. Ethylenediamine dihydrochloride
12. Cobalt chloride
13. p-tert-Butylphenol formaldehyde resin
14. Epoxy resin
15. Carba mix
16. Black rubber mix
17. Cl + Me-Isothiazolinone
18. Quaternium-15
19. Methylbromo glutaronitrile
20. p-Phenylenediamine
21. Formaldehyde
22. Mercapto mix
23. Thiomersal
24. Thiuram mix
25. Diazolidinyl urea
26. Quinoline mix
27. Tixocortol-21-pivalate
28. Gold sodium thiosulphate
29. Imidazolidinyl urea
30. Budesonide
31. Hydrocortisone-17-butyrat
32. Mercaptobenzothiazole
33. Bacitracin
34. Parthenolide
35. Disperse Blue 106
36. Bronopol

handy patch test system that comes in preloaded chambers which can be stuck straight onto the patients back from the package (Fig. 21.5).

Among the most common sensitisers are metals (nickel, cobalt, chromium), perfumed compounds (fragrances, balsams), colophony (a resin), corticosteroids, preservatives and other ingredients in topical products (Kathon CG, thimerosal, lanolin, ethylenediamine), antibiotics (neomycin), dyes (paraphenylenediamine, azo colours), rubber chemicals (thiurams, mercaptobenzothiazole), formaldehyde, plastic chemicals (epoxy, acrylates, phenolic resin), anaesthetics



**Fig. 21.4** Contact Allergic Dermatitis to PPD in hair die



**Fig. 21.5** Positive patch test results

(benzocain, cinchocain), plant allergens (primin), etc. (Table 21.6). Nickel is the most common contact allergen in women probably from piercing earlobes at a young age. Cobalt is one of the most common allergens in men and is commonly found in rubber, metals and cement.

Special patch test series are available in tertiary referral center for specific occupational dermatitis (e.g. hairdresser series, dental series and photographer series). Patch testing is of no value in investigating urticaria and is difficult to perform in children.

Very rare cases of type 1 hypersensitivity reactions, including anaphylaxis, have been reported during patch testing so full resuscitation facilities should be available [3]. Antihistamines do not need to be stopped during a patch test.

Patients who are prone to contact allergic dermatitis (e.g. develop a rash from cheap earrings = nickel allergic) should be careful when introducing a new skin care product. If they are worried, they may perform an “open patch test”

by applying the new cream to the volar aspect of the forearm twice a day for 1–2 weeks. If an eczematous reaction occurs, they should avoid the product.

A new **atopy patch test** has been developed for patients with atopic eczema which tests for a Type 4 (delayed T-lymphocyte hypersensitivity reaction) to common allergens such as house dust mite, grass pollen, cat dander, cow’s milk, hen eggs, wheat and soy products. Compared to the skin prick tests and IgE and RAST tests (which test for type 1, IgE mediated hypersensitivity reactions), the atopy patch test appears to be more specific but less sensitive [4].

A **skin prick test** helps identify immediate, Type 1 IgE mediated allergic reactions and is useful when looking for airborne allergens (e.g. animal dander, house dust mite, pollen, moulds, etc.) and some food allergies which can occasionally be responsible for exacerbations of atopic eczema. Drops of the various allergens (Table 21.7) are placed on the skin (usually the



**Fig. 21.6** Skin prick test positive to dog and house dust mite

**Table 21.7** Common allergens tested in a skin prick test

Feather Mix
Cat
Dog
Horse
Rabbit
House Dust Mite I
House Dust Mite II
Tree I (early)
Tree II (Mid)
Grass Mix
Weed Mix
Mould Mix I
Mould Mix II
Latex
Whole Egg
Cow’s Milk
Codfish
Crab
Wheat Flour
Peanuts
Rape Seed Oil
Hazelnut
Brazil Nut
Sesame Seed
Neg. Control
Pos. Control

forearm or back) and punctured intradermally with a lancet introducing a tiny amount of the allergen into the skin. Results are read 15 minutes later. A positive reaction causes a wheal (bump) and flare (redness) reaction larger than the negative control (Fig. 21.6). The size of the wheel may quantify the severity of the reaction. This test is also useful to see if a patient has grown out of their allergy. More limited series can be done in small children (e.g. house dust mite, cow's milk, egg, peanut, positive and negative controls). Skin prick test is unsuitable in urticaria as the skin is generally hyper-reactive and it can be difficult to perform on small children. Antihistamines should be stopped 48 hours before a skin prick test [5]. Resuscitation equipment including adrenaline should be available in the extremely rare risk of a severe allergic reaction to the skin prick test solution [6].

An IgE and RAST (Radio-Allergo Sorbent Test) blood test are also useful for identifying airborne allergens and some food allergies in atopic eczema and chronic urticaria (Table 21.8). Like the skin prick test it helps identify immediate, Type 1, IgE mediated allergic reactions. The RAST test can be carried out even when a patient is on anti-histamines. The significance of both the RAST and a skin prick test can be difficult to interpret if the patient has coexisting allergic rhinitis or asthma. False positive and false negative results may occur, especially for weak reactions. These tests are best carried out by doctors experienced in allergy testing and in conjunction with a detailed allergy history, examination and other allergy tests such as an exclusion diet.

Adverse reactions to foods may occasionally be responsible for precipitation or aggravation of urticaria or atopic eczema. These reactions may be immunologically mediated (food allergy) or non immunologically mediated (food intolerance) and the reaction may be obvious or hidden.

**Table 21.8** Common allergens tested in a RAST test

**Food screen** = Codfish, egg white, milk, peanut, wheat and soya milk.

**Asthma screen** = House dust mite, cat, dog, timothy grass, aspergillus and silver birch

A careful history and food diary may help to identify the offending food. Skin prick testing and RAST testing may help identify food allergies but not food intolerance. The definitive test for adverse reactions to foods is a careful **exclusion diet** followed by food challenging under medical supervision by a doctor and dietitian who are experienced in this area. The most commonly implicated foods which may aggravate atopic eczema are dairy produce, eggs, nuts and wheat. Food additives such as the azo dyes and the benzoate preservatives can occasionally provoke urticaria or atopic eczema but this may be difficult to identify. Children on long term restricted diets should be reviewed annually by a dietitian to ensure their diet is nutritionally adequate.

Aspirin (salicylates) and NSAIs containing drugs are one of the most common identifiable causes of acute and chronic urticaria. Since aspirin is hidden in many drugs the patient may not be aware of the reaction. Many natural and processed foods also contain small amounts of salicylates and if taken in sufficient quantity can provoke an urticarial rash. Fruits such as apples, sultanas, raisins, oranges and strawberries are particularly rich in salicylates. Artificial salicylates are also found in flavored sweets, ice cream, soft drinks and cake mixes. It is worth trying all troublesome cases of chronic urticaria on an aspirin free, low salicylate, coloring and preservative free diet since there is no accurate test for identifying sensitivities to these products. However, the diet should be stopped after six weeks if there is no significant improvement.

## 21.8 Conclusion

Allergy tests performed by doctors are not 100% sensitive or specific. False positives and false negatives can occur. All allergy tests need to be carried out by a doctor with experience in these tests and should only be interpreted following a careful history and a thorough physical examination. A negative allergy test does not necessarily rule out a food allergy and if the clinical fea-

tures are suspicious enough, an exclusion diet followed by a reintroduction of the food may be warranted.

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# Generalised Rashes in Adults

22

David Buckley

## Key points

- The diagnosis of a generalised rash in adults may be obvious from the distinctive morphological features of each individual lesion and the distribution of the rash throughout the body.
- It is important to try to make an accurate diagnosis so as to initiate the correct management plan.
- Persistent, troublesome or progressive rashes may need more thorough investigations or referral.
- Clinical signs such as vesicles, blisters, burrows, mucosal involvement or a fever may help in making a diagnosis.
- When faced with an unusual generalised rash, it is more likely to be an unusual presentation of a common problem rather than a rare disease.

## What to tell the patient

- If you develop a new, generalised, itchy rash and someone close to you is also itching, scabies is a possible cause.
- Almost any drug (both prescribed and over the counter) can cause a generalised rash. Tell your doctor about all medicines you are taking

including tablets, creams, patches, implants, the pill, the coil, vitamins, tonics, and herbal medicine.

## 22.1 Introduction

A rash is a widespread eruption of lesions. Many patients present to their doctor with a generalised rash. The diagnosis may be obvious from the distinctive morphological features of each individual lesion and the distribution of the rash throughout the body (e.g. psoriasis presents with silvery-white scaly plaques with well demarcated borders on the back of the elbows and the front knees) (Fig. 22.1). In some cases the signs and symptoms may be less well developed because the patient may be presenting at the early stages of the disease or may have more than one overlapping problem. The classical clinical features of the rash may sometimes be obscured by previous treatment such as topical or oral steroids which can make the diagnosis more difficult.

## 22.2 Clinical Features and Diagnosis

Many different dermatological conditions can produce similar rashes. For example, a morbilliform rash is one that looks like measles, an eczematous rash looks like eczema, psoriasisiform

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**Fig. 22.1** Chronic plaque psoriasis

looks like psoriasis and an urticarial rash looks like urticaria. Sometimes a single skin disorder can result in rashes with various clinical appearances (e.g. psoriasis can present as guttate psoriasis, small plaque psoriasis, flexural psoriasis, plaque psoriasis or palmer plantar pustular psoriasis). Many rashes look different early in their evolution or if they have been present for months or years when scratching may result in secondary skin changes which may mask the underlying condition. It is important to try to make an accurate diagnosis so as to initiate the correct management plan. If a specific diagnosis cannot be made then the doctor should attempt to make a reasonable and comprehensive differential diagnosis considering all possibilities including common, uncommon and rare rashes. It is important to rule out life threatening diseases that may present with a rash and immediately treat and refer these patients, for instance, a rash from meningo-coccal septicaemia, erythroderma, staphylococcal scalded skin syndrome or toxic epidermal necrolysis/Steven-Johnson syndrome (TEN/

SJS). It is also important to identify infectious rashes that may pose a threat to others especially pregnant women. Childhood exanthems (Chap. 27), pregnancy rashes (Chap. 25) and blistering eruptions (Chap. 23) will be covered in their respective chapters.

### 22.3 Differential Diagnosis

Difficult to diagnose rashes may sometimes be managed by symptomatic treatment and review after a few weeks where clues in history or more classical clinical features of a particular rash may become more apparent. Other rashes may need investigations or referral depending on these symptoms, the risk of serious underlying pathology and the patient's ideas, concerns and expectations.

Many patients with an acute generalised, maculo-papular rash with no symptoms improve spontaneously or with simple measures such as emollients and avoiding soaps and other irritants, (e.g. guttate psoriasis, viral rashes). Sometimes, no definite diagnosis is made. More persistent, troublesome or progressive rashes may need a more thorough history taking (Table 22.1), investigations (Table 22.2) or referral. When faced with an unusual generalised rash it is more likely to be an atypical presentation of a common problem rather than a rare disease. Rare diseases like mycosis fun-

**Table 22.1** History taking in patient with a generalised rash

Symptoms—fever, itch, pain?
Contact with other with a rash?
Chronic illness (e.g. diabetes, HIV, Rheumatoid SLE)
Personal or family history of atopic disease, psoriasis or other skin disease.
Insects
Plants
Hobbies
Recent travel
Occupation
Drugs (topical, oral, systemic, prescribed, over the counter, alternative)
Alcohol
Pets/animals
Sexual history
Chemical exposure

goides (a cutaneous T-cell lymphoma of the skin) do occasionally show up in primary care. It tends to present initially with clinical features of a more benign disease such as eczema but the rash persists and evolves despite treatment, which should prompt the doctor to do further investigations such as a skin biopsy or refer the patient.

Mycosis fungoides improves with topical corticoids but later reappears. It can present with ill-defined plaques or as a generalised rash. These patients may go through many skin biopsies taken at different periods of their life with the rash until eventually a biopsy shows evidence of a T-cell lymphoma. Do not be afraid to repeat a skin biopsy even if the patient had a previous one taken a year or so before.

Like all challenging problems in medicine, when faced with a difficult to diagnose generalised rash, it is important to take a careful, focused history (Table 22.1). Symptoms such as fever, itch or pain should be identified. Patients with a rash and a fever usually implies infection but inflammatory conditions, drug eruptions, rheumatologic diseases and erythroderma can also cause a fever and a flu-like illness. Serious life threatening conditions that present with a rash and fever such as meningococcal septicaemia should always be considered as sometimes

the rash can precede the fever but the patient is usually unwell.

Itch is a common sign in certain skin conditions and some cause an intense itch. Pain in the skin with a generalised rash is unusual but can occur with herpes zoster, cellulitis, TEN/SJS, staphylococcal scalded skin syndrome or some drug rashes.

Scale and blanchable erythema implies epidermal (superficial) skin pathology such as psoriasis, tinea and pityriasis rosea. Where there is a generalised rash with no scale, this usually implies deeper, dermal pathology such as urticaria or erythema multiform.

The characteristics of the individual lesions such as size, shape, colour and the presence or absence of scale should be carefully examined with good light and magnification (Table 22.3). Dermoscopy when available can be helpful in certain circumstances such as visualising the scabies mite at the end of a scabies burrow (Fig. 22.2a, b, c, and d).

The distribution of a generalised rash can be very helpful in making a diagnosis (e.g. whether the rash is primarily truncal, primarily flexural or involve other areas of the body such as the palms and soles, the scalp, the mucous membranes, nails or nipples) (Table 22.4).

Certain clinical signs such as the Koebner phenomenon (i.e. the development of typical lesions in the site of trauma) are characteristic of psoriasis, lichen planus and viral warts.

Nikolsky sign (i.e. easy separation of the epidermis from the dermis with lateral pressure) is associated with staphylococcal scalded skin syndrome and TEN/SJS. Other clinical signs such as the rash clearing in one area in less than twenty-four hours, only to recur in a different area over the body are typical of urticaria. On the other hand, an urticarial rash that lasts more than twenty-four hours in any one area of the body may imply urticaria vasculitis, especially if there is tenderness, pain and bruising on the site of the rash. Other signs such as palmer hyper-linearity, infraorbital folds (Dennis-Morgan lines) and lichenification may suggest atopic eczema.

**Table 22.2** Tests to consider in difficult to diagnose generalised rashes

– Full blood count, urea and electrolytes, liver function tests, glucose, erythrocyte sedimentation rate, thyroid function tests
– Urinalysis
– Auto-antibodies screen including antinuclear antibodies
– VDRL + syphilis screen
– A.S.T.O. + throat swab
– Coeliac antibodies
– IgE + RAST
– HIV
– Skin scrapings for yeast or fungal stain and culture
– Skin microscopy for scabies
– Chest Xray (? Sarcoid,? TB)
– Skin biopsy
– Immunofluorescence (direct + indirect)

**Table 22.3** Morphology of individual lesions in a generalised rash

Size					
1–10 mm		1–25 cm		Variable	
Folliculitis		Plaque psoriasis		Atopic eczema	
Keratosis pilaris		Small plaque psoriasis		Contact dermatitis	
Guttate psoriasis		Discoid eczema		Drug eruption	
Insect bites		Tinea corporis			
Lichen planus		Urticaria			
Nodular prurigo (prurigo nodularis)					
Shape					
Disk shape		Annular		Oval	Linear
Discoid eczema		Tinea corporis		Pityriasis rosea	Contact dermatitis
Guttate psoriasis		Small plaque psoriasis		Phytophotodermatitis	
		Glanuloma annulare		Koebner phenomenon	
Colour					
White	Pink or Red	Purple	Red-brown	Blue-black	Brown
Vitiligo	Viral rash	Lichen planus	Secondary syphilis	Vasculitis. Meningococcal septicaemia	Morphoea. Post-inflammatory
Post inflammatory	Drug rash				
Lichen Sclerosis					

## 22.4 Investigations

Any unusual scaly rash which is asymmetrical and not responding to topical treatments such as steroids should have skin scrapings taken and examined for fungal stain and culture. Scabies is a microscopic diagnosis and is confirmed by either seeing the mite in the burrow with dermatoscope or better still, removing the mite from the burrow with a number fifteen scalpel blade and placing it on a microscope for everyone to see.

Skin biopsies can be helpful in certain conditions (e.g. lichen planus, lupus, granuloma annulare) but can be confusing or misleading in other conditions, particularly viral exanthems, atopic eczema and drug eruptions. When biopsying a rash it is best to do an elliptical excision incorporating some of the involved and uninvolved skin which can be orientated for the pathologist by placing a suture in a specimen in the uninvolved end of the biopsies. Fresh lesions or a new area of rash should be biopsied and a detailed clinical

history of the rash with any relevant clinical findings should be sent to a pathologist with a special interest in dermatohistopathology. The clinical diagnosis and a list of differential diagnosis should be sent with the specimen. Discussing the case with the pathologist is often helpful in trying to reach a diagnosis. If you cannot make a reasonable assessment of what the rash might be on clinical grounds alone, it may be better to refer the patient to a colleague with more experience in dermatology who can give the pathologist a reasonable differential diagnosis to work from. Immunofluorescence of the involved and/or perilesional skin sometimes required, particularly for blistering eruptions but the specimens has to be sent to the lab in liquid nitrogen or Michel's transport medium to enable the pathologist to carry out immunofluorescence.

Most generalised rashes can be divided into scaly and non-scaled rashes and further divided into whether they are intensely itchy or whether there is little or no itch (Table 22.5).



**Fig. 22.2** (a) Scabies rash; (b) scabies burrow on the foot; (c) scabies burrow and mite (at 7 o'clock) as seen on dermoscopy. (d) scabies's burrow on the wrist

## 22.5 Generalised, Scaly, Non Itchy or Mildly Itch Rashes

If the rash has a sharply demarcated border and primarily affecting the flexures, particularly the back of the elbows, the front of the knees and the low back then psoriasis is a strong possibility. There are often other clues to the diagnosis such as scalp involvement, nail involvement, ear and perianal involvement (see Chap. 15).

If the generalised scaly, non itchy rash is primarily on the trunk it could be due to pityriasis rosacea, pityriasis versicolour, seborrhoeic dermatitis, sub-acute lupus, a drug eruption, ichthyosis, tinea infection or secondary syphilis.

### 22.5.1 Pityriasis Rosacea

This causes a generalised scaly rash with little or no itch mainly seen in children and young adults par-

**Table 22.4** Distribution of a generalised rash

Primarily truncal	Guttate psoriasis
	Pityriasis versicolour
	Pityriasis rosea
	Sub-acute lupus
	Folliculitis
Flexural involvement	Atopic eczema
	Flexural psoriasis
	Scabies
	Tinea
	Lichen planus
Palms + soles involved	Contact dermatitis
	Palmoplantar pustulosis
	Urticaria
	Scabies (in babies)
	Tinea
	Secondary syphilis
	Erythema multiforme
	TEN/SJS
Genital involvement	Scabies (in men)
	Psoriasis
	Seborrhoeic dermatitis
	Drugs
	Lichen planus
	Pemphigus vulgaris
	TEN/SJS
Scalp Involvement	Psoriasis
	Seborrhoeic dermatitis
	Dermatitis herpetiformis
	Discoid lupus
	Tinea
	Folliculitis
Nipple involvement	Scabies (in women)
	Contact dermatitis
	Atopic eczema
Nail involvement	Psoriasis
	Tinea
	Lichen planus
	Drugs
	Crusted scabies
	Darier's disease
Mouth involvement	Lichen planus
	Pemphigus vulgaris
	Drug
	TEN/SJS
Ear involvement	Psoriasis
	Seborrhoeic dermatitis
	Atopic eczema
	Contact dermatitis
	Discoid lupus

(continued)

**Table 22.4** (continued)

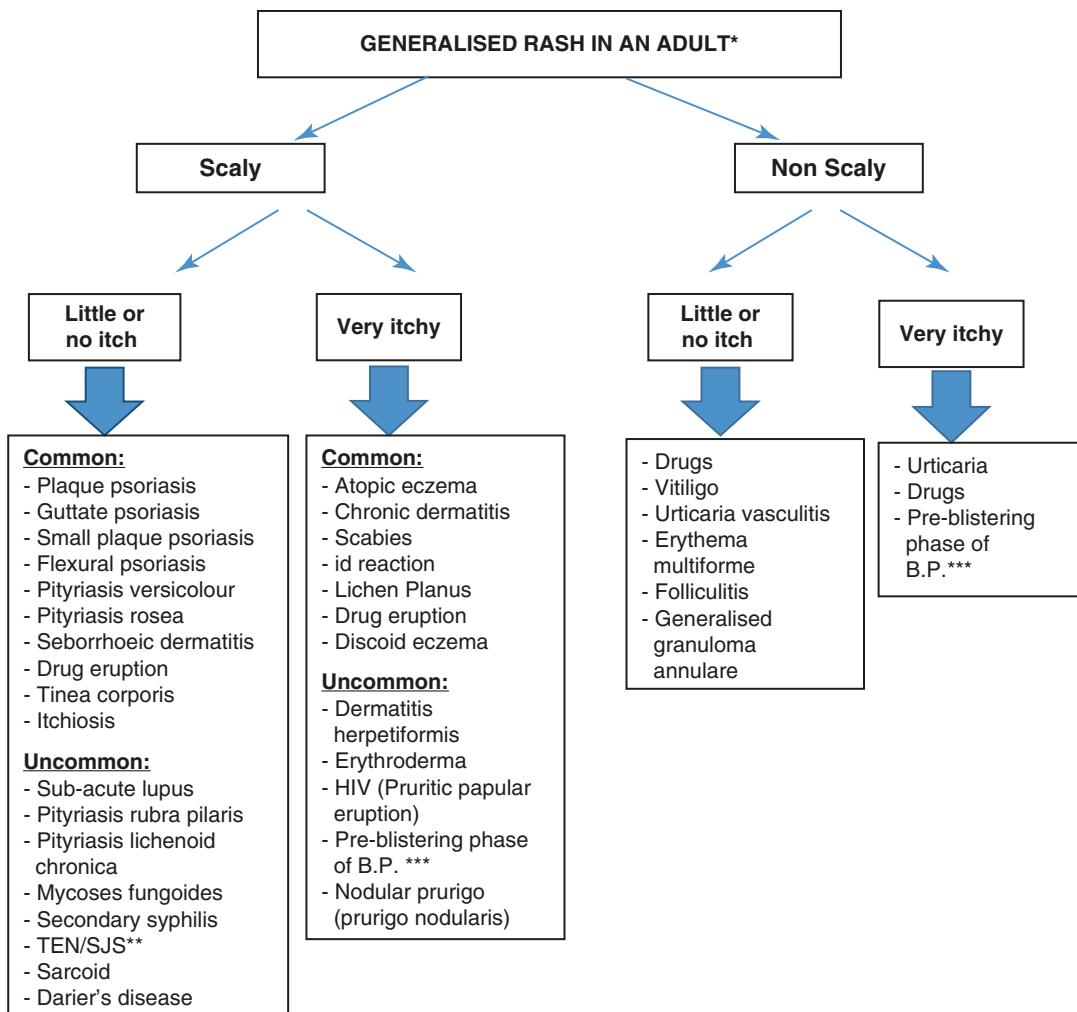
Peri-anal involvement	Psoriasis
	Seborrhoeic derm
	Atopic eczema
	Contact Dermatitis
	Lichen planus

ticularly in the spring and autumn. The cause is unknown although viral triggers such as reactivation of the herpes virus type 6 and 7 (which cause roseola in infants) are often implicated. Diagnosis is clinical as there are no haematological or histological diagnostic criteria. It begins as a scaly, annular or discoid rash 1 to 3 cm in diameter usually found somewhere on the trunk (herald patch) (Fig. 22.3). This can sometimes be confused with ringworm. The herald patch is absent or undocumented in 20% of cases.

After 5 or 10 days a generalised rash appears on the trunk and proximal limbs characterised by multiple oval shaped lesions some with a scaly border (collarette of scale) whose long axis measures 1 to 2 cm and which run in the direction of the dermatomes given the characteristic fern tree pattern on the back [1] (Fig. 22.4a, b, c). The rash usually lasts around 6 or 12 weeks and then clears spontaneously. Treatment is symptomatic and is usually with emollients and avoiding soaps and other irritants. If there is itch a potent topical steroid applied once daily to the body can help. For more severe extensive, itchy rash, acyclovir 400 mg three times a day for seven days can be considered if the patient is seen early [2]. Relapse is unusual. Pityriasis rosea in pregnancy may cause miscarriage or premature delivery in some women.

### 22.5.2 Pityriasis Versicolour (Tinea Versicolour)

Pityriasis versicolour (PV) is a slightly scaly rash affecting the trunk and upper arms usually in young adults. There is normally little or no itch. It is sometimes called tinea versicolor, although the term 'tinea' should strictly refer to infection with a dermatophyte fungus. PV is caused by the

**Table 22.5** Common and rare causes of a generalised rash in an adult

\*Excluding febrile rashes, childhood exanthema, pregnancy rashes and blistering disease

\*\*TEN Toxic epidermal necrolysis, SJS Stevens-Johnson syndrome

\*\*\*B.P. Bullous pemphigoid

proliferation of the lipophilic fungus of the genus *Malassezia* (formerly known as *Pityrosporum*) which is part of the normal flora of human skin and can be seen on microscopy of skin scrapings. The patient usually presents with a hypopigmented or hyperpigmented macular rash with fine scales. Pityriasis comes from the Greek word pityra which means scale. The colour of the rash may vary with sun exposure hence the name (versicolour). It can present itself in brown, pink or white colours (Fig. 22.5a, b).

PV is usually diagnosed clinically but a fast, cheap way to confirm the diagnosis is to take a

piece of transparent adhesive tape (sellotape or scotch tape), place it over a small round PV plaque, rub to stick as much as possible and then pull off (it is not painful at all!). The tape will show an area with scales in the exact shape as the lesion in the skin. If this piece of tape is placed on a microscope slide with some fungi staining (even blue ink will do), spores and hyphae of *Malassezia* will be seen which are often likened to spaghetti and meatballs. After treatment, placing and tearing off the tape will no longer show any scales. The infection is no longer present; only the residual hyperpigmentation is still visible.



**Fig. 22.3** Pityriasis rosae with a herald patch (larger lesion on the left upper back)

This condition can be treated by a topical anti-fungal cream or ketoconazole (“Nizoral®”) shampoo used as a lotion, rubbed from the neck down to the upper thighs and down the arms to the wrists front and back for 15 minutes nightly for seven days. This should clear the fine scale but the pigmented changes can persist for a number of weeks or months after the treatment. Getting a mild tan will help it speed up the resolution of the irregular pigmentation. Relapse is common and some patients have to treat themselves every spring.

Oral antifungal such as itraconazole (200 mg daily  $\times$  7 days) or fluconazole (300 mg once weekly for 1 to 3 weeks) can be helpful in more severe resistant cases. Oral terbinafine is not effective for malassezia infections such as pityriasis versicolor.

Pityriasis versicolor should not be confused with vitiligo which causes loss of pigment and white patches on the skin. Vitiligo has no scaling or itch and it most commonly begins on the hands and feet (see Chap. 47).

### 22.5.3 Seborrhoeic Dermatitis

This most commonly affects the face, the scalp and groin, but more severe cases can cause an itchy rash particularly over the sternum and between the shoulder blades on the back (see Chap. 16).

### 22.5.4 Subacute Lupus Erythematosus

This is a very rare rash in primary care but generally presents as a non-itchy or mildly itchy, annular, slightly scaly rash mostly on the trunk in adults. It is often aggravated by ultraviolet light and heals without scarring (see Chap. 51).

### 22.5.5 Drug Eruptions

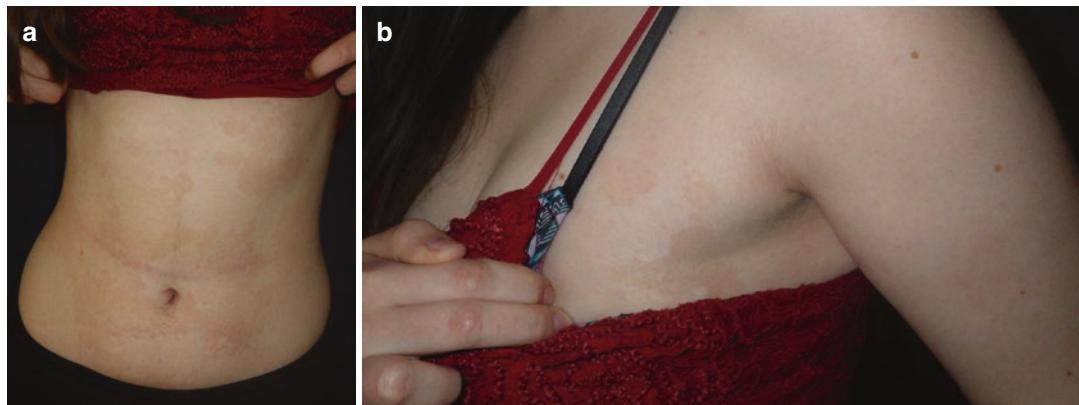
Almost any drug can cause a generalised scaly rash but the more common offenders include non steroid anti-inflammatory, aspirin, anti convalescents, antifungals, antibiotics and chemotherapeutic agents. The diagnosis of a drug eruption might be obvious from the timing of the introduction of the drug and the commencement of the rash. The time between starting the medication and the rash appearing can vary from days to months in some cases. Some rashes might only develop after a course of the drug has been completed. Many elderly patients are on multiple drugs which can make it difficult to identify the culprit. Stopping or substituting suspected drugs, which sometimes has to be done in rotation, maybe the only way to rule out a drug eruption.

### 22.5.6 Ichthyosis

Ichthyosis is an autosomal dominant disease where there is a loss of functioning in the gene that encodes for the protein filaggrin which helps to form an effective skin barrier. Patients with ichthyosis have extremely dry (fish scale) skin that develops in childhood and can get worse around puberty. It affects the extensor aspects of the limbs, scalp, central face and trunk. Skin



**Fig. 22.4** (a) Pityriasis rosea with oval patches running in the direction the dermatomes. (b) Pityriasis rosea oval patches with colorette of scale. (c) Colorette of scale in a discoid patch in pityriasis rosea



**Fig. 22.5** (a, b) Pityriasis Versicolour

folds are usually spared. Atopic eczema is found in approximately 50% of people with ichthyosis. In the absence of atopic eczema they may be very little or no itch. Management is by frequent applications of emollients and avoiding soaps and other irritants.

### 22.5.7 Tinea Corporis

Tinea infections (also called ringworms) are common and usually present as one or two solitary annular, scaly, slightly itchy rash on any part of the body. Most cases are from animal sources and

some forms of tinea corporis can be quite extensive on the trunk or body such as the rash from microsporum canis (from cats and dogs). The original lesions are typically almost perfectly annular, with individual lesions not too large. The diagnosis is often obvious with a typical annular rash and a history of being in contact with an animal (e.g. a new kitten at home). The classical features are sometimes hidden by previous treatments such as potent topical steroids (tinea incognito). Always consider a fungal infection for any scaly rash where the diagnosis is unclear and a skin scraping should be taken for fungal stain and culture particularly if there is a history of animal contacts. Treatment of tinea is normally straight forward with either topical or oral anti fungals or both. Every effort should be made to isolate this source and treat it (e.g. cattle, dogs) (see Chap. 31).

### 22.5.8 Secondary Syphilis

Syphilis has recently had resurgence, partially as a result of migration, foreign travel and HIV disease. It usually starts with a painless sore (ulcer) at the infected site such as the genitalia or mouth known as a chancre (primary syphilis). This is followed weeks or months later with flu-like symptoms and a widespread rash (secondary syphilis). If left untreated tertiary syphilis may develop months or years later causing various systemic problems including heart, eyes, brain and bone disease.

Once infected the incubation period before developing a primary chancre is anything from 10 to 90 days (average 21 days). The chancre usually heals in 4 to 8 weeks without treatment and may be hidden inside the vagina or anus and may go undetected. Secondary syphilis develops 3 weeks to 3 months after the primary chancre and causes a widespread, slightly scaly, reddish-brown rash on the trunk and limbs. It classically causes rash on the palms and soles (see Fig. 36.19 in Chap. 36). There may be a flu-like illness and lymphadenopathy.

Diagnosis is usually made by microscopy of swabs taken from the primary chancre or in later stages by blood tests such as VDRL. False positive

tests can occur during pregnancy, other infections, with drug abuse, connective tissue disease and ageing. Syphilis is the great mimicker. It can resemble many skin conditions and diagnosing it is only possible if one includes it in the screening test. Treatment of syphilis is normally carried out by infectious disease departments as contact tracing, test of cure and screening other STDs is important.

Other rare causes of a generalised scaly non itchy rash include pityriasis rubra pilaris, pityriasis lichenoides chronica, mycosis fungoides, TEN/SJS (Chap. 23), generalised sarcoid (Chap. 51), and Darier's disease (Chap. 28).

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## 22.6 Generalised, Scaly, Itchy Rashes

The most common causes of a generalised, scaly, itchy rash is atopic eczema, chronic dermatitis and scabies. If a patient presents with a new generalised itchy, scaly rash together with a history of being in contact with others with and itch, then scabies should be considered. If burrows are found, this makes the diagnosis even more likely. An intensely itchy, generalised, scaly rash with small vesicles especially in the backs of the elbows and the front of the knees might suggest dermatitis herpetiformis. Other less common generalised, itchy, scaly rashes include lichen planus (Chap. 19) drug eruptions, the pre blistering phase of bullous pemphigoid (see Chap. 23) and nodular prurigo (prurigo nodularis). Other causes include some of the following:

### 22.6.1 Discoid Eczema

This is most commonly seen in adults and usually causes discreet, coin shaped erythematous, scaly plaques occurring particularly on the extremities in young adult males. The rash is usually intensely itchy. The condition usually runs a course of one to two years before resolving spontaneously. It can sometimes be confused with a contact dermatitis or fungal infection. Treatment is with emollients, avoiding irritants and potent topical

steroids. Allergy testing is usually unnecessary and un-rewarding.

### 22.6.2 Dermatitis Herpetiformis

This is an intensely itchy, generalised, scaly, eczematous rash that has a characteristic distribution on the extensor surfaces, the scalp and the buttocks. There are often tiny vesicles but these are usually scratched off fairly quickly because of the intense itch and then appear as excoriations (see Chap. 23).

### 22.6.3 Erythroderma

This is a condition that causes widespread redness, scaling and itching of the skin affecting 90% of the skin due to an underlying inflammatory disease. It is sometimes called the “red man syndrome”. There are many causes but it is usually as a result of an exacerbation of a pre-existing skin condition such as psoriasis, dermatitis (atopic, contact, or seborrhoeic types), pityriasis rubra pilaris, cutaneous T-cell lymphoma (e.g. Mycosis fungoides) or a drug eruption. In up to 25% of cases no underlying cause can be found.

The patient is often toxic with a fever and a flu-like illness. This can be a medical emergency and most patients require hospital admission for rehydration, supportive care, diagnosis and specific treatment of the underlying cause if found.

### 22.6.4 Pruritic Papular Eruption of HIV

This is one of the most common skin eruptions seen with HIV infection. The cause is unclear. It is not strictly a rash. It usually presents as multiple discreet intensely red bumps symmetrically distributed on the trunk and limbs. The face, mucus membranes, palms and soles are usually spared. Because of its non-specific features any patient with an unusual extensive itchy eruption should have their bloods checked for HIV particularly if they are felt to be in a high risk group. Treatment can be difficult

but most cases will respond to potent topical steroids, emollients and anti-histamines. Phototherapy may help in more resistant cases and some patients may respond to anti retroviral therapy.

### 22.6.5 Nodular Prurigo

This is also known as prurigo nodularis. It is not strictly a rash, rather a generalised eruption. It causes intensely itchy, scaly, sometimes warty nodules, 1 to 3 cm in diameter. They are mostly found on the arms and legs in adults (Fig. 22.6). The nodules can occur on any part of the body and they are usually grouped (Fig. 22.7). The cause is unknown. It is more common in patients with a personal or family history of atopic disease. It is sometimes associated with underlying diseases such as iron deficiency anaemia, chronic renal failure, coeliac disease or HIV infection. A biopsy may be required to rule out other diseases. Treatment can be very difficult. Emollients, soap substitutes, potent topical steroids or tacrolimus may help. More resistant cases may need intralesional steroid injections, steroids under occlusion (e.g. “Haelan Tape®”) cryosurgery, systemic antihistamines, tricyclic antidepressants (e.g. amitriptyline), anticonvulsants such as gabapentin (“Neurontin®”) and other treatments aimed at reducing the itching (see Chap. 50). Some patients may respond to UVB phototherapy, oral steroids or other systemic agents such as methotrexate or systemic retinoids. Patients with nodular prurigo often suffer from insomnia and may become depressed or even suicidal.



**Fig. 22.6** Nodular prurigo (Prurigo Nodularis)



**Fig. 22.7** Prurigo Nodularis (Nodular prurigo)

## 22.7 Generalised, Non Scaly Rashes With Little Or No Itch

Apart from drugs and vitiligo, the three most common presentations for these symptoms would be folliculitis, urticaria vasculitis and erythema multiforme.

### 22.7.1 Generalised Folliculitis

Folliculitis occurs as a result of inflammation in the hair follicle (pilo-sebaceous unit). This causes red papules and/or pustules where the hair exits the skin. This may only be apparent when the area is examined with good light and magnification. The area affected by folliculitis may be localised (scalp, face or beard area, etc.) or generalised. Generalised folliculitis is more likely to occur in hairy individuals and on hairy parts of the body such as the chest and back in men or the legs in women.

Generalised folliculitis can be caused by infection, occlusion, irritation, drugs or various skin diseases. Swabs from a pustule may help identify bacterial causes such as *staph aureus* which usually responds to topical or oral antibiotics (e.g. flucloxacillin x 2 to 6 weeks). Spa pool (jacuzzi) folliculitis is usually due to *pseudomonas aeruginosa* and usually clears spontaneously once the patient stays away from the pool. More severe resistant cases may need treatment with oral ciprofloxacin.

Yeast infections such as *malassezia folliculitis* is caused by *Pityrosporum ovale* and usually presents as a monomorphic, acne like eruption on the chest and upper back. It is more common in patients who are overweight, have Down's syndrome or who are immunosuppressed (e.g. diabetes, HIV, obesity, pregnancy). It sometimes occurs after a course of oral broad spectrum antibiotics or oral steroids. Swabs may fail to isolate the yeast and a skin biopsy may be necessary to make the diagnosis. *Malassezia folliculitis* responds to dealing with the underlying problem and topical or oral anti yeast medications such as ketoconazole or itraconazole. Potent topical steroids can aggravate infective folliculitis caused by bacteria or yeast.

Gram negative folliculitis can occur in acne patients on long term oral antibiotics such as tetracyclines. It usually presents as an acute pustular rash on the face and/or body in an acne patient. It can occasionally cause nodules or cysts. Swabs may help confirm the diagnosis. It usually responds to stopping the anti acne drug and giving trimethoprim or amoxicillin for a few weeks. Some of the patients who develop gram negative folliculitis will eventually end up on isotretinoin ("Roaccutane®").

Greasy moisturisers can also cause folliculitis due to occlusion of the hair follicle. Rubbing greasy moisturisers downwards in the direction of the hair growth can help prevent folliculitis or alternatively the patient could use a less greasy product. Coal tar products can also cause folliculitis.

Eosinophilic folliculitis can occur in individuals who are immunosuppressed (e.g. HIV or cancer patients) and is usually diagnosed by

finding the characteristic histological features on a skin biopsy. Treatment is by dealing with the underlying cause and some cases may respond to NSAIDs, oral tetracyclines or oral metronidazole.

### 22.7.2 Urticular Vasculitis

This is a form off cutaneous vasculitis which resembles urticaria but unlike urticaria, the itchy rash can remain on any one area of the body for more than 24 hours. On close examination there maybe tiny inflamed blood vessels and light bruising. Sometimes the rash is painful or burning rather than itchy. Petechiae (small spots of bleeding under the skin) may appear. Some patients may have systemic symptoms such as joint pains, fever, abdominal pains, lymphadenopathy or photosensitivity. Diagnosis is usually made by a skin biopsy. Treatment can be symptomatic with anti-histamines and non-steroidal anti-inflammatory. More troublesome cases of urticarial vasculitis require Dapsone, colchicine, anti-malarial, oral steroids or other immunosuppressive agents (see Chap. 54).

### 22.7.3 Erythema Multiforme

This is a hypersensitivity reaction triggered by infections such as herpes simplex virus or mycoplasma pneumonia. It usually presents with a sudden eruption of a sharply demarcated, round, pink, red macula rash that become more raised and gradually enlarge to form plaques which can range from 1 to 7 cm in diameter. The centre of the plaques usually darkens in colour to cause the typical “target legion” or “bulls eye” lesion (Fig. 22.8). They maybe only a few or perhaps hundreds of target legions seen throughout the body (Fig. 22.9). Legions usually resolve spontaneously after 2 or 3 days. More severe cases can cause vesicles or blisters. Mucus membrane involvement is common especially in the mouth with swelling, redness and blister formation.



**Fig. 22.8** Orf for 10 days and erythema multiforme for 2 days in a farmer



**Fig. 22.9** Erythema multiforme

The diagnosis is usually clinical but histology can sometimes help in more difficult to diagnose cases.

Most cases settle spontaneously after a few days. Treatment, when required, usually involves treating the underlying infection which is normally herpes simplex. Symptomatic treatment with potent topical steroids for a rash on the body and anti-histamines can help. Severe cases may respond to oral steroids. Recurrent erythema multiforme is usually treated by a continuous course of oral anti-virals for 6 to 12 months. More severe cases may require a treatment with dapsone, antimalarials or other immunosuppressive drugs. Erythema multiforme usually resolves spontaneously without scarring over a course of a few days or few weeks. It does not progress into toxic epidermal necrolysis or Stevens Johnson’s syndrome (TEN/SJS) which are nearly always caused by a drug eruption.

## 22.8 Generalised, Non Scaly, Very Itchy Rashes

Apart from urticaria, drugs would be the most common cause of a generalised, very itchy, non scaly rash on the body. Often, there are secondary skin changes such as excoriations from scratching. The pre-blistering stage of bullous pemphigoid can also cause an urticarial, intensely itchy rash which can last weeks or months before the blisters appear.

## 22.9 Conclusion

Generalise rashes are common in primary care. Some are obvious form the history and examination [3, 4]. Others may need special tests and investigations to try to find out the underlying diagnosis. Symptomatic treatment with emollients, soap substitutes and topical steroids (pro-

vided there is no signs of a fungal infection) may be appropriate initially but if the rash continues and simple tests do not reveal the underlying cause, the patient may need to be referred to a colleague with more experience in dermatology.

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# Blistering Eruptions

23

David Buckley

## Key Points

- With the exception of chickenpox, a generalised or wide spread blistering rash can be serious and may need urgent investigation and treatment.
- Rare primary blistering disorders can be difficult to diagnose, and may require special skin biopsies for histology, immuno-fluorescence imaging and electromicroscopy.
- Severe extensive blistering eruptions may need hospital admission and treatment with high dose steroids or antibiotics.

## What to tell the patient

- Most localised blisters are usually due to innocent problems such as a localised burn, trauma, cold sores or an insect bite.
- More generalised blistering rashes may be more sinister and will require immediate investigation and treatment.
- Chickenpox is one of the most common causes of a generalised blistering rash. While it can be relatively harmless in children it can be more serious in adults, newborns, pregnant women and in people who have a compromised immune system.

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## 23.1 Introduction

A blister is a fluid filled, circumscribed elevation of the epidermis. Small blisters (<1 cm in diameter) are called vesicles and larger ones are called bullae. Blisters may be filled with serous fluid, puss or blood. A solitary blister or a localised group of blisters are usually not associated with any serious pathology. However, with the exception of chickenpox, a generalised or wide spread blistering rash can be much more dangerous. Blistering rashes can be divided into three main groups: common benign blistering disorders; common skin disorders that occasionally blister; and rare primary blistering eruptions (Table 23.1).

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## 23.2 Common Benign Blistering Disorders

**Burns** are the most common cause of blisters seen in general practice. The clinical features are usually the same, regardless of the cause (heat, cold, electricity, chemical or UV radiation). Immersing the area in running cold water for 20 minutes is helpful if the burn is seen early. Burst any tense blisters with a sterile pin and drain the excess fluid. Leave the top of the blister intact as it acts as its own natural biological dressing. A potent or super potent topical steroid applied in the first 24 hours may usually reduce the inflammatory reaction for a small

**Table 23.1** Causes of vesicles/bullae

Primary Cutaneous Disease:
Pemphigus,
Bullous Pemphigus,
Dermatitis Herpetiformis,
Contact Dermatitis,
Erythema Multiforme,
Stevens-Johnson syndrome,
Toxic Epidermal Necrolysis,
Varicella Zoster Virus,
Herpes Simplex,
Hand-foot-and-mouth disease,
Staphylococcal scalded-skin syndrome,
Scarlet Fever,
Toxic Shock Syndrome,
Exfoliative Erythroderma Syndrome
Systemic Diseases:
Paraneoplastic pemphigus,
Porphyria Cutanea Tarda,
Porphyria Variegata

localised burn. “Cool Jel®” (WaterJel tech) which is a topical cooling gel can be applied immediately after a burn. Antiseptic creams such as silver sulphadiazine 1% cream (“Flamazine®”) is popular and may prevent infection [1]. Other alternatives include honey, “Mepitel®” or “Adaptic®” (see Chap. 38).

First aid management of more extensive burns includes cooling the area with tepid running water (not ice), keeping the patient warm and giving appropriate analgesia. Cooling the area may reduce the damage caused by heat and gives relief to the patient. Layers of kitchen “Clingfilm” (do not wrap circumferentially around a limb as swelling may lead to constriction) or layers of sterile gauze soaked with saline should be placed over the burns and the patient should be transferred to hospital or a burns unit. Extensive burns may lead to dehydration and hypotension and may need emergency resuscitation with IV fluids, etc. [2].

If the burn needs to be dressed, gauze impregnated with an antiseptic such as Chlorhexidine (“Bactigras®”) or an open mesh silicon based wound contact layer such as “Mepitel®” (“Safetac®”) or “Adaptic®” (“Systagenix®”) are the most suitable as they are less likely to stick to the burn. Keeping the area clean is essential to avoid infection. Burns on the face, hands and

genitalia, and deep or extensive burns need special care and attention. If there is any risk of scarring, contractures or dehydration, it might be wise to refer the patient to a surgical colleague or a burns unit. Tetanus prophylaxis should always be considered. Remind the patient to protect the area from the sun and UVL until complete healing to avoid excess hyperpigmentation.

**Traumatic blisters** such as friction blisters can be managed the same way as burns.

**Chickenpox** (varicella-zoster virus) is another common blistering disorder. The itchy vesicles are usually small and symmetrically distributed mostly on the trunk and face. They pass through the stages of papule, vesicle, pustule and crust. There may be a preceding flu-like symptoms (Chap. 27). Atypical cases, particularly in adults, neonates, anyone who may be immunocompromised or in pregnant women, may not have the usual clinical features and may cause confusion. These high risk groups may need antiviral treatment.

Both **herpes simplex** and **herpes zoster** may also present as a localised vesicular or bullous eruption. These conditions are dealt with in Chap. 32.

### 23.3 Common Skin Disorders That Occasionally Blister

**Insect bites** are a very common but often missed cause of blisters (bullous papular urticaria). The blisters can vary in size from a few millimeters to a large tense bulla measuring 2–4 cm in diameter (Fig. 23.1). There may be only one, or a cluster of blisters, usually on an exposed area of the body. Most patients cannot remember the actual bite. Burst tense blisters and apply a potent topical steroid for 24 hours, which should reduce the inflammatory reaction. Avoid using topical anti-histamines because of their high sensitising potential. Oral sedative anti-histamines are much safer and more effective at relieving itch, but are usually only given at night as they may cause drowsiness. Eliminating the source of the insects (animals, plants, etc.) and the use of insect repellants may help.

Impetigo can occasionally blister (**bullous impetigo**) when there is an intense inflammatory reaction to the staphylococcus. Treatment is with topical or systemic antibiotics or both, depending on the severity.

Severe eczema or dermatitis can occasionally cause vesicles or bullae, particularly on the hands or feet where it is known as **pompholyx** (dyshidrotic eczema). Because the skin in these areas is thick, the vesicles do not burst easily and appear more like itchy papules (Fig. 23.2a and b). In many cases of pompholyx, no obvious cause can be found. Some cases are due to an allergic con-

tact dermatitis or an id reaction to tinea pedis, particularly in young women. Soaking hands or feet in potassium permanganate (1:10,000) together with a systemic antibiotic is useful in weepy, infected cases. Potent topical steroids are also helpful, once infection is cleared. Occasionally, systemic steroids may be necessary in severe unresponsive cases.

**Scabies** can occasionally cause vesicles to develop around the wrists and fingers, but the typical signs and symptoms of scabies should still be present.

**Phytophotodermatitis** can present itself as bullae. It can be recognised by the bizarre pattern of the streaky rashes and possibly blisters, which usually correspond to the places where the plant substance has touched the skin: it is very common in arms and hands. Poison ivy, giant hogweed and bergamotta from citrus peel are common causes.

Another common cause of local bullae is **drug photoallergic reactions** which may happen after several days of exposure to sun. They usually appear in photoexposed areas like the face and the dorsum of hands. A drug that frequently causes this reaction is piroxicam. The diagnosis is simple once the patient is asked about recent medication (Chap. 49).



**Fig. 23.1** Insect bites

## 23.4 Rare Primary Blistering Eruptions

These disorders usually cause widespread blistering throughout the body. Although uncommon in general practice, they can cause life threatening infection if not taken care in time, dehydration or



**Fig. 23.2** (a) Pompholyx. (b) Pompholyx triggered by Tinea Pedis

metabolic disturbances. Some patients have an associated occult neoplasm which should be searched for. Diagnosis can be difficult, often involving special skin biopsies for histology, immuno-fluorescence imaging and electromicroscopy (Tables 23.1 and 23.2). Patients suspected of having any of these disorders should be referred to a specialist for confirmation of the diagnosis and initiation of treatment.

**Bullous pemphigoid (BP)** usually occurs in the elderly. It often starts with itchy, burning, urticarial plaques which particularly affect the flexural areas, especially the groin and axilla. The urticarial plaques can last weeks or months before blisters develop (pre-blistering bullous pemphigoid) (Fig. 23.3a and b). Blisters, which are large, tense and filled with clear, yellow fluid, arise on the erythematous urticarial skin. When the blisters rupture they leave a painful raw eroded area which may easily become secondarily infected. The area usually heals without scarring. The mucous membranes are rarely involved, except in the rare cicatricial pemphigoid type when the ocular, oral and genital mucosae are particularly affected. Very high doses of systemic steroids under specialist supervision will usually cause a remission. Potent or super potent topical steroids can be helpful in milder, localised cases. Some cases may respond to doxycycline 100 mg twice a day.

**Pemphigoid gestationis** is a rare form of bullous pemphigoid which occurs in the second trimester of pregnancy and may be associated with increased foetal and maternal morbidity (see Chap. 25).

**Pemphigus vulgaris (PV)** is less common but more serious than bullous pemphigoid. It occurs in a younger age group (40–60 years) and over 50% of patients present with painful oral ulcerations several weeks or months before the generalised skin eruption. Flaccid blisters which easily rupture appear on normal skin and may remain localised for several months but eventually become more generalised with the scalp, face, flexures and pressure areas being particularly affected. The blister sites heal with some bleeding and crusting but no scarring. Downward pres-

sure on an intact blister produces lateral extension of the lesion (positive Nikolsky's sign). This is a useful sign and may help differentiate pemphigus vulgaris from bullous pemphigoid (where the Nikolsky's sign is negative).

PV can be a serious and sometimes fatal condition. Fortunately high dose oral steroids usually induce a remission. These patients may be changed to drugs like azathioprine after the condition is controlled with oral steroids. They should be managed by specialists in immune skin conditions.

Pemphigus vulgaris may occasionally be caused by drugs such as d-penicillamine, rifampicin and phenylbutazone.

One easy way to remember the type of blistering that occurs is that pemphigoid cause **d**eep, **t**ense blisters whereas pemphigus causes **s**uperficial, **f**laccid blisters.

**Dermatitis herpetiformis** (DH) causes an auto-immune, pruritic, vesiculo-bullous eruption which may occur at any age but the most common age of onset is between the third and fourth decade. It is twice as common in males as in females. Most patients present with intensely itchy erythematous macules which evolve into vesicles within 36 hours. Scratching removes the roof of the vesicle and relieves the itch. Thus, excoriation and scarring may be the principal physical sign, and vesicles may be hard to find (Fig. 23.4a, b).

The characteristic distribution of this symmetrical rash, which usually involves the backs of the elbows, scalp, forehead, buttocks and the front of the knees, should make one suspect DH. Most patients (80%) with DH have gluten sensitive enteropathy with partial villous atrophy on jejunal biopsy which is similar but usually less severe than coeliac disease. DH may be considered a cutaneous manifestation of coeliac disease. Most do not have any GI symptoms. Intestinal lymphomas may develop after many years.

Diagnosis is usually by skin biopsy of fresh vesicles which should show the characteristic features of DH with sub epidermal blisters. Direct immunofluorescence reveals IgA immunoglobu-

**Table 23.2** Comparison of bullous (blistering) diseases

	Bullous Pemphigoid	Pemphigus Vulgaris	Dermatitis Herpetiformis	Linear IgA	Erythema multiforme	SJS/TEN <sup>a</sup>	SSSS <sup>b</sup>
Age of onset	Elderly	Middle age	All ages esp. 30's-50's	Elderly	All ages	All ages	Neonates and young children
Sites of involvement	Flexures on an erythematous urticarial background	Onset in mouth. Blisters on normal skin	Elbows, knees, buttocks, scalp. Intense itch	Limbs initially then generalised	Palms, dorsum of hands. Extensor limb → spreads centrally	Trunk → Face → Limbs	Axillae, groin Nose, ear Nappy area, umbilicus
Mucous membrane involvement	Rarely	Common (in >50%)	No	Common (50%)	Sometimes	Common	Crusts in nose sometimes
Types of blisters	Large tense deep blisters	Flaccid superficial blisters	Intensely itchy flaccid vesicles	Itchy flaccid grouped vesicles	Bullae in target lesions	Flaccid bullae	Flaccid bullae and skin peeling off in sheets
Histology	Sub-epidermal bullae = deep	Intra epidermal blisters = superficial	Sub epidermal blisters	Sub epidermal blisters	Sub epidermal	Full thickness epidermal and epithelial necrosis	Intra epidermal cleavage beneath and within the stratum granulosum
Immunofluorescence	Linear deposits of IgG at basement membrane	Intracellular deposition of net like IgG in epidermis	Papillary deposition of IgA	Linear IgA along basement membrane	Negative or upper dermal vascular fluorescence	Negative	Negative
Associated features	Other auto immune diseases	Other auto immune disease	Coeliac disease in 80%	Maybe caused by drugs or underlying cancer	May be caused by herpes simplex, mycoplasma or drugs	Fever. Critically ill	Exotoxin producing Staphylococcus aureus infection. Very sick child: fever, irritable
Treatment	High dose oral steroids, super potent topical steroids	High dose oral steroids. Can be fatal	Dapsone. Gluten free diet. Super potent topical steroids	Dapsone or oral steroids	Potent topical steroids or oral steroids	Stop offending drug. IV fluids. I.C.U.	Flucloxacillin. IV fluids. I.C.U. contact tracing

<sup>a</sup>SJS/TEN = Stevens Johnson Syndrome/Toxic Epidermal Necrolysis<sup>b</sup>SSSS = Staphylococcal Scalded Skin Syndrome



**Fig. 23.3** (a) Pre-blistering phase of bullous pemphigoid. (b) Bullae in an 82-year-old man with bullous pemphigoid

lin in dermal papillae. Coeliac antibodies may be positive and a jejunal biopsy should be carried out, looking for subtotal villous atrophy. Treatment of DH is with dapsone, which stops



**Fig. 23.4** (a) Dermatitis herpetiformis with tiny excoriated vesicles on the abdomen. (b) Dermatitis herpetiformis with vesicles on the posterior elbow

the itch and vesicle formation dramatically within 24–48 hours in most cases. This drug needs careful monitoring by doctors experienced in using it. Patients taking dapsone should be counselled about the risks associated with it, which include haemolytic anaemia, agranulocytosis, methaemoglobininaemia, and peripheral neuropathy. Regular blood tests (full blood count and reticulocyte

count) are necessary. A strict gluten free diet may enable the maintenance dose of dapsone to be lowered or stopped. Potent or super potent topical steroids may suppress the disease locally. A gluten free diet usually helps.

**Linear IgA dermatosis** is a rare autoimmune skin disorder in which blisters form in the skin and mucous membranes. It is most common in the elderly. The itchy vesicles usually start on the limbs but can spread all over the body. 50% of patients will have blisters and ulcers on the lips and inside the mouth. Vesicles and bullae tend to be grouped together on various parts of the body. New vesicles may develop in a ring around an old one (the ‘string of beads’ sign). Eye involvement can occasionally occur.

Linear IgA can be associated with inflammatory bowel disease, other auto immune diseases, drugs and underlying malignancy (e.g. Lymphoma, solid tumours, etc.). Skin biopsy shows sub epidermal blistering and direct immunofluorescence shows immunoglobulin IgA along the basement membrane of the epidermis in a linear pattern. Most cases respond to dapsone or oral steroids.

**Erythema multiforme** (EM) usually presents as crops of erythematous macules, erupting symmetrically on the dorsal aspect of the hands, wrists, and feet, and around the knees and elbows. Lesions measure 1–2 cm in diameter with an annular configuration (target lesions). Milder cases may be difficult to differentiate from urticaria.

In more severe cases the lesions may be large and blister (bullous EM) (Fig. 23.5). Oral involvement may occur. EM may occur at any age and can be provoked by a number of agents including herpes simplex, mycoplasma infection, drugs (sulphonamides, tetracycline, phenylbutazone, barbiturates, contraceptive pill), autoimmune diseases, carcinoma and lymphomas. Approximately 50% of cases no precipitating factor is found. Topical steroids are helpful in minor cases. Systemic steroids may be necessary in bullous EM.

**Stevens Johnson Syndrome/Toxic Epidermal Necrolysis** is a very rare, acute, serious, and potentially fatal skin reaction in which



**Fig. 23.5** Bullous erythema multiforme

there is sheet-like skin and mucosal loss. They are now considered the same disease and distinct from erythema multiforme. They are almost always caused by rare drug reactions to many different types of drugs like antibiotics, antifungals, antivirals, allopurinol, NSAIDs and anti-convulsants. In its most severe form there may be fever, widespread bullous eruptions and painful, hemorrhagic oral and genital mucosal ulcerations. It may begin with morbilliform rash and progress to target lesions and flaccid blisters. The Nikolsky sign is positive in areas of skin redness. This is a skin finding in which the top layers of the skin slip away from the lower layers when slightly rubbed. Skin biopsy is usually required to confirm the clinical diagnosis and to exclude **Staphylococcal Scalded Skin Syndrome** (SSSS) and other generalised blistering rashes. Patients are usually very sick and require immediate withdrawal of the offending drug and treatment in intensive care.

**Staphylococcal scalded skin syndrome (SSSS)** is caused by the release of exotoxins from toxicogenic strains of the bacteria *Staphylococcus aureus*. It is characterised by red blistering skin that looks like a burn or scald, hence its name. It most commonly occurs in neonates and young children under the age of 5 years old. It may occasionally occur in immunosuppressed adults. SSSS starts from a localised staphylococcal infection of the toxicogenic strain that is a producer of exotoxins. Outbreaks of SSSS often occur in childcare facilities. Asymptomatic adult carriers of *Staphylococcus aureus* may introduce the bacteria into the nursery. About 15–40% of healthy

humans are carriers of *Staphylococcus aureus*, but children are more vulnerable to infection because they have not had the opportunity to develop immunity to the toxins. SSSS usually starts with fever, irritability and widespread redness of the skin. Within 24–48 hours fluid-filled flaccid blisters form in the armpits, groin and body orifices such as the nose and ears. These rupture easily, leaving an area that looks like a burn. In newborns, lesions are often found in the nappy area or around the umbilicus. Diagnosis is confirmed by skin biopsy and sending tissue for culture and sensitivity. Treatment is usually with flucloxacillin which is sometimes given IV in more severe cases. The child may also need general supportive care in ICU. Recurrence is rare, as infection usually results in the child acquiring lifelong protective antibodies against staphylococcal exotoxins. Close contacts, such as family members and health care workers may need to be screened to eradicate the organism in asymptomatic carriers. Strict hand washing and the use of hand sanitisers will reduce the risk of spreading the organism.

**Epidermolysis bullosa** is a rare group of inherited disorders characterised by blistering of the skin following trauma. Most cases begin in childhood. Milder cases may be confined to the hands and feet and heal without scarring. Severe cases may be extensive, involving the skin, gastrointestinal and respiratory tracts, causing scarring, disability and often an early death. This is sometimes referred to as the ‘butterfly syndrome’.

**Porphyria cutanea tarda** is due to an abnormality of porphyrin metabolism. Excess uroporphyrins and coproporphyrins derived from the liver are found in the urine. PCT may be precipitated by oestrogen, chloroquine or excess alcohol. Small blisters appear on sun exposed skin of the forehead, backs of the hands and following trauma. They heal to leave small scars and milia cysts. Facial hypertrichosis (excess hair) may be prominent. Some patients may have cirrhosis and elevated total body iron stores. Treatment is with phlebotomy and avoiding any known precipitating factors.

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## 23.5 Conclusion

Generalised blistering eruptions are usually a sign of alarm. They can be potentially life threatening and may need special investigations such as skin biopsy for histology and immunofluorescence. Severe extensive blistering eruptions may need hospital admission and treatment with high dose steroids or antibiotics.

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## References

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# The Ageing Skin

24

David Buckley

## Key Points

- The burden of skin disease in the elderly is much higher than in the younger population as a result of various factors including photo ageing, underlying chronic disease, intrinsic ageing, immobility, trauma, nutritional deficiency, and polypharmacy.
- Treating skin diseases in the elderly can be more challenging as a result of various factors including failing physical and mental health, difficulty with manual dexterity, poor eyesight, isolation, poor nutrition and financial restraints.
- Certain skin conditions are far more common in the elderly such as xerosis (dry skin), seborrhoeic keratosis, senile (Bateman) purpura pruritus, varicose eczema, chronic actinic dermatitis, blistering disorders and skin cancers.
- While melanoma can occur at any age after puberty, almost half (44%) occur in people over the age of 65 and 55% of deaths from melanomas occur in this age group.

## What to tell the patient

- Many of the skin changes seen in the elderly such as wrinkles, skin thinning, freckling, actinic keratosis, telangiectasia and purpura are more likely due to the accumulative effects

of ultraviolet radiation (photoageing) rather than chronological ageing.

- Drugs are a common cause of itch and rashes in the elderly. Almost all drugs have the possibility to cause itch or rash.
- Seborrhoeic keratosis and cherry angioma (Campbell de Morgan spots) are common, harmless benign growths in the elderly and require no treatment unless for cosmetic reasons.

## 24.1 Introduction

Life expectancy is increasing year on year. The Central Statistics Office in Ireland predicts that the number of people aged 65 will rise from 532,000 in 2011 to almost 1.4 million in 2046. The burden of skin disease in the elderly is much higher than in the younger population as a result of various factors including photo ageing, underlying chronic disease (Table 24.1), intrinsic ageing, immobility, trauma, nutritional deficiency, and polypharmacy. Therefore there will be a far greater demand for skin care in the elderly in the community in the future.

## 24.2 Photoageing

Many of the skin changes seen in the elderly such as wrinkles, skin thinning, freckling, actinic keratosis, telangiectasia and purpura are more likely

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**Table 24.1** Chronic disease that can be associated with skin problems in the elderly

• Arteriosclerosis
• Venous insufficiency
• Diabetes
• Congestive cardiac failure
• Chronic liver or renal disease
• Thyroid disease
• Rheumatoid arthritis
• Parkinson's disease
• Immunodeficiency
• Obesity

due to the accumulative effects of ultraviolet radiation (photo ageing) rather than chronological ageing. When the skin on an elderly person's face is compared to that of his/her buttocks it is obvious that a lot of the signs of "ageing" are in fact due to chronic exposure to ultraviolet light and it is possible to slow down or prevent these changes by being sun smart (wearing an SPF 50 sun block, a broad-brimmed hat and taking extra vitamin D) at a younger age.

The structure and function of the skin changes with age. The epidermis becomes thinner and more porous with a loss of the skin barrier function. This results in elderly patients being more susceptible to eczema and dermatitis (irritant and contact). The stratum corneum loses its ability to retain water and the skin dries out. Cell replacement and wound healing is slower in the elderly. The dermis becomes thinner with loss of elasticity and connective tissue resulting in wrinkles (rhytides), furrows, sunken cheek and sagging of the skin (solar elastosis). This can also lead to broken veins on the face and neck (telangiectasia) and easy bruising (senile purpura) which can be aggravated if the person is on anticoagulants. The eccrine sweat glands shrink as we get older and the pilosebaceous units produce less oil resulting in dry skin. This is why acne is rare in the elderly but eczema is more common. The Langerhans cells decrease, affecting the immune responsiveness of the skin. This can lead to the decline in cell mediated immunity, resulting in problems such as skin cancers and shingles. Melanocytes are more active in the elderly, particularly in the

sun exposed areas, resulting in irregular pigmentation (solar lentigo) and hyper-pigmentation.

Some of the changes could be slowed down, improved or reversed by certain cosmetic procedures such as topical retinoids, chemical peels, HRT, lasers, IPL, "Botox®", fillers, microneedling and cosmetic surgery. Arteriosclerosis, smoking and genetic factors can accelerate skin ageing.

Elderly people can suffer from many of the chronic skin conditions seen in younger people such as psoriasis, urticaria, drug eruptions and scabies. Certain skin conditions are far more common in the elderly such as xerosis (dry skin), seborrhoeic keratosis, cherry angioma, senile (Bateman) purpura, pruritus, varicose eczema, chronic actinic dermatitis, blistering disorders and skin cancers (Fig. 24.1). Non-melanoma skin cancers (BCC's and SCC's) and pre cancers (actinic keratosis and Bowen's disease) are far more common in the elderly, primarily as a result of an excessive accumulation of ultraviolet radiation over the years. Melanoma can occur at any age after puberty, but almost half of all melanomas (44%) occur in people over the age of 65 and 55% of deaths from melanomas occur in this age group.

### 24.3 Senile Purpura (Bateman Purpura or Actinic Purpura)

This is an extremely common skin problem especially in forearms in the elderly with fair skin. It causes multiple bruises (purpura) as a result of



**Fig. 24.1** Brusing form aspirin and clopidogrel in a 75 year old

rupture of delicate blood vessels which can occur spontaneously or after minor trauma. It is not dangerous but unsightly. It is a form of sun damage and it is the result of thinning skin and weakening of the blood vessels. It gets worse when the patient is taking aspirin, anticoagulants or steroids (Fig. 24.1). Topical or oral vitamin C and topical alfa hydroxiacid can help reducing this annoying condition.

## 24.4 Seborrhoeic Keratosis

Seborrhoeic keratosis (SK) (also called seborrhoeic warts or basal cell papilloma) is probably the most common benign growth in the skin. They are rare in those under the age of forty. Many older patients can have a small number of SK, particularly on their trunk while others can have numerous SK all over their trunk, face and scalp. This pre-disposition to SK can sometimes run in families. Sudden onset of many SK may be a sign of underlying malignancy (Leser-Trelat syndrome) but this is extremely rare.

SK are usually easily to diagnose clinically with the characteristic raised, brown, waxy, rough, scaly, stuck-on appearance. They are usually round or oval, measuring anything from 5 mm to 20 mm, sometimes even larger. They are normally multiple and have characteristic features when viewed under a dermatoscope (Chap. 46). Care should be taken as they can be confused with moles, solar lentigo, lentigo maligna, melanomas or pigmented BCCs. Dermoscopy may help differentiate these various lesions but if there are any doubts about the diagnosis, a biopsy should be taken. If there is a suspicion of a melanoma, the complete lesion with a 2 mm of clear skin all around should be removed for histology or urgent referral to specialist should be done. If there is a low suspicion of malignancy, multiple punch biopsies from suspicious areas throughout the lesion as judged by an expert in dermoscopy may be performed to rule out any serious pathology.

Although seborrhoeic keratosis are not dangerous, some patients may want at least some of them removed for cosmetic or comfort reasons. No treatment should ever be undertaken unless

there is the certainty on the diagnosis. The simplest and most successful way to remove them is either by shave biopsy or curettage under local anaesthetic followed by light cryosurgery to prevent relapse (a five second freeze and one freeze-thaw cycle). Thin SKs can be treated with cryosurgery, sparing the need for local anaesthesia. Bleeding is usually light and can be controlled with aluminium chloride. The removed specimen should always be sent for histology to confirm the diagnosis.(see Chap. 57).

## 24.5 Cherry Angioma (Campbell de Morgan Spots)

These are very common benign vascular growths, most commonly seen on the trunk in the elderly. They are usually bright red or purple macules or papules measuring 2 mm to 10 mm in diameter and are most commonly found on the trunk but sometimes on the face (Fig. 24.2). They never cause problems but some patients may want them removed for cosmetic reasons. The simplest treatment is with a vascular laser but if this is unavailable they can be removed by electrocoagulation. If in doubt about the diagnosis, take a punch or shave biopsy under local anaesthetic.

## 24.6 Generalised Pruritus

Pruritus means a desire to itch. A severe itch is as distressing as a severe pain and can lead to serious impairment in quality of life. Chronic itch is



**Fig. 24.2** Cherry angioma in a 68-year-old man

a common presentation in the elderly. This may or may not be associated with an underlying rash. The itch usually results in scratching which can cause excoriations and low grade infection of the skin. These secondary changes from scratching may cause a rash or may hide the more typical clinical features of an underlying rash (e.g. the vesicles of dermatitis herpetiformis are usually quickly scratched away as they are intensely itchy).

Pruritus can be peripheral, due to stimuli occurring in the skin, or can be central, when itching is perceived as occurring in the skin although it originates in the central nervous system.

There are many causes of generalised pruritus in the elderly (Table 24.2) and every effort should be made to identify and treat any of these under-

**Table 24.2** Possible causes of pruritus in the elderly

**Dermatological:**

- Xerosis (dry skin)
- Infestation (e.g. scabies, bed bugs, body lice, etc.)
- Infection (bacterial, viral, fungal)
- Lichen planus
- Nodular prurigo
- Dermatitis/eczema
- Pemphigus vulgaris
- Bullous pemphigoid
- Dermatitis herpetiformis
- Mycoses fungoides (Cutaneous T-cell lymphoma)

**Metabolic:**

- Renal/liver failure
- HIV
- Diabetes
- Thyroid disease
- Parathyroid disease
- Hypervitaminosis A
- Iron deficiency anaemia
- Neuropathy
- Underlying malignancy
- Nutritional deficiency
- Paraneoplastic (Lymphoma, leukaemia, or cancer of the lung, prostate or stomach)

**Psychogenic:**

- Lichen simplex chronicus (neurodermatitis)
- Prurigo nodularis
- Neurotic excoriation
- Delusion of parasites

**Drugs:** (see Table 24.4)

**Table 24.3** Investigation of generalised pruritus

**Full history:** (contacts, animals, recent illness, medical history, family history, foreign travel, alcohol, tobacco)

**Drug history:** (OTC and prescribed, oral and topical, see Table 24.4)

**Physical examination:** (e.g. burrows, vesicles, blisters, lymphadenopathy, hepatosplenomegaly)

**Lab investigations to consider:**

Full blood count

Erythrocyte sedimentation rate

C-reactive protein

Urea and electrolytes

Estimated glomerular filtration rate

Liver function tests

Calcium, phosphate, alkaline phosphatase

Thyroid function tests

Iron, B12, folate

Fasting glucose

HbA1C

Urinalysis

Chest x-ray

Skin scraping

Coeliac antibodies

Allergy testing (skin patch test, Skin prick test, IgE + RAST test)

Skin biopsy

Immunofluorescence

lying factors (Table 24.3). The itch or rash may be a sign of an underlying malignancy (paraneoplastic syndrome). By far the most common cause is xerosis or dryness of the skin, which happens to everybody with ageing. As the skin dries out, it loses its natural skin barrier function which makes the person more prone to irritants (soaps, shampoos and bubble baths), allergens (creams and dressings) and infections. Excessive dryness of the skin may be aggravated by excessive washing, too much heat or low humidity, which often occurs in nursing homes.

A common form of dryness occurs in the lower legs in elderly patients called eczema craquele or asteatotic eczema. This presents as dry, itchy, scaly skin with a cracked porcelain-like appearance. Varicose veins with secondary varicose eczema are another common cause of dry itchy skin in the lower legs in the elderly.

Xerosis can be managed by moisturising liberally with a safe, greasy moisturiser rubbing

downwards at least twice a day. Moisturisers with menthol which can be stored in the fridge may help soothe hot, itchy skin. Trimming finger nails and wearing cotton gloves at night may also help. Elderly patients with dry skin should also avoid soaps and other irritants such as shower gels, shampoos, bath additives, perfumes, etc. Suitable soap substitutes such as aqueous cream, “Elave wash®”, “Elave shampoo®” and “Aveeno wash®” should be used instead. If there are any signs of eczema then a potent topical steroid should be applied once at night to the affected area in the body for two to four weeks, while continuing to moisturise and avoid soap and other irritants. Antipyretics such as sedating antihistamines, amitriptyline or hydroxyzine (“Ucerax®”) at night may help to relieve the itch and promote sleep. These may cause drowsiness and predispose to falls and accidents, particularly in the elderly. Table 24.3 outlines some investigations to consider when seeking to identify the cause of generalised pruritus.

## 24.7 Cutaneous Adverse Drug Reactions

An adverse drug reactions (ADR) is an unintentional reaction to a drug used at the recommended dose for a specific disease. ADR are more common in the elderly probably because they are on more drugs and because their capacity to clear the drugs from the body is reduced. ADR may be immunologically or non-immunologically mediated. Immunological reactions can be immediate (type 1, IgE mediated—occurs within minutes to hours) or delayed (type 4 reaction, cell-mediated—can take hours or days to develop). Severe ADR such as Stephens Johnson Syndrome/TEN are caused by complex immunological reactions and usually start 4 to 28 days after starting a drug.

Drugs are a common cause of itch and rashes in the elderly. Almost all drugs have the possibility to cause itch or rash. Table 24.4 lists the more common offenders. Cutaneous ADR reactions may present in various ways such as an itchy morbilliform eruption (exanthema), acute

urticaria, angioedema, anaphylaxis, acneform rash, drug induced pemphigus, erythema multiforme (with or without mucosal involvement), fixed drug eruptions, hypersensitivity vasculitis, photosensitive reactions, or drug hypersensitivity syndrome (also known as drug reaction with eosinophilia and systemic symptoms—“DRESS”).

Elderly patients are often on a number of different drugs that could be responsible. Sometimes it may be obvious that a particular drug is responsible as the rash or itch may start around the time the drug was commenced. In other occasions, the rash or itch may not start for weeks or months after commencing the offending drug. If there is a strong suspicion that the drug is responsible, then the drug should be stopped or substituted with a different class of drug for a month and then reintroduced. If the itch or rash improves while off the drug and relapses once the drug is reintroduced, it is highly likely that it was the cause. The cycle of stopping and restarting individual drugs may have to be repeated sequentially for a number of different suspected drugs. Consultation with the doctor who initiated the drug (e.g. the cardiologist or the endocrinologist, etc.) may be necessary, especially for critical drugs such as anticoagulants, cardiac drugs, anti-hypertensive drugs, diabetic drugs, psychotropic drugs, etc. If a patient develops a drug eruption associated with a fever, mouth ulcers, skin pain or a positive Nikolsky sign, they may need urgent assessment as they may go on to develop a more severe drug reaction such as Stephens Johnston Syndrome/TEN. High risk drugs include antibiotics (e.g. trimethoprim), anti fungals (e.g. terbinafine), allopurinol and anticonvulsants.

## 24.8 Bed Sores (Pressure Sores) or Decubitus Ulcers

Pressure sores are much more common in the elderly because their skin is thinner, weaker and more prone to trauma from falls or constant pressure over bony prominences as a result of immobility. Pressure ulcers may not only affect the skin but also the underlying muscle, connective

**Table 24.4** Principal drugs able to induce puritis without skin changes

Drug group	Examples
Antihypertensive drugs	ACE inhibitors Angiotensin II antagonists (sartans) $\beta$ -Adrenoceptor antagonists ( $\beta$ -blockers) Calcium channel blockers Methyldop Sildenafil
Antiarrhythmic drugs	Amiodarone
Anticoagulants	Ticlopidine Fractionated heparins
Antidiabetic drugs	Biguanides Sulfonylurea derivates
Hypolipemic drugs	HMG-CoA reductase inhibitors (statins)
Antibacterials and chemotherapeutics	Penicillins Cephalosporins Macrolides Carbapenems Monobactams Quinolones Tetracyclines Lincosamides Streptogramins Metronidazole Rifampin (rifampicin) Thiamphenicol Trimethoprim/sulfamethoxazole (cotrimoxazole) Antimalarials
Psychotropic drugs	Tricyclic antidepressants Selective serotonin reuptake inhibitors Antipsychotics
Antiepileptic drugs	Carbamazepine Fosphenytoin Oxcarbazepine Phenytoin Topiramate
Cytostatics	Chlorambucil Paclitaxel Tamoxifen
Cytokines, growth factors, and monoclonal antibodies	Granulocyte-macrophage colony-stimulating factor Interleukin-2 Matuzumab Lapatinib Epidermal growth factor receptor inhibitors
Plasma volume expanders	Hydroxyethyl starch
Others	Antithyroid agents NSAIDs Corticosteroids Sex hormones Opioids Inhibitors of xanthine oxidase

Ref: Nicoletta Cassano, Gianpaolo Tessari, Gino A. Vena, Giampiero Girolomoni. Chronic Pruritus in the Absence of Specific Skin Disease: An Update on Pathophysiology, Diagnosis, and Therapy. Am J Clin Dermatol. 2010;11(6):399–411

tissue, tendons, cartilage or bone. Prevention of pressure sores in the elderly is vital. Once the skin breaks down it is more difficult to get it to heal again. 40% of all nursing care is devoted to skin care and decubital care. Elderly patients should be encouraged to keep mobile as much as possible. If they are already immobile for whatever reasons, their vulnerable areas, such as the buttocks, ischial tuberosity and the heels should be protected by regular turning, good skin care and appropriate padding or cushions (e.g. sheep skin heel pads, sheepskin rugs, tubular cushions, ripple mattresses, etc.). If a patient does develop pressure sores, the area needs to be relieved of pressure, elevated and dressed with an appropriate ulcer dressing (see Chap. 38). Island dressings (“Melolin®”, “Primapor®”), and semi-permeable dressings (“Opsite®”, “Tegaderm®”) are not suitable for pressure sores. Secondary infection may need to be treated with an oral antibiotic such as flucloxacillin if not penicillin allergic for at least one to two weeks. Surgical debridement may be required if there are thick eschars. Underlying aggravating factors such as poor nutrition, diabe-

tes, thyroid disease, anaemia and incontinence need to be assessed and managed.

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## 24.9 Other Common Skin Dermatoses in the Elderly

Leg ulcers, blistering disorders, solar damage and skin cancer are all common in the elderly and are discussed elsewhere in this book.

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## 24.10 Conclusion

Skin problems in the elderly are becoming more common as a result of a number of factors including an increasing aging population, chronic UVL exposure, increasing incidence of multiple morbidity and polypharmacy. Treating skin diseases in the elderly can be more challenging as a result of various factors including failing physical and mental health, difficulty with manual dexterity, poor eyesight, isolation, poor nutrition and financial restraints.



# Skin Diseases in Pregnancy

25

David Buckley

## Key Points

- Certain skin conditions may improve in pregnancy while others get worse. It can be unpredictable and can vary from person to person and from pregnancy to pregnancy.
- Pregnant women can occasionally develop skin problems that are unique to the pregnancy state (atopic eruption of pregnancy, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy and pemphigoid gestationis).
- Many STDs may have no symptoms and so all pregnant women should be screened for certain conditions such as HIV, syphilis, hepatitis B and hepatitis C.
- Treatment of skin problems in pregnancy can be difficult as great care needs to be taken not to harm the unborn child.

## What to tell the patient

- Most rashes in pregnancy have no adverse effects on the unborn child.
- Rashes with a fever or suspected STDs (sexually transmitted diseases) in pregnancy should be assessed as soon as possible as they may have adverse effects on an unborn child.
- Melasma (also called chloasma) is more common in pregnancy and women with melasma

should protect their skin from ultraviolet light.

- Temporary diffuse hair thinning is common after a pregnancy (telogen effluvium) and nearly always regrows within 6 to 12 months.

## 25.1 Introduction

Pregnant women can occasionally develop skin problems that are unique to the pregnancy state. However, the majority of skin problems seen in pregnant women are either chronic skin diseases that can vary in extent or severity during pregnancy or incidental skin conditions that can occur at any time (e.g. scabies, tinea infection, drug eruptions, urticaria, etc.). Certain skin changes that can occur in pregnancy may need urgent assessment by an obstetrician such as febrile rashes as they may pose a risk to the foetus. STDs may also present during pregnancy and could be transmitted to the child transplacentally or during the delivery. Table 25.1 outlines some investigations that may be required in a pregnant woman presenting with an itchy rash where the diagnosis is unclear.

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**Table 25.1** Investigation to consider for itch in pregnancy:

Full blood count
Erythrocyte sedimentation rate
Urea and electrolytes
Liver function tests
Thyroid function tests (TFTs)
Glucose
B12, Folate, Ferretin
Serum bile acid levels
Urinalysis
Skin biopsy
Immunofluorescence of skin and blood



**Fig. 25.1** Spider naevi on the chest and one seborrhoeic keratosis

## 25.2 Skin Changes During Pregnancy

Various hormonal factors occurring during pregnancy can cause changes in the skin. The most obvious are striae that occur on the abdomen and sometime on the arms and legs. Telangiectasia and spider nevi can occur on the face and chest (Fig. 25.1). Varicose veins can also be more troublesome in pregnancy as a result of hormonal changes and as a result of pressure effect of the unborn child on the venous system. Erythema nodosum can occur in pregnancy and most cases settle spontaneously in 6 to 8 weeks.

Melasma (also called chloasma) is also common in pregnancy and causes blotchy hyperpigmentation on the face that usually resolves after the pregnancy is over (Fig. 25.2). Women with melasma should protect their skin from ultraviolet light. Some cases of melasma can persist for months or years after the pregnancy, especially if the woman is using hormonal contracep-



**Fig. 25.2** Pregnancy rashes. Mild melasma on the face of patient on the oral contraceptive pill for many years

tives such as the oral contraceptive pill, the mini pill, progesterone containing implant or a hormone containing IUD.

All moles tend to darken evenly during pregnancy and those on the abdomen or breast may enlarge as a result of expanding skin. However, if a woman develops a new mole or has an existing mole that changes in size, shape or develops uneven colour throughout the mole, it should be checked by a doctor with experience in skin cancer and dermoscopy as melanoma accounts for 8% of malignant tumours arising in pregnancy. Suspicious moles should be removed with a 2 mm border of clear skin for histological diagnosis if there is any suspicion of a melanoma.

Hormonal changes during pregnancy can cause hair thinning during or after the pregnancy in some women (telogen effluvium) but the hair usually regrows within 6 to 12 months of the baby being born.

Because of the hormonal and immunological changes that occur in pregnancy, the severity of pre-existing chronic skin diseases such as acne, rosacea, eczema and psoriasis may vary considerably during pregnancy. Changes can vary between individuals and diseases (some conditions improve during pregnancy while others deteriorate). Treatment of chronic skin diseases can also be more challenging in pregnancy as certain topical and systemic drugs might potentially harm the unborn child.

Most patients with psoriasis improve in pregnancy but 10–20% of cases can get worse. Potent topical steroids, including those combined with calcipotriol, can be used in pregnancy but should be limited to 200 g in total during pregnancy [1, 2]. Narrow band UVB can also be used for troublesome psoriasis or acne in pregnancy.

Acne often improves in early pregnancy but may worsen in the third trimester as maternal androgen levels increase. Acne neonatorum (an acne eruption in the newborn) may occur as a result of passive transfer of maternal androgens across the placenta during the third trimester. Rosacea usually gets worse in pregnancy. Topical treatments such as benzoyl peroxide or azelaic acid can be safely used for acne and rosacea in pregnancy in troublesome cases. Topical and oral retinoid should be avoided during pregnancy as they are highly teratogenic. Oral erythromycin can be used after the first trimester of pregnancy for troublesome acne or rosacea. Clarithromycin or azithromycin are the preferred systemic treatment for troublesome acne in the first trimester. Oral tetracycline should be avoided in pregnancy because of its effect on the development of foetal bones and teeth. Topical metronidazole can be safely used for rosacea in pregnancy (see Chap. 6).

Pityriasis rosea is thought to be due to the human herpesvirus 6+7 and can predispose to prematurity or miscarriage.

## 25.3 Specific Dermatoses of Pregnancy

### 25.3.1 Atopic Eruption of Pregnancy

This is the most common dermatosis in pregnancy and accounts for fifty per cent of patients seen in a typical pregnancy skin clinic [3] (Table 25.2). About 20% of patients with atopic eruption of pregnancy have pre-existing eczema. The other 80% experience atopic skin changes for the first time or after a long remission (for example since childhood). The itch usually starts in early pregnancy. Two thirds of cases have typical flexural atopic eczema while one third have a papular eruption on the trunk and limbs with prurigo nodules on the shins and arms.

Skin in atopic eruptions of pregnancy is very dry and should respond to liberal applications of a safe greasy moisturiser. More troublesome cases may require potent topical steroids on the body (see Table 25.3). Severe cases may require oral steroids although, if given in the first trimester, they can increase the risk of cleft lip and palate. UVB phototherapy is safe in pregnancy but may aggravate melasma. Sedating antihistamines (e.g. “Piriton®” or “Phenegran®”) are safe in pregnancy (see Table 25.4). The rash often recurs in subsequent pregnancies, particularly if there is a personal or family history of atopic eczema. The unborn child is unaffected by atopic eruption of pregnancy but the child may develop atopic eczema in childhood as a result of inheriting the atopic gene.

### 25.3.2 Polymorphic Eruption of Pregnancy

This is the second most common specific dermatosis in pregnancy and accounts for approximately 22% of specific dermatoses of pregnancy. It was previously known as puritic, urticarial, papules and plaques in pregnancy (PUPPP). It occurs in approximately 1 in 160 to 1 in 200 pregnancies. The itch usually starts in the third trimester or immediately post-delivery. It usually presents as pruritic urticarial papules that coalesce into plaques and eczematous skin. The rash usually begins in the striae on the abdomen but spare the peri-umbilical area. The rash may then spread to the buttocks, proximal thighs and in severe cases it can become more generalised. It usually spares the face, breasts, hands and soles. The itch can be quite intense and can be relieved by simple emollients and avoidance of soaps and other irritants. Subsequent skin lesions can become polymorphic with small vesicles, erythema, targetoid and eczematous lesions.

A potent topical steroid and antihistamines may be required for intense itch on the body (see Tables 25.3 and 25.4). Severe cases may require a short course of oral steroids. Tacrolimus may be used with caution in pregnancy as it is a large molecule and has low adsorption.

Polymorphic eruption of pregnancy usually resolves within 4 to 6 weeks of the baby being

**Table 25.2** Itch in pregnancy table

	Onset trimester			Typical clinical features	Flexural rash	Vesicles or Bullae	Peri-umbilical involvement	Can cause foetal damage	Diagnosis	Treatment	Can recur in subsequent pregnancy
	1 <sup>st</sup>	2 <sup>nd</sup>	3 <sup>rd</sup>								
Atopic eruption in pregnancy	+	++	+++	Dry skin flexural rash Atopic history	+	-	+/-	-	History & clinical features ? ↑ IgE	Emollients & topical steroids	+
Polymorphic eruption of pregnancy	+	++		Papulo-urticarial rash starts in striae on the abdomen	-	+/-	Spared	-	History + clinical features	Emollients + topical or oral steroids	-
Intrahepatic cholestasis in pregnancy	+	++		Intense itch starts on palms & soles 10% jaundiced	-	-	-	+	Serum bile acid levels	Early delivery/ ursodeoxy- cholic acid	++
Paroxysmal nocturnal hemoglobinuria	+	++		Vesicles & blisters on urticated erythema, starts on abdomen	-	++	++	+	Skin biopsy and blood for immunofluorescence	Topical or oral steroids, Immuno-globulin	++
Scabies	+	+	+	Itchy rash & burrows	+	-	+/-	-	Isolated mite or view with dermoscope	Permethrin 5%	-
Urticaria	+	+	+	Urticarial rash & dermatographism	-	-	+/-	-	History & clinical features	Sedating anti-histamines	-
Drug eruption	+	+	+	Polymorphic	-	-	-	-	Rash starting after drug introduced. Clears on stopping drug.	Stop the drug. Emollients & topical steroids	-

**Table 25.3** Use of topical steroids (TS) in Pregnancy

When using potent TS do not use more than 200–300 g throughout the pregnancy.
Do not use more than 50 g of potent topical steroids per month.
Very potent topical steroids should be avoided in pregnancy.
Potent TS should be used with caution especially in the third trimester.
Potent TS should not be used on striae if possible.

**Table 25.4** Use of antihistamines in Pregnancy

Old fashioned sedating antihistamines such as chlorpheniramine ("Piriton®") or promethazine hydrochloride ("Phenergan®") are considered safe in pregnancy.
Sedating antihistamines are probably the safest antihistamine in the first trimester. Loratadine or cetirizine are considered safe in the second trimester.
All antihistamines should be avoided in the third trimester if possible.

born. It is most common in primigravidae and in multiple pregnancies (twins). Recurrence in subsequent pregnancies is unusual except in multiple pregnancies or with polyhydramnios. It is usually considered a benign condition and is not harmful to the unborn child.

### 25.3.3 Intrahepatic Cholestasis of Pregnancy (ICP)

ICP is also known as puritis/prurigo gravidarum. It is caused by a defect in the excretion of bile from the liver, resulting in rising levels of bile acid in the blood. This can be as a result of genetic, hormonal and environmental factors. The itch usually begins in the second or third trimester and is more common in multiple pregnancies. It occurs in between 0.4% to 1% in pregnant women in Western Europe [4]. There is considerable ethnic variation and it is far more common in women from South America, Scandinavia and the Baltic States. The itch usually starts on the palms and soles but then quickly involves the whole body. There is usually no rash but there

can be excoriations from scratching. The itch typically increases in severity until the baby is delivered. Occasionally (less than 10% of patients) it can cause dark urine and/or pale stools, jaundice and nausea. It is diagnosed by measuring the serum bile acid levels in the blood. This is not part of a routine liver function test and has to be requested specifically by the doctor when sending bloods. Routine LFT's may be normal particularly at an early stage of ICP.

ICP can cause an increased risk of premature labour, foetal distress and still births, particularly in severe cases. Treatment involves delivering the baby at the earliest possible time but usually not before 36 or 37 weeks of pregnancy. Ursodeoxycholic acid will reduce bile acids in the blood stream and can relieve the symptoms. It is not licensed for use in pregnancy but can be used off licence and is considered safe for the mother and baby. ICP tends to recur in subsequent pregnancies and can also recur if the woman is put on the oral contraceptive pill.

### 25.3.4 Pemphigoid Gestationis (PG)

This is a rare autoimmune blistering eruption that usually begins in the second trimester. It occurs in approximately 1:50,000 pregnancies [5]. It was previously known as "Herpes Gestationis" although it has nothing to do with the herpes virus. It presents as an intensely itchy rash on the abdomen but, unlike polymorphic eruption of pregnancy, it can involve the peri-umbilical region. It causes intense, pruritic, urticarial papules and plaques which might become more generalised and eventually can cause tense blisters. Improvement in late pregnancy can occur but it often (in 70% of women) flares up in the post-partum period and this can last for weeks or months. It is more common in multiple pregnancies and multiparous women. Mucous membrane involvement is rare.

Diagnosis can be confirmed by direct immunofluorescence of peri-lesional skin as well as by indirect immunofluorescence of serum. PG can cause prematurity and small for dates babies, par-

ticularly in more severe and extensive disease. As a result of passive transfer of antibodies from the mother to the foetus, about ten per cent of newborns may develop mild skin lesions which resolve spontaneously within days to weeks. Treatment of PG is usually in a specialist dermatology and obstetrics centre and may involve the use of potent topical steroids or oral steroids. Immunosuppressant's may be required after the pregnancy.

Recurrence of PG usually occur earlier with subsequent pregnancies or if the woman is put on hormonal contraceptives.

#### **25.4 Febrile Rashes**

Certain viral illnesses, that are common in childhood, may be passed onto a pregnant woman, particularly if she has younger children at home. All pregnant women should be fully vaccinated before planning a pregnancy and should have their blood checked to confirm protection against rubella and varicella zoster virus. If a pregnant woman is exposed to rubella, measles, or erythema infectiosus (fifth's disease), she may need urgent assessment as she may require specific immunoglobulin and close monitoring. Chickenpox (varicella zoster) can put the mother and foetus at risk. Passive immunity with varicella zoster immunoglobulin to seronegative mothers within 72 hours of exposure to the virus may prevent or reduce the severity of maternal infection. If a woman or newborn is infected with chickenpox he/she should be treated early with acyclovir.

#### **25.5 Sexually Transmitted Diseases (STD)**

Women who are pregnant are obviously sexually active and can become infected with the same STDs as women who are not pregnant. Many of these STDs may have no symptoms and so all pregnant women should be screened for certain conditions such as HIV, syphilis,

hepatitis B and hepatitis C. In the United States of America the center for disease control recommends screening all pregnant women for chlamydia.

Chlamydia and gonorrhoea may or may not cause symptoms in the pregnant woman but can cause problems such as premature delivery, premature rupture of membranes and low birth weight. The baby can get infected during delivery and it can cause infections such as conjunctivitis or pneumonia in the new-born.

Genital warts are very common and can occur for the first time in pregnancy or get worse during pregnancy. Although they are not usually considered dangerous to the unborn child, extensive genital warts may require caesarean section to prevent the newborn developing laryngeal papillomatosis.

Genital herpes may be transmitted to the child during delivery and a caesarean section may be required if the mother has active genital herpes around the time of delivery. Acyclovir is considered safe in pregnancy for treatment or prophylaxis. Women with recurrent genital herpes may require prophylactic acyclovir form 36 weeks of pregnancy till after the baby is born.

Prevention of STDs during pregnancy is vital and the best protection is by using condoms and/or remaining in a long-term, monogamous relationship with a partner who has been tested and known to be uninfected.

#### **25.6 Conclusion**

Certain skin conditions may improve in pregnancy while others get worse. It can be unpredictable and can vary from person to person and from pregnancy to pregnancy as to how pregnancy can affect an underlying chronic skin condition such as atopic eczema, psoriasis or acne.

Women who are pregnant are relatively immune-suppressed and are prone to certain skin conditions that are unique to the pregnancy or rare outside pregnancy. Treatment of skin problems in pregnancy can be difficult as great care needs to be taken not harm the unborn child.

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## **Part V**

### **Paediatric Dermatology**



# Paediatric Dermatology

26

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## Key points

- Certain skin conditions are more common in children (e.g. atopic eczema, warts, molluscum contagiosum and keratosis pilaris).
- Children's skin barrier function and immune system is less well developed than adults and they are more prone to irritants, allergens and infections.
- Children are more likely to present with congenital abnormalities (e.g. haemangioma and congenital naevi) and inherited diseases than adults.
- Treating skin disease in children can be more challenging as many of the treatment we use in adults such as potent topical steroids or oral anti-fungal tablets are more likely to cause side effects in children or are not licensed to use in children.
- Infantile seborrhoeic dermatitis behaves differently from adult seborrhoeic dermatitis and may be a completely different condition.
- Infantile psoriasis usually presents like seborrhoeic dermatitis but the rash is more deeply red, inflamed, extensive and difficult to treat.
- Scabies in babies can include burrows on these soles of the feet and lesions on the face. This is not seen in older children or adults.

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## What to tell the patient (parents)

- Nappy rash should be managed by removing all irritant chemicals that might aggravate the problem and replacing them with a bland soap substitute and a simple moisturiser combined with a barrier cream such as zinc and castor oil.
- Infantile acne usually resolves spontaneously after 6–12 months but if troublesome may require treatment with topical agents such as benzoyl peroxide or a topical antibiotic.
- Keratosis pilaris often runs in families and usually improves as the child gets older.
- Impetigo contagious is as the word implies, very contagious. Careful hand washing and using a separate towel for the infected child is important to protect the rest of the family. The child needs to be kept out of school or crèche till the crusts dry out.

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### 26.1 Introduction

Children may suffer from many of the common dermatology problems that affect adults such as eczema, psoriasis, urticaria, scabies and pityriasis rosea. However, certain skin conditions are more common in children (e.g. atopic eczema, warts, molluscum contagiosum and keratosis pilaris). There are other skin conditions that occur almost exclusively in childhood or behave differently in childhood and this chapter will deal

specifically with these problems. Febrile rashes are dealt with in the chapter of childhood exanthems (see Chap. 27).

Infants and children's skin is different from adult skin. Their skin barrier function and immune system is less well developed and they are more prone to irritants, allergens and infections. In addition, children are more likely to present with congenital abnormalities (e.g. haemangioma or congenital naevi) and inherited diseases (see Chap. 28 on genodermatoses).

## 26.2 Neonatal Milia

Milia (milk spots) are very common and can be found in up to 50% of newborns. They are caused by blocked eccrine sweat gland and are common around the eyes, nose, cheeks, forehead and chest. Each papule is about 1 or 2 mm across and are pearly-white or yellowish. Clusters and crops are common in newborns. They are harmless, cause no symptoms and will resolve spontaneously as the child gets older. Isolated milia can occur in adults especially on the cheeks and around the eyes. If they need to be removed for cosmetic reasons it is best to puncture the surface with a small needle under sterile conditions and gently squeeze out the contents or remove the contents with a micro-curette.

## 26.3 Napkin Dermatitis

Napkin dermatitis is probably one of the most common skin complaints that doctors see in babies. This arises as a result of a number of factors such as irritation from urine and stool, sensitivity to various wet wipes, nappies, washes and creams and sometimes infections. If the rash is confined to the nappy area and does not involve the flexures (creases), then it is most likely just simple napkin dermatitis. If the rash involves the creases and there are patches of red eczematous, scaly skin on other parts of the body, then the nappy rash may be a sign of more generalised skin problem such as seborrhoeic dermatitis or psoriasis. Ironically, napkin dermatitis is unusual

in children with atopic eczema. This may be because of the urea in urine, combined with the occlusive effects of the nappy, may help hydrate the skin and prevent eczema in the nappy area.

The management of nappy rash involves removing all irritant chemicals that might aggravate the problem (soaps, perfumed baby wipes, nappy changing creams with a lot of perfumes or preservatives). These should replaced with bland soap substitutes (e.g. "Elave®" wash or aqueous cream) or clean the delicate perianal skin with water and a wet flannel (bidet). Use a simple moisturiser combined with a barrier cream such as zinc and castor oil with each nappy change. Use eco friendly nappies that are not bleached with chemicals containing chlorine.

If there is still a lot of redness and inflammation, 1% Hydrocortisone cream or ointment may have to be applied, once daily at night time, for not more than five nights until the condition settles. Some children may develop a group a streptococcal infection in the nappy area. If they are in pain, are off their feeds or have a fever they may need treatment with a topical and/or an oral antibiotic (e.g. mupirocin ointment and/or flucloxacillin). Bizarre or unusual rashes on the nappy area might make one think of non-accidental injury.

Sometimes there is a secondary yeast infection, such as candida. This usually presents with a flexural rash in the groin creases and satellite lesions spreading out beyond the main body of the rash. If candida is suspected then a topical anti-yeast agent such as miconazole nitrate cream ("Daktarin®") or clotrimazole cream ("Canestan®") should be used for at least 2 weeks. If the yeast infection is associated with a lot of inflammation, then combining 1% Hydrocortisone with an anti-yeast agent should help (e.g.: "Daktacort®" or "Canestan HC®"). These can be applied once a day for about 2 weeks. All irritants should also be removed and the area moisturised with zinc and castor oil with every nappy change. Antifungal topical products can be irritating themselves especially in occlusive areas and should be stopped after 1–2 weeks. Leaving the nappy area open for a few minutes or a few hours may help but is usually impractical in a baby that is not toilet-trained. Changing

to cloth nappies may help but may be impractical for a busy parent with other young children. Breast fed babies tend to have more runny stools but are less likely to develop nappy rash. Apart from lactose intolerance, food allergies are rarely the cause of nappy rash so changing the child's diet is unlikely to help.

## 26.4 Infantile Seborrhoeic Dermatitis

Infantile seborrhoeic dermatitis behaves differently from adult seborrhoeic dermatitis and may be a completely different condition. Infants who develop seborrhoeic dermatitis do not necessarily go on to develop seborrhoeic dermatitis in adulthood. The most common presentation with seborrhoeic dermatitis is "cradle cap" where the small baby develops non-itchy, thick scales on the top of the scalp. This is harmless and a self-limiting condition. Bland emollients or almond oil and lifting off the scales with a fine comb will help. Tar based shampoo such as "T gel®" or "Capasal®" may also help. If there is a lot of inflammation, 1% Hydrocortisone may be required for a few weeks. Ointments containing salicylic acid such as "Cocois®" should be diluted or used very sparingly or left on for a shorter period of time than in adults as large quantities can cause systemic absorption and toxic reactions.

Children with seborrhoeic dermatitis often develop a more generalised eczematous rash similar to atopic eczema (See Chap. 16). Unlike atopic eczema these children have little or no itch and rarely scratch. The napkin area is usually involved in seborrhoeic dermatitis but is usually spared in atopic eczema. Some children may have an erythematous patch over the sternum, which is quite typical of seborrhoeic dermatitis.

Fortunately, seborrhoeic dermatitis will resolve spontaneously as the child gets older and is usually cleared by the time they get out of nappies. Treatment is with bland emollients, soaps substitutes and 1% Hydrocortisone ointment for any inflamed areas. It is important to reassure the parents that the child does not have atopic eczema, which is a much more serious and trou-

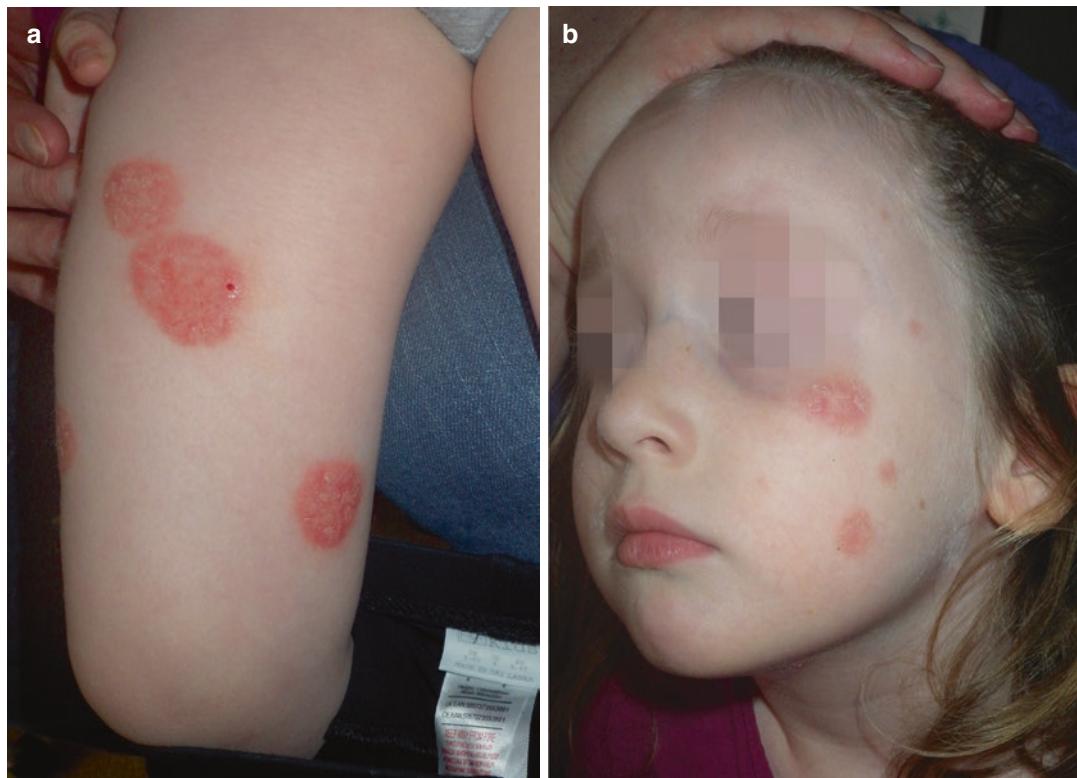
blesome disease. They are usually happy to know that the child is in no distress as the rash does not itch. It is also comforting for them to know that the rash will clear completely, usually by the time the child is 2 years old.

## 26.5 Infantile Psoriasis

Some doctors believe seborrhoeic dermatitis and psoriasis are related conditions and may be part of a spectrum. Infantile psoriasis usually presents like seborrhoeic dermatitis but the rash is more deeply red, inflamed, more extensive and more difficult to treat. It is more likely if there is a family history psoriasis and may be triggered by stress or infection. In infancy it often appears in the nappy areas as napkin dermatitis but can progresses into a more generalised, non-itchy, slightly scaly rash on the scalp, chest, elbows, knees or flexures. The scales are not usually as pronounced as in adults. Instead, the rash is usually deeply red, shiny, macular with a sharp demarcation between the involved and uninvolved skin, just like flexural psoriasis in adults. The infant or child may later develop more classical features of plaque psoriasis on the body and scalp (Fig. 26.1a, b). Nail involvement is rare in infants and children but can sometime occur in isolation without any skin signs.

Older children may present with classical guttate psoriasis with multiple non itchy scaly plaques spread throughout the body in a symmetrical distribution but often sparing the face. It may follow a sore throat.

Treatment in infants and young children should be individualised to the child's age, as well as the extent, distribution, type and severity of the psoriasis (see also Chap. 15). However, most mild to moderate cases will respond to bland emollients, soap substitutes and mild to moderate topical steroids. Tar preparations, calcipotriol and dithranol are all safe to use in children but weaker concentrations may be required. "Dovobet®" and "Enstilar Foam®", which are a combination of calcipotriol and betamethasone, should be avoided in infants and children less than 12 years old, as the steroid component is too potent for this age group.



**Fig. 26.1** (a) Psoriasis in a 5 year old. (b) Psoriasis in a 5 year old on the face

For more troublesome psoriasis in children, calcipotriol ("Dovonex®") can be applied in the morning and a moderately potent topical steroid such as clobetasone butyrate ("Eumovate®") can be applied at night for 4–6 weeks. The topical steroid can then be stopped and the calcipotriol continued till the psoriasis clears.

Tacrolimus ("Protopic®") can be helpful particularly for psoriasis on the face, flexures and nappy area but this is an unlicensed indication. Occasionally systemic treatment with methotrexate, cyclosporine biological agents or phototherapy may be necessary in severe cases under specialist supervision.

Some children can develop **pityriasis amiantacea** where there are thick scaly plaques adherent to the hair on the scalp in localised patches. This is often associated with seborrhoeic dermatitis or psoriasis. Scalp ringworm (*tinea capitis*) and head lice should be excluded. Pityriasis amiantacea can usually be managed with ointments containing tar and salicylic acid

such as "Cocois®". This may have to be diluted down 50% for small children and should only be applied on relatively small areas of the scalp. The ointment is normally left on for an hour or two and then washed off with tar based shampoo. Fine combing may help to lift off the scale. There may be temporary alopecia but the hair will grow back once the rash resolves. If there is a lot of inflammation, 1% Hydrocortisone or a moderately potent topical steroid such as clobetasone butyrate ("Eumovate®") may be applied once daily for a few weeks.

## 26.6 Infantile Acne

Small babies are prone to infantile acne. This is thought to be due to maternal androgens passing onto newborn baby, who then develops comedones (blackheads and whiteheads), papules and pustules usually on the cheeks and nose (Fig. 26.2). This usually resolves sponta-

neously after 6–12 months but if troublesome may require treatment and topical agents such as benzoyl peroxide or a topical antibiotic such as erythromycin. More severe cases may require referral to a specialist for systemic treatment such as erythromycin or trimethoprim for a few weeks or few months. It is important to note that oral tetracycline antibiotics should not be used in children under the age of 12 years old as they are associated with impaired bone growth, permanent discolouration of teeth and enamel hypoplasia in children [1–2]. Very severe resistant cases with scarring may need referral to a paediatric dermatologist for oral isotretinoin (“Roaccutae®”).

It is unclear if children with infantile acne are more prone to developing troublesome acne in their teens. If pre-pubertal children over the age of 2 years old present with acne they should be referred to a paediatrician for investigations for possible underlying hormone abnormalities such as Congenital Adrenal Hyperplasia, Cushing syndrome or an androgen secreting tumour.



**Fig. 26.2** Infantile acne

## 26.7 Keratosis Pilaris

This is a form of localised folliculitis caused by a disorder of keratinisation. This is very common in children and young adults. Cells get stuck in the hair follicles causing tiny plugs of keratin and gives a “goose pimple” appearance to the skin. This causes a coarse texture like sandpaper and is most commonly found in the outside of the upper arms, the outside of the thighs and on the cheeks (Fig. 26.3a, b). On the face there can be considerable erythema (Fig. 26.4). It often runs in families and is more common in children with atopic eczema. It usually improves as the child gets older. Treatment includes moisturisers or keratolytics such as urea, salicylic acid, tretinoin, adapalene or vitamin D analogues such as calcipotriol. A cream or lotion with 10% urea or higher for the body and 3% for the face can be tried. This can be applied, rubbing it downwards twice a day and can take months to see a good improvement. If this does not help, adapalene gel (“Differin Gel®”) can be tried but this can cause dryness and irritation in some patients so it should be applied sparingly to the affected areas on the face and body on alternate days initially.

## 26.8 Impetigo

This is more common in children than in adults. It is usually caused by *Staphylococcus aureus* or *Streptococcus pyogenes*. It usually presents as an asymmetrical “sore” with a yellowy gold coloured crust and exudate on exposed areas. It can spread to other parts of the body and to other children. As it is contagious, careful hand washing and using a separate towel for the infected child is important to protect the rest of the family. The crusty plaques are usually round, oozing and expand as the infection progresses. Milder cases will usually respond to topical antibiotics such as fusidic acid cream (“Fucidin Cream®”), which should be applied after gently removing the crust with an antiseptic wash. All orifices of the body should be treated regardless of impetigo location to ensure a focus for re-infection is cleared. More severe or extensive cases such as bullous impe-



**Fig. 26.3** (a) Keratosis pilaris. (b) Keratosis pilaris



**Fig. 26.4** Keratosis pilaris with ulerythema affecting the lateral eyebrows

tigo may require oral or systemic antibiotics usually with flucloxacillin. Children should be kept out of school until the crusts have dried out.

## 26.9 Tinea Capitis (Scalp Ringworm)

Tinea capitis is a dermatophyte infection that is far more common in children than adults. Although there is a wide local variation of the causative organisms throughout the world, *T.tonsurans* (human spread) and *T.canis* (cat or dog ringworm) are the most common fungus causing scalp ringworm. It usually presents with

a round patch of hair loss (alopecia) with associated redness, scaling, inflammation and possible pustules on the involved area of the scalp. Diagnosis is by taking skin scrapings or plucking hair from the involved area and sending them for fungal stain and culture. As it can take 2–4 weeks to get results back, treatment is normally initiated on clinical grounds alone. Tinea capitis always requires systemic treatment because topical anti-fungal agents do not penetrate down to the deepest parts of the hair follicle. Oral anti-fungals such as griseofulvin 10–15 mg/kg/day to be taken with food for 6–12 weeks may be required to clear scalp ringworm [3]. However, it is difficult to source this drug. Other anti-fungals that can be effective include terbinafine and itraconazole. Oral terbinafine (“Lamisil®”) is not licenced for children under 2 years of age (usually <12 kg) and the use of oral itraconazole (“Sporanox Capsules®”) in paediatric patients is not recommended unless it is determined that the potential benefit outweighs the potential risks. These oral agents work faster than griseofulvin, but still take 4–6 weeks to clear fungal scalp infections. All children in the family should be checked for scalp ringworm and also treated. Topical antifungal such as ketocanozal (“Nizoral shampoo®”) or terbinafine cream may reduce the risks of transmission while the systemic treatment is working. If tinea canis is diagnosed then the animal source should be identified and treated.

Some children can develop a more deep-seated fungal infection of the scalp with a secondary severe inflammatory response causing a large, boggy, oozing mass on the scalp known as a **kerion**. There may be localised enlarged lymph nodes and a lower grade fever which could be mistaken for an abscess. A kerion needs to be diagnosed and treated quickly because neglected cases can cause permanent scarring alopecia in the affected area of the scalp. The child will need to be treated with oral anti-fungals for at least 6–12 weeks. If there are signs of secondary bacterial infection, antibiotics may be necessary such as flucloxacillin for 1–2 weeks. The role of oral steroids is controversial but may be required if there is a lot of inflammation (1 mg/kg/day for

7 days). There is **no** role for incision and drainage or any other surgery procedures for a kerion.

## 26.10 Scabies in Babies

Scabies can present as a generalised itchy eczematous rash in babies. It can often be confused with other itchy rashes such as atopic eczema. There is usually history of the child being in contact with somebody else with an itch. The classical signs of scabies in babies is burrows on these soles of the feet. Infants with scabies can have lesions in the face too. This is not seen in older children or adults. The diagnosis can be confirmed by seeing the mite at the end of a burrow with a dermatoscope or removing the mite from the end of a burrow with a number fifteen scalpel blade or a green needle and lifting it onto a microscope slide. It can then be viewed by the doctor and parents. This usually guarantees 100% compliance with the treatment once the parents see the mite.

Treatment is similar to adults except the scalp and face should also be covered with a scabieside in children less than the age of 2 years old. Permethrin 5% cream (“Lyclear Dermal cream®”) is safe to use in children over the age of 2 months old. Like adults, it should be applied for 8–12 hours and then washed off. One further application should be applied 1 week later. All close household contacts and babysitters should also be treated simultaneously regardless of whether they are itching or not (see Chap. 35). For children under 2 months of age, the recommendation is 7% sulfur preparation for three consecutive nights and this may have to be repeated weekly for a few weeks.

**Infantile acropustulosis** causes small, itchy blisters and pustules on the palms and soles of infants in the first 2–3 years of life. It can occur with scabies or post scabies but in some cases there is no history of scabies. Dermoscopy is very helpful to try to identify scabies mites. When it occurs without scabies it usually responds to emollients, moderately potent topical steroids and sedating antihistamines if it is very itchy. Most cases resolve by the time the child is 3 years old.

## 26.11 Conclusion

Children are more prone to congenital and allergic diseases. They are also more prone to infectious illnesses. Treating skin disease in children can be more challenging as many of the treatment used in adults such as potent topical steroids or oral anti-fungal tablets are more likely to cause side effects in children or are not licensed to use in children.

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# Exanthems and Infectious Rashes in Childhood

27

David Buckley

## Key points

- Childhood exanthems are often caused by infections and present themselves in a wide spectrum of rashes usually with systemic symptoms such as fever and a flu-like symptom.
- Mass childhood vaccination now results in some previously common childhood exanthems being seen very rarely (e.g. measles and rubella).
- If a pregnant woman or an immune-compromised person is inadvertently exposed to the chickenpox virus, then giving varicella zoster immune globulin as soon as possible but within 96 hours of the initial contact can reduce the severity of disease but may not prevent it.
- Scarlet fever occurs when the bacteria that causes Group A beta haemolytic streptococcal tonsillitis or impetigo release an erythrogenic toxin that causes tiny pinkish red spots all over the body.
- If meningococcal disease is suspected then systemic benzoyl penicillin should be given IV or IM prior to hospital transfer.

## What to tell the patient/parent

- Chickenpox is contagious for 1–2 days before the rash appears and for 7 days after the first day of the rash appearing.

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- Most cases of measles nowadays are seen in children who have not completed their full course of the MMR vaccine.
- Congenital rubella can be prevented by ensuring that all women of child bearing age have their immunity status checked before trying to become pregnant.
- Meningococcal disease can cause a life-threatening septicaemia and/or meningitis which can kill within hours. The child is usually critically ill with a high fever, nausea, vomiting and perhaps signs of shock such as cold hands and feet. Limb pain is a common presenting sign. 50–70% have a non blanching rash.

## 27.1 Introduction

**Exanthem** is another term for a rash. Childhood exanthems are often caused by infections and cause a wide spectrum of rashes usually with systemic symptoms such as fever and a flu-like symptom. Some exanthems can also be caused by drugs such as antibiotics or non-steroidal anti-inflammatory drugs (NSAIDs) which can confuse the picture and make an accurate diagnosis difficult.

Childhood exanthems usually occur in infants and young children over the age of 6 months old as maternal antibodies protect the infant in the first few months of life. Mass childhood vaccina-

tion now results in some previously common childhood exanthems like measles and rubella being seen very rarely. There are many other childhood infectious diseases (mostly viral) that usually present with a rash, with or without a fever. Some have typical clinical features while others may need laboratory confirmation, especially for public health reasons. Many children can present with a non specific rash with or without a fever that can be associated with a number of harmless viral upper respiratory tract infections. Others may present early or late in an illness making it difficult to recognise the classical features of the rash. In some cases it is the associated features and the history of the illness that may give the clue to the diagnosis rather than the rash itself. All childhood infectious illnesses are more serious if the child is immune-suppressed (HIV/AIDS, transplant patients, or on systemic steroids). This chapter will also discuss the more potentially serious childhood infectious rashes and those that may require the child to be isolated or kept away from pregnant women (Table 27.1) [1, 2].

## 27.2 Chickenpox (Varicella)

Chickenpox is one of the most common childhood rashes. Although a vaccine is available in some countries (USA) it is not given routinely in Ireland. The child usually presents with a mild flu-like illness and small papules mainly on the trunk and face. These usually develop over the course of 24 hours into very small clear, fluid filled vesicles (Fig. 27.1). There is often a history of the child being in contact with somebody else with chickenpox. Although chickenpox is usually harmless and self-limiting, it can lead to pitted scars, especially on the face and can rarely lead to more serious complications (cellulitis, viral pneumonia or encephalitis) particularly in immune-compromised children (children with diabetes, HIV or on chemotherapy). Children with chickenpox usually do not have a fever so if the child becomes unwell with a high temperature he/she should be investigated for more serious complications. The varicella virus will respond to an oral or

intravenous anti-viral such as acyclovir provided it is started early enough in an attack (Table 27.2). However, most children do not need anti-virals. They should be treated symptomatically with antiseptic baths, soothing lotions and sedating antihistamines at night to relieve itch. Fusidic acid cream mixed with 1% hydrocortisone cream ("Fucidin H®") can be used for lesions on the face to try and suppress the inflammatory response and reduce the risks of secondary infection and scarring. Ibuprofen should be avoided as it very rarely can cause a severe skin infection in children with chickenpox. Paracetamol is safe. Children who have been in contact with the virus and expose themselves to sunlight can develop lesions limited to the sun-exposed areas.

Children with chickenpox should be kept away from pregnant woman. The incubation period for chickenpox is 10–21 days before the rash develops. Children are infectious until 7 days after the first day of the rash. If a pregnant woman or an immune-compromised person is inadvertently exposed to the chickenpox virus, then giving varicella zoster immune globulin within 96 hours of the initial contact can reduce the severity of disease but may not prevent it.

Chickenpox is contagious for 1–2 days before the rash appears and for 7 days after the first day of the rash appearing. The varicella virus remains dormant in the dorsal root ganglion for life after infection and may reactivate at a later date to cause shingles (herpes zoster).

## 27.3 Measles

Measles is now a rare but serious and potentially life threatening illness in children. Mass vaccination with the MMR (measles mumps and rubella) vaccine began in Ireland around 1989. Most cases nowadays are seen in children who have not completed their full course of the MMR vaccine. Children with measles are usually very sick with high fever, conjunctivitis, a cough and runny nose. They can develop white spots (Koplik's spots or sign) on the inside of their cheeks, which usually appear 1 or 2 days before they develop

**Table 27.1** Childhood infectious rashes

Disease	Causative organism	Incubation (days)	Period of contagiousness	Duration of rash (days)	Days from onset of illness to rash	Fever	Flu-like illness	Red blanchable rash	Vesicles or bullae	Mucous membranes (nose/eyes)	Special features	Treatment	Special precautions
Measles	Measles virus	7–14	2–4 days before rash, until 2–5 days after rash clears	3–7	3rd–5th	+++	+++	+++	–	++	Koplik spots inside cheeks 1–2 days before rash.	Symptomatic, isolation	Inform public health.
Rubella	Rubella virus	12–23	1 week before symptoms until 7 days after the rash appears	3–5	1st–2nd	+	+	–	–	+	Posterior cervical glands. May have polyarthralgia. Petechial rash on palate	Symptomatic	Inform public health and avoid pregnant woman.
Rosacea infantum	Herpes virus types 6 + 7	7–14	Clears most infectious during high fever before rash appears	1–2	4th	+++	+	+	–	–	Rash appears on 4th day as temperature settles	Symptomatic	
Erythema infectiosum (fifth disease)	Parvovirus B19	4–14	Before rash until few days after	5–10	1st–2nd	+	+	–	–	–	Red “slapped” cheeks. Lace like rash.	Symptomatic	Avoid pregnant woman.
Chickenpox (varicella)	Varicella	8–21	2 days before symptoms until all vesicles have crusted.	10	0–2nd	+/-	+/-	–	+ vesicles	–	Small vesicles start on trunk & face. Avoid Ibuprofen	Symptomatic. Antivirals. Varicella - zoster immunoglobulin for pregnant women	Avoid pregnant woman.
Hand, foot and mouth disease	Coxsackie virus A16	3–5	7–10 days. Stools infectious for 1 month	7–10	1st–3rd	+	–	–	On hands, feet & mouth	–	Small pink patches on hands & feet → greyish blisters	Symptomatic	Avoid pregnant woman.
Scarlet fever	Group A, Beta-hemolytic streptococcus	2–5	1 day before to 1 day after treatment begins	2–7	1st–2nd	++	+/-	+	–	+/-	Sore throat or impetigo. Strawberry tongue. Rash feels like sandpaper. Pastia lines in folds of skin	Oral or IV penicillin	

(continued)

Table 27.1 (continued)

Disease	Causative organism	Incubation (days)	Period of contagiousness	Duration of rash (days)	Days from onset of illness to rash	Fever	Flu-like illness	Red blanchable rash	Vesicles or bullae	Mucous membranes (nose/eyes)	Special features	Treatment	Special precautions
Glandular fever infectious mononucleosis	Epstein-Barr virus (herpes type 4)	4–6 weeks	May be for 1 year after symptoms clear	Varies	3rd–10th	+	+	In 10% of cases (90% if given Amoxicillin)	–	–	Sore throat and tonsillar glands enlarged.	Symptomatic	
Gianotti—Crosti syndrome (infantile papular acrodermatitis)	Various viruses	Varies	Varies	2–8 weeks	Varies	+	+	–	+/-	Rash spreads from thighs to buttox to outer arms to face. May be asymmetrical.	Rash spreads from Monospot +	Symptomatic	
Kawasaki disease	Not infectious	Not infectious	Not infectious	Varies	Varies	+>5 days	+	+	–	+ glove & stocking rash. Very irritable child. Strawberry tongue.	Glove & stocking rash. May develop complications = medical emergency.	IV immunoglobulin	Refer to paediatrician.
Staphylococcal scalded skin syndrome	Methicillin sensitive <i>Staphylococcus aureus</i>	1–4	1–2 days after antibiotics begun	2	2nd	+	+/-	–	+ Bullae –	Positive Nikolsky sign.	Positive Nikolsky sign.	IV flucloxicillin	Refer to paediatrician.
Meningococcal disease	Neisseria meningitidis	2–10	7 days before symptoms to 1–2 days after treatment started	Varies	8–24 hours	+/-	+/-	– (petechial in 60% of cases)	–	+/-	Petechial rash in 60% of cases. Limb pain. Cold hands and feet. May have meningeal signs. Medical emergency.	Give Benzoyl penicillin I.V. or I.M. and transport to hospital immediately.	Close contacts may need prophylactic treatment.



**Fig. 27.1** Chickenpox in a 9-year-old boy

**Table 27.2** Treatment of varicella (chickenpox) with Zovirax® (aciclovir) in severe cases or immunocompromised children: double strength suspension 400 mg/5 ml/100 ml

Dosage	Zovirax® double strength suspension
0–2 years	2.5 ml qid × 5 days = 50 ml
2–6 years	5 ml qid × 5 days = 100 ml
6–12 years	10 ml qid × 5 days = 200 ml
Adults and adolescents <sup>a</sup>	Use Zovirax® 800 mg Tablets, one tablet five times a day for 7 days

Dosing in children may be more accurately calculated as “Zovirax®” 20 mg/kg bodyweight (not to exceed 800 mg) four times daily

<sup>a</sup>Adults and adolescents may be treated with “Valtrex®” (Valacyclovir Hydrochloride 500 mg tablets) 1000 mg TID × 7 days

a generalised rash. The rash is morbilliform (measles like) but because of mass vaccination, many young doctors may never have seen a case of measles. The rash is macular (flat), erythematous (red) and blanches with pressure. It consists of red spots ranging from 0.1 cm to 1 cm in diameter. The spots may all join together, especially on the face to cause diffuse redness and diffuse

non-itchy red blanchable rash and is usually accompanied with a high fever. The rash usually starts on the face and behind the ears on the fourth or fifth day of the febrile illness and quickly becomes more generalised while sparing the palms and the soles. The rash begins to fade with fine brown scales after 3 or 4 days. Diagnosis is usually made for public health reasons using a viral nasopharyngeal swab and throat swabs for polymerase chain reaction. Blood can also be checked for measles IgM and IgG antibodies.

Treatment is usually symptomatic with supportive measures and isolation of the child at home or in hospital, if required.

An infected child is contagious from 2 days before any symptoms develop to at least 5 days after the onset of the rash. The incubation period is usually from 7 to 14 days.

## 27.4 Rubella

Rubella (German measles) is a far less serious illness than measles. It is now uncommon because of mass vaccination. It is important to diagnose rubella because of the high risk to the unborn child if a non-immune, pregnant woman is exposed to the virus.

The incubation period is between 12 and 23 days. A child is infectious from 7 days before the rash develops until 7 days after the rash appears. The rubella is most contagious when the rash is erupting. Infected children should be isolated at home until 7 days after the rash has appeared.

A child usually presents with a low grade fever, sore throat and runny nose. The rash usually begins on the face and spreads to the neck, trunk and limbs over the course of 1 or 2 days. It consists of light pink or red spots about 2 mm or 3 mm in diameter. The rash can be quite extensive and lasts 3–5 days. Itch may or may not be present. As the rash fades, the skin may dry out and scale. Enlarged posterior cervical glands almost always occur with rubella. Diagnosis can be confirmed by isolating the virus from throat swabs, blood, urine or spinal fluids. There is no specific treatment for rubella. The child should be kept well hydrated and treated with paracetamol for fever or pain.

Congenital rubella can be prevented by ensuring that all women of child bearing age have their immunity status checked before trying to become pregnant. If they have no immunity against the rubella virus then they should be vaccinated and should avoid pregnancy for at least 3 months after receiving the vaccine.

## 27.5 Roseola Infantum

This is a common viral illness caused by a particular strain of the herpes virus (type 6 and 7). It most commonly occurs in children less than 18 months old. The child usually has a high fever. There are usually no signs of an upper respiratory tract infection or meningeal infection present. The rash usually appears on day 3–5 of the febrile illness as the fever begins to subside. It causes small pink or red spots, 2–5 mm in diameter which blanch on pressure. The rash mostly occurs on the trunk but can spread to the face and limbs. The rash usually only lasts a few hours to 2 or 3 days at most. There may be erythematous papules on the soft palate and uvula known as Nagayama spots. The treatment is symptomatic for this usually mild and self-limiting illness.

The incubation period for roseola is approximately 7–14 days after exposure. The rash does not need to be treated as it usually not itchy. Temperature control is important particularly for children who are prone to febrile convulsions.

## 27.6 Fifth Disease (Slapped Cheek Syndrome)

Fifth disease is also known as **erythema infectiosum**. It is caused by the parvovirus B19 virus. It usually presents in an otherwise well child with red hot burning cheeks. General mild lace or network type rash on the limbs and trunk usually appears a few days (4 or 5) after the red cheeks appear. The rash fades spontaneously after a few days or a few weeks. Clusters of cases in families

or in schools can occur. By the time the rash appears, the child is no longer infectious and they can remain in school. The parvovirus B19 can be harmful to the unborn child, so infected cases should be kept away from pregnant woman. Treatment is usually symptomatic with cooling creams for the burning cheeks and paracetamol. The name “fifth disease” comes from its place on the French historical classification of childhood skin rashes which include measles (first disease), scarlet fever (second disease), rubella (third disease) and Dukes’ disease (fourth disease).

## 27.7 Kawasaki Disease

This is a rare but potentially fatal childhood exanthema of unknown cause. It was first described in Japan in 1967. It is more common in boys and in children of Asian descent. It usually presents as a very irritable young child under the age of 5 with a high, swinging fever and four of the following five cardinal signs of disease:

1. Rash—morbilliform, macular papular, erythematous or target like
2. Oral signs—strawberry tongue, red mouth, cracked lips
3. Eye signs—conjunctivitis without exudate
4. Peripheral limb signs—red swollen hands and feet
5. Lymphadenopathy often only on one side of the neck

An affected child may not have all the cardinal signs and some features may appear while others may disappear during an infection. There are no diagnostic tests so suspected cases should be seen by the paediatrician as 20% of untreated cases develop coronary artery damage. Treatment is usually with intravenous immunoglobulins. Children with Kawasaki disease are often unusually irritable. Kawasaki disease is not infectious and so the patient does not need to be isolated. Some recent cases have been associated with COVID-19 infections (see Chap. 33).

## 27.8 Gianotti-Crosti Syndrome

This is a characteristic response of the skin to a viral infection (like Hepatitis B, Epstein Barr (glandular fever virus), enterovirus, echovirus or respiratory syncytial virus). It mostly occurs in children between the ages of 6 months and 12 years and clusters may occur. Symptoms of URTI may precede a non-itchy papular rash which first develops on the thighs and buttocks and then spreads over the following few days to the outer arms and face. The trunk is usually spared and the rash may be asymmetrical. There may be a mild fever and adenopathy. The rash usually fades spontaneously over the following 2–8 weeks. Children suspected of having this rash should have blood tests for Hepatitis B, monospot and other viral illness.

## 27.9 Scarlet Fever

This occurs when the bacteria that causes Group A beta haemolytic streptococcal sore throats or impetigo release an erythrogenic toxin that causes tiny pinkish red spots all over the body. It is most common from the age of 2–10 years old. It usually has a 1–4 days incubation period. Older children usually develop lifelong protective antibodies against the streptococcal toxins. Scarlet fever usually starts with a fever, sore throat, swollen, red, strawberry tongue and a flu-like illness. The scarlet blotches usually develop on the second day of the illness and progress to give a sunburn appearance with palpable goose pimples (sandpaper texture). The classic red streaks (pastia lines) may appear in the folds of skin especially in the axillae and antecubital fossa. Scarlet fever usually responds to oral or intravenous Penicillin V or Erythromycin if the child is allergic to penicillin. The fever and constitutional symptoms should settle quickly with treatment and long-term complications from streptococcal toxins such as rheumatic fever and glomerulonephritis are fortunately rare. The rash fades with peeling over the following few days or weeks.

## 27.10 Infectious Mononucleosis (Glandular Fever)

Glandular fever is caused by a human herpes virus Type 4, also known as Epstein Barr Virus (EBV). It can occasionally be caused by cytomegalovirus or Toxoplasma gondii. Glandular fever usually presents in teenagers and young adults with a low grade fever, fatigue, a sore throat and glands in the neck. Saliva is the primary method of transmitting, hence the name “kissing disease”. Approximately 10% of cases will develop a faint generalised non-itchy rash, which first appears on the trunk and upper arms and later spreads to the face and lower arms. Another possible rash that can occur with glandular fever is a more intensely itchy, morbilliform rash that appears on the extensor surfaces and pressure points 7–10 days after inappropriate treatment with a beta-lactamase antibiotic such as ampicillin or cephalosporin (Fig. 27.2).



**Fig. 27.2** Infectious mononucleosis and a morbilliform rash as a result of the patient being given amoxacillin

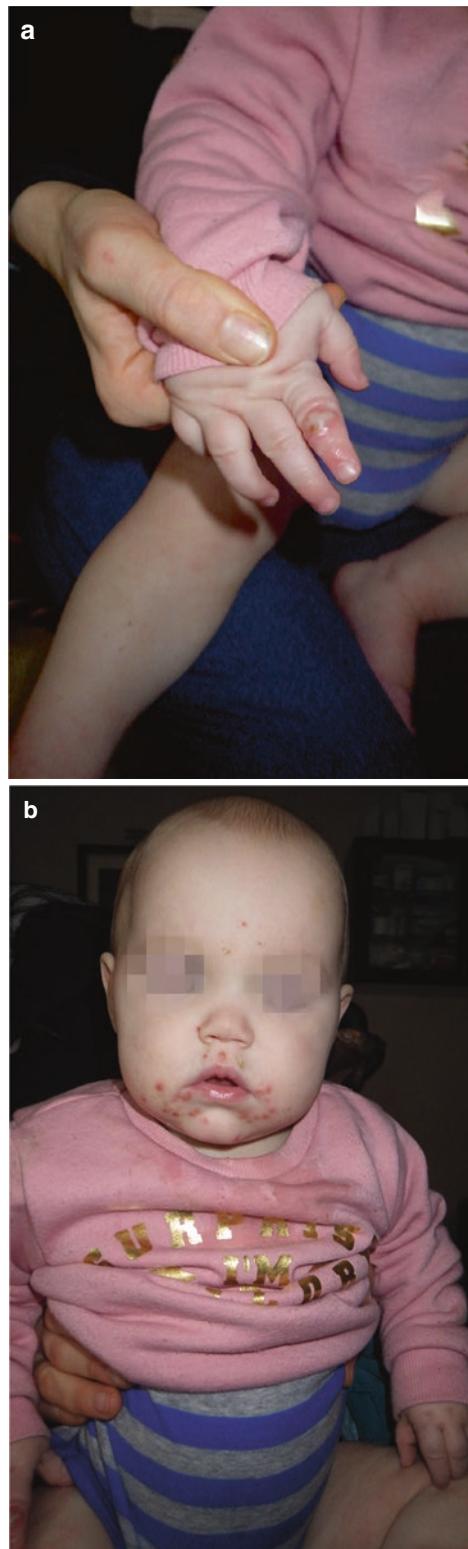
Although this presents like a drug rash, it is not a true drug allergy and will not reoccur if the patient gets the same antibiotic at a later date when he/she does not have glandular fever. This is one of the reasons why children with sore throat requiring antibiotics should be given penicillin V and not amoxicillin.

Glandular fever is usually confirmed by doing a blood test for a monospot which is positive. A lymphocytosis can be found. The treatment is usually symptomatic, although prolonged fatigue for weeks or months can occur in some cases.

## 27.11 Hand, Foot and Mouth Disease

This is a mild and short lived viral infection that most often occurs in children under the age of 5. It usually presents with blisters on the hands, feet and mouth (Fig. 27.3a, b). Clusters can occur within a family or school. It is caused by an enterovirus infection, usually coxsackievirus A16. The incubation period is usually 3–5 days when the child then develops a mild fever, sore throat and a flu-like illness. Pink patches develop on the front and back of the hands and feet, which are quickly followed by small greyish blisters. The rash usually resolves without scarring within a week. Small mouth ulcers may result in loss of appetite. Treatment is non-specific and symptomatic with analgesia, good skin care and a soft diet. Good hand washing techniques will reduce the spread of the disease. Stools can be infective for up to a month after the illness. Infected children should be kept away from pregnant women.

A recent coxsackievirus A6-associated enterovirus outbreak in Ireland was responsible for a potentially more widespread, severe, and varied disease than classic hand foot and mouth disease. This is known as **eczema coxsackium** and can be confused with bullous impetigo, eczema herpeticum, vasculitis, and primary immunobullous disease. It is more common in children with atopic eczema. These children usually have an extensive rash with widespread



**Fig. 27.3** (a) Hand foot and mouth disease. (b) Hand foot and mouth

vesicles, bullae, and/or erosions similar but less severe than classical hand, foot and mouth disease. These children are usually not very sick and most recover spontaneously after a few weeks.

## 27.12 Meningococcal Disease

This is a serious acute bacterial infection caused by *Neisseria meningitis*. It can cause a life-threatening septicaemia and/or meningitis which can kill within hours. The child is usually critically ill with a high fever, nausea, vomiting and perhaps signs of shock such as cold hands and feet. Limb pain is a common presenting sign. Some children may have little or no fever especially if they have been given antipyretics recently, if they are seen very early in the course of the disease or if they are in shock. It can occur at any age but it is most common in otherwise healthy children aged from 6 months to 4 years of age. Fifty to seventy percent of patients will develop a petechial rash (red or purple spots from leaking capillaries) that, unlike the measles rash, will not blanch on pressure. The rash may progress to bruising or even frank necrosis in extreme cases. Some children may have meningeal signs.

Meningococcal disease is a medical emergency and the child needs to be transferred to hospital immediately. If meningococcal disease is suspected then systemic benzoyl penicillin should be given IV or IM prior to hospital transfer.

Mass vaccination with the meningococcal C conjugate (Men C) vaccine started in Ireland in the year 2000 and the incidence of meningitis C infections has reduced by 96% since then. However, meningitis C only accounted for 30% of cases of meningococcal disease prior to mass vaccination. Meningitis B is now the predominate cause of meningococcal disease in Ireland. An effective vaccine against *Neisseria meningitis* sub group B was introduced into the childhood vaccination schedule in the UK in September 2015 and in Ireland in December 2016 (see Chap. 54).

## 27.13 Staphylococcal Scalded Skin Syndrome (SSSS)

SSSS is caused by methicillin sensitive staphylococcus aureus (MSSA). This organism can release two exotoxins that cause blistering and bullae. The condition usually occurs in newborns, young children and immuno-compromised adults. It usually presents with a fever and erythema. Bullae (large blisters) form and rupture to leave areas of denuded skin which look like burns or scalds. In newborns the lesions are most commonly found on the perineum and/or the peri-umbilical area while the extremities are more common in older children. Some cases could be confused with a non accidental injury. This condition is infectious and clusters of cases have been identified. Cases need to be isolated and strict hand hygiene needs to be implemented. Treatment is usually with penicillinase resistant anti-staphylococcal antibiotics such as flucloxacillin. Some children can be very sick and may need supportive treatment in intensive care.

Children with SSSS have very delicate skin which can tear even when lightly rubbed and results in exfoliation of the outer most layer (positive Nikolsky sign). Mucous membranes are usually spared, which is in contrast to Stevens Johnson syndrome, which is usually caused by an allergic reaction to various medications resulting in sheet like skin and mucosal loss.

Protective antibodies against staphylococcus exotoxins are usually acquired after an infection, therefore recurrence is rare.

## 27.14 Conclusion

Most childhood rashes are harmless and self limiting. Many are due to non-specific viruses and do not require any specific treatment. Occasionally a child may present with a fever and a more serious rash that may require urgent treatment or isolation. Certain childhood infectious diseases that cause a rash can be dangerous to pregnant women, so serology and cultures may be required

to make an accurate diagnosis to assess the risk to any pregnant women in close contact with the child. Diseases like measles and rubella are now quite rare because of mass vaccination. This means that many younger doctors may never have seen a child with these viral illnesses and may find it difficult to diagnose these rashes on clinical grounds alone.

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# Genodermatoses: Inherited Skin Diseases

28

David Buckley

## Key points

- Genodermatoses are genetic skin conditions which can be classified into these three categories: chromosomal defect, a single gene defect or polygenic. The condition may be inherited or due to a new mutation.
- Some genodermatoses are obvious at birth (congenital) while others may only become apparent in childhood or adult life (e.g. tuberous sclerosis).
- In albinism, lack of pigment can result in severe visual problems and makes the patient particularly sensitive to UV damage and skin cancer.

## What to tell the patient

- **Most cases of neurofibrosis 1** are mild but neurological manifestations can occur in up to 40% of patients especially intracranial tumours, spinal cord tumours, peripheral nerve tumours and epilepsy.
- 60–70% of patients with tuberous sclerosis will have epilepsy or learning disabilities which may be present from birth or develop in adolescence or early adult life.

## 28.1 Introduction

Genodermatoses are skin disorders that are inherited as a result of a genetic (chromosomal) defect. Some may run in families (inherited skin disorders) while others may occur as a result of a new mutation. Some are obvious at birth (congenital) while others may only become apparent in childhood or adult life (e.g. tuberous sclerosis). Patients and families may need genetic counselling to assess the risk of having more children with a certain chromosomal disorder.

## 28.2 Down Syndrome (Trisomy 21)

Most children with Down's syndrome are diagnosed at birth or shortly after. They usually have normal skin at birth but can develop various skin problems as they get older including an increased risk of developing dry skin, skin infections, atopic disease, psoriasis, autoimmune disease and premature aging of the skin. Young adults with Down's syndrome are more likely to develop truncal folliculitis on their presternal and interscapular area due to Malassezia folliculitis which responds well to oral itraconazole. Children with Down's syndrome are more likely to develop alopecia areata and hidradenitis suppurativa.

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## 28.3 Neurofibromatosis

This is a relatively common autosomal dominant condition but almost 50% of cases have no family history. It occurs in 1 in 3000 people. There are two main types of neurofibromatosis (**type 1** and **type 2**):

### 28.3.1 Neurofibromatosis Type 1 (von-Recklinghausen's = NF1)

NF1 represents 85% of all cases.

It is a rare (1 in 4000 births) autosomal dominant disease characterised by the presence of two or more of the following:

- (a) Six or more *café-au-lait* spots greater than 5 cm in pre-pubertal children and greater than 15 cm in adults
- (b) Axillary or inguinal freckling (70% of cases)
- (c) Two or more neurofibromas
- (d) Two or more Lisch nodules (small circle pigmented iris hamartomas—best seen with a slit lamp)—(90% of cases)
- (e) Optic glioma
- (f) A distinctive osseous lesion (e.g. sphenoid dysphasia or thinning of the long bone cortex)
- (g) A first degree relative with NF1

*Café-au-lait* macules are usually the first sign of the disease and they appear in all children with NF1 by the age of 4. They are sharply defined, light brown macules, varying in size from 0.5 cm to 50 cm, but the majority are less than 10 cm in children.

Neurofibromas occur as a result of benign tumours surrounding nerves which present as soft, lilac-pink, sessile dome shaped or pedunculated tumours mostly on the trunk and limbs ranging in size from a few millimetres or several centimetres in diameter. They can be present in their hundreds (Fig. 28.1).

Elephantiasis neurofibromatosis is caused by neurofibromas of the nerves with associated overgrowth of subcutaneous tissue and skin which can produce gross disfigurement as



**Fig. 28.1** Neurofibromatosis type 1

depicted in the movie “The Elephant Man”. However, most cases of NF1 are mild and never develop any major complications.

Neurological manifestations occur in 40% of patients especially intracranial tumours, spinal cord tumours (**which can lead to scoliosis**), peripheral nerve tumours and epilepsy. This **can result** in both behavioural and learning difficulties. Some patients can have internal organ involvement with neurological, GI, GU or cardiovascular problems. Genetic counselling is important as 50% of children of parents with NF1 are likely to be affected by the disease.

### 28.3.2 Neurofibromatosis Type 2 (**Bilateral Acoustic** **Neurofibromatosis**)

This is a separate entity to NF1 with the affected gene on chromosome 22. Like NF1, patients can develop *café-au-lait* spots and cutaneous fibro-

mas but they also develop acoustic neuromas as well as other CNS tumours. The usual age of first symptoms (i.e. hearing and balance problems) is around 20-years of age (range 2–52) and cataracts are present in 80% of the cases.

## 28.4 Tuberous Sclerosis

Tuberous sclerosis is an autosomal dominant disorder of hamartoma formation in many organs especially in skin, brain, eyes, kidney and heart. It is uncommon with an incidence of 1 in 10,000. Approximately 60–70% of cases are thought to be new mutations. The typical skin lesions are as follows:

- (a) Angiofibromas—these usually appear from the age of 3–10 years old. They are firm, discreet, reddish brown, telangiectatic papules ranging from 1 mm to 10 mm in diameter on the cheeks and chin and often mistaken for acne (Fig. 28.2).
- (b) Shagreen patch—this is an irregular, thick, macular, papular, soft skin coloured plaque usually in the lumbo-sacral area that causes no symptoms (Fig. 28.3)
- (c) Periungual fibromas—these appear at or after puberty as smooth, firm, fleshy, warty benign growths arising from the proximal nail folds and can grow to 5 mm or 10 mm long (Fig. 28.4a, b).
- (d) Ash leaf-shaped white macules—these are white ovoid macules measuring 1–3 cm usually on the trunk or limbs (Fig. 28.5). They are usually the first cutaneous sign of tuberous sclerosis. They may be present at birth or shortly after and can be a clue to the diagnosis in a child with epilepsy. However, these macules are often seen in normal babies without tuberous sclerosis.

60–70% of patients with tuberous sclerosis will have epilepsy, intellectual disabilities and developmental delay which may be present from



**Fig. 28.2** Tuberous sclerosis with tiny angiofibroma on the nose



**Fig. 28.3** Tuberous sclerosis with a Shagreen patch

birth or develop in adolescence or early adult life. Tumours may also occur in the heart, kidney, lungs, GI tract and brains.



**Fig. 28.4** Tuberous sclerosis: (a) periungueal fibroma with possible fungal nail infection, and (b) periungueal fibroma with secondary nail dystrophy



**Fig. 28.5** Cafe au lait spots, ovoid white (ash leaf) macules and Shagreen patch in tuberous sclerosis. Photo courtesy of Dr Myriam Raquel González Oviedo

## 28.5 Ehlers-Danlos Syndrome

Ehlers-Danlos syndrome is a genetic connective tissue disorder resulting in fragile and hyperelastic skin, hypermobile and easily dislocatable joints, scoliosis and fragile blood vessels (easy bruising). Internal organs may be involved including the heart valve (mitral valve prolapse) GI, GU, eyes, spine and gums. The skin feels soft and boggy. Some patients may have hyper-mobile joints without skin involvement. Clinical features including osteoarthritis, usually first appear in young adults.

## 28.6 Darier's Disease

This is a rare autosomal dominant disease that causes a disruption of keratinisation resulting in various abnormal skin and nail manifestations. Patients often have a family history of Darier's disease. They usually present in late childhood or in early adult life with greasy, scaly papules on the face and trunk in a seborrhoeic dermatitis pattern. It can sometimes spread in a dermatomal distribution. It can also affect the flexures. It may be mistaken for other skin conditions such as acne, psoriasis and seborrhoeic dermatitis. Subtle signs such as longitudinal red or white streaks on the nails and palmer pits may help make the diagnosis. Histology is usually characteristic in Darier's disease.

The clinical features are variable with many patients having mild disease while others may have extensive disease. Patients with Darier are more prone to skin bacterial and viral infections such as widespread herpes simplex.

Treatment will depend on the severity of the disease. Mild cases may only require emollients and photo protection. More troublesome cases may require topical or oral retinoids.

## 28.7 Albinism

Albinism is an autosomal recessive (but some forms are X-linked) genetic deficiency of melanin pigment production. Oculocutaneous albinism (OCA) affects the eyes, hair and skin, whereas only the eyes are affected in ocular albinism (OA). While most people with albinism have very light or white skin and hair, levels of pigmentation can vary depending on the type of albinism. OA, which is much less common, involves only the eyes, while skin and hair may appear similar or slightly lighter than that of other family members.

Approximately 1 in 17,000 people in Europe have one of the types of albinism, although it is much more common in East Africa. Lack of pigment results in severe visual problems and makes the patient particularly sensitive to UVL damage and skin cancer. Sun glasses and sun protection

for the skin with high SPF sun blocks, hats and appropriate clothing is vital from birth. Life expectancy is not affected provided the patient does not succumb to skin cancer.

Very rare genodermatoses such as **Epidermolysis bullosa** are covered in other chapters (see Chap. 23) or should be reviewed in major text books in dermatology (see Chap. 67).

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## 28.8 Conclusion

Genodermatoses are genetic skin conditions which can be classified into these three categories: chromosomal defect, a single gene defect or can be polygenic. The condition may be inherited or due to a new mutation. Early diagnosis is important to limit morbidity and mortality. Genetic counselling is vital if the affected person is planning a family.



# Congenital Nevi, Melanocytic Naevi (Moles) and Vascular Tumors in Newborns and Children

29

David Buckley

## Key points

- Melanoma is extremely rare in children under the age of 12 years.
- Spitz Nevus is a pink or brown dome shaped nodular nevus that usually grows rapidly. They are sometimes removed for histology as they can resemble a melanoma clinically.
- Blue naevi are harmless.
- Congenital melanocytic nevi less than 20 cm in diameter have a very low potential to turn malignant and should usually be left alone in childhood. Check up by specialists with dermoscopic experience is suggested.
- The most common lesion in children under the age of 1 year old that may need urgent evaluation by a dermatologist is a large rapidly growing strawberry nevus (capillary haemangiomas) on the face, especially if it is adjacent to vital structures such as the eyes, nose or mouth.

## What to tell the parents

- Stork bite birthmarks (Nevus Flammeus Nuchae) are harmless and will fade spontaneously.
- Most strawberry naevi will regress and clear without scarring after a few years.

- Small capillary haemangioma <5 cm (Strawberry Haemangioma) on the trunk, limbs and scalp are probably best left untreated as 90% will clear spontaneously by the time the child is 9 years old. Follow up is important until obvious signs of regression are present. They should not be confused with capillary malformations (Port wine stains) which do not clear and in fact tend to get darker and thicker with time.
- If an existing or new mole is changing in size, shape or colour or looks completely different from all the other moles (the ugly duckling sign), it needs to be checked by a doctor with experience in dermoscopy and lesion recognition.

## 29.1 Introduction

Small babies often present with red or brown naevi or vascular tumors which can be of considerable concern to the parents but are rarely dangerous or life threatening. Naevi can present at birth (congenital) or may develop during childhood (acquired) after the age of 2 years old. Although generally divided into vascular (arise from blood vessels) or melanocytic (arise from melanocytic, pigment producing cells), the term “mole” should be reserved to melanocytic lesions while vascular lesions are referred to as tumors and/or malformations.

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## 29.2 Congenital Vascular Lesions

**Naevus simplex** is the most common congenital nevus seen in children. It is usually due to a minor vascular malformation. It presents as faint, flat, pink capillaries on the back of the neck (Nevus Flammeus Nuchae or “stork bits”), eyelids or the forehead (“angel’s kiss”) (Fig. 29.1). It occurs in approximately a third of all children. The ones on the face usually fades after a year or two. No treatment is necessary and the parents should be reassured they fade spontaneously as the child gets older. Nevus Flammeus Nuchae may persist into adult life.

**Strawberry Haemangioma** (capillary haemangiomas) occur in almost 10% of children and are more common in girls, in premature babies, low birth weight babies, twins and triplets. They are due to a benign tumour of the endothelial cells that line the blood vessels causing an abnormal collection of normal blood vessels under the skin. They are not normally present at birth but usually develop in the first few weeks of life and grow rapidly. 60% occur on the head and neck. Their location will determine their color: superficial ones tend to be brilliant red; deep ones are blue subcutaneous nodules. “Mixed” hemangiomas are tumors partly located in the surface and partly deep seated. They have an elevated red brilliant strawberry part with an underlying blue deep seated part. 80% of haemangiomas have reached 80% of their maximum size by the time the child is 3–6 months old. Small capillary haemangiomas ( $<5$  cm) on the trunk, limbs and



**Fig. 29.1** Naevus simplex (Angel’s kiss) present in an 18-month-old baby



**Fig. 29.2** Strawberry haemangioma in an 11-month-old child

scalp are probably best left untreated as 90% will clear spontaneously by the time the child is 9 years old (Fig. 29.2). Children with multiple haemangiomas ( $>5$ ) should have an ultrasound of the liver to rule out liver haemangiomas. Haemangiomas on the face, neck or in the midline of the body may be a risk factor for haemangioma in the airway which usually presents as difficulty feeding, stridor or hoarseness. Urgent treatment may be required for very large or rapidly growing capillary haemangiomas especially if they are interfering with vital structures such as the eyes, nose and mouth or if they are ulcerating. Most will respond to oral propranolol with or without steroids under paediatric supervision. Topical beta blockers such as timolol maleate (“Timoptol® 0.5% eye drops” normally used for glaucoma—one drop applied to the haemangioma daily) and a topical steroid such as clobetasol (“Eumovat®”) may help for small haemangiomas or while waiting for an urgent appointment for a more serious haemangioma. Other interventions have been used like steroid infiltrations and vascular lasers.

In Ireland there is a National Pilot for Photo-Triage of Referrals for Haemangiomas. General Practitioners can email a photograph of the haemangioma together with the child's date of birth via a secure e mail known as Healthmail to: hse.haemangioma@hse.ie

A paediatric dermatologist will review the photograph within five working days and contact the general practitioner via phone or email with a clear referral pathway dependent on the geographical location of the patient.

**Port Wine Stains (Nevus Flammeus)** are vascular malformations that are usually present at birth. It occurs in 1 in 300 births. Unlike capillary haemangiomas, port wine stains are usually permanent and will grow as the child gets bigger. In adults, they can get thick and papular (Fig. 29.3). They can occur on any part of the body and usually follow the lines of the dermatomes. When they occur on the face, they can cause considerable cosmetic concern. Occasionally, there may be part of syndromes which include involvement of the underlying cerebral leptomeninges and eye that can cause neurological defects and visual disturbance (Sturge-Weber syndrome). Sturge-Weber syndrome should be suspected when a PWS involves the forehead (especially if it involves both sides) or more than one dermatome.

Port wine stains are best treated when the child is very young as the skin is thinner and vessels are more susceptible to laser treatment. Depending on the size, this laser sessions can be done under topical or local anesthesia. Numerous laser treatments may be required over many

months or years to improve the cosmetic appearance particularly when the port wine stains affect the face.

### 29.3 Congenital Melanocytic Nevi

**A congenital nevus (mole)** is a proliferation of benign melanocytes, also known as a birthmark, that develops before or shortly after birth. They are usually classified according to their size (Table 29.1). They are usually round or oval and can be any shade of brown or pink (Fig. 29.4). They can be smooth (macular) or bumpy (papular). They are usually permanent but can get rougher, warty and hairy as the child gets older (Figs. 29.5 and 29.6). Small CN have a very low

**Table 29.1** Congenital melanocytic nevi classification

Small	<1.5 cm
Medium	1.5–10 cm
Large	10–20 cm
Giant	>20 cm (e.g. bathing trunk nevi)



**Fig. 29.4** Congenital nevus (mole) in a 2 year old



**Fig. 29.3** Port Wine Stains (Nevus Flammeus) on the thigh



**Fig. 29.5** Congenital nevus (mole) in a 2-year-old girl



**Fig. 29.6** Warty congenital naevus in an adult

potential to turn malignant but it may be advisable to have a specialist in dermatoscopy evaluate it. Giant CN need to be under close monitoring by specialists in melanocytic lesions. Any change or outgrowth needs to be biopsied urgently for evaluation to rule out the presence of melanoma. If a medium or large congenital nevus needs to be removed for cosmetic reasons, this should be done by a specialist when the child is old enough to tolerate local or general anaesthetic (e.g. >12 years old).

## 29.4 Acquired Melanocytic Nevi

**Acquired nevi** usually develop as the child is older (>2 years old) and are generally smaller than congenital nevi. Most acquired moles present in adulthood first appear in childhood. Teenagers and young adults have the greatest

number of moles and some resolve spontaneously as the person gets old. The average Irish person has approximately 20–50 moles in adulthood. Acquired nevi are usually small, pink, tan or brown coloured and may be flat or slightly raised. They all grow uniformly as the child gets bigger (Fig. 29.7). The development of a melanoma from an acquired mole is extremely rare in a child under the age of 12. However, if an individual mole is changing in size, shape or colour or looks completely different from all the other moles (the ugly duckling sign), it should be monitored closely. The patient should be referred or the mole removed if there is a suspicion of a melanoma.

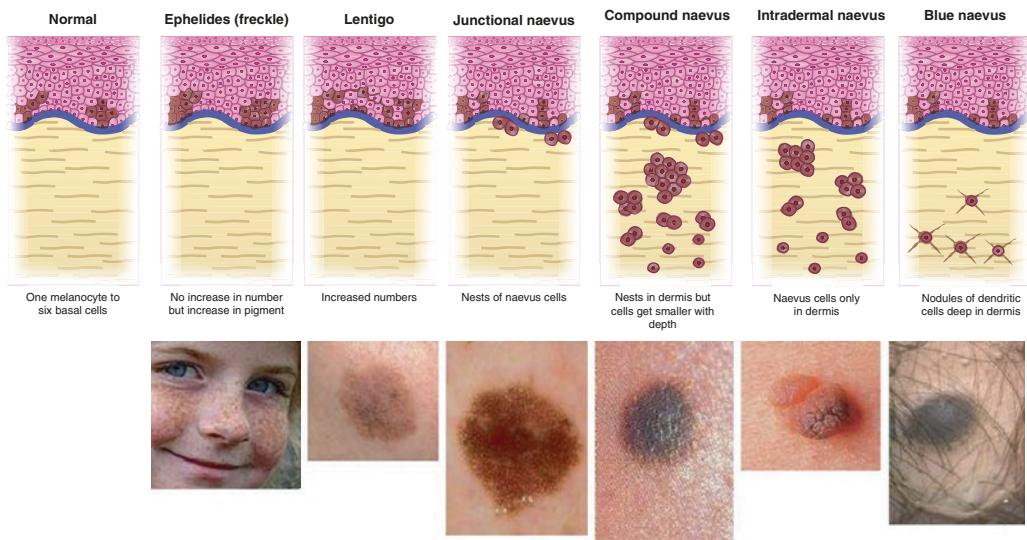
**Ephelids** (freckles) are small 1–2 mm brown macules usually found on the exposed sites such as the face, arms and legs in children and can number from one or two up to hundreds. They usually appear after sun exposure and fade in the winter. They should be differentiated from **juvenile lentigines** (2–10 mm) which appear early in childhood and solar lentigo (2–20 mm) that occur in the elderly from excessive UVL.

There are a number of unusual congenital nevi such as the following:

**Café au lait macules** are light, brown coloured (colour of coffee with milk), oval, flat, hairless moles that can appear in early childhood. Approximately 10–30% of the general population have an isolated cafe au lait macule. If they are multiple (>6) and large (>5 cm in children or >15 mm in the postpubertal age) they may be a marker for neurofibromatosis type 1 (cafe au lait macules occur in 95% of cases of NF type 1), tuberous sclerosis, Albright syndrome or Fanconi anaemia.

**Sebaceous nevus** (also called **nevus sebaceus**) is a rare birthmark that is usually found on the scalp in children. It is a benign hair follicle tumour and is usually a solitary, oval, yellow-orange patch with no hair. They become more warty in adolescent and early adult life (Fig. 29.8). A small proportion can develop into a BCC or SCC. They can usually be left untreated but should be biopsied if they suddenly grow, bleed or become tender, which may be a sign of

## Nevi types & Pathology:



**Fig. 29.7** Acquired Naevi. Adapted from: <http://www.slideshare.net/vmshashi/pathology-of-skin-common-disorders>



**Fig. 29.8** Sebaceous naevus in a 16year old

malignant transformation, but if this was to occur it would usually be in adulthood.

**Blue naevi** are deeply pigmented with a deep blue or slate grey colour. They may be flat or raised. They are harmless moles that are generally stable and show typical features under a dermatoscope. They usually appear first in older children and teenagers. No treatment is required unless for cosmetic reasons.

**Halo nevus** (also known as “Leukoderma acquisitum centrifugum,” “Perinevoid vitiligo,” and “Sutton nevus”) usually occurs in children

or young adults where a stable mole develops a symmetrical ring of hypo-pigmentation around the border measuring approximately 0.5–1 cm wide. This is usually a sign that the mole is regressing and the mole will disappear in time. No treatment is required provided the mole in the centre is not getting bigger, changing in shape or developing into different colours. The hypo-pigmented ring around the mole should be protected with a sun block, as this area will burn easily. In some children it can occur in several or even all naevi. It tends to have no relevance.

**Spitz Nevus** is a pink or brown dome shaped nodular nevus that usually grows rapidly for a few months on the face or limbs in a child (Fig. 29.9a). They usually stop growing after a few months and may remain stable for months or years. Many will disappear spontaneously in time. Although these are benign, there is a rare form named atypical Spitz nevus with unknown clinical prognosis. They can have clinical and histologic traits that share features with melanoma. They should be removed.

**Spider naevus** is an acquired vascular malformation which are common in children, in pregnant women, in women on oestrogen containing contraceptives and in liver disease (Fig. 29.9b). They arise as a result of a solitary dilated blood vessels which has tiny radiating tributaries that look like spiders legs (hence the name). They disappear on compression (e.g. with a glass slide) and the central large blood vessel refills first followed by the small tributaries once the compression is released. They often disappear spontaneously. If they persist and are unsightly they can easily be removed with electrosurgery, cryosurgery or laser treatment.

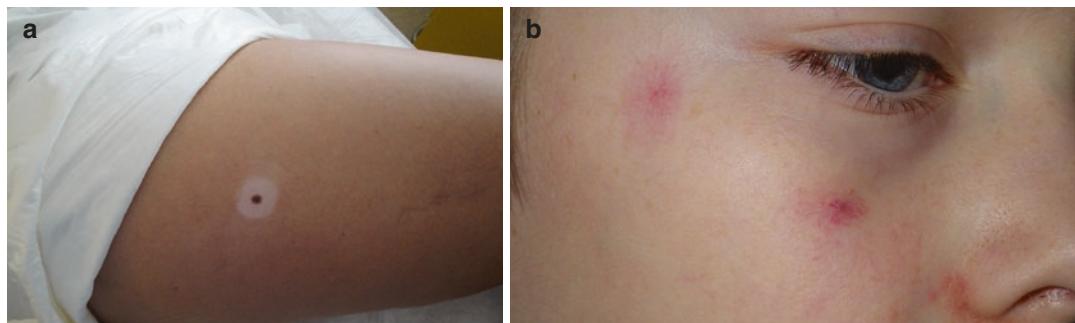
**Linear epidermal nevus** is due to an overgrowth of the epidermis and usually present at birth or during early childhood. They present as brown macules or papules in a linear (straight line) fashion along the lines of embryonic development (lines of Blashko) (Fig. 29.10). They are usually found on the trunk or limbs and are normally unilateral (Fig. 29.11). They often become more raised and warty as the child gets older. They are harmless and benign and usually do not require any treatment unless for cosmetic reasons.

Naevus **depigmentosus** (also called achromic naevus) present as well-defined pale patch on the trunk or limbs in newborns or children. Unlike vitiligo where there is complete absence of pigment, with naevus depigmentosus the affected area is hypopigmented but not completely white. In the affected areas the melanocytes do not produce enough melanin. Naevus depigmentosus

can cause isolated hypopigmented patches or can be segmental or linear. The ash-leaf macules seen in tuberous sclerosis are oval in shape and, although they may look similar to achromic naevi, they usually present as multiple lesions. Treatment of naevus depigmentosus is usually not necessary but cosmetic camouflage will help if on exposed sites.



**Fig. 29.10** Linear epidermal naevus in a 14 year old



**Fig. 29.9** (a) Compound naevus with some Spitzoid features on histology. It looks like a halo naevus but the hypopigmentation is from band aids. (b) Spider naevi on a 10 year old



**Fig. 29.11** Linear epidermal naevus in an adult

**Mongolian Blue Spot** is also known as lumbosacral dermal melanocytosis. It causes a blue-grey staining of the skin that usually affects the lower back and buttocks in newborns although they can be present mostly anywhere in the skin surface. The patches can vary in size from a few centimetres to covering the whole of the buttocks. Occasionally, other sites such as the face or limbs can be affected. The skin on the affected areas is entirely normal with no textural changes and no excessive hair growth. It is more common in children with dark skin types. No treatment is required and the discolouration usually clears spontaneously as the child reaches school going age.

**Becker naevus** is an unusual large acquired mole that develops usually on the upper trunk in older children or young adults. They are far more common on boys than girls. It is usually light brown and can have associated hair growth. Some can cover half the upper chest wall or shoulder (Fig. 29.12). They are harmless and are usually



**Fig. 29.12** Becker naevus in an 18 year old

left untreated as they have little or no potential to turn malignant. Sun protection will make them less obvious and some adults might want the excessive hair removed for cosmetic reasons.

## 29.5 Conclusion

Nevi are common in childhood. Melanoma is extremely rare before puberty. Rapidly growing strawberry nevus (capillary haemangiomas) on the face, especially if it is adjacent to vital structures such as the eyes, nose or mouth, atypical Spitz nevi and changing congenital melanocytic moles are emergencies that justify a specialist intervention.

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**Part VI**

**Infections, Infestations and Bites**



# Common Bacterial Skin Infections in General Practice

30

David Buckley

## Key points

- Various physical, chemical and immunological alterations to the skin can disrupt the skin barrier function, predisposing to bacterial penetration.
- Treatment of bacterial infections will depend on the severity (depth) of infection, the sensitivity of the organism to antibiotics and the patient's immune competency.
- Most bacteria that cause skin infections are ones that colonise the skin or mucus membranes.
- Superficial bacterial infections such as impetigo and folliculitis are usually caused by *staphylococcus aureus* and respond to flucloxacillin.
- Deeper infections such as cellulitis are caused by beta haemolytic streptococci and usually respond to Penicillin V (phenoxymethypenicillin). More severe infections may need a combination of penicillin v and flucloxacillin at high doses orally or intravenously.
- All patients with bacterial skin infections should have their blood sugar checked.

## What to tell the patient:

- Finish your complete course of antibiotics as prescribed—otherwise you run the risk of the infection coming back and becoming resistant to commonly used antibiotics.
- Careful hand washing and using your own face cloths and towels is important to prevent others getting your infection.
- Do not smoke or drink alcohol when you have an infection as they will delay wound healing.

## 30.1 Introduction

Skin infections are usually obvious with a combination of redness, swelling, heat and pain. Superficial infections may have exudate or pus. With more deep seated infection, temperature and systemic upset with flu like symptoms may occur. Treatment will depend on the severity (depth) of infection, the presumed organism and the patient's immunocompetence.

Skin infections usually result from an imbalance between the pathogenic power of micro-organisms and the immunological defences of a patient. Various physical, chemical and immunological alterations to the skin can disrupt the skin barrier function, predisposing to bacterial penetration. Once the bacteria has penetrated the skin's defensive layer and their virulent factors have overcome local host defences, tissue inva-

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**Table 30.1** Bacterial skin infections

<b><i>Staphylococcus Aureus</i></b>
(Usually <u>superficial</u> skin infections—epidermis)
Sensitive to flucloxacillin (or clarithromycin if penicillin allergic)
Impetigo
Folliculitis
Boils/carbuncles
Exacerbation of eczema
Cellulitis (1/3 of cases)
<b><i>Streptococcus Pyogenes</i></b>
(Usually <u>deep</u> skin infection—dermal)
Sensitive to Penicillin V or Benzoylpenicillin (or clarithromycin if penicillin allergic)
Erysipelas ( <i>Group A Beta haemolytic streptococci</i> )
Cellulitis (2/3 of cases)
(Impetigo sometimes)

sion and infection occur. Loss of the skin barrier may be caused by lacerations, bites, surgical wounds, scratching, burns, ulcers, inflammatory dermatoses (eczema/dermatitis), viral or fungal infections. Skin infections located on the groin, fingers, toes and the head are more likely to become complicated.

Most bacteria that cause skin infections are ones that colonise the skin or mucus membranes. Gram-positive bacteria are the most frequently isolated with a prominence of *staphylococcus aureus* and *streptococcus pyogenes* (Table 30.1). The particular type of skin infection that these organisms cause will largely be determined by how they invaded the skin barrier and the depth of invasion (Fig. 30.1).

Group B Streptococci (*Streptococcus agalactiae*) are frequently identified in diabetic patients. *Pseudomonas aeruginosa* is often isolated from lower extremity infections particularly in cases of peripheral vascular disease or puncture wounds and in cases involving hydrotherapy (e.g. Jacuzzi folliculitis).

## 30.2 Bites

Gram-negative organisms are more likely in animal or human bites, surgical infections and in IV drug users, and usually require a beta-lactamase antibiotic such as a amoxicillin/clavulanic acid or clarithromycin if allergic to penicillin. Good sur-

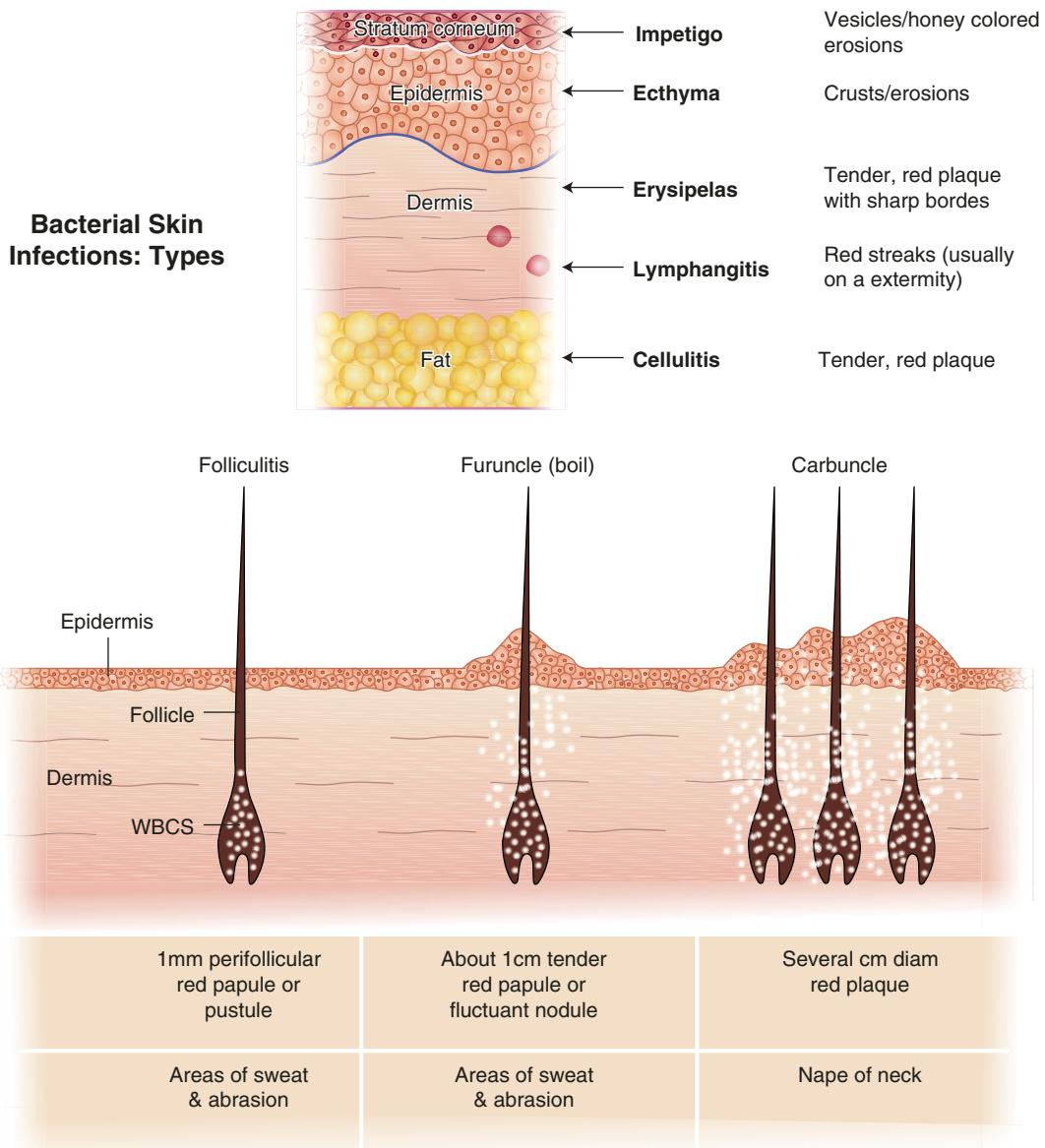
gical toilet under local anaesthetic is most important for bites. With animal bites consider tetanus risk and for human bites consider HIV, Hepatitis B and C risk.

Although specific bacteria may cause a particular type of skin infection, considerable overlap in clinical presentations remain.

Most patients are treated empirically at presentation pending culture results if taken. The empiric choice of antibiotic treatment must cover the most likely organism. Thus it is important to consider where and how the infection was acquired. An uncomplicated localised superficial skin infection in an immunocompetent host will often respond to topical antibiotics such as fusidin acid ("Fucidin cream®"). Deeper and more extensive infections, particularly in patients who are immunocompromised, will need systemic treatment either orally for moderate infections or intravenously for severe infections. Once the causative agent and its susceptibility has been identified, treatment should be switched to a narrow spectrum antibiotic. Swabs for culture and sensitivity are not always necessary for skin infections in general practice but should be considered in more severe infections, recurrent infections or where resistant organisms are suspected (e.g: nursing home patients and patients with chronic wounds in the community).

## 30.3 Impetigo

*Staphylococcus aureus* is most commonly known for causing the honey coloured, crusty lesions seen in impetigo ("aureus" is the Latin for golden) (Fig. 30.2). This is most common in pre-school children and newborns, and can be quite infectious, spreading to siblings and classmates. It most commonly involves the face and exposed sites. The children are usually otherwise healthy. It usually starts with some small vesicles that rupture and develop a golden crust. After a few days it can spread locally. Satellite lesions occur around the original site and sometimes on distant sites. It can sometimes be confused with a herpes simplex or ringworm, as lesions are sometimes vesicular, discoid or annular. Sometimes a blister



**Fig. 30.1** Bacterial skin infection chart. With permission of <http://www.slideshare.net/vmshashi/pathology-of-skin-common-disorders>. WBCS white blood cells

develops (bullous impetigo) most commonly in the skin folds and when this occurs it is more likely as a result of toxin producing *staphylococcus aureus* which is a localised form of *staphylococcus* scalded skin syndrome.

Milder cases may respond to topical fusidic acid ("Fucidin cream®"). Resistance to fusidic acid is becoming increasingly more common.

Fortunately resistance to fusidic acid is not stable and will usually fade if the drug is stopped. Therefore "Fucidin cream®" should be used for short courses of not more than 2 weeks, and courses should not be repeated for at least 6–12 weeks if possible. For more severe or widespread infection, oral flucloxacillin (or clarithromycin if allergic to penicillin) for at least 7–14 days should



**Fig. 30.2** Impetigo is usually caused by a bacteria called *Staphylococcus aureus*

be added to topical “Fucidin cream®”. Patients and carers should be instructed about careful hand washing and should be advised to use their own towels and face cloths to prevent cross infection.

#### 30.4 Folliculitis

This hair follicle infection is usually caused by staphylococci when the bacteria penetrate down through the hair shaft to cause a deep-seated infection in the hair follicles. This obviously occurs only in the hairy parts of the body, such as the beard area, scalp or on the trunk in men or in the legs or groin in women (Fig. 30.3). Close inspection with good light and a magnifying lens will show multiple small areas of erythema around the hair shafts, which sometimes progress onto small papules and pustules. Like impetigo, minor cases might respond to topical fusidic acid but more deep seated or wide-



**Fig. 30.3** Folliculitis of the posterior scalp

spread infection will require a long course of oral flucloxacillin for 2–6 weeks. For resistant cases, nasal swabs should be taken to rule out nasal carriage as a focus for re-infection. This can be treated with topical nasal antibiotics such as neomycin (“Naseptin®”) or mupirocin (“Nasal Bactroban®”). Caution should be taken as there are patients who are allergic to neomycin.

Folliculitis can sometimes be due to trauma, such as from shaving or waxing. In addition, applications of tar, oils, or greasy ointments can also lead to folliculitis. When prescribing greasy moisturisers, such as emulsifying ointment, doctors should always instruct the patient or carer to rub the ointments downwards, as rubbing ointments upwards can irritate hair follicles and lead to folliculitis.

Folliculitis can sometimes be caused by **gram-negative organisms** such as pseudomonas or *E. coli* especially in acne patients taking oral tetracyclines. They usually respond to stopping the tetracyclines and giving amoxicillin/clavulanic acid, trimethoprim or isotretinoin (“Roaccutane®”).

**Swimming pool or Jacuzzi folliculitis** is also due to gram negative organisms such as pseudomonas and may resolve spontaneously by staying out of the water or by treatment with ciprofloxacin. Yeasts such as *Pityrosporum ovale* can cause a low grade folliculitis on the trunk in young adults and it responds to topical or oral anti-yeast medications such as ketoconazole. Ingrown hairs in the neck in men can cause a folliculitis like reaction (**pseudofolliculitis barbae**) which can

be managed by physically removing the ingrowing hairs or by growing a beard (see Chap. 36).

### 30.5 Boils (Furuncles or Carbuncles)

These can be considered as a localised severe folliculitis, which again is most commonly due to *staphylococcus aureus*. The point of entry again is usually through the hair follicle, but the infection spreads locally to cause a painful, tender boil, which can sometimes progress on into a skin abscess. All patients with boils or abscesses should be checked for diabetes. Since this is a deep-seated infection, topical treatments are usually ineffective and treatment involves giving high dose oral or intravenous Flucloxacillin (oral clarithromycin if the patient is penicillin allergic) for at least 7–14 days. If an abscess is fluctuant and pointing, it should be incised and drained (I+D), either under local or general anaesthetic. Unless there are signs of surrounding cellulitis or systemic upset (fever and chills) antibiotics are usually unhelpful and unnecessary for an abscess and I+D is usually all that is required. In more severe cases swabs should be taken to identify the responsible organism and its sensitivity to antibiotics. For recurrent infections “Milton®” baths should be considered (see Chap. 66).

### 30.6 Secondary Infection of Eczema

The most common cause of an acute exacerbation of atopic eczema, particularly in children, is secondary infection, usually by *staphylococcus aureus*. Most cases need systemic antibiotics, usually with flucloxacillin (or clarithromycin if penicillin allergic) for at least 7–14 days at the maximum dose allowed for the particular age group.

### 30.7 Methicillin-Resistant *Staphylococcus Aureus* (MRSA)

MRSA was considered a hospital infection up until a few years ago. It is now commonly found in the community. Many patients in nursing homes and patients in the community with chronic wounds, such as leg ulcers can be infected by MRSA. MRSA is not more virulent than ordinary *staphylococcus aureus* but when it causes an infection it is harder to treat because of its resistance to multiple drugs. Good wound care should help heal MRSA infected wounds.

Many patients in nursing homes and those discharged from hospitals may be carriers of the MRSA strain without having any clinical infection. With such patients, simple hygienic precautions such as wearing gloves and prudent hand washing should be applied by the health care provider. Washing with an antibacterial wash such as “Hibiscrub®” for 2 weeks and applying mupirocin ointment to the nostrils twice a day for 5 days may help eradicate carriage.

### 30.8 Leg Ulcers

Patients with chronic leg ulcers should have their wounds swabbed to identify if they are carrying MRSA. In some cases the MRSA may be colonising the wound and may not be responsible for any obvious underlying clinical infection. Therefore, even in the presence of MRSA, good wound care with topical antiseptics, elevation and compression when indicated, should help to heal the wound (see Chap. 37). If there is obvious clinical infection, such as pain, erythema, heat and swelling, the patient may respond to high dose oral clindamycin. It is important to identify MRSA in the wound to protect health care workers, other residents in a nursing home, and family members from getting contaminated and spreading the MRSA to other people.

A green discharge may indicate pseudomonas infection. This can be difficult to clear but if localised may respond to acetic acid (vinegar or “EarCalm®”) or “Flamazine®” cream. More severe cases may need oral or parental ciprofloxacin.

## 30.9 Streptococci Infections

### 30.9.1 Erysipelas

This is a deeper infection than impetigo, usually affecting the superficial dermis. It usually develops suddenly with redness, heat, swelling and tenderness in the affected area. It spreads out rapidly from the original site, which is often a small area of broken skin as a result of a small patch of localised eczema (Fig. 30.4). The patient often feels feverish and has flu-like symptoms and perhaps a low-grade temperature. The rash usually has a well demarcated, erythematous, palpable border. The face and lower legs are the most common sites infected. Vesicles and bullae may appear after a few days. Lymphangitis and regional lymph node enlargement are sometimes associated. It is most commonly caused by beta haemolytic streptococcus and responds to high dose oral or parenteral penicillin (benzoylpenicillin IM or IV or oral penicillin V in high doses). Erysipelas-like cellulitis can sometimes be caused by staphylococcus aureus and so in severe infections it may be necessary to combine peni-

cillin V and flucloxacillin orally in high doses, or by intravenous injections. Rest and elevation are important, particularly if a lower limb is involved.

### 30.9.2 Cellulitis

This causes a similar but deeper and more diffuse infection of the lower dermis than erysipelas. Again the patient usually presents with a painful, red, hot and swollen rash, which can spread rapidly. The patient may have a low-grade fever and flu-like symptoms. It often involves the lower leg and a leg ulcer or a break in the skin from a fungal infection of the feet can be the portal of entry for the streptococcus (Fig. 30.5). Lower leg cellulitis may sometimes be confused with other causes of a painful red leg such as inflammatory varicose eczema, DVT, and allergic or irritant dermatitis. If there is bilateral erythema, no fever and a normal white blood cell count, the diagnosis is unlikely to be cellulitis. Treatment of cel-



**Fig. 30.4** Erysipelas caused by *Group A Streptococcus* bacteria which penetrated the skin as a result of eczema of the ear



**Fig. 30.5** Cellulitis from infection between toes on the plantar aspect of foot

lulitis is the same as erysipelas with high dose Penicillin V and adding flucloxacillin for more severe infections (clarithromycin can be used if penicillin allergic). Recurrent cellulitis may respond to phenoxymethy penicillin tablets 250 mg twice a day for 12 months.

### 30.10 Conclusion

Superficial bacterial infections such as impetigo and folliculitis are usually caused by staphylococcus aureus and respond to flucloxacillin.

Deeper infections such as cellulitis are caused by beta haemolytic streptococci and usually respond to Penicillin V (phenoxymehtyplenicillin). More severe infections may need a combination of Penicillin V and flucloxacillin at high doses orally or intravenously. Infections of surgical wounds and bites are often caused by gram negative organisms and require a beta-lactamase antibiotic such as amoxicillin/clavulanic acid. All patients with skin infections should be screened for diabetes and patients with unresponsive or chronic infections should be screened for MRSA.



# Fungal and Yeast Infection of Skin, Hair and Nails

31

David Buckley

## Key points

- Ringworm (tinea corporis) is not always ring shaped and not all annular rashes are due to ringworm.
- Fungal infections are usually scaly, unilateral and asymmetrical.
- Potent topical steroids dampen down the inflammatory response to the fungus but usually promote its spread, resulting in a more widespread, unilateral, diffuse, non specific rash that may not be easily identified as fungal in origin (*Tinea incognito*).
- If there is any suspicion that a skin, hair or nail problem is due to a fungal infection, skin scrapings, plucked hairs or nail clippings may be taken for fungal stain and culture.
- In a suspected case of fungal nail infection, confirm the diagnosis by sending nail clippings for fungal stain and culture before starting treatment.
- *Tinea manuum* usually causes a dry, slightly scaly rash on the skin creases of the palm of the hand. It is usually unilateral and can be associated with athlete's foot ("two feet, one hand syndrome").

## What to tell the patient

- Ringworm can spread from animal to humans but not usually from humans to humans.
- If you are prone to athlete's foot, do not walk around barefoot. Wear leather soles in the winter and open sandals in the summer.

## 31.1 Introduction

Some fungal infections of the skin are obvious, can be diagnosed clinically and treated empirically without having to take samples for laboratory analysis (Table 31.1). A good example is an annular rash with raised, red, scaly borders and fading centres on the arm of a farmer (Fig. 31.1). This is most likely to be ringworm (**tinea corporis**)

**Table 31.1** Common fungal and yeast skin and nail infections

Tinea Pedis (Athlete's foot)
Tinea Corporis (Ringworm on the body)
Tinea Cruris ("Jock itch" of the groin)
Tinea Manuum (Hand infection)
Tinea Unguium (Onychomycosis—fungal nail infection)
Tinea Capitis (Scalp ringworm)
Tinea Barbae (Beard ringworm)
Tinea Incognito (when masked with a potent topical steroid)
Tinea Versicolor ( pityriasis versicolor)

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**Fig. 31.1** Typical ringworm



ris) and can be treated with a topical antifungal such as terbinafine (“Lamisil<sup>®</sup>”, “Lanafine<sup>®</sup>”). Identifying and treating the source (cows, dogs, cats, etc.) is helpful to prevent re-infection and also to prevent other family members from getting infected.

## 31.2 Ringworm

Ringworm is not always ring shaped and not all annular rashes are due to ringworm. Further confusion can arise if the patient has self-treated a rash with a myriad of creams from well meaning pharmacists, friends and family (Fig. 31.2). These treatments can alter the classical appearance of the rash particularly, if applying a potent topical steroid. Potent topical steroids dampen down the inflammatory response to the fungus but usually promote its spread, resulting in a more widespread, unilateral, diffuse, non specific rash that cannot easily be identified as fungal in origin (**Tinea incognito**) (Fig. 31.3). This can often look like a patch of eczema or psoriasis prompting the doctor to use even more potent steroids. Any attempts at stopping the steroid will usually result in a rebound exacerbation of the rash which may become more scaly and pustular (Table 31.2).

If there is any suspicion that the rash is due to a fungal infection, skin scrapings should be taken



**Fig. 31.2** Tinea Corporis. This patient also had tinea curis not responding to 1% hydrocortisone mixed with an miconazole antifungal cream



**Fig. 31.3** Tinea incognito from using betamethasone valerate cream on a fungal infections

**Table 31.2** Clues that a rash may be fungal or yeast in origin

Asymmetrical rash
Unilateral rash
Annular rashes
Slightly raised scaly borders
Ill defined borders
Satellite lesions
Animal contacts
Patients of African origin
Unresponsive or worsening with potent topical steroids
Rebound of the rash when potent topical steroids are stopped

for fungal stain and culture (Table 31.2, Fig. 3.1 in Chap. 3). Results can take 2–4 weeks. It can be difficult to grow a fungus in the laboratory, especially if the patient is already on oral or topical antifungals or if they previously cleaned the skin with povidine iodine. False negatives may occur.

Not only will the culture confirm that you are dealing with a fungal infection but it will also give you clues as to the likely source of the infection (from animals, humans, the soil, etc) and guidance as to the most appropriate treatment. While waiting for the results, you may have to treat on a best guess basis. It is reassuring to know you have sent skin scrapings particularly, if you are tempted to try topical steroids while awaiting results.

The choice of treatment for a fungal skin infection is dependent on the probable organism (dermatophyte or yeast infection) and the extent and severity of the infection. For localised dermatophyte infections a topical allylamine antifungal such as terbinafine, for 1–2 weeks, is usually sufficient. For more extensive, severe infections add in a longer course of oral terbinafine.

Some rashes can be due to a yeast (*Candida*) infection rather than a dermatophyte. Terbinafine has little or no anti-yeast activity, whereas imidazole antifungus such as, ketoconazole (“Nizoral®”) or itraconazole (“Sporanox®”) have strong anti-yeast activity. Other imidazole antifungal such as miconazole (“Daktarin®”) or clotrimazole (“Canesten®”) have both antifungal and anti-yeast activity and also a weak antibacte-

rial effect. These can be a good choice if you are unsure if the infection is fungal, yeast, bacterial or a mixed infection such as athlete’s foot or a groin infection.

### 31.3 Tinea Pedis

Tinea Pedis nearly always starts as an itchy rash between toes with scaly, white, macerated skin, most commonly between the third, fourth or fifth toes. One foot is usually worse than the other. Most cases are caused by *Trichophyton* (*T. rubrum* or *T. Interdigitale*). Occasionally, a yeast infection such as candida may be responsible.

Tinea pedis sometimes causes an allergic (“id”) reaction that causes pompholyx (blistering eczema) on the soles of the feet and on the hands. This is more common in young women. If you see a person with pompholyx on the hands, always look at their feet, as you may find tinea pedis, which may be the cause. Treating the tinea pedis with an antifungal and the pompholyx with a potent topical steroid usually clears the rash. It is important to advise patients with tinea pedis to wear open sandals in the summer and leather soled shoes in the winter. They should also be cautioned not to walk around barefoot anywhere, particularly in pools, gyms and changing rooms. Footwear can harbour yeast or fungi, so it is important to treat all shoes with an antifungal powder such as miconazole (“Daktarin®”) daily for 1 week.

Tinea pedis may also cause small cracks or breaks in the skin allowing bacteria to penetrate, resulting in cellulitis on the foot or leg. While it is important to treat the cellulitis with an antibiotic, it is also important to treat the tinea pedis with an antifungal to prevent relapse. **Potassium permanganate** soaks with a 1% solution (1:100) can be helpful for weepy infected tinea paedis and can be antifungal and antibacterial.

It is not uncommon to find autoinoculation from feet to groin: the same patient that presents with tinea pedis may also have evidence of tinea cruris and vice versa.

Feet need to be kept dry and the interdigital space should be airdried (a hair dryer in cold air

modality can do the job). Talcum powder helps reduce moisture.

Patients with hyperhidrosis on the sole may develop pitted keratolysis, an infection caused by several bacterial species including corynebacterium among others. The sole presents with punched out pits and bad smell caused by the sulfur compounds produced by the infecting microorganisms. It responds to topical (e.g. fusidic acid cream) or oral antibiotics (e.g. erythromycin). Keeping the area dry is very important.

#### 31.4 Tinea Manuum

Tinea manuum usually causes a dry, slightly scaly rash on the skin creases of the palm of the hand. It is usually unilateral and can be associated with athlete's foot ("two feet, one hand syndrome") (Fig. 31.4). Skin scrapings from the scaly palm creases usually clinch the diagnosis.

#### 31.5 Tinea Cruris

Tinea cruris causes an itchy, red, scaly rash in the groin creases but it can be very difficult to differentiate from intertrigo, eczema, seborrhoeic dermatitis or psoriasis of the groin (Figs. 31.5 and 31.6). Skin scrapings can help to identify if there is fungal infection present. Topical imidazole antifungals are usually better than terbinafine as they have anti-yeast and antibacterial effects as

**Fig. 31.4** Tinea manuum on the right hand



well as antifungal effects. Sometimes combining an imidazole antifungal with 1% hydrocortisone can help to dampen the inflammatory aspects of the infection until the antifungal takes effect (e.g. "Daktacort®", "Canesten HC®"). Underlying causes should be identified and managed to prevent relapse (diabetes, obesity, poor hygiene, antiperspirants, soaps, bubble baths, etc).



**Fig. 31.5** Tinea curis. This patient also had Tinea Paedis



**Fig. 31.6** Tinea curis

## 31.6 Tinea Capitis

Tinea capitis is more common in children under the age of 12. It is usually caused by infections from cats, dogs or cattle. It causes round patches of hair loss, but unlike alopecia areata, it also causes skin inflammation with redness and scaliness of the skin, in the area of hair loss (Fig. 31.7). Skin scrapings or plucked hair from the affected areas may grow the fungus. Treatment is with oral terbinafine for 4–6 weeks (Table 31.3). Terbinafine should not be used in pregnancy. Griseofulvin is more effective for *Microsporum canis* (from cats, dogs or rabbits) (15–25 mg/kg/day with food for 6–12 weeks) but it can be difficult to source in some countries. Oral itraconazole is another alternative for *M. canis* infection. Occasionally, tinea capitis can cause a severe allergic reaction resulting in a boggy, purulent, oozing mass on the scalp with regional lymphadenopathy (a **kerion**) (Fig. 31.8). Treatment may require a combination of oral antifungals, oral antibiotics and oral steroids. If not treated early, a kerion may cause permanent scarring and a bald patch. Tinea capitis is more common in patients of African origin and *Trichophyton violaceum*



**Fig. 31.7** Tinea capitis in a 7 year old

**Table 31.3** Choice of drug treatment of tinea capitis according to organism isolated [1]

Trichophyton tonsurans	Terbinafine
Trichophyton violaceum, soudanense	Terbinafine
Microsporum canis	Griseofulvin or itraconazole
Microsporum audouinii	Griseofulvin or itraconazole



**Fig. 31.8** Kerion in the posterior scalp in a 16 year old farmer's son. The cattle had ringworm

*laceum* is the most common organism isolated in this group. It usually responds to oral terbinafine and ketocanazole shampoo may prevent person to person spread.

**Tinea barbae** is most commonly found in the beard area in farmers and causes an inflammatory, papular, pustular, crusty, unilateral rash that responds to 3–4 weeks of topical or oral terbinafine.

## 31.7 Tinea Unguium (Onychomycosis)

Tinea unguium can often be confused with other conditions that cause nail dystrophy such as psoriasis, paronychia or onychogryphosis (Fig. 31.9). Fungal toenail infections are usually harmless and asymptomatic. The only indication for treatment is if it is causing pain or for cosmetic concerns. If the patient is insistent on treatment, nail clippings from the infected nail should be taken by the doctor using a good nail clipper as proximally as possible to include the whole thickness of the discoloured, thickened, crumbly nail with some subungual debris for fungal stain and culture. This can be painful and the doctor needs to warn the patient that there may be some light bleeding after the nail clippings are taken. Even with a good nail sample, 30–50% of infected nails may fail to grow the fungus in the lab. Do not accept nail clippings that the patient may provide themselves as they



**Fig. 31.9** Tinea unguium. Clippings grew *Trichophyton rubrum* in the finger nails and toe nails

are usually taken far too distally to be useful. It is best to withhold treatment until the results are back, as the type of organism involved will dictate the most appropriate treatment. Mild, superficial fungal nail infections may respond to topical treatment such as amorolfine (“Loceryl®”) nail lacquer, twice weekly for six for finger nails and up to 12 months for toenails.

40% Urea ointment (“Canespro Fungal Nail Treatment®”) can be used to debride the damaged infected nail by daily applications for 2–3 weeks under occlusion. After this, treatment with a topical antifungal such as terbinafine cream or amorolfine nail paint will penetrate better and be much more effective at clearing the fungus.

Infection can be due to a dermatophyte (e.g. *Trichophyton rubrum*), yeast (*candida*) or moulds. If a dermatophyte infection is isolated (e.g. tinea rubrum) and two thirds of the nail is affected and the infection involves the nail bed, treatment is usually with oral terbinafine 250 mg daily for 3–4 months. Pulse treatment with itraconazole is an option: each pulse is 200 mg in the morning, 200 mg in the evening for a week followed by 3 weeks with no treatment. For feet tinea unguis, three pulses are required; for hand nail infection, two pulses. Yeast infections respond better to oral itraconazole. Bloods, including full blood count, renal and liver function tests, should be checked before starting oral therapy and repeated 1 month into a 3 or 4 month course of treatment to ensure no adverse reactions. Success rates range from 40% to 80% with oral therapy.

It can take 6–12 months after completing oral treatment before the nail grows out clear and it is important to explain this to the patient at the outset. The reported rate of clinical recurrence of onychomycosis ranges from 10% to 53%, regardless of the treatment method used [2].

Laser and IPL treatment as well as photodynamic therapy have been shown recently to have some antifungal and anti-yeast effect by heating the subungual skin and killing the organisms. However the evidence of the effectiveness of laser treatment of onychomycosis is limited [3] (see Chap. 41).

### 31.8 Tinea (Pityriasis) Versicolor

Tinea (Pityriasis) Versicolor causes a low grade, faint, slightly itchy, slightly scaly, blotchy rash mainly on the trunk in adults. The colour of the rash can vary from brown to white (versicolor) according to the seasons and depending on which area of the skin is involved (Fig. 31.10). Skin scrapings should identify the offending commercial yeast (*Malassezia*) but skin scrapings are rarely necessary as the diagnosis is usually obvious on clinical grounds. Treatment is with a topical anti-yeast agent such as ketoconazole. Ketoconazole shampoo can be used as a lotion and 5–10 ml can be applied from the neck down to the thighs and down to the wrists for 15 min daily for 7 days. The patient needs to be warned that the scale and itch will go immediately after treatment but the pigment changes can take 6–12 weeks to fade. Relapses are quite common and



**Fig. 31.10** Pityriasis versicolor

some patients have to treat themselves every spring. Malassezia yeast can also cause a truncal folliculitis (*pityrosporum folliculitis*), which may require systemic anti-yeast treatment such as ketoconazole or itraconazole. It is important to maintain treatment on the scalp (on a week basis, leaving the product on wet scalp for 10 min before rinsing) in patients with frequent recurrences.

### 31.9 Conclusion

Fungal infections can affect any part of the skin, hair or nails. They can mimic many other conditions. If there is any suspicion if a fungal infection tissue (skin scrapings, plucked hairs, nail clippings, etc) should be sent for fungal staining and culture. It can take a month to culture a fungus in the laboratory. If there is a high index of suspicion of a fungal infection and treatment is required immediately (e.g. scalp ringworm, painful tinea cruris, etc) empirical treatment should be started and the patient reviewed in a few weeks when the results of fungal culture are available. If

there is a low index of suspicion that an eczematous rash is fungal in origin (but it cannot be ruled out), a sample should be sent to the laboratory and the patient treated with a topical steroid if necessary. If subsequent tests show a fungus, the steroid can be stopped and antifungal treatment initiated. Sometimes combining a weak topical steroid with a topical antifungal (e.g. "Daktacort®" or "Canasten HC®") can be helpful in dampening down an inflammatory dermatosis in the face or flexures while waiting for fungal cultures to return.

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# Cutaneous Viral Skin Infections

32

David Buckley

## Key Points

- For patients who get frequent, troublesome attacks of cold sores, valaciclovir hydrochloride, 500 mg tablets (“Valtrex®”) four tabs twice a day for 1 day is very effective once started at the earliest sign of an attack.
- A recurrent vesicular eruption on the same part of the body is most likely due to herpes simplex.
- Genital herpes is sexually transmitted and like all STDs the patient should have a complete STD screen and contact tracing.
- If a pregnant woman or person who is immunocompromised is exposed to a case of chickenpox and their immune status is unknown, they should be considered for varicella-zoster immunoglobulin and systemic antivirals.
- Treating shingles with oral antivirals at an early stage may lessen the incidence and severity of post herpetic neuralgia, particularly in those over the age of 50, once treatment can be started within 72 h of the first appearance of the rash.

## What to Tell the Patient

- Do not kiss anyone if you have a cold sore.
- Do not have sex if you have a break out of genital herpes.
- Shingles is not contagious.
- If you are over the age of 50 and you suspect you may have shingles, seek medical advice immediately as treatment works best if started as early as possible.
- Molluscum contagiosum are harmless and most resolve spontaneously within 6–12 months.

## 32.1 Introduction

The most common skin conditions caused by viral infections are herpes simplex, varicella zoster, viral warts, molluscum contagiosum, herpangina and orf.

## 32.2 Herpex Simplex

The herpes simplex virus (HSV) type 1 causes cold sores which are characterised by recurrent eruptions of a vesicular rash that crusts over and heals completely without scarring over the course of 1–2 weeks. Although the lips are the most common area affected ('herpes simplex labialis'), cold sores can occur on any part of the body (as herpes

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simplex chronicus). Another eruption that repeatedly appears on the same area of the body is a fixed drug eruption. This usually presents as a well defined, round or oval patch of redness and swelling of the skin which sometimes blisters. It heals within a few weeks once the drug is stopped but recurs every time the drug is reintroduced.

The first attack of HSV Type 1 is usually in childhood and may be asymptomatic. Sometimes it may cause a painful stomatitis in the mouth and lips that can cause difficulty eating and swallowing (herpetic gingivostomatitis). There may be fever, malaise and swollen glands. This usually resolves spontaneously within 1–2 weeks without treatment. It can also make its first presentation as a herpetic whitlow.

Once infected, the virus will remain dormant and resistant to treatment in the dorsal root ganglion for the rest of the patient's life. It may erupt at any stage to cause the classical cold sore. There are many possible triggers for cold sores (Table 32.1), but they can often erupt for no par-

ticular reason. Treatment is often ineffective once the lesions have fully developed.

Avoidance of triggers is the best way to prevent cold sores but this is not always possible. Treatment with topical antivirals such as acyclovir ("Zovirax®") is of limited benefit and will only work if the treatment is started at the earliest possible stage; preferably at the tingling stage (if it occurs) before the vesicles appear. It has to be applied five times a day for 5 days. For those with frequent attacks having a tube readily available is useful so that treatment can be started at the earliest possible time.

Once the vesicles have appeared it is questionable if topical acyclovir will help. There are very few good clinical trials on the value of topical acyclovir. It may well be that a simple anti-inflammatory with some topical antibiotics such as fusidic acid cream with 1% hydrocortisone ("Fucidin H®") may be as effective or even more effective at this stage. Even keeping the area dried with topical disinfecting alcohol can work better than any cream treatment.

For patients who get frequent, troublesome attacks of cold sores, having a standby course of antiviral tablets to start at the earliest possible time in an attack can be very effective and safe (e.g. valaciclovir hydrochloride, 500 mg tablets ("Valtrex®") four tabs twice a day for 1 day only) (Table 32.2).

For patients with frequent recurrent severe attacks of cold sores, prophylactic treatment with

**Table 32.1** Triggers for a herpes simplex viral infection

Minor trauma at the site of infection.
Febrile illness.
Excessive ultraviolet light.
Hormonal triggers (e.g. with periods every month).
Emotional stress (e.g. with exams).
Post surgery on the face (e.g. laser, deep chemical peel, dermabrasion).
Post dental surgery.

**Table 32.2** Valaciclovir hydrochloride, 500 mg tablets ("Valtrex®") dosage recommendations for adults

Indications	Dosage for adults	Duration
<b>Cold sores (herpes labialis)</b>	Two × 2 g doses taken 12 h apart (i.e. 4 tabs BD)	1 day
<b>Genital herpes:</b>		
Initial episode	500 mg every 12 h	10 days
<b>Genital herpes:</b>		
Recurrent episode	500 mg every 12 h	3–5 days
<b>Genital herpes:</b>		
<i>Suppressive therapy:</i>		
(a) Immunocompetent patients	500 mg every 24 h	6–12 months
(b) Alternate dose for immunocompetent patients with more frequent infections	1 g every 24 h Or 500 mg every 12 h	6–12 months
(c) HIV-1-infected patients	500 mg every 12 h	6–12 months
<b>Herpes zoster</b>	1 g every 8 h	×7 days

systemic antivirals may be necessary. These can also be used for patients who are at high risk of developing cold sores after procedures such as chemotherapy, radiotherapy, laser treatment, chemical peels or dermabrasion (e.g. valaciclovir hydrochloride, 500 mg tablets ("Valtrex®") two tabs once a day for as long as there is a risk). For more than five attacks per year, a continuous low dose treatment can work: 500 mg per day of valaciclovir for 6–12 months.

The HSV type 1 can cause severe, extensive, painful, monomorphic vesicles and erosions with lymphadenopathy and a fever especially in patients who have a background of atopic eczema (eczema herpeticum or Kaposi varicelliform eruption). For extensive infection systemic antivirals should help but again should be started at the earliest possible stage in an attack (Table 32.2). Severe infection with HSV may need hospital admission for IV antivirals.

When HSV Type 1 affects the finger, it can cause a herpetic whitlow which can sometimes be an occupational hazard for dentists and dental assistants. This is why dentists should wear gloves for all oral examinations. It is also common in children where it is caused oral gingivitis. It presents as vesicles, pain and burning sensation.

### 32.3 Genital Herpes

This is a much less common but more serious HSV infection which is usually caused by the Type 2 HSV but can sometimes be caused by Type 1 HSV. Like cold sores, the diagnosis can often be made clinically with the characteristic vesicular eruption recurring time and again in the same area (penis, vagina, anus, thighs or buttocks) and healing without scarring. The eruption is painful and can take a few weeks to heal. There is usually pain on passing urine (dysuria) but unlike a UTI, there is no increase in frequency. Occasionally the pain can be so severe the patient may present with urinary retention. Passing urine

while having a bath may help. It is usually sexually transmitted and like all STDs, the patient should have a complete STD screen and contact tracing which is probably best carried out in an STD clinic, especially if it is a first attack. The diagnosis can be confirmed by taking special PCR viral swabs from fresh lesions. Treatment of genital herpes usually requires a systemic antiviral treatment to be started as early as possible in an attack. For those with frequent attacks, suppressive maintenance treatment with an oral anti-viral medication for 6–12 months may be helpful (Table 32.2).

Patients are infectious during an attack and so should avoid sex at this time. Condoms will help prevent infection. Child birth during an attack of genital herpes could possibly infect the newborn child. Once the lesions are fully healed the patient is not usually infectious until the next attack of herpes. More useful information is available from the British Association for Sexual Health and HIV ([www.bashh.org](http://www.bashh.org)) and the Herpes Virus Association ([www.herpes.org.uk](http://www.herpes.org.uk)).

### 32.4 Erythema Multiforme

This is a hypersensitivity reaction typified by a generalised rash that has the characteristic target lesions (Fig. 32.4). The most common trigger is the HSV (cold sores). This can sometimes be associated with blisters, erosions and ulcers in the lips, mouth and genitalia which can be painful and debilitating. The rash should clear spontaneously after a few weeks but can recur with every attack of cold sores in some patients. Treatment of erythema multiforme is usually with topical or oral steroids combined with systemic antivirals for a severe attack. Prevention may require treatment with long-term oral antivirals so as to prevent cold sores.

HSV may also trigger an attack of erythema nodosum with the characteristic red, tender, maculo-papular, non scaly rash on the shins and sometimes on the forearms.

### 32.5 Varicella Zoster

Chickenpox (varicella) is considered a harmless childhood viral infection that occurs in most children. It causes a generalised vesicular eruption, mainly affecting the face and trunk. It can be associated with a low grade fever. Most children recover spontaneously without complications within 1–2 weeks. It has an incubation period of 10–21 days. The patient is infectious from 2 days before the appearance of the rash until all the vesicles have crusted over which usually takes 5–10 days. Characteristically, the rash develops in crops so that the spots are in various forms of development at any affected area (vesicles, crusts) and lesions are very itchy. If a person has a severe attack or is immunocompromised (diabetes, chemotherapy, leukaemia, HIV, etc.) they should be treated with systemic antivirals. The varicella virus can be harmful to an unborn child so children with chickenpox should, if possible, avoid contact with pregnant women.

If a pregnant woman or person who is immunocompromised is exposed to a case of chickenpox and their immune status is unknown, they should be considered for varicella-zoster immunoglobulin and systemic antivirals.

Chickenpox can leave small punched out scars which can be unsightly and permanent, particu-

larly if they occur on the face. Using a topical anti-inflammatory with an antibiotic such as fusidic acid cream combined with 1% hydrocortisone cream (“Fucidin H cream®”) on the facial lesions twice a day to 7–14 days may help prevent scarring (see Chap. 50). Oatmeal bath treatments can help reduce the itching.

Among possible complications are: pneumonia, encephalitis and Reye syndrome. For the firsts two, inform the patient to let you know for any breathing difficulties or headache. To avoid Reye syndrome, never give aspirin to a child with varicella.

### 32.6 Shingles (Herpes Zoster)

Once the acute chickenpox infection clears the virus will remain dormant in the dorsal root ganglia of the spinal cord. It can remain dormant there for the rest of the patient’s life. It may be re-activated spontaneously or by various triggers and when it erupts it causes shingles. This can occur at any age but is more common and more problematic in the elderly. Shingles usually presents with the characteristic unilateral, vesicular or bullous eruption running in a dermatomal distribution (e.g. one side of the face, one side of the chest or down one arm, or one leg) (Fig. 32.1a, b). It



**Fig. 32.1** (a) Shingles after 4 days in a 22-year-old patient on methotrexate. (b) Shingles in a 67-year-old after 4 days

can be painful and sore and usually settles spontaneously within 2–3 weeks. Occasionally it can be very inflammatory, leaving permanent scars and chronic pain and tenderness in the area which can last more than 1 month. This is known as postherpetic neuralgia. It is more common in patients over the age of 50. There is some evidence that treating shingles with oral antivirals at an early stage may lessen the incidence and severity of postherpetic neuralgia. Systemic treatment should be considered for shingles, particularly in those over the age of 50 once it can be started within 72 h from the first appearance of the rash (Table 32.3). There is some limited evidence that systemic treatments with antivirals may be effective even up to 7 days after the onset of the rash, particularly in the high risk groups [1].

Shingles can be triggered by sun burn. Vesicles will appear exclusively in sun-damaged areas for a few days before the rash appears. Shingles can be preceded by pain or discomfort in the affected area for a few days before the rash is visible. The prodromal pain can be confused with other conditions, depending on where the eruption occurs: e.g. face = migraine, chest = myocardial infarction, abdomen = cholecystitis or appendicitis, etc. When shingles affects the ophthalmic branch of the facial nerve, the eye could be in danger of corneal scarring and so an ophthalmic opinion should be sought immediately. Healing of the cornea tends to take longer than the skin.

The varicella zoster virus (VZV) may be shed from shingles lesions and can cause chickenpox in a non immune child or adult. Despite popular myth, it is not possible to get shingles from

**Table 32.3** Indications for systemic antivirals for shingles (start within 72 h of the appearance of the rash)

Patients more than 50 years of age
History of the rash for less than 72 h (possibly 7 days in severe cases)
Ophthalmic herpes zoster
Non-truncal disease (e.g. the face, scalp, ear, arms, legs or genitalia)
Immunosuppressed patients
Moderate to severe rash
Moderate to severe pain

another patient with shingles. However, clusters of cases of shingles have been reported. It is suggested that contact with someone with chickenpox or shingles may cause one's own dormant virus to reactivate. Immunosuppressed patients can have a rare presentation of shingles as a generalized form similar to chickenpox.

In patients with shingles, a sudden onset of headache or respiratory difficulty should alert the doctor to the possibility of meningeal or pulmonary infection, which will require hospitalization and IV medication.

### 32.7 Post-Herpetic Neuralgia

Postherpetic neuralgia causes neuropathic pain which can be described as burning, shooting, itching or as a stabbing hypersensitivity in the area which can last for more than 1 month after an attack of shingles. This can be a very painful and debilitating condition particularly when it occurs on the face (Fig. 32.2). It does not usually respond to standard analgesics such as paracetamol or nonsteroidal anti-inflammatories. Tramadol may be more effective in some patients. Tricyclic antidepressants such as amitriptyline taken at night can be very helpful particularly if there is night pain or insomnia. The dose should be started at 10 mg 1 or 2 h before going to bed and be gradually increased until the patient has a good night's sleep without drowsiness the following morning.



**Fig. 32.2** Post herpetic neuralgia × 3 year on the face of a 77 year old lady

More specific treatments with anti epileptic drugs such as gabapentin (e.g. "Neurontin®") or pregabalin (e.g.: "Lyrica®") may be necessary [2]. These should be started at a low dose and gradually titrated upwards until a therapeutic response is obtained or side effects insured. Topical treatments such as lignocaine patches ("Versatis®") are of limited value and not practical on the face. Topical capsaicin ("Axsain®"), which is derived from chilli peppers, can act as a counter irritant which may be of help in some patients.

Transcutaneous electrical nerve stimulation (TENS) or nerve blocks may be required in severe cases. Combining treatments such as "Lyrica®", "Amitriptyline" and "Axsain®" may be tried, but at this stage the patient should probably be referred to a pain management clinic.

It is estimated the 1 one 3 people in the USA will suffer from shingles in their lifetime. In some countries there are vaccines against chickenpox for children and against shingles for people over the age of 50 ("Zostavax®"). A new inactivated shingles vaccine is available in some countries ("Shingrix®") which appears to be more effective at preventing shingles and last longer [3].

## 32.8 HIV

The HIV/AIDS can present in many different ways including in the skin. Any person with severe eczema, severe psoriasis, severe seborhoeic dermatitis, unusual rashes, recurrent or giant molluscum contagiosum, multidermatomal shingles, oropharyngeal candida, lymphadenopathy, neutropenia or lymphopenia should be tested for HIV regardless of their sexual history.

## 32.9 Herpangina

This is usually caused by an enteroviral infection such as coxsackie virus.

It is most commonly found in children and presents with multiple painful mouth ulcers which can make eating and drinking difficult.

There may be a low grade fever. Red spots appear in the mouth and throat at the start of the illness. The red spots develop into small blisters which eventually form a tiny yellowish ulcer with a red rim. It is usually a mild, although painful, self limiting infection and most cases settle spontaneously within 7–10 days. Treatment is symptomatic with analgesics, fluids and a soft diet. Some cases can be due to herpes simplex virus but the treatment is usually the same.

## 32.10 Orf

This is an unusual viral infection caused by the parapox virus usually from young sheep or goats. It is most commonly found in sheep farmers, their families, vets and butchers. It presents on an exposed area of the body such as the hand, arms or face as a painful, small, red nodule that may blister. The nodule can last weeks and grow to 3–5 cm in diameter. Most cases cause a solitary nodule but groups of a few nodules can occur (Fig. 32.3). There may be streaks up along the lymphatic channels and localised lymphadenopathy. The orf nodule can last up to 6 weeks and can be complicated by secondary infection and cellulitis. Orf can rarely be associated with erythema multiforme (Fig. 32.4), toxic erythema and bullous pemphigoid.

Treatment of orf is symptomatic and most cases resolve spontaneously. The wound should be dressed and any secondary bacterial infection



**Fig. 32.3** Orf in a sheep farmer



**Fig. 32.4** Orf causing erythema multiforme



**Fig. 32.6** Molluscum on a patient on methotrexate and hydroxychloroquine for rheumatoid arthritis



**Fig. 32.5** Molluscum contagiosum in a 9 year old



**Fig. 32.7** Molluscum contagiosum going into spontaneous resolution

treated if it occurs. Incision and drainage is unhelpful but shave excision, cryosurgery or imiquimod may help in some cases.

## 32.11 Warts

See Chap. 34.

## 32.12 Molluscum Contagiosum

This is a harmless pox virus infection most commonly seen in children with dry skin or eczema. The pox virus may cause an immune response resulting in secondary eczema in the infected area (this is known as an id reaction or autosensitisation dermatitis). They usually appear as clusters of small, white, dome shaped nodules with a central umbilical top (Fig. 32.5). They vary in size from 1 mm up to 6 mm in diameter (Fig.

32.6). If squeezed, a white, cheesy material appears through the open punctum on the top of the dome. They usually resolve spontaneously within 6–18 months. They often become red and inflamed which is a sign that that individual lesion is regressing (Fig. 32.7).

Treatment is usually not necessary unless they are very unsightly or uncomfortable. As molluscum are associated with dry skin, moisturising the skin in the affected area with a thick, grease moisturiser may help speed up the resolution. “Crystacide®” contains hydrogen peroxide, which is known as a disinfectant and has some antibacterial, anti fungal and anti viral activity. It has been used by some dermatologists for molluscum. The cream can be applied twice a day for up to 21 days. “Crystacide cream®” may form a dry film on the skin. It also contains propylene glycol and salicylic acid that

can cause skin irritation and it should be stopped if this occurs. Patients should be warned it can bleach fabrics.

The simplest and most effective treatment, if required, is very light cryosurgery for 2–3 seconds as, unlike viral warts, molluscum are extremely sensitive to low temperature. Molluscum on the genitalia or pubic area may be sexually transmitted and a full STI screen should be carried out looking for other asymptomatic infections such as gonorrhoea, chlamydia, syphilis, HIV or hepatitis B and C. Giant or extensive molluscum contagiosum may be associated with an underlying immunodeficiency such as HIV.

### 32.13 Conclusion

Viruses can cause harmless, nuisance skin infections such as warts or cold sores (herpes simplex). However, they can also cause painful and

potentially life threatening infections such as genital herpes or HIV. Viruses can be difficult to isolate, transport and culture in the laboratory. Most viral infections can be diagnosed clinically but more difficult to treat or extensive viral rashes may need referral for specialist investigation and treatment (e.g. ophthalmic herpes or zoster, genital herpes or HIV).

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# COVID-19 and the Skin

33

David Buckley

## Key Points

- COVID-9 is a new coronavirus that can affect the skin in various ways either directly or indirectly.
- Any new, unusual rash appearing in a patient during the pandemic should be viewed with suspicion, especially if the patient has a fever, respiratory symptoms or has been in contact with a known or suspected case of COVID-19 in the previous 2 weeks.
- New ways of practising dermatology is required during this pandemic and some of these, especially teledermatology, will hopefully remain and flourish in the future.
- Sending photos by emails to your doctor and/or live video consultations are safe, convenient, and quick ways of getting an opinion from your doctor about many common skin conditions including new rashes.
- Skin lesions similar to chilblains, urticaria, chickenpox, rashes and even blushing tones in the skin (levido reticularis) have been associated to COVID-19 infection. If you develop any such symptoms, contact your doctor.

## What to Tell the Patient

- If you develop a new rash during the pandemic, especially if you are sick with a cough, shortness of breath or have a fever, please phone your doctor's office for telephone advice before seeing them face to face.
- If you have a cough, shortness of breath, a fever or if you have been in contact with a known or suspected case of COVID-19 in the last 2 weeks please do not call to the doctor's office without talking to the receptionist by phone in advance.

## 33.1 Introduction

This chapter will discuss how COVID-19 can affect the skin, what patients on immunosuppressants for skin diseases should do and how patients with skin disease can be safely assessed using teledermatology.

## 33.2 Skin Manifestations of COVID-19

An interesting paper published by a Spanish group in the British Journal of Dermatology in 2020 [1] presented a large number patients with COVID-19 related skin conditions. All patients with a skin eruption of recent onset (previous 2 weeks) with no clear explanation and who were suspected or confirmed as having COVID-19,

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were included. 375 cases from dermatologists all over Spain were collected during the peak of the COVID 19 pandemic. 5 clinical patterns were described:

1. Acral areas of erythema with vesicles or pustules (pseudo-chilblain) (19%) (Fig. 33.1)
2. Other vesicular eruptions (chickenpox like) (9%)
3. Urticarial lesions (19%) (Fig. 33.2)
4. Maculopapular eruptions (47%)
5. Livedo or necrosis (6%) (Figs. 33.3 and 33.4)

The severity of associated disease followed a gradient, with pseudo-chilblain the less severe form and livedo or necrosis, the most severe and associated to increasing percentages of pneumonia and intensive care unit requirements.



**Fig. 33.2** New urticarial rash on the legs of a girl with possible COVID 19



**Fig. 33.1** Covid toes (chilblains) Fig. Copyright Dr Katrina Fernandez-Dunsterville



**Fig. 33.3** New livedo type rash on the legs of girl with possible COVID-19

It was noted that it is unusual that one virus could cause several different dermatological patterns. Some of these conditions (especially urticaria and maculopapular rashes) could be also related to drug rashes or maybe due to other



**Fig. 33.4** New petechial rash in an adult with COVID 19

viruses (especially parvovirus). The incidence of herpes zoster appeared to have increased in the study period but no causal relationship was found.

In one small hospital based study from Italy, up to 20% of COVID-19 cases had a rash associated with the disease (not with the treatment) [2]. The true incidence of rashes associated with COVID-19 in the community is unknown. However, it is well known that COVID-19 can affect the lungs and cause a pneumonitis. Likewise, it can affect skin and cause a vasculitis. Beware of any new unusual rashes especially in a patient with respiratory symptoms, fever or in a patient who has been in contact with a known or suspected case of COVID-19. If in doubt use personal protective equipment (PPE).

Many of the drugs used to treat more severe cases of COVID-19 can cause drug eruptions, a possible explanation for hospital cases or discharged patients that develop a new rash.

### 33.3 Hand Eczema/Dermatitis

COVID-19 is a lipophilic virus which means it likes fat and can penetrate the lipid barrier of the skin and lurk underneath. Therefore everyone should wash with soap (a detergent that breaks down lipids) and water to help clean the hands of the virus.

People with hand eczema/dermatitis have reduced lipids in the skin on their hands and

normally have to avoid soap. However, because of COVID-19, it is now recommended that even those with hand dermatitis use a non-perfumed soap to wash their hands and pat dry with a disposable paper towel (not hand dryers) which can also be used to turn off the tap, flush the toilet and on the door handle of the bathroom. Hand sanitiser gels may be used if soap and water is not available. After washing, the patient with hand eczema/dermatitis should moisturise liberally with a perfume free, preservative free hand moisturiser. They can use a light hand moisturiser every 1–2 hours during the day (e.g. “Aveno Dermexa®”, “Neutrogena Hand Cream®”, “Doublebase®” or “Epiderm Cream®”) and a greasy moisturiser (e.g. “Emulsifying ointment BP®” or “Epiderm ointment®”) at night under cotton gloves so as not to stain the bedclothes.

The WHO recommends that health care workers (HCW) clean their hands by rubbing them with an alcohol-based gel, as the preferred means for routine hand antisepsis if hands are not visibly soiled. HCW should wash their hands with soap and water when hands are visibly dirty or visibly soiled with blood or other body fluids and after using the toilet [3].

People with hand eczema still need to use waterproof gloves for all wet work and should use a soap-free shower gel, soap-free shampoo, and soap-free conditioner. Pain, oozing, crusting or a sudden deterioration of hand eczema/dermatitis may indicate that the skin has become infected. If this occurs the patient should consult with their doctor as they may need antibiotic tablets, steroid ointments, lab tests or allergy investigations. (See more useful tips in our patient information leaflet on “Hand care tips for people with hand eczema/dermatitis during COVID-19” in Chap. 66).

### 33.4 Immunosuppressants and COVID-19

Some patients with severe psoriasis, psoriatic arthritis, atopic eczema, hidradenitis suppurativa, systemic lupus and blistering diseases may be on immunosuppressants such as oral steroids, methotrexate, azathioprine or biologic agents to control their disease. According to the Irish Health

Service Executive (HSE), being on one immunosuppressive treatment does not appear to increase the risk of getting a COVID-19 infection (Coronavirus). There is no evidence to date that being on one immunosuppressive treatment puts a patient at higher risk of severe disease with COVID-19. However, as other infections can cause severe illness in people who are on immunosuppressive treatment, these patients should take extra care [4, 5].

Patients should not stop their immunosuppressants during the pandemic without consulting with their GP or their hospital specialist. They should socially isolate and work from home if possible. If they are on immunosuppressants and have significant co-morbidities (e.g. people over 70 year old or those with a chronic disease) they should try to avoid contact with other people as much as possible during the pandemic.

Patients on high dose oral steroids (e.g. 20 mg/day or greater for 2 weeks in adults and children >10 kg) should not stop them suddenly even if they get sick with a fever. They should consult with their doctor if they are sick. Topical and inhaled steroids do not increase the risk of getting infections or having adverse outcomes if they get an infection such as COVID-19. Topical calcineurin inhibitors (e.g. tacrolimus “Protopic”) for atopic dermatitis in otherwise healthy adults does not result in significant systemic absorption or immunosuppression. Hydroxychloroquine, which is sometimes used in dermatology, is not considered an immunosuppressant.

Some patients with less severe skin diseases may choose to take a holiday from their immunosuppressants if they feel the risks of staying on the immunosuppressants outweighs the risks of recurrence of their disease during the pandemic. However, this decision should only be taken after discussing the issue with their doctor as the COVID-19 pandemic may go on for many months. Patients on two immunosuppressants such as methotrexate and oral steroids ( $\geq 5$  mg/day) or a biological agent with oral steroids should try to avoid contact with other people as much as possible during the pandemic. Patients should phone their GP or consultant if they have coronavirus or symptoms of coronavirus and are

on immunosuppressants. They should do this before taking the next dose of treatments. Patients who usually have regular blood tests while on immunosuppressants should continue to have these during the pandemic.

**Oral isotretinoin (“Roaccutane”)** is not an immunosuppressant but there may be a theoretical risk of increased COVID-19 viral load due to its drying effects on the mucous membranes [6]. Patients should discuss the risks vs. benefits of oral isotretinoin with their doctor. Some may choose to take a holiday from their treatment. Adequate contraception and monthly pregnancy tests have to be continued if women stay on oral isotretinoin but this could be done by video consultation. Blood testing should be continued for all patients and should be carried out before starting, 1 month after starting and then every 3 months until they have finished the course.

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### 33.5 Teledermatology

The WHO defines telemedicine as “healing from a distance”. This is obviously a very safe way for health care workers (HCW) to assess patients who may have COVID-19 and is also a safe way for the patients to consult with their doctor or nurse. The simplest form of telemedicine is a phone call but this form of communication is difficult in assessing dermatology problems as it is such a visual speciality. It still has the advantage of maintaining communication with the patient which can give comfort and reassurance, especially when treating older patients. During the COVID-19 confinement, thousands of phone calls were made allowing physicians to give basic support and advice. A live video consultation allows the doctor to see the patient and pick up on non-verbal cues as well as seeing the skin. To diagnose new conditions, a good picture is always the best option. If the patient sends a good quality photo of their rash or skin lesion by email/sms in advance of a phone call it will help greatly in trying to make a diagnosis or to plan further investigations and treatments. Unlike a face to face consultation in pandemic times, teledermatology avoids the risk of infection to both

the patient and the doctor: neither of them will need to wear a mask which can hinder communication. Patients can show the doctor their medications and the doctor can demonstrate how to apply the treatments. Videocall plus image are the best combination. Many people use Facetime, WhatsApp video and Skype on their smart phones or PCs for personal and family use. These platforms are not safe or secured enough for clinical use but they have been used in emergency out of hours situations during the COVID-19 pandemic. There are various encrypted, GDPR compliant platforms available to carry out live video consultations such as doxy.me and Zoom Pro. Some Irish practice management software packages such as MyClinic365, Nuahealth and Wellola also incorporate video in their software.

Medical indemnity companies such as the Medical Protection Society (MPS) recommend that the safest way to carry out live video consultations is with patients who are already registered with the practice and with patients resident in the same country as the treating doctor. New and referred patients who choose to have a video consultation should fill out a new patient information form, read a patient information leaflet on video consultation and read and preferably sign a consent form in advance of the video consultation.

At the start of the video consultation check that the video and audio are working (“I can see and hear you, can you see and hear me?”). At the end of the consultation ask the patient to repeat back the management plan to ensure they understand all the advice they have been given.

Teledermatology and live video consultations can be used for many common skin, hair and nail problems such as acne, eczema, psoriasis, assessing rashes and for pre and post op assessments. However, video consultations are not suitable for lesions suspicious of melanoma where dermoscopy is required and is not appropriate if a procedure is necessary (e.g. removal of warts, dressing an ulcer, taking samples for lab analysis).

Payment can be taken in advance by credit card over the phone for private patients. Prescriptions can be sent to the patient’s phar-

macy by secure email and the patient can be sent an email after their visit with relevant patient information leaflets, links to relevant websites, follow up arrangements and an evaluation feedback form on their video consultation.

Video consultations usually take a little longer than face to face consultations for the doctor but it saves the patient time and money as they may not have to take time off work or arrange child minding and they have no travel expenses. It is ideal for younger patients who are tech savvy. It might be more complicated for some older patients or those without a smart phone or good WiFi connectivity.

### 33.6 Dermoscopy

Dermoscopy may pose a risk of transmission of COVID-19 especially when carried out on the hands and face [7]. If it has to be carried out, the doctor and patient should sanitize their hands and wear masks and gloves. Non-contact dermoscopy should be carried out if possible. Otherwise consider wrapping the dermatoscope in cling film and disinfect the dermatoscope before and after the procedure with alcohol wipes. Figuring and sharing the dermoscopy image for teaching and referrals will minimise touching the patient.

### 33.7 Conclusions

COVID-19 has presented unique challenges both in diagnostics and how health care workers approach and handle patients. It has given us an opportunity to explore new ways of practising medicine to ensure the safety of our patients, our staff and ourselves especially with the use of teledermatology. In the US, doctors have estimated that they have progressed teledermatology more in the first 2 months of the COVID-19 pandemic than they had in the previous 10 years.

Warning: Printed advice about COVID-19 may be out of date as soon as it is published. Please check your government’s health service websites for regular updates.

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# Management of Warts in General Practice

34

David Buckley

## Key Points

- Cutaneous viral warts are caused by the Human Papilloma Virus (HPV) which is a double-stranded DNA virus with many strains that can affect various parts of the body.
- Warts are less common in the elderly and can occasionally be confused with a hyperkeratotic lesion such as an actinic keratosis, BCC or SCC.
- All patients with genital warts should have a full STI screen and contact tracing.

## What to Tell Patients

- Almost everyone will get warts at some stage in their life.
- Most warts clear spontaneously within 2 years especially in children.
- Not all warts need to be treated, especially in children.
- Apart from genital warts, HPV is not highly contagious and the HPV will not penetrate intact skin. They usually need a portal of entry such as a crack or a cut in the skin and a susceptibility to be infected by the virus.
- Most topical wart treatments work better if applied after paring down the hard keratin

with an emery board or blade every night before applying the topical agent and continue the treatment for at least 6–12 weeks.

- When used properly, salicylic acid has a cure rate of 75%.

## 34.1 Introduction

Warts (*Verruca Vulgaris*) are a benign, viral, epidermal skin tumour that affects almost everyone at some stage in their life. Some studies suggest that up to 30% of children and young adults may have warts at any given time [1].

Apart from ano-genital warts, which are a risk factor for cervical, anal, penile and oropharyngeal cancer, most warts are harmless and most clear spontaneously within 1–2 years without any treatment especially in children and immunocompetent adults. Spontaneous remission has been shown to occur in half of children within a year and in two thirds within 2 years [2]. Sometimes warts can continue to grow and spread to other parts of the body. The most common indication to treat warts is for cosmetic or comfort reasons. Warts in children are probably best left untreated unless there are major cosmetic considerations (e.g. warts on the face) or a cause of pain (e.g. plantar warts). Doctors often underestimate the amount of embarrassment patients suffer with their warts especially if they are on exposed areas (hands or face) and the

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patient is working with the public or is a food handler (Fig. 34.1).

Ano-genital warts present unique problems as they are usually sexually transmitted and may predispose to cervical, anal, penile and oropharyngeal cancer (Fig. 34.2). All patients with genital warts should have a full STI screen and contact



**Fig. 34.1** Filiform wart on the nose pre-shave biopsy for histology in a 75 year old female with Type 2 Diabetes

tracing. It is usually best carried out within a STI clinic.

### 34.2 Clinical Features and Diagnosis

Infection of keratinocytes by the human papilloma virus (HPV) causes hyperkeratinisation and epidermal thickening. This results in papules and nodules with thick keratin or papillomatous surface. Diagnosis is usually obvious especially in children but warts can be more difficult to diagnose in certain areas such as the feet and the genital areas. Biopsy may be necessary to confirm the diagnosis before initiating treatment if there is any doubt about the diagnosis on visual inspection (Fig. 34.3a, b).

There are many types of warts which can affect various parts of the body (Table 34.1).



**Fig. 34.2** Genital warts



**Fig. 34.3** (a) Warty growth with an underlying NMCS (Bowen's disease). (b) Warty growth post removal scab with an underlying area of Bowen's disease



They can vary in size from 1 to 2 mm up to 20 mm and more (Fig. 34.4a, b, c). They may be solitary, multiple or grouped together (mosaic) (Fig. 34.5a, b). Paring down a wart usually reveals tiny bleeding or thrombosed capillaries.

**Table 34.1** Types of warts

<b>Common warts</b> ( <i>verruca vulgaris</i> )—mostly on the hands and knees
<b>Flat (plain) warts</b> ( <i>verruca plana</i> )—found on the face in some women
<b>Filiform warts</b> ( <i>verruca filiformis</i> )—usually found near the mouth and nose
<b>Periungual warts</b> —usually from picking and biting around the nails.
<b>Butcher's warts</b> —from repeated minor trauma.
<b>Kissing warts</b> —autoinoculation
<b>Plantar wart</b> , or <i>verruca</i> ( <i>verruca plantaris</i> )—on the soles of the feet and toes and grow inwards
<b>Mosaic warts</b> —a cluster of small warts coalescing together to look like one large one
<b>Genital wart</b> ( <i>condyloma acuminatum</i> ) = usually in the genitalia, perianal area or mouth.

The wart virus can display the Köebner phenomenon. This is where the wart seeds into a scratch or scar. This is sometimes helpful in diagnosing warts, such as a line of plain warts in a scar. The Köbner phenomenon can also cause warts to recur in the scar of previous surgery such as cauterity, excisional or cryosurgery, if the initial treatment is sub optimal and does not clear all the warts.

### 34.3 Differential Diagnosis

Warts are less common in the elderly and can occasionally be confused with a hyperkeratotic actinic keratosis, a BCC or an SCC. If in doubt take a biopsy or refer the patient for a second opinion. Table 34.2 lists other common conditions that can be confused with viral warts. Corns and *verruca* can be easily confused (Table 34.3). A verrucous periungual or ungual lesion in the elderly is most probably an SCC and not a wart.



**Fig. 34.4** (a) Warts on the knees in a 9 year old. (b) Wart on the lip. (c) Periungual warts in a picker and biter



**Fig. 34.5** (a) Mosaic of plantar warts. (b) Mosaic of plantar wart

**Table 34.2** Differential diagnosis for viral warts

Hyperkeratotic actinic keratoses
Cutaneous horn
Skin tags
Seborrhoeic keratoses
Molluscum contagiosum
Corns and callaces
Scars
Keloids
BCC/SCC/Melanoma
Sebaceous gland hyperplasia
Syphilitic condylomata

**Table 34.3** How to differentiate a plantar wart from a corn

Plantar wart	Corn
- On any part of the sole of the foot	- Over pressure points
- Thrombosed capillaries	- No vascular structures
- More tender when squeezed	- More tender when pressed
- Well demarcated (white ring)	- Poorly demarcated
- More common in young people	- More common in those >50 years old

### 34.4 Pathophysiology

HPV is not highly contagious and the HPV will not penetrate intact skin. The virus requires a portal of entry such as a crack or a cut in the skin to gain access. Biting and picking around the nails is probably the commonest cause of periungual warts. Biting a wart can be the cause of Heck's disease (focal epithelial hyperplasia which only affects the lining of the mouth). HPV infections of the genital tract can also be transmitted to the respiratory tract of a newborn child causing juvenile-onset recurrent respiratory papillomatosis. There are many subtypes of the HPV virus which have a predilection of infect various parts of the body (Table 34.4).

**Table 34.4** HPV subtypes causing warts

Wart type	HPV subtype
Common	1, 2, 4, 57
Plantar	1, 2, 4, 57
Mosaic	2
Plane	3, 10
Oral	6, 11, 32
Anogenital	6, 11, 16, 18, 31, 33, 35, 55, 56, 58

There are many unanswered questions about the simple common wart. Why do some people appear to be immune while other, apparently healthy individuals can be plagued by persistent and recurrent warts?

Patients with a depressed immune system (diabetes, HIV, chemotherapy, lymphoma, transplant patients, etc.) are more vulnerable to warts, find it more difficult to clear them and often relapse after treatment. In older patients, particularly if they are immunosuppressed, such as transplant patients, the wart virus may have onchogenic potential and can predispose to skin cancer such as squamous cell carcinomas. This may explain the greater incidence of SCC compared to BCC on the dorsum of the hand.

### 34.5 Treatment

Research into warts and their treatment are scanty and most of what is published is of poor quality and design. A recent Cochrane systemic review of 60 trials on warts showed that 46 (77%) were classified as low quality, that heterogeneity between trials was high, and analyses were often inappropriate or misleading. Comparison of different treatments is often difficult to assess, as the exact techniques used for treatment of the warts is not always described.

The decision to treat warts has to be made on a case by case basis according to the experience of the doctor, patient's preference and the application of evidence based medicine (Table 34.5).

**Table 34.5** Treatment options

- No treatment
- Traditional cure/placebo
- Topical salicylic acid
- Topical glutaraldehyde
- Cantharidin
- Cryosurgery
- 5% imiquimod ("Aldara")
- Topical podophyllotoxin
- 3% Formalin soaks
- Monochloroacetic acid crystals embedded in 50% salicylic acid
- Pulse dye laser
- Photo dynamic therapy
- Curettage and cauterity
- Systemic retinoids
- Intralesional bleomycin
- Intralesional interferon
- Intralesional immunotherapy
- Diphenylcyclopropenone (DCP) contact immunotherapy

If warts have to be treated (for pain or cosmetic concerns), the first line of treatment is usually with topical treatments such as salicylic acid or a traditional cure. **Traditional cures** probably get their reputation by the fact that most warts will regress and clear spontaneously within time. A placebo has a 27% success rate [3]. One should never underestimate the power of persuasion, wishful thinking and the ability of mind to cure the body. The exact type of traditional cure seems less important than the enthusiasm of the person imparting the technique. Traditional cures probably work best in children who are more suggestible, naïve and gullible than adults. Warts are also more likely to clear spontaneously in this age group. Popular traditional cures include holy (wishing) wells, banana skin, duct tape, first spit in the morning, the crossroads treatment, etc.

There are many over the counter topical agents used to treat warts in children and adults. Some of these are keratolytic such as **salicylic acid**

**Table 34.6** Examples of salicylic acid based topical treatments for viral warts (% salicylic acid)

“Cuplet + gel®”	11%
“Vericaps plasters®”	11%
“Salatac gel®”	12%
“Salactol paint®”	16.7%
“Duofilm®”	16.7%
“Occlusal paint®”	26%
“Verrugon ointment®”	50%
“Pickles ointment ®”	50%

(Table 34.6). A 26% strength is safe and effective for hands and feet (not the face), in adults and children. 50% salicylic acid can be helpful for resistant plantar warts in adults and children over the age of 6 years old. Others topical agents are verrucidal such as **glutaraldehyde 10%**. Success in treatment is often dependent on the enthusiasm and persistence of the patient or parent. Most will work better if applied after paring down the hard keratin with an emery board or blade every night before applying the topical agent and continuing the treatment for at least 6–12 weeks. When used properly, salicylic acid has a cure rate 75% [4]. Most patients and parents fail to persist with these topical treatments unless it is clearly outlined to them how to apply the treatment and how long the treatment will take.

**3% formalin soaks** daily for 6–12 weeks can sometimes help clear mosaic plantar warts (Fig. 34.5a, b). The plantar wart should be pared down nightly with an emery board or nail file and then the wart is soaked in a bowl of formalin or cotton wool soaked in formalin for 10–20 min. The surrounding healthy skin can be protected with “Vaseline®” if necessary.

**Monochloroacetic acid crystals** embedded in 50% salicylic acid cream can be helpful in isolated plantar warts less than 10 mm in patients who are not suitable for cryosurgery or other topical agents [5]. The cream and crystals are held in place over the wart using a corn plaster and tape for 3 days. This treatment causes a sterile abscess which lifts the wart from the surrounding skin. The abscess needs to be incised and drained (which can be difficult in children) and the plantar wart can be shelled out leaving a clean ulcer which will usually heal in 2–3 weeks.

**Cantharidin** is derived from the blister beetle, *Cantharis vesicatoria*. It causes epidermal

cell death, acantholysis, and clinical blister formation through its action on mitochondria. Cure rates have been reported to be as high as 80% for common, plantar and periungual warts [3]. It is relatively painless on application but the resulting blisters can be sore. This agent is, as yet, only available in Canada and China.

**Photo-Dynamic Therapy (PDT)**, is sometimes used for treating warts but it is time consuming, expensive and results are variable.

**Pulse dye lasers** can be effective when treating warts that are not too bulky. Removing the thick keratin is important before laser treatment as the light will not penetrate thick dark keratin. Like cryosurgery, this can be painful and may require local anaesthetic. One of the advantages is that there is little or no swelling or blistering following laser treatment but you may get bruising that can last one week. It is thought to work by blocking the small feeding capillaries, thus causing an ischemic necrosis.

**Intralesional bleomycin or interferon** can be an effective treatment for resistant warts but is, once again, expensive, very painful and only available in some hospital dermatology departments [6]. **Intralesional immunotherapy** using various antigens such as the measles, mumps and rubella (MMR) vaccine or candida skin test antigen causes a delayed hypersensitivity reaction, increasing the ability of the immune system to recognise and clear the HPV [7].

One small placebo-controlled clinical trial used **oral zinc sulphate** (10 mg/kg daily up to a max of 600 mg/day) to treat recalcitrant warts. Complete clearance was reported in 87% of the treatment group versus no clearance in the placebo group [8, 9]. No serious side-effects were reported apart from nausea (16%), mild gastric pain (3%) and itching sensation (3%). The total daily dose should be taken over three doses with milk or yogurt for two months. It should not be used in pregnancy or when breast feeding. Zinc deficiency is thought to lead to reduced immune capacity.

**Diphenylcyclopropenone (DCP)**, also known as diphenylyprone, is a skin sensitising agent used occasionally to treat warts and alopecia areata by contact immunotherapy. Initial sensitisation to DCP is required for the treatment to work. A small test patch of high concentration

DCP (2%) is left in contact with the skin for 2–3 day to induce contact allergy. The doctor or nurse then applies a weaker concentration of DCP to the warts once weekly. It is extremely easy for health care professionals to become sensitized the DCP so it is usually only used in certain dermatology hospital departments.

### 34.6 Ano-Genital Warts

**Podophyllotoxin** (“Warticon®” or “Condyline®”), which has anti mitotic activity, was the most common way to treat genital warts. However, treating genital warts with **5% imiquimod cream** (“**Aldara®**”) might be better as it will help clear the clinical and sub clinical warts (Fig. 34.6). “Aldara®” is a topical immune response modifier that stimulates the patient’s own immune response to help clear HPV. It should be applied overnight three times a week, for 6–12 weeks. Patients should be warned that the warts can become irritated and painful during this treatment. Recent surveys have

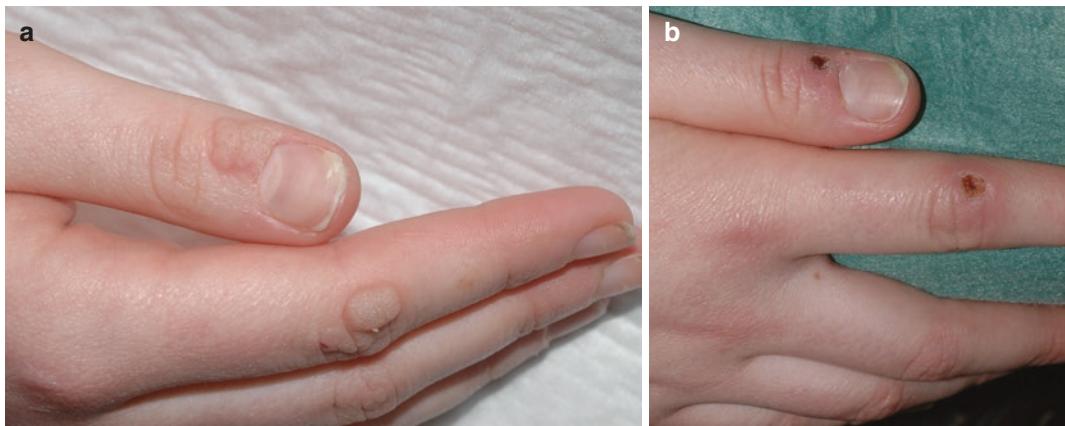
shown that combining “**Aldara®**” with **cryosurgery** seems to be more effective than either treatment alone (“cryoimmunostimulation”) [10]. It is best to consider this combination when there are large, numerous or persistent genital warts. There are different protocols: one is to treat with “**Aldara®**” alternate days for the first 2 weeks. Then treat the warts with relatively gentle cryosurgery under topical anaesthetic (5–10 seconds freeze, one freeze thaw cycle) and continue “**Aldara®**” immediately post cryosurgery, applying it on alternate days for another 1 week. This cycle can be repeated at least once if necessary. There is some evidence that this combination can also help clear non ano-genital warts [11]. Very large genital warts may have to be surgically removed under local or general anaesthetic followed by cryosurgery and/or imiquimod 5% for any residual small warts [12]. All patients with ano-genital warts should have a STI screen and their partner(s) should also be checked out for STIs. Ano-genital warts in children should raise the suspicion of child sexual abuse and if there are any concerns an STI screen and referral to paediatrics and social services should be considered.



**Fig. 34.6** Genital warts pre-cryosurgery and imiquimod

### 34.7 Vaccination

In recent years safe effective vaccines have been introduced against certain strains for the HPV for teenage girls (and boys in some countries such as Australia and the US). This has dramatically reduced the incidence of genital warts and hopefully cervical cancer in women and anal, penile and oral cancers in men eventually. “**Gardasal 9®**” is the vaccine used in Ireland and protects against Human Papillomavirus Vaccine Types 6, 11, 16, 18, 31, 33, 45, 52 + 58 which are the strains predominantly linked with cervical cancer (Table 34.4). “**Gardasil 9®**” vaccine is made from tiny proteins that look like the outside of the real human papillomavirus (HPV). The vaccine does not contain any live virus, or even killed virus or DNA from the virus, so it cannot cause cancer or other HPV-related illnesses. In Ireland it is currently given free of charge to teenage girls in schools and to men who have sex with men and people who are HIV positive (via the STI clinics).



**Fig. 34.7** (a) Periungual and common warts. (b) Periungual + common warts 1 month post-cryosurgery

## 34.8 Conclusion

Some patients (or their parents) with unsightly or painful warts do not want to use a cumbersome, uncomfortable, time consuming treatment with topical agents such as salacylic acid that may only have a 75% cure rate after 12 weeks [13]. They will often prefer treatment with **cryosurgery** (with local anaesthetic if necessary), which should clear the warts in over 90% of cases with one single session of cryosurgery and the wound usually heals up in 2–3 weeks without scarring [14–17] (Fig. 34.7a, b). Cryosurgery for warts is discussed in Chaps. 58 and 59.

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# Bugs and Bites

35

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## Key Points

- The cardinal sign of scabies is burrows, which harbour the mite, eggs and multiple brown feces (scybala).
- Most men with scabies have nodules on their penis, which, when identified, can help in the diagnosis.
- In women, an itchy, eczematous rash on the nipples is very common with scabies.
- If a tick is infected by a Gram-negative spirochete bacteria known as *Borrelia burgdorferi* (the causative agent of Lyme disease), the patient may develop the typical rash of erythema chronicum migrans 1–3 weeks after the bite.
- The typical rash of erythema migrans and a history of a recent tick bite is sufficient to make a diagnosis of Lyme disease and initiate therapy immediately, which is usually amoxicillin 500 mg three times a day for 2 weeks.
- The generalised itch and rash of scabies are primarily due to an allergic “id reaction” to the mite, eggs and feces (scybala) in the burrows. You do not have mites everywhere you are itching.
- All household contacts, boyfriend/girlfriend and childminders of people with scabies should be treated for scabies at the same time, regardless of whether they have an itch or not.
- Most biting ticks in Ireland do not transmit Lyme disease.

## What to Tell the Patient

- Scabies is contagious. You can get it from sexual contact, sleeping on infested sheets or prolonged contact with the skin of a person that is infested. You cannot get it from casual contact like shaking hands.

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## 35.1 Introduction

Insects and mites are a common cause of various skin problems seen in general practice. Although most are harmless and only cause temporary itch or rash, some biting insects can transmit potentially life threatening infections such as malaria, dengue, chikungunya, yellow fever and Zika infection (all mosquito transmitted), leishmaniasis (sandfly transmitted) or Lyme disease (from tick bites).

## 35.2 Scabies (*Sarcoptes scabiei var. hominis*)

If a young person develops a generalised itchy rash for no apparent reason and with no previous history of skin problems, one should always consider sca-

bies (see also Chap. 22). Clues to the diagnosis include close household contacts or friends also having an itch and the rash not responding to simple treatments such as emollients and topical steroids. Scabies can occur in all social classes and has nothing to do with a lack of hygiene. Scabies is contracted from other humans and not from animals.

Men with scabies can have nodules on their penis, which, when identified, can help in the diagnosis. Men rarely volunteer this information and you have to go looking for the nodules. They are extremely itchy and are secondary inflammatory response to scabies. They do not usually disappear after scabies treatment and may need to be treated with topical corticoids. In women an itchy, eczematous rash on the nipples is very common with scabies.

The cardinal sign of scabies is burrows, which harbour the female mite and eggs. They are most commonly found between the fingers, on the sides of the hands, on the flexor aspect of the wrists and on the side of the feet. In babies, burrows are often found on the soles of the feet (Fig. 35.1). A careful search with good light and magnification usually reveals burrows. Some patients might only a handful of burrows spread around the body, which are not always easily found without good light and magnification. When examined under a dermatoscope, the mite can be seen at the advancing edge of the burrow and has the shape of a fighter jet plane (see Chap. 46).

The best way to confirm the diagnose of scabies is to remove the mite using a number 15 blade and a magnifying lens from the advancing

edge of the burrow and then place it on a microscope slide for inspection. The mite has an unusual sticky quality, which helps it to stick to the tip of the blade when the burrow is picked. The mite is barely visible with the naked eye but is easily identified with a microscope. By showing the patient the mite under the microscope, 100% treatment compliance is guaranteed by the patient and by all household contacts! If the doctor cannot isolate the mite, scybala or eggs, a careful history and physical examination may be sufficient to make a clinical diagnosis of scabies or a suspected diagnosis of scabies. A confident diagnosis can be made using dermoscopy in experienced hands (Table 35.1 and see Chap. 22, Fig. 22.2a–c).

Despite popular myths, scabies is not extremely contagious. It does not fly or hop. It crawls very slowly from human to human. This requires a “heat bridge” such as prolonged holding hands, cuddling up on the couch or sharing a bed.

**Table 35.1** Summary of 2018 IACS criteria for the diagnosis of scabies

<b>A: Confirmed scabies</b>
<i>At least one of:</i>
1: Mites, eggs or feces on light microscopy of skin samples
2: Mite visualized on individual using dermoscopy
<b>B: Clinical scabies</b>
<i>At least one of:</i>
1: Scabies burrows
2: Typical lesions affecting male genitalia
3: Typical lesions in a typical distribution and two history features
<b>C: Suspected scabies</b>
<i>One of:</i>
1: Typical lesions in a typical distribution and one history feature
2: Atypical lesions or atypical distribution and two history features
<b>History features</b>
1: Itch
2: Close contact with an individual who has itch or typical lesions in a typical distribution

*Notes: A diagnosis of Clinical and Suspected scabies should only be made if other differential diagnoses are considered less likely than scabies*

Ref: Daniel Engelman, L. Claire Fuller, Andrew C. Steer. Consensus criteria for the diagnosis of scabies: A Delphi study of international experts. For the International Alliance for the Control of Scabies Delphi panel Published: May 24, 2018. <https://doi.org/10.1371/journal.pntd.0006549>; <http://bit.ly/2VqXRz9>



**Fig. 35.1** Scabies in 3-months-old baby

**Table 35.2** The amount of permethrin 5% w/w (“Lyclear Dermal Cream®”) required to treat a person from the neck down to the toes for two applications

Adult	60 g (2 tubes)
Child 5–15 years old	30 g (1 tube)
Child 1–5 years old	15 g (half a tube)
Child 2 months to 1 year old	7.5 g (quarter tube)
Less than 2 months old	Contraindicated

Treatment involves rubbing on a scabicide cream such as permethrin 5% (“Lyclear Dermal Cream®”) from the neck downwards to every part of the body, excluding the face and scalp. The lotion has to be left on for 12 h and the treatment repeated only once, one week later (to destroy any hatching eggs). All household contacts, boyfriend/girlfriend and childminders should be treated in exactly the same way and on the same day, regardless of whether they have an itch or not as some people can have scabies and no itch. Sufficient quantities of cream need to be prescribed (Table 35.2) and written instructions how to treat scabies will help with compliance (see patient advice leaflets). Permethrin 5% is not usually recommended in children less than 2 months old, in pregnancy and in lactating women. Avoid permethrin if the skin is severely irritated with many cracks as it can be absorbed. In babies it should be applied all over including the face and scalp but avoiding the mouth and eyes. Permethrin 5% is not licensed in children under the age of 2 months but crotamiton 10% cream (“Eurax”) can be used instead in this age group applying it daily ( $\times 24$  h) for 3 consecutive days from the top of the head down to the toes [1].

Another option is 8 or 10% precipitated sulfur in petrolatum ointment. It is the drug of choice for infants younger than 2 months of age and for pregnant or lactating women. The cream should be applied nightly for three consecutive nights and washed off 24 hours later. It should be reapplied 3 days per week for 2 or 3 weeks. The major drawback to sulfur treatment is the unpleasant odor.

The incubation period for scabies can be up to 4 weeks for the first infestation. Not everyone with scabies itches. The generalised itch and rash of scabies are primarily due to an allergic “id reaction” to the mite and eggs in the burrows. This is also known as autosensitisation dermatitis or

**Table 35.3** Reasons why a person with scabies is not responding to treatment:

Incorrect diagnosis.
Incorrect application of treatment.
Re-infection from an untreated close contact.
Irritation from repeated application of scabies treatments.
Post-scabies itch (can last up to 6 weeks).
Scabies resistant to the insecticide.
Crusted scabies.

autoeczematisation. Some patients can develop an intensely itchy, generalized eczematous rash that keeps them awake at night. Other patients seem to be relatively immune to the mite with little or no itch or rash yet can be infectious to others.

The mite can survive for 2–3 days away from human skin. Therefore, a person can get infested by reusing the same cloth (pyjamas, sheets, towels,...), by sleeping on a hotel that has not changed sheets and had previously been used by somebody with scabies, or by using the same towels. This is the reason why scabies is very easily transmitted in human groups with close contact (nursing home, infant day care facilities...). Simply laundering clothing and bed sheets that have been in contact with the skin at 50°, or using a dryer, or ironing the fabric will usually kill the mites. For crusted scabies, mattresses, soft furniture and flooring should be thoroughly cleaned and left in the sun for a few hours if possible and occasionally the house may have to be fumigated.

Table 35.3 outlines the most common reasons for treatment failure. Repeated application of scabies creams or lotions can irritate the skin and lead to an itchy rash that can mistakenly be diagnosed as persistent scabies.

After successfully treating scabies, the itch and rash should slowly fade over the next 2–6 weeks. Emollients, potent topical steroids and sedating antihistamines can help to ease the itch, once the treatment has been completed.

Some elderly patients and immunosuppressed patients can develop crusted (Norwegian) scabies where they have thousands or millions of mites on their skin, which can include their scalp with ironically little or no itch. These patients are highly infectious and difficult to treat. They may need treatment with topical permethrin and an oral broad

spectrum anthelmintic such as ivermectin (200 µg/kg body weight stat dose and repeated at least once more 7 to 14 days later). Ivermectin may also be required in large institutional outbreaks such as in nursing homes. It should not be used in children less than 15 kg, in pregnancy or in breast feeding mothers. Ivermectin is usually well tolerated but it may cause nausea, headache and itch. Moxidectin, which is commonly used in veterinary practice to treat a range of parasites, may be suitable for humans as a single once off dose for scabies, but it is not yet licensed or available for human use [2].

### **35.3 Head Lice (*Pediculus Humanus Capitis*)**

Head lice most commonly affect children of school going age and do not affect animals. Like scabies, the mites require a “heat bridge” that occurs with prolonged head to head contact. They will not survive for long without feeding so they are not usually caught from other people’s brushes or hats. However, unlike scabies, head lice can be seen with the naked eye. Mature adult lice are approximately 3 mm in length (the size of a small sesame seed). They are very shy and usually scurry into dark crevasses of the hair once light is shone on them. It is far easier to see the eggshells (“nits”), which are cemented onto the hair shaft and grow out with the hair. These empty shells are harmless and do not necessarily imply active infection. The most common place to find the lice and eggs is behind and above the ears.

The head lice feed on blood from the scalp. With heavy infestation, the multiple bites on the scalp can cause intense itch and sometimes secondary bacterial infection with cervical lymphadenopathy on the back of the neck. This will require treatment with an oral antibiotic such as flucloxacillin as well as treatment to eradicate the head lice.

The best way to treat head lice -in addition to the specific treatment- is by fine combing the hair when it is wet. Applying some conditioner makes it easier to fine comb. Good light and patience is required. If lice are found, they can be killed by squishing them on a hard surface. Fine combing has to be repeated every 4 days until no living lice

can be found. For heavy infestation, cutting the hair short or shaving the scalp helps.

Physical insecticides such as dimeticone (“Hedrin®”) or isopropyl myristate (“Full Marks®”) work by suffocating the lice by smearing them over the scalp and leaving them on for 15 min. The treatment should be repeated 1 week later as these products do not kill the eggs. All family members need to be checked and treated simultaneously if live lice are found.

Head lice have developed high levels of resistance to chemical insecticides with failure rates of up to 87% with permethrin and 64% with malathion [3].

For severe resistant cases oral ivermectin could be considered at an increased stat dose of 400 µg/kg body weight and repeated once 8 days later [4]. Because its overuse may result in resistance, oral ivermectin should be reserved for more difficult-to-treat cases.

Natural products such as tea tree oil and nerolidol have been used with success against lice and their eggs [5].

### **35.4 Pubic Lice (Crabs)**

Crabs are much less common than head lice but should be treated in a similar fashion. Shaving pubic hair is not as radical as shaving the scalp and allows for easier detection and treatment of



**Fig. 35.2** Papular urticaria from insect bites

the lice. Sexual partners may also need to be checked and treated if infected.

### 35.5 Papular Urticaria

Papular urticaria is a common reaction to insect bites and is commonly seen in the summer months. Despite the name it is not a true form of urticaria. Most insect bites occur around the ankles, wrists and face (exposed areas). These cause itchy papules of varying intensity, depending on the sensitivity of the patient. Some people can develop vesicles or bullae from the bites (Fig. 35.2). Eradicating the source (e.g., animals, plants, etc.) and using insect repellents are the best ways to prevent papular urticaria. Potent topical steroids and sedating antihistamines at night should relieve any itch. In Ireland, these bites are a nuisance but not dangerous. In tropical countries these can be much more serious as insect bites can transmit serious infections such as malaria, yellow fever or dengue fever.

### 35.6 Bed Bugs (*Cimex lectularius*)

Bed bugs are blood-sucking parasites that can also cause papular urticaria and sometimes blistering reactions. The oval shaped, flat, reddish-brown insect is up to 5 mm long, which can be seen with the naked eye. Like head lice, they are very shy and hop into dark corners when exposed

to light. All you may find are signs of “spotting” around the bed structures, walls and mattress where the bugs have defecated. They can survive for months in furniture and bedding without feeding. They usually feed at night without detection by the host. They generally bite two or three times and then rest. This causes groups of two or three papules in a linear fashion on exposed areas such as the face, neck and arms. They can be eradicated by general vacuuming, laundering at 60° and by encasing a mattress in plastic. Chemical control with insecticides can be helpful although there may be health consequences when humans are exposed to insecticides. There are also problems with the development of insecticide resistant bed bugs. Fumigation by commercial companies is sometimes necessary. They are very difficult to eradicate and a real headache issue specially for large cities like New York where there is mass infestation of bedbugs.

### 35.7 Tick Bites and Lyme Disease

Most tick bites do not cause problems for humans but occasionally they can cause serious infections such as Lyme disease, tick borne relapsing fever, Rocky Mountain spotted fever, tick-borne encephalitis and other serious infections, particularly when travelling overseas.

Many cases of **Lyme disease** in Ireland are caused by tick bites while travelling overseas, especially in parts of central, eastern and north-



**Fig. 35.3** (a) Tick attached to the skin but not yet fed. (b) Tick on dermoscopy = not fed yet

ern Europe (including Scandinavia) and parts of Asia, the US and Canada. Indigenous cases are becoming more common with the highest incidence in Kerry and Galway. Ticks are mainly found in grassy and wooded areas, including urban gardens and parks. Tick bites may not always be noticed. The tick usually attaches itself to the skin for 24–48 hours. If detected, the tick needs to be removed carefully with a fine tweezers. It is important to avoid squeezing it, as this will increase the risk of transmitting infection if the blood from the engorged tick is injected back into the host's body ("A Tick in Time Saves



**Fig. 35.4** (a) Typical erythema migrans of Lyme disease. Credit: Ffurler/CC BY-SA (<https://creativecommons.org/licenses/by-sa/4.0/>). (b) Insect bite hypersensitivity reaction

Lyme") [6]. Most tick bites do not transmit Lyme disease and immediate removal of the tick reduces the risk of transmission (Fig. 35.3a, b).

The main reservoir host for ticks in Ireland are woodland birds, deer, farm animals, pets and small hairy animals such as mice and squirrels. Due to their breeding patterns, the tick population is highest in late spring and early summer. If the tick is infected by a Gram-negative spirochete bacteria known as *Borrelia burgdorferi sensu lato* (the causative agent of Lyme disease), 70–80% of patients will develop the typical rash of **erythema chronicum migrans (also called erythema migrans)** (Fig. 35.4a). These skin lesions typically become apparent approximately 7–14 days (range, 3–30 days) after the tick has detached or was removed and should be at least 5 cm in largest diameter for a secure diagnosis. The erythematous rash spreads centrifugally and can extend to 10–20 cm with central clearing giving a "bull's eye" appearance. The rash usually occurs at the site of the bite, which is commonly on the leg, back, groin or axilla. It may be accompanied by flu-like symptoms. Occasionally there can be more than one **erythema migrans** rash on the body. The rash will resolve spontaneously without treatment. Left untreated, Lyme disease may lead on to long term neurological, rheumatological or cardiac complications.

An erythematous skin lesion present while an *Ixodes* tick is still attached or which has developed within 48 h of detachment is most likely a tick bite hypersensitivity reaction (i.e., a noninfectious process), rather than erythema migrans. Tick bite hypersensitivity reactions are usually <5 cm in largest diameter, sometimes have an urticarial appearance, and typically begin to disappear within 24–48 h (Fig. 35.4b). In contrast, an early primary erythema migrans lesion usually increases in size over this time frame. To differentiate between the 2 processes, it may be useful to mark the borders of the skin lesion with ink and then observe for 1–2 days without antibiotic therapy [7].

The typical rash of erythema migrans and a history of a recent tick bite are sufficient to make a diagnosis and initiate therapy immediately. The 2018 NICE guidelines [8] recommend doxycycline 100 mg twice per

**Table 35.4** Antibiotic choices summary form BMJ

	Adults 12+ and children >45 kg	Children <12 years old
First choice <sup>a</sup>	Doxycycline 200 mg/day orally × 21 days	9–12 years <sup>b</sup> (<45 kg) Doxycycline: Day 1: 5 mg/kg in 2 divided doses Day 2–21: 2.5 mg/kg/day orally (Doxycycline not recommended for children <9 years)
Second choice <sup>a</sup>	Amoxicillin 1 g × 3 times a day orally × 21 days	Children <33 kg: Amoxicillin 30 mg/kg × 3 times per day × 21 days orally
Third choice <sup>a</sup>	Azithromycin 500 mg daily orally × 17 days	Children <50 kg: Azithromycin 10 mg/kg daily × 17 days orally

Specialist advice may be required for children, pregnant women, severe or resistant cases

<sup>a</sup>Consider a second course of an alternative antibiotic for people with ongoing symptoms

<sup>b</sup>Doxycycline has no marketing authorisation for children <12 years. However, use in children aged 9 years and above is accepted specialist practice. Informed consent should be obtained, and full responsibility taken by the prescriber

day × 21 days as a first line treatment for non pregnant adults. Amoxicillin 1000 mg 3 times per day × 21 days is the recommender second line treatment and azithromycin 500 mg daily × 17 days is recommended as a third line treatment. Ceftriaxone 2 g twice a day for 21 days is recommended as a first line treatment of Lyme disease affecting the central nervous system in adults with doxycycline 200 mg twice a day for 21 days a second line option in adults (see Table 35.4).

“Most people who are infected with Lyme disease have a circular, red rash surrounding the site of a tick bite, that may be accompanied by muscle and joint aches and less commonly, facial (Bell’s) paralysis,” said USA Lyme disease expert Paul Auwaerter. “The symptoms are sometimes alarming, but with proper diagnosis and antibiotic treatment almost all will go away within a few weeks”.

Blood tests and skin biopsy can help to identify the infection in more severe or chronic cases

but are difficult to interpret and are probably best assessed by a microbiologist or infectious disease consultant. Antibodies to *Borrelia burgdorferi* using ELISA or immunofluorescence assay can be checked and if positive they should be confirmed by Western immunoblot. Positive antibodies can be detected for many years after successful treatment. Sometimes the organism can be cultured or detected by polymerase chain reaction (PCR) on a skin biopsy. Treatment of chronic Lyme disease is complex and needs to be carried out by an infectious disease specialist. More information is available on the NICE guidelines [8].

Measures recommended by the IDSA (Infectious Disease Society of America) to reduce the risk of infection include the use of protective clothing, insect repellents containing 20–30% DEET (N,N-diethyl-m-toluamide), checking the entire body for ticks daily if in an infectious area, and prompt careful removal of attached ticks before transmission of infection can occur (usually takes 24–36 h for the tick to attach to the skin and transmit the infection) (Table 35.4).

According to the IDSA, routine use of antimicrobial prophylaxis or serologic testing is not recommended to prevent Lyme disease after a recognized tick bite. A single dose of doxycycline may be offered to adult patients (200 mg dose) and to children ≥8 years of age (4 mg/kg, up to a maximum dose of 200 mg) when all of the following circumstances exist:

- (a) the attached tick can be reliably identified as an adult or nymphal *I. scapularis* tick that is estimated to have been attached for ≥36 h on the basis of the degree of engorgement of the tick with blood or on certainty about the time of exposure to the tick (Figs. 35.2 and 35.3),
- (b) prophylaxis can be started within 72 h of the time that the tick was removed,
- (c) ecologic information indicates that the local rate of infection of these ticks with *B. burgdorferi* is ≥20%, and
- (d) doxycycline is not contraindicated (e.g. doxycycline is contraindicated in children under 9 years of age and in pregnant women).

Infection of  $\geq 20\%$  of ticks with *B. burgdorferi* generally occurs in parts of New England, in parts of the mid-Atlantic States, and in parts of Minnesota and Wisconsin, but not in most other locations of the United States.

Kirstein and colleagues [9, 10] reported infection rates of *B. burgdorferi* s.l. in questing ticks collected in a number of Irish forests and national parks of between 3.5 and 26.7% in a paper published in 1997 (including 3.5% in Glenveagh and 18.8% in Killarney National park; 26.7% in Avondale, 4.4% in Lough Key and 16.4% in Portumna Forest Parks and 24.2% in Garinish Island).

Patients who have had a tick bite should be shown pictures of the rash of erythema migrans and advised to report to a doctor if they develop a similar rash or a flu-like illness within 1 month of being bitten.

### 35.8 Conclusion

Biting insects and scabies are a common cause of human misery. In the tropics, insects can transmit potentially life threatening diseases. Resistance to insecticides, climate change and an increase in international travel may result in some of these tropical illnesses becoming more common in Europe and North America.

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## **Part VII**

### **Regional Dermatology**



# Regional Dermatology

36

David Buckley

## Areas Covered

- Head and neck
- Scalp
- Beard area
- Forehead and cheeks
- Ears
- Eyelids and inner canthus
- Nose
- Lips and mouth
- Neck
- Trunk
- Flexural areas
- Hands
- Feet
- Female and male genitalia
- Peri anal area
- Lower legs

## Key Points

- Common rashes such as atopic eczema, contact or irritant dermatitis, seborrhoeic dermatitis, psoriasis and fungal infections can all look very similar and diagnosis may only be apparent by taking a detailed history and doing a thorough physical examination looking at the distribution of the rash and other clues as to the diagnosis.

- Treatment of the same condition may vary depending on which part of the body is involved.
- Asymmetrical or unilateral rashes are more likely to be due to infections such as tinea, impetigo, herpes or shingles.

## What to Tell the Patient

- Almost all hand rashes will be helped with a good, hypo-allergenic, greasy moisturiser, avoiding soaps and other irritants and the careful use of gloves for wet jobs.
- Most foot rashes will be helped by wearing leather soled shoes and cotton socks in the winter and open, hypoallergic leather soled sandals in the summer.
- Avoid walking barefoot as foot rashes may be infectious to others or may make you prone to picking up infections such as verrucae or athlete's foot.

## 36.1 Introduction

Certain rashes and lesions have a predilection for certain areas on the body. For instance, xanthelasma generally only occurs on the medial aspects of the upper and lower eyelids and intertrigo generally only occurs on the folds of skin such as under the breasts or lower abdomen and groin, especially in obese individuals.

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Other rashes and lesions may occur in many different areas of the body but are more commonly found in certain areas (e.g., psoriasis classically affects the scalp, back of the elbows and the front of the knees; viral warts most commonly occur on the fingers and hands). Some generalised rashes such as eczema and psoriasis may be confined to certain areas of the body if they are mild or present in the early stages of their development or if they are partly treated.

## 36.2 Diagnosis and Clinical Features

When primarily confined to a localised area, certain common rashes such as atopic eczema, contact or irritant dermatitis, seborrhoeic dermatitis, psoriasis and fungal infections can all look very similar. Diagnosis may only be apparent by taking a detailed history (including previous medical history, family history, occupation and hobbies) and doing a thorough physical examination of all the skin looking for discrete clues to the diagnosis (e.g. nail pitting may suggest psoriasis or coexisting asthma may suggest atopic eczema).

The age of the patient may help when making a diagnosis. Younger patients are more likely to suffer from atopic eczema, congenital naevi and infectious rashes such as warts and impetigo. Adults are more likely to suffer from psoriasis and other inflammatory skin diseases such as lichen planus and occupational related skin diseases such as contact irritant dermatitis. The elderly are more likely to suffer from photodamaged skin and degenerative diseases such as actinic keratosis, non-melanoma skin cancers, diabetic related skin diseases, and skin problems associated with varicose veins such as varicose eczema or ulcers. Because the elderly are often taking far more medications, drug related skin problems are more common in this age group.

The distribution of a rash may help in the diagnosis. Symmetrical rashes are more likely to be due to eczema or psoriasis whereas asymmetrical or unilateral rashes are more likely to be due

to infections such as tinea, impetigo, herpes or shingles. Sometimes investigations such as swabs for culture and sensitivity, skin scrapings for fungal stain and culture or skin biopsies for histological diagnosis may be required to clinch the diagnosis. Superficial basal cell carcinoma (BCC), actinic keratosis, Bowen's disease, squamous cell carcinoma and amelanotic melanoma may all look similar and often require a biopsy to make a definitive diagnosis.

An overlap between two conditions is not uncommon on the body, such as contact allergic and contact irritant dermatitis on the hands or tinea pedis and pompholyx on the feet.

## 36.3 Lesions and Rashes on the Head and Neck



All the common dermatoses such as eczema/dermatitis, psoriasis, acne and rosacea may occur on the head and neck. These rashes are usually obvious from their clinical features and distribution. Bacterial (e.g. impetigo, cellulitis, erysipelas), viral (e.g. herpes simplex, herpes zoster, viral warts, molluscum contagiosum), and fungal infections (e.g. tinea capitis, corporis, barbae) are all common on the head and neck.

The head and neck are exposed so photosensitive rashes and lesions aggravated by excessive ultraviolet light are much more common in these areas.

Solar elastosis, solar lentigo, actinic keratosis, cutaneous horns, non-melanoma skin cancer (Bowen's disease, BCC and SCC and keratoacanthoma) and melanoma including lentigo maligna are often found on the head and neck.

## 36.4 Scalp Rashes and Lesions

### 36.4.1 The Hair Bearing Scalp

The most common scalp problem is **dandruff**. This presents with white, dusty, scales on the scalp that can be very noticeable on dark clothing. There may or may not be an itch. There is no redness or inflammation of the scalp. Treatment is with an anti-dandruff shampoo such as Selenium Sulphide ("Selsun®") or Zinc Pyrithione ("ZP11®", "Head & Shoulders®") all of which have weak antifungal activity. Tar shampoos may also help. For more resistant cases ketoconazole ("Nizoral shampoo®") or ciclopirox olamine ("Stieprox shampoo®") are effective, safe and convenient. These should be used twice a week for 1 month and then once a week indefinitely to prevent relapse.

**Seborrhoeic dermatitis** may be considered a more severe form of dandruff where there is redness and scaliness of the hair bearing areas of the scalp. It is caused by sensitivity to the commensal *Pityrosporum ovale* yeast in susceptible individuals. There is normally evidence of seborrhoeic dermatitis on other parts of the body such as the eyebrows, nasolabial folds, beard area, over the sternum, between the shoulder blades, in the

groin, perianal area and sometimes on the penis. Treatment is with an anti-dandruff and tar based shampoo as outlined above for dandruff. If seborrhoeic dermatitis affects the body then the shampoo should be lathered up on the scalp and then applied to the affected areas of the body and left to soak in for 2 or 3 min before rinsing it out. Conditioner should be avoided on sensitive scalps but can be used on the hair. More troublesome seborrhoeic dermatitis on the face and body usually responds to weak topical steroids mixed with an anti-yeast agent such as miconazole ("Daktacort®") or 1% Clotrimazole ("Canesten HC®"). More severe cases usually respond to tacrolimus ("Protopic®") although it is not licensed for this indication.

**Psoriasis** commonly affects the hair-bearing areas of the scalp and presents with thick heaped up scales and a sharp demarcation between the involved and uninvolved skin (Fig. 36.1). There are usually signs of psoriasis on other parts of the body. Severe psoriasis can cause temporary hair thinning. Most topical treatments will not work until the thick scales are removed. The best way to do this is with a tar and salicylic acid product such as "Cocois®" which can be rubbed into the thick scales and left on for a few hours and then washed out with a tar based shampoo. This usually removes the thick scales after 5–7 days and then a more specific anti-psoriasis treatment such as potent topical steroids (e.g. betamethasone) or a steroid combined with calcipotriol ("Dovobet gel®") can be used. "Dovobet gel®" should be applied at night and washed out in the morning. Dithranol is also effective in the scalp once the



**Fig. 36.1** Scalp psoriasis

scales are removed but it may stain light blonde or white hair.

**Pityriasis amiantacea** is similar to a localised patch of psoriasis in the scalp in children but the scales attach to the hair and are elevated from the scalp as the hair grows out (Fig. 36.2). Treatment is also similar to psoriasis. The scales can be softened and removed with a tar and salicylic acid ointment, followed by a moderately potent topical steroid for a few weeks.

**Head lice** can cause an itchy scalp and may become secondarily infected in more severe cases. The most obvious sign of head lice are nits which are empty egg shells cemented onto the hair shaft close to the scalp. The presence of nits does not confirm active infestation. Careful inspection of the whole scalp using a fine comb



**Fig. 36.2** Pityriasis amiantacea in a 8 year old boy

should be undertaken looking for living, moving lice which are about the size of a sesame seed. If a living, moving louse is found the scalp should be treated with a head lice lotion or shampoo such as 1% permethrin ("Lyclear cream rinse<sup>®</sup>") or 0.5% malathion ("Quellada<sup>®</sup>" or "Derbac M<sup>®</sup>"). All household contacts should have their heads closely inspected and treated only if living, moving lice (not just nits) are found.

**Tinea capitis** is a form of ringworm that is caused by dermatophyte fungus. It is most commonly seen in children. The most common cause is *Microsporum canis* from cats and dogs. Cattle ringworm (*tinea verrucosum*) can also affect the scalp. *Tinea tonsurans* and *tinea violaceum* may also cause scalp ringworm and can be spread from person to person. These fungi are more common in people of Afro Caribbean descent.

Scalp ringworm usually presents with a localised patch of hair loss associated with an underlying inflammatory, scaly scalp rash. The diagnosis can be confirmed by taking skin scrapings or plucking hair for fungal stain and culture. Treatment is usually with an oral antifungal such as terbinafine or itraconazole for at least 4–6 weeks. If animal fungus is found on culture, the animal source should be identified and treated. Antifungal shampoos such as ketoconazole may help prevent infection in other family members.

A **kerion** is a thick boggy mass on the scalp as a result of an aggressive immunological reaction to scalp ringworm seen in some children (almost never in adults). Treatment is with a prolonged courses of oral anti-fungal medication and oral steroids may sometimes be required to dampen down the inflammatory reaction during the course of antifungal treatment. Oral antibiotics may be required if there is secondary bacterial infection. A kerion should never be incised and drained or surgically removed.

**Eczema** of the scalp is common and often caused or aggravated by hair care products such as shampoos, conditioners, perms and colours. Patients usually present with a generalised tight, itchy, scaly scalp and there may be signs of eczema in other adjacent areas such as the ears, the hairline and the forehead. A search should be

made for the offending hair product which may be obvious from the history or may only be found after allergy testing such as a skin patch test. Treatment is usually with a potent topical steroid such as betamethasone ("Betnovate Scalp Application<sup>®</sup>") or a potent steroid shampoo such as clobetasol propionate ("Etrivex<sup>®</sup>") which should be applied as a lotion to the scalp for 15 min and then washed out in the shower. Gentle hair care products such as a soap-free shampoo should be used until the scalp has recovered. Conditioner should only be used on the ends of the hair and not on the scalp.

#### 36.4.2 Scalp Rashes and Lesions on the Non-hairy Areas of the Scalp

The most common rash on non-hairy areas of the balding scalp is **actinic keratosis** which usually occurs in the scalp of balding men, who spend a lot of time outdoors or who have worked in hot climates. **Photosensitive rashes** may also occur on the balding scalp (e.g. lupus and actinic dermatitis).

### 36.5 Scalp Lesions

**Pilar cysts** (also known as a trichilemmal cysts) are common on the hairy or bald scalp and are similar to a sebaceous cysts but have no punctum and rarely become infected. They only need to be removed if they are uncomfortable or unsightly. Other benign lesions that can occur on the scalp include **seborrhoeic keratosis** and **viral warts**. **Solar lentigo**, **actinic keratosis**, **non-melanoma skin cancer**, **lentigo maligna** and **melanoma** may all occur in the scalp, especially if there is hair thinning or balding.

**Sebaceous naevi** most commonly occur on the scalp. It is a non-melanocytic congenital skin hamartoma consisting of a benign overgrowth of the epidermis, sebaceous glands, hair follicles, apocrine glands and connective tissue. They usually appear in childhood as a solitary, small, bald patch with yellow-orange skin in an oval or linear shape.

As they mature in adulthood they become more rough and warty in appearance (Fig. 36.3). Sebaceous naevi do not require treatment unless they have transformed into a malignancy (mostly BCC), a rare but possible occurrence, or for cosmetic reasons. Histology reveals characteristic thickening of the epidermis with mature hair follicles and prominent sebaceous glands. Malignant transformation usually presents as a fleshy, ulcerating, bleeding growth within the naevus which should be biopsied for histological diagnosis and to plan definitive treatment which usually involves surgical removal of the complete naevus.

A boggy scalp abscess in a child's scalp is usually a **kerion** (see above under fungal infection) and should never be incised and drained (see Chap. 31).



**Fig. 36.3** Sebaceous naevus on the scalp since birth in a 16 year old

## 36.6 Beard Rashes

There are a number of rashes directly associated with beard growth. The most common is **pseudofolliculitis barbae** (also known as ‘shaving bumps’ or ‘razor bumps’) which is caused by hair emerging from the follicle at an acute angle and curling around to grow back into the skin, thus causing a foreign body type reaction with papules and pustules (Fig. 36.4a, b). Treatment is by trying to lift out the ingrown hairs. This may be achieved by rubbing the skin in the opposite direction to which the hairs are emerging from the skin with a buf-puf (a hard facial sponge) so as to make the hairs stand up and then shaving them in the same direction. A topical antibiotic with or without 1% hydrocortisone may also help. The ultimate treatment for resistant cases is growing a beard or removal of the beard with lasers or IPL (intense pulsed light) treatment.

**Sycosis barbae** is a type of folliculitis of the beard area caused by *Staphylococcus aureus* and characterised by discrete papules and pustules which originate in the hair follicles. The condition may affect the beard area diffusely or in a localised patch which may become encrusted. Treatment is with a prolonged course of topical or oral antibiotics. For chronic conditions, the use of topical antibiotics like clindamycin after shaving for a prolonged period of time can help in this chronic condition. Nasal carriage of *Staphylococcus aureus* should be treated if found.

**Tinea barbae** is unusual nowadays and is caused by animal ringworm. It usually presents as an extensive scaly pustular rash on the cheek or chin in farmers. Affected hairs can be easily dislodged. Fungal staining and culture of the scales or plucked hair are diagnostic. Secondary infection is not uncommon so a positive bacterial culture may be misleading. Treatment is with oral terbinafine (“Lamisil tabs®”) or itraconazole (“Sporanox®”) for 2–4 weeks.

**Herpetic folliculitis (Herpetic sycosis)** can occur as a deep seated infection in the beard area from HSV type 1 which may be spread from a cold sore through shaving. It may be recurrent in the same area every time and patchy especially on the upper lip. Treatment is with systemic anti-virals.

**Warts** are common in the beard area, on the cheeks and anterior neck and can be particularly difficult to treat. They usually respond to cryosurgery but relapse is common. Sometimes combining cryosurgery with 5% imiquimod cream (“Aldara®”) can help eradicate the virus. Growing a beard may help as it eliminates the trauma of shaving which may be spreading the virus. Flat warts can respond to topical retinoic acid in cream for long periods of time.

**Seborrhoeic keratosis, actinic keratosis, non-melanoma skin cancer and melanoma** can occur in the beard area where they may be camouflaged or hidden by a beard.



**Fig. 36.4** (a) Pseudofolliculitis barbae in a 22 year old. (b) Same patient—pseudofolliculitis barbae—close up

### 36.7 Forehead and Cheek Lesions and Rashes

Some lesions and rashes occur most commonly on the forehead and cheeks. **Sebaceous gland hyperplasia** results in enlarged sebaceous glands most commonly found on the forehead and cheeks in middle aged and elderly patients. It appears as small pinkish, yellow nodules up to 3 mm in diameter. There are often prominent blood vessels around the lesion. There may be a central hair follicle with yellowish lobules (Fig. 36.5). Sebaceous gland hyperplasia can be confused with a BCC. Sebaceous gland hyperplasia is completely harmless and benign. They can be unsightly and can be removed by shave biopsy and light cautery.



**Fig. 36.5** Sebaceous gland hyperplasia on the forehead

Specimen should be sent for histology to ensure there is no underlying malignancy.

Other benign tumours that may be found in the face include **sebaceous adenoma** (SA) and **trichilemmoma**. Sebaceous adenomas are usually small nodules, 2–4 mm in diameter, with a smooth surface and yellow colour, sometimes with a central umbilication. They usually occur on the face or scalp in middle aged or older individuals. Some nodules can grow to over a centimetre in diameter and appear as yellow, speckled, smooth surfaced papules or nodules. Sebaceous adenomas can be confused with sebaceous gland hyperplasia when they are small. They can resemble a BCC when larger. They are usually diagnosed histologically. It is extremely rare for sebaceous adenomas to transform into a malignancy (a **sebaceous carcinoma**); however, a suspicious SA needs to be excised and biopsied. Patients with multiple sebaceous adenoma may have the **Muir-Torre Syndrome**. This is a rare hereditary autosomal dominant cancer syndrome. People with this syndrome are prone to developing cancers of the colon, breast and genitourinary tract. They may also develop other sebaceous tumours, such as sebaceous epithelioma or sebaceous carcinoma and other skin lesions such as keratoacanthoma. Sebaceous naevi which are usually found in the scalp are not usually related to Muir-Torre syndrome.

Other rare lesions that can occur on the nose and cheeks include the typical red, scaly butterfly rash of **systemic lupus erythematosus**.

**Angiofibromas** are a common feature of tuberous sclerosis and usually appear as small, pink or red papules across the cheeks and nose in a butterfly distribution. They usually appear between the ages of 3 and 10 years old and increase in size and number as the child gets older. Other cutaneous features of tuberous sclerosis such as periungual fibromas, a shagreen patch in the lower lumbar area, multiple ash leaf macules and café au lait spots may help confirm the diagnosis. Epilepsy is present in about 70% of patients with tuberous sclerosis. Angiofibromas on the face are harmless but can be removed by shave biopsy and light cautery if required for cosmetic reasons.

**Milia** are common around the eyes, cheeks, nose and forehead in newborns but can also occur on the face in adults. They are made up of multiple tiny (1–2 mm) keratin filled papules at the base of a hair follicle or sweat gland. In neonates they usually resolve spontaneously. In adults if they are unsightly they can be removed by pricking with a needle and removing the content with a micro-curette. Some may clear with topical retinoids, a chemical peel or microdermabrasion.

**Syringomas** are similar to milia but are more common in adults and may be solitary. They are benign sweat gland tumours and are commonly found on the upper cheeks and lower eyelids especially in women. They are small (1–2 mm) flesh coloured, white or yellow papules and may be distributed symmetrically on the face. Treatment when required is similar to that for milia.

**Keratosis pilaris** is common on the sides of the cheeks, upper outer arms and outer thighs in children and young adults. It gives the skin a sandpaper feel as a result of multiple tiny keratin plugs in the affected areas. It usually responds to moisturising with a urea based moisturiser (e.g. "Eucerin®" or "Calmurid®") or applying a topical retinoid or retinoid like agent such as adapalene gel.

**Flat (plain) warts, molluscum contagiosum, melasma, telangiectasia and spider naevi** are all common on the cheeks. **Dermatosis papulosa nigra** appears as small pigmented papules on the cheeks in adult of African or Asian descent, especially women. It is considered a variant of seborrhoeic keratosis.

**Dermatomyositis** is a rare autoimmune condition that can present with a reddish-purple (heliotrope) rash mainly on the eyelids, face, upper chest and upper arms in adults. It can be associated with proximal muscle weakness which can predate the rash, present at the same time or occur after the rash has appeared. Some cases may have an underlying malignancy that should be screened for (see Chap. 51).

## 36.8 Rashes and Lesions on the Ear

**Atopic eczema, contact dermatitis, seborrhoeic dermatitis and psoriasis** can all occur on the ear. **Discoid lupus erythematosus (DLE)** is

often found on the external auditory canal and presents as a scaly rash.

**Chilblains (pernio)** can occur on the ears and cause itchy and/or tender, red or purple bumps as a reaction to cold. They are considered a local form of vasculitis. They are most common in children and in the elderly and are sometimes aggravated by sun exposure and heat. They can sometimes be associated with lupus erythematosus (Chilblain lupus) with lupus erythematosus, Raynaud's phenomenon and recently with Covid-19. Chilblains are difficult to treat but may respond to potent topical steroids. Keeping the ears warm should help. Severe cases may respond to vasodilators such as oral nifedipine.

**Juvenile Spring Eruption** causes an eczematous rash in the spring and summer, usually in boys and young men. It causes itchy, red lumps which evolve into blisters and crusts on the light exposed areas of the ears which heal with minimal or no scarring. It is considered a localised form of polymorphic light eruption. Symptoms can be relieved with moderately potent topical steroids and emollients. Hats and sun blocks should be used in the spring and early summer to protect the ears and letting the hair grow longer may also help. This is probably why girls are less commonly affected.

**Superficial basal cell carcinoma, actinic keratosis, Bowen's disease, keratoacanthoma, squamous cell carcinoma and melanoma** can all present as an isolated lesion on the ears that does not clear with emollients and topical steroids (Fig. 36.6). Any isolated lesion that persists for more than 3 months should be biopsied for histological diagnosis.



**Fig. 36.6** Keratoacanthoma R ear; grew rapidly over 2 weeks

### 36.9 Ulcers, Blisters and Nodules on the Ears

Chilblains and juvenile spring eruption can cause blisters on the ears and are detailed above.

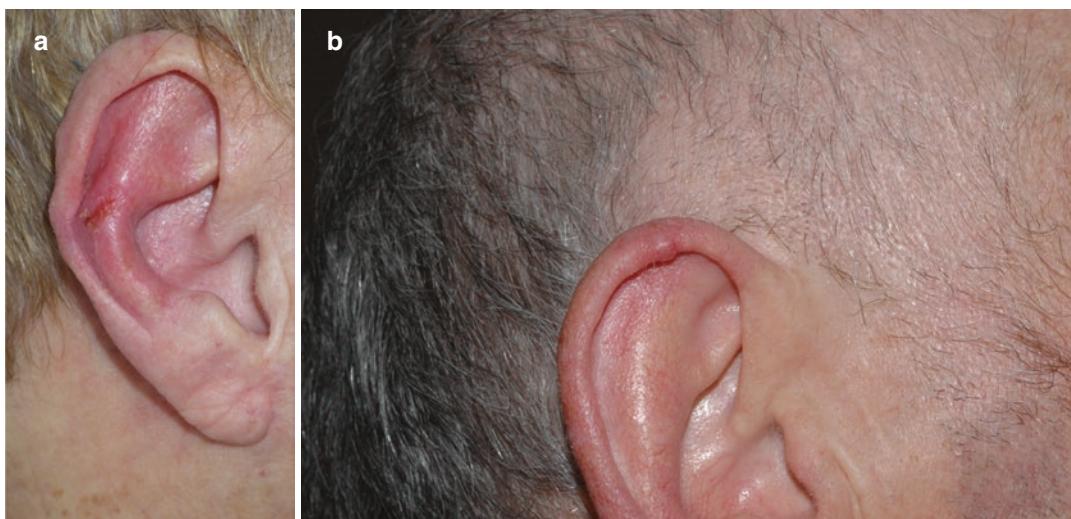
**Chondrodermatitis nodularis helicis (CNH)** usually presents as tender, painful lumps on the upper anterior helix of the ear in middle aged and elderly men and women. The lumps are usually 5–10 mm in diameter and are painful in bed at night when lying with the affected ear on the pillow (Fig. 36.7a, b). There may be a central, small discharging ulcer in the middle of the nodule. It can often be confused with skin cancer. If there are any doubts, a biopsy should be taken. Treatment is by avoiding lying on the affected side in bed at night. Some cases may respond to a potent topical steroid or intralesional steroid injection. More resistant cases may need to be treated with cryosurgery or surgical excision.

**Seborrhoeic keratosis, viral warts, molluscum, non-melanoma skin cancer and melanoma** can all appear as isolated nodular lesions on the ears. A biopsy may be required if the diagnosis is not obvious clinically. **Sebaceous cysts** sometimes occur on the earlobe and surgical excision is the only way to remove them.

### 36.10 Rashes and Lesions on the Eyelids and Inner Canthus

**Atopic eczema, contact allergic or irritant dermatitis, seborrhoeic dermatitis and psoriasis** can all affect the eyelids. Treatment is difficult as the delicate eyelid skin is very sensitive even to weak topical steroids. Tacrolimus (“Protopic®”) might be a safer choice for more severe resistant cases. Eczema and dermatitis are more common on the upper eyelid whereas periorificial dermatitis is more common on the lower eyelid.

**Periorificial dermatitis** (previously known as perioral dermatitis) usually occurs around the mouth but can sometimes occur around the lower lateral eyelids in conjunction with the typical perioral rash or sometimes in isolation around the eyelid with no peri-oral involvement (periocular dermatitis). This condition is aggravated by topical steroids which should be stopped. Treatment usually requires 1 or 2 months treatment with an oral anti-acne type medication, such as lymecycline to clear the rash. Milder cases may respond to a topical antibiotic such as erythromycin (e.g. “Zinerit®”), clindamycin (e.g. “Dalacin T®”) or metronidazole (e.g. “Rozax®”).



**Fig. 36.7** (a) Chondrodermatitis on the ear in a woman. (b) Chondrodermatitis nodularis helicis in a woman

**Blepharitis** is inflammation of the eyelid margin and may have many causes including staphylococcal infection, seborrhoeic dermatitis, rosacea and contact allergy to eye care products such as eyeliner, eye drops or contact lens solution. Products with neomycin may cause eyelid blepharitis. Flaking and crusting may occur where the eyelash exits the skin. The eyes themselves may become itchy or sore. Some cases of blepharitis can become infected and appear as a **stye (hordeolum)** which is usually due to staphylococcal aureus and should respond to chloramphenicol eye drops or ointments.

Treatment of blepharitis involves good eyelid hygiene with the use of warm compresses and washing the eyelid margin with a cotton wool swab, using a mixture of water and an anti-dandruff shampoo such as ketoconazole. Severe cases may require treatment with a weak topical steroid or oral antibiotics for a week. Great care must be taken to avoid getting steroid into the eye. Severe cases may need antibiotics or weak topical steroids for a week. If there is an eczematous reaction on the eyelids it may respond to topical tacrolimus ("Protopic®"). **Ocular rosacea** can cause blepharitis and can be the presenting feature of the disease in some cases. Milder cases may respond to good eyelid hygiene and topical antibiotics such as fusidic acid. Resistant cases may require topical ciclosporin drops, oral tetracycline or oral isotretinoin. In ocular rosacea and seborrhoeic dermatitis in this area, it is important washing the eyes daily with a soap free wash or dilute ketoconazole shampoo.

**Superficial BCC, actinic keratosis, Bowen's disease, squamous cell carcinoma and melanoma**

**noma** can all affect the eyelids and inner canthus so any isolated, scaly patch, ulcer or nodule that does not clear after 3 months may need to be biopsied (Fig. 36.8). Other benign nodular lesions on the eyelid margins could be due to **warts, molluscum or seborrhoeic keratosis**.

**Behcet's disease** (see below under female genital ulceration) is a very rare form of vasculitis that can cause uveitis associated with mouth and/or genital ulceration.

**Xanthelasma** is unique to the eyelids and is an accumulation of fat under the skin. The lesions are usually symmetrical and most commonly found on the medial aspect of the upper and/or lower eyelids (Fig. 36.9a, b).

The cause of xanthelasma is unknown but despite popular myths, it is not normally associated with underlying hyperlipidemia. Treatment can be difficult because of the delicate skin on the eyelid and the proximity to the eyeball. Most cases can be treated successfully by surgical excision, shave biopsy, cautery or cryosurgery. Topical trichloroacetic acid can also be helpful



**Fig. 36.8** Nodular BCC (rodent ulcer) lower eyelid



**Fig. 36.9** (a) Xanthelasma. (b) Mild xanthelasma

but great care must be taken to avoid getting this acid in the eye.

**Chalazion (meibomian cyst)** usually presents as a painless, firm, lump on the upper or lower eyelid, caused by inflammation of the oil glands of the eyelid. Unlike a stye, a chalazion is usually found in the substance of the eyelid rather than the eyelid margin. If it becomes painful and red, it may be a sign of bacterial infection requiring oral antibiotics, though this is uncommon. This should be treated with topical and oral antibiotics. Once the infection is cleared, a chalazion can be surgically removed by incision and drainage via the inside of the eyelid under local anaesthetic. A chalazion forceps is used to evert the eyelid and stabilise the chalazion as it is incised and drained from the inside. A drop of topical antibiotic such as fusidic acid eye ointment ("Fucidin eye ointment®") can be instilled into the eyelid margins and a firm eyepatch should be applied for 24 h after drainage of a chalazion.

Other conditions affecting the eyelids include cellulitis and angio-oedema. Herpes zoster may also present with unilateral vesicular periorbital eruption, which requires referral to ophthalmology if corneal involvement occurs.

**Hidrocystoma** (also known as cystadenoma, Moll gland cyst or sudoriferous cyst) present as a translucent jelly-like cyst arising on an eyelid (Fig. 36.10). A solitary translucent eyelid cyst is usually due to an apocrine hidrocystoma. Multiple cysts on the lower eyelid are usually due to eccrine hidrocystomas. Hidrocystomas may be confused with a BCC but the content of a hidrocystoma can usually be easily emptied by simply



**Fig. 36.10** Hidrocystoma of the eyelid

puncturing it with a needle. They are harmless and benign but may need to be removed for cosmetic or comfort reasons.

### 36.11 Nasal Lesion and Rashes

Certain rashes and lesions are particular to the nose. **Rhinophyma**, which is as a result of the swelling of the nose in patients with rosacea, is rare but disfiguring. The underlying rosacea should be treated with appropriate topical and/or oral therapies. The patient needs to protect their nose from ultraviolet light. Surgical treatment is sometimes necessary to debride and debulk the deformity on the nose.

**Lupus Pernio** is a form of cutaneous sarcoid that presents as a bluish red or violaceous nodules and plaques on the nose, cheeks or ears. It is more common in females from 45 to 65 years old and in those of African descent. It can be associated with systemic sarcoidosis which may show up on a chest x-ray.

The diagnosis of lupus pernio is confirmed by performing a skin biopsy and treatment usually involves potent topical steroids or immunosuppressants such as oral steroids.

Lupus pernio should not be confused with **lupus vulgaris** which is a rare form of cutaneous tuberculosis. This is caused by *Mycobacterium tuberculosis* which is the same organism that causes pulmonary TB. Cutaneous TB is very uncommon but may occasionally be seen in patients from India, China or Africa. Lupus vulgaris present as small, sharply defined, reddish-brown lesions with a gelatinous consistency (called apple jelly nodules), which can persist for months or years and can lead to disfigurement. Diagnosis is mainly from skin biopsy, skin culture, tuberculin skin testing (Mantoux test) and chest x-ray, which may show signs of pulmonary TB. Treatment of cutaneous TB is usually with a combination of anti-TB drugs given orally over several months.

**Filiform warts** often occur on the entrance to the nostrils, especially in children. These usually respond well to the cryosurgery under topical anaesthesia.

Actinic keratosis, Bowen's disease, NMSC and melanoma may also present on the nose as with other parts of the face.

### 36.12 Rashes and Lesions on the Lips and in the Mouth

**Contact allergic and irritant dermatitis** can occur on the lips. There can be many causes including lipstick, toothpaste (Fig. 36.11), soaps and other irritants. **Lip licking** is common in children and can cause an irritant dermatitis which causes discomfort and further licking. Nickel allergy may also present as **cheilitis**. Patients with atopic eczema can have lip involvement. Treatment is usually with lip emollients, avoiding soaps, other irritants and allergens and the use of a weak topical steroid such as 1% hydrocortisone ointment. SLS (sodium lauryl sulfate) free toothpastes such as "Sensodyne Pronamel Gentle Whitening Toothpaste®" may also help.

**Periorificial dermatitis** (POD) can occur near the lips but, by definition, does not extend up to the vermillion border of the lip. It usually presents as a red rash made up of multiple micropapules above or below the lips or sometimes all the way around the mouth. Occasionally the rash can occur under the lower lateral eyelids either in isolation or in conjunction with the perioral rash. It is almost always found in young females and is usually aggravated by topical steroids. It can be differentiated from irritant dermatitis such as lip

licking dermatitis, by a border of clear skin between the rash and the vermillion border which is seen in perioral dermatitis and not irritant dermatitis. Treatment of POD is by withdrawing topical steroids and giving 1 or 2 months of an oral anti-acne medication such as lymecycline. There may be a flare of the rash in the first week of stopping the steroid. Reducing the potency or frequency of application of the topical steroid gradually over 2 weeks may sometimes be appropriate but this may result in it taking longer for the POD to clear.

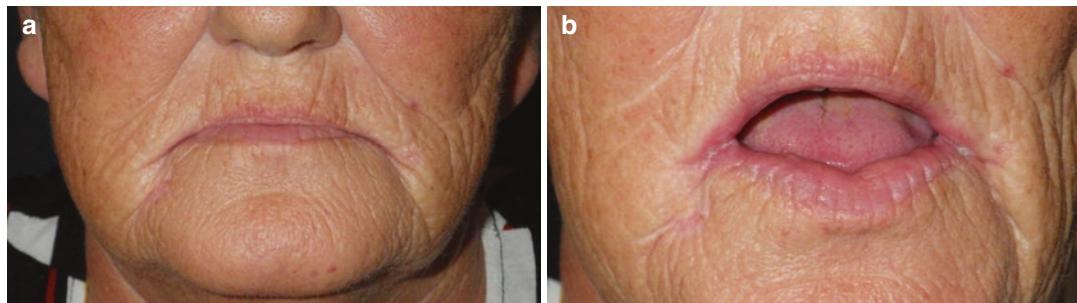
Milder cases may respond to a topical antibiotic such as erythromycin (e.g. "Zinerit®"), clindamycin (e.g. "Dalacin T®") or metronidazole (e.g. "Rozax®").

**Actinic cheilitis** usually affects the lower lip and causes a linear scaly rash along the vermillion border of the lip. It mostly occurs in those who spend a lot of time outdoors through work or hobbies. Treatment is usually with cryosurgery, topical actinic keratosis treatments such as 5-fluorouracil, imiquimod or photodynamic therapy. A biopsy may be necessary before treatment to rule out an underlying malignancy, particularly if there is a nodule or ulcer on the damaged lip. Sun protection is vital with a high SPF lip cream or ointment to prevent progression to skin cancer. Psoriasis rarely affects the lips.

**Angular stomatitis** is a common inflammatory condition affecting the corners of the mouth. It is often due to a number of factors such as drooling from the angle of the mouth causing a contact irritant dermatitis and with an associated mixed bacterial or yeast infection. Angular stomatitis is more common in the elderly, especially if they wear poorly fitting dentures (Fig. 36.12a, b). It can also occur in children who get oral thrush or in patients who are generally unwell with systemic problems, to include poor nutrition, iron or B12 deficiency, or those who are immunosuppressed such as patients with diabetes or on systemic steroids or oral isotretinoin. The patient usually presents with painful cracks on the corner of the mouth with oozing and crusting. Treatment involves removing any irritants and moisturising liberally with a safe, greasy lip moisturiser. Topical anti-infective agents such as



**Fig. 36.11** Cheilitis of lips possibly from sodium lauryl sulfate (SLS) in toothpaste



**Fig. 36.12** (a) Angular stomatitis in a 72-year-old female. (b) Angular stomatitis in the same female

imidazole anti-fungal, combined with a weak topical steroid such as 1% hydrocortisone ointment, applied once or twice daily for 1–2 weeks, should help to reduce inflammation and clear any infection. Frequent moisturising with a safe greasy moisturiser should be recommended. “Sensodyne®” toothpaste which is SLS free may also help. More resistant cases may respond to ketoconazole cream with or without 1% hydrocortisone ointment or tacrolimus ointment (“Protopic®”).

Certain medication such as oral retinoids used for acne or psoriasis can often cause severe dryness of the lips with cheilitis and angular stomatitis. Symptoms can be relieved by reducing the dose of the retinoid and with topical treatments as outlined above.

**Oral leukoplakia** is a pre-malignant condition that can present on the lips, tongue or buccal mucosa, (inside of the cheeks) as a white, macular plaque which is more common in the elderly, smokers and those with excessive alcohol intake. Diagnosis is usually by skin biopsy and treatment is usually either by surgical excision or cryosurgery. Patients should be followed up throughout life, as a small percentage may progress and develop into a squamous cell carcinoma.

**Oral lichen planus** can affect the lips and buccal mucosa. It is considered a T cell-mediated autoimmune disease of unknown cause. About 50% of people with cutaneous lichen planus will have oral lichen planus also. It is more common in adults and in women. It may be asymptomatic or can cause pain, discomfort, nodules or ulcers. It can present with a symmetrical, white, lace like pattern on the buccal mucosa (inside of the

cheeks). **Erosive lichen planus** (ELP) can cause red, tender erosions mainly in-between the gums and lips. Diagnosis is often clinical which may be obvious from cutaneous signs elsewhere. More unusual cases may need a biopsy of the lips or buccal mucosa to diagnose ELP. ELP has slightly increased risk of **oral cancer**. Treatment involves removing any underlying causes such as drugs (e.g. gold, some antibiotics, NSAID’s, antihypertensives, cholesterol lowering drugs and heart disease medication). Patch testing may be required to rule out contact allergy. Smokers should be encouraged to stop. Good oral hygiene by brushing the teeth, flossing regularly and using dilute chlorhexidine mouth wash may help. Toothpaste should be free from sodium lauryl sulphate (e.g. “Sensodyne®” toothpaste). Some cases of oral lichen planus can be very painful with ulceration leading to scarring. This can make eating difficult. Topical steroids in an oral paste, gel or spray can often help. Tacrolimus (“Protopic®”) can also help. Severe cases may need referral to tertiary care for systemic treatment such as steroids, retinoids, cyclophosphamide or dapsone (see Chap. 19).

**Orofacial granuloma** is a rare disease of unknown origin that causes swelling of the lips and the surrounding skin. It is usually asymptomatic but can become quite unsightly. It is often idiopathic (orofacial granulomatosis) but can be associated with other granulomatous disorders such as Crohn’s disease or sarcoidosis (see Chap. 51). Biopsy shows a noncaseating granulomatous reaction. Swelling from the granulomatous infiltration may cause lymphatic blockage resulting in a rubbery swelling of the upper or lower lips and



**Fig. 36.13** Orofacial granulomatosis. Histology showed granulomatous inflammation

surrounding skin. It can look like a bad cosmetic lip fillers injection (Fig. 36.13). Investigations should include routine bloods, a chest X-ray to rule out sarcoidosis or TB and endoscopy if there are any bowel symptoms. Tests for underlying TB may also be necessary. Treatment is difficult but milder cases may respond to topical, intralesional or oral steroids, topical tacrolimus or oral tetracyclines. More resistant cases will need to be referred to a dermatologist for specialist investigations and treatment.

### 36.13 Ulcers and Nodules on the Lips and in the Mouth

The most common ulcerating lesion on the lip is **herpes simplex**. The diagnosis is usually obvious from the history of recurrent blistering eruption which dries into a crust and heals within 2 weeks, leaving no scars. The blisters erupt in exactly the same place during each attack. Treatment is with topical acyclovir started at the earliest possible stage in an attack. More severe cases may need systemic anti-viral such as valaciclovir 500 mg twice daily for up to 10 days, and for recurrent episodes 500 mg twice daily for 3–5 days in adults. Valaciclovir 2000 mg twice a day for 1 day in adults is convenient and effective if started as early as possible in an attack. For severe, recurrent cases, suppressive therapy may be required such as valaciclovir 500 mg, once daily for 6–12 months. Sun protection of the lips may also help (see Chap. 32).

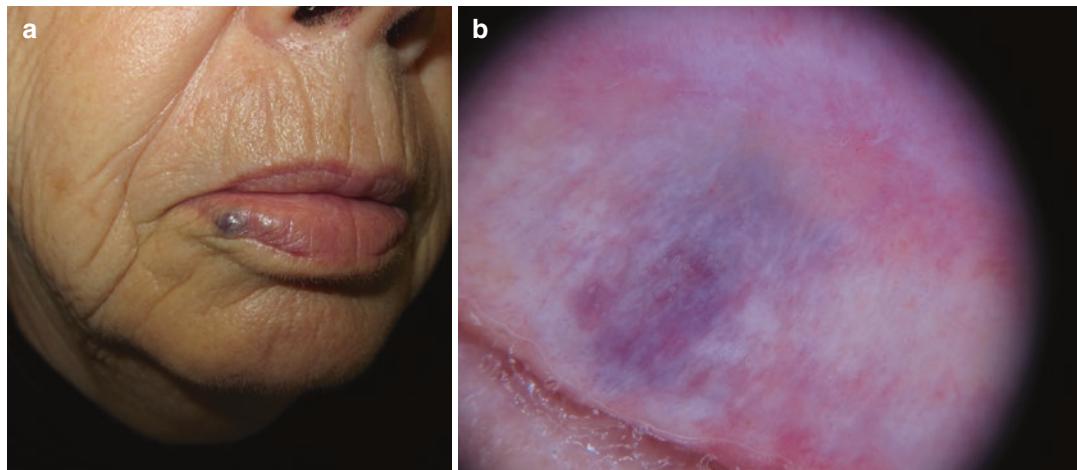


**Fig. 36.14** Aphthous ulcers. Photo courtesy of Dr Myriam Raquel González Oviedo

**Friction** from poorly fitting dentures or dental caries can also cause mechanical ulcers or blisters on the lips or in the mouth.

**Aphthous ulcers** can occur inside the lips or on the buccal mucosa inside the cheeks or under the tongue. Although not serious, they can be very painful and debilitating. They are very common and can occur in up to 20% of the population. They usually begin as a round, yellowish macule surrounded by a red halo. The yellow macule breaks down into a punched out ulcer which is covered by a loosely attached white, yellowish or greyish membrane. They may be singular or multiple. They can be recurrent. Most are between 3 and 10 mm in size and heal spontaneously within 1–2 weeks (Fig. 36.14). They are more common in childhood, young adults and in females. The exact cause is unclear but traumatic and viral causes have been suspected. Patients with recurrent aphthous ulcers should have routine blood tests to include B12, folate, ferritin, TFT's and a diabetic screen. They should also be checked for coeliac disease, herpes simplex, Crohn's disease, Behcet's disease and HIV. Treatment is usually symptomatic with an oral anaesthetic gel and systemic analgesics if required.

Larger, more painful, persistent ulcers may need treatment with a topical steroid such as triamcinolone in an oral paste, tacrolimus ointment or cauterization with a silver nitrate stick. Severe recurrent aphthous ulceration may respond to oral colchicine (0.5 mg TID) or oral thalidomide (from 50 mg up to 300 mg/day but this is extremely



**Fig. 36.15** (a) Venous lake on lower lip. Photo courtesy of Dr Paola Pasquali. (b) Dermoscopy of the same venous lake on lower lip. Photo courtesy of Dr Paola Pasquali

teratogenic and rarely used nowadays). Ulcers that fail to heal should be biopsied to rule out an underlying malignancy.

Rare causes of lip and mouth ulcers include **pemphigus vulgaris**, **bullous pemphigoid** and the primary chancre of **syphilis**. **Squamous cell carcinoma** can present as a nodular, ulcerating, lip lesion and any suspicious lesion on the lip not fading after 6–12 weeks, should be biopsied, to rule out an underlying malignancy.

The causes of nodular lesions on the lip include **molluscum contagiosum** and a mucocele of the lip. **Mucoceles** usually present as a soft, round, fluid filled, pale pink lump, most common on the inside of the lower lip, usually between 5 and 15 mm in diameter. They are caused by trauma to the duct of a small salivary gland leading to extravasation of mucous. They are not a true cyst as they are not lined by epithelium. They are usually painless but troublesome as they can be unsightly, irritating and can make eating difficult when large. Some mucoceles resolve spontaneously but large or more persistent ones should be removed surgically or can be destroyed using cryosurgery.

**Viral warts** are common on the lips in children but usually resolve spontaneously. Warts inside the mouth in adults are unusual and may be sexually transmitted.

The most common vascular lesion on the lip is a **venous lakes**. They are normally bluish-black

or slate gray in colour, but may also be red or purple (Fig. 36.15a, b). They can usually be diagnosed clinically with a dermatoscope but a biopsy should be taken if there is any doubt about the diagnosis as a melanoma may present in a similar fashion. Venous lakes are harmless varicose veins and can be removed surgically with a punch biopsy, cryosurgery or with a pulsed dye laser if required.

### 36.14 Neck Rashes and Lesions

Rashes which are particular to the neck include **Poikiloderma of Civatte**. This presents as discolouration and textural changes on the side of the neck but sparing the shaded area under the chin, which is usually seen in fair skinned women in their 40s and 50s. It presents as an asymptomatic staining of the skin with mixtures of brown or red as a result of hyperpigmentation and telangiectasia. The texture of the skin is also altered with multiple tiny papules as a result of prominent hair follicles giving a roughened “plucked chicken skin” appearance (Fig. 36.16). There is no scaling or itch. The cause is not clear but hormonal factors and ultraviolet light are probably involved. Contact dermatitis from spraying perfume on the neck may also aggravate this condition. Poikiloderma of Civatte is completely harmless and only a cosmetic problem. Treatment is difficult but avoidance of UVL and



**Fig. 36.16** Poikiloderma of Civatte in a 49-year-old female

irritants such as perfumes may help. Hydroquinone may help reduce hyperpigmentation while topical retinoids such as tretinoin could restore atrophic skin. Intense pulsed light treatment has been used to treat the hyperpigmentation and pulsed dye laser for the telangiectasia. Cosmetic camouflage and appropriate clothing such as high collars help hide the appearance of the rash.

**Contact allergic dermatitis** is also relatively common around the neck. This may be as a result of clothing or jewellery in contact with the affected area. The most common allergen is nickel. Patients may also develop contact allergic dermatitis on their neck from nail care products such as nail varnish, nail varnish removers, artificial nails or the adhesive used to hold them on. Diagnosis can be confirmed by skin patch allergy testing.

**Lichen simplex** or **neurodermatitis**, can also present on the neck, with asymmetrical lichenified or thickened skin due to chronic scratching and is more common in people with anxiety and/or obsessive compulsive disorder. It can also affect the genitals, wrists, forearms and lower legs and is detailed further below.

**Skin tags (acrochordon)** are very common around the neck in patients who are overweight and in darker skin phototypes. They are harmless and benign. They can be removed by cutting or cauterity under local anaesthetic for cosmetic reasons, if required. Cryotweezers are useful for multiple small tags especially in patients with blood born infectious where cutting and blood exposure should be avoided. Skin tags can sometimes be confused with seborrhoeic keratosis, viral warts or molluscum contagiosum. If



**Fig. 36.17** Folliculitis keloidalis present for 10 year in a 42-year-old male

there are any doubts, some or all of the removed specimen should be sent for histology.

**Folliculitis keloidalis** (also called acne keloidalis nuchae or acne keloidalis) is a rare form of folliculitis. It can cause keloid scarring and alopecia of the nape of the neck. This condition is more common in men and in patients of African descent. It can be associated with ingrown hairs, pressure from shirts or straps, obesity and metabolic syndrome in some patients (Fig. 36.17). Treatment can be difficult but it may respond to long courses of oral acne treatments (e.g. lymecycline, doxycycline or trimethoprim) or clindamycin and rifampicin for 3 months. It may respond to oral isotretinoin sparing the need to use long term oral antibiotics. Keloids may respond to intralesional cryosurgery or intralesional steroid injections. Fortunately the hair can usually be grown to hide the scars.

**Acanthosis nigricans** appears as thickened, darkened, velvety skin in the flexures such as around the neck and in the axilla. Skin tags are commonly found in and around the plaques (Fig. 36.18). It mostly occurs in adults and can be associated with obesity, diabetes, and occasionally can be a paraneoplastic manifestation. It is more common in patients with dark skin types. Sudden onset of this condition may occur as a result of an underlying malignancy, especially GI. Treatment, if requested for cosmetic reasons, includes hydroxy acid, urea, salicylic acid or retinoid creams. More severe cases can be helped by shave biopsy, dermabrasion or laser therapy.

All types of **skin cancers** are more common in the neck as it is an exposed site. Any isolated



**Fig. 36.18** Acanthosis nigricans in a 24-year-old obese man with a high sugar intake but no diagnosis of diabetes

new or changing lesion should be viewed with suspicion and biopsied if there is any possibility of a skin cancer.

### 36.15 Rashes and Lesions on the Trunk



All common dermatoses such as acne, eczema/dermatitis, psoriasis and seborrhoeic dermatitis can occur on the chest, abdomen and back. Rashes that are usually confined to the trunk include **pityriasis rosea** and **pityriasis versicolor**.

All forms of **skin tumours**, both benign and malignant, can be found on the trunk. The most common benign growths include **seborrhoeic keratoses** which are most commonly found on the trunk. **Cherry angiomas** (also known as Campbell de Morgan spots) are very common on the trunk. They are benign haemangiomas that are common in adults over the age of 40. They can be bright red, purple or blue. The cause is unknown. They are completely benign and are best treated with a pulsed dye laser, if required, for cosmetic purposes.

**Keloids** can develop spontaneously on the trunk areas, particularly in patients of African descent or they can arise from scars or acne.

The **nipples and areola** can be involved in various skin problems including atopic eczema, contact dermatitis, irritant dermatitis and friction (jogger's nipples). Psoriasis rarely affects the nipple. Women with scabies often develop an eczematous rash on the nipples from a generalized allergic dermatitis to the mite or eggs elsewhere on the body. Eczematous rashes of the nipples usually respond to a week topical steroid such as 1% hydrocortisone ointment. Potent steroids should be avoided on the sensitive skin on the nipple and areola. Tacrolimus ("Protopic®") could be used in more severe eczematous rashes in this area.

Paget's disease of the breast can present as a unilateral rash around the nipple and may be an indication of an underlying ductal carcinoma in situ or invasive breast cancer. Any unilateral nipple rash not responding to simple treatments should be biopsied. Skin tumours such as skin tags, fibro-epithelial polyps, seborrhoeic keratosis, moles and warts can affect the nipples. BCC, SCC and melanoma have been reported on the nipple-areolar complex but are extremely rare.

Sore and/or cracked nipples are common in breast feeding women. It is often as a result of the baby not latching on properly and the mother may need the assistance of a lactation consultant. Resting the area by expressing the milk for a few days allowing the skin to heal may help. Other

simple measures include moisturisers, avoiding soaps on the area and nipple shields. If there are any signs of infection it is usually due to *Staphylococcus aureus* and should respond to topical or oral antibiotics (e.g. flucloxacillin).

### 36.16 Flexural Rashes

Rashes confined to the flexures (e.g. axillae, groin, sub-mammary, lower abdomen under the fat pads) can have many causes. In an overweight patient, **intertrigo** is one of the more common causes. It is usually caused as a result of friction, sweating and low grade mixed bacterial and yeast infection. Weight loss and good skin hygiene is probably the best way to clear the rash. Symptoms can be relieved by avoiding soaps and other irritants and lubrication with a suitable moisturiser such as one containing zinc and castor oil. A weak topical steroid combined with a broad spectrum anti-effective agent such as miconazole ("Daktacort<sup>®</sup>") or clotrimazole ("Canesten HC<sup>®</sup>") once at night for a few weeks may help. More resistant cases may respond to ketoconazole cream with or without 1% Hydrocortisone ointment or tacrolimus ointment ("Protopic<sup>®</sup>").

Excessive sweating may be a contributing factor (see Chap. 11). It helps to wear a cotton bra or placing a cotton handkerchief between the skin and synthetic fabric underwear.

Tinea infection can often affect the groin creases (**tinea cruris**). The rash is usually red with an ill-defined edge and sometimes, satellite lesions spreading beyond the main area of the rash are found. Tinea cruris often coexists with tinea pedis (athlete's foot) so it is worth checking the feet. The diagnosis can usually be made clinically but if there is any doubt, skin scrapings should be taken for fungal stain and culture. Most cases respond to topical antifungal treatment but resistant cases may require systemic antifungal treatment such as terbinafine or itraconazole tablets for at least 2 weeks.

**Flexural psoriasis** is usually red and non-scaly with sharply demarcated border between the involved and uninvolved skin. Treatment is usually with a weak or moderately potent topical steroid. Resistant cases may respond to tacrolimus ("Protopic<sup>®</sup>"). Potent topical steroids should be

avoided in the flexures, as the occlusive effects of the skin folds increase the absorption of the steroid and the risks of skin atrophy, striae and systemic effects.

**Erythrasma** can cause of a flexural rash, particularly in the groin, intergluteal folds, perianal area and axillae. It causes a characteristic reddish-brown, slightly scaly, rash with a sharp border of irregular contour. It may be itchy. Diagnosis can be confirmed by Wood's lamp exam which fluoresces a characteristic coral red color or skin scrapings which show a bacteria called corynebacterium. This is usually treated with a topical antibiotic such as fusidic acid (e.g. "Fucidin cream<sup>®</sup>") or an oral antibiotic such as erythromycin or tetracycline.

**Lichen sclerosus et atrophicus** may affect the groin or the perianal area causing shiny, ivory white, smooth surfaced plaques or papules which may cause a symmetrical atrophic itchy rash. Diagnosis is confirmed by biopsy and most cases will respond to a potent topical steroid. This condition may be premalignant so careful follow-up is advised.

**Impetigo, folliculitis and boils** are not uncommon in flexural skin particularly where there is hair such as under the arms or in the groin. Patients with these problems should be screened for diabetes.

If the patient develops multiple, deep, painful boils, nodules and abscesses with sinuses and scars in the axillae, groin, under the breasts or buttocks that persist for more than 6 months, **hidradenitis suppurativa** should be considered (see Chap. 11). This is a rare but debilitating condition that usually occurs after puberty and is more common in women and in smokers. It may be familial. The condition can persist for years and cause pain, discharge and odour which can be very distressing for the patient. Some cases may respond to prolonged courses of oral acne medication such as tetracycline, trimethoprim or the oral contraceptive pill (e.g. "Dianette<sup>®</sup>"). Topical clindamycin may also help. Severe persistent cases may respond to a 12 week course of clindamycin 300 mg BD and rifampicin 300 mg BD. This turns the tears and urine red. This combination can occasionally cause pseudo-membranous colitis and so all patients should be warned to report to the doctor if they develop diarrhoea while on this treatment.

Severe cases may require oral retinoids, dapsone or biologics such as infliximab, adalimumab or anakinra. Intralesional steroids may help. Some patients may benefit from surgical interventions such as de-roofing or excision and grafting of the affected areas (see Chap. 11).

**Atopic eczema, contact irritant dermatitis, contact allergic dermatitis and seborrhoeic dermatitis** can all occur in the flexures but the diagnoses should be apparent from the history and a general physical examination. Skin cancer is very rare in the flexures as these areas are relatively photo-protected. **Seborrhoeic keratosis** can occur in the flexures, especially under the breasts.

**Acanthosis nigricans** is a rare flexural rash that causes darkened thickened velvety plaques in the axilla, groin and around the neck (as discussed above in section on neck rashes).

### 36.17 Hand Rashes and Lesions



**Hand dermatitis** is one of the most common presentations in general practice. Patients usually present with dry, scaly, itchy hands. The first question to always ask is about their occupation and hobbies. People working in wet jobs (hairdressers, nurses, dairy farmers, plasterers) may develop **contact irritant dermatitis** as a result of constant wetting of the skin and the overuse of soaps and detergents. Treatment of hand dermatitis involves avoiding all soaps and other irritants and the careful use of gloves. Patients should be advised to use a soap substitute for hand washing, showering and shampooing. They should be advised to moisturise their hands hourly initially for the first few weeks with the greasiest moisturiser they can tolerate. They may need a lighter moisturiser during the day while at work and a greasy moisturiser to use at home in the evening and at night before going to bed (see Chaps. 62 and 66). A potent topical steroid may be required for the badly affected areas at night before going to bed but this will never work alone. Topical steroids should always be combined with good hand care, moisturising and avoidance of soaps and other irritants.

Resistant cases of hand dermatitis may require a skin patch allergy test to rule out co-existing underlying **contact allergic dermatitis** to products such as chromate (in cement and leather) or nickel (see Chap. 21). Underlying tinea pedis (athlete's foot) can sometimes cause an allergic “**id**” reaction resulting in hand dermatitis. All patients with hand dermatitis should have their feet examined. If tinea pedis is found and treated it may help to clear the hand rash.

**Pompholyx** is a severe form of blistering eczema of the palms and the palmar aspects of the fingers and can sometimes affect the feet. As the skin in these areas is thick, the blisters do not burst easily and may appear as itchy papules under the skin. Treatment is with potent or super potent topical steroids and good hand care as outlined above. Severe cases of hand dermatitis may need to be referred to a consultant dermatologist for phototherapy or systemic treatment such as oral steroids, azathioprine, methotrexate, ciclosporin or oral retinoids such as alitretinoin (“Toctino®”).

Weeping, crusting and pain usually indicates a **secondary bacterial infection** which should respond to a suitable systemic antibiotic such as flucloxacillin and soaking the hands in a 1:10,000 solution of potassium permanganate.

Fungal infection of the hand is rare (**tinea manuum**). It usually presents as a unilateral scaly rash which emphasises the skin creases in people involved in wet work. Diagnosis can be confirmed by sending skin scrapings for fungal stain and culture. Treatment is usually with a topical antifungal such as terbinafine cream and good hand care. More severe resistant cases may require oral terbinafine for 2 weeks.

**Erosio interdigitalis blastomycetica** is an unusual Candida infection of the inter-digital space on the hand in those involved in wet work. This can be treated with an imidazole antifungal cream and hand care advice.

**Tylosis** (hereditary focal or generalised palmaroplantar keratoderma) is an uncommon autosomal dominant condition that presents with thick, scaly plaques on the palms and soles usually over pressure points in children and adults. It may be associated with oesophageal cancer. Treatment is by paring, emollients, keratolytics (e.g. salicylic acid), vitamin D analogues (calcipotriol) or topical or systemic retinoids.

**Scabies burrows** are usually found in the interdigital web spaces, on the wrists and around the ankles.

Secondary **syphilis** can cause a reddish-brown blotchy rash on the palms of the hands (Fig. 36.19) and soles of the feet and is usually associated with a generalised non-itchy rash on

the body, which could be confused with guttate psoriasis, pityriasis rosea or a drug eruption. Always test for syphilis in a patient with a reddish rash on the palms. Another common condition for a rash in this location is erythema multiforme minor.

**Granuloma annulare** often presents with an annular rash with a raised nodular edge on the dorsum of the hands. Diagnosis is clinical and confirmed by biopsy. The main histologic feature is the presence of palisading granulomas composed of necrobiotic (brightly eosinophilic) collagen and abundant mucin surrounded by histiocytes, fibroblasts, and lymphocytes (see Chap. 51 for other causes of granulomas on histology). Treatment is usually with a potent topical steroid and some cases may respond to topical tacrolimus. Granuloma annulare can occasionally be associated with diabetes so all patients should have their blood sugar checked. It can sometimes become more generalized.

**Lichen planus** often appears on the dorsum of the hands and on the anterior wrists with typical reddish purple, small, scaly plaques with a lace curtain-like network through the rash (see Chap. 19).

**Porphyria cutanea tarda** is a rare condition which causes erosions and blisters on the dorsum of hands. This is due to excessive porphyrins which cause photosensitivity. This can be hereditary or acquired. The acquired forms can be linked with excessive alcohol consumption, oestrogen medication or haemochromatosis (see Chap. 51).

**Hand warts** are normally easy to recognise. However, an isolated warty growth on the dorsum of the hand or fingers and in patients over the age of 50 may be a non-melanoma skin cancer and should be biopsied.

**Actinic keratosis, Bowen's disease and squamous cell carcinoma** can all occur on the dorsum of the hands because of chronic sun exposure. BCCs are rare on the hands. **Solar lentigo and seborrhoeic keratosis** can also occur on the dorsum of the hands. **Melanoma** (pigmented or non-pigmented) may occur on the hands or around or under the nails. **Ganglions** usually present adjacent to joints on the fingers or the wrists (Fig. 36.20). They are usually firm but



**Fig. 36.19** Secondary syphilis. Photo courtesy of Dr Myriam Raquel González-Oviedo



**Fig. 36.20** Ganglion over the distal interphalangeal joint



**Fig. 36.21** Chilblains in a 19-year-old female

slightly fluctuant and puncturing or aspirating the nodule will reveal a colourless, jelly material which will confirm the diagnosis and may be curative.

**Chilblains** (sometimes called pernio) are small, itchy, painful, red swellings that most commonly occur on the fingers or toes but can occur in other areas such as the nose or ears (Fig. 36.21). Chilblains are caused by an abnormal skin reaction to cold. Most chilblains resolve after a few days without treatment. Keeping the hands and feet warm will help prevent chilblains.

**Raynaud's** (also known as Raynaud's syndrome or Raynaud's phenomenon) often presents on the fingers. It is a disorder of the small blood



**Fig. 36.22** Raynauds and chilblains in a 20-year-old female

vessels which go into spasm as result of various triggers such as cold or stress. The fingers (or less commonly the toes) initially go white and as they warm up they go blue and finally red as the blood flow returns (Fig. 36.22). It can be associated with pain, numbness or tingling. It may be a primary disease or rarely can it be linked with underlying auto-immune conditions such as lupus or rheumatoid arthritis. It is important to advise the patient to keep their hands warm by using ski gloves or hand warmers. Potent topical steroids may help painful chilblains. Peripheral vasodilators such as oral nifedipine may be required for more severe cases of Raynaud's especially in the winter.

**Psoriasis** of the hands usually presents as either typical scaly plaques on the dorsum of the hands and fingers or as palmopustular psoriasis on the palms (also known as palmoplantar pustulosis). It may present as an erythematous, scaly, fissuring rash, indistinguishable from hand eczema.

Treatment involves the usual hand care advice. Stable plaque psoriasis on the dorsum of the hands can be treated in the same way as plaque psoriasis on the body. Palmopustular psoriasis (palmoplantar pustulosis) can be difficult to treat and this is one of the few places where a potent topical steroid can be useful for psoriasis. Tar ointments are also helpful. This rash can be associated with smoking and smokers should be strongly encouraged to stop as it

may help the rash. More resistant cases may require ultraviolet light therapy or systemic treatment with etretinate ("Tigason®") or acitretin ("Neotigason®") which are oral retinoids (vitamin A derivatives) or with methotrexate (see Chap. 15).

**Hyperhidrosis** of the palms can be helped with aluminium chloride 20% ("Anhydrol Forte®" or "Driclor®"), iontophoresis or Botulinum toxin ("Botox®") injections (see Chap. 12).

**Digital mucous cysts** most commonly occur distal to the distal interphalangeal (DIP) joint of the fingers. They can also occur on the toes. They can be considered a form of ganglion with a tract extending from the DIP joint to the cyst. There may be a groove on the adjacent nail from pressure on the nail plate (Fig. 36.23). They are harmless and usually asymptomatic but patients sometimes want treatment for cosmetic or comfort reasons.

In a study of about 100 patients, surgery yielded the highest cure rate for digital mucous cysts (95%) compared with sclerotherapy (77%), cryotherapy (72%), corticosteroid injection (61%), and expression of cyst contents by repeated puncturing with a green needle under sterile conditions (39%) ( $P < 0.001$ ) [1].

Acute and chronic paronychia are common and are discussed elsewhere (Chap. 41).



**Fig. 36.23** Digital myxoid cyst with nail groove and possible tinea unguium

### 36.18 Rashes and Lesions on the Feet



Many of the rashes and lesions that occur on the feet are similar in their presentation and treatment to those that occur on the hands (e.g. **contact allergic dermatitis, pompholyx, psoriasis, warts and ganglions**). Other conditions such as fungal infections (**tinea pedis**) and **corns** are far more common on the feet while skin cancer (especially BCC) is less common on the feet. The feet are relatively photo-protected but **squamous cell carcinoma** and **melanoma** can occur on the soles or dorsum of the feet, on the toes and under

the nails. Diabetes can lead to various foot problems including a persistent **tinea pedis**, **viral warts** and **foot ulcers**.

**Tinea pedis** is the most common scaly rash on the feet. It is uncommon before puberty and usually presents as an itchy, moist, white, fissuring rash between the outer toe web spaces and the plantar aspects of the toe. Treatment involves using an imidazole anti-fungal cream and general foot care advice (Table 36.1). Resistant cases may require soaking of the feet in a 1% (1 in 100) solution of potassium permanganate (see Chap. 31). If required, the diagnosis can be confirmed by sending skin scrapings for fungal stain and culture.

Oral antifungal treatment may be indicated for more widespread tinea infection of the feet. Oral terbinafine is effective for a dermatophyte infection and oral itraconazole is more effective if candida species are isolated.

**Eczema/dermatitis** of the feet is less common than on the hands. A contact allergy to leather or rubber or other chemicals in footwear can be confirmed by patch testing. Management is similar to that of hand eczema with the usual foot care advice.

**Juvenile plantar dermatosis** is usually seen in school children and presents as a symmetrical, glazed, eczematous, fissuring rash on the soles of the feet (Fig. 36.24). It mainly affects the forefoot, sparing the instep. It is not a contact dermatitis but may well be related to the occlusive effects of rubber soled shoes. Treatment involves general foot care advice together with the use of a suitable moisturiser and a moderately potent topical steroid. The majority of cases will clear spontaneously in the teenage years.

**Table 36.1** General foot care advice

Never walk barefoot
Dry carefully between the toes after showering
Wear leather soled sandals in the summer
Wear leather soled shoes in the winter with cotton socks
Avoid soaps and bubble baths



**Fig. 36.24** Juvenile plantar dermatosis. Photo courtesy of Dr Myriam Raquel González Oviedo

**Psoriasis, palmoplantar pustulosis** and **pompholyx** of the feet should be managed as they would be in the hands (see above). **Hyperhidrosis** (excessive sweating) can cause soggy feet. Treatment involves advice on general foot care (Table 36.1). Aluminium Chloride 20% ("Anhydrol Forte®" or "Driclor®") can be very helpful as an antiperspirant. Iontophoresis can be useful in resistant cases (Chap. 12).

**Pitted keratolysis** presents with clusters of punched out pits, mainly affecting the forefoot or the heel in patients with sweaty feet (Fig. 36.25). There is usually a strong odour as a result of several bacterial species including corynebacterium infecting the skin. The pitting is due to destruction of the horny cells (stratum corneum)



**Fig. 36.25** Pitted keratolysis in a 21 year old male

by proteases that are produced by the bacteria. The bad odour is due to sulphur compounds produced by the bacteria. Treatment is with topical antibiotics such as fusidic acid cream and keeping the feet dry. Severe resistant cases may need oral erythromycin for 1 or 2 weeks.

A thickened, scaly callus with fissuring is a common problem on the heel of the feet. There is often no obvious underlying disease or inflammation but it can be sore and uncomfortable. The best management is by paring back the dead skin with a blade or a pumice stone. Heel balms such as "Flexitol Heel Balm®" (25% urea) applied twice a day for a few weeks may also help. Using it under occlusion at night with cling film may enhance the effect. More severe fissuring may need the assistance of a podiatrist for paring or the fitting of heel pads. Painful fissures can be sealed with tissue glue.

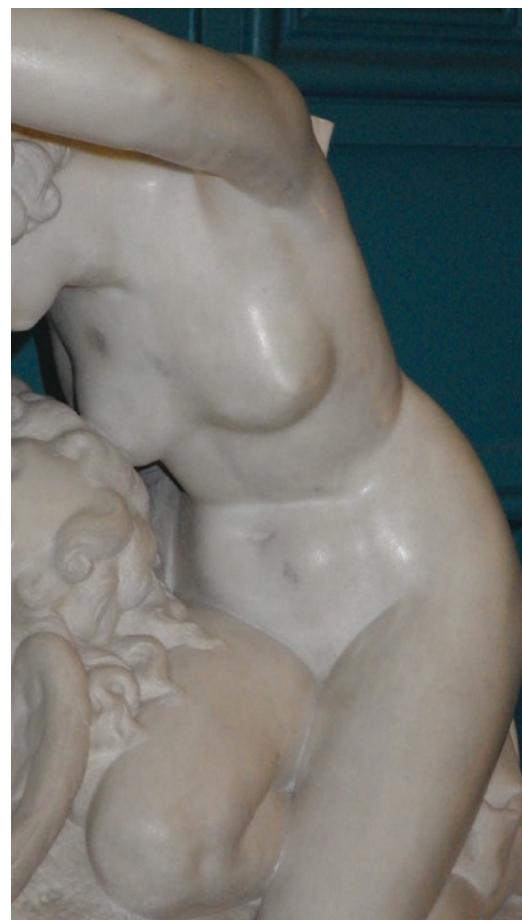
**Palmoplantar keratoderma** causes severe thickening of the skin on the palms and soles. It can be inherited, acquired, and occasionally associated with an underlying internal malignancy (e.g. the Howell-Evans' syndrome). Tylosis is inherited and can be associated with oesophageal carcinoma. Acquired diffuse palmoplantar keratoderma has occasionally been associated with carcinoma of the bronchus.

Treatment is usually with keratolytics such as salicylic acid and/or high dose urea containing creams.

### 36.19 Genital and Perianal Rashes and Lesions

Rashes, ulcers and lesions on the genitalia and perianal skin can lead to embarrassment, fear and depression. The patient may delay presenting to the doctor because of fear of infection or cancer. More severe disease can affect urinary and sexual function. You need to ask about the possibility of STDs and sexual preferences. Genital and perianal rashes and lesions in children may raise suspicions of non-accidental injury or sexual abuse.

#### 36.19.1 Female Genital Rashes, Ulcers and Lesions



Women often present to their doctor with symptoms of itch or discharge around the vaginal area. By far the most common cause is **vaginal candidiasis (thrush)**. *Candida albicans* is a harmless yeast that can be found asymptotically in approximately 20% of non-pregnant adult women in the vagina. An overgrowth of candida can cause a heavy, white, curd-like vaginal discharge, with burning or itch in the vagina and vulva. It can occur in pregnancy, in patients with diabetes or immunodeficiency and in patients on the contraceptive pill and antibiotics. *Candida albicans* can also proliferate if there is an underlying skin condition such as psoriasis, lichen planus or lichen sclerosus in the vagina.

Diagnosis is normally clinical and treatment can be with simple topical anti-yeast creams or pessaries. Resistant or recurring cases may need oral anti-fungal medication such as fluconazole (150 mg stat and 50 mg OD  $\times$  2 weeks) or itraconazole. In recurrent cases, if the male sexual partner is symptomatic with balanitis, he may also need to be treated with an anti-yeast cream on the penis for 7 days. Swabs should be taken in resistant or recurring cases to rule out other infections such as trichomonas and bacterial vaginosis.

**Trichomonas** is a common sexually transmitted infection caused by a protozoan parasite, *trichomonas vaginalis*. It may be asymptomatic or can cause a thin, yellow vaginal discharge with an offensive odour. There may also be itch or burning in the vagina or vulva. Treatment of trichomonas is usually with metronidazole which can be used in pregnancy and lactation. The male sexual partner should also be treated at the same time.

**Bacterial vaginosis** is due to an imbalance of the amount of bacteria in the vagina. This could cause a thin, white discharge with an offensive odour. Bacterial vaginosis is more common after antibiotics or when there is decreased oestrogen production. It may also occur if a woman has an IUD or has a number of sexual partners. Apart from the discharge, there are normally no other symptoms such as itch or pain. Treatment is not always necessary, particularly in asymptomatic cases. If there is an offensive discharge or if a woman is pregnant, she may require treatment with metronidazole.

Women with vaginal itch, soreness or discharge who are at risk of STIs (sexually transmitted infections), should have a full STI screen, particularly for gonorrhoea and chlamydia.

General skin conditions such as **seborrhoeic dermatitis, psoriasis, contact and irritant dermatitis and atopic eczema** can all affect the delicate vulval skin and cause itch (**pruritus vulvae**) (Table 36.2). Diagnosis is usually obvious from the history and a thorough physical examination of the whole skin, scalp and nails looking for clues to the diagnosis elsewhere. Treatment is usually similar for all these conditions (Table 36.3). The woman should be advised to

**Table 36.2** Pruritus vulvae—*aetiology*<sup>a</sup>

- Candida
- Bacterial vaginosis
- Trichomonas
- Contact allergic dermatitis
- Contact irritant dermatitis
- Seborrheic dermatitis
- Psoriasis
- Atopic eczema
- Lichen simplex
- Lichen sclerosus
- Lichen planus
- Neoplastic (VIN, Extra-mammary Paget's, SCC)

<sup>a</sup>There may be more than one cause

**Table 36.3** Pruritus vulvae: general advice

- General measures for delicate vulval skin:
- Avoid soaps and bubble baths
  - Use a soap free wash
  - Moisturise with a perfume free, fragrance free moisturiser
  - Wear loose fitting cotton underwear. Do not add fabric softner
  - Avoid nylon tights
  - Change sanitary towels frequently
  - Avoid horse riding and bicycles
  - Avoid potent topical steroids unless treating specific dermatoses such as lichen sclerosus or lichen planus
  - Avoid scratching if possible
  - Take a sedating antihistamine or amitriptyline at night if itch is keeping you awake
  - Use unbleached or minimally processed toilet paper or a wet flannel (bidet)
  - Avoid wet wipes

avoid all soaps and other irritants such as bubble baths and douches. She should use a soap free wash and a bath emollient with no detergents or perfumes and be encouraged to use cotton underwear, loose fitting clothing and avoid nylon tights. Patients should use unbleached or minimally processed toilet paper and avoid wet wipes to see if symptoms abate. Check the chemical content of personal hygiene products and clothing detergents for possible allergens or contact irritants. A skin patch allergy test may be required to check for contact allergic dermatitis in resistant cases [2].

1% Hydrocortisone ointment is safe and effective on delicate vulval skin. Stronger topical steroids should be avoided unless there is a specific dermatosis such as lichen planus or lichen sclerosus. Tacrolimus ("Protopic®") 0.1% ointment can be useful in resistant cases of psoriasis, eczema or dermatitis in the vulva. Superimposed clinical infections should be suspected and many women may respond to a combination of 1% hydrocortisone with an imidazole anti-fungal such as clotrimazole ("Canesten HC®"). Be careful as some topical antimicotic medication can be irritating in itself.

**Lichen simplex (neurodermatitis)** can cause a persistent itch in the vulval skin which may be due to habit scratching or may arise because of an underlying localised skin condition such as eczema, dermatitis or psoriasis. Constant scratching can irritate delicate vulval skin leading to more itch and more scratching, thus setting up a vicious cycle. Emollients, avoidance of soaps and other irritants and using topical steroids or topical calcineurin inhibitors (tacrolimus) may help to break the cycle.

**Lichen sclerosus (also known as lichen sclerosus et atrophicus)** is an auto-immune condition that can cause an itchy or painful rash in the vulva and perianal skin. It does not usually affect the mucous membranes but involvement at the edge of mucocutaneous junctions may lead to introital narrowing. It is ten times more common

in women than in men. It is most common in women in their 50s.

Vulval lichen sclerosus may be localised to one small area or may extensively involve all the vulva and sometimes perianal skin. It can cause severe itch and sometimes pain. It can cause adhesions and scarring which can make sexual intercourse uncomfortable or impossible. It usually presents as white, crinkled or thickened patches of skin that are extremely itchy and may scar. Diagnosis is confirmed by skin biopsy. Treatment is usually with potent or super potent topical steroid ointments. Patients should be instructed to apply the topical steroid once daily, accurately to the affected areas for 1 month, then alternate days for 1 month and then twice a week for 1 month. A regular maintenance treatment with moderately potent or potent topical steroid may need to be continued once or twice a week for a number of months to prevent recurrence. Some cases may respond to tacrolimus. Women should also be advised to moisturise with a perfume-free, fragrance-free, greasy moisturiser and should avoid soaps and other irritants. Sedating antihistamines or amitriptyline may be required at night if the itch is keeping the woman awake. Resistant cases may require systemic treatment such as oral steroids, hydroxychloroquine or methotrexate. Sexual intercourse may require abundant vaginal lubrication.

Vulval lichen sclerosus is associated with an increased risk of vulval intra-epithelial neoplasia (VIN), vulval cancer and anal cancer (SCC). Any patient with lichen sclerosus in the vulva or perianal area, who develops a lump or a sore that does not heal, should have a biopsy to rule out cancerous transformation.

Lichen sclerosus can occasionally affect non-genital skin (extra genital lichen sclerosus). It usually presents as one or more white, dry plaques on the inner thighs, buttocks, lower back, abdomen, under the breasts, neck, shoulders or axillae. The plaques have a wrinkled surface and waxy, thickened texture. Extra genital lichen

sclerosus is usually not as itchy as the vulval form and does not appear to predispose to cancer. Treatment is similar to genital lichen sclerosus with potent topical steroids or tacrolimus.

**Lichen planus** is a T cell-mediated autoimmune disease which causes chronic inflammatory skin changes. It is usually generalized and can affect almost any part of the body including the mucosal lining of the mouth, vulva and perianal skin. It causes reddish-purple papules and plaques which may have a fine white, lace-like pattern called Wickham striae. It can sometimes be hypertrophic, bullous or erosive. It can cause scarring and adhesions which can make sexual intercourse painful or impossible.

When it affects the vulval skin, the diagnosis can be suspected by examining the rest of the skin, hair and nails which may show typical features of lichen planus elsewhere. Skin biopsy of the vulval skin may be required to confirm the diagnosis. Treatment is usually with good skin care as outlined above for lichen sclerosus. In addition, lichen planus normally responds to potent or super potent topical steroids daily for 6–8 weeks. Some cases do well with topical calcineurin inhibitors such as tacrolimus ointment (“Protopic®”). Severe extensive cases may require a course of oral prednisolone for 1–2 months (see Chap. 19). Long-standing erosive lichen planus of the vulva can occasionally predispose to squamous cell carcinoma and therefore if a patient develops a sore or a lump in the rash, a biopsy should be taken.

**Atrophic vaginitis** can affect some women after the menopause. It usually responds to oestrogen vaginal creams.

**Extra-mammary Paget's disease** is an uncommon cancer which can cause a chronic eczematous rash on the skin around the anogenital region in males or females. It most commonly occurs on the vulva in women between the ages of 50 and 60. It usually presents as an itchy plaque in the groin, on the genitalia or in the perianal area. Thickening of the skin, erosion, bleeding and sec-

ondary infection can occur from chronic scratching. The plaques usually fail to respond to topical treatments including topical steroids. The diagnosis is confirmed by biopsy and treatment is usually surgical with wide local excision. Recurrence is common, so long-term follow-up should be arranged. Twenty percent of cases of extra-mammary Paget's have an underlying malignancy such as cervical or anal cancer.

**Vulvodynia** usually occurs in adult women, between the ages of 20 and 60. It usually causes pain, burning, stinging, irritation or rawness in the vulval area that lasts at least 3 months without any clear identifiable underlying cause. It can have a profound effect on a woman's quality of life, affecting all areas such as sitting down, exercising, riding a bicycle, horse riding or sexual intercourse. Chronic cases can lead to anxiety and depression. As the cause is unknown, treatment is difficult. Women should be instructed on general measures for delicate vulval skin and use silicon based lubricants during sexual intercourse (Table 36.3). Medication such as amitriptyline may help if the symptoms are keeping the patient awake at night. Some patients may require psychotherapy and behavioural modification. Some women will respond to anti-depressants. Strong reassurance that there is no underlying serious medical issue can sometimes help.

### 36.19.2 Ulcerating and Nodular Lesions in the Vulva

**Genital warts and molluscum** can occur on vulval skin. **Seborrhoeic keratosis** can also present as a nodular lesion in the same area. As the skin is hairy, **folliculitis** is not uncommon. STIs such as **syphilis** can occasionally cause an ulcer in the genital skin. Persistent ulcers or nodular lesions may need to be biopsied to rule out **malignant disease** such as VIN, extra-mammary Paget's, SCC or melanoma (Table 36.4).

**Table 36.4** Possible causes of genital ulceration in both females and males

Infections:
– Herpes simplex
– Syphilis
– Other STIs (e.g.: Lymphogranuloma venereum, Chancroid, etc.)
Inflammatory:
– Lichen planus
– Lichen sclerosus
– Scratching
Autoimmune:
– Behcet's disease
– Bullous disease
– Aphthous ulcers
– Pyoderma gangrenosum
Pre-malignant:
– Penile intraepithelial neoplasia (PIN)
– Erythroplasia of Queyrat
– Bowen's disease
Malignant:
– SCC
– Melanoma
– Extra-mammary Paget's
Trauma

**Fibroepithelial polyps and skin tags** are common around the genital skin.

An infected **Bartholin's gland** can cause swelling and sometimes discharge which is usually unilateral and responds to oral antibiotics. More severe cases may need incision and drainage.

**Genital herpes** can cause a recurrent, painful, vesicular eruption in the genital area (see Chap. 32).

**Behcet's disease** is a rare form of autoimmune vasculitis that can present with ulcers in the mouth, genitalia, eyes and sometimes a skin rash. There may be systemic symptoms such as fatigue, fever, nausea and joint pains. It is most common and more serious in people from the Mediterranean basin, Middle East and Far East. Diagnosis is usually clinical but investigations for vasculitis should be carried out (see Chap. 54). Treatment depends on the severity and area involved but some cases may need systemic anti-inflammatory medications such as oral steroids, azathioprine, colchicine, cyclophosphamide, thalidomide, or infliximab.

## 36.20 Male Genital Rashes and Lesions



Normal anatomical structures such as **angiofibromas** (**pearly penile papules**) at the base of the glans or **hypertrophic sebaceous glands** (**fordyce spots**) on the shaft of the penis are often confused as a disease process by anxious men but reassurance is all that is required.

### 36.20.1 Rashes on the Penis and Scrotum

One of the most common genital rashes in men is **candida** infections, especially if they are uncircumcised. This may be sexually transmitted. His partner may not have symptoms. Clinical features include **balanitis** (redness, soreness and itchiness of the glans) and some-

times a discharge. Good hygiene such as retracting the foreskin and washing away any smegma in the shower using a soap free wash or just water may help prevent a yeast infection. Treatment is usually with a topical imidazole anti-fungal cream and a man's sexual partner should also be treated. A random blood sugar should be done to rule out diabetes. Empirical treatment is usually sufficient and swabs should only be taken in resistant cases.

**Seborrhoeic dermatitis** and **psoriasis** can sometimes affect the penis and scrotum (Fig. 36.26). Involvement of genital skin occurs in 30–40% of patients with psoriasis. Psoriasis can occur on genital skin without affecting other parts of the body in 2–5% of cases of psoriasis.

When psoriasis occurs on the shaft of the penis, it may look red and scaly as in other area of the body. It is usually well demarcated with little or no itch. Under the foreskin, psoriasis and seborrhoeic dermatitis can look quite similar with redness, oozing and itchiness. There are usually no scales because of the occlusive effects of the foreskin. Psoriasis and dermatitis in this area can be aggravated by lack of hygiene or the excessive use of soaps. The diagnosis is usually confirmed by finding signs of psoriasis or seborrhoeic dermatitis in other parts of the body. The

diagnosis is usually clinical and treatment is usually with a weak topical steroid, combined with an imidazole anti-fungal. More resistant cases may require tacrolimus ointment, once daily for 1–3 weeks and once or twice a week after that to prevent relapse. “Dovobet®” and “Dithranol®” should be avoided in this sensitive genital skin.

**Contact allergic dermatitis** (e.g. from condoms) or **contact irritant dermatitis** (e.g. from soaps or shower gel) can sometimes affect the genitalia. **Atopic eczema** can also cause an itchy rash on the penis or scrotum. The patient usually has obvious signs of atopic eczema on other parts of the body. Treatment is usually by avoidance of allergens and irritants, moisturising and using a weak topical steroid such as 1% hydrocortisone or tacrolimus for more resistant cases.

**Burning scrotum** is a relatively common presentation in general practice. The man may or may not have a rash on the scrotum. If there is a rash, then a search for clues on other parts of the body (e.g. scalp, flexures, nails and perianal area) may help make the diagnosis. Sometimes there is little or no rash but there may be redness with intense itch or a burning sensation. This may be partially psychogenic and the patients may require a lot of reassurance that there is no infection and no cancer present. Skin scrapings for fungal stain and culture are sometimes required and a skin biopsy for histological diagnosis may be needed in non-responsive cases. A skin patch test may help identify a contact allergic dermatitis.

Treatment involves avoiding irritants such as shower gels, soaps, bubble baths and baby wipes. Patients should be advised to use a soap free wash and to moisturise frequently with a hypoallergenic, fragrance free, perfume free moisturiser. White cotton boxer shorts should be encouraged and they should be washed in non-biological powders and given a good rinse cycle. Patients should be encouraged to wear loose trousers. Topical steroids should be avoided in the delicate scrotal skin unless there is evidence of a definite steroid responsive dermatosis such as psoriasis, seborrhoeic dermatitis or eczema. In these conditions only weak topical steroids should be used. Indeed, potent topical steroids



**Fig. 36.26** Balanitis involving the foreskin and prepuce is termed balanoposthitis. This case is caused by seborrhoeic dermatitis

may sometimes be the cause a red burning scrotum resulting in a rosacea type rash or a rash resembling peri-oral dermatitis. Topical tacrolimus may be safer in this area when treating an inflammatory dermatitis such as eczema or psoriasis not responsive to a weak topical steroid. If there is signs of a rosacea or a peri-oral dermatitis like rash on the scrotum, it may respond to oral anti acne type antibiotics such as lymecycline for 1 or 2 month.

Systemic anti-pruritic medication like a sedating antihistamine such as promethazine hydrochloride (e.g.: "Phenergan®") or amitriptyline may be required especially if the burning sensation keeps the patient awake at night. Anecdotal reports have suggested that gabapentin or doxycycline orally may help [3–5]. Some patients may need psychiatric assessment to rule out an underlying psychological cause.

**Lichen planus** can also affect male genital skin. It is usually part of a generalised itchy rash. Unlike lichen sclerosus, it can also affect mucous membranes. When lichen planus affects the glans of the penis, it causes a reddish-purple, itchy, sore rash that may be erosive with vesicles, bullae and scars. It is sometimes necessary to take a biopsy to confirm the diagnosis. Lichen planus on genital skin is usually treated with moderately potent or potent topical steroids for a few weeks until the condition comes under control and then maintenance treatment with a mild or moderate potent topical steroid. More resistant cases may respond to tacrolimus. When lichen planus affects the genitalia, it can occasionally be pre-malignant. Cases that are resistant to treatment or develop ulcers or nodules should be biopsied to rule out malignant transformation.

**Lichen sclerosus** is a rare rash that may affect the tip of the penis in uncircumcised men. It can also affect the perianal skin. Early cases may have little or no symptoms. More advanced disease can cause itch, soreness and sometimes a tight band around the foreskin making an erection and intercourse painful. The rash can sometimes cause adhesions of the foreskin to the glans which may require surgical correction. Diagnosis is usually confirmed by skin biopsy. Treatment is usually with potent or very potent topical steroids

for 1 month and then weaning down to less potent steroids. More severe cases may require circumcision. Lichen sclerosus is considered premalignant and so patients should be followed-up to look for signs of malignant transformation such as resistance to treatment or the development of a nodule or an ulcer on the affected area.

**Zoon's balanitis (plasma cell balanitis)** is a condition that affects uncircumcised men mostly over the age of 50 years. It causes an uncomfortable, glistening, moist, brown or white rash on the foreskin which may become tethered to the glans of the penis that could make retraction of the foreskin painful or impossible (Fig. 36.27). Most will have no pain and the main concerns are aesthetic and that they may have a sexually



**Fig. 36.27** Zoon's (Plasma cell) balanitis in a 56-year-old male

transmitted disease. Diagnosis is can be confirmed by biopsy and treatment includes promoting hygiene, retracting the foreskin regularly to clean the area, Zinc oxide diaper creams after bathing, potent topical steroids or tacrolimus. Circumcision is sometimes the best option for unresponsive cases.

**Erythroplasia of Queyrat** is a rare form of penile intraepithelial neoplasia which is a pre-cancerous disease of the penis. It presents as a moist, shiny, isolated plaque, often on the glans or inner prepuce of uncircumcised men. The diagnosis is usually confirmed by biopsy and treatment is usually surgical by a urologist.

**Bowenoid papulosis** is a rare form of intraepithelial neoplasia which is a pre-cancerous skin condition. It is linked with the human papilloma (wart) virus. It presents as multiple reddish brown scaly papules on the shaft of the penis (Fig. 36.28). The diagnosis is normally confirmed by biopsy. Some cases resolve spontaneously. Treatment, if required, is usually with topical wart treatments, cryosurgery or other surgical techniques.

**Condyloma lata** is caused by secondary syphilis. It can present as greyish-white moist raised patches on the penis and can be easily confused with genital warts (condyloma acuminata). All suspicious lesions on the penis in men should be investigated with a VDRL blood test to rule out syphilis.

**Extra-mammary Paget's disease** is a rare intraepithelial adenocarcinoma which presents as a slowly, expanding, erythematous plaque with



**Fig. 36.28** Features of lichen sclerosis and Bowenoid papulosis in a 23-year-old male. Histology was inconclusive

an area of erosion or white scale on the scrotum or peri-anal skin. Diagnosis is usually confirmed by biopsy and treatment is surgical. It may be associated with an underlying malignancy.

Vaccination against HPV in pre-pubertal males may prevent against genital warts and HPV associated anogenital intracellular neoplasia and invasive SCC in the future.

### 36.20.2 Lesions on the Penis and Scrotum

**Genital warts** can occur on any part of the penis and scrotum. They sometimes spread into the urethral meatus. Diagnoses are usually clinical and all cases should have a full STI screen and contact tracing. Treatment options include podophyllin, imiquimod or cryosurgery alone or combined (see Chap. 32). Condoms should be used until all warts are cleared. Some cases will clear spontaneously. All cases should be followed up until all visible warts are cleared.

**Molluscum contagiosum** can sometimes present as tiny, white, dome-shaped papules with a central umbilicated crater on the shaft of the penis or the pubic area. They are usually multiple and often sexually transmitted. Unlike viral warts, they are not associated with penile intraepithelial neoplasia or cervical cancer in women. Molluscum usually responds to very light cryosurgery (3–5 s) with or without topical anaesthesia.

Scrotal cysts are also known as **pilar cysts** or trichilemmal cyst. They are a keratin-filled cyst that originates from the outer hair root sheath. Unlike a sebaceous cysts (epidermoid cysts), pilar cysts do not have a punctum. They are most commonly found in the scalp and the scrotum. They are usually small and painless but may be multiple (Fig. 36.29). Although they are harmless, patients often want them removed for cosmetic reasons.

**Angiokeratomas** are benign vascular malformation of capillaries that can occur on the scrotum or vulva. They are harmless and usually do not require treatment. If they are big or bleed, they can be treated with cryosurgery or lasers.



**Fig. 36.29** Scrotal cysts in a 21 year old

**Non-melanoma skin cancer** of the penis or scrotum is rare but penile intraepithelial neoplasia, Bowen's disease and SCC can occur on the shaft, glans or scrotum. **Melanoma** can also occur on the glans penis, the shaft of the penis or the scrotum.

Skin cancer on the penis usually has a poor prognosis partially because embarrassment may delay presentation. Any suspicious growth, ulcer or rash on the penis or scrotum that fails to respond to treatment should be biopsied to rule out malignancy. Most confirmed cases are treated surgically by a urologist.

**Scabies** in men almost always cause itchy nodules on the shaft of the penis and this sign can be used to help confirm the diagnosis of scabies.

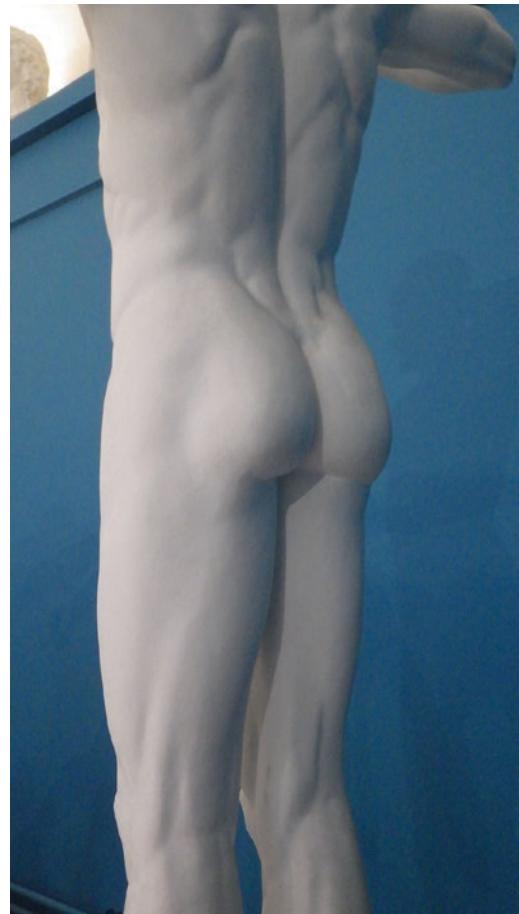
**Folliculitis** can also affect hairy parts of the body, particularly in the pubic area and on the scrotum.

### 36.20.3 Genital Ulceration in Men

Various infectious, inflammatory, autoimmune, pre-malignant and malignant conditions can cause genital ulceration (see Table 36.4). **Genital herpes** can cause a recurrent, painful, vesicular eruption in the genital area (see Chap. 32).

Rare causes of ulcers on the genitalia include drug eruption, Behcet's disease (see above under female genital ulceration) and dermatitis artefacta.

### 36.21 Perianal Itch, Rashes and Lesions



Itch and soreness around the perianal area is a very common presentation in general practice. There can be many causes but most patients can be accurately diagnosed by a careful history and a thorough physical examination of the perianal area and of the skin, hair and nails generally. A rectal examination should also be carried out. Sexually transmitted infections, unusual sexual practices and sexual abuse may have to be considered in the differential diagnosis.

**Rectal bleeding** should always be taken seriously and investigated thoroughly. It cannot be assumed it is due to haemorrhoids ("piles"). Always consider the possibility of a colon/rectal malignancy.

In children and adults **anal fissures** are common and painful. Fissures may lead to constipation which may further exacerbate the problem. There may be bleeding on defaecation and rectal examination may be difficult or impossible because of pain. The diagnosis is usually obvious by inspecting the perianal area which should reveal a linear split of the mucosa. Treatment involves oral analgesics, stool softeners, extra fiber in the diet and topical glyceryl trinitrate ("Rectogesic<sup>®</sup>") twice a day for 8 weeks. This relaxes the smooth muscle and anal tone. Like sublingual GTN spray, it can cause a headache in 30% of patients. Topical anaesthetics such as 1–2 ml of lidocaine applied to the sore area immediately before defecation daily for 14 days may also help. Resistant cases should be sent for a surgical opinion.

**Threadworms** may cause perianal itch in children and adults. The worms are usually visible on careful inspection of the stool or the perianal area, particularly at night. A good light and magnification will help to see the tiny worms which look like small pieces of thin, white cotton threads, 2–13 mm in length, which wriggle around. The adult worm lives in the intestine for up to 6 weeks. At the end of their life cycle, the female lays her eggs around the perianal area, especially at night and this is what leads to itch. Scratching transfers the eggs to the fingers which can then lead to auto-infection or cross infection to other family members, if the hands are not washed carefully. Treatment is usually with an

oral anti-helminthic such as mebendazole, 100 mg stat, which is repeated once more, 2–4 weeks later. All family members and household contacts should be treated whether they have symptoms or not. Mebendazole should not be used in children under the age of 6 months, in pregnancy or in breast feeding women.

All family members should be instructed to carefully wash their hands first thing in the morning and every time they use the toilet. Undergarments and bedclothes should be washed regularly. These measures can usually break the life cycle of threadworms, provided they are continued for at least 6 weeks and can be used in children under 6 months, pregnant women and breast feeding women where drug therapy should be avoided.

**Haemorrhoids** are enlarged vascular mucosal cushions in the anal canal. They are a common cause of perianal itch, pain and bleeding. They can be aggravated by constipation, straining, pregnancy or chronic cough. It is important to realise that symptoms such as bleeding, pain or itch may be due to other more sinister causes even in the presence of haemorrhoids. Persistent bleeding usually requires referral for sigmoidoscopy or colonoscopy to rule out more serious disease such as cancer of the rectum or colon.

Haemorrhoids may be helped by simple measures such as stool softeners, drinking plenty of water and anaesthetic or steroid suppositories for a maximum of 7–14 days. Used longer than this, they may cause sensitisation, skin atrophy and contact dermatitis.

**Prolapsed thrombosed piles** (perianal haematoma) usually present as a painful lump in the perianal area which may require incision and drainage of the clot under local anaesthetic to obtain relief. More troublesome haemorrhoids need referral for other surgical treatments.

Generalised skin conditions such as **seborrhoeic dermatitis, psoriasis, atopic eczema, contact irritant or contact allergic dermatitis** are all common causes of itch and soreness in the perianal skin. A good history and a general physical examination will often help to make an accurate diagnosis (Table 36.5). Treatment of all these conditions is similar (Table 36.6).

**Table 36.5** Possible causes of pruritus ani:

- Hairy skin
- Sweating
- Micro-organisms (candida, bacteria)
- Soaps and other irritants such as anaesthetic or analgesic creams, ointments or suppositories.
- Wet wipes
- A weak anal sphincter leading to faecal leakage
- Constipation
- Spicy foods
- Excessive alcohol or caffeine
- Tight clothing
- Scratching
- HPV (ano-genital wart) infection

**Table 36.6** General measures for itchy dermatoses in the perianal area

- Avoid soaps and other irritants (bubble baths, baby wipes, topical analgesics or anaesthetics)
- Wash with a soap free wash
- Apply abundant Vaseline or "Zinc and castor oil" if doing sports which generate excess friction like biking or long walks or runs.
- Wear loose fitting cotton underwear and loose trousers or a dress. Do not wash your underwear with fabric softener.
- Avoid scratching
- Sedating antihistamines or amitriptyline may help relieve itch at night
- Avoid constipation
- Use a weak topical steroid (1% hydrocortisone) with or without an imidazole anti-fungal (e.g. "Daktacort®", "Canesten HC®") if recommended by the doctor.
- Tacrolimus, applied once nightly, for up to 3 weeks and once or twice a week after that can be helpful in more resistant cases
- Avoid potent topical steroids
- Use unbleached or minimally processed toilet paper or a wet flannel (bidet).
- Avoid wet wipes

**Group A streptococcal and staphylococcal infection** can cause a superficial bacterial infection in the perianal skin especially in children under the age of 10 years. Any painful, red, inflamed, pussy rash should be swabbed for culture and sensitivity and if a bacterial infection is suspected, it should be treated with a broad spectrum antibiotic such as flucloxacillin.

A painful, purulent rash in the perianal area should also raise the suspicion of an STI such as **gonorrhoea** especially in men who have sex with men.

**Erythrasma** is an uncommon cause of a non-itchy, flexural rash, particularly in the groin, intergluteal folds, perianal area (discussed above under flexural rashes).

Rare skin diseases such as **lichen planus** or **lichen sclerosus**, which more commonly occur in the vaginal or penile skin, can also affect the perianal area. Diagnosis is usually by skin biopsy and treatment is usually with a potent topical steroid or tacrolimus.

Infections such as **anogenital warts (Condyloma Acuminata)**, **molluscum contagiosum**, **condyloma lata** (secondary syphilis) and **herpes simplex** can present as lesions or ulcers in the perianal area.

Neoplastic disease such as **anal intraepithelial neoplasia (AIN)**, **Bowen's disease**, **SCC** or **Extramammary Paget's** can also occur in the perianal area. Any unusual symptoms such as bleeding, lumps, ulcers, persistent itch, discharge or unusual polyps or skin tags should be biopsied for histological diagnosis. Anal cancer is more common in patients who have been infected with the HPV virus (e.g. genital warts). It is also more common in men who have sex with men, people who engage in anal sex and in chronic skin conditions such as lichen sclerosus or lichen planus. Vaccination against the HPV virus may reduce the incidence of anal cancer in the future. Safe sex such as using condoms and limiting the number of sexual partners may also help reduce the incidence of anal cancer.

**Pruritus ani** is usually defined as itch in the perianal area without any obvious underlying skin disease. There may be some perianal erythema and excoriation but usually no signs of obvious eczema or psoriasis elsewhere. The cause is often multifactorial which can lead to chronic irritant dermatitis (Table 36.5). It is more common in men probably because they are more hairy and sweaty. Treatment involves avoiding all items mentioned in Table 36.5 and more specific treatment as outlined in Table 36.6. Most cases can be managed by avoiding soaps and other irritants and frequent moisturising with a safe greasy moisturiser and barrier cream (e.g. Vaseline or Zinc and Castor oil). Many cases are caused by friction of the skin in the perianal area which is aggravated by hairy skin and sweat. Also tiny amounts of faeces may leak from the anal sphincter.

ter which will further aggravate the rash and itch. Cleaning the perianal area with perfumed baby wipes or bleached toilet paper may also cause problems.

The toilet paper industries use chlorine and chlorine dioxide to bleach it which can cause a build-up of dioxin in the toilet tissue. Formaldehyde, which is used to improve the wet-strength and other important characteristics of paper and paper products can also irritate delicate perianal skin. Bisphenol A (BPA) can be found in high concentrations in paper products, including recycled toilet paper. Toilet paper which is brown or beige in colour is usually made without bleach but lacks softness and comfort. Look for toilet paper with the following labels: *BPA-free paper products, TCF (Totally Chlorine-Free items) or PCF (Processed Chlorine-Free)*. If unsure, it may be safer to clean the delicate perianal skin with water and a wet flannel (bidet) [6].

Nothing stronger than 1% hydrocortisone should be used on the delicate perianal skin which is similar to the skin on the eyelids. Combining the 1% hydrocortisone with a broad spectrum anti-infective agent such as an imidazole antifungal like miconazole (“Daktacort®”) or clotrimazole (“Canesten HC®”) may help if there is an associated low grade infection. The thin skin in the perianal area combined with the occlusive effects of the folds of skin in this area makes it very vulnerable to steroid damage such as erythema, skin thinning and skin infection. If there are obvious signs of skin infection a swabs for bacterial culture and sensitivity and skin scrapings for fungal stain and culture should be taken (Fig. 36.30). The patient may require

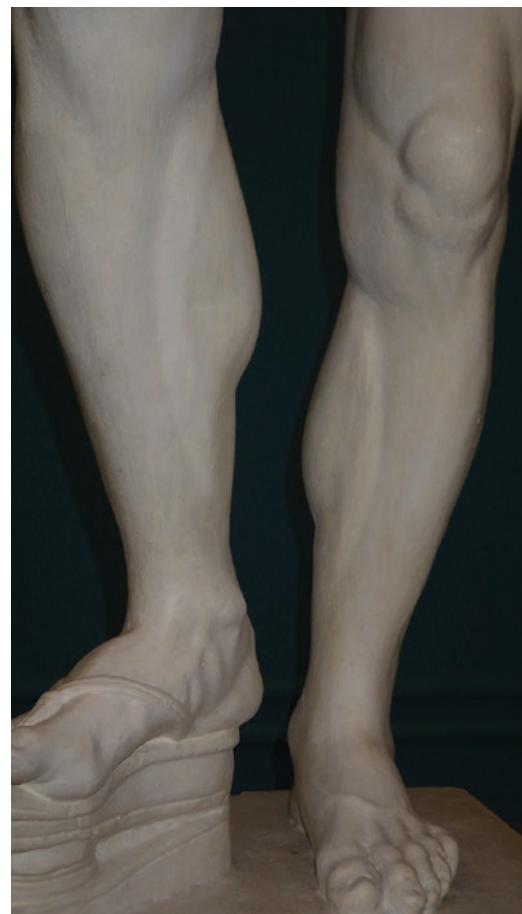
treatment with an oral antibiotic or antifungal for a few weeks to clear the infection which is usually secondary to the underlying pruritus ani.

Potent topical steroids can cause a steroid rosacea type problem in the perianal area and patients may have difficulty weaning themselves off them as they may get a rebound of their symptoms if the steroid is stopped abruptly. It is probably kinder to wean down to a moderate potency steroid and later 1% hydrocortisone over a few weeks. More difficult to treat cases may respond to tacrolimus (“Protopic®”) but the patient should be warned that 50% of patients who commence tacrolimus get a transient erythema and irritation of the skin on the first week of use. This usually settles on the second and subsequent weeks (see Chap. 64).

### 36.22 Lower Leg Rashes, Lesions, Ulcers and Blisters



**Fig. 36.30** Tinea cruris causing pruritus ani. Skin scrapings grew Trichophyton rubrum



Many generalised rashes may also occur on the **pretibial** region such as psoriasis, atopic eczema and lichen planus. However, certain other conditions are most commonly found on the lower leg and particularly in the pretibial region (see also Chap. 24).

**Erythema ab igne** is often found on the pretibial region caused by sitting too close to the fire, from hot water bottles or laptops places on the legs. It causes a mottled reticular discolouration (Fig. 36.31). There is no specific treatment but the condition usually resolves once the heat source is removed.

**Asteatotic eczema (eczema craquele)** is most commonly seen on the lower legs in the elderly due to excessive drying of the skin. It



**Fig. 36.31** Erythema ab igne from hot water bottles in a 17 year old

presents as a dry, scaly rash with irregular erythematous fissures like “crazy paving”. Treatment is by moisturising with a greasy moisturisers and avoiding soaps and other irritants.

**Lichen simplex chronicus (neurodermatitis)** is common in the lower leg and usually occurs as a result of chronic scratching and itching. It often presents as an isolated, scaly, eczematous patch on the lower shin which is constantly being scratched by the hands or the opposite leg. Treatment is the same as most forms of eczema. The patient should be advised to moisturise liberally with safe, greasy moisturisers and avoid soaps and other irritants. A potent topical steroid may help to ease the itch and break the itch-scratch cycle and occlusion of the steroid may be necessary with plastic kitchen wrap (cling film, shrink wrap, Saran wrap, cling wrap or food wrap).

**Cellulitis and erysipelas** are common in the lower leg and can often start with a small scratch or scrape in the skin of the foot or lower leg. Some cases arise from broken skin due to tinea pedis. The red, hot, tender rash spreads up the leg and in more extensive cases the patient may have flu-like symptoms. Treatment is either with oral or intravenous antibiotics, rest and elevation.

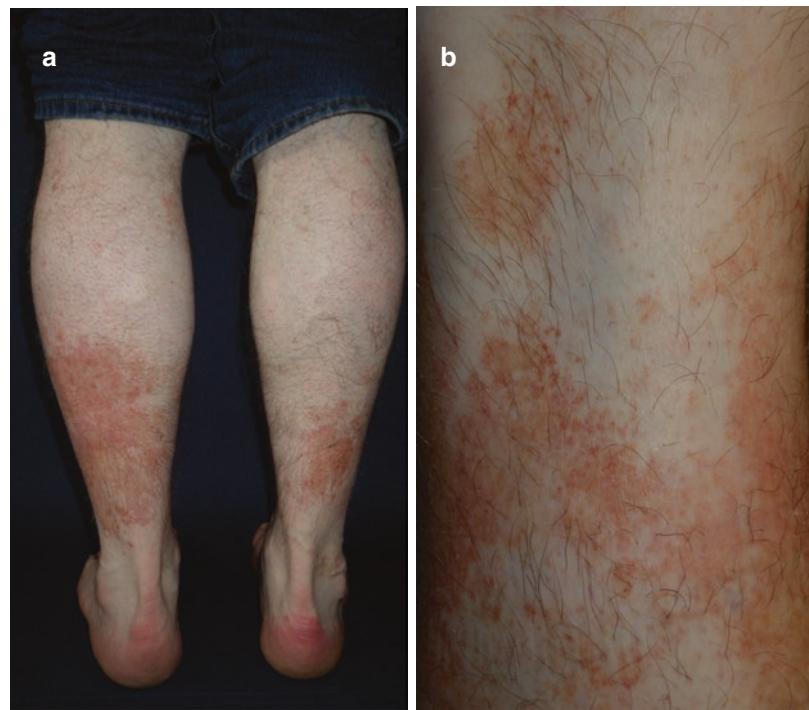
Cellulitis can sometime be confused with a red, swollen leg that can occur as a result of a deep vein thrombosis (DVT). If there are any doubts, a Doppler study should be carried out urgently as DVT can be potentially life threatening if it progresses to a pulmonary embolism.

**Schamberg's disease (also known as “progressive pigmentary dermatitis of Schamberg”)** is a chronic discolouration of the skin found in the lower legs. It causes rusty, brown/orange, patchy, non-blanching purpura that begins around the ankles and progresses up the lower leg over a period of months or years. ‘Cayenne pepper’ type spots develop at the edge of old lesions (Fig. 36.32a, b). There are usually no symptoms and there are no obvious causes or effective treatments. Cosmetic camouflage may help to disguise the rash.

**Purpura** is due to leakage of blood vessels which causes deposition of hemosiderin into the skin. This results in a purple to orange discolou-

**Fig. 36.32 (a)**

Schamberg's disease (Progressive pigmented purpura) in a 51-year-old male. **(b)** Schamberg's disease in the lower leg



ration of the skin. Purpura may be due to a low platelet count (thrombocytopenia) or a vascular disorder such as vasculitis.

**Vasculitis** causes inflammation of the blood vessels which also causes leakage of hemosiderin into the skin resulting in palpable purpura. Causes include Henoch-Schonlein purpura or collagen vascular disease such as SLE, rheumatoid arthritis or systemic sclerosis. All cases of suspected cutaneous vasculitis should have their urine and blood pressure checked as there may be co-existing nephropathy (see Chap. 54).

**Senile purpura** occurs as a result of lack of connective tissue support for blood vessels causing leakage of blood into the skin. It can occur in old age or after using topical or systemic steroids. Bruising occurs after minor trauma especially on the hands and lower legs.

**Necrobiosis lipoidica** causes well-defined, round or oval shaped plaques on the front of the shins and is strongly associated with diabetes. The rash may precede diabetes for many years. Seventy percent of patients with this granulomatous condition will develop diabetes at some stage. The reported prevalence of necrobiosis

lipoidica in patients with diabetes is 1–2%. It is three times more common in women than in men. The affected areas are atrophic and occasionally may ulcerate after minor trauma. The plaques are usually on the front of the shins and have a characteristic brown edge with yellow discolouration in the centre and obvious telangiectasia (Fig. 36.33a, b). The condition is very difficult to treat but the actively inflamed lesions, which have raised, mauve borders, may respond to potent topical steroids. Plaques should be protected from trauma with dressings or shin guards.

**Pretibial myxoedema** occurs in about 10% of patients with hyperthyroidism or thyrotoxicosis (Grave's disease). Waxy plaques or nodules, which can be pink, flesh coloured or yellow, develop on the skin on the front of the shin (Fig. 36.34). There may be a "peau d'orange" effect. Management is with a potent topical steroids and treating the underlying hyperthyroidism.

**Erythema nodosum** presents with red, hot, tender nodules or plaques that most commonly occur on the shins but may also occur on the forearms (Fig. 36.35). Lesions can be more than 10 cm in diameter and fade over the course of a



**Fig. 36.33** (a) Necrobiosis lipoidica in a 42-year-old with no diabetes. (b) Necrobiosis lipoidica close up



**Fig. 36.34** Pretibial myxoedema in a female with hypothyroidism



**Fig. 36.35** Erythema nodosum post-sore throat in a 22-year-old woman

few weeks, leaving residual bruising which fades eventually.

Table 36.7 outlines the common causes of erythema nodosum. See Table 36.8 for investigations to be carried out in a case of EN. Treatment is by addressing the underlying cause. Anti-inflammatories and analgesics such as paracetamol or codeine may help. Rest and elevation should be advised. Elasticated support stockings may be required if there is significant swelling. Most cases resolve spontaneously within 3–6 weeks.

### 36.22.1 Ulcers and Blisters on the Pretibial Area

The most common cause of ulcers in the pretibial area is **varicose ulcers**. Less common causes include **arterial ulcers**, **malignant ulcers** and ulcers associated with collagen vascular disease such as rheumatoid arthritis and SLE. Necrobiosis lipoidica can ulcerate from minor trauma and can prove very difficult to heal. Tropical ulcers can occur in patients who travel to the tropics.

**Table 36.7** Common causes of erythema nodosum

- Streptococcal sore throat
- Pregnancy
- Tuberculosis
- Drugs (e.g. sulphonamides, the oral contraceptive pill)
- Sarcoidosis
- Ulcerative colitis
- Crohn's disease
- Various bacterial, viral and fungal infections

**Table 36.8** Investigation for erythema nodosum

- Routine bloods including Full blood count, erythrocyte sedimentation rate, urea and electrolytes, liver function tests, Glucose
- ASOT = Antistreptolysin O (ASO) titer
- Throat swab
- Serum ACE (sarcoid?)
- Mycoplasma titre
- Urinalysis
- Chest X-Ray

**Pyoderma gangrenosum** is a rare ulcerating condition that most commonly occurs in on the lower legs which can be associated with ulcerative colitis, Crohn's disease, rheumatoid arthritis, chronic active hepatitis and multiple myeloma. About 50% of cases have no associated risk factors. It can present as a rapidly growing painful ulcer with a raised, over-hanging, purple edge and a yellow, honeycomb like base. Ulcers can vary from 1 to 2 cm in diameter. Some cases present as multiple, small, punched-out ulcers (Fig. 36.36). Treatment usually requires high dose oral steroids. Less severe cases may respond to topical steroids or tacrolimus. Other treatment options include Dapsone, oral anti-inflammatory antibiotics such as minocycline or biological agents such as anti-TNF therapies (e.g. infliximab) which can also be used for treating an underlying inflammatory bowel disease if present. Appropriate wound dressings, depending on the stage of the ulcer, should help heal the ulcer. If there is any coexisting evidence of venous hypertension, high compression stockings or bandaging may be required but the patient may not be able to tolerate this because of pain within the ulcers. Secondary infection may need treatment with systemic antibiotics.



**Fig. 36.36** Pyoderma gangrenosum with painful leg ulcers in a 67-year-old female with ulcerative colitis. Her ulcers responded to oral steroids



**Fig. 36.37** BCC in the upper calf

### 36.22.2 Lesions on the Lower Legs

**Actinic keratosis, Bowens disease, SCC, BCC and melanoma** (pigmented or non pigmented) can all occur on the lower legs because of sun exposure especially in women as they wear dresses. They can present as ulcers, isolated scaly patches or nodules (Fig. 36.37). Biopsies should be performed on any suspicious lesion to confirm the diagnosis and plan treatment.

**Dermatofibroma** are most commonly found on the legs and are more common in women. They classically present as a firm nodule in the skin with central scarring and erythematous or hyperpigmented borders measuring 5–10 mm in diameter. They may display the “dimpling sign” where the surrounding skin puckers when the lesion is squeezed from the side. They are harmless and usually resolve spontaneously but it can take years. They are sometimes removed for cosmetic or comfort reasons.

**Warts** are common on the knees on children and may will resolve spontaneously within 1–2 years.

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### 36.23 Conclusion

Being familiar with the location and distribution of common rashes and lesions will help make a diagnosis when a rash or lesion occurs in localized areas of the body. Treatment of the same condition may vary depending on which part of the body is affected. For instance, potent topical steroids may be appropriate for treating eczema on the body but not on the face or groin.

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# Leg Ulcers: A Treatment Programme

37

David Buckley

## Key Points

- Most leg ulcers (80%) are due to varicose veins which cause venous stasis and varicose ulcers.
- The mainstay of treatment for varicose ulcers is a combination of elevation, exercise and compression bandaging or hosiery (stockings).
- Patients with leg ulcers should be managed holistically by looking beyond the ulcer and ensuring the patient has no underlying conditions that may delay wound healing.
- There is no single ulcer dressing that will deal with all situations. It is best to identify the most appropriate dressing for each stage of the healing process.

## What to Tell the Patient

- Elevating your leg by 30 degrees above the horizontal for 2 hours morning and evening by lying out on the couch and placing your legs on the arm rest.
- Walk for an hour every day and avoid standing in the one spot.
- Eat well, maintain a healthy weight and do not smoke if you have leg ulcer.

- If you have varicose veins and varicose ulcers you should wear high compression support stockings daily for the rest of your life.

## 37.1 Introduction

Most patients with leg ulcers suffer from either pain, malodorous discharge or difficulty with mobility. When poorly managed, many of these patients can take months or years to heal and have a high incidence of relapse. This places a huge financial strain both on the patient and on the state. GPs and nurses are ideally placed to provide a holistic treatment program for patients with leg ulcers.

## 37.2 Clinical Features and Diagnosis

Before treating a leg ulcer it is important to examine the patient generally to identify and treat any underlying factors which may delay healing such as smoking, obesity, anaemia, diabetes, hypothyroidism, cardiac failure, steroids and non-steroidal anti-inflammatory drugs (Table 37.1). The patient's nutritional status and home environment should be assessed. All patients with leg ulcers will benefit from elevating their leg by 30 degrees above the horizontal for 2 hours morning and evening by lying out on the couch and placing

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their legs on the arm rest. They should also be encouraged to walk for an hour every day and avoid standing in the one spot (see Chap. 66).

The next stage is to make an accurate diagnosis of the exact aetiology of their ulcer (Table 37.2). Most leg ulcers (80%) are due to varicose veins which cause venous stasis and varicose ulcers. Varicose ulcers usually have a sloping edge and are found mainly around the medial malleolus, ankle or lower calf (Figs. 37.1 and 37.2). The mainstay of treatment for varicose ulcers is a combination of elevation, exercise and

**Table 37.1** Investigation of chronic or non-healing wounds

- Full blood count, erythrocyte sedimentation rate
- Urea and electrolytes,
- Liver function tests.
- Glucose
- Thyroid Function Tests (TFT's)
- HbA1C
- B12, Folate, Ferritin
- Antinuclear factor
- Rheumatoid factor
- Urinalysis
- Doppler ankle/arm pressure
- Skin biopsy (if malignancy suspected)
- Patch test (if contact allergic dermatitis suspected)
- Send tissue, exudates or swab for C + S if clinically infected

**Table 37.2** Causes of leg ulcers

Varicose hypertension	~80%
Arterial insufficiency	~10%
Malignant	~5%
Vasculitis (e.g. rheumatoid arthritis, SLE, diabetes, pyoderma gangrenosum, etc.)	~5%

**Fig. 37.1** Varicose ulcer



compression bandaging or hosiery (stockings). This encourages venous return by activating the muscle pumps which in turn reduces venous hypertension and promotes healing. Compression should be commenced if a wound is not healing within 2 weeks [1]. To prevent delays in treatment, class 1 (17 mmHg compression) (flight) stockings can be prescribed provided there are no risk factors for arterial insufficiency (smoking, diabetes, arteriosclerosis, etc.) while awaiting Doppler studies of the arterial pressures in the lower legs (ABPI) [1].

Arterial ulcers account for approximately 10% of leg ulcers. Roughly half of these will also have clinical features of venous insufficiency which can mask the true aetiology. Arterial ulcers are mainly found on the foot. They are usually deep, painful and have rolled punched out edges. There may be other signs of arterial insufficiency such as weak or absent pulses, pallor, coldness and shiny, hairless skin. Peripheral vasodilators might help but resistant



**Fig. 37.2** Varicose ulcer + varicose eczema

cases should be referred for vascular assessment and perhaps surgery.

### 37.3 Differential Diagnosis

A small percentage of patients with leg ulcers (10%) are due to systemic illnesses that cause vasculitis such as rheumatoid arthritis, SLE, diabetes, pyoderma gangrenosum, etc. Successful management of these will depend on optimum control of the underlying condition, together with good wound management.

Trauma to the leg can cause an ulcer but most traumatic ulcers will heal in a few weeks with



**Fig. 37.3** Ulcers in both lower legs failing to heal after a few years. Biopsies showed both were due to BCC

simple wound care provided there is a good blood supply to the leg and no varicose veins.

A leg ulcer which fails to heal, despite appropriate management, should be biopsied to rule out malignancy (BCC, Bowens, SCC or melanoma) (Fig. 37.3).

### 37.4 Pathophysiology

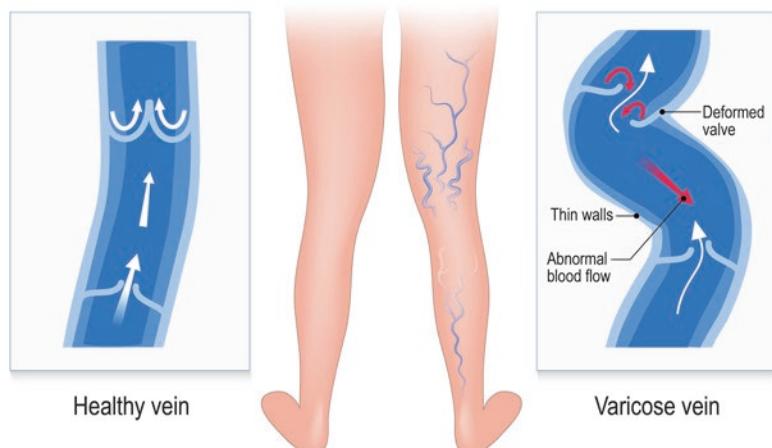
Varicous ulcers occur because of venous stasis which may arise from previous DVT, phlebitis or long standing primary varicose veins. This creates a reversal of flow with venous blood passing backwards from the deep to the superficial venous systems through incompetent perforators, causing venous hypertension which can lead to varicose eczema and/or varicose ulcers (see Fig. 37.4).

### 37.5 Treatment

Compression is the most important strategy when treating varicose ulcers since this will deal with the underlying cause (i.e. venous hypertension). However, compression bandaging or hosiery might further compromise blood supply if there is any arterial insufficiency. Therefore, it is very important to check the

**Fig. 37.4** Aetiology of varicose veins. VARICOSE VEINS© [designua]/123RF.COM Image ID 78422387. Media Type: Vector. [https://es.123rf.com/photo\\_78422387\\_varicose-veins-and-normal-vein-medical-illustration.html?vti=mpdgcfdxm6p4a7ktbn-3-50](https://es.123rf.com/photo_78422387_varicose-veins-and-normal-vein-medical-illustration.html?vti=mpdgcfdxm6p4a7ktbn-3-50)

## VARICOSE VEINS





**Fig. 37.5** Measuring the ankle systolic pressure with a vascular ultrasound device and a sphygmomanometer

pedal pulses. If they are very weak, impalpable or difficult to assess because of overlying ulcers, swelling or oedema, then a doppler ultrasound should be used to check the ankle pressure. This is simple to do in the GP's surgery but requires a special vascular probe for the Doppler (not a maternity Doppler probe) and a manual sphygmomanometer. The systolic BP is measured in the arm using the Doppler and then on the leg by placing the cuff around the calf and listening for the pulse to reappear over the dorsalis pedis or posterior tibial artery with the vascular Doppler while deflating the cuff (Fig. 37.5). The ratio between the ankle and the arm systolic blood pressure (ankle-brachial index = ABI) is normally  $>1$  and should be at least  $>0.8$  if compression bandaging is to be used (Table 37.3). If the ABI is  $>1.3$  it may imply incompressible or calcified arteries which is often seen in patients with diabetes, end stage renal disease and the very elderly. In these patients more sophisticated tests by a vascular surgeon may be necessary before applying compression bandaging or stockings.

## 37.6 Wound Care

Expensive primary wound dressings on venous ulcers are no substitute for adequate compression with bandaging or compression hosiery

**Table 37.3** Ankle brachial index (ABI). Who is suitable for compression bandaging or hosiery?

ABI value	Interpretation	Recommendation
Greater than 1.4	Calcification of the vessel?	Refer to vascular specialist
1.0–1.4	Normal	High compression bandaging or hosiery
0.9–1.0	Acceptable	High compression bandaging or hosiery
0.8–0.9	Some arterial disease	Low compression bandaging or hosiery
0.5–0.8	Moderate arterial disease	Refer to vascular specialist
Less than 0.5	Severe arterial disease	Refer to vascular specialist

(stockings). Doctors and nurses too often feel the need to use something medicated to clean the wound when simply swabbing the ulcer with a piece of gauze or bathing it with sterile saline would suffice. There is no single ulcer dressing that will deal with all situations. It is best to identify the most appropriate dressing for each stage of the healing process (see Chap. 38) [2].

Sending routine bacterial swabs on leg ulcers for analysis is generally unhelpful, since most will be colonised with a whole host of organisms that will not delay healing. If the ulcer shows clinical signs of infection, such as swelling, redness, heat or malodorous or green discharge, tissue or discharge should be sent for culture and sensitivity especially looking for multi resistant *Staphylococcus aureus* (MRSA). The infected wound should be dressed with a tulle dressing impregnated with an antiseptic such as chlorhexidine, ("Bactigras®", made by Smith & Nephew) or povidone-iodine ("Inadine®", "Systagenix®" or "Betadine®", made by Mundipharma Pharmaceuticals). For persistent ulcers, especially when complicated with resistant organisms, a silver impregnated dressing may help, such as "Actibisorb Silver®", "Acticoat®" or "Aquacel Ag®". The patient

should also be commenced on a broad spectrum oral antibiotic for at least 2 weeks. Clean wounds can be dressed with a non-sensitising, non-adherent wound contact layer, such as knitted viscose e.g. “N-A dressing<sup>®</sup>”, made by Johnson & Johnson.

Hydrocolloid dressings such as “Duoderm<sup>®</sup>”, “Comfeel<sup>®</sup>” or “Granuflex<sup>®</sup>” create a healing environment where humidity, temperature and gaseous concentrations are controlled and the wound is protected from infection and trauma. These dressings should not be used on infected wounds.

Heavy exuding ulcers may be dressed with an absorbent dressing such as a polyurethane foam dressings (“Lyofoam<sup>®</sup>”, “Polymem<sup>®</sup>” or “Allevyn<sup>®</sup>”) or a calcium alginate dressing such as “Curasorb<sup>®</sup>” or “Kaltostat<sup>®</sup>”.

If there is surrounding varicose eczema, this may be treated with a potent topical steroid ointment for a few weeks. However, these should never be placed in or near an ulcer.

If there is extensive weeping eczema, then an impregnated paste bandage such as “Ichthopaste<sup>®</sup>” (zinc + ichthamol) or “Viscopaste PB7<sup>®</sup>” (zinc) can be used from the toe to the knee with or without a local dressing over the ulcerated area. A ‘pleat’ technique is used to apply the paste bandages rather than winding them around the leg. This stops the bandage from tightening when it dries out. Full support bandaging needs to be applied outside the impregnated bandage provided there is no PVD.

Allergic contact dermatitis may develop if potentially sensitising substances are left near the wound or in contact with the skin. This can cause an eczematous rash at the point of contact with the dressing. Some patients may also develop an eczematous rash in distant sites such as the trunk or arms as a result of auto-sensitisation dermatitis (also called an “id reaction”).

### 37.7 Compression Bandaging

Bandaging skills are best learned by clinical demonstration. Doppler ankle pressure should be checked if there is any risk of PVD. After the ulcer is dressed appropriately, a foam dressing, or a few layers of gauze should be placed over the primary dressing to absorb exudates. This will also protect the wound and concentrate the force of the compression bandage to the ulcerated side of the limb.

The leg should then be bandaged from the toes to just below the knee with an elasticated compression bandage such as “Actico<sup>®</sup>” + “FlexiBan<sup>®</sup>” padding (made by L&R Medical UK Ltd), “Coban 2<sup>®</sup>” (made by 3M) or “SurePress<sup>®</sup>” High Compression bandaging over a “SurePress<sup>®</sup>” Adsorbent Padding (made by Convatec).

The bandages should be applied with equal pressure up the limb which gives maximum compression pressure at the ankle (30 mm–40 mm/HG) (Fig. 37.6). Adsorbent padding can be used to protect pressure points and to build up the ankle circumference to between 18 and 25 cm if it is much smaller than the calf [3]. Compression bandaging can be painful in the first week. Analgesics and sleeping tablets may be necessary during this time. Diuretics may also be required in the first few weeks, to reduce oedema. Patients should be instructed to elevate their leg at least 30 degrees above the horizontal for 2 hours morning and evenings. They should avoid standing in the one spot. Walking should be encouraged while in compression bandaging or hosiery to activate the muscle pumps and encourage venous return.

Healing ulcers should be interfered with as little as possible. The dressing should be reapplied when the bandage becomes soaked with exudate or becomes malodorous. This is usually once or twice a week. Progress should be monitored by measuring or photographing the wound periodically. Skin grafting techniques such as pinch grafts may be necessary on slow to heal wounds.

**1. Measure**

Measure the ankle. Reshape/protect the limb (if necessary) by applying suitable sub-compression padding such as FlexiBan® padding.

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**2. Secure**

Secure padding with two turns at the base of the toes and with a figure of eight at the ankle before using a 50% overlap spiral up the leg to 2 cm below the knee/popliteal fossa. Re-measure the ankle.

---

**With an ankle circumference of 18 cm–25 cm after padding:**

**3. Check**

Before applying Actico® 10 cm check that the foot is correctly positioned ‘toes to nose’ to maximise ankle movement. Hold Actico® with tension and apply two turns from the base of the toes. Roll Actico® across the dorsum keeping tension. An extra turn may be applied in the middle of a long foot

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**Fig. 37.6** Application guide for Actico short stretch compression bandaging (Used with the permission of L&R Medical UK Ltd)



#### 4. Enclose

Enclose the ankle and heel with a figure of eight

Start to spiral up the leg using the 'Lock & Roll' method (50% overlap – Full stretch). Roll the Actico® round the back of the leg whilst ensuring it is locked out at full stretch. Continue with the 'Lock & Roll' method up the limb in a spiral



#### 5. Finish

Finish the Actico® 3 cm below the popliteal fossa. Cut off excess bandage and tape if necessary. Ensure layers are bonded.

**With an ankle circumference more than 25 cm after padding:**



#### 6. Repeat

Apply a second Actico® 10 cm in the opposite direction. Secure with two turns with tension over the ankle and on the third turn follow the 'Lock & Roll' method as before in Steps 4 & 5. Finish as before.

**Fig. 37.6** (continued)

## 37.8 Compression Stockings (Hosiery)

Patients with varicose ulcers should be assessed for suitability for either sclerotherapy or ligation and stripping of the veins. Once the ulcer is healed all patients should be measured and fitted for medical compression stockings (hosiery) which provide 30 mm–40 mm of compression at the ankle (e.g. “Mediven Plus®” compression class 2 or “Alleviant Ulcer Care Kit®” by Jobskin, or “Activa Compression Hosiery”). Sizing can be judged by measuring the smallest diameter at the ankle and the largest diameter at the calf. The correct size can then be chosen from the manufacturers table using these measurements. Compression hosiery should not be used if there is any arterial insufficiency.

Patients who have healed their varicose ulcer should wear compression stockings for the rest of their life even if they have had vein surgery. The vast majority of patients can manage with stockings that go to the knee even if the varicose veins are palpable above the knee. Below knee stockings are easier to put on and off and are more comfortable. Most patients should buy two pairs; they can be washing one pair while wearing the other. Both pairs should be discarded after 100 washes (approximately 6 months) and replaced with new pairs as the elastic will degrade as the stockings are repeatedly washed. There are various techniques for assisting with putting on and

taking off the stockings, as this can be difficult especially for the elderly with weak hands or arthritis (“Medi Butler®”, “Easy Slide®”, “Actiglide Application Aid®”).

## 37.9 Conclusion

When dealing with leg ulcers, it is important to make an accurate diagnosis as the treatment will be dependent on the underlying cause. Most leg ulcers in primary care are caused by venous hypertension from varicose veins. With appropriate primary wound dressings, compression bandaging and a holistic approach to patient management (e.g. good diet, no smoking, exercise, weight management, etc.) most varicose ulcers less than 20–30 mm should heal in 6–12 weeks. Small ulcers (<10 mm) with mild varicose veins may heal with compression stockings, without the need for compression bandaging.

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3. [https://www.youtube.com/watch?v=en\\_dr0ErC2o](https://www.youtube.com/watch?v=en_dr0ErC2o)



# Wound Care

38

David Buckley

## Key Points

- To optimise wound healing the patient's general health needs to be assessed to rule out any underlying causative or aggravating factors such as diabetes, anaemia, hypothyroidism, congestive cardiac failure, autoimmune disease, smoking, nutritional deficiency, skin cancer, pressure or venous hypertension.
- Wound dressing will vary according to the cause of the wound, the stage of wound healing, the amount of exudate and whether there is infection present or not.
- If there are clinical signs of infection such as pain, fever, malodour, pus, tenderness and redness, systemic antibiotics will almost always be necessary.
- If a wound is not healing despite adequate care, an underlying malignancy such as an SCC, a BCC or a melanoma may be present and a skin biopsy may be necessary.

## What to Tell the Patient

- If you have a slow to heal wound please ensure you are eating well and maintain a healthy weight.
- Rest and elevation with help wound healing.
- Avoid smoking and non-steroidal anti-inflammatory drugs (NSAIDs) as they delay wound healing.

- A biopsy might be needed to be taken to understand the cause of an ulcer.

## 38.1 Introduction

When presented with a patient with a wound it is important to find out the cause, establish the stage of wound healing and create an environment to optimise wound healing. Sometimes it may be obvious what caused the wound such as a pressure sore, a burn, trauma or a surgical wound. Other times it may be more difficult as the ulcer may have developed spontaneously. Certain investigations may be necessary to establish the cause and to deal with it. For example an ulcer on the lower leg could be due to venous insufficiency, peripheral vascular disease or a skin cancer (Fig. 38.1). The management of each situation would be different [1].



**Fig. 38.1** Moderately differentiated squamous cell carcinoma on the lower leg for 9 months

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It is important to look beyond the wound in an attempt to optimise wound healing in a holistic way. The patient's general health needs to be assessed to rule out any underlying contributing or aggravating factors such as diabetes, anaemia, hypothyroidism or other chronic conditions such as chronic renal failure, chronic liver failure, congestive cardiac failure, haematological disorders, autoimmune disease or nutritional deficiency (Table 38.1). All patients with chronic wounds should be encouraged not to smoke as this will invariably delay wound healing. Likewise, non-steroidal anti-inflammatory medication should also be avoided if possible as they too will delay wound healing.

The "TIMES" acronym was developed by international experts in wound management as a framework for a structured approach to wound bed preparation and to optimise wound healing [2] (Table 38.2).

**Table 38.1** Investigation of chronic or non-healing wounds

- Full blood count
- Erythrocyte sedimentation rate
- Urea and electrolytes
- Liver function tests
- Glucose
- Thyroid function tests (TFT's)
- HBA1C
- B12, Folate, Ferritin
- Antinuclear factor
- Rheumatoid factor (RF)
- Urinalysis
- Doppler ankle/arm pressure
- Skin biopsy (if malignancy suspected)
- Patch test (if contact allergic dermatitis suspected)
- Send tissue, exudates or swab for C + S if clinically infected

**Table 38.2** TIMES Acronym for assessing wounds

- |  |  |
|--|--|
| T = Tissue—is it sloughy or necrotic                     |  |
| I = Infection  |  |
| M = Moisture (dry or moist)                              |  |
| E = Edges (=advancing, static or getting smaller)        |  |
| S = Surrounding skin (=healthy, eczematous or macerated) |  |

## 38.2 Tissue

Wound care is an important but often confusing area especially in primary care where the nurse or doctor may have limited access to the vast array of dressings that are available. It is probably best to be familiar with a limited range of easily available dressings that will be suitable for the majority of wounds one is faced with in primary care. The wound dressing will vary according to the cause of the wound, the stage of wound healing, the amount of exudate and whether there is infection present or not. (Tables 38.3 and 38.4) [1]. A careful assessment of the wound should be carried out at the beginning of treatment and periodically during the course of wound healing as different dressings may be required at different stages of wound healing. Measuring the size of the wound, drawing a stencil of the wound outline and/or photographing the wound are very useful ways of judging the speed of wound healing (Fig. 38.2).

### 38.2.1 Post-operative Wound Dressings

After simple skin surgery in uncomplicated cases, wounds can often be left uncovered especially on the face and scalp where it might be difficult to apply dressings. If there is light bleeding or oozing from a wound post-operatively then a simple non-adherent island dressing such as "Primapore®" or "Opsite Post-Op®" or "Melolin®" may be all that is required. Some doctors like to apply an antiseptic powder such as chlorhexidine or an antiseptic spray (e.g. "Opsite Spray®") to the healthy surgical wound before applying a non-adherent dressing.

### 38.2.2 Epithelializing Wounds

If the wound is healthy, non-infected and has little or no exudate, a low adherent, porous, knitted viscose dressing such as "Profore

**Table 38.3** Description of dressing

Type	Example	Indication	Advantage	Disadvantage	Contra-indications
Island dressing, Slightly absorbent, non-adherent with adhesive tape	Primapor® Opsite Post-Op®	Post-op + low exudating wounds	Absorbs Sterile barrier	Low absorbent Allergy to adhesive	Moderate or heavy exudate
Non-adherent, Moist tulle gras dressings	Chlorhexidine = Bactigras® or povidone-iodine = Iridine® or Iodoflex®	Wounds healing by secondary intention	Non-stick, Moist. Antibacterial Iodine good for over granulation	Not absorbent May promote hyper-granulation Requires secondary dressing	Allergy
Non-adherent dry dressing	Tricotex® Porefore WCL® Melonin®	Low exudate wounds, healing by primary intention	Non allergic	No absorption, Requires secondary dressing	Sticks to wound
Calcium alginate from seaweed	Curasorb® Kaltostat®	Exudating or bleeding wound	Forms moist gel Haemostatic Reduces pain Packs cavity	Requires secondary dressing	Dry wound or hard eschar
Hydrogel absorbent polyurethane + 60% water	Hydrosorb gel sheet®	Debrides wounds	Transparent Keeps wound moist Non adherent	Not if heavy exudate	Infected wounds third degree burns
Polyurethane foam dressing	Lyfofoam® Polymen® Allevyn® Biatain®	Mild to moderate exudates	Moist Absorbent Barrier	Can be used as a primary or secondary dressing	Dry wound or hard eschar Allergic infection
Hydrocolloid dressing	Garnuflex® Duoderm® Comfeel®	Burns and small abrasions	Moist	Low absorbent	Not if infection Not if muscle tendon or bone exposed
Hydrofibre	Aquacel®	Necrotic sloughy wounds + healing by secondary intention	Form soft moist absorbent gel	Secondary dressing needed	Dry wounds
Odour absorbent	Actisorb silver® Clinisorb®	Malodorous wounds	Mask odour	Clinisorb requires primary dressing	Do not cut
Silver dressings	Actisorb silver® Acticoat silver® Aquacel Ag®	Infected wounds	Kills pathogens including MRSA + VRF	May become resistant Acticoat silver = activate with sterile H <sub>2</sub> O	Allergy can be toxic
Negative pressure wound therapy	Vacuum assisted closure device E.g.: VAC dressing® or PICO® single use suction dressing	Difficult to heal wounds	Blood flow Oedema Discharge Moist Protective barrier	Requires power	Nor for osteomyelitis or malignant wounds

**Table 38.4** Wound dressing selection chart

Wound bed description	Aim of care	Exudate	Primary dressing	Secondary dressing
Post-op	Promote healing by primary intention	Low Moderate	Primapore® Opsite Post-op®	— —
Infected wound	Reduce bacterial burden	Low Moderate	Bactigras®, Iodine® or Iodoflex® Acticoat Sivler®	Surgical gauze Or Lysofoam
Exuding or bleeding wound	Absorb exudate + maintain moist wound environment	Moderate Heavy	Granuflex®, Duoderm® or Comfeel® Actisorb Silver® or Kaldostat® or Curasorb®	Surgical gauze Or Lysofoam
Over granulating wounds	To reduce granulation	Low to heavy	Silver nitrate stick + Biatain® or iodine dressing	Nothing or surgical gauze
Epithelializing wounds	Promote maturation	Low Moderate	Tricotex® or Profore WCL® or Duoderm® Comfeel® or Biatain®	Surgical gauze Or Lysofoam
Malodorous wounds	Reduce odour + may need systemic antibiotics	—	Actisorb silver® Acticoat silver® Metronidazole gel or iodine®	Clinisorb
Yellow sloughy wounds	May need surgical debridement	Low Moderate Heavy	Comfeel® Curasorb® Aquacel®	Surgical gauze Or Lysofoam
Black leathery necrotic wounds	Debride + moisten	Low	Hydrocollide (Comfeel®) or hydrogel (Hydrosorb®) or hydrofiber (Aquacel®)	Surgical gauze Or Lysofoam
Difficult to heal wounds	Rule out infection + malignancy	Moderate to heavy	VAC® dressing	By tissue viability nurse
Burns	Cool, moisten, adsorb exudate	Moderate	Cooljel® sheets or gel or Mepitel® or Adaptic®	Surgical gauze Or Lysofoam

WCL® (“wound contact layer”) or “Tricotex NA®” (non adherent) may be suitable and this should be covered by a secondary absorbent dressing such as sterile surgical gauze or “Lysofoam®”. “Trioctex®” is non-allergic and has a very low potential to cause sensitisation. “Mepitel®” or “Adaptic®” are also useful in this situation.

These are dry dressings whereas a moist wound environment has been shown to increase epithelialisation and stimulate proliferation. Hydrocolloid dressings such as “Duoderm®”, “Comfeel®” or “Granuflex®” can be used if there is low exudate as they form a gel that provides a

moist wound environment. They are not suitable if there is infection, where tendons, muscles or bones are exposed or if the wound requires frequent changes.

### 38.2.3 Over Granulating Wounds

These are usually healthy, clean, uninfected wounds but the granulation tissue is too prominent and may slow healing. “Biatain®” is a polyurethane foam dressing which is significantly more absorbent and less likely to leak and more cost effective than “Allevyn®”. It can be useful

**Fig. 38.2** Varicose ulcer

for granulation or epithelising wounds with light to heavy exudate and may be left in place for up to seven days. It is not suitable for dry wounds, necrotic wounds or hard eschar. Silver nitrate caustic pencils moistened with water and dabbed onto the wound daily for 3 days may also help. Iodine wound contact layer such as "Inidine®" or "Iodoflex®" may also help reduce the development of excessive granulation tissue.

#### **38.2.4 Malodours Wounds**

Activated charcoaled ("Clinisorb®") helps to absorb odour but cannot be applied directly to the wound. It should be used as secondary dressing only as it will be deactivated if it comes in contact with exudate or becomes wet. Odours are usually produced by a bacteria including anaerobes such as bacteroides and chlrostridium species and aerobic bacteria including proteus, klebsiella and pseudomonas species. Metronidazole orally or in a gel formulation may help deal with anaerobes. "Actisorb Plus®" or "Acticoat Silver®" or "Aquacel Ag®" are good for infected, malodorous wounds and can be used as a primary dressing.

#### **38.2.5 Yellow, Sloughy Wounds**

This may be a sign of a biofilm which is bacteria and other organisms embedded in a thick, slimy, yellowish, jelly barrier of sugar and protein (Fig. 38.3). The biofilm protects the microorganism from external threats such as antibiotics and delays

**Fig. 38.3** Varicose ulcers with a biofilm

wound healing. The best way to deal with biofilm is by regular manual debridement. Broad spectrum microbiocidal antimicrobials such as silver, iodine, chlorhexidine or honey dressings may prevent the reestablishment of the biofilm [3].

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### **38.3 Infections**

The body has a fantastic capacity to heal wounds and if a wound is not healing or is getting worse then every effort should be made to find out why. Apart from not dealing with underlying causes, the most common reason a wound will not heal is infection, usually bacterial. Swabbing an open wound will nearly always grow organisms but they may not be aggravating the wound and could be merely commensals. It is far more important to look for clinical features of infections such as pain, tenderness, redness in the surrounding skin adjacent to the wound or ulcer or delayed healing. Many wounds will ooze and have exudate which is not necessarily a sign of infection unless frank pus is present. The odour from the wound may sometimes alert one to the presence of infection such as anaerobes. A green discharge may suggest pseudomonas.

If there are clinical features of wound infection then a swab should be taken to try and identify the pathogen and to rule out resistant bugs such as MRSA or pseudomonas. If there are clinical signs of infection, systemic antibiotics will be almost always necessary as topical antibiotics or antiseptics would rarely be sufficient. The choice of antibiotic, the delivery route, the dose and the duration can vary from case to

case but if there are signs of cellulitis in the surrounding skin then *staph aureus* or *staphylococcus pyogones* are the most likely organisms and most will respond to high dose flucloxacillin either orally or systemically for at least ten to fourteen days. If the patient is systemically unwell (febrile, feverish, hot and cold) then they will more than likely require intravenous antibiotics. For more severe infections benzoyl penicillin may be added to flucloxacillin to ensure adequate coverage. Swabs may establish which organism is involved and there sensitivities to various antibiotics. If anaerobes are isolated metronidazole may be necessary either topically or orally.

**Bleach (“Milton®”) baths** (see Chap. 13 on atopic eczema in children) may help clear skin pathogens, especially *staph aureus*, in people with recurrent infective exacerbations of their atopic eczema.

**Potassium permanganate** crystals is an oxidising agent and acts as a disinfectant. A dilution of 1:10,000 in water (light pink colour) is very useful for blistering eruptions such as pompholyx or infected tinea paedis, especially if it is associated with bacterial or fungal infections. Potassium permanganate comes in crystals or tablets (“**Permitabs®**” 400 mg = one tablet to 4 litres of boiled cooled water). The affected area should be soaked in the solution for 5 minutes. Patients should be warned it will stain the skin and nails brown for a few weeks.

Resistant organisms such as *pseudomonas*, multi resistant *Staphylococcus aureus* (MRSA) or *Vancomycin-resistant Enterococci* (VRE) can prove difficult to treat and may require the input of a tissue viability nurse or a microbiologist. Even in the presence of MRSA, wounds will often heal once wound healing is optimized by dealing with the underlying cause, ensuring the patient is as healthy as possible and applying appropriate wound dressings. Obviously, great care needs to be taken when assessing and treating patients with infected wounds, especially MRSA, to ensure the patient or the healthcares do not transfer the pathogens to other patients (see Table 38.3 on MRSA management).

It is important to bear in mind that if a wound is not healing despite adequate care an underlying

malignancy such as a SCC, a BCC or a melanoma may be present and a skin biopsy from the edge of the wound may be necessary. Punch biopsies may be sufficient but if using a three or four millimetre punch, a number of samples may need to be taken from the edge of the ulcer at different sides. An elliptical biopsy from the edge of the ulcer including some involved and unininvolved skin is ideal but may be more difficult to carry out especially in the lower leg or in the very elderly as the surgical scar may prove difficult to suture and may break down in the presence of infection.

Prophylactic oral or systemic antibiotics may be required in special circumstances such as **animal bites or human bites**. The organisms involved include a mixed anaerobes and streptococcal. Gram negative coccus and *Pasteurella multocida* are also common. Any penetrating injury should be explored, irrigated and delayed closure may be necessary. Antibiotic cover is usually mandatory and co-amoxicilav (625 mg three times a day for adults) is the antibiotics of choice for initial prophylaxes or the treatment of established infection before culture and sensitivity results are available. Tetanus prophylaxes and rabies treatment may have to be considered for animal bites. Post-exposure prophylaxes for hepatitis B and HIV should be considered for human bites.

### **38.3.1 Wound Dressings for Infected Wounds**

As mentioned previously, systemic antibiotics may be necessary. An appropriate wound dressing may also help fight infection and promote wound healing. Chlorhexidine is a broad spectrum antiseptic that rarely causes contact sensitivity and resistance is rarely a problem. “**Bactigras®**” (made by Smith and Nephew) is a tulle gras dressing containing chlorhexidine that can be safely placed over the wound in one or two layers and is easy to remove at the next wound dressing session. “**Inadine®**” is a similar tulle gras dressing containing povidone iodine which is an excellent antiseptic. “**Iodoflex®**” is a paste containing iodine which can also be placed over a wound or in cavities. Iodine dressings should be

replaced once the rusty brown colour starts to fade. A maximum of three sheets should be used at any one time. Care needs to be taken when using iodine dressings in children, pregnant woman, in patients with hypothyroidism and patients on lithium. The biggest problem with iodine dressings is that it can sting and be painful, especially for the first twenty-four hours of application. Some patients can be sensitive or allergic to iodine. All tulle gras dressings and pastes will have to be covered with a secondary wound layer such as sterile surgical gauze or a multilayer absorbent dressing which will absorb exudate (e.g. "Lyofom®" or "Lyofom extra®"). This can then be fixed to the skin with hypoallergic tape, tubular dressings or bandaging (see Table 38.4).

Compression bandages or compression stockings from the toe to the knee can be applied over the secondary wound layer for varicose ulcer once the peripheral vascular disease is ruled out by checking the ankle BP using a vascular doppler.

For persistent ulcers, especially when complicated with resistant organisms, a silver impregnated dressing may help such as "Actiborb Silver®", "Acticoat®" or "Aquacel Ag®". These dressings are a lot more expensive than simple tulle gras dressings and will also need a secondary wound layer. The silver binds with the organisms and provides up to seven days of broad spectrum anti-microbial action even against resistant organisms such as MRSA, VRE and pseudomonas. These dressings also absorb fluid and maintain a moist homed environment. They may need a secondary wound layer such as surgical gauze or a foam dressing such as "Lyofom®". The antimicrobial barrier properties of "ACTICOAT®" remain effective for a minimum of 3 days. "ACTICOAT 7®" remains effective for a minimum of 7 days. These dressings should be moistened with sterile water (do not use saline) which should be allowed to soak in for 2 minutes before applying the dressing to the wound surface, either side down.

MRSA may also respond to mupirocin cream ("Bactroban®") twice a day for 14 day. Munuka honey with a high UMF that is specifically made for wound care may also help clear MRSA.

## 38.4 Moisture

### 38.4.1 Heavily Exudating or Bleeding Wounds

Obviously what is needed here is a dressing with a lot of soakage that does not stick to the wound. Polyurethane form dressings such as "Lyofom®", "Polymem®" or "Allevyn®" can be used as a primary dressing but the doctor needs to read the instructions carefully to ensure the correct side of the foam dressing goes against the wound. Foam dressings can be used as a primary or secondary dressing.

Calcium alginate dressings such as "Curasorb®" or "Kaltostat®" are derived from a natural polysaccharide in seaweed and form a gel when they come in contact with the wound keeping the environment moist. They also absorb exudate. They usually require a secondary dressing.

"Aquacel®" is a soft, dressing composed of hydrochloride fibres (sodium carboxymethyl-cellulose) used in the management of heavy exuding wounds and is 20% more absorbent than alginates. As "Aquacel®" absorbs exudate, it converts from a dry dressing to a soft gel sheet. It can be covered with a foam dressing or sterile surgical gauze and can be left on for a maximum of seven days. "Aquacel®" should only be used on heavily exuding wounds; otherwise the dressing will adhere to the wound. If this happens, irrigation with 0.9% sodium chloride will facilitate removal. For low exuding wounds try a hydrocolloid dressing such as "Comfeel®" or an alginate dressing such as "Curasorb®".

### 38.4.2 Dry, Black, Necrotic, Leathery Wounds

These wounds may need surgical debridement of thick eschars which may require topical, local or general anaesthetic. Do not debride black heels or toes without first getting a vascular surgeon assessment as there is often an underlying vascular insufficiency. A hydrocolloid dressing such as

“Comfeel®” or a hydrogel such as “Hydrosorb®” may help. A hydrofiber dressing such as “Aquacel®” might also work.

## 38.5 Edges

If the edge of the wound is extending outwards or is static it is usually a sign of suboptimal wound care. There may be infection, poor choice of wound dressings, skin cancer or failure to deal with underlying issues such as diabetes, malnutrition pressure or venous hypertension. On the other hand a contracting wound edge and a shrinking wound is a good sign of optimal wound and patient care. In surgical debridement, some bleeding on the edge is a good sign.

## 38.6 Surrounding Skin

If the skin surrounding the wound is unhealthy it will delay wound healing. Common causes are varicose eczema which can be managed with emollients, soap substitutes, a moderately potent or potent topical and compression bandaging or hosiery provided there is no peripheral vascular disease. Maceration from heavy exudates can damage surrounding skin and should be managed with adsorbent dressings as mentioned above under “moisture”. Infection may also spread to the surrounding skin (cellulites) which should be dealt with urgently to avoid further skin breakdown.

If there are signs of solar damage in the surrounding skin (e.g. solar elastosis, solar lentigo, actinic keratosis) it makes it more likely that the wound or ulcer may be malignant in origin.

## 38.7 Negative Pressure Wound Therapy

These electronic suction devices provide negative pressure to the wound bed via an open cell foam sponge placed in the wound and secured, air tight, with adhesive tape. They promote localised blood flow, reduce oedema, and promote granulation

and epithelialisation. Vacuum assisted closure (“VAC®”) devices are usually only available in specialised surgical units and are usually fitted and supervised by specially trained tissue viability nurses. “PICO®” is another battery operated wearable suction dressing available from Smith and Nephew. They are suitable for difficult to heal wounds. Necrotic wounds with thick eschar need to be debrided before a “VAC®” dressing as the “VAC®” would dry out the wound and make the eschar more difficult to remove.

## 38.8 Conclusion

The most important aspect of dealing with a wound is to ensure there is no infection or malignancy and to deal with any underlying problems the patient may have such as poor nutrition, smoking, obesity or poorly controlled diabetes. The cause of the wound should also be addressed (e.g. pressure sores need positional variation and padding; varicose ulcer need compression bandaging or hosiery).

There is not one dressing that will suite all wounds. In primary care, most wounds will do well with a primary contact layer such as chlorhexidine (e.g. “Bactigras®”), povidone-iodine (e.g. “Iodine®” or “Iodoflex®”) or a non adherent layer (e.g. “Tricotex®”). A foam dressing (e.g. “Lyofoam®”) or cotton gauze makes a good secondary layer if there is exudates. For an excellent review see Mary Martin’s YouTube video [4].

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# The Red Leg

39

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## Key Points

- There are a number of conditions that are more commonly found on the lower leg mainly because of gravitational effects or venous hypertension.
- An *itchy* red lower leg may be caused by eczema/dermatitis, psoriasis or tinea infection.
- A *painful* red lower leg can be more sinister and may be caused by infection, an ulcer or a DVT (Deep Vein Thrombosis).

## What to Tell the Patient

- Rest and elevation is important when managing a red leg.
- If your red leg becomes painful please contact your doctor immediately as you may be developing an infection or a clot.

## 39.1 Introduction

Patients often present to their doctor with one red leg. There are a number of conditions that are more commonly found on the lower leg mainly because of gravitational effects or venous hypertension. Rashes on the lower leg may be part of a more generalised skin problem that happens to be

more prominent on the lower leg such as atopic eczema or psoriasis.

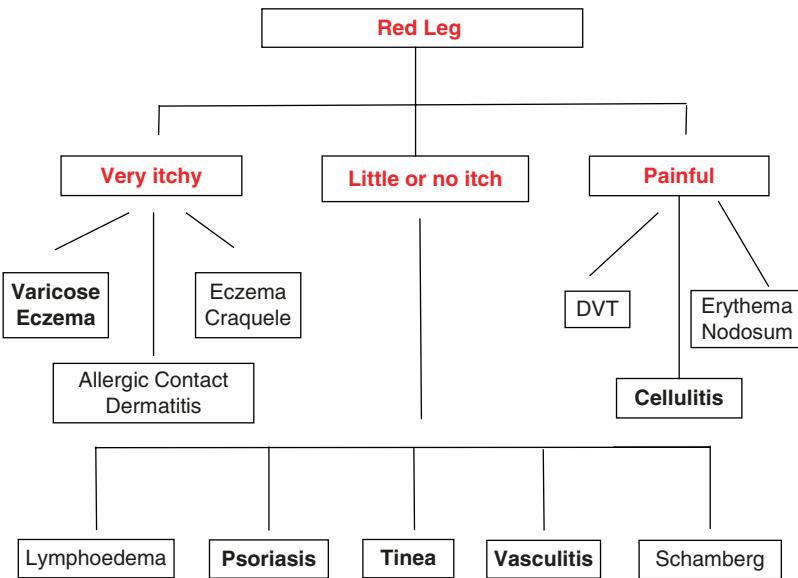
If both legs are red then the diagnosis may be due to a generalised skin condition such as psoriasis or atopic eczema, a systemic disease such as vasculitis or directly related to venous hypertension such as varicose eczema. One leg may be more severely affected than the other. More often than not, the patient will present with one red leg. A careful history will often help to clinch the diagnosis (Fig. 39.1 and Table 39.1). In particular, it is important to establish if the red leg is itchy or sore. It is also important to assess the general health of the patient and in particular to check for a fever and look for any possible underlying precipitating factors such as varicose veins, diabetes, thyroid disease, cardiac failure, peripheral vascular disease, pregnancy or an abdominal tumour which may compromise the venous return in the legs.

## 39.2 Itchy Red Leg

Itch usually implies that there is eczema or dermatitis. It is usually accompanied by a dry, red, scaly rash. By far the most common cause is **varicose eczema** which occurs as a direct result of

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**Fig. 39.1** Causes of red leg



**Table 39.1** Other skin conditions that are more likely to occur on the lower legs and may cause redness include the following

- Actinic keratosis
- Bowens disease
- Nacobiosis lipoidica
- Pyoderma gangrenosum
- Pretibial myxoedema
- Prurigo nodularis
- Lymphoedema

varicose veins secondary to venous hypertension. The veins may not always be obvious because of the rash. Standing the patient up and examining the whole leg from the groin down usually reveals the underlying cause.

Like all eczemas, the dry skin will need moisturising and the patient should be instructed that a greasy moisturiser should be applied to hairy legs in a downward direction to avoid folliculitis. Advise to avoid soaps and other irritants. Wet wraps or paste bandages can sometimes be helpful on the lower leg. Potent topical steroids applied for 1 to 2 weeks can usually relieve the itch and should dramatically improve the rash. A patient should be advised to avoid topical antibiotics, topical anti-histamines and products rich in colourings and preservatives, because patients with varicose eczema have a defective skin barrier and are prone

to developing **allergic contact dermatitis**. Some patients with more severe varicose eczema or contact dermatitis may develop a more generalised itchy rash on other parts of the body due to an id reaction (auto-sensitisation dermatitis). If the eczema is infected (weepy, crusty or sore) then a systemic antibiotic maybe necessary for 1 to 2 weeks. The most important part of the management of patients with varicose eczema is to treat the underlying cause which is the varicose veins and venous hypertension. It is best dealt with by either compression hosiery (provided there is no evidence of peripheral vascular disease) or varicose veins surgery (see Chap. 37).

**Asteatotic eczema** (eczema craquelé) is also known as xerotic (dry) eczema. There is a distinctive rash, most commonly found on the lower legs in elderly patients. The skin is dry and there usually is a network of superficial cracks, like cracks in a porcelain cup or crazy paving, hence the name (craquelé). It is usually itchy and worse in the winter and in situations of low humidity such as sitting too close to a hot fire or having the room too hot. It can be aggravated by smoke and other irritants which further dry out the stratum corneum. There may be excoriation from scratching. Diagnosis is usually clinical and the treatment involves the application of greasy moisturisers in a downward

direction. Soaps and other irritants should be avoided. A potent topical steroid maybe required for 1 to 2 weeks in more severe cases.

### 39.3 Red Leg with Little or No Itch

**Psoriasis** can have a predilection for the lower legs in some patients, particularly if there are underlying varicose veins which can irritate the skin and result in a Koebner phenomenon which causes the psoriasis to break out in irritated skin.

**Tinea corporis** can occur on any part of the body and if inadvertently treated with a potent topical steroid it can cause a more widespread red, scaly rash and the annular raised border that usually occurs with ringworm may be absent because of the use of topical steroid (tinea incognitio). It is usually unilateral, asymmetrical and skin scrapings for fungal stain and culture should help clinch the diagnosis.

**Schamberg Disease (Capillaritis)** is relatively common and usually presents as a reddish, brown, **cayenne** peppered discolouration of the lower legs especially around the ankles and the dorsum of the foot. It occurs mainly in young men who are otherwise healthy and asymptomatic. More extensive cases can cause a macular, non scaly, symmetrical, bilateral reddish brown rash spreading up the shins. There is usually little or no itch. The aetiology is unknown but it is caused by capillaritis leading to capillary leakage. Occasionally it can be drug induced (e.g. amlodipine, aspirin, diuretics). It can appear after prolonged or vigorous exercise, especially during warm weather. Treatment is usually not necessary and most cases will resolve spontaneously in time.

**Vasculitis** is a rare condition caused by inflammation of the blood vessels (Table 39.2). This normally presents as a raised purpuric rash (palpable purpura) with or without ecchymoses (bruising) which can occur on any part of the body but has a predilection for the lower legs. Mild cases may have little or no symptoms whereas severe cases can cause itch, pain, necrosis and ulceration. It is usually symmetrical and can be more generalised (see Chap. 54).

An acute form of vasculitis (**leukocytoclastic vasculitis**) can present suddenly and can be asso-

**Table 39.2** Causes of vasculitis

Infective causes
Auto immune
Compliment mediated
Drug induced (antibiotics, anti-coagulants, NSAIDs, thiazides)

**Table 39.3** Investigation of vasculitis

Full blood count
Urea and electrolytes
Liver function tests
Erythrocyte sedimentation rate
C-reactive protein
Hepatitis B & C screen
Auto antibody screen
Urinalysis
ASOT
Chest X-ray
Immunoglobulins
Cryoglobulins
Anti neutrophil cytoplasmic antibodies
Skin biopsy

ciated with constitutional symptoms such as fever, abdominal pain, malaise and arthralgia.

**Henoch-schonlein purpura** is a form of acute vasculitis seen in children and can cause renal damage in up to 50% of cases.

**Sub Acute Vasculitis** can look more like urticaria but the rash can remain in the one area for days rather than hours which occur in classical urticaria (**urticular vasculitis**). This chronic form of vasculitis can last weeks or months with no constitutional symptoms.

With the acute form of vasculitis it is very important to rule out **meningococcal septicaemia** especially, if the rash suddenly appears, is asymmetrical and associated with constitutional symptoms or a fever.

Treatment of vasculitis will depend on the underlying cause (Table 39.3). Most cases of vasculitis will need specialist referral for further investigation and management.

### 39.4 Painful Red Leg

**Cellulitis and Erysipelas** can occur on any part of the body but is relatively common on the lower leg. The organisms that cause cellulitis normally need a portal of entry to penetrate into the deeper layers of the skin. This can arise if there are fis-

sures or scratch marks from itchy eczema or from tinea infection. The infection usually causes a red, hot, tender rash that spreads up the leg over a matter of a few hours to a few days. The patient may have flu-like symptoms and a fever. Milder cases may respond to oral flucloxacillin combined with penicillin v and elevation. More serious and well established cellulitis will probably need treatment with intravenous flucloxacillin and benzoyl penicillin. (see Chap. 30). Recurrent cellulitis may respond to phenoxyethylpenicillin tablets 250 mg twice a day for 12 months.

**A Deep Venous Thosmbosis (DVT)** often presents as a painful, tender calf muscle. There is sometimes swelling, redness and heat in the affected area. There are usually no obvious skin changes. DVTs can occur in conjunction with other skin problems such as varicose veins and varicose eczema. This can make the diagnosis more difficult. A history of immobility, recent flights, recent surgery or drugs that can predispose to clotting, such as the oral contraceptive pill would make the diagnosis of a DVT more likely.

A d-dimer blood test is a sensitive but non-specific test for a DVT. A raised level is suggestive (though not conclusive) of a DVT. A negative test suggests a low possibility of DVT and rules out the possibility of a DVT in up to 97% of cases. Doppler ultrasound or venogram is the best way to confirm the diagnosis.

**Erythema nodosum** is a panniculitis that causes red, tender, hot plaques usually on the shins. It may be associated with a flu-like illness and can have many causes including infections, pregnancy, drugs, sarcodiosis, etc (See Chap. 54).

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### 39.5 Conclusion

A red leg may be caused by a serious, potentially life threatening problem, particularly if associated with pain or fever. All cases need thorough investigation to find out the underlying cause. Urgent referral may be required for serious problems such as cellulitis or a DVT.

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## **Part VIII**

### **Hair and Nail Problems**



# Hair Loss and Hair Growth

40

David Buckley

## Key Points

- Hair loss can be devastating for some patients.
- By far the most common form of hair loss is male or female patterned hair loss (androgenic alopecia) which often runs in families.
- Diffuse hair thinning all over the scalp may indicate a systemic problem, a deficiency or may be drug related.
- Patchy hair loss may be due to alopecia areata.
- If there is a scaly scalp associated with hair loss it usually implies a skin condition such as psoriasis, eczema, or a fungal infection.
- Scarring alopecia is usually due to a deep seated skin problem and often requires a skin biopsy for diagnosis. Treatment should be initiated quickly, before the alopecia becomes more widespread, as it is usually irreversible once scarring develops.
- Sudden onset patterned or severe hair loss or hirsutism may indicate an underlying hormonal problem and will need thorough investigation.
- Treatment of alopecia and hirsutism is dependent not only on the efficacy, but also on practicability, safety, patient preference and cost.

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## What to Tell the Patient

- Male patterned hair loss is a natural phenomena for many men and most men learn to cope with it eventually.
- Don't believe all the hype you read about cures for baldness in the ads in the newspapers or on the internet—the vast majority are not true.
- Minoxidil ("Regaine Scalp Foam®") is one of the very few treatments approved for patterned hair loss in men and women but it usually only helps you hold onto what you have. Significant re-growth is unusual.
- Hair transplants using follicular transfer can produce dramatic results but is too expensive or too invasive for many people.
- Laser hair removal is safe and effective especially for those with dark hair and light coloured skin.

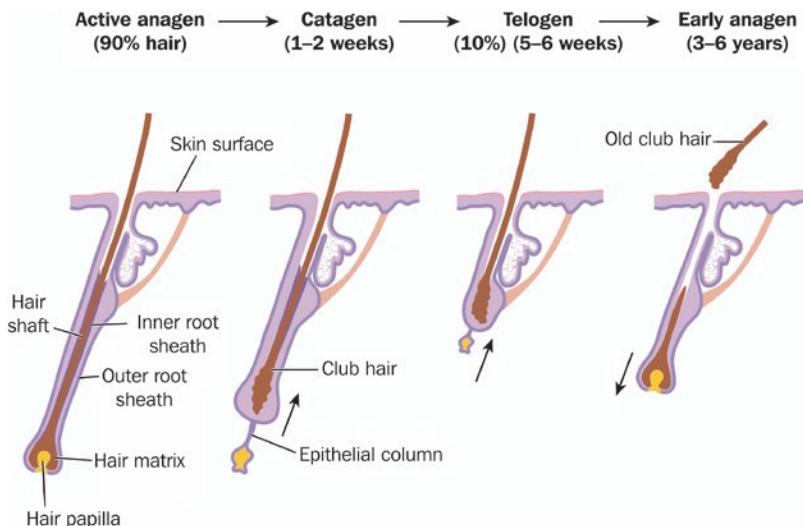
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## 40.1 Introduction

Alopecia is the Latin word for hair loss. There are many different types and causes.

The rate of hair growth and loss is dependent on many factors, including genetics, race, sex, age and hormonal influences. Too much hair in the wrong place or too little in the right place can cause severe psychological distress for some people. The word 'baldness' should be avoided when talking to patients. 'Hair loss' or "hair thinning" are much kinder expressions. When seeing a patient with hair problems, it is important to assess their ideas,

**Fig. 40.1** Cycle of hair growth. Copyright: PeterLamb@123RF.com. [https://www.123rf.com/photo\\_44493517\\_stock-vector-hair-growth-cycle-showing-active-anagen-phase-catagen-telogen-and-early-anagen-phases-created-in-ado.html](https://www.123rf.com/photo_44493517_stock-vector-hair-growth-cycle-showing-active-anagen-phase-catagen-telogen-and-early-anagen-phases-created-in-ado.html)



concerns and expectations with regard to their problem, as this will dictate how aggressive we need to be with our assessments and treatments.

The growth of facial, trunk and limb hair in the male and of pubic and axillary hair in both sexes is clearly dependent on androgenic stimulation at puberty. Scalp hair differs in that its growth does not require androgenic stimulation.

Paradoxically, in genetically predisposed subjects, androgen is required for post pubertal hair deficiency on the vertex and fronto-temporal region in men and the vertex alone in women (androgenic alopecia or patterned hair loss). This is why one can sometimes see men who are balding on the top of their scalp but very hairy on their chest and back.

Human hair, like in many animals, grows in cycles (Fig. 40.1). The growth phase (**anagen**) in the scalp may last for three years or more. Under normal circumstances 80–90% of hair follicles on the human scalp are in the anagen phase at any one time. The rate or speed of hair growth is about 1.25 cm per month, or about 15 cm per year. This is followed by a resting phase (**catagen**) when the hair stops growing. The hair then falls out some weeks later (**telogen** phase). Approximately 100 hairs are lost each day. These hair cycles can vary according to physical, psychological, seasonal and environmental factors. The average adult has approximately 100,000 hairs on their scalp.

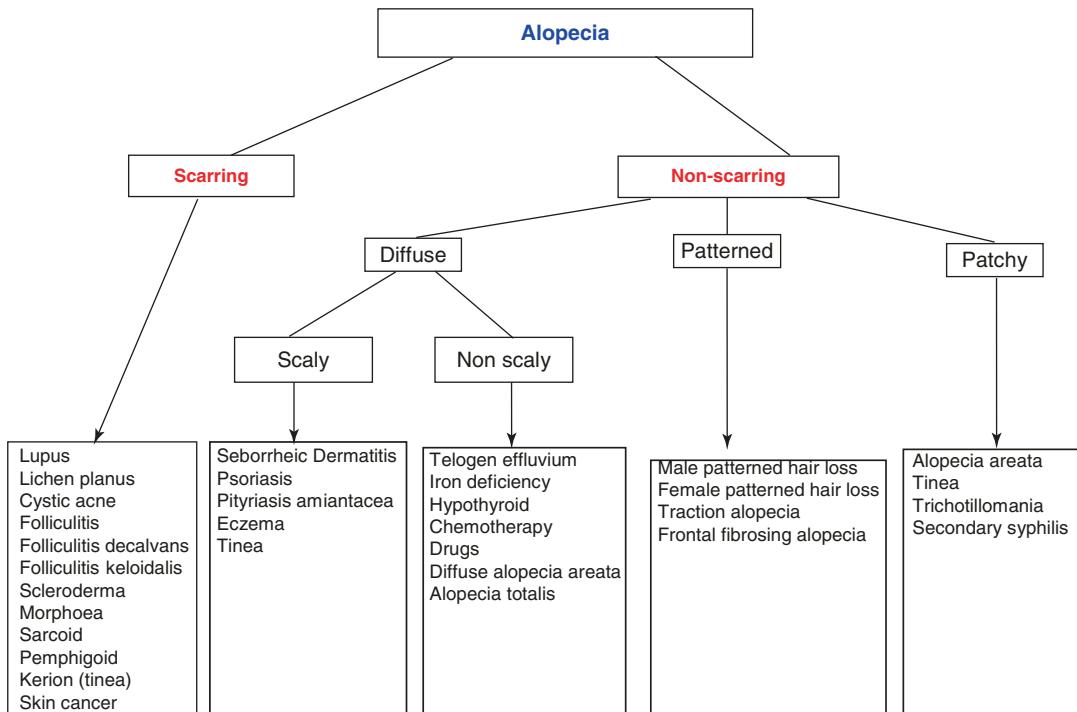
Alopecia is best divided into scarring (cicatricial alopecia) and non-scarring causes. Non scarring alopecia can be further sub divided into

patterned, patchy and diffuse hair loss (Fig. 40.2). Table 40.1 shows some investigations that may have to be considered with assessing a patient for hair loss.

## 40.2 Patterned, Non Scarring Alopecia

Androgenic alopecia (AGA) (“male pattern hair loss”) is by far the most common type of hair loss seen in post pubertal males [1]. The fronto-temporal and vertex areas are affected initially but it can eventually lead to total hair loss on the top of the scalp with preservation of the sides and back of the scalp (Fig. 40.3). Onset can occur at any time after puberty. The tendency towards androgenic alopecia is inherited from both parents but there can be variable expression of the genes. Family history is still the best guide to prognosis. By the age of 70, 80% of men and up to 40% of women have signs of AGA [2]. For most men, counselling to help them to accept their problem is probably the best approach.

Some men will be happy to grow and fashion their hair to cover the affected areas. Volumising shampoo and conditioners may help. (e.g. “Nanogen Thickening Treatment Shampoo®”). Another cosmetic option is hair fibers (e.g. “Nanogen Hair Thickening Fibres®”). This releases thousands of microscopic colour-matched hair fibres, which bind electrostatically to individual hairs giving the appearance of a fuller head of hair. These can



**Fig. 40.2** Causes of alopecia (Hair loss)

**Table 40.1** Investigation to consider for hair loss

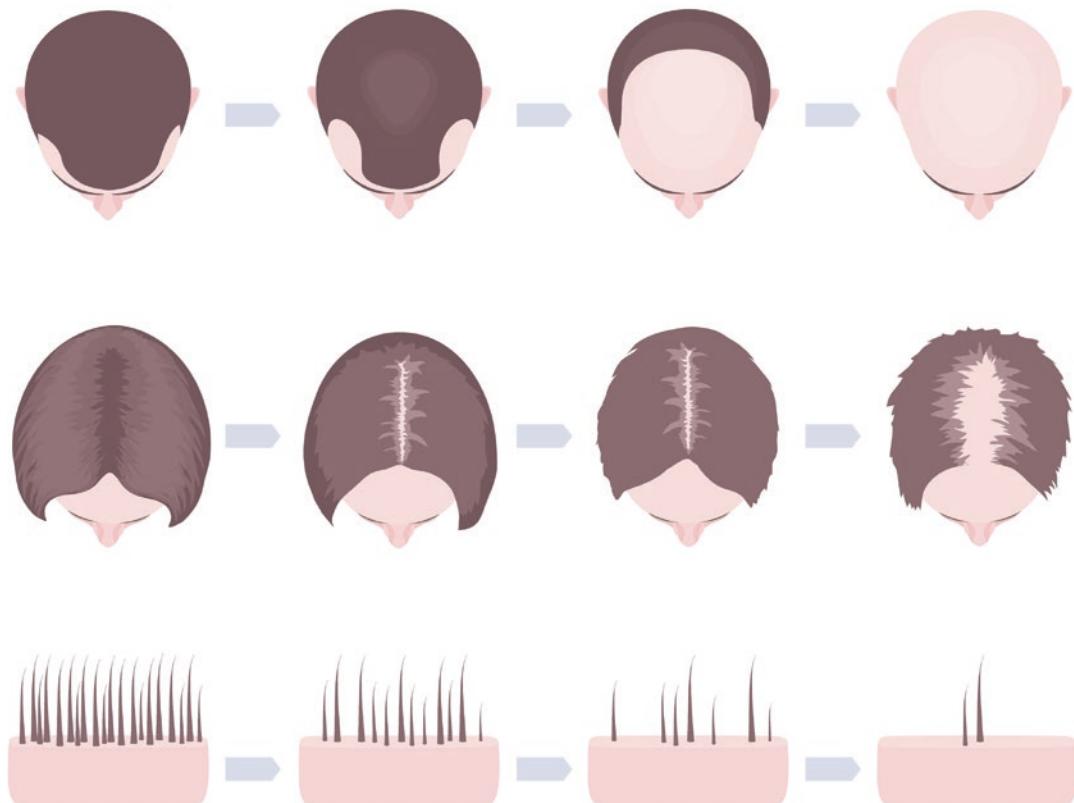
Full blood count
Erythrocyte sedimentation rate
Urea and electrolytes
Liver function tests
Thyroid function tests (TFT's)
Antinuclear factor (ANF)
Glucose
HbA1C
B12, Folate and Ferritin
VDRL for syphilis
ANF and auto antibody screen
Swab for culture and sensitivity
Skin scrapings for fungal stain and culture
Scalp biopsy especially for scarring alopecia
17-Hydroxyprogesterone (17-OHP)
Dehydroepiandrosterone sulfate (DHEAS)—an androgen synthesized almost exclusively by the adrenal cortex
Acute ACTH stimulation test (Synacthen test) for later onset congenital adrenal hyperplasia
PCOS investigations (see Chap. 9). FSH/LH, Oestradiol, Sex hormone binding globulin, Free Testosterone, Prolactin (check bloods on the first week after a period)

be made wind and waterproof with various locking mists (e.g. "Nanogen Fibre Locking Spray®"). Temporary scalp dyes can safely blend scalp and hair colour together to conceal areas of thinning

hair (e.g. "Nanogen Aquamatch Waterproof Concealer®").

Vitamin supplements promoted for hair and nail growth such as biotin 2.5 mg/day (a B complex vitamin also known as vitamin H that may improve the keratin infrastructure as found in "Viviscal®" and "Pantogar®") have low evidence of efficacy and should be avoided in pregnant women. Platelet rich plasma injections have been tried but there is not enough evidence to recommend it at present [2]. There is some weak evidence that low level laser light, visible red light and LED light therapy may have some beneficial effects in androgenic alopecia [3]. There are thousands of herbal and homeopathic products available on the internet and in retail outlets that are promoted for hair loss but the vast majority have little or no benefits and have not gone through any serious clinical trials. People with hair loss are often desperate, many are gullible and naive and may try anything that has glossy marketing and convincing websites, despite lack of scientific evidence that they actually work. Doctors can help in trying to point the patient with hair loss in

# HAIR LOSS



**Fig. 40.3** Patterned hair loss. Top row is in men; middle row is in women. HAIRLOSS© [naumas]/123RF.COM Image ID 95514985. Media Type: Vector. [https://es.123rf.com/photo\\_95514985\\_male-and-female-pattern-hair-loss-set-stages-of-baldness-in-men-and-women-number-of-follicles-on-sca.html?vti=ns5daeljqrn20chkm-1-39](https://es.123rf.com/photo_95514985_male-and-female-pattern-hair-loss-set-stages-of-baldness-in-men-and-women-number-of-follicles-on-sca.html?vti=ns5daeljqrn20chkm-1-39)

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the right direction when choosing hair care products and supplements that at least have some evidence of efficacy (see Chap. 66).

Others may choose to shave their scalp. Some men are happy to wear a hair piece or wig. **Hair transplantation using follicular unit transfer**, when performed by an experienced surgeon, can give excellent results once there is not extensive hair loss but is very expensive. Hair transplants are usually combined with oral and/or topical hair loss products such as minoxidil and finasteride (as mentioned below) to reduce postoperative progression of androgenic alopecia.

**Minoxidil** (“Regaine Scalp Foam®”) is approved by the US Food and Drug Administration (FDA) for use in androgenic alopecia in both sexes. Although its exact mechanism of action is unclear, minoxidil opens potassium channels and was originally approved as an oral treatment for hypertension [4, 5]. Minoxidil prolongs the anagen phase of the hair cycle by inducing the transition from telogen to anagen hairs [6].

However, results to date with topical minoxidil have been disappointing. Significant regrowth, which is clinically apparent, is rare. In most patients there is a slowing down of the rate of hair loss and some patients are quite happy to accept

even this limited response. The 5% solution or foam once a day has been shown to be as effective as the 2% solution applied twice a day but may cause more scalp irritation [2]. Topical minoxidil should not be used if there is any underlying scaly disease or irritation. It should be continued twice a day for at least 4 to 6 months before deciding if there is any improvement. There may be a temporary shedding of hair in the first 4 to 6 weeks of use before re-growth starts and all patients should be warned of this possibility before starting minoxidil. If there is some improvement, minoxidil can be continued once a day or three times per week for months or years. Best results occur if it is started early in the hair loss process. If the treatment is stopped any hair that has been ‘saved’ will fall out. Minoxidil should not be used in pregnancy and when breastfeeding.

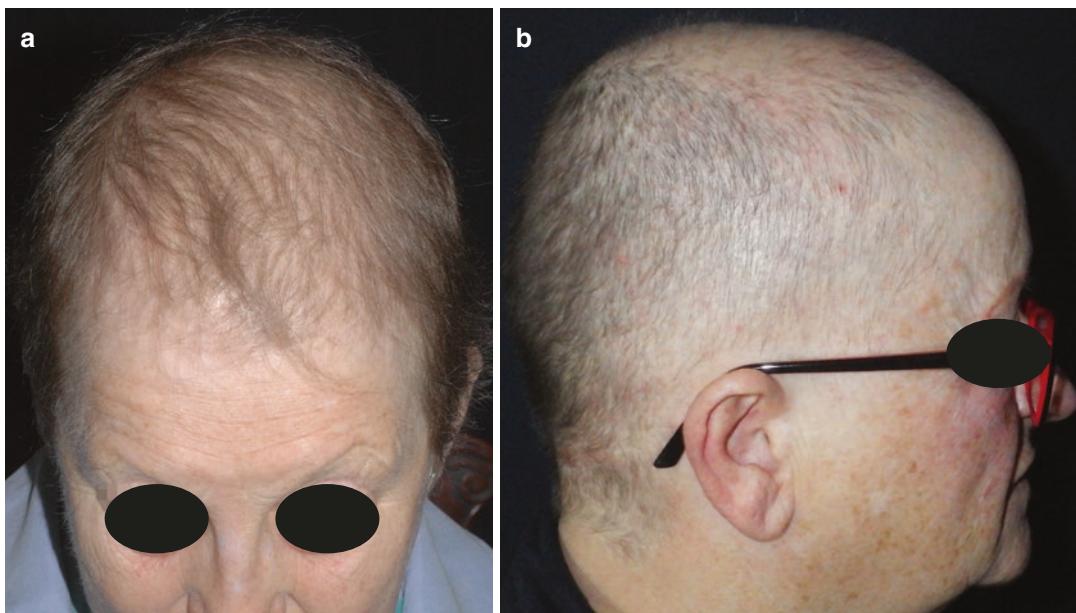
**Oral finasteride** is a specific type II 5-alpha reductase inhibitor most commonly used for benign prostatic hypertrophy. This drug also slows down the rate of hair loss in men especially if it is begun early in the disease process. It can work well with topical minoxidil. It is available in the US as “Propecia®” which comes in a 1 mg tablet and is taken once per day. In countries where it is not licensed, some doctors prescribe a quarter of a 5 mg finasteride tablet daily (e.g. “Proscar®” 5 mg tabs-one quarter of a tablet daily) for androgenic alopecia in men. Some research suggests that some patients may develop permanent sexual dysfunction, (decreased libido, erectile dysfunction, depression) even after the drug is stopped. This has led to a series of law suits in the US and the establishment of the “Post Finasteride Syndrome Foundation” in the US which appears to be backed by legal firms touting for business. A recent large study published in the BMJ in 2016 provide evidence that in the alopecia population ( $n = 12,346$ ), the risk of erectile dysfunction was not increased in users of finasteride 1 mg compared with unexposed men with alopecia [7]. New topical versions of finasteride 0.1% have been formulated which has been shown to have effects similar to oral finasteride in one small study [8]. Topical scalp lotions with minoxidil and finasteride combined are now commercially available and have been shown to be safe and effective in some small studies [9].

Oral finasteride should not be used in men with a history of depression or sexual dysfunction. Finasteride cannot be used in women of child bearing age as it is teratogenic. It has been used off license at a dose of 5 mg/day in post-menopausal women with some success [10]. Dusaseride 0.5 mg daily (a more potent 5-alpha-reductase inhibitor) has been used alone or in combination with topical minoxidil for patterned hair loss in men and post menopausal women in some studies but it is not licensed for this indication yet.

**Female pattern hair loss (androgenic alopecia = AGA)** is of later onset, is less severe and progresses less rapidly than in men. The hairline is preserved but there is more diffuse thinning in the vertex (Figs. 40.3 and 40.4a, b) [1]. It may begin at any time after puberty but usually accelerates after the menopause. Routine bloods including serum ferritin and thyroid function tests (TFTs) should be checked. If the ferritin is below 70 ng/mL an iron supplement should be given for 3 to 6 months together with advice on an iron rich and vitamin C rich diet. If the patient is anemic she will need investigations as to the cause. Volumising shampoos and lightening the hair color so it blends in with the scalp may help. Hair fibers and scalp dyes as mentioned for men can also make a big difference to some women with hair thinning. Hair extensions are another option for some women. Follicular transfer hair transplant, although more popular in men, is also an option for women.

Early onset female patterned hair loss may be a feature of polycystic ovarian syndrome (PCOS) or later onset congenital adrenal hyperplasia.

Topical minoxidil (“Regain 5% foam®”) can help if started early in the hair loss process. Some cases may respond to low doses of cyproterone acetate (e.g. “Dianette®”) especially if she has other indications for the oral contraceptive pill, such as for contraception, menstrual problems, acne or polycystic ovarian syndrome (PCOS). “Dianette®” can only be used in women of reproductive age and should be avoided in smokers and those with hypertension, hypercholesterolemia, obesity or a history of venous thromboembolism (VTE). All patients on “Dianette®” need to be warned about the possibility of venous



**Fig. 40.4** (a) Female pattern hair loss in a 67-year-old woman. (b) Female pattern hair loss in a 61-year-old woman

thromboembolism (VTE) and be given the warning signs to look out for.

Spironolactone can act as an anti-androgen and can help female pattern hair loss and hirsutism. It is teratogenic so effective contraception should be used in women of child bearing age on this drug. It is usually started at a small dose (25 mg/day) and gradually increased to a maximum of 200 mg/day if required and tolerated. It can cause hyperkalaemia and electrolytes should be monitored regularly. Combinations of topical minoxidil and systemic treatments such as “Dianette®” or spironolactone are sometimes used.

**Traction alopecia** usually results from plaiting or weaving the hair too tightly. It most commonly occurs in people of Afro-Caribbean descent and is usually found in the temples.

**Frontal fibrosing alopecia**, as the name implies, causes permanent alopecia with itch in the frontal area of the scalp in adult women [11] (Table 40.2) (Fig. 40.5a, b). There is perifollicular erythema and scaling in the frontal area that can lead to permanent non scarring alopecia. There is also loss of villous hairs, marooned hairs and orphaned hairs. Eyebrows may also be affected. The prevalence of frontal fibrosing alopecia has increased ten-fold over the past decade [12]. The cause is unknown but it can

be considered a variant of lichen planus of the scalp (lichen planopilaris) but hormonal factors may be involved, as most cases occur in women after the menopause. Other possible triggers including, neurogenic inflammation, smoking, UV filters (e.g. Oxybenzone), and ingredients in leave-on facial products. The receding hair line can be unsightly but many women can cover it up by growing a fringe. Diagnosis is usually by a biopsy and early referral to a skin specialist is advisable as treatments may delay the progression of the disease. Once the hair is lost it will not re-grow so early treatment is important. Treatment options include potent topical or intra-lesional steroids, oral steroids, anti-inflammatory antibiotics such as doxycycline, or antimalarial tablets. Topical minoxidil may be combined with some of these other treatments. 5-alpha-reductase inhibitors (e.g. finasteride 1 to 5 mg OD or dutasteride 0.5 mg orally  $\times$  3 times per week) may help in some cases but is off license and can only be considered in post-menopausal women. Topical calcineurin inhibitors (e.g. “Protopic”) may help eyebrows to regrow. Topical bimatoprost 0.03% ophthalmic solution (“Lumigan®”), which is licensed for open angle glaucoma, has been used as a once-daily topical treatment for eyelash and eyebrow hair thinning.

**Table 40.2** Common types of hair loss in women [10, 11, 13]

Type	Clinical description	Causes
Alopecia areata (AA)	Non-scarring; no signs of erythema or skin inflammation; occurs in patches; acute onset	Uncertain etiology; possibly autoimmune in origin
Chronic telogen effluvium (CTE)	Non-scarring; early shedding of hair; not associated with thinning hair	Idiopathic and multifactorial; typically no specific trigger
Central centrifugal cicatricial alopecia (CA)	Inflammatory in origin, irreversible hair loss and follicular ostia; common in African American women	Etiology and pathology unknown; hairstyle can play a role
Female pattern hair loss (FPHL)	Progressive and gradual hair loss in characteristic patterns; non-scarring	Some hormonal causes; role of genetics unclear, possibly polygenic
Frontal fibrosing alopecia (FFA)	Scarring form of hair loss; characterised by hairline recession; associated with decrease/loss of eyebrows; mostly occurs in postmenopausal women	Inflammatory; cause unknown
Telogen effluvium (TE)	Acute, diffuse hair loss of <50%; non-scarring; duration approximately 3 months; transient but may precede FPHL	Can be due to disease, medications, pregnancy, dietary deficiencies, autoimmune disorders, emotional distress

Table from: Update on female pattern hair loss: advances in diagnosis and treatment, Medscape Education 2016



**Fig. 40.5** (a) Frontal fibrosing alopecia in a 64 year old woman which is now dormant. (b) Frontal fibrosing alopecia and thin eyebrows laterally  $\times 10$  years in a 61 year old woman

### 40.3 Patchy Non Scarring Alopecia

**Alopecia areata** presents with a characteristic coin shaped area of hair loss varying in size from 1 cm in diameter to 10 cm or more (Fig. 40.6a, b). Many patients develop only one or two small areas that grow back spontaneously within six to 12 months. Some patients may have a few large areas or multiple small areas of alopecia areata which coalesce to create significant cosmetic disfigurement (Fig. 40.7). Occasionally patients may lose all their scalp hair (**alopecia totalis**)

(Fig. 40.8) or all of their scalp and body hair (**alopecia universalis**). The more extensive or chronic cases are less likely to resolve spontaneously and will respond less well to treatment.

The cause of alopecia areata is unknown but it is thought to be autoimmune in origin and it may be associated with other autoimmune diseases such as type 1 diabetes, thyroid disease, vitiligo, etc. Screening for these conditions should be done in patients with alopecia areata. Unlike other autoimmune diseases, the end organ in alopecia areata (the hair root) is not damaged and the potential for regrowth is retained.



**Fig. 40.6** (a) Alopecia areata with good white regrowth. (b) Alopecia areata beard area of a 32-year-old male



**Fig. 40.7** Alopecia areata in an Indian woman

Treatments when necessary are non specific and difficult to assess because of the spontaneous remission which often occurs. Topical or intradermal steroids, short contact dithranol and phototherapy have all been tried with varying degrees of success. Severe cases may respond to immunosuppressants such as methotrexate or some of the newer biological agents (eg: ustekinumab). Psychological support and advice about hair styling and wigs can be helpful in resistant cases. For patients with pale skin, dyeing the hair a light color may help camouflage the bald patches against the white scalp. In December 2008 the US Food and Drug Administration approved a prostaglandin analogs, 0.03% bimatoprost solution ("Latisse®"), identical to the ophthalmic solution for glaucoma treatment ("Lumigan®"), for increasing eyelash length, thickness and darkness in patients with hypotrichosis of the eyelashes, or persons



**Fig. 40.8** Alopecia totalis in a 44-year-old woman

who desire lengthy eyelashes. There is good evidence it can help in patients with alopecia areata affecting the eyelashes [13].

Information for patients with alopecia areata is available from the The National Alopecia Areata Foundation (NAAF) website: [www.naaf.org](http://www.naaf.org)



**Fig. 40.9** Moth-eaten alopecia of secondary syphilis. Left, far view; right, close-up view

**Trichotillomania (formerly called trichotillomania or hair pulling)** is an extreme form of self inflicted traction alopecia which is most commonly seen in adolescent girls who usually have an underlying psychological problem (see Chap. 53).

**Secondary syphilis** can cause patchy “moth eaten” hair loss (Fig. 40.9). Syphilis is still “the great mimicker.” There may be other features of secondary syphilis such as a generalised rash, a patchy rash on the palms or soles, or condylomata lata, but alopecia can be the only presenting feature of syphilis. Syphilitic alopecia can mimic alopecia areata both clinically and on histology. The bald patches in alopecia areata tend to be more well defined and have “exclamation mark” hairs. Tinea capitis can resemble moth-eaten syphilitic alopecia, but skin scrapings or plucked hairs should indicate the presence of fungus. Trichotillomania can also present as moth-eaten alopecia, but the history and normal findings on scalp biopsy should help differentiate these conditions.

#### 40.4 Diffuse Non Scarring Alopecia Without Scale

An underlying systemic disorder such as hypothyroidism, hypopituitarism, secondary syphilis, anaemia or iron deficiency should be excluded. Numerous drugs can cause hair loss such as anti-

epileptics, anti-coagulants, the oral contraceptive pill, lithium, ibuprofen, oral isotretinoin, cytotoxics, chemotherapeutic agents, anti-hypertensives and diuretics.

Pregnancy, a high fever or severe psychological distress may stimulate many follicles to pass into the telogen phase simultaneously with a resultant hair fall a few weeks or months later (**telogen effluvium**). This is always followed by good regrowth without any treatment within six to twelve months. The pull test is an examination to roughly judge active hair shedding. 50–60 hairs are grasped by thumb, index and middle fingers. The fingers should slide along the hair shaft gently pulling them upwards. The pull test is positive when more than 10% of the grasped hair can be pulled out. In patients with androgenic alopecia the pull test is positive only in the active phase and on the top of the scalp.

Alopecia areata can rarely be extensive affecting the whole scalp with diffuse thinning without the characteristic circular patches.

#### 40.5 Diffuse, Non Scarring Alopecia with Scale

Generalised skin conditions that may affect the scalp such as seborrhoeic dermatitis, psoriasis, pityriasis amantacea, tinea capitis and eczema/dermatitis can all cause diffuse temporary hair

thinning especially when the itch and scale is severe. Treating the underlying condition usually results in full hair regrowth. Thick scales can be removed with a tar and salicylic acid ointment such as "Cocois®", which can be applied for a few hours and washed out with a tar based shampoo. Anti dandruff shampoos such as "Nizoral®" or "Stieprox®" will help seborrhoeic dermatitis. "Dovobet gel®" will help clear scalp psoriasis and pityriasis amiantacea. Scalp eczema should clear with a steroid scalp lotion or steroid shampoo. A soap free shampoo should be used if there is scalp eczema. Skin scrapings and/or plucked hairs should be sent for fungal stain and culture if there is a suspicion of fungal infection. Dark suits and tops should be avoided as they show off the dandruff scale.

## 40.6 Scarring Alopecia (Cicatricial Alopecia)

Active disease will show erythema and scaling with symptoms such as itch or pain. Inactive disease may simply present as a white atrophic permanent hairless patch. Scarring alopecia from developmental defects or physical injury should be evident from the history. Early aggressive treatment of scarring alopecia is vital, as hair regrowth will not happen once scarring is developed.

A fresh patch of scarring alopecia in a child should be considered due to ringworm (**tinea capitis**) until proven otherwise. Most cases clear completely with oral antifungals but if the lesion becomes secondarily infected with bacteria or if a kerion occurs, permanent scarring alopecia may result.

Bacterial **folliculitis** of the scalp can cause diffuse small scattered areas of scarring alopecia. It is more common in Afro-Caribbeans as they tend to use a lot of oily products in the scalp. Swabs of pustules may grow *staph aureus* which should respond to a course of flucloxacillin for 2 to 4 weeks.

**Folliculitis decavans** is a rare, abnormal immunological reaction to infection with *staph aureus* that is more common in men. It can cause

a progressive permanent scarring alopecia with pustules and crusts around the affected hair follicles (Fig. 40.10). More advanced cases can cause white scarring with clumping of the hairs in the scars like a dolls scalp. It does not respond to flucloxacillin. Some cases may respond to anti-acne type treatment such as lymecycline 408 mg OD or BD, Doxycyclin 100 mg OD or BD or trimethoprim 300 mg BD for a few months. Resistant cases may respond to rifampicin and clindamycin 300 mg of each BD for 12 weeks. Oral isotretinoin is another option for resistant cases. Topical or intralesional steroids may help.

**Folliculitis keloidalis** is a rare form of folliculitis that can cause keloids and scarring alopecia in the posterior scalp and the nape of the neck, especially in Afro-Caribbean adult males (Fig. 40.11). Folliculitis keloidalis is also called acne cheloidalis nuchae or acne keloidalis, even though this condition is not in any way related to acne.



**Fig. 40.10** Folliculitis decavans post-laceration of the scalp



**Fig. 40.11** Acne keloidalis nuchae on a 24-year-old obese man

The cause is unknown and treatment is very difficult. Some cases may respond to topical or intralesional steroids. Other treatment options include oral tetracycline as an anti-inflammatory, a three-month course of clindamycin or oral isotretinoin. Large keloids may respond to laser treatment or cryosurgery. Oral isotretinoin has been used with some good results.

**Lichen planus** (called **lichen planopilatus** when it affects the scalp) can cause scarring alopecia with perifollicular hyperkeratosis (Fig. 40.12). It may occur in isolation or can be associated with lichen planus in other areas of the body (see Chap. 19). It is diagnosed by skin biopsy and treatment is usually with potent topical steroids or intralesional steroids. More resistant cases may require systemic treatment with doxycycline 100 mg daily for 12 months, oral steroids (1 mg/kg/day x 15 days and tapering off over 4 to 6 months), or cyclosporine.

Scalp manifestations of a more widespread dermatosis such as **lupus erythematosus**, **scleroderma**, **morphea**, **sarcoid** or **pemphigoid** may all cause scarring alopecia. A complete examination of all the skin, including the nails, mucous membranes and genitalia may provide clues as to the exact aetiology. In difficult cases a skin biopsy for histology and immunofluorescence should help to make a diagnosis. A **primary or secondary skin cancer** should always be considered in unusual cases.



**Fig. 40.12** Lichen planopilatus × 4 years with features of frontal fibrosing alopecia (no eyebrows) and female pattern baldness in a 63 year old woman

## 40.7 Hirsutism

Many women develop mild growth of fine villus hair on their face and body which they either ignore or manage themselves or with the help of a beautician. This should not be confused with hirsutism which is defined as a heavy growth of coarse terminal hair in areas where normally only men have hairs such as moustache, beard area, chest, belly, and back (Fig. 40.13). It is not always easy to decide where normal 'hairiness' ends and hirsutism begins. Hirsutism must also be distinguished from **hypertrichosis** which is defined as excessive growth of hair on the face and body, not in a male pattern, which may occur as a result of thyroid disease, anorexia nervosa, porphyria, certain drugs (e.g., ciclosporin, diazoxide, minoxidil, minocycline) or an underlying neoplasm.

Up to 5% to 10% of women are hirsute. The most common cause is polycystic ovary syndrome. Hirsutism can lead to psychological distress, low self esteem, depression, and social isolation.

Hair follicles on the face and body are influenced by circulating androgens (which stimulate hair growth), oestrogens (which retard hair growth) and by the sensitivity of the hair follicles to circulating hormones. Acne and female pattern hair loss are also under androgen control and may accompany hirsutism especially in polycystic ovary syndrome (PCOS) (Fig. 40.14).



**Fig. 40.13** Hirsutism in a woman with polycystic ovarian syndrome (PCOS)



**Fig. 40.14** PCOS + male pattern hair loss in a 33 year old woman

The most common cause of **hirsutism** is '**idiopathic**'. Most of these patients, when investigated thoroughly, can be shown to have minor hormonal abnormalities (Table 40.3). Another less common cause is **PCOS**. This usually presents with hirsutism combined with other signs of androgen excess such as obesity, menstrual irregularities, relative infertility, acne, female pattern alopecia or enlarged polycystic ovaries.

Both of these causes can be treated with anti-androgens. Mild cases may respond to low doses of cyproterone acetate (e.g. "Dianette<sup>®</sup>") or an oral contraceptive pill with a progestogen such as desogestrel. More severe cases may need much higher doses of cyproterone acetate or spironolactone (25 to 100 mg OD) to achieve results. These treatments may also help female patterned hair loss. Response can be slow and can take 6–12 months or treatment before assessing if they are responding. Eflornithine ("Vaniqua<sup>®</sup>") is

**Table 40.3** Investigation to consider for hirsutism

(Early morning bloods and on the first week after a period)
Full blood count
Erythrocyte sedimentation rate
Urea and electrolytes
Liver function tests
Thyroid function tests (TFT's)
Glucose and HbA1C
FSH/LH
Oestradiol
Sex hormone binding globulin
Free Testosterone
Prolactin
US ovaries
17-Hydroxyprogesterone (17-OHP)- a marker unique for congenital adrenal hyperplasia
Dehydroepiandrosterone sulfate (DHEAS) -an androgen synthesized almost exclusively by the adrenal cortex
Acute ACTH stimulation test (Synacthen test) for later onset congenital adrenal hyperplasia
Twenty four hour urine free cortisol if signs and symptoms of Cushing's syndrome.
Magnetic resonance imaging (MRI) or computed tomography (CT) of the adrenals

an ornithine decarboxylase inhibitor and can reduce hair growth when applied topically for at least two to four months.

Some cases of hirsutism are due to **late onset congenital adrenal hyperplasia (CAH)** which can present with hirsutism, acne, alopecia, anovulation, and menstrual dysfunction. Late onset CAH may be difficult to differentiate from polycystic ovary syndrome (PCOS). Random 17-Hydroxyprogesterone (17-OHP) concentrations may be within the normal range for individuals with later onset CAH. Thus, the acute ACTH stimulation test (**Synacthen test**) remains the gold standard to measure the ability of the adrenal cortex to respond to ACTH by producing cortisol appropriately.

**Cushing's syndrome** may also cause hirsutism. Most cases should be sent to an endocrinologist for further investigation and treatment.

Very occasionally a **hormone secreting ovarian, adrenal or pituitary tumour** may present with sudden onset hirsutism. **Drugs** which can cause hirsutism include androgenic oral contraceptive pills, anabolic steroids, phenytoin and corticosteroids.

**Table 40.4** Hirsutism; reasons to refer

- Moderate or severe hirsutism not adequately controlled with local (cosmetic) treatment
- Rapidly progressive hirsutism
- Late onset hirsutism
- When associated with other signs of hormonal imbalance, including menstrual irregularities, infertility, galactorrhoea, deep voice, increased muscle bulk, female pattern hair loss, clitoromegaly or enlarged ovaries.

Most patients with milder degrees of hirsutism do not require investigations and can be managed with local cosmetic treatments such as bleaching or depilatory creams. Patients should be reassured that plucking, waxing or shaving will *not* make hairs grow any faster, darker or thicker. Electrical epilation (electrolysis) offers some hope for a permanent hair reduction in localised areas. Laser or IPL can be very helpful for more extensive hair removal and work best on patients with dark hair and light colour skin (skin type 1 or 2). Patients with skin type 3 or 4 skin and excessive hair growth may respond to an Nd:YAG laser. More severe cases of hirsutism, especially if associated with other signs of hormonal imbalance, need to be referred to an endocrinologist for further investigation and treatment (Table 40.4).

## 40.8 Conclusion

Hair loss and excessive hair growth can be devastating for some people, especially women. While most people, when faced with hair thinning, worry that they may lose all their hair and end up wearing a wig, fortunately this degree of hair loss is rare especially in young patients. With careful history taking, examination and in some cases, laboratory investigations, most patients with hair thinning can be helped. Advice on hair styling, hair colouring and volumising shampoos may also help. Hair transplants, while too expensive for many, can give fantastic results in some patients. Excessive hair growth in women may need investigation and many cases can be managed by simple methods from a beautician or with lasers or IPL machines.

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# Nail Problems in General Practice

41

David Buckley

## Key Points

- Fungal nail infections should be confirmed by sending nail clippings to the lab for fungal stain and culture before commencing systemic treatment.
- It can be very difficult to distinguish fungal nail infection from psoriasis.
- Chronic paronychia is usually caused by damage to the cuticle and can result in secondary nail changes.
- Ingrown toenails that cause infections requiring systemic antibiotics will usually need surgery to permanently resolve the problem.
- The nails may give clues to underlying diseases.

## What to Tell the Patient

- Your cuticles are very important and delicate. Do not pick, bite or over manicure them as they act as a seal between the nail and the surrounding skin.
- Fungal nail infections may be somewhat unsightly but are usually harmless and do not always require treatment.
- If you develop unexplained pigmentation or a growth under the nail, check with a doctor as skin cancer can sometimes present like this.

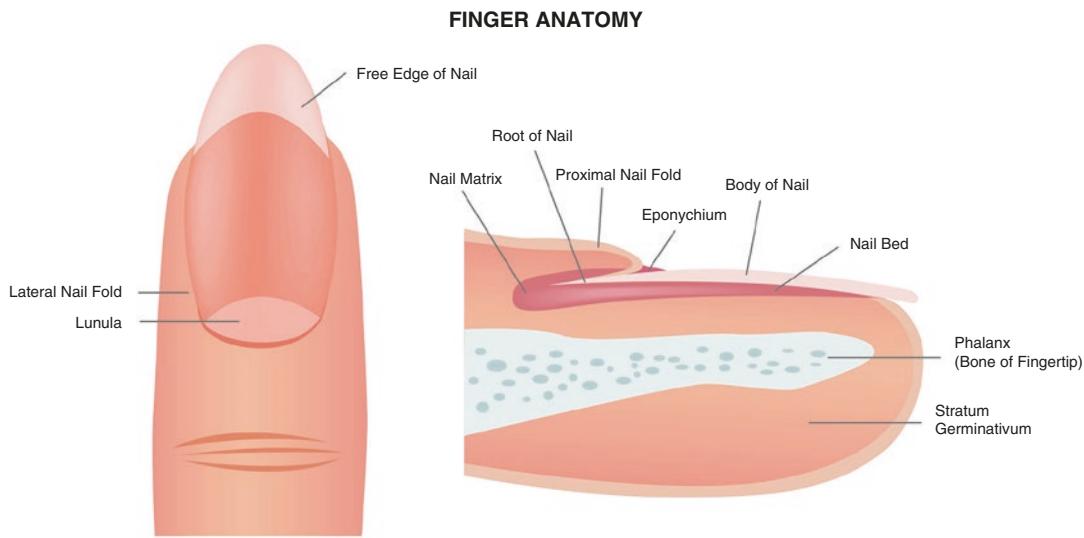
## 41.1 Introduction

Nails have an important function in fine movements such as picking up small objects. They also protect the fingertips from trauma and their cosmetic function should not be underestimated (Fig. 41.1). It is only when we have a problem with our nails that we realise how important they can be. On average, fingernails are replaced every six months and toenails every 18 months to two years.

## 41.2 Local Factors Affecting the Nail

**Trauma** is the most common cause of nail dystrophy. Nail biting or picking is often subconscious and can lead to damage to the nail plate directly or indirectly through damage to the cuticle. Cuticle damage through manicuring, harsh nail varnish removers, soaps, detergents, constant wetting of the hands, picking or biting may cause ridging or rippling of the nail plate or an infection in the folds of skin adjacent to the cuticle (paronychia).

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**Fig. 41.1** Cross section of a nail. Finger Anatomy© GritsalakKarat@123RF.com Image ID Media Type: Vector. [https://www.123rf.com/photo\\_67560349\\_structure-of-a-nail-human-nail-anatomy.html?vti=mslyc6jgjlmt1v5if-1-1](https://www.123rf.com/photo_67560349_structure-of-a-nail-human-nail-anatomy.html?vti=mslyc6jgjlmt1v5if-1-1)



**Fig. 41.2** Nail dystrophy secondary to nail picking and cutical damage



**Fig. 41.3** Joggers toes

**Median Nail Dystrophy of Heller** is a central dystrophy involving the thumb nail caused by habitual picking of the cuticle at the nail base. It is an impulsive behaviour and before attempting any medication, it is important to explain to the patient the cause and to try to help them to stop picking or biting (Fig. 41.2).

Nails can detach from the nail bed. It is called **onycholysis**. The color of the separated part will not be pink but white. It is very frequently misdiagnosed as fungal infection. Psoriasis or trauma are common causes. In toenails it may happen after long walks, running or after sports like foot-

ball (where there is continuous hitting to the tip of the nail off the footwear). This can also cause subungual haematoma which can cause the nail to fall off a few months later but fortunately a healthy new nail will grow up under the old nail as it slowly detaches spontaneously (Fig. 41.3). Keeping the toe nails short reduces the chances of separating the nail from the nail bed.

**Acute paronychia** is usually caused by *Staphylococcus Aureus* penetrating the periungual area as a result of cuticle damage. The nail fold becomes suddenly swollen, red, and painful. Pus may accumulate in the paronychial space.

Systemic antibiotics combined with an incision and drainage when necessary is the treatment of choice. The patient should be advised on correct nail care which should include warning them against damaging the cuticle by manicuring. They should keep their hands dry as much as possible by the careful use of gloves and avoid soaps and other irritants.

**Chronic paronychia** is usually caused by damage to the cuticle which allows *Candida Albicans* and a variety of other non-virulent organisms and irritants to penetrate the periungual space over weeks or months. It presents as chronic swelling, tenderness and redness in the area of the nail fold with accompanying non-specific nail plate irregularities, including ridging and discolouration. Treatment involves careful hand and nail care to allow the cuticle to re-grow, together with a topical imidazole antifungal cream. Verbal and written instructions should be given to the patient on how to keep their hands and cuticles dry at all times by the careful use of gloves and how to avoid soaps, shampoos and other irritants (see Chap. 66). Recovery may take many months.

**Lamellar dystrophy (onychoschizia)** causes horizontal splitting and peeling of the distal ends of the nails, like slate splitting in a quarry, usually in adult women. It is usually caused by repeatedly exposing the nails to harsh soaps, shampoos and detergents. Treatment is by good hand care as outlined above.

**Dermatophyte (ringworm) infection** of the nail (onychomycosis) usually presents as white or yellow discolouration of the nail plate which may

become crumbly and distorted with heaped up debris under the free edges (Table 41.1) (Figs. 41.4 and 41.5). The prevalence of onychomycosis has been reported to be as high as 23% across Europe [1] and becomes more common as we age, with poor foot hygiene, peripheral vascular disease and in those who are immunosuppressed. Onychomycosis of the toe is often associated with tinea pedis (athlete's foot) suggesting that the skin is the main source of fungal organisms that infect the nail. The most common organism is *Trichophyton Rubrum* which also causes tinea paedis. It is usually painless and shows little signs of inflammation. Only about 50% of discoloured or dystrophic-appearing nails have a fungal infection confirmed with dermatophyte on culture.

**Tinea unguium (onychomycosis)** may resemble many other nail disorders such as psoriasis

**Table 41.1** Presentation of fungal nail infection

- **Distal and lateral subungual onychomycosis (DLSo)**  
Usually caused by dermatophyte infection, is usually associated with tinea pedis and is the most common presentation.
- **Superficial white onychomycosis (SWO)**  
It presents as white chalky plaque on the surface of the proximal nail plate, almost exclusively on the toenails.
- **Proximal subungual onychomycosis (PSO)**  
Usually associated with paronychia and most commonly caused by *Candida* especially in those with wet jobs.
- **Total dystrophic onychomycosis (TDO)**  
Complete destruction of the nail plate from long standing, end-stage disease progressing from any of the above types of nail infection.

**Fig. 41.4** Tinea unguium in an 18 year old



**Fig. 41.5** Tinea unguis. *Trichophyton rubrum* was isolated



**Table 41.2** Differential diagnosis of fungal nail infection

- Onychogryphosis (thickening and distortion of the nail, typically of the big toe, usually due to previous nail trauma).
- Trauma (tight shoes, nail biting, runner's toes).
- Eczema (irritant or allergic contact dermatitis of periungual skin causing secondary nail changes).
- Lichen planus.
- Subungual melanoma.
- Psoriatic nail disease.
- Bacterial paronychia causing secondary nail infection—e.g. *Pseudomonas* spp. infection.
- Systemic disease—e.g. thyroid disease, diabetes, peripheral arterial disease.
- Rare systemic disorders—e.g. keratosis follicularis (Darier's disease), yellow nail syndrome.
- Idiosyncratic drug reaction (especially tetracyclines, quinolones and psoralens).

riasis or lichen planus, so nail clippings and subungual debris should be taken for fungal stain and culture to confirm the diagnosis before starting treatment (Table 41.2). Clippings should be taken as far proximal as possible by the doctor (not the patient) and sent to the lab in a sterile urine sample bottle. The patient should be warned that taking clippings can be painful and may cause some bleeding. Results can take a month to come back from the lab. False negatives can occur especially if the patient has already used topical or oral antifungals. Candida is sometimes isolated but it may be an opportunistic infection on top of a primary nail disease such as psoriasis.

Some patients may choose not to have treatment, particularly if only the toenails are affected. Many factors need to be considered before deciding to treat including the degree of discomfort, the visual appearance of the nail, the patient's general health, drug interactions, the risks of side effects and cost of treatment. If the patient insists on treatment, a twice weekly application of amorolfine ("Loceryl®") nail paint for 6 months for finger nails and 12 months for toenails is convenient and may be effective for very superficial nail plate infections. Mechanical debridement of the nail plate is recommended before the application of topical antifungals. 40% Urea ointment ("Canespro Fungal Nail Treatment®") can be used to debride the damaged infected nail by daily applications for 2 to 3 weeks under occlusion. After this, treatment with a topical antifungal such as terbinafine cream or amorolfine nail paint will penetrate better and be much more effective at clearing the fungus.

The most popular oral therapy for established fungal nail infection is terbinafine ("Lamisil®") 250mg daily for six weeks for finger nail infection and three months for toenails. This should not be started until the infection has been confirmed by fungal culture. Routine bloods (full blood count, urea and electrolytes, and liver function tests) should be taken before starting a course of terbinafine and the bloods should be repeated after one month, as rare cases of liver damage and severe drug eruptions have been reported with this drug.

Terbinafine interacts with rifampicin and cimetidine and may cause nausea. Not all cases will respond and relapse is not uncommon. The patient should be warned in advance that the nails will still look dystrophic at the end of the course of treatment. It could take another 6 months for finger nails and 12 months for toenails before a new, uninfected, normal nail to grow out.

Sprinkling an antifungal powder into all the footwear regularly may help prevent re-infection. Replacing insoles of shoes and discarding old footwear may help prevent relapse. Wearing leather shoes in the winter and open sandals in the summer may help. The patient should also avoid walking barefooted, especially in the gym, at the swimming pool and even in the family home.

Itraconazole may also be used for fungal infection of the nails and is a better choice if there is a yeast infection in the nail. Itraconazole 200mg can be taken once daily for 3 months or can be used as pulsed therapy (200mg twice a day for one week every month for 3 months). Bloods should be checked before treatment and one month into a course of treatment. Itraconazole can interact with warfarin, antihistamines, anti-psychotics, digoxin, H<sub>2</sub>-receptor antagonists, some statins and phenytoin. "Loceryl nail lacquer®" is sometimes combined with systemic therapies for full thickness nail infections.

Studies have shown mycotic cure rates of 76–82% and a relapse rate of 20% for terbinafine and a mycotic cure rates of 63% for itraconazole [2, 3]. These treatments are not licensed in children and should not be used in pregnancy. (see patient information leaflet on treating fungal nail infection, Chap. 66).

**Mold infections** of the nails such as *Scopulariopsis Brevicaulis* and *Fusarium* species are rare and can be very difficult to treat. Options include removal of the nail followed by topical and oral antifungals for at least 3 months.

Lasers such as Nd:YAG or diode have been shown to have some benefit in fungal nail infections. They heat the infected tissue with infrared radiation which is thought to kill fungi after a few sessions. Some lasers have been approved by the FDA for treating fungal nail infection (see Chap. 31). Photodynamic Therapy (PDT) has also been used with some success in fungal nail infections.

All the various types of benign and malignant skin **tumors** may occur around or under the nail bed. Periungual warts are common in nail pickers or biters. They can be unsightly and difficult to treat (see Chaps. 34 and 59).

**SCC and melanoma** (pigmented and non-pigmented) can all present as a subungual growth that can lift and destroy the nail plate. They have sometimes been mistaken for ingrown toenails or nail trauma.

A **pigmented junctional naevus (mole)** may occur in the nail matrix which can give rise to a dense band of pigment running proximally to distally in the nail plate.

It may indicate the presence of a junctional naevus which may be undergoing malignant transformation. The safest approach with suspicious pigmented change under the nail plate is to arrange to have the nail plate removed and excise the pigmented area for histology.

A **subungual haematoma** is usually obvious with a history of trauma or as a result of repeated minor trauma of the nail against the front of the shoe known as "runners toe" (also called "jogger's toe", "dancer's toes", "tennis toe" or "skier's toe") (Fig. 41.3). A subungual haematoma will cause the nail to slowly separate from the nail bed and the nail will eventually fall off but a new nail will invariably regrow. If there is any doubt about the diagnosis the nail should be removed and if the pigmentation is found in the nail plate a biopsy should be taken to rule out a melanoma. Dermoscopy can help differentiating pigment from blood. If still in doubt, open up a small window on the nail over the "dark area" and drop some hydrogen peroxide. If it bubbles, it is blood. Otherwise, take a biopsy. **Subungual melanoma** may cause extension of brown or black pigment from the nail bed, matrix or nail plate to the adjacent cuticle and proximal or lateral periungual skin (Hutchinson's nail sign).

**Myxoid cysts** (also called a mucous cyst or a digital ganglion cyst) are common and occur between the distal interphalangeal (DIP) joint and the base of the nail. It forms a small cystic lesion with a smooth, shiny, almost translucent surface which may give rise to a longitudinal depression on the nail plate (Fig. 36.23. Chap 36). Like gan-

glions, a myxoid cyst is linked to the distal interphalangeal joint (DIP) by a tiny sinus tract. The simplest treatment is to puncture the cyst with a needle and squeeze out the clear jelly-like contents under sterile conditions. This may have to be repeated a few times. Other options include cryosurgery, aspiration and steroid injection or surgical removal [4].

**Subungual exostosis** occurs as a result of a bony spur that grows up from the terminal phalanx pushing the nail upwards usually on one side. It is most commonly found under the 1<sup>st</sup> toenail in young adults and can be confirmed on X-ray. It is harmless but is normally removed by an orthopedic surgeon for comfort and cosmetic reasons.

**Ingrown toenails** most commonly occur on the first toe as a result of incorrect nail cutting (i.e. cutting down the side of the nail instead of cutting straight across the top), tearing the nail with the fingers or poorly fitting footwear (Figs. 41.6, 41.7, and 41.8). Some cases can be hereditary as the size and shape of the nail can be inherited. In many cases the nail is too large for the toe resulting in ingrowing, especially if there is pressure from ill fitting shoes or incorrect nail cutting or breaking the toenail with the fingers.

Infected ingrown toenails should be treated with a topical or oral antibiotic. Milder cases may respond to a potent topical steroid with a topical antibiotic such as "Fucibet Cream®" for 1–2 weeks which may reduce the infection and swelling sufficiently to alleviate the problem. It may help to put a tiny piece of cotton under the nail on the affected side to help lift the side skin away from the nail. Sometimes the chiropodist or podiatrist can help.

Removing part or the entire nail will resolve the problem in the short term but up to 80% will become ingrown again once the nail grows back. The most successful and suitable treatment in general practice is to remove a quarter of the nail on the affected side under ring block anesthesia and to ablate the underlying nail matrix with 80%

**Fig. 41.6** Ingrown toenails in a 35 year old



**Fig. 41.7** Ingrown toenails. Pincer nails in a 33 year old



**Fig. 41.8** Ingrown toenail of the right first toe. This patient had a chemical matricectomy using phenol (phenolization) on the left hallux 9 years before



fresh phenol for three minutes in a bloodless field. The nail will not regrow in this area and there is a relapse rate of less than 5% [5]. Radiosurgery can also be used instead of phenol to destroy the nail bed after a wedge resection. Consider doing a bilateral wedge resection even if the opposite side is not ingrown at the time of surgery, as a high number of patients will develop another ingrown toenail on the opposite side of the toe at some stage in the future.

Overgrowth of the nail plate (**onychogryphosis**) most commonly occurs as a result of trauma or can occur spontaneously in the elderly. Treatment is either palliative by trimming, usually by a chiropodist, or radical where the affected nail is removed and the nail matrix treated with phenol or radiosurgery as above, thus ensuring no regrowth (Figs. 41.9 and 41.10).

White spots in the nails (**leukonychia**) are very common and, despite popular beliefs, are not due to calcium deficiency. **Leukonychia** causes whitening of the nails which may occur as a result of minor trauma, superficial tinea infection, metabolic disease, systemic disease or drugs. One of the rare causes of whitening of the nail plate is **hereditary leukonychia** which may involve all of the nail (leukonychia totalis), only part of the nail (leukonychia partialis), or can appear as one or more transverse bands (leukonychia striata) (Fig. 41.11) or white spots (leukonychia punctata). The bands usually appear at the base of the nail and gradually grow up to the free end. The nails are smooth on the surface and do not show any other changes. This is usually



**Fig. 41.9** Onychogryphosis in a 47 year old

inherited as an autosomal dominant trait. It is harmless and there is no known treatment.

### 41.3 Nail Changes Associated with Common Dermatoses

Nail changes can provide a clue in diagnosing certain systemic diseases and some nail changes can be a presenting feature before other signs of a systemic disease become clinically apparent [6].

**Psoriasis** may cause characteristic changes in the nails which can be useful in diagnosis. Pitting, onycholysis (partial separation of the distal nail from the nail bed) and subungual thickening are frequently seen (Fig. 41.12). Occasionally these psoriatic nail changes may be found in a patient without other signs of psoriasis elsewhere. They may precede the onset of psoriasis. Nails are



**Fig. 41.10** Onychogryphosis



**Fig. 41.11** Transverse leukonychia



**Fig. 41.12** Pitting in psoriasis

more likely to be involved in fingers that are affected by psoriatic arthritis. Pitting may also be seen in eczema, alopecia areata and vitiligo. In addition to psoriasis, onycholysis may also be caused by trauma, eczema, pregnancy, thyroid disease, anemia and certain drugs. Patients with psoriasis may coincidentally develop fungal infection of the nails. Taking nail clippings for fungal stain and culture may help identify if there is a fungal infection present.

Treatment of psoriatic nail dystrophy is difficult. Some cases clear spontaneously or during systemic treatment of the psoriasis. Using a potent topical steroid lotion, which can be flooded under the nail if onycholysis is present, may help some cases and is worth trying for 6 to 12 weeks provided nail clippings for fungal stain and culture are negative.

**Eczema** of the distal fingers which involve the cuticles may be associated with rippling and thickening of the fingernails.

**Lichen planus** may cause nail changes in up to 10% of cases such as longitudinal ridging, loss of lustre or brittle nails. Permanent loss of the nails may occur in atrophic lichen planus (see Chap. 19). **Alopecia areata** and **vitiligo** may also be associated with temporary or permanent shedding of the nails.

**Systemic lupus erythematosus** and **dermatomyositis** may be associated with dilated nail fold capillaries just proximal to the cuticles. **Tuberous sclerosis** can cause periungual fibromas (see Chap. 28) and **Darier's disease** can cause longitudinal ridging with V-shaped notches at the distal end of the nail plate. Recently, a red half-moon nail sign has been described as a novel manifestation of coronavirus infection [7].

## 41.4 The Nail in Systemic Disease

A systemic illness or injury may cause temporary decreased growth in the nail matrix which will subsequently become apparent as a transverse ridge across all the nails (**Beau's lines**) which will grow out over the following few months. It is possible to date an illness retrospectively by the position of these lines (e.g. if the line is half way up the finger nails the insult occurred approximately 3 months previously as it takes six months for a finger nail to grow).

**Koilonychia** (spoon nails) is seen in chronic iron deficiency anaemia but may also be seen in thyroid disease, as a result of constant contact with industrial oils or occasionally as a familial disorder. Addison's disease may cause diffuse nail pigmentation. The **yellow nail syndrome** is manifest by thickened yellow nails which are curved on their long axis. This is often associated with lymphoedema and bronchiectasis. These nail features may also be found with hypothyroidism, nephrotic syndrome and certain drugs.

**Clubbing** of the fingernails can be idiopathic or secondary to an underlying condition like chronic lung disease, bronchogenic carcinoma or cyanotic heart disease. Clubbing may also occur as a rare inherited condition and with thyroid disease, primary biliary cirrhosis, ulcerative colitis or Crohn's disease.

Linear (splinter) haemorrhage in the distal half of the nail bed may be caused by trauma, bacterial endocarditis, mitral stenosis, hypertension or dialysis.

Peripheral vascular disease, Raynaud's, lichen planus and systemic sclerosis may cause atrophic nail changes where part of the nail is completely destroyed and replaced by the proximal nail fold skin and cuticle which grows down over and through the nail plate, giving rise to the triangular formation known as **pterygium**.

## 41.5 Conclusion

Nail changes can give us clues to underlying dermatosis and illnesses. They should be examined with good light and magnification, as sometimes the signs may be subtle (e.g. nail pitting). Cuticle damage, which can lead to chronic paronychia, is one of the more common causes of finger nail dystrophy. Traumatic onycholysis is the most common cause of nail dystrophy which is frequently misdiagnosed as tinea infection. Tinea infection is common in toe nails and the most common fungus is the same one that usually causes athlete's foot (*Trichophyton rubrum*). Fungal infection of the nails can look like psoriasis of the nails and the two can coexist. Nail clippings should always be taken to confirm a fungal infection before starting a course of oral antifungal treatment.

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**Part IX**

**Lesion Recognition**



# Lesion Recognition

42

David Buckley

## Key Points

- With careful examination, using good light and magnification, most lesions can be easily recognised and diagnosed with the naked eye by doctors with training in lesion recognition.
- Overlying crust or scale should be removed carefully with a scalpel blade to reveal the base of a lesion.
- Dermoscopy helps greatly in making an accurate clinical diagnosis of both benign and malignant lesions, especially melanomas and BCCs.
- Any lesion where a confident clinical *named* diagnosis cannot be made should be viewed with suspicion and the patient should be referred to a colleague with more experience in lesion recognition or the lesion should be biopsied at the earliest opportunity.
- Skin lesions do not always present as the classical text book description.

## What to Tell the Patient

- You should go to see your doctor if you have a new or changing growth, “sore” or mole, especially if it looks different than any other lesion on the body or if it is tender or bleeding.

## 42.1 Introduction

A lesion is any single area of altered skin. It may be solitary or multiple. Lesions may be **macular** (flat), **papular** (raised), or **nodular** (solid) (Table 42.1). Lesions can be further subdivided according to their configuration, such as their shape or outline. Some lesions can be discoid (coin shaped), linear (in a line), or annular (lesions grouped in circle like a ring). Lesions can also be categorised according to their colour: pigmented (brown, black, gray or blue) or non-pigmented (skin coloured, red, purple or white).

With careful examination, using good light and magnification, most lesions can be easily recognised and diagnosed clinically by a doctor experienced in lesion recognition. Overlying crust or scale should be removed carefully with a scalpel blade to reveal the base of the lesion. A good dermatology atlas or website with lots of

**Table 42.1** Termology in lesion recognition

**Macular** = a small area of flat colour change <1 cm diameter

**Papular** = a small, raised palpable lesion <1 cm diameter

**Nodular** = a solid, enlargement of a papule in three dimensions: height, width and length > 1 cm in diameter

**Ulcer** = full thickness loss of epidermis or dermis and may involve subcutaneous tissue. An ulcer heals with a scar. It may be covered with an **eschar** (scab)

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photos is very useful when trying to identify lesions (e.g. [www.dermnetnz.org](http://www.dermnetnz.org) or [www.dermis.net](http://www.dermis.net) or [www.dermatlas.net](http://www.dermatlas.net)). VisualDx (<https://www.visualdx.com/>) is a diagnostic clinical decision support system. Their large collection of dermatologic skin images are used algorithms to enhance diagnostic accuracy, aiding physician to make therapeutic decisions.

## 42.2 Benign versus Malignant Skin Lesions

The main decision to make when examining a lesion is to decide whether it is benign or malignant. Even more important is not to miss a melanoma which, unlike most other forms of skin cancer, can grow rapidly, spread beyond the skin quickly and can be life threatening, even in young adults. A recent study in general practice showed that almost a quarter of the invasive melanomas seen had little or no pigment (amelanotic or hypomelanotic) [1]. Melanomas can mimic other tumours and so all skin lesions should be examined carefully to rule out a melanoma, whether they are pigmented or non-pigmented (Tables 42.2 and 42.3).

**Table 42.2** The Buckley four point warning signs for skin cancer

### Skin Cancer Screening

These are the warning signs that a lesion (a growth, a sore or freckle or a mole) that is present or changing for more than six to twelve weeks may be turning cancerous in adults: -

#### New Cancers Do Show

- **N**ew - A **n**ew growth, sore, freckle or mole in the last 6 to 12 weeks.
- **C**hanging - A growth, sore, freckle or mole that is **c**hanging in size, shape or color over the past 6 to 12 weeks.
- **D**ifferent - A growth, sore, freckle or mole that looks, feels or behaves **d**ifferently from any other growth, sore or mole on the body (the "ugly duckling").
- **S**ore - A growth, sore, freckle or mole that is **s**ore, tender to touch, bleeding or itchy and will not heal after 6-12 weeks.

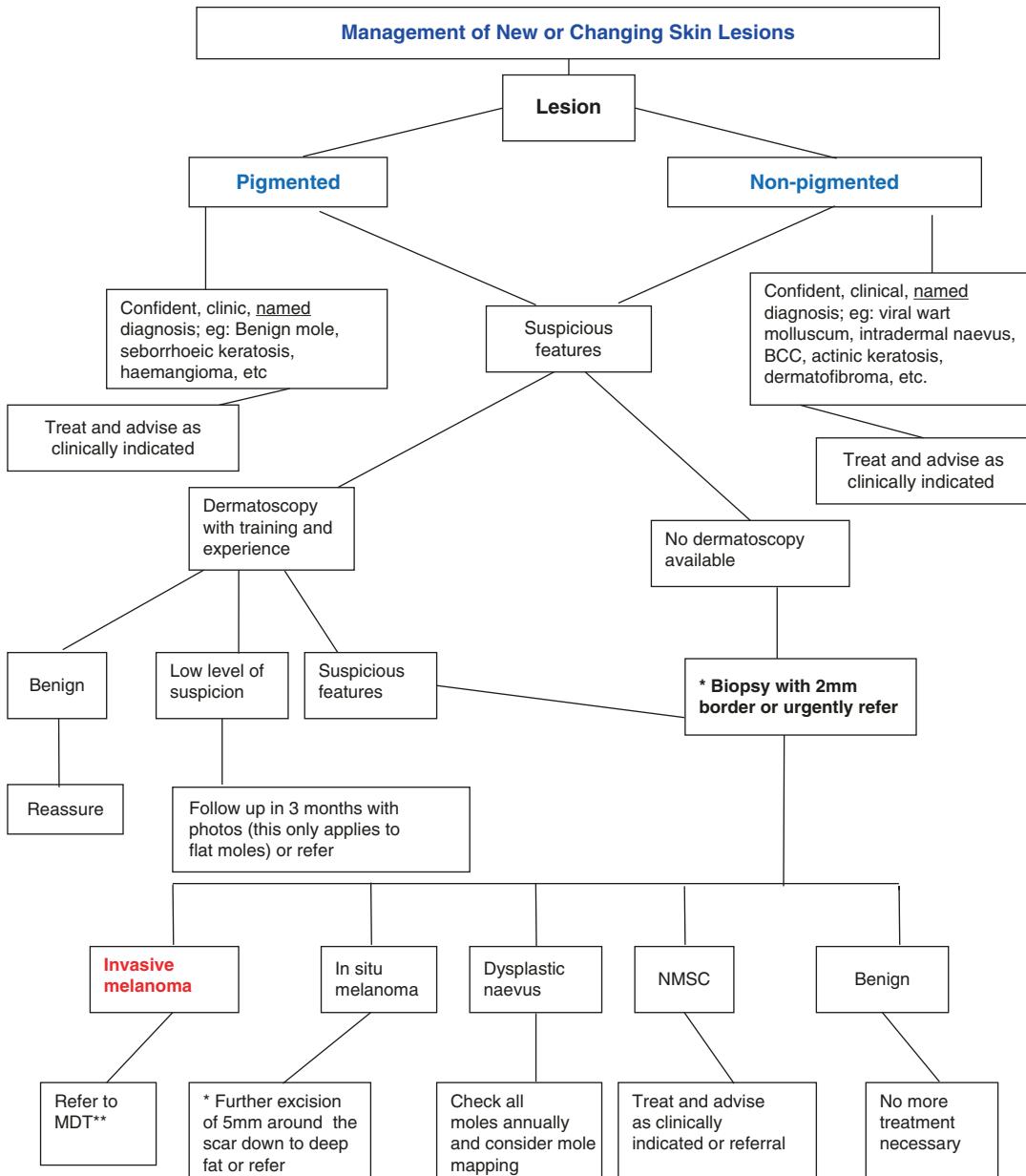
If a growth, sore, freckle or mole shows one or more of these warning signs it should be checked by your doctor. The more warning signs, the greater the risk of skin cancer. Early detection saves lives!

**Table 42.3** Warning signs for skin cancer (both melanoma and NMSC)

- A "mole" that is changing in size, shape or colour
- A "mole" that looks completely different than all the other surrounding moles (the ugly duckling sign)
- A new lesion which continues to grow especially if it is bleeding, crusting or tender
- An ulcerating lesion that fails to heal despite good wound care after a few weeks
- A persistent, isolated, scaly plaque which fails to clear despite appropriate topical therapies

The recently launched NCCP guidelines on melanoma in Ireland encourage all GPs to refer all lesions suspicious of melanoma to a Pigmented Lesion Clinic which are usually located in tertiary referral hospitals [2]. However, the standard of lesion recognition amongst GPs is very varied. Some are highly skilled and experienced including dermoscopy training while others have little or no training in lesion recognition. There is already evidence that Pigmented Lesion clinics are being overrun with mostly harmless benign lesions leading to excessively long waiting times for those who turn out to have a melanoma [3, 4]. Pigmented lesion clinics may also fail to pick up non-pigmented melanomas. In a recent audit of the melanomas in general practice, amelanotic melanomas were biopsied as quickly as pigmented melanomas (average of 9 days vs 8 days) [1]. Studies have shown that the outcome is not affected by who carries out the initial excision of a suspicious lesion or where the initial diagnostic excision is carried out (e.g. by a GP with experience in skin surgery versus a dermatologist, or a plastic surgeon) [5-11].

The Primary Care Surgical Association has an algorithm for assessing skin lesions with a view to ruling out melanomas [12] (Table 42.4). Any lesion where a confident clinical *named* diagnosis cannot be made should be viewed with suspicion and the patient should be referred to a colleague with more experience in lesion recognition or the lesion should be biopsied at the earliest opportunity (Table 42.3). When biopsying a lesion suspicious of a melanoma, a complete excisional biopsy should be performed, removing the entire lesion with a 2 mm border of clear skin all around and including the upper subcutis and a generous cuff of fat. The specimen should be sent

**Table 42.4** PCSA Management of New or Changing Lesions Algorithm

to a histo-pathologist with experience in examining skin lesions and melanomas. The Primary Care Surgical Association recommends that only doctors with experience in skin surgery and skin cancer should carry out this type of work. GPs should not treat invasive melanomas. If an inva-

sive melanoma is found then the patient should be referred immediately and urgently to the regional melanoma multidisciplinary team (MDT) for further staging and treatment.

According to the PCSA guidelines, if a suspicious pigmented lesion is seen by a GP who does

not have experience in skin cancer and skin surgery, then the patient should be referred urgently to a colleague who has these skills or else to a pigmented lesion clinic or a plastic surgeon, which are located in regional or university hospitals throughout Ireland. Referral to the pigmented lesion clinics should be via the NCCP cancer referral template. The choice of whether to refer to a colleague or to the regional pigmented lesion clinic will be dictated by how quickly an appointment can be made for the patient, how quickly a diagnostic excisional biopsy can be carried out if required, the level of suspicion of a melanoma and the preference of the patient. Studies have shown that biopsies of suspicious skin lesions in primary care by GPs with experience in skin cancer and skin surgery can lead to more rapid diagnosis and a quicker pathway to definitive treatment of the patient found to have melanoma. This is also lower costs and more convenience to the patient, especially those who live outside major urban areas [1].

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### 42.3 Dermoscopy

Dermoscopy helps greatly in making an accurate clinical diagnosis of both benign and malignant lesions, especially pigmented lesions (see Chap. 46). In Australia, a dermoscope is as common as a stethoscope in general practice. Dermoscopic images are easy to take with a digital camera attached to the dermoscope and are very useful when following up flat pigmented lesions where the diagnosis of melanoma is unlikely. The clinical and dermoscopy features can be followed up after three months, looking for a change in the lesion. This is known as serial digital dermoscopic imaging. Clinical and dermoscopy images are also useful as they can be shared on various on-line forums to be viewed by other experienced doctors who may give an opinion on the possible diagnosis. When referring a patient, a clinical history and dermoscopy images can alert the doctor receiving the referral about the nature of the lesion and the likelihood of it being a melanoma (see Chap. 6).

*Nodular* lesions should not be monitored. Even if a nodular lesion has a low suspicion of

melanoma, it should be excised or referred urgently, as nodular melanoma can grow rapidly and spread beyond the skin early and are therefore not suitable for follow-up monitoring.

Dermoscopy is a science in itself. Interpretation of lesions using dermoscopy requires at least a proper training course on basic dermoscopy by an expert dermatologist. Ongoing training in more intermediate and advanced dermoscopy is also advisable. The University of Wales College of Medicine run a distant learning course on dermoscopy over twelve weeks which is easily accessible to most doctors, as most of the teaching is done online [13]. The Primary Care Surgical Association ([www.pcsa.ie](http://www.pcsa.ie)), the Primary Care Dermatology Association of Ireland ([www.pcdis.com](http://www.pcdis.com)) and the Primary Care Dermatology Society (UK) ([www.pcds.co.uk](http://www.pcds.co.uk)) also run beginners and advanced dermoscopy courses. The International Dermoscopy society is a free access society with journal, dermoscopedia, congresses and course information (<https://dermoscopy-ids.org/>).

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### 42.4 Clinical Examination of Skin Lesions

When examining an individual lesion, use good light and magnification and pay particular attention to the size, colour, shape, volume and texture of the lesion. Also assess whether the lesion is tender or not. Any adjacent lesions and skin lesions on all other parts of the body should be assessed, as clues may be found that may help clinch the diagnosis. For example, if there is a pigmented, slightly scaly, waxy, stuck on lesion on the patient's forehead and the patient has multiple seborrhoeic keratosis on their back, then the most likely diagnosis would be a seborrhoeic keratosis on the forehead.

Solitary lesions, particularly if they are new, growing, crusting, bleeding or tender to touch should always be viewed with suspicion, as these lesions are more likely to be malignant. On the other hand, multiple lesions which all look similar are usually benign such as stable moles, warts, molluscum contagiosum or seborrhoeic keratosis.

In Chap. 43, pigmented lesions will be discussed and the following chapter we will look at non-pigmented lesions. It is important to realise that there is a lot of crossover. Lesions that are normally non-pigmented may present as a pigmented lesion such as a pigmented BCC and lesions that are normally pigmented such as a melanoma, may present as a non-pigmented lesion (e.g. amelanotic or hypomelanotic melanoma). Other lesions such as intradermal naevi may be pigmented or non-pigmented, even in the same patient.

Skin lesions do not always present as the classical text book description. In fact it can often be impossible to differentiate a BCC from an SCC or an actinic keratosis from an area of Bowen's disease or an early SCC on clinical grounds alone. Some tumours may have mixed pathology (e.g. basosquamous carcinoma) and occasionally two tumours may grow beside each other (collision tumours). In primary care the most important decision is to decide if the lesion is suspicious and warrants further investigation such as biopsy or referral. The exact diagnosis will be made with the assistance of the pathologist.

## 42.5 Conclusion

Experience in lesion recognition will only be achieved by comprehensive training and experience. A good atlas or website may also help gain experience in lesion recognition. The most important factor when faced with a suspicious lesion is to consider if there is any possibility it could be a melanoma. If the doctor cannot make a confident, named diagnosis on clinical grounds, the patient should be referred to a colleague with more experienced in lesion recognition and dermoscopy. If the doctor with experience in lesion recognition and dermoscopy cannot make a clinical diagnosis, then the safest option is to organize to have the lesion removed completely for histological diagnosis giving the histopathologist as much clinical

information as possible together with a reasonable list of differential diagnoses.

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# Pigmented Lesions

43

David Buckley

## Key Points

There is only one rule when assessing suspicious pigmented (or non pigmented) lesions—"if in doubt, cut it out or refer to a colleague with more experience in skin lesion recognition".

## What to Tell the Patient

Most melanomas appear as a new mole or naevus in adults. If you find a new mole and you are more than 40 years old, show it to your doctor.

If a mole (new or existing) is changing in size, shape or colour it should be viewed with suspicion and shown to your doctor.

## 43.1 Introduction

The word pigment is derived from the Latin word meaning "colour or colouring". A pigmented lesion is any lesion that shows any shades of brown, black, gray or blue (Table 43.1).

Pigmented lesions are further divided into:

- Macular (flat)
- Ulcerated (break in the skin)
- Nodular (solid and raised up off the skin)

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**Table 43.1** Normal skin pigmentation is influenced by the following

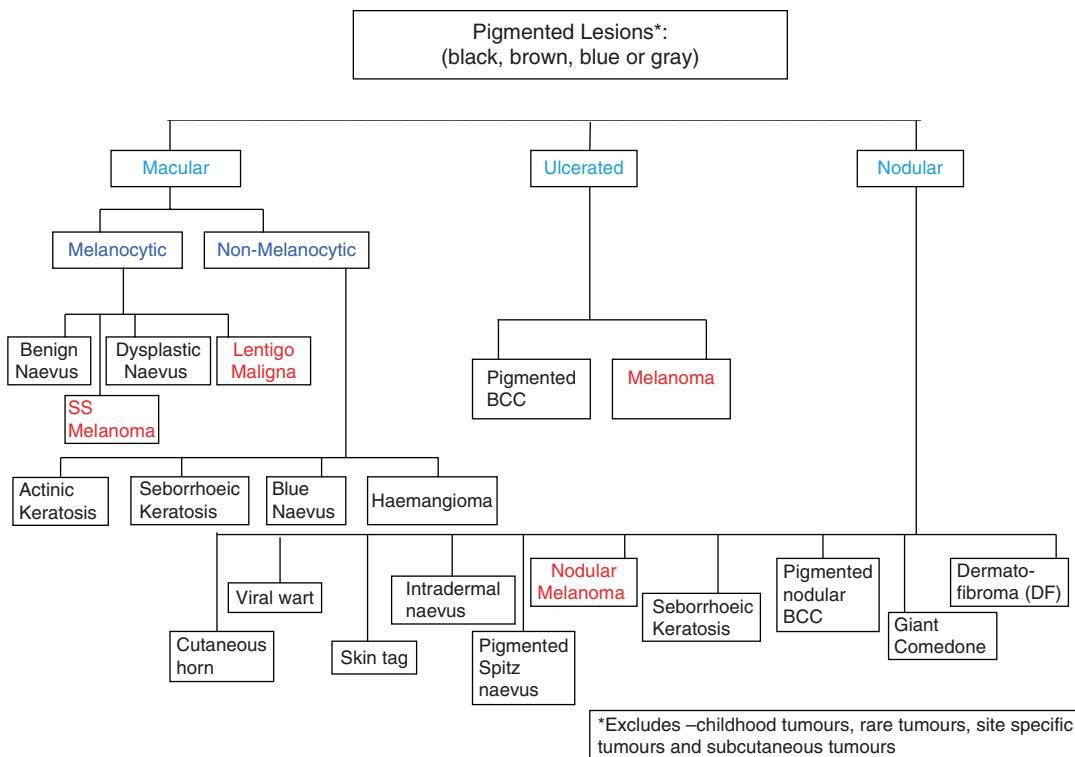
- Degree of vascularity
- Amount and depth of melanin (e.g. black = in epidermis, blue = in dermis)
- Presence of carotene
- Thickness of the horny layer (keratin)

The algorithm (Fig. 43.1) covers common and dangerous skin lesions but does not include rare or unusual lesions. In addition, this chart does not take into consideration the age of the patient. Obviously, certain lesions are more common in certain age groups. For example, children tend to have congenital moles, adults tend to get seborrhoeic keratosis and elderly patients get pigmented actinic keratosis and pigmented non-melanoma skin cancers. Melanomas can occur at any age but are extremely rare before puberty.

Pigmented macular lesions can be further subdivided into melanocytic (i.e. derived from melanocytes) or non-melanocytic, by the use of dermoscopy in trained hands.

### 43.1.1 Pigmented Macular (Flat) Lesions

Any brown, black, gray or deep blue flat lesion should always be examined carefully to rule out a superficial spreading malignant melanoma or a dysplastic naevus. The suspicion should be even



**Fig. 43.1** Algorithm of pigmented skin lesions

higher if there is a history of a change in the lesion such as change in size, shape or colour. Benign moles are stable and do not change and so the history is very important when assessing moles. Sometimes there may be no history available as the mole may be on a part of the body that is not easily visible to the patient (on the back, on the calf or on the sole of the feet). There is only one rule when assessing suspicious pigmented (or non pigmented) lesions—“if in doubt, cut it out or refer to a colleague with more experience in skin lesion recognition”.

A useful diagnostic test is the revised 7-point checklist (Table 43.2). This test was found to have a high sensitivity, but low specificity. This means, they are good at catching up the bad guys (melanomas) but they also come with a fairly high numbers of false positives (excisions of lesions suspected as melanomas that were not),

**Table 43.2** Revised 7-point checklist for assessing risk of melanoma

Suspect melanoma if there are 1 or more **major signs** in a mole:

1. Change in **size** (diameter or height getting bigger)
2. Change in **shape** (notched or ragged border)
3. Change in **colour** (2 or more irregular colours including white)

3 or 4 **minor signs** without a major sign can also indicate a need to biopsy suspicious moles:

1. Inflammation
2. Crusting or bleeding
3. Sensory change (itch or soreness)
4. Diameter ( $\geq 7$  mm) (but melanomas can be as small as 3 mm)

leading to possibly unnecessary biopsies and increased patient anxiety [1–3].

If in doubt, it is definitively better to have a suspicious lesion cut out and get the result showing it was benign than leaving a melanoma undiagnosed.

Another useful sign of **melanoma** is “the ugly duckling sign”. This is where there is a mole that looks completely different from all the other moles on the patient’s body. Unless there is a very definite history that the ugly duckling mole is not changing, it is safer to remove it for histological diagnosis.

71% of melanomas arise as brand new lesions while only 29% arise from within an existing mole or freckle which starts to grow larger or change in shape or colour [4].

Patients who have had a previous melanoma or a non-melanoma skin cancer or patients with a family history of these lesions or pancreatic cancer are more at risk of developing melanomas. Other high risk patients are those with fair skin that burn easily (Fitzpatrick skin type 1 and 2), patients with multiple moles ( $>100$ ), particularly if they are large and have an irregular edge or colour (dysplastic naevi).

Older patients can develop **lentigo maligna**, which is considered a melanoma in situ. These are usually slow growing, flat lesions with irregular colour and edges that usually occur on the face or other exposed areas of the body. In the elderly they can grow to quite a large size and they are often mistaken for simple lentigos (sunspots) or seborrhoeic keratosis. Even though they grow slowly, these in situ melanomas may eventually progress into a nodular melanoma (lentigo maligna melanoma).

Lentigo maligna present a unique challenge, as they are often large, mostly on the face and usually occur in elderly patients with significant comorbidities and limited life expectancy. All lentigo maligna patients should be offered surgical excision, but some refuse. Imiquimod 5% cream (“Aldara®”) and cryosurgery have shown some success in patients who refuse surgery.

Other lesions that may be pigmented and flat are pigmented actinic keratosis (they are usually flesh coloured and slightly rough to the feel), a flat seborrhoeic keratosis (they are usually nodular and scaly), a blue naevus or a haemangioma. The last two lesions are normally easily diagnosed with dermoscopy for those with training and experience in this technique.

### 43.1.2 Pigmented Ulcerated Lesions

Ulcerating pigmented lesions could be a melanoma (always ask: is it changing in size, shape or colour?) or a pigmented ulcerating BCC. These lesions should be excised completely for histological diagnosis or referred to a colleague with more experience in skin lesion recognition.

### 43.1.3 Pigmented Nodular (Raised Up) Lesions

Nodular-pigmented lesions can be further subdivided into:

- scaly/warty nodules
- smooth dome shaped nodules
- fleshy ulcerating nodules

### 43.1.4 Pigmented, Scaly/Warty Nodules

The most common lesion to present in this way is a **seborrhoeic keratosis (SK)** (also called a seborrhoeic wart or basal cell papilloma). These usually have a raised, scaly, waxy surface and a “stuck-on” appearance with a sharply demarcated border on the skin and are common in people over the age of 40 years old. They can be solitary or multiple, may be small or can grow up to 1–2 cm in diameter and can occur on any part of the body apart from the palms and soles but are most common on the trunk and face. They are usually brown but can be flesh coloured, yellow, brown, gray or black. (Figs. 43.2, 43.3 and



**Fig. 43.2** Seborrhoeic keratosis

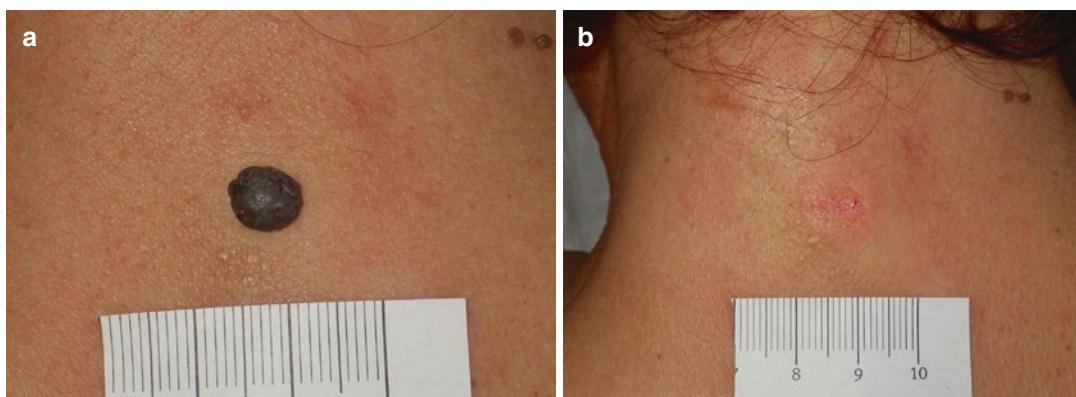
**43.4a, b).** On the face they can be flat (macular) and can be difficult to distinguish from an actinic keratosis, Bowen's disease, melanoma or lentigo maligna (Fig. 43.5). A sudden eruption of multiple new SKs may be associated with underlying adenocarcinoma of the breast, stomach, ovaries or uterus. This is known as the sign of Leser-Trélat.

Dermoscopy can be very helpful in making the diagnosis as SKs have a number of typical dermoscopy features such as milia-like cysts, comedo-like openings, cerebriform ("brain-like") surface and hairpin vessels (see Fig. 43.6).

There are a number of clinical variants of SK and some can be only diagnosed with histology (see Table 43.3).



**Fig. 43.3** Seborrheic keratosis



**Fig. 43.4** (a) Seborrheic keratosis posterior neck. (b) Same seborrheic keratosis immediately post-shave biopsy and aluminium chloride for haemostasis

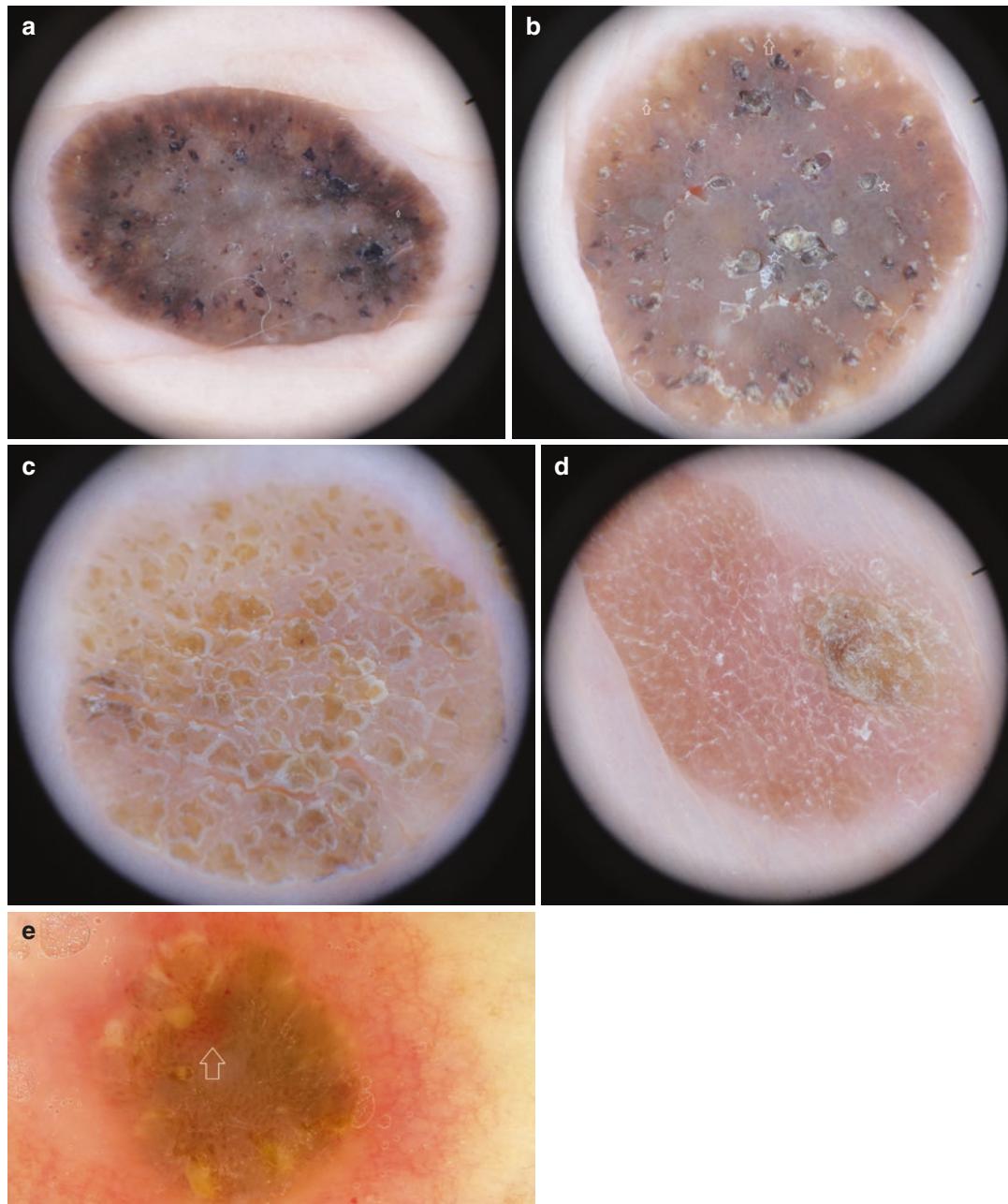
SKs are harmless and benign but are sometimes removed if they are unsightly or uncomfortable or if the clinical and dermoscopy diagnosis is not clear. When a SK becomes itchy, bleeds, becomes red or inflamed or if it looks completely different than all the others, it should be biopsied (Table 43.4).

They can be easily removed by curettage or shave biopsy under local anaesthetic or by cryobiopsy (freezing, shaving and applying a haemostatic solution). The way they easily separate from the underlying skin and lack of any underlying skin abnormality apart from light capillary bleeding, further supports the diagnosis of a seborrheic keratosis. Always send removed tissue for histology. Haemostasis can be easily achieved with 20% aluminium chloride on a cotton bud (Fig. 43.4a, b). When removed by curettage, it is recommended to freeze the base with light cryosurgery (5 second freeze with one freeze-thaw cycle), as otherwise they will recur.

Other lesions that present as a scaly, warty, pigmented nodule are viral warts, pigmented skin tags (fibro-epithelial polyps), hyperkeratotic actinic keratosis, lichenoid keratosis (a regressing seborrheic keratosis) or solar lentigo, inverted follicular keratosis, SCC, BCC or melanoma (Figs. 43.7, 43.8, and 43.9a, b). Some of these lesions can be diagnosed clinically but if there is any doubt about the diagnosis they should be removed for histology (Fig. 43.10).



**Fig. 43.5** Examples of various types of seborrhoeic keratosis



**Fig. 43.6** Dermoscopy features of SK. (a) SK showing fingerprint like structures, (b) SK showing milia-like cysts and comedo-like openings, (c) SK showing fissures and ridges, (d) SK showing network like structures, (e) SK showing hairpin vessels

#### 43.1.5 Pigmented, Nodular, Smooth, Dome Shaped Lesions

There are a number of lesions that can present like this. The most common would be intrader-

mal naevi (Fig. 43.11) and skin tags. The most serious is a nodular melanoma. Nodular melanomas can spread rapidly beyond the skin and generally have a much worse prognosis than superficial spreading melanomas. Any lesions

**Table 43.3** Clinical variants of SK

Clinical Subtypes	Typical Location	Diagnostic Features
Stucco keratosis	Lower extremities, particularly on ankles	Dry surface, rough; hard, opaque papules; gray-white; easily scrapes off
Dermatosis papulosa nigra	Face	Common on dark-skinned individuals; small hyperpigmented papules; dark brown to black
Inverted follicular keratosis	Face, especially on cheek and upper lip	Firm white-tan to pink papules; usually solitary
Large cell acanthoma	Face or neck, including eyelids	Sharply demarcated papule or plaque; skin-colored to hypopigmented or hyperpigmented; solitary lesion
Lichenoid keratosis	Upper chest or forearms	Often scaly; nonscaly papule or plaque are usually pearly; pink to pink-brown
Macular SK	Sun-exposed areas	Flat, oval, tan-brown patches; increase with age

Noile K, et al. *J Cutan Med Surg.* 2008;12:203-210

**Table 43.4** Differential diagnosis of a SK

Melanoma
BCC
Bowen's/SCC
Actinic keratosis
Wart (commonly on palms and soles, and have pinpoint dots = thrombosed vessels)
Melanocytic naevus
Skin tag/fibroepithelial polyp
Eccrine poroma (benign sweat gland tumour)

suspicious of a nodular melanoma should be dealt with like a breast lump (i.e. they should be either biopsied or referred and seen by a more experienced colleague within 1–2 weeks of presentation). Other less serious lesions that can present like this are haemangiomas, blue naevus, spitz naevus, and dermatofibromas.

**Spitz naevi** are typically dome-shaped, red, reddish-brown (classic Spitz) or darker nodules (pigmented Spitz) and may be up to one or two centimetres in diameter. It usually appears on the face or limbs of children and grows rapidly for a few months. They are benign but can be confused with a melanoma and are usually excised for histological diagnosis.

**A blue naevus** is a dark blue colour because the pigment cells (melanocytes) are deeper in the skin than commoner brown moles and freck-



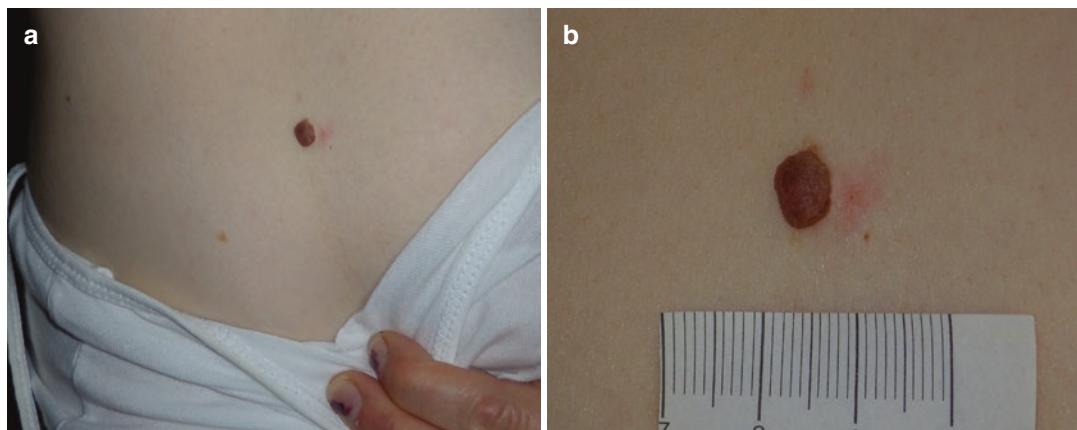
**Fig. 43.7** Solar lentigo



**Fig. 43.8** Solar lentigo on a 73 year old

les. They are harmless, benign and do not need biopsy if stable (Fig. 43.12).

**Dermatofibromas** are usually flesh coloured but may be pigmented or may have a pigmented rim around the outside. Dermatofibromas have a



**Fig. 43.9** (a) Fibro epithelial polyp, (b) Same fibro epithelial polyp close up



**Fig. 43.10** Melanoma 4.5 mm deep on histology

very distinctive and unusual feel. When pinched between your fingers they feel like a firm pebble in the skin. They may also show dimpling (*peau d'orange*) of the surrounding skin when squeezed (Fig. 43.13a, b). On dermoscopy they usually show a central white area surrounded by a faint pigment network. Dermatofibromas are common on women's legs and arms. They are benign and some can resolve spontaneously but this may take many years. Most remain for life. Sometimes patients want them removed for cosmetic reasons, because they itch, when they are raised and make shaving difficult (on the legs in

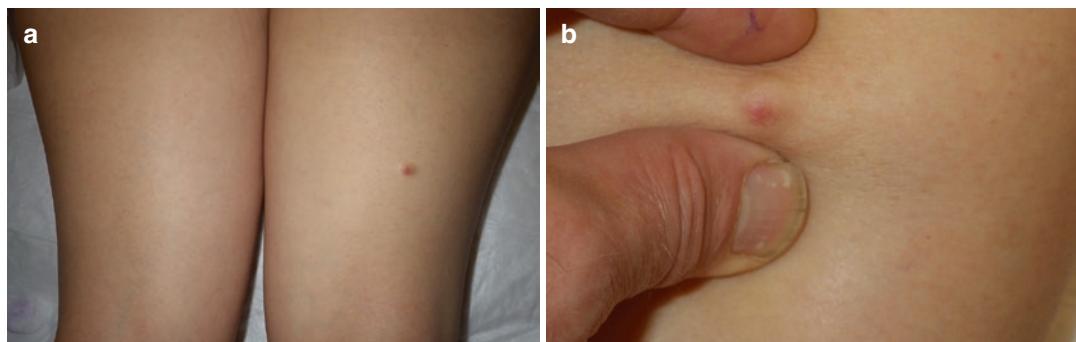


**Fig. 43.11** Intradermal naevus chin



**Fig. 43.12** Blue naevus on the dorsum of the hand

women) or if there is any doubt about the diagnosis. The easiest way to remove them is by punch biopsy or elliptical excision to the edge of the lesion.



**Fig. 43.13** (a) Dermatofibroma on the posterior part of the thigh, (b) Dermatofibroma showing the dimpling sign when squeezed

#### 43.1.6 Pigmented, Fleshy, Ulcerated, Nodule

The main differential diagnosis here would have to be a nodular melanoma which would be a very worrying sign as these usually have a very poor prognosis. They need to be referred on urgently for histological diagnosis and further management. A pigmented nodular BCC that starts to ulcerate can also present like this.

#### 43.2 Conclusion

Most pigmented lesions on the skin are derived from moles or seborrhoeic keratosis. If there is any suspicion that a pigmented lesion could be a melanoma it should be referred to a colleague with more experience in lesion recognition. Most melanomas emerge as new lesions. If a mole

(new or existing) is changing in size, shape or colour it should be viewed with suspicion and biopsied or referred as it could be a melanoma.

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# Non-pigmented Lesions

44

David Buckley

## Key Points

While most **melanomas** are pigmented, some have little or no pigment and can present as a non-pigmented macule or nodule.

- Any new or changing lesion, regardless of its colour, should be viewed with suspicion and biopsied unless a confident, clinical, named diagnosis can be made based on the history, visual inspection and dermoscopy examination.

The algorithm (Fig. 44.1) covers common and dangerous skin lesions but does not include rare or unusual lesions. It also excludes location specific lesions such as lesions that might be commonly found on the eyelid (xanthelasma), the ear (chondrodermatitis nodularis helicis), the fingers (ganglion) or the toes (ingrown toenail). Figure 44.1 only applies to epidermal lesions and excludes subcutaneous lesions such as lipomas and sebaceous cysts.

## What to Tell the Patient

- Any new or changing sore, especially those that look different or are tender or bleeding should be shown to your doctor.

## 44.1 Introduction

A non-pigmented lesion can be skin coloured, red, purple or white.

Non-pigmented lesions can be subdivided into (Fig. 44.1):

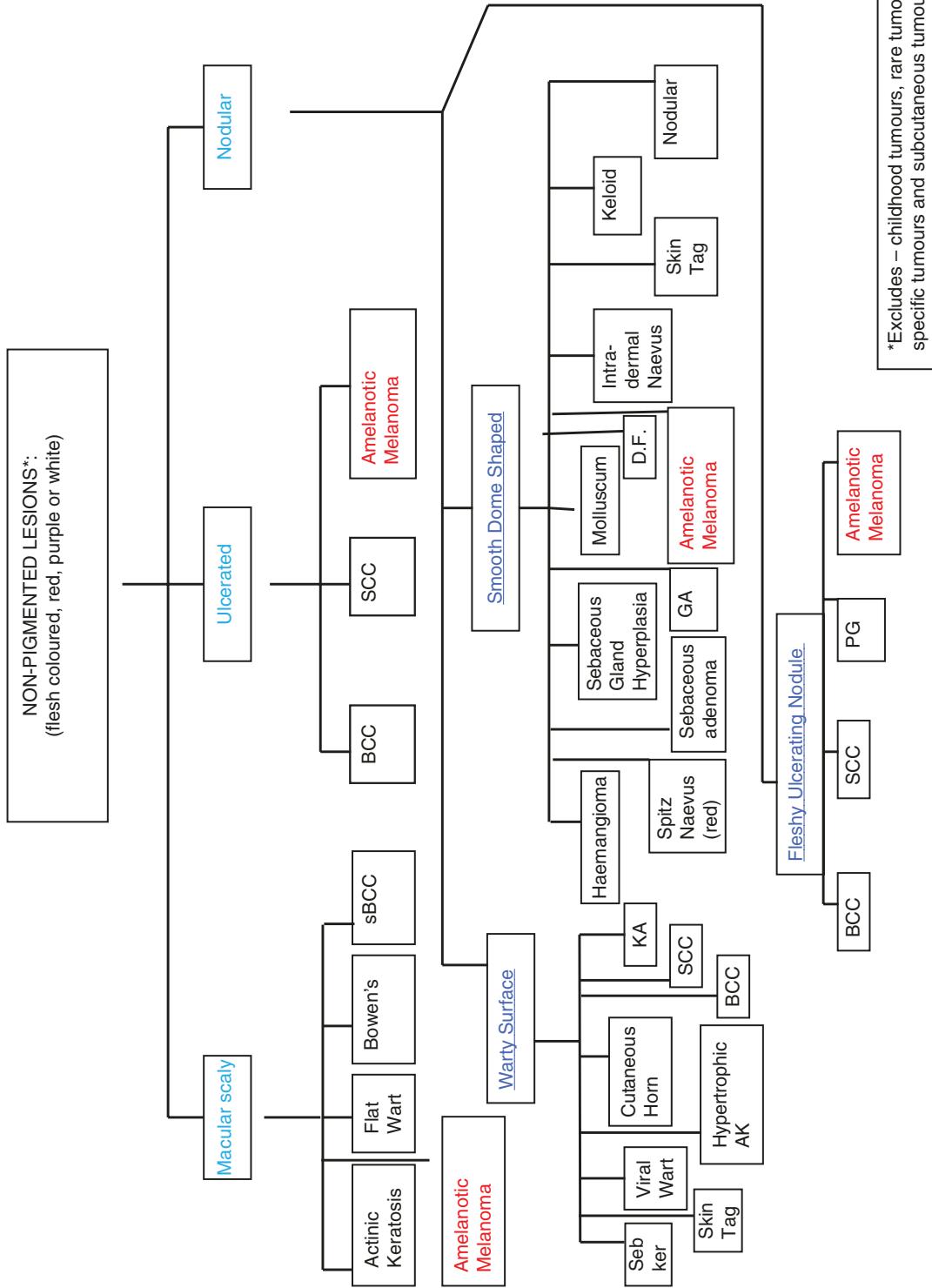
- Macular (flat)
- Ulcerated (broken skin)
- Nodular (solid and raised)

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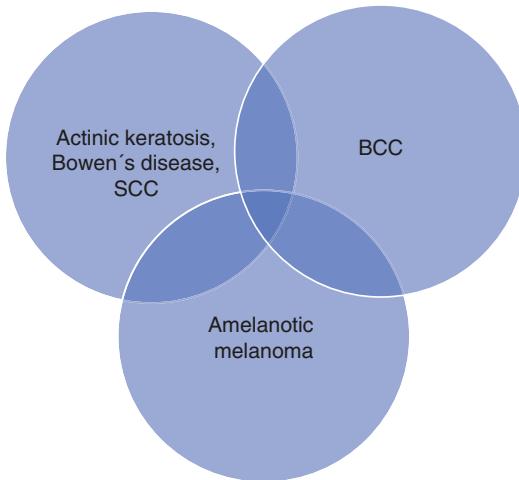
## 44.2 Non-pigmented Macular Lesions

**Actinic keratoses** are usually flat and non-pigmented although they usually have a slightly rough texture on palpation. Sometimes they can be scaly or hypertrophic but paring them down usually reveals the true nature of the lesion. However, it can sometimes be almost impossible to differentiate an actinic keratosis from an area of Bowen's disease, an early squamous cell carcinoma (SCC), a superficial BCC or an amelanotic melanoma by naked eye recognition (Fig. 44.2). Dermoscopy can help make a diagnosis but if there are any doubts about the diagnosis a biopsy should be taken to make an accurate diagnosis before deciding on the most appropriate treatment which includes excision, cryosurgery, 5% fluorouracil ("Efudex®"), imiquimod ("Aldara®,") or photodynamic therapy.



\*Excludes – childhood tumours, rare tumours, site specific tumours and subcutaneous tumours

**Fig. 44.1** Non-pigmented lesions graph (AK = actinic keratosis, KA = keratoacanthoma, GA = granuloma annulare, DF = dermatofibroma, PG = pyogenic granuloma)



**Fig. 44.2** Differential diagnosis of a non-pigmented isolated scaly patch

While most **melanomas** are pigmented, some have little or no pigment and can present as a non-pigmented macule. It has been shown that taking a punch or incisional biopsy from a non-pigmented lesion that subsequently turns out to be a melanoma, will not alter the prognosis once the melanoma is fully excised as soon as possible after the punch biopsy [1, 2]. While it is usually better to remove the complete lesion for histology, a punch or incision biopsy may be appropriate in certain circumstances if the clinical suspicion of melanoma is low [3]. At times, complete excision is not practical for clinical, technical or other reasons, so partial biopsy may be necessary. This may be considered where the lesion is large or on a site where total excision may cause cosmetic or functional impairment or where the patient has significant comorbidities. Multiple biopsies may be required for larger lesions such as a large pigmented macular lesion on the face to differentiate a solar lentigo or seborrhoeic keratosis from a lentigo maligna. All biopsies should include the most suspicious or invasive zones as judged by dermoscopy.

**Seborrhoeic keratosis** is usually raised, rough, oily and pigmented. However, you can sometimes get non-pigmented, flat seborrhoeic keratosis. The diagnosis may be obvious clinically especially if there are numerous other seborrhoeic keratosis in the area. Dermoscopy can

help to diagnosis seborrhoeic keratosis. However, if there are any doubts a biopsy should be taken to confirm the diagnosis.

**Flat warts** can also present as non-pigmented macular lesions. Although we call them flat, they are usually slightly raised, papular and multiple. They are often quite inconspicuous and are more easy to see with a magnifying light or a dermatoscope. They are most commonly found on the face and the back of the hands.

#### 44.3 Non-Pigmented Ulcerating Lesions

Bleeding, oozing, scabby or ulceration in a lesion is usually a bad sign unless there is an obvious cause (e.g. picking, friction, trauma, etc.). If an ulcerating lesion does not heal with good wound care, the possibility of an underlying malignancy should always be considered. The site of the ulcer may help in the diagnosis. If it is on the lower leg it may due to **varicose ulcer, arterial ulcer, diabetic ulcer** or a **pressure sore**. If the ulcer is on a light exposed area (face, neck, ears, scalp, forearm, dorsum of the hands or the lower legs in women) then it may be due to skin cancer (ulcerating **BCC**, ulcerating **SCC** or an ulcerating **melanoma**). If an ulcer does not heal with appropriate treatment, then a biopsy should be taken from the edge of the lesion to confirm the diagnosis and to make a treatment plan.

#### 44.4 Non-Pigmented Nodular Lesions

These can be further subdivided into the following subtypes:

- Scaly/warty nodules
- Fleshy ulcerating nodules
- Smooth dome shaped nodules

##### 44.4.1 Non-Pigmented Scaly/Warty Nodules

The most common lesion to present in this way is a **viral wart**, which is usually obvious because of

its site (usually on the hands) (Fig. 44.3) and the age of the patient (usually children and young adults). An isolated warty lesion on a light exposed area of an older patient (greater than 50 years old) should raise suspicions, as it may in fact be a **hypertrophic actinic keratosis**, a **BCC**, an **SCC** or a **keratoacanthoma** (Fig. 44.4).

**Skin tags (Acrochordons)** can be warty looking but are usually obvious because of their long history and their location (around the eyes, neck, in axilla, under the breast in women and in the groin) (Fig. 44.5a, b). A **cutaneous horn** can look warty and should always be removed with a good cuff of underlying skin as they may arise out of an underlying SCC.

**Seborrhoeic keratosis** are usually scaly, warty, pigmented, stuck-on lesions but can sometimes be non-pigmented. **Hypertrophic actinic keratosis**, **lichenoid keratosis** and **inverted follicular keratosis** can often be difficult to differentiate clinically and may require a biopsy to confirm the diagnosis.

**Keratoacanthomas (KA)** are interesting as they usually grow rapidly over the course or



**Fig. 44.3** Wart on the dorsum of the hand of a 67 year old male

2–6 weeks and may resolve spontaneously over the following 3–6 months. Keratoacanthomas have a flesh coloured nodular base with a characteristic central keratin plug. The problem with keratoacanthomas is that they can be almost impossible to differentiate from a **squamous cell carcinoma** both clinically and histologically. KAs are probably best considered a variant of an **SCC** (e.g. **SCC-KA type**) and as such it is best to excise them completely with at least a 4 mm margin of clear skin all around the lesion. If a KA is



**Fig. 44.4** Squamous cell carcinoma



**Fig. 44.5 (a, b)** Skin tags (Fibroepithelial polyps)

not easy to excise because of its size or site, an incisional biopsy taking a half or three-quarters of the tumour should help the pathologist make an accurate diagnosis before planning definitive surgery. When a KA is cut into for an incisional biopsy, it sometimes stimulates spontaneous remission.

#### 44.4.2 Non-Pigmented Fleshy Ulcerating Nodules

**Pyogenic granulomas** usually grow rapidly and may arise out of a puncture wound. They are a fleshy growth that ooze and bleed easily (Figs. 44.6 and 44.7). Although they can resolve spontaneously, it is best to remove them by shave biopsy or curettage followed by cautery to base as they can bleed profusely once removed. It is vital to send the removed lesion for histology.



**Fig. 44.6** Pyogenic granuloma on the neck of a 12 year old patient

The possibility of an **amelanotic (non-pigmented) melanoma** should always be considered even if the lesion looks typical of a pyogenic granuloma (Fig. 44.8). This is another



**Fig. 44.7** Pyogenic granuloma on the scalp of an 8 year old



**Fig. 44.8** Pyogenic granuloma on an arm that appeared 4 months before. It is mandatory to rule out a melanoma

good reason to remove all pyogenic granulomas for histological diagnosis.

**Basal cell carcinomas, squamous cell carcinomas** and **keratoacanthomas** can occasionally present like this and any new fleshy ulcerating nodule that appears for no reason and continues to grow should be biopsied for histological diagnosis. If the lesion is large (20 or 30 mm) and the suspicion of melanoma is low then a few punch biopsies or an incisional biopsy taken from strategic areas of the tumour should help clinch the diagnosis.

#### 44.4.3 Non-Pigmented Smooth Dome Shaped Nodules

These can be further subdivided into:

- Red lesions
- Small discrete lesions usually less than 5 mm
- Larger lesions usually greater than 5 mm

#### 44.4.4 Red, Smooth, Dome Shapes Nodules

A **haemangioma** (Fig. 44.9) or **Spitz naevus** (Fig. 44.10a, b) can present like this and are usually easily identified with a dermatoscope. An **amelanotic melanoma** could also present like this so if there are any doubts, a lesion should be excised for histological diagnosis.

The most common angioma is the **cherry angioma** (also called Campbell de Morgan spots or senile angioma) which are harmless and benign (Fig. 44.11). They are usually multiple

and are most commonly found on the trunk and face in people over the age of 40 years old. They can be bright red (cherry), purple or deep blue. They can vary in size from 1 to 2 mm up to a centimetre in diameter. They usually have characteristic dermoscopy features (red lacunae). If there is any doubt about the diagnosis they should be removed completely for histology. If they are unsightly or uncomfortable they can be easily treated with cryosurgery, electrosurgery or laser.



**Fig. 44.9** Capillary haemangioma



**Fig. 44.10** Spitz naevus in the arm of an 11-year-old child



**Fig. 44.11** Cherry angioma and seborrhoeic keratosis on the back of a 68 year



**Fig. 44.12** Intradermal naevus present for years

#### 44.4.5 Small, Discrete, Smooth, Dome-Shaped, Non-pigmented Nodules

**Intradermal naevi** are very common on the face and grow very slowly over years. They do not normally bleed, crust or ulcerate (Fig. 44.12). When they get large they can be unsightly or can catch in clothes or jewellery. They can easily be removed by shave biopsy and light cautery and tissue should always be sent for histology.

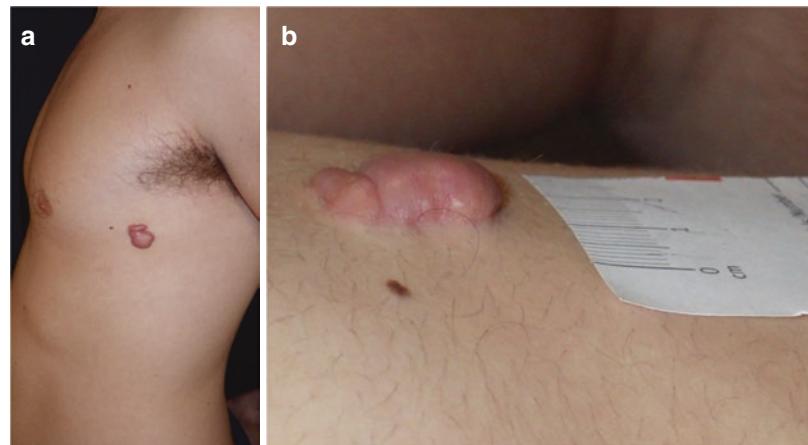
**Sebaceous gland hyperplasia** are also common on the forehead and upper cheeks in middle aged and elderly patient and can sometimes be mistaken for a small nodular BCC. They are usually multiple, small (~3 mm), discrete, yellowish nodules with tiny telangiectasia but no ulceration (Fig. 44.13). A biopsy should be taken if there is any doubt about the diagnosis. They can be treated by cryosurgery or electrocoagulation for cosmetic reasons if required.



**Fig. 44.13** Sebaceous gland hyperplasia on the forehead

**Sebaceous adenomas** can look similar to sebaceous gland hyperplasia. They present as a slowly growing, small (2–4 mm), smooth, yellow, sometimes speckled papules with central umbilication on the skin of the face or scalp in adults. They can sometimes grow to up to 2–5 cm in diameter and occasionally can turn malignant. Multiple sebaceous adenoma may be a marker for the **Muir-Torre Syndrome** with a high risk of developing internal malignancies, particularly the GI tract and GU tract.

**Molluscum contagiosum** are usually small, pearly white nodules with a central open pore at the top. If they are squeezed, they can sometimes expel a cheesy white material. They are usually multiple and clustered together. They are most common in children especially those with atopic eczema and dry skin. They resolve spontaneously after 6–12 months in most children but if there is pressure to treat for cosmetic reasons, the most successful treatment is a very light three second freeze with

**Fig. 44.14 (a, b) Keloids**

liquid nitrogen cryosurgery, as they are extremely sensitive to the cold. This very light freeze is relatively painless and most children can tolerate it without local or topical anaesthetic. Large or multiple molluscum in an adult should make you suspicious of underlying HIV infection. Hydrating cream helps resolve part of the problem.

**Dermatofibromas** have a characteristic, pebbly feel within the skin and are most commonly found on the arms and legs in adult women (see Chap. 43).

#### 44.4.6 Large (>5 mm), Discrete, Smooth, Dome Shaped, Non-pigmented Nodules

If a lesion like this is new and growing, a **nodular BCC** is as strong possibility. The possibility of a BCC would be further heightened if the lesion were oozing, bleeding, crusting or tender. However, **keloids** (Fig. 44.14a, b) and **sebaceous cysts** can also present like this. Keloids usually follow trauma but can arise spontaneously, especially in acne prone skin and in Afro-Caribbean patients. **Sebaceous (epidermoid) cysts** normally have a visible punctum, which helps to

make the diagnosis. **Pilar (trichilemmal) cysts** occur in the scalp and have no punctum but contain similar smelly, soft cheesy material as found in a sebaceous cyst. **Granuloma annulare** can be smooth and dome-shaped but is usually made up of a number of nodules in an annular shape.

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## 44.5 Conclusion

Any new or changing lesion, regardless of its colour, should be viewed with suspicion and biopsied unless a confident, clinical, named diagnosis of a benign lesion can be made on the history, visual inspection and dermoscopy examination if available.

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2. Bong JL, Herd RM, Hunter JA. Incisional biopsy and melanoma prognosis. J Am Acad Dermatol. 2002;46(5):690–4.
3. Clinical practice guidelines for the management of Melanoma. 2008, p. 35. [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/cp111.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/cp111.pdf)



# Cancer and Pre-Cancer of the Skin

45

David Buckley

## Key Points

- The doctor needs skills and knowledge to be able to decide which lesions to ignore, which to treat and how, which to review, which to biopsy and which to refer.
- Any skin lesion where a definitive, named, clinical diagnosis cannot be made should be viewed with suspicion and the lesion should be biopsied or referred to a colleague with more experience in lesion recognition.
- The doctor should always have a high index of suspicion for any new lesion (pigmented or non-pigmented) that is changing in size, shape or colour or looks different from any other lesion on the body and where a confident, named, clinical diagnosis cannot be made.
- Any lesion that is new, firm and growing (NFG) should be viewed with suspicion.
- The incidence of transfer from an actinic keratosis to a squamous cell carcinoma is estimated to be anything from 0.5 to 2% per year but the true figure is unknown.
- Although considered low risk, 5% of Bowen's disease will progress to SCC so it is important to treat them in the majority of patients.
- Many non-melanoma skin cancers (NMSCs) will be small cancers, and relatively simple to excise. Certain locations (the H area of the face

which includes the nose, ears and lower lip) are prone to metastasise early and others (especially the scalp) may require skin grafting once removed surgically.

- Unlike squamous cell carcinomas (SCCs), most basal cell carcinomas (BCCs) have little or no potential to spread beyond the skin and are therefore not life threatening. They can be locally destructive and can cause significant morbidity and health expenditure.
- Diagnosis of BCCs can often be made clinically or with dermoscopy and should be confirmed by biopsy (punch or shave biopsy). Who treats it and how it is treated will depend on the histological subtype, depth of tumour, size in diameter, location, the age and general health of the patient and the skills and experience of the treating doctor.
- Melanoma may develop from an existing mole but most arise de novo.
- The prognosis and definitive treatment for melanoma will be dictated by the depth of the tumour (Breslow thickness) and other high risk features such as ulceration, higher Clark levels (4 or 5), high mitotic activity, lympho-vascular invasion and whether or not there is local or distant metastasis. Early detection saves lives.

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## What the Patient Should Know

- A new mole or growth that is changing, different from any other mole or growth on the skin or is sore or tender should be checked by a doctor. ("New Cancers Do Show").
- Unlike other skin cancers, melanomas can occur at any age but are more common as one gets older.
- A new mole that appears after the age of 40 years old should be viewed as suspicious, and the older the patient, the more suspicious you should be.
- Less than half of all melanomas develop from existing moles. The majority (60–80%) arise from brand new moles.

## 45.1 Introduction

There are a number of common invasive skin cancers (BCC, SCC, melanomas) (see Fig. 45.1). While the classical, clinical textbook description may help diagnose a particular type of cancer, often in primary care it may be difficult, if not impossible to tell if a lesion is cancerous and which particular type of skin cancer it is without

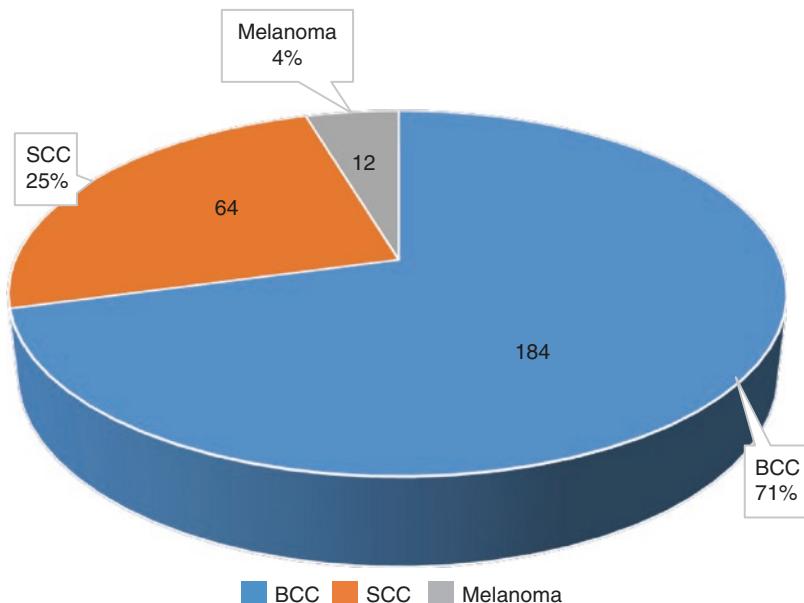
relying on dermoscopy or a skin biopsy. Taking a punch or shave biopsy is a simple procedure that can be easily carried out in primary care. The skill is deciding which lesions to ignore, which to treat and how to treat, which to review, which to biopsy and which to refer.

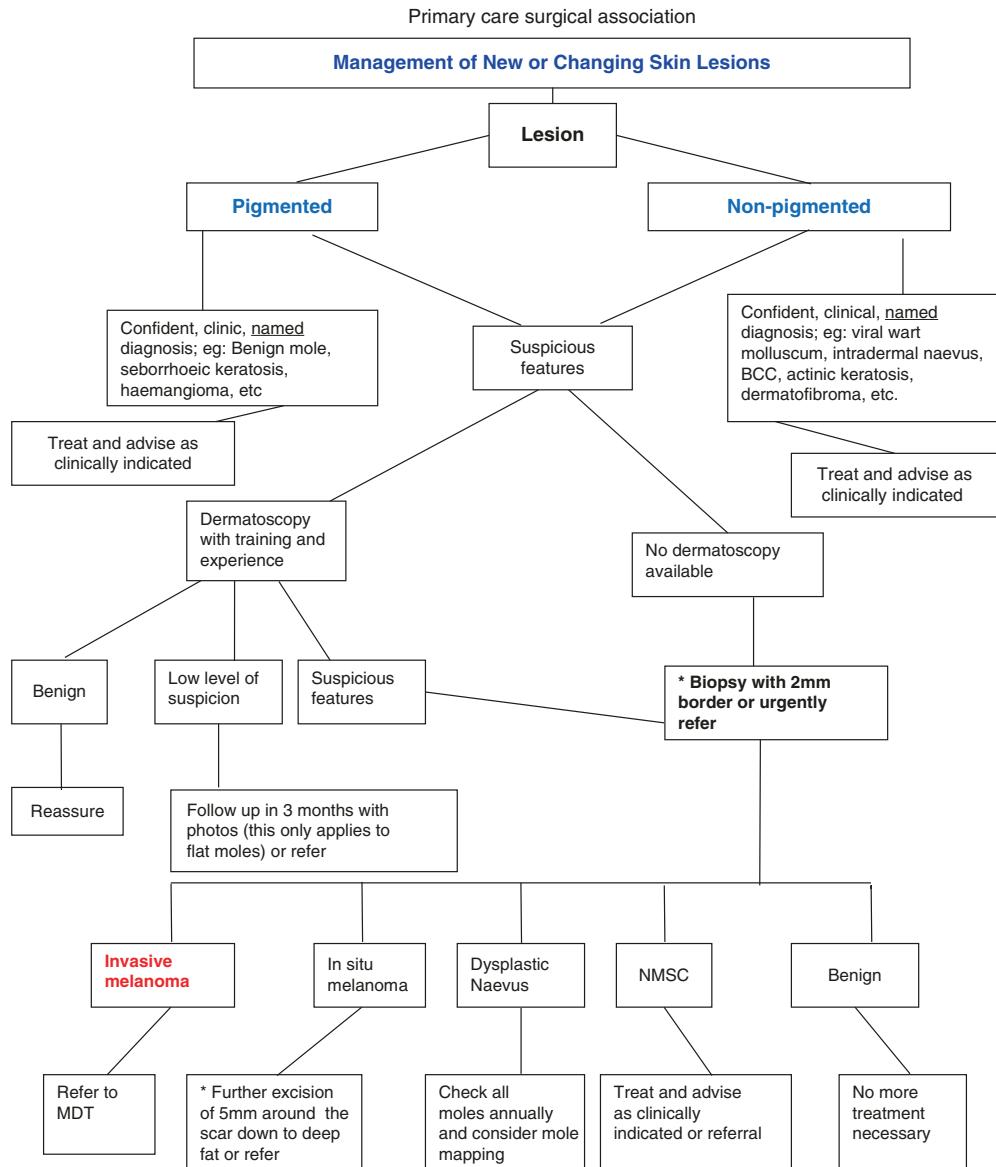
There are only about ten common benign lesions that occur on the skin (Table 45.1). All doctors should be familiar with these and be able to make a confident, clinical named diagnosis. Any skin lesion where a definitive, named, clinical diagnosis cannot be made should be viewed with suspicion and the lesion should be biopsied, excised or referred to a colleague with more experience in lesion recognition (Fig. 45.2).

**Table 45.1** Top ten common benign skin lesions

Benign moles (including congenital naevi, intradermal naevi, blue naevus, etc)
Viral warts
Molluscum contagiosum
Seborrhoeic keratosis
Dermatofibroma
Pyogenic granuloma
Sebaceous cyst
Skin tag
Sebaceous gland hyperplasia
Haemangioma (including cherry angiomas)

**Fig. 45.1** All invasive skin cancers histologically diagnosed in one of the authors practice (DB) over 4 years





\*Only doctors with experience in skin surgery should biopsy or excise

**Fig. 45.2** PCSA algorithm on suspicious skin lesions

Another warning sign for skin cancer is the “NFG sign”. This stands for **n**ew, **f**irm and **g**rowing. If a lesion shows these 3 signs it should be viewed with suspicion.

The term “malignant melanoma” is no longer used. “Melanoma” is sufficient to describe this type of tumour as all melanomas are malignant. 90% of invasive melanomas arise in the skin, 5%

in the eye and the remainder in a variety of sites including the vulva (0.5%), nasal cavity (0.4%) and the anus (0.2%) according to the National Cancer Registry Ireland.

Dermoscopy is extremely useful in making a diagnosis and differential diagnosis of unusual skin lesions but requires extra training. One study indicated that GPs who used dermoscopy after a

**Table 45.2** When to suspect a melanoma when assessing a suspicious skin lesion according to age groups

Babies and children: Almost never
Teenagers: Usually benign
Adults <30 years: Probably benign
Adults >30 years: Suspicious
Adults >40 years: Probably malignant
Adults >50 years: Usually malignant

1 day training course were able to increase their sensitivity for detecting melanoma by 25% [1]. Dermoscopy is *not* optional for any doctor who has a special interest in skin lesion recognition and skin cancer detection.

Artificial intelligence (AI) and deep-learning algorithms using machine learning may well completely change the way we screen for and diagnose skin cancer in the near future. Such algorithms are already showing evidence of outperforming humans on specific tasks such as image recognition in radiology, self-driving cars, in drones and soon in the diagnosis of some cancers [2]. Apps are already available for smartphones which can help individuals track their moles looking for change over time (e.g. “Miiskin” or “SkinVision”).

Most people have developed all their moles by the time they are 40. A new mole after this age is more suspicious, and the older the patient, the more suspicious a new mole is (Table 45.2). In the elderly it can be sometimes difficult to tell the difference between a suspicious mole and a seborrhoeic keratosis but dermoscopy in trained hands can help.

## 45.2 Epidemiology

There is a significant increase in the incidence of skin cancer worldwide. For instance, in Ireland there are approximately 11,000 cases diagnosed every year, with four people dying every week as a result of skin cancer. Incidence rates of non-melanoma skin cancers (NMSC) have risen by 31% in males and 27% in females in the last 10 years (2005–2014) and rates for melanoma have increased by 73% in men and 23% in women during the same period, making it the

fastest rising, preventable cancer in Ireland [3]. By 2040, it is estimated that there will be 33,000 cases of skin cancer diagnosed each year in Ireland.

Melanoma is still a rare cancer, accounting for only 9% of all skin cancers. There were 968 cases and 159 deaths per year from melanoma in Ireland between 2012 and 2014. There are significantly more deaths in Ireland from melanoma than from cervical cancer (156 v 96 in 2014) (Table 45.3). In 2014 melanomas breached the 1000 cases per year mark in Ireland (1041 cases to be exact) which means melanomas have almost trebled in the last 20 years.

On the other hand, NMSCs are the most common cancer in Ireland and they represent almost one-third of all invasive cancers. While deaths from NMSCs are rare, they still account for approximately one-third (30%) of all skin cancer deaths in Ireland between 2012 and 2014. NMSC cause a huge burden of disease, can cause considerable morbidity and are a huge drain on precious resources in health services.

Dividing skin lesions into pigmented and non-pigmented lesions can be helpful, since most melanomas are pigmented and most non-melanoma skin cancers are non-pigmented. However, a small percentage of melanomas can have no pigment (amelanotic) (Fig. 45.3) and can be easily confused with benign lesions such as a pyogenic granuloma or with non-melanoma skin cancers. Conversely many benign skin lesions such as naevi (moles), seborrhoeic keratosis, dermatofibroma and some basal cell carcinomas can be pigmented. Atypical looking seborrhoeic keratosis should be referred to a skilled dermoscopist or biopsied to rule out a melanoma (Fig. 45.4a, b; Table 45.4).

Non-pigmented (amelanotic) melanomas tend to be larger, deeper and have a worse prognosis than pigmented melanomas, probably because of delay in presentation and treatment (Fig. 45.5a, b). One small study in primary care showed that almost a quarter (23%) of all invasive melanomas had little or no pigment [4].

Most skin cancers occur as a result of excessive natural or artificial UV light from the sun or sunbeds. Sudden bursts of excessive UVL on

**Table 45.3** Deaths Occuring (Number) by Sex, Cause of Death and Year in Ireland (2014)<sup>a</sup>

	Female	Male	Both sexes
	2014	2014	2014
C00-D48 neoplasms	4379	4839	9218
C15 malignant neoplasm of oesophagus	114	270	384
C16 malignant neoplasm of stomach	144	225	369
C18 malignant neoplasm of colon	217	282	499
C19 malignant neoplasm of rectosigmoid junction	139	169	308
C20 malignant neoplasm of rectum	68	101	169
C22 malignant neoplasm of liver and intrahepatic bile ducts	139	187	326
C25 malignant neoplasm of pancreas	240	274	514
C26 malignant neoplasm of other and ill-defined digestive organs	54	73	127
C34 malignant neoplasm of bronchus and lung	872	1060	1932
<i>C43 malignant melanoma of skin</i>	77	79	156
C44 other malignant neoplasms of skin	30	52	82
C50 malignant neoplasm of breast	735	5	740
C51 malignant neoplasm of vulva	17	0	17
<i>C53 malignant neoplasm of cervix uteri</i>	96	0	96
C54 malignant neoplasm of corpus uteri	102	0	102
C56 malignant neoplasm of ovary	278	0	278
C64 malignant neoplasm of kidney, except renal pelvis	77	146	223
C67 malignant neoplasm of bladder	93	156	249
C71 malignant neoplasm of brain	123	169	292
C80 malignant neoplasm without specification of site	130	119	249
C85 other and unspecified types of non-Hodgkin's lymphoma	86	109	195
C90 multiple myeloma and malignant plasma cell neoplasms	68	94	162
C92 myeloid leukaemia	48	76	124

<sup>a</sup>From [www.cso.ie](http://www.cso.ie) and [www.ncri.ie](http://www.ncri.ie)

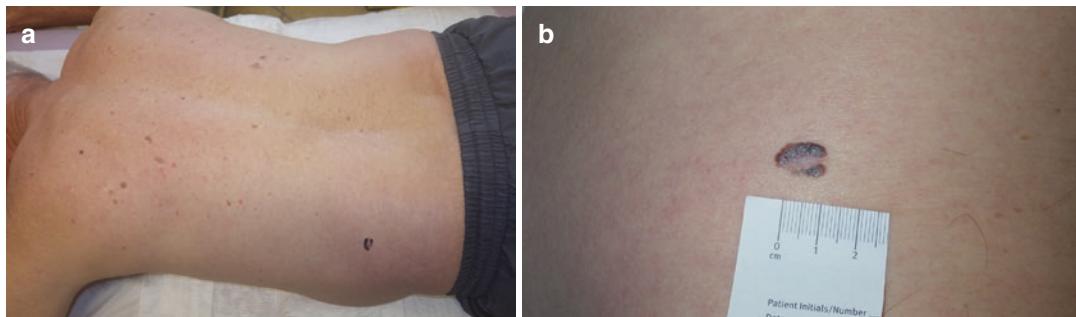


**Fig. 45.3** An amelanotic nodular melanoma 3.9mm deep present for 2 years on the left arm in a 52-year-old female. The patient died 9 months later from metastatic disease

skin that is not accustomed to it, resulting in blistering sunburn is one of the major risk factors for melanoma. Lower levels of UVL exposure over many years is a major risk factor for non-melanoma skin cancer which are more common on exposed sites of the body. Recent studies have suggested that increasing cumulative doses of hydrochlorothiazide, which is commonly used as a diuretic and antihypertensive, may cause an increase in NMSCs possibly as a result of its photosensitizing action [5].

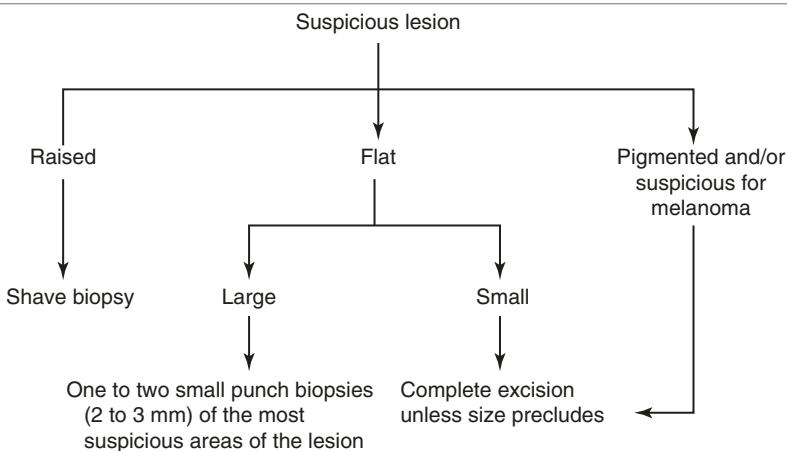
### 45.3 Algorithms for Skin Cancer Detection

Various algorithms have been devised to assist in the clinical examination of suspicious pigmented skin lesions, including the US based “ABCD rule” (Table 45.5; Figs. 45.6 and 45.7), the UK



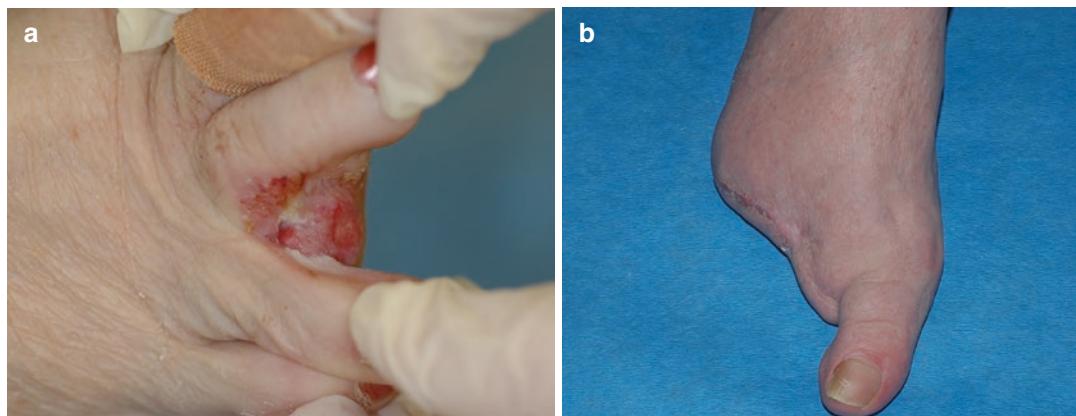
**Fig. 45.4** (a) Melanoma (SSM) on the low back in a man with many of seborrheic keratoses. This was an “ugly duckling”. (b) Close up of the same melanoma

**Table 45.4** Biopsy of Suspected Skin Cancer



*NOTE: This decision tree is designed to assist the physician but cannot replace the physician's judgment and may not be applicable in all situations.*

Ref: October 15, 2004 ◆ Volume 70, Number 8 [www.aafp.org/afp](http://www.aafp.org/afp) American Family Physician  
Diagnosis and Treatment of Basal Cell and Squamous Cell Carcinomas

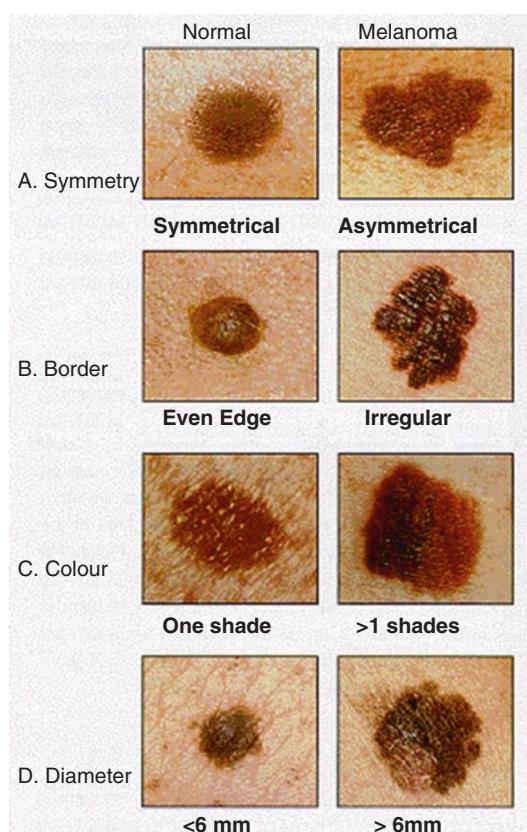


**Fig. 45.5** (a) Amelanotic melanoma 7.1 mm deep. It was present for 12 months. (b) Same patient 6 years later

based “Revised 7-point checklist” (Table 45.6), and the “ugly duckling” sign where a suspicious

**Table 45.5 ABCD rule for assessing the risk of a mole developing into a melanomas**

Asymmetry—the two halves of the area may differ in shape
Border—the edges of the area may be irregular or blurred, and sometimes show notches
Colour—this may be uneven. Different shades of black, brown and pink may be seen
Diameter—most melanomas are at least 6 mm in diameter. Report any change in size, shape or diameter to your doctor



**Fig. 45.6** ABCD rule for diagnosing melanoma

**Fig. 45.7** Evolution of a mole: Change in size



lesion stands out as being different than any other moles on the body. However, these screening tests will fail to detect amelanotic melanomas and most NMSCs. While these screening tools may be useful for doctors and other health professionals, they are not suitable for the general public as the terms are difficult to understand.

The “**Buckley 4 point skin cancer check list**” screens for all skin cancers, both NMSCs and melanomas (pigmented and non-pigmented) (Table 45.7). This incorporates the “ugly duckling sign” which is a very useful aid in diagnosis of melanomas and NMSCs. It encourages the public to look for new or changing moles and skin lesions (sores or growths). This screening tool puts the warning signs in a logical sequence and includes only commonly used, easily understood terms = A ***new*** mole or growth that is ***changing*** (in size, shape or colour), looks ***different*** from any other mole or growth on the skin and is ***sore*** or tender. A useful mnemonic to help remember these warning signs is: “**New Cancers Do Show**”. A mole or growth that shows any one of these four warning signs has to be viewed with suspicion and the more warning signs that are found, the greater the chance that it is a skin cancer.

**Table 45.6** Revised 7-point checklist for assessing risk of melanoma

Suspect melanoma if there are 1 or more major signs in a mole:

1. Change in size (diameter or height getting bigger)
2. Change in shape (notched or ragged border)
3. Change in color (2 or more irregular colours including white)

3 or 4 minor signs without a major sign can also indicate a need to biopsy suspicious moles:

1. Inflammation
2. Crusting or bleeding
3. Sensory change (itch or soreness)
4. Diameter ( $\geq 7$  mm) (but melanomas can be as small as 3 mm)

**Table 45.7** The Buckley 4 point Skin Cancer Check List

These are the warning signs that a lesion (a growth, a sore, a freckle or a mole) that is present for more than 6–12 weeks may be turning cancerous:

A useful mnemonic to help remember these warning signs is:

**“New Cancers Do Show”**

- New—A new growth, sore, freckle or mole especially if you are older than 40 years
- Changing—A growth, sore, freckle or mole that is changing in size, shape or colour over the past few months
- Different—A growth, sore, freckle or mole that looks, feels or behaves different than any other growth, sore or mole on the body (the “ugly duckling”)
- Sore—A growth, sore, freckle or mole that is sore, tender to touch, bleeding or itchy and will not heal after 6–12 weeks

If a growth, sore, freckle or mole shows one or more of these warning signs it should be checked by your general practitioner. The more warning signs, the greater the risk of skin cancer. Early detection saves lives!

Once a suspicious skin lesion is detected it should be examined by a doctor (GP or dermatologist) trained in dermoscopy and a decision to biopsy, refer, observe or reassure can then be made. The desire not to miss a melanoma has to be balanced by the need to reduce unnecessary excisions.

The exact type of skin cancer and definitive management plan can only be decided after a histological diagnosis is made (Fig. 45.8: PCSA management of NMSC). Lesions suspicious of melanoma should be referred urgently to a pigmented lesion clinic. Other suspicious skin lesions may be biopsied in primary care provided the GP has training and experience in lesion recognition and skin surgery. If a skin cancer needs to be referred to a plastic surgeon or dermatological surgeon, sending clinical and dermoscopic photographs with a detailed history and biopsy results should speed up the referral process and the pathway to definitive treatment in most cases.

## 45.4 Skin Biopsy

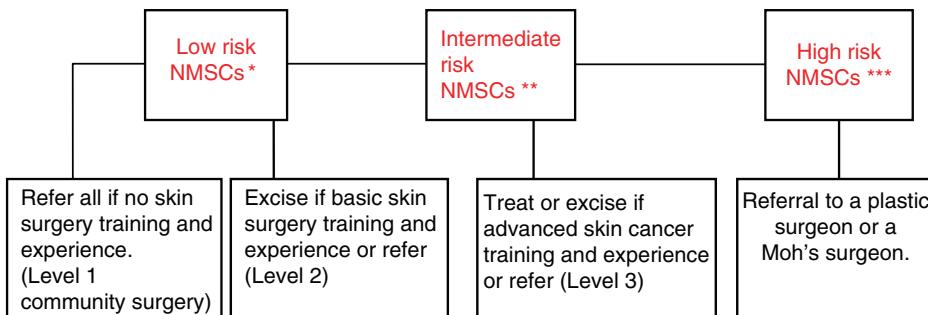
Punch and shave biopsies are a quick and easy way to diagnose many skin lesions (Table 45.4). However, they are not suitable for lesions suspicious of melanoma, whether pigmented or not. In this situation it is best to remove the whole lesion with a 2 mm border of clear skin all around and a generous cuff of subcutaneous fat. The NCCP and NICE guidelines currently recommend that these excisions should only be carried out in a specialist referral centre such as a Pigmented Lesion Clinic or a plastic surgery clinic.

With the exception of lentigo maligna, a punch biopsy is not recommended for lesions suspicious of melanoma, as the biopsy may be taken from an area that may not be representative of the whole lesion and the melanoma may be missed. The Australian and New Zealand melanoma guidelines suggest taking multiple punch biopsies from the most suspicious areas of a lesion as judged by dermoscopy where the whole lesion cannot be easily removed and the chance of a melanoma is low but a definitive diagnosis is required (e.g. possible solar lentigo or a seborrhoeic keratosis, pigmented AK or lentigo maligna).

When taking a skin biopsy, it is essential to include the patient’s name, date of birth, address, skin type, gender, date biopsy was taken and the anatomical location of the lesion. It is recommended to specify the type of biopsy being taken (incision, excision, punch, shave, curettage, etc), the size of the lesion and the clinical margins if a complete excision is undertaken. If orientation sutures are used on the specimen, their location should be included on the histopathology form. It is important to give a brief history and clinical description of the lesion along with a differential diagnosis. If the GP cannot make a reasonable clinical diagnosis and differential diagnosis it may be better to refer the patient with the lesion intact to a colleague with experience in lesion recognition, dermoscopy and skin surgery.

Primary Care Surgical Association.

Guidelines on the management of non-melanoma skin cancers (NMSC)



\* Low risk NMSCs = tumours <10mm on the body between the clavicles and the knees (excluding genital lesions).

\*\* Intermediate risk NMSCs = Facial tumours <10mm and body tumours <30mm provided they show no high risk features

\*\*\* High risk NMSCs:

Recurrent excised

Cosmetic concerns

Large tumours (>10mm on face or >30mm on body)

Large tumours on the vertex of the scalp or below the knees

Tumours involving the nasolabial, pre- or post-auricular folds or genitalia.

Poorly differentiated SCCs, or those with perineural, perivascular or lymphatic involvement.

Deeply invasive NMSC's especially those involving the lips, ears, nose or eyelids. SCCs in scars or sinuses

Tumours with indeterminate margins or tethered to underlying structures (eg: Morpheaic BCCs)

Immunosuppressed patients

Rare aggressive tumours (eg = Acantholytic, spindle or desmoplastic SCCs, Merkel tumours or dermatofibrosarcoma protuberans)

Where there is a risk of metastatic spread (eg: regional lymph nodes enlarged)

**Level 1 Procedures** = sutures lacerations and I+D abscess but no extra training in elective skin surgery.

**Level 2 procedures** = ICGP Minor surgery course or equivalent and training in lesion recognition.

**Level 3 procedures** = Training and accreditation in advanced skin surgery, dermoscopy and performing high volume or complex work including selected NMSCs on the face.

**Fig. 45.8** Guidelines on the management of non-melanoma skin cancers (NMSC)

## 45.5 Actinic Keratosis (AK) (Solar Keratosis)

AKs have been considered pre-cancerous but their malignant potential may have been overestimated in the past. Their presence is considered a risk marker for developing skin cancer as they represent a past history of excessive sun exposure. They are usually found on exposed skin in older patients with fair skin, especially those working or spending a lot of time outdoors, such as farmers, fishermen, sailors, gar-

deners and golfers. There is usually other signs of UV damage such as wrinkles (solar elastosis), freckles and solar lentigos (sunspots).

AKs are frequently multiple and sometimes there is evidence of sun damage over a wide area (especially the bald scalp, forehead or the dorsum of the hands) where the adjacent skin has evidence of solar elastosis which is termed a “field change” (Fig. 45.9). In these areas there may be sub-clinical changes which can lead to AKs and possibly SCCs eventually in the area (“field of cancerization”). The incidence of transfer from an actinic keratosis to a squamous cell carcinoma

is estimated to be anything from 0.5 to 2% per year but the true figure is unknown.

The risk of malignant transformation of an average AK into a SCC in 1 year is less than 1/1000 [6]. Over a 10-year period, a person with an average of 8 AKs has a 6–10% chance of developing an SCC [7]. Studies have shown that up to 60% of the squamous cell carcinomas begin as actinic keratoses and that there is histologic evidence of contiguous actinic keratoses in 97% of the squamous cell carcinoma lesions that arise on sun damaged skin [8]. Actinic keratosis on the lower lip (actinic chelitis) is 2.5 times more likely to progress to SCC than AK of the skin (Fig. 45.10).

Larger, thicker or hyperkeratotic AKs are more likely to progress to an SCC [9]. Conversely, smaller AKs can sometimes resolve spontaneously without treatment.



**Fig. 45.9** Field defect with flat and hyperkeratotic AKs



**Fig. 45.10** Moderately differentiated SCC lower lip

Not all AKs progress from small AKs to hyperkeratotic AKs and onto to SCC. Some small AKs can progress directly into SCC.

AK incidence is extremely high in those on immunosuppressants (e.g. organ transplant patients) and SCC incidence is nearly 100 times higher in these patients than the general population, matched for age and skin type [10].

AKs are usually diagnosed clinically with the characteristic features of a slightly red, dry, roughened macule (flat lesion) varying in size from 2 to 10 mm or more in diameter (Fig. 45.11). They are often more easily felt than seen. There is no bleeding, crusting or ulceration. They are usually multiple. Hyperkeratotic AKs have a thick scale which should be pared down to see what is underneath. If it is an AK there should be no underlying ulceration. Hyperkeratotic AKs may also be termed “Bowenoid AKs” when they begin to develop a thickened, red appearance. Pigmented AKs may mimic other pigmented lesions such as seborrhoeic keratosis, solar lentigo and lentigo maligna. AKs are usually diagnosed clinically or with a dermatoscope. If there is any doubt about the diagnosis of an AK, it is best to biopsy the lesion or refer the patient to a colleague with more experience on lesion recognition for a definitive diagnosis (Tables 45.8 and 45.9).

Most dermatologists feel it is worthwhile treating AKs. There are many methods (Table 45.10 and Fig. 45.12). Some are targeted at the individ-



**Fig. 45.11** AK on the dorsum of the hand

ual lesion such as cryosurgery, curettage and cauterity, shave biopsy, excision, laser ablation, cytotoxic agents such as 5-fluorouracil (“Efudex®”) or an immune response modifier such as imiquimod 5% (“Aldara®”). Other treatments are more suitable for treating the affected area (field treatment) such as the dorsum of the hand, the scalp or the forehead. Field directed treatments target both clinically apparent and sub

clinical lesions simultaneously. Field treatments can be carried out with various topical agents such as 5-fluorouacil (“Efudex®”), Diclofenac gel (“Solaraze®”), imiquimod 3% (“Zyclara®”) or photodynamic therapy (PDT). These treatments can cause considerable and unpredictable irritation and soreness and are best used under the supervision of a doctor with experience in topical field therapies as careful patient counselling is required pre-treatment. Some doctors treat the larger, thicker, more hyperkeratotic AKs first with cryosurgery and then treat the whole field with a field based topical treatment once the cryosurgery wound has healed. Alternatively, treating the field first with topical therapies and later the residual lesions with cryosurgery may also be successful. Unresponsive lesions should be biopsied or referred.

Imiquimod is an immune modulator that regulates Tool-like receptor-7 induced cytokine production and its use has been approved for the treatment of AK (“Zyclara 3%®”) or Bowen’s disease, superficial BCC and genital warts (“Aldara 5%®”) (Table 45.11). “Aldara®” comes in tiny sachets and only one sachet should be used per application. A single-use sachet is sufficient to cover an area of 20 cm<sup>2</sup>.

“Zyclara®” comes in similar size sachets but up to 2 sachets can be used per day. However, it is best to use only one and distribute its content carefully to reduce systemic reactions. It should be applied once daily before bedtime to the area affected by multiple AKs (“the field”) and 2 sachets should be enough to cover the dorsum of both hands or the

**Table 45.8** Differential diagnosis for AKs

Seborrhoeic keratosis
Solar lentigo (sunspot)
Flat warts
Bowen’s disease
SCC
BCC
Melanocytic naevus (mole)
Melanoma

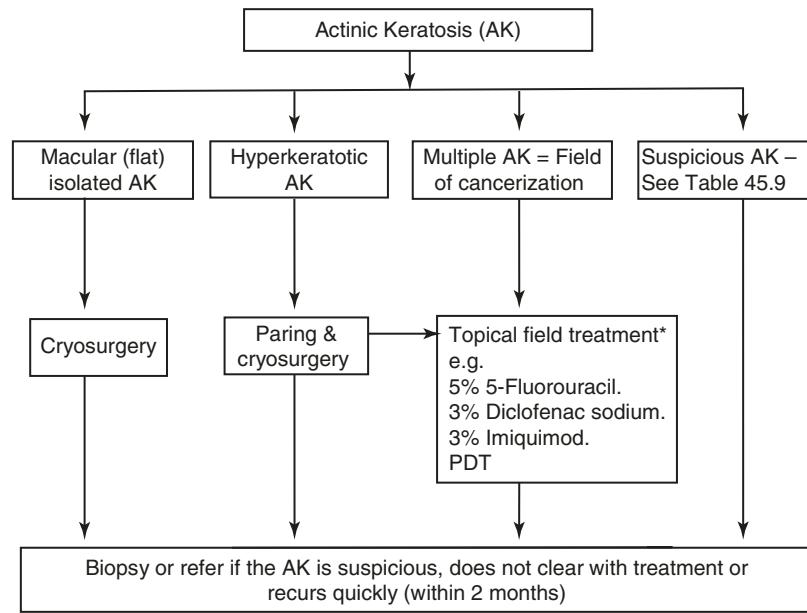
**Table 45.9** Clues that a biopsy may be indicated for an AK

Hyperkeratotic AK
>10 mm
Induration
Inflammation
Ulceration
Bleeding
Rapid expansion
Tender on palpitation
Rapid recurrence after treatment (e.g. 2 months)
Persistence post treatment
Immunosuppressed patients
Organ transplant patients
High risk areas: lip, ear, dorsum of the hand

**Table 45.10** Topical treatment for actinic keratoses (AK), Bowen’s disease and superficial BCCs (sBCC)

Treatment	AK	Bowen’s	sBCC	Duration of treatment	Irritation
Diclofenac gel (“Solaraze®”)	+	-	-	60–90 days	-/+
Imiquimod 3% (“Zyclara®”)	+	-	-	2 weeks on +2 weeks rest—Repeat once	++ (variable)
Imiquimod 5% (“Aldara®”)	+	+	+	4–12 weeks	+++ (variable)
5-Fluorouracil (“Efudex®”)	+	+	+	2 weeks on +2 weeks rest—Repeat twice	++
Photodynamic therapy (PDT)	+	+	+	Once + repeat once after 7 days	+/-

**Fig. 45.12** Treatment algorithm for actinic keratosis



PDT = photodynamic therapy

\*may be combined with paring and cryosurgery  
for isolated hyperkeratotic AK's in the field.

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**Table 45.11** Imiquimod 5% ("Aldara®") for treatment of actinic keratosis, Bowen's disease, superficial BCCs (sBCC), lentigo maligna and genital warts

Diagnosis	Treatment protocol	Repeat treatment
Actinic keratosis	3 times a week for 4 weeks	May need second cycle
Bowen's and sBCC	5 times a week for 6 weeks	May need second cycle
Lentigo maligna <sup>a</sup>	7 times a week for 12 weeks	
Genital warts	3 times a week up to 16 weeks	Until clearance of visible warts

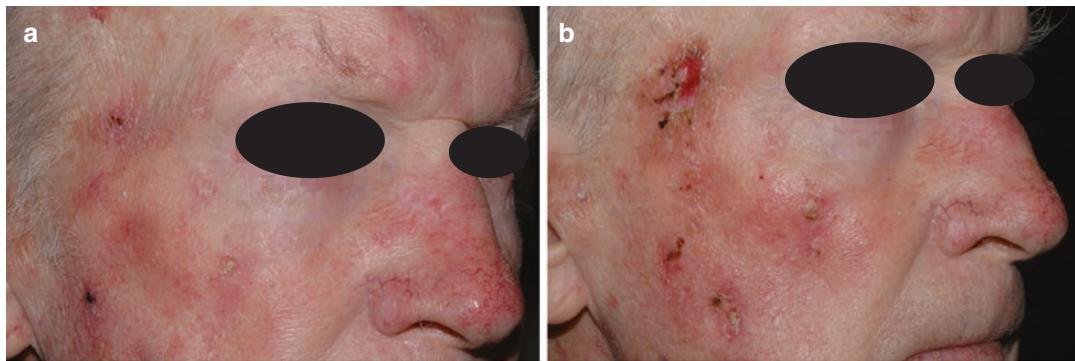
<sup>a</sup>not licensed for this use

forehead or the balding scalp. It is applied daily for 2 weeks followed by a 2 week rest period before applying it again for a further 2 weeks.

Diclofenac ("Solaraze®") is a nonsteroidal, anti-inflammatory drug that inhibits cyclo-

oxygenase-2 resulting in reduced prostaglandin synthesis [11]. Raised prostaglandins have been linked with sun damage and AKs [12]. "Solaraze cream®" can be useful for small superficial AKs and, unlike 5-fluorouracil or "Zyclara®", it causes very little irritation. However, it has to be applied twice a day for 3 months, may not clear larger, more hyperkeratotic AKs and relapse is not uncommon. Occasionally patients can be allergic to diclofenac and can develop local severe reaction that may require stopping the medication.

Ingenol mebutate ("Picato®") is an extract of a common plant, petty spurge or milk weed (*Euphorbia peplus*) which is grown in Australia. It is thought to work by causing rapid lesion necrosis and specific neutrophil-mediated, antibody-dependent cellular cytotoxicity. Only one tube should be used per day and it should be washed off after 6 hours. Great care is required to avoid get-



**Fig. 45.13** (a) Actinic keratosis before treatment with ingenol mebutate. (b) Same patients 6 days post treatment. Similar reactions might occur with 5-fluorouracil and imiquimod 3%

ting it into the eyes where it can be very irritating. The content of one tube covers a treatment area of 25 cm<sup>2</sup> (e.g. 5 × 5 cm) which is equivalent to the area on the dorsum of one adult's hand. It comes in 2 strengths (0.015% which should be applied to the face or scalp daily for 3 days or the 0.05% which can be used on the body daily for 2 days). Reactions tend to appear 3–4 days after the last application (Fig. 45.13a, b). In 2020 the European Medicine Agency withdrew "Picato" (ingenol mebutate), concluding that this medicine may increase the risk of skin cancer and that its risks outweigh its benefits.

"Zyclara®" and "Picato®" can cause considerable, unpredictable irritation in some patients when used for field directed treatments of AKs and solar elastosis. The more severe the reaction the better the result but some patients may not be able to tolerate these treatments. Significant reactions that the patient hates, the physician loves to see as it means the treatment is working. It may be kinder to the patient to delay treatment until a quite time in the patient's life when they have no major work or social engagements planned within the following 2 weeks post treatment.

Photodynamic therapy (PDT) involves applying aminolevulinic acid (ALA) or methyl aminolevulinic (MAL) cream under occlusion for

3 hours to the lesion or area being treated. This is a pro-drug that is intracellularly metabolised to protoporphyrin IX, a photosensitising molecule. When this is activated by exposure to light from an LED lamp (for 9–10 minutes usually under local anaesthetic) or daylight, free radicals and reactive oxygen species are generated which are cytotoxic and selectively kill the pre-cancer (AK's or Bowens disease) or cancer cells such as superficial BCCs [13]. While the treatment itself may be painful in some patients, there is little post-operative irritation and usually excellent cosmetic results. Aminolevulinic acid (ALA and MAL) cream is very expensive and the success rate when treating Bowen's disease and superficial BCCs may not be as good as other methods used to treat these lesions [14].

5-fluorouracil ("Efudex®") is a cytotoxic agent that can selectively destroy certain cancer or pre-cancerous cells. It is used for the topical treatment of superficial pre-malignant and malignant skin lesions including AKs, Bowen's disease and superficial basal-cell carcinoma. A recent study published in the New England Journal of Medicine showed 74.7% of patients treated with 5-fluorouracil (twice a day × 1 month) cleared their AKs 12 months from the end of therapy versus 53.9% who received imiquimod, 37.7% given

MAL-PDT and 28.9% treated with ingenol mebutate [15].

All patients with AKs, solar elastosis (skin thinning from excessive UV exposure) or skin cancer should be advised to protect their skin from ultraviolet light with the careful use of clothing, a broad-brimmed hat and high factor sunblocks such as SPF 30 or greater. This degree of photo-protection will put the patient at risk of vitamin D deficiency so these patients should take a vitamin D supplement daily (800–2000 IU a day) especially in the winter provided there are no contraindications (e.g. renal stones, renal failure, hypercalcaemia, etc).

## 45.6 Bowen's Disease (SCC In Situ)

Bowen's disease (also known as intraepidermal SCC or SCC in-situ) is quite common on the face and lower legs, especially in women. They are generally macular (flat), red and slightly scaly but usually there is no ulceration or bleeding (Fig. 45.14). They may resemble an actinic keratosis or a superficial BCC or even an SCC. Dermoscopy may help in making the diagnosis but a biopsy should always be taken before definitive treatment. For larger lesions (>10 mm) two or more biopsies may be necessary and the leading edge is more likely to con-

firm the diagnosis rather than taking biopsies from the centre of the lesion. Although considered low risk, 3–5% of Bowen's disease will progress to SCC so it is important to treat them in the majority of patients. Up to 10% of Bowen's disease on the genitalia will progress to SCC. However, if they are found in a very frail elderly patient in a difficult to treat site (e.g. lower legs), it might be more appropriate to keep them under observation rather than treat them. If they show signs of progressing to an SCC (becoming ulcerated or nodular) curative treatment may have to be considered.

Because they are superficial, Bowen's disease may respond to topical treatment such as 5-fluorouracil ("Efudex®") (Fig. 45.15) or 5% imiquimod (Aldara®) (Tables 45.10 and 45.11). However, this is a slow, and potentially painful treatment that requires a coordinated, well-motivated patient, as the whole treatment can take weeks or months of nightly treatment. This may be difficult or impossible in an elderly patient, living alone with a physical or mental disability. This treatment can also be distressing in a younger patient who is working as they may have to put up with an unsightly, scabbing lesion for 6–12 weeks during the treatment phase which can be distressing, particularly if the lesion is on the face, hands or lower legs.



**Fig. 45.14** Bowen's disease (SCC in-situ) on the cheek



**Fig. 45.15** Bowen's disease before treatment with 5-fluorouracil ("Efudex®")

Small lesions in easily accessible sites can be excised with a 4 mm border but this may prove difficult especially in the lower leg or the face and may represent over treatment of a superficial, pre-cancerous lesion. Cryosurgery is very successful for treating Bowen's disease when carried out under experienced hands. It usually requires a 30 second freeze and one freeze-thaw cycle. This can be painful and is best given under local anaesthetic. The complete lesion and 4 mm of clear, unaffected surrounding skin should be frozen as fast as possible and once the ice-ball reaches the treatment borders, it should be maintained by intermittent bursts of liquid nitrogen spray for 30 seconds. This causes considerable swelling, oedema and crusting which can take 2–4 weeks to heal (Fig. 45.16a, b). Cryosurgery is not suitable for larger lesions (>10–20 mm) and is not recommended in the lower leg as healing time can be extremely slow in this area (see Chap. 58).

In experienced hands, curettage followed by cautery (usually mono-polar diathermy) is a very effective treatment for treating Bowen's disease if the procedure is undertaken three times (the cauterised area is curetted and again cauterised twice more in a single session). The material from the first curettage is sent for histology, the subsequent material curetted after cautery is of

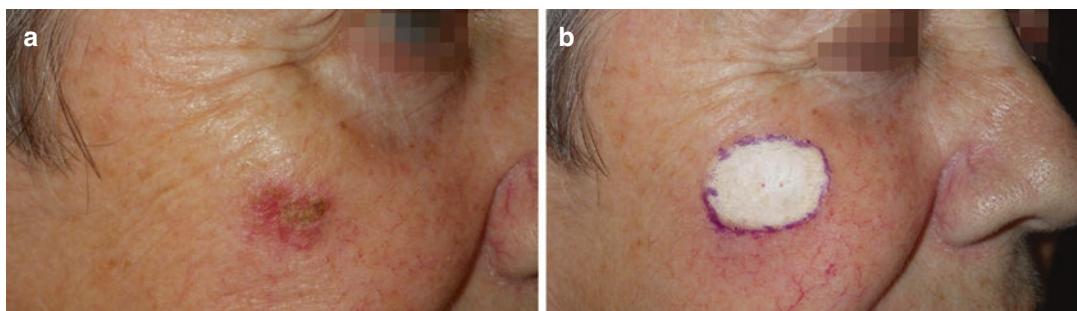
no diagnostic use; it just acts to provide a deeper clearance of the superficial lesion.

## 45.7 Squamous Cell Carcinomas (SCCs)

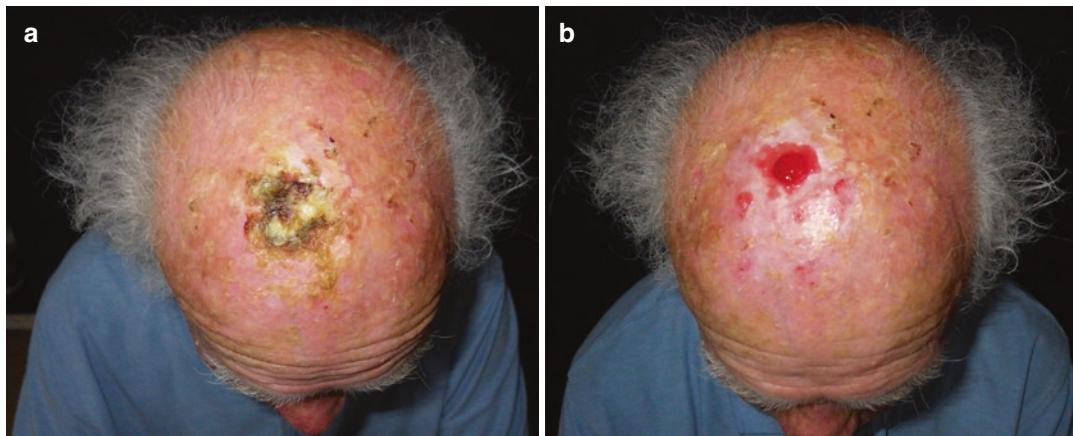
Some SCCs arise de novo from apparently normal skin. Others develop from a pre-existing actinic keratosis (Fig. 45.17a, b), from an area of Bowen's disease or from an area of chronic inflammation or ulceration (e.g. a leg ulcer that fails to heal despite appropriate management). SCCs are usually red, scaly and may have some superficial ulceration (Fig. 45.18). They can be flat or nodular (Fig. 45.19). They are usually raised, growing and tender. Diagnosis is confirmed by biopsy such as a punch biopsy or an incision or excision biopsy.

SCCs may occur on all areas of the body including the mucous membranes and genitals, but are most common in areas frequently exposed to the sun, such as the rim of the ear, lower lip, face, balding scalp, neck, dorsum of the hands, or on the arms and legs.

The majority of SCCs are low risk but approximately 2–5% of SCCs metastasize. The aggressiveness of an SCC is described by its degree of differentiation. A mildly differentiated SCC is considered low risk of metastases, a moderately



**Fig. 45.16** (a) Bowen's disease before treatment with cryosurgery; (b) same patient immediately after treatment



**Fig. 45.17** (a) Patient with erosive pustular dermatosis of the scalp with underlying Bowen's disease and SCC; (b) same patient after scab removal



**Fig. 45.18** SCC and actinic keratoses on the scalp of a fisherman

differentiated SCC is considered medium risk and a poorly differentiated SCC is considered high risk and aggressive. High risk SCC tumours may also have perineural or vascular invasion which indicates an even greater risk of metastasis. Larger deeper tumours, especially those tethered to underlying structures are also considered high risk. SCCs involving the lip (mostly the lower lip) and ear tend to metastasise early. All high risk SCCs should be referred to a plastic surgeon or dermatological surgeon without delay (Table 45.12). Draining lymph nodes should be palpitated and their presence or absence should be recorded at the time of presentation or referral (Fig. 45.20).



**Fig. 45.19** Two SCCs (moderately differentiated) on the right cheek

Treatment is usually with excision including a 4 mm border of clear skin all around for low risk SCCs. High risk SCCs may need much wider margins and adjunct radiotherapy.

**Table 45.12** High risk NMSCs

Recurrent tumours
NMSC in areas where there are cosmetic concerns
Large tumours (>10 mm on face or > 30 mm on body)
Large tumours on the vertex of the scalp or below the knees
Tumours involving the naso labial folds or the pre or postauricular folds
Poorly differentiated SCCs
Deeply invasive NMSCs (>4 mm deep) especially those involving the lips, ears, nose or eyelids
Tumours with indeterminate margins or tethered to underlying structures (e.g. Morphoeic BCCs)
Immunosuppressed patients
Rare aggressive tumours (e.g. Acantholytic, spindle or desmoplastic SCCs, Merkel tumours or dermatofibrosarcoma protuberans)
Where there is a risk of metastatic spread (e.g. Regional lymph nodes enlarged)
≤1 mm histological clearance

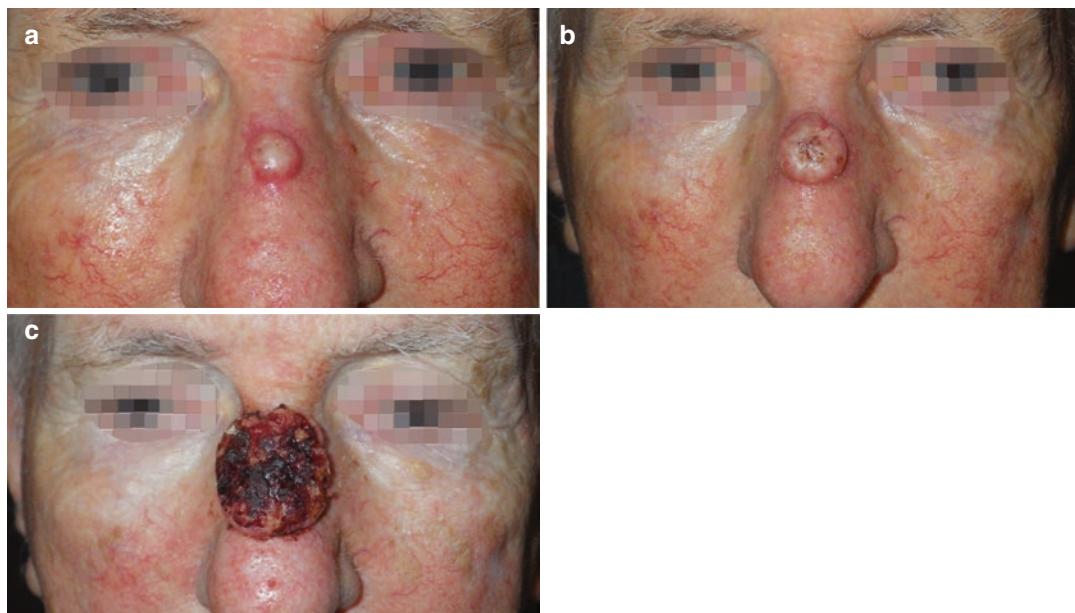
**Fig. 45.20** Metastatic disease from an SCC

Small well differentiated SCCs may be treated with aggressive cryosurgery but this should only be carried out by doctors with extensive experience in this procedure. It involves freezing the debulked tumour and at least 4 mm of surrounding unininvolved skin under local anaesthetic with liquid nitrogen spray under temperature control for 30 seconds and 2 freeze thaw cycles. This will cause considerable swelling and blistering but the lesion usually heals with an acceptable scar within 4–8 weeks.

SCCs may lurk under a cutaneous horn, especially in the elderly so suspicion should be high if there is any erythema or thickening of the base of a cutaneous horn. It is best to remove them by deep shave or excision biopsy and always send the specimen for histological diagnosis (Fig. 45.21).

**Keratoacanthoma (KA)** usually appears as a nodular lesion with a central keratin plug in the light exposed areas in the elderly that can grow rapidly within 4–6 weeks (Fig. 45.22). The natural history of a KA is that it may regress spontaneously over 3 or 6 months. However, it can sometimes be difficult if not impossible to tell the difference between a KA and a well differentiated SCC both clinically and histopathologically (Fig. 45.23a–c). When sending a possible KA for histology it is best to send the whole lesion and clearly indicate on the pathology form that the lesion grew rapidly and that you are considering a KA as well as an SCC in the differential. KAs

**Fig. 45.21** Cutaneous horn on left temple. Histology showed a hyperkeratotic actinic keratosis**Fig. 45.22** Keratoacanthoma (SCC/KA subtype) which grew to this size in 6 weeks



**Fig. 45.23** (a) Keratoacanthoma which grew to this size over 3 weeks. (b) Same patient 2 weeks later. (c) Same patient 9 weeks after the first photo. It was fully excised with a skin graft

are best considered a variant of SCC and should be treated by complete surgical excision including 4 mm of clear skin all round. Smaller KAs (<10 mm) may be suitable for removal by shave excision and subsequent cryosurgery to the base by experienced cryosurgeons as outlined above for SCCs.

Unlike BCCs and Bowen's disease, SCCs have the potential to spread and metastasize to local lymph nodes or beyond and can be life threatening. Patients who are immunosuppressed (e.g. renal transplant patients) are more likely to develop multiple, aggressive SCCs and in fact skin cancer is the leading cause of death in solid organ transplant patients. These patients need careful follow-up to ensure prevention, early detection and timely treatment of any new lesions.

## 45.8 Basal Cell Carcinomas (BCCs)

Unlike SCCs, most BCCs have little or no potential to spread beyond the skin and are therefore not usually life threatening. Although slow growing, they can spread insidiously under the skin



**Fig. 45.24** BCC which required amputation of the nose

and can be locally destructive, especially morpheaic, sclerosing or the micronodular sub-types with perineural or vascular involvement. In certain high risk areas growth can occur into vital structures (e.g. nerves or arteries) and may be life threatening (Fig. 45.24). BCCs grow slowly with an estimated growth of 0.5 mm over 10 weeks in

facial BCC's and 0.7 mm over 8.7 weeks for head and neck tumours [16, 17]. Be wary of treating younger patients (under 30), those with genetic susceptibility such as Gorlin syndrome (naevoid basal cell carcinoma syndrome), immune-suppressed patients, tumours over joints, large tumours, morphoeic tumours and any recurrent BCCs (Table 45.12). These are probably best referred to a specialist.

The classical text book description of a nodular lesion with raised, pearly white edges and an ulcerating centre ("rodent ulcer" or a nodular or ulcerative BCC) is but only one of many ways BCCs present (Fig. 45.25). There are a number of subtypes of BCCs (Table 45.13 and Fig. 45.26) all with different clinical and/or histological features. Superficial BCCs (sBCC) are flat, may be scaly and are not usually ulcerated (Fig. 45.27a, b). They can be easily confused with an AK, Bowen's disease, a well differentiated early SCC, or an amelanotic melanoma (Figs. 45.28 and 45.29). A seborrhoeic keratosis or a flat wart may also resemble a sBCC. The various subtypes of BCCs can be pigmented and mimic a mole or a

melanoma. Morphoeic (sclerosing), infiltrated and micronodular BCCs can spread insidiously under the skin and their borders are ill defined (Fig. 45.30). Basosquamous BCCs have features of both BCCs and SCCs and should be treated as SCCs.

Dermoscopy usually shows typical features of a BCC which can help not only distinguish them from other benign and malignant lesions but also help delineate the borders. Where the diagnosis is clinically certain and excision is considered the best treatment option, a punch biopsy is not required; indeed for smaller lesions it may make subsequent management difficult as the lesion may disappear from sight post biopsy. Photography pre-biopsy can be helpful in recording the location.

Otherwise, diagnosis is usually by punch or shave biopsy and treatment will depend on the subtype, depth of tumour, size in diameter, location and the age and general health of the patient (Table 45.14). Treatment will also depend on the experience of the treating doctor.

Defining high and low risk BCCs is a contentious issue and has historically been based on location (especially the so called H region of the face: around the eyes, nose, lips and ears) and subtype (morphoeic, micronodular). However, a more pragmatic approach is to state that any BCC that cannot be marked with an adequate border and completely excised both laterally and deeply is to be considered high risk for that surgeon and should be referred.

Low risk BCCs are probably best excised with 4 mm border all round. Higher risk or recurrent BCCs should be referred to a team where a range of treatment options can be considered including Mohs microscopically controlled surgery and radiotherapy. The most important aspect for the management of BCCs is to get the initial treatment correct and curative first time round; recurrences can be very difficult to manage. The edge of the lesion should be marked and include a 4 mm safe margin of clear skin all around the tumour. Refer any lesion that either cannot be safely excised with adequate margins or where excision is likely to cause complications (e.g. interfere with adjacent structures).



**Fig. 45.25** BCC with raised, pearly, nodular border and ulcerating center

**Table 45.13** Subtypes of Basal cell carcinomas

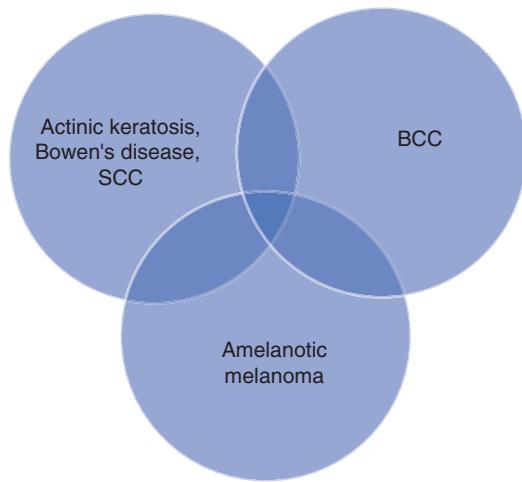
Nodular BCC (nBCC)
Ulcerate (uBCC = rodent ulcer)
Superficial spreading (sBCC)
Pigmented
Morphoeaform
Sclerosing
Basosquamous (metatypical)
Mixed infiltrative
Micronodular

**Fig. 45.26** Pathology of skin—common disorders (adapted from: Shashidhar Venkatesh Murthy, A/Prof & Head of Pathology at James Cook University. <http://www.slideshare.net/vmshashi/pathology-of-skin-common-disorders>)

Basal Cell Carcinoma:		
Type	Features	Picture
Nodular BCC	<ul style="list-style-type: none"> <li>•Most common</li> <li>•Small, shiny, pink lump</li> <li>•Prominent BV network</li> </ul>	
Superficial BCC	<ul style="list-style-type: none"> <li>•Often multiple</li> <li>•Pink or red scaly irregular plaques</li> </ul>	
Morpheic BCC	<ul style="list-style-type: none"> <li>•sclerosing BCC</li> <li>•scar-like</li> <li>•perineural spread</li> </ul>	
Pigmented BCC	<ul style="list-style-type: none"> <li>•Brown, blue or grey</li> <li>•Like melanoma</li> <li>•Nodular or superficial</li> </ul>	
Basosquamous BCC	<ul style="list-style-type: none"> <li>•Mixed BCC &amp; SCC</li> <li>•more aggressive</li> </ul>	



**Fig. 45.27** (a) sBCC on the bridge of the nose. (b) Same patient highlighting the borders of the sBCC by stretching the skin



**Fig. 45.28** Differential diagnosis of a non-pigmented isolated scaly patch



**Fig. 45.29** Superficial BCC on the cheek

Small (<10 mm) well defined BCCs in areas that are difficult to excise such as the inner canthus (Fig. 45.31), nose and ears may be treated successfully by cryosurgery. Cryosurgery should only be carried out by doctors with extra training and experience in treating selected cases of non melanoma skin cancers. Temperature control should be available to ensure temperature of less than -50 degrees



**Fig. 45.30** Sclerosing BCC

**Table 45.14** Treatment options for BCC's

Surgical excision
Plastic surgery (flaps + grafts)
Cryosurgery
Imiquimod 5% ("Aldara")
Photodynamic therapy
Curettage and cauterity
Radiosurgery
Mohs micrographic surgery

centigrade are reached under the tumour during treatment in selected cases. Biopsies (punch or shave) should always be taken before cryosurgery and can help shrink the tumour before definitive treatment which should be planned for about 2 weeks later when the tumour has shrunken and histology is back.

Superficial BCCs do not usually require excision. They can be treated with cryosurgery or with topical therapies such as imiquimod 5% ("Aldara®") (Fig. 45.32a-c) and PDT (Tables 45.10 and 45.11). Curettage and cauterity is a popular technique in the USA for treating small localised BCCs in experienced hands. Radiotherapy was popular in the past

and is now only used for large BCCs especially in the elderly, in some difficult to treat areas and post-surgery for high risk BCCs (whenever there is perineural invasion) and for some SCCs. Radiotherapy may leave unsightly scars which develop over time and surgical procedures in previously irradiated tissue is very difficult if the tumour is not cleared or recurs post-radiotherapy.

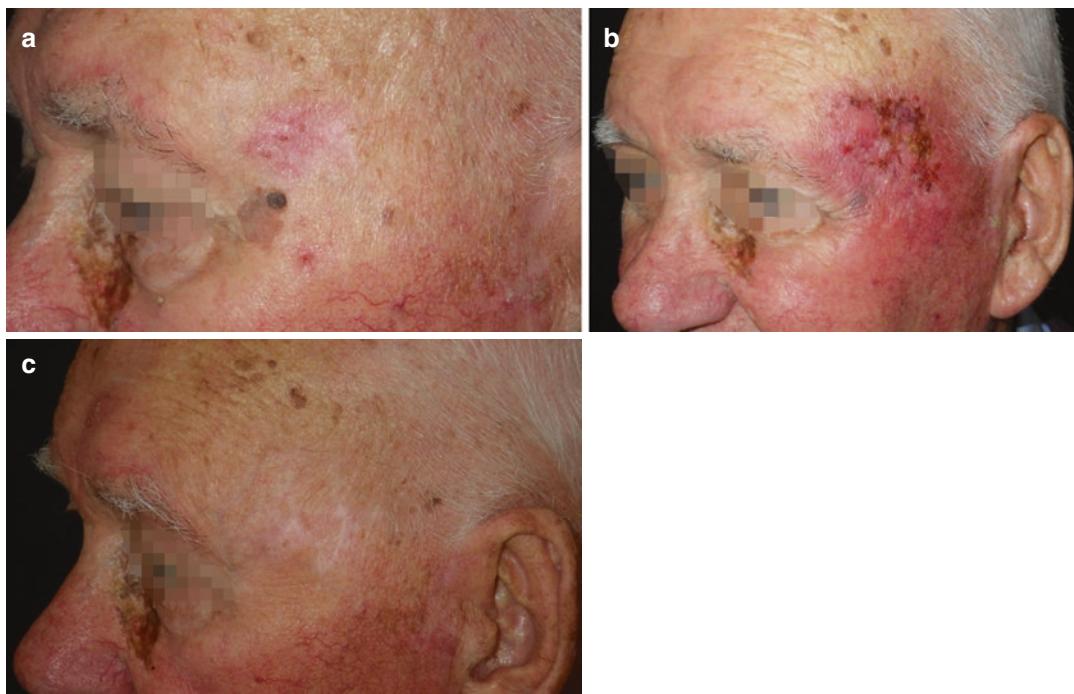


**Fig. 45.31** nBCC before shave biopsy, cauterity and cryosurgery

5-Fluorouracil (5-FU), a chemotherapy drug approved to treat internal cancers, is FDA-approved for superficial BCCs, with similar cure rates to imiquimod. The cream is gently rubbed into the tumour twice a day for 4 weeks followed by a rest period of 4 weeks. This cycle of treatment may have to be repeated up to 2 more times. Side effects are variable but redness, irritation, and inflammation usually occur.

Most methods for treating BCCs have a greater than 90% cure rate. Mohs micrographic surgery is the gold standard when treating BCCs and has a cure rate of 97–99% in experienced hands (Table 45.15). However, because this treatment method is time consuming, labour intensive and expensive, it should be carried out only on high risk tumours (Table 45.12).

Mohs micrographic surgery involves excising the tumour, marking the edges and immediately checking the borders using frozen sections while the patient is still in the operating theatre to ensure the entire tumour has been removed. If any residual tumours are found in a particular



**Fig. 45.32** (a) sBCC (25 mm) left temple before treatment. (b) Same patient after 6 weeks treatment with imiquimod 5%. (c) Same patient 3 years later

**Table 45.15** Treatment options of BCCs versus 5-year cure rates<sup>a</sup>

Treatment option	5-year cure rate (%)
Cryosurgery	94–99
Curettage and cauterity	92–98
Excision	90–97
Flaps and grafts	91–98
Imiquimod 5%	76–89
Photodynamic therapy	70–94
Radiotherapy	90–91
Mohs surgery	97–99

<sup>a</sup>In selected cases

side of the lesion, more skin is removed from that area and is again immediately assessed by frozen section to ensure complete clearance. Once all the edges are clear, the resulting defect is closed by a variety of methods depending on the size and location of the wound (primary closure, flap, graft, partial closure or healing by secondary intention). “Slow Mohs” is a similar technique but the second or subsequent excisions are carried out a few hours later or the following day after the initial excision, before finally closing the defect.

For extraordinarily rare cases of metastatic BCC or locally advanced BCC that become dangerous and even life-threatening, targeted hedgehog inhibitors such as oral **vismodegib (Erivedge™)** or **sonidegib (Odomzo®)** are now available but because of their prohibitive cost and side effect profile they are reserved for selected severe cases (destructive and/or metastatic) not suitable for surgery or radiotherapy.

## 45.9 Melanoma

Melanoma is the most dangerous skin cancer. Some types of melanoma such as the nodular subtype can grow rapidly and spread quickly beyond the skin within a few weeks or a few months and can be fatal (Fig. 45.33). Others can remain in-situ for many years before becoming invasive (e.g. lentigo maligna) (Fig. 45.34). Studies have shown that the median increase in thickness of superficial spreading melanomas is



**Fig. 45.33** Melanoma on the left calf which was 7.5 mm deep on histology



**Fig. 45.34** Melanoma in situ, lentigo maligna subtype

0.12 mm per month, for lentigo maligna is 0.13 mm per month, and for nodular melanoma is 0.49 mm per month. It is also reported that one-third of melanomas grew 0.5 mm per month or more, a finding that highlights the need for definitive diagnosis and treatment at the very earliest opportunity [18]. Unlike other skin cancers, melanomas can occur at any age but are more

common as one gets older. Melanomas in children have been reported but are extremely rare before the age of 12 years.

Most people have developed all their moles by the age of 40. A new mole after this age should be viewed as suspicious, and the older the patient the more suspicious the new mole becomes. People with more than 50 moles which are 2 mm or greater in diameter on their whole body have an increased risk of developing a melanoma. Less than half of all melanomas develop from existing moles. The majority (60–80%) arise from brand new moles. Other risk factors for melanoma include having red or fair hair, fair skin, light-coloured eyes, having suffered blistering sunburn in the past or having used sun beds in the past.

Many genes are implicated in the development of melanoma and melanoma is more likely in those with a personal or family history of pancreatic cancer or astrocytoma. Patients with multiple dysplastic naevi or a previous personal or family history of melanoma are also considered high risk. Patients with a family history of dysplastic naevus syndrome are more likely to develop melanoma.

Some people are considered high risk patients because of their outdoor occupation or hobbies or as a result of foreign travel to sunny places. Acute, intense, intermittent blistering sunburns, especially on areas of the body that are not usually exposed to the sun, is the greatest risk factor for the development of sun exposure-induced melanoma. This sun-associated risk factor is different from that for non-melanoma skin cancers (BCC, SCC, Bowens), which are associated with prolonged, long-term sun exposure. Lentigo maligna melanoma is an exception to this rule, because it frequently appears on the head and neck of older individuals who have a history of long-term sun exposure.

Any new moles or change in an existing mole should be examined carefully for signs of melanoma. In patients with multiple moles, it is important to look for the one that is changing while all the others remain unchanged. The “ugly duckling sign” is also useful. This is where one mole looks completely different from all the others in the area or on the body. If the “ugly duckling mole” is showing any unusual features such

as change in size, shape or colour, itch, oozing or crusting, it should be referred for a second opinion and probable excision biopsy. When examining a patient’s moles with a dermatoscope, most patient’s moles will have one pattern. The ugly duckling will be the mole that is dermoscopically different from all the others.

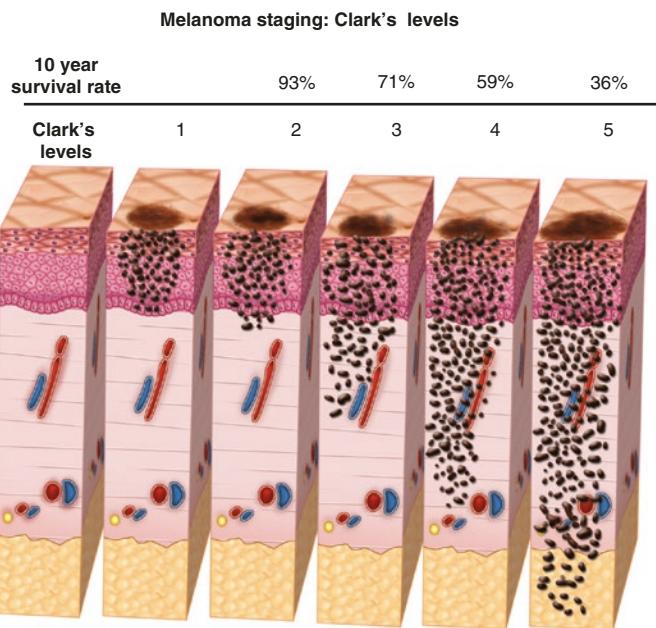
Most but not all melanomas are pigmented, so any pigmented lesion that is changing in size, shape or colour needs to be carefully assessed. There are a number of benign and pre-malignant mimickers that can present like a spreading pigmented macule such as a seborrhoeic keratosis, pigmented AK, solar lentigo (sunspot), pigmented BCC and lentigo maligna. If there is any suspicion of a melanoma, the lesion should be removed with a 2 mm border of clear skin for histological diagnosis. This is usually carried out in a specialist referral centre such as the Pigmented Lesion Clinic or a plastic surgery clinic. If a melanoma is diagnosed, prognosis and definitive treatment will be dictated by the depth of the tumour (Breslow thickness) (Table 45.16) and other high risk features such as ulceration, Clark level 4 or 5, high mitotic activity, lymphovascular invasion and whether or not there is local or distant metastasis. Breslow thickness is expressed in millimeters and measures depth from the granular layer of the epidermis to the deepest part of the tumour. Breslow thickness is strongly correlated with melanoma survival (Table 45.16; Figs. 45.35 and 45.36). If the Breslow thickness

**Table 45.16** Five year survival rate for primary melanoma

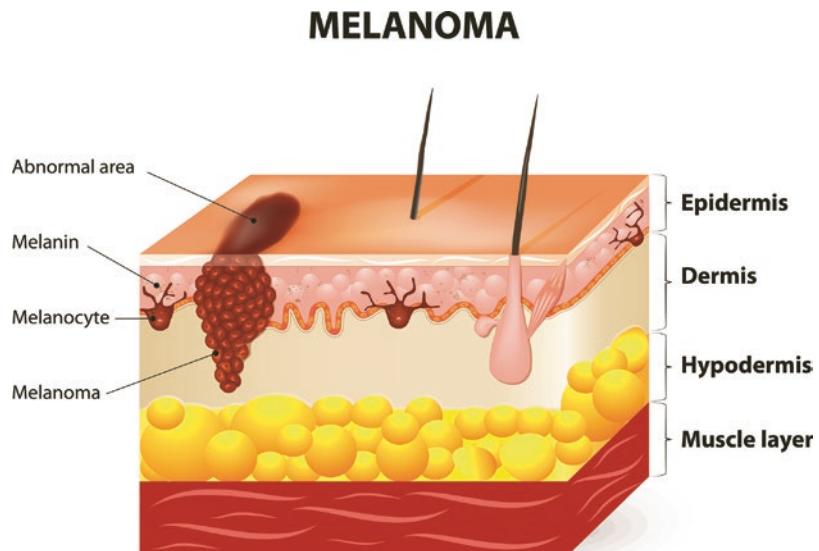
Breslow thickness of melanoma	Five year survival <sup>a</sup>
Dysplastic naevi	100%
Melanoma in situ and lentigo maligna	95%–100%
<1 mm	95%–100%
1.01–2 mm	80%–90%
2.01–4 mm	60%–75%
>4 mm	50%

<sup>a</sup>Survival rates are worse if there is ulceration, high mitotic rate, local lymph node involvement or distant metastases  
Ref: Marsden JR et al. Revised UK guidelines for the management of cutaneous melanoma 2010. Br. J. Dermatol. 2010, 163, 238–256

**Fig. 45.35** Breslow depth and Clark's levels for melanoma



**Fig. 45.36** Invasive melanoma penetrating deep into the dermis.  
(Copyright designua@123RF.com)



is  $>1$  mm the patient is often sent for a sentinel lymph node biopsy (SLNB) which is a biopsy of the first node where the cancer is likely to spread from the primary tumour. If the sentinel node is positive for tumour cells it has important implications for staging the tumour, judging the prognosis and planning further treatment which should

be carried out in a specialist melanoma centre. Naevi and lesions suspicious of melanoma should be excised with a 2 mm margin and no wider in order to allow sentinel node biopsy to take place if deemed advisable. Sentinel node biopsy involves injecting dye into the excision site and then surgically removing the draining (sentinel)

**Table 45.17** Current recommendations for surgical margins of excision of a histologically confirmed melanoma are as follows

In situ lesions:	0.5 cm margin
Lesions ≤ 2 mm in thickness:	1 cm margin
Lesions >2 mm in thickness:	2 cm margin

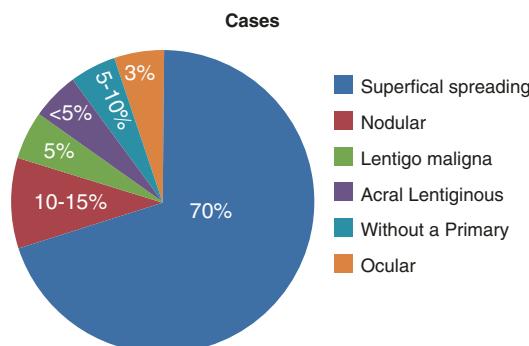
Ref: Michielin O et al. Cutaneous melanoma: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. Ann Oncol (2019)

lymph node. If the lymph node contains melanoma the patient should be considered for systemic treatment.

The treatment of melanoma is surgical. However, patients with deep primaries (> 4 mm deep) or regional lymph-node involvement are classified as high risk and should be considered for medical therapy as well as surgical therapy. The recommended surgical margins can vary according to which guidelines you refer to (Tables 45.16 and 45.17) and guidelines are constantly being updated so referral to a specialist melanoma centre is recommended for up to date treatment of melanoma.

If a macular lesion is large or situated in a cosmetically sensitive area and the suspicion of melanoma is low, an incision biopsy or one or a number of punch biopsies may be appropriate according to the Australian and New Zealand melanoma guidelines. However, this is at variance to the UK's NICE guidelines. The biopsies should be taken from the most abnormal area of the lesion as judged by dermoscopy.

A shave biopsy is usually contraindicated, as it may compromise pathologic diagnosis and make it difficult to determine the Breslow thickness. However, the National Comprehensive Cancer Network (USA) suggests that shave biopsy is acceptable when the index of suspicion is low, and a broad shave biopsy may help optimize diagnostic sampling in cases of lentigo maligna (melanoma in situ) [19]. In cases where a shave biopsy was done and a melanoma is found, a complete excision biopsy of the lesion should be performed in a regional melanoma referral centre as soon as possible to determine the depth and extent of the lesion.



**Fig. 45.37** Subtypes of melanomas

Melanoma is a histological diagnosis and can only be confirmed by a histopathologist. No single histologic feature is pathognomonic for melanoma, many characteristic features exist. The diagnosis is arrived at by the pathologist finding a combination of diagnostic criteria. This is subjective and best carried out by a histopathologist with a special interest and experience in dermatohistopathology and melanoma in particular. More than one pathologist should view all suspicious slides especially those suspicious of dysplastic naevi and melanoma in situ. All invasive melanoma slides need to be sent to a specialist melanoma centre.

Figure 45.37 outlines the different types of melanomas.

**Dysplastic naevi** are moles that show some atypical features on histology but do not fulfil the full criteria for a melanoma. The level of dysplasia may be mild, moderate or severe. It is not clear what proportion, if any, of dysplastic naevi might progress on to become a melanoma if left undetected. Patients with multiple dysplastic naevi, especially if there is also a family history of dysplastic naevi syndrome, are more likely to develop a melanoma. Any severe dysplastic naevus should be removed with a safety margin as if it was a melanoma in situ.

**Melanoma in situ** occurs when the cancer cells are confined to the epidermis above the basement membrane and so have no metastatic potential while in the in situ stage (Fig. 45.34). They are usually diagnosed after the initial excision with a 2 mm border. Treatment involves excising the scar with a 5 mm border all around including a gener-

ous cuff of subcutaneous fat and further investigations are not usually necessary.

**Superficial spreading melanoma (SSM)** is the most common subtype and accounts for about 70% of all melanomas. They are often seen in young people and most commonly found on the back in men and on the legs in women. They are usually slow growing and macular (flat) initially but will show signs of change in size, shape or colour (Fig. 45.38). Dermoscopy and clinical photographs can be helpful in assessing a flat pigmented lesion where the diagnosis is uncertain or where there is no clear clinical history. Photographs can be shared with colleagues by email or on various online discussion forums but ensure all images



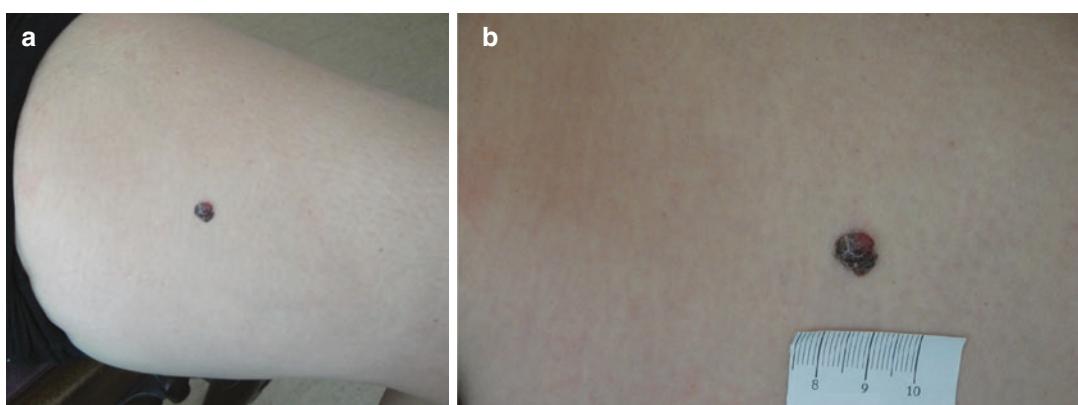
**Fig. 45.38** Superficial spreading melanoma 0.5 mm deep

conform to the requirements of GDPR in terms of patient's consent and confidentiality. Where the risk of melanoma is low and the history is uncertain, *flat* pigmented lesions may be photographed preferably with dermoscopy images and reviewed in 3 months, looking for changes that might suggest malignant transformation. However, *nodular* lesion that are suspicious for melanoma (pigmented or non pigmented) should always be biopsied immediately as nodular melanomas can grow rapidly and metastasize early.

SSM initially go through a horizontal growth phase which can last 12 to 18 months before going into a more dangerous vertical growth phase.

**Nodular (elevated) melanomas** grow rapidly and spread early, often within 6 months of appearing (Fig. 45.39a, b). If there is any doubt about a nodular pigmented lesion or a nodular non-pigmented lesion, particularly if it is changing in size shape or colour, it should be excised with a 2 mm border for definitive diagnosis or referred urgently to a colleague with more experience in lesion recognition.

**Acral lentiginous melanomas** arise in the palms of the hands and fingers, the soles of the feet or toes, under the nails (subungual) or on the genitalia. It is the most common melanoma in African-Americans and Asians and makes up approximately 75% of melanomas in this group. They may or may not be pigmented. Any suspicious lesions on the hands or feet should be referred for a second opinion and/or biopsy (Fig. 45.5a, b).



**Fig. 45.39** (a) Nodular melanoma left thigh 4.5 mm deep, (b) Close up of same melanoma on left thigh

**Subungual melanomas** may be pigmented or non-pigmented. A pigmented subungual melanoma can be difficult to differentiate from a subungual haematoma or a subungual benign naevus. If the pigment spreads from under the nail to involve the periungual skin (Hutchinson's nail sign) then the nail should be removed and the underlying lesion biopsied as this is a sign that the lesion may be a melanoma.

Melanomas under the nail may present as a fleshy pigmented or nonpigmented growth that may lift and distort the nail plate and can occasionally be confused with an ingrown toenail or a fungal nail infection. Where there is any doubt about a subungual growth or suspicious pigmentation the patient should be referred to a specialist. Acral melanomas have a poor prognosis as they tend to metastasize early.

**Amelanotic melanomas** are classically described as skin colored but may be red, pink, or erythematous. Amelanotic melanomas are particularly dangerous as the lack of pigment can lull the patient and the doctor into a false sense of security. These lesions may have a very small amount of pigment, (hypo-melanotic) that may only be apparent with a magnifying lens or with a dermoscope. Amelanotic melanomas represent 2–20% of all melanomas depending on the source of the statistics [20] although a recent small GP based study showed that almost a quarter of all melanomas had little or no pigment [4]. Amelanotic melanomas often evade diagnosis by masquerading as other pathology. They may be macular (flat) or nodular (raised). Amelanotic melanomas may resemble other benign or malignant lesions such as pyogenic granuloma, BCC, AK, Bowen's disease or SCC (Figs. 45.3 and 45.5). The doctor should always have a high index of suspicion for any new lesion (pigmented or non-pigmented) that is changing in size, shape or colour or looks different from any other lesion on the body and where a confident, named, clinical diagnosis cannot be made. All lesions suspected of being a pyogenic granuloma should have tissue sent for histology to rule out amelanotic melanoma.

**Lentigo maligna** are pre-invasive melanomas that grow slowly. They are most commonly found on the face and exposed skin in the elderly (Fig.

45.34). They can grow slowly over many years and become quite large (10–30 mm) and some may eventually develop into a **lentigo maligna melanoma** particularly the larger ones. It is estimated that only 3–5% of lentigo maligna will become invasive [21]. Malignant transformation of a lentigo maligna to a lentigo maligna melanoma can sometimes take 10–15 years or more and is usually clinically apparent as the macular lentigo maligna becomes papular or nodular and there may be bleeding or superficial ulceration. If this happens the patient should be referred for immediate excision provided the patient is fit enough for what might be major surgery. It can be difficult to distinguish a lentigo maligna from a solar lentigo or a macular seborrhoeic keratosis on the face in the elderly. Dermoscopy may help. If there are any doubts it may be safer to remove the whole lesion for histology or if this is difficult, at least take a number of punch biopsies from the most suspicious areas as indicated by dermoscopy in trained hands. Reflectance confocal microscopy can help specialists diagnose ambiguous lesions.

Lentigo maligna in a younger patient (under the age of 70) are best excised with at least a 5 mm border of clear skin. Large lentigo maligna in a frail, elderly patient with multiple comorbidities can present a therapeutic challenge, as the patient may succumb to other incidental illnesses before the lentigo maligna has had time to develop into a lentigo maligna melanoma. Lentigo maligna may respond to 5% imiquimod ("Aldara®") or cryosurgery but these treatments should be reserved for patients with lentigo maligna who cannot or will not undergo excisional surgery. Immunocryosurgery is also an option for these selected patients (see Chap. 58).

## 45.10 Merkel Cell Carcinoma (MCC)

This is a rare, aggressive and often fatal neuroendocrine skin cancer that is linked with the polyomavirus (MCPyV) in 80% of cases. The incidence has been increasing probably because of increasing longevity and the increased use of immunosuppression [22]. The "AEIOU" characteristics can help identify these tumours. They are



**Fig. 45.40** Merkel cell carcinoma in a 85 year old female

usually **asymptomatic** (A), and **expand rapidly** (E). They are more common in patients who are **immunosuppressed** (I), although over 90% of patients with MCC are not immunosuppressed. They are more common in **older** patients >50 years old (O) and in **UV light exposed skin** (U) (Fig. 45.40).

MCC are often confused with benign lesions such as cysts, folliculitis or lipomas as they present as a smooth, non-ulcerating, non scaly nodule. However, if a lesion is non-tender, growing rapidly and occurs in an elderly or immunosuppressed patient in a light exposed site, a biopsy should be taken. These rare tumours need to be managed by a multidisciplinary team consisting of plastic surgery, radiotherapists and oncologists. They need very wide local excision and possibly local radiotherapy as local recurrence is very high (up to 50%). Some more advanced MCCs may respond to immunotherapy, including programmed cell death protein 1 (PD1) pathway blockade therapy. Electrochemotherapy has also been used successfully for treating more resistant MCC.

## 45.11 Dermatofibrosarcoma Protruberans (DFSP)

This is a rare soft tissue sarcoma that develops in the deep layers of skin. It grows very slowly and there are usually no symptoms unless it gets very large and can press on adjacent structures. It is more common in women in their 20s to 50s and

in the Afro-Caribbean population. Metastases are rare (~5%) but the tumour can invade local structures such as tendons, muscle or bone.

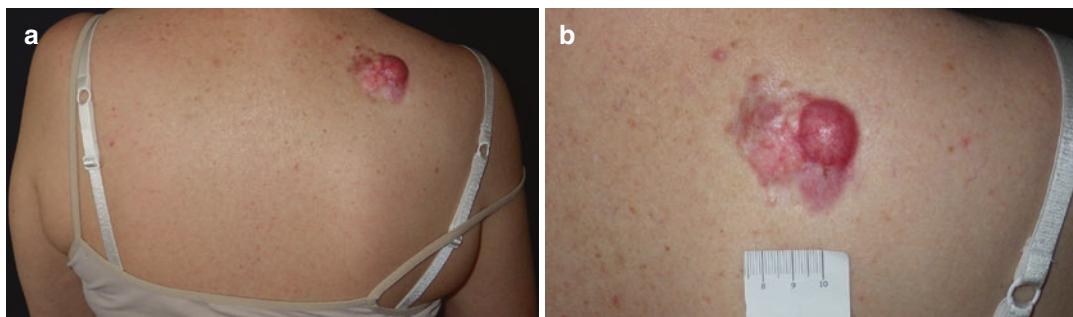
DFSP usually presents as a flat or slightly raised patch of skin that feels rubbery or hard to the touch. It often looks like a scar or keloid that may be violet, reddish brown or skin colored. Some cases can grow very large because they usually do not cause any pain, bleeding or crusting and are often mistaken for benign lesions such as a lipoma, epidermoid cysts (sebaceous cyst) or keloid (Fig. 45.41a, b).

Treatment is by wide local excision and more advanced cases may benefit from radiotherapy or targeted therapy such as oral **imatinib** which is in a class of medications called kinase inhibitors which works by blocking the action of the abnormal protein that signals cancer cells to multiply.

**Mycoses fungoides** (cutaneous T-cell lymphomas): Mycosis fungoides is a rare type of cutaneous T-cell lymphoma (thymus-derived). It is a type of non-Hodgkin lymphoma. Despite the name this condition has nothing to do with fungus. It usually presents as a red, slightly scaly, itchy rash on the skin that can easily be confused with eczema/dermatitis or psoriasis and may respond to potent topical steroids. In more advanced diseased, tumours and ulcers may develop in the skin after many years of the rash. Diagnosis is usually made on skin biopsy. In patients with what looks like “chronic eczema” in large plaques, always take a biopsy. Sometimes, the biopsies need to be repeated over time and the reports may diagnose “chronic dermatitis” especially early in the disease. In some cases it takes years until the biopsy shows MF. Treatment options include potent topical steroids, phototherapy, oral retinoids or chemotherapy. The prognosis is usually good and most patients can be controlled although not necessarily cured of their disease.

## 45.12 Kaposi Sarcoma

This is a very rare form of cutaneous sarcoma that is now most commonly associated with HIV infection where the human herpes virus 8 is



**Fig. 45.41** (a) Dermatofibrosarcoma protuberans (DFSP) growing for 8 years on a 47-year-old female's back, (b) Same DFSP (close up)

thought to be responsible. However, there are some types not associated with HIV such as the classic type (mostly found in Mediterranean men), the endemic type most commonly seen in tropical Africa and the iatrogenic type in people immune-suppressed due to various medical treatments.

Kaposi sarcoma usually presents as single or multiple red or purplish macules, papules and nodules on the skin or mucous membranes including in the mouth, nose, and throat. It may also cause enlarged lymph nodes and may affect internal organs. Initially, the lesions are small and painless but they can ulcerate and become painful. Diagnosis is usually by skin biopsy of a new lesion and treatment varies considerably depending on the underlying cause and extent of the disease. HIV related KS may respond to anti-retroviral treatment.

### 45.13 Paget's Disease

Paget's disease is a rare skin cancer characterised by a chronic eczema-like rash of the nipple and adjacent areolar skin mostly found in women in their 50s and 60s. Mammary Paget's disease is associated with an underlying cancer, either *in situ* adenocarcinoma of the breast or a more widespread infiltrating cancer.

A similar condition known as extramammary Paget's disease can be found in the vulva, penis or perianal skin in men or women. Any eczematous rash on the nipple or ano-genital skin that does not respond to topical treatments such as steroids or topical calcinurea inhibitors should be biopsied to rule out a malignancy. Extramammary Paget's disease is usually associated with an underlying malignancy such as cervical or anal cancer.

### 45.14 Secondary Skin Cancer

Some internal malignancies may metastasize to the skin. Although this is rare, the most common ones are breast (Fig. 45.42), bowel, lung, oropharyngeal or ovarian cancer. An occult melanoma may metastasize to the skin and the primary site may never be found. The scalp is a very common site for skin metastasis. The clinical features may alert the doctor such as multiple new skin lesions in a patient with a known internal malignancy. Histology may also help alert the doctor to a possibility of an underlying malignancy.

Skin metastasis may present as one or more firm, round or oval, mobile, non-painful nodules. They may be rubbery, firm or hard in texture and vary in size from a few millimetres to several centimetres in diameter. These may be skin coloured, red, blue or black.



**Fig. 45.42** Breast cancer with cutaneous secondaries in a 83 year old female

## 45.15 Conclusion

Skin cancer can present as a mole or lesion that is new or changing, looks different from any other lesion on the body or is sore. The exact type of skin cancer and subsequent management can only be decided after taking a skin biopsy of the lesion if clinically indicated. All patients with any type of skin cancer are at high risk of developing another skin cancer and they should be advised to protect their skin from UVL (see Chap. 49) and should take extra vitamin D orally (approx 800–2000 iu/day especially over the winter). All patients with skin cancer should be educated on the warning signs of skin cancer and should be encouraged to check their own skin from head to toe at home monthly for the rest of their lives. Recent studies have shown that patients who have already had one NMSC have a 40% chance of developing a second within 5 years. If a patient has had more than one NMSC they have a 82% chance of developing another within 5 years [23]. Patients with NMSC are at increased risk of developing a new melanoma. All patients with melanoma or more than one NMSC should have an annual skin check for life with a doctor. UVL protection and early detection are the best defence against skin cancer.

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# Dermoscopy for the General Practitioner

46

David Buckley

## Key Points

- Dermoscopy is not strictly diagnostic, but with training it can increase the sensitivity and specificity of the clinical diagnosis of melanoma and BCC.
- The “ugly duckling” sign, which is useful when examining moles with the naked eye (i.e. looking for the mole that looks completely different from all other adjacent moles), can also be applied to the dermoscopic skin review.
- The dermoscopy image can be stored as a digital photograph to evaluate changes over time and sharing images for second opinion.
- Dermoscopy is essential for any doctor with a special interest in skin lesion recognition and skin cancer detection.

## 46.1 Introduction

Dermoscopy (also known as dermatoscopy, epiluminescence microscopy or skin surface microscopy) is a quick, non-invasive, method that can be used for the diagnosis of various skin lesions. When used in conjunction with careful history and a thorough physical examination, dermoscopy can increase the clinician’s diagnostic accuracy, especially when evaluating possible skin cancers.

However training is essential and any doctor with a special interest in skin lesion recognition and skin cancer detection should get trained [1].

## 46.2 The Dermatoscope

The dermatoscope is essentially a hand held microscope that uses magnifying optics (10X) and trans-illuminating light sources (usually LED lights) which, unlike a magnifying glass, will allow the doctor to visualise deeper structures located within the epidermis, the dermal-epidermal junction and the papillary dermis.

Conventional dermoscopy uses an oil or gel interface to limit the amount of reflected light, allowing better visualisation of pigment and structures within the skin. Newer devices use LED bulbs with cross polarising light filters which eliminates the need for oil/gel and will allow visualisation of deeper structures including blood vessels. A good hand held dermatoscope should cost roughly €500–€1200. Most dermatoscopes can be attached to a smartphone using the appropriate attachment (usually commercially available with the dermatoscope). This gives a unique possibility to get a second opinion on a lesion, especially if not trained in dermoscopy.

While dermoscopy was initially developed by dermatologists to assess pigmented lesions such as moles, it is now used by many clinicians

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including an increasing number of GPs to diagnose a wide variety of skin problems. These include non-pigmented lesions, hair and scalp (trichoscopy), sub-ungual structures, nail folds and parasite infestations such as scabies. It can also be used to delineate the borders of skin tumours prior to excision and the assessment of where best to take a biopsy when sampling a lesion for diagnosis. In Australia, where there is a very high incidence of skin cancer, GPs consider the dermatoscope to be as essential as a stethoscope.

Dermoscopy is not strictly diagnostic, but with training it increases the sensitivity and specificity of the clinical diagnosis of melanoma and BCC. An important and useful result of dermoscopy has been the reduction in the number of biopsies of common benign lesions that have characteristic dermoscopic features such as benign naevi, blue naevi, seborrhoeic keratoses, haemangiomas and dermatofibromas [1]. These lesions may mimic skin malignancies on naked eye examination. Dermoscopy can help decide whether to reassure the patient, review after 3 months, biopsy the lesion or refer the patient to someone with more experience.

### 46.3 Melanocytic Lesions

Dermoscopy is an invaluable tool that will help the doctor to decide if a pigmented lesion is

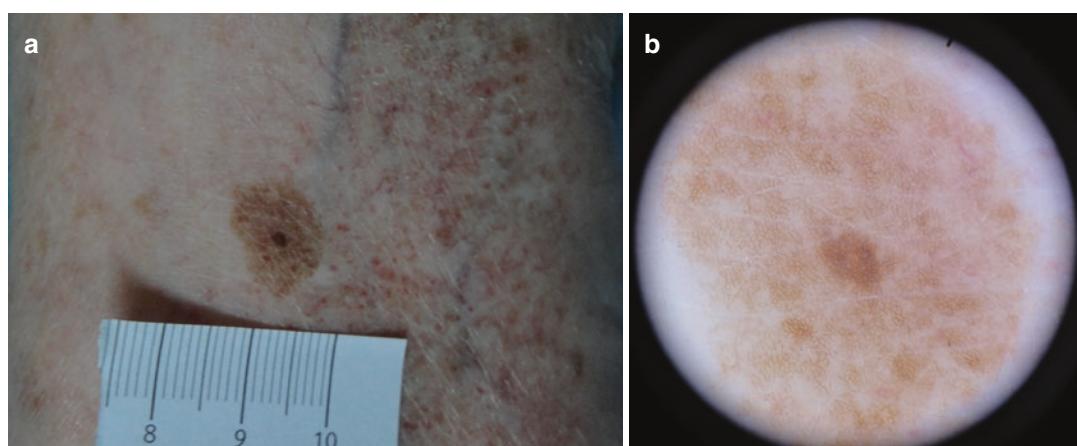
melanocytic (i.e. from a mole) or not. The presence of pigmented networks (Fig. 46.1a, b), globules, dots or streaks favour a melanocytic lesion (Fig. 46.2) [1].

Dermoscopy will also help doctors to identify features which may indicate that a melanocytic lesion is benign (homogeneous colour and structure) (Fig. 46.1a, b), suspicious (red and brown or three colours or irregular edges) (Fig. 46.3a, b) or malignant (blue/white veil, radial streaking, irregular dots or globules or an irregular network) (Fig. 46.4a, b).

Many common non-melanocytic (i.e. lesions that are not naevi but may be pigmented or not) lesions have characteristic dermoscopic features which will help in their diagnosis (Fig. 46.2) (Figs. 46.5a, b, 46.6a, b, 46.7a, b, and 46.8a, b). Non-melanocytic lesions that do not have characteristic dermoscopy features as outlined in Fig. 46.2 should be biopsied to rule out amelanotic melanoma.

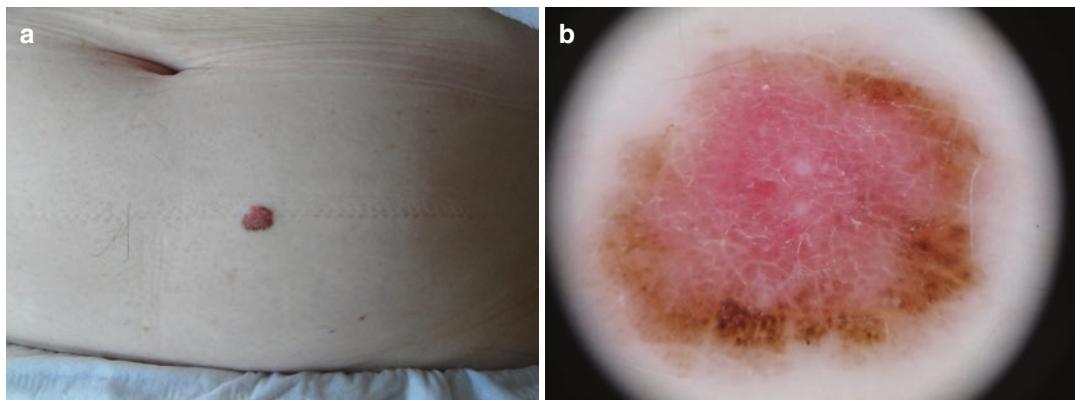
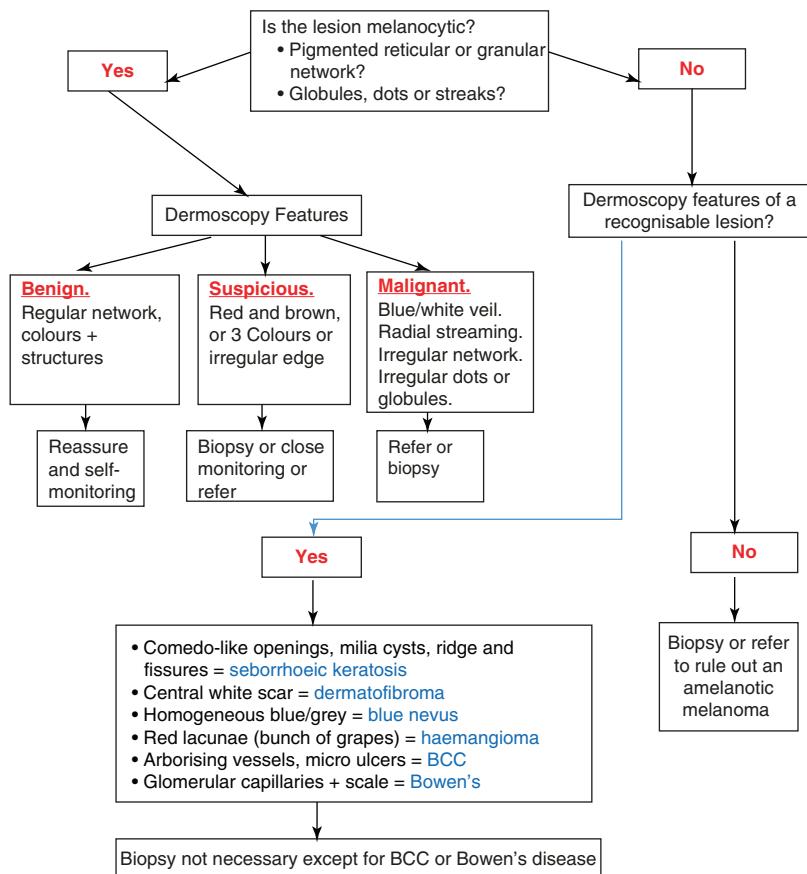
In the absence of specific colours or structures, the blood vessel morphology can assist in making the correct diagnosis. Care should be taken when using contact dermoscopy not to press too hard on the lesions as this will empty the blood vessels and hide their shape and size (Figs. 46.9a, b, 46.10a, b, 46.11a, b, 46.12a, b, 46.13a, b, and 46.14a, b).

Without formal training, the use of dermoscopy may result in poor performance compared with naked eye examination. Dermoscopy



**Fig. 46.1** (a) Stable mole on the foot. (b) Dermoscopic image of the same stable benign mole

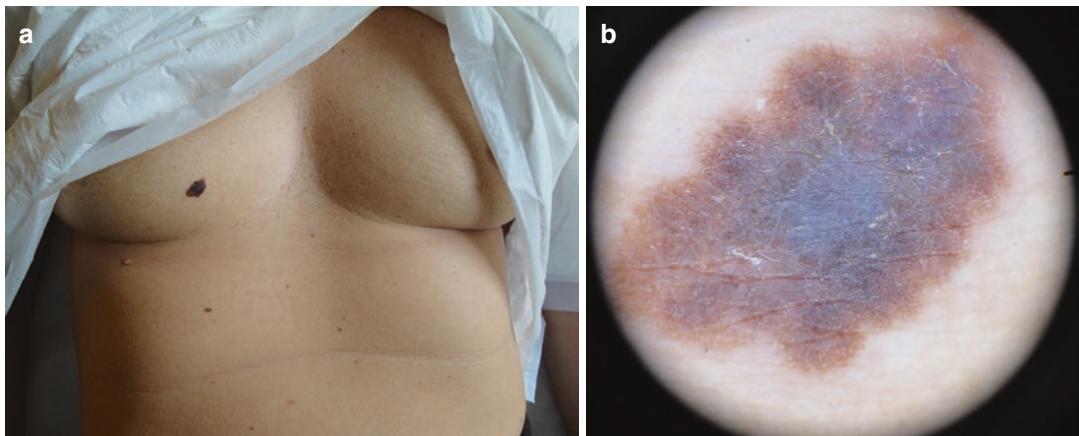
**Fig. 46.2** Basic diagnostic algorithm for Dermoscopy



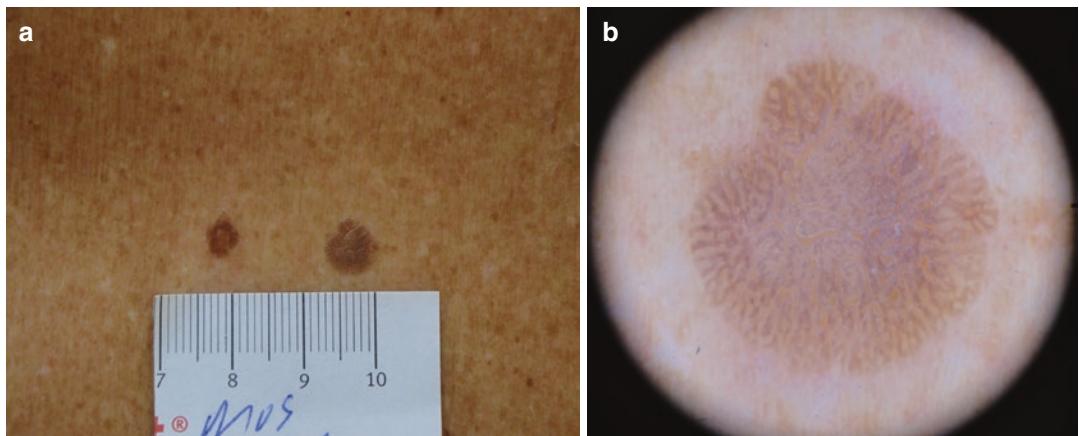
**Fig. 46.3** (a) Severely dysplastic naevus on the abdomen with features of Spitz naevus on histology. (b) Dermoscopic image of the same naevus

requires the user to learn and understand new terminology in relation to colours and structures, specific to dermoscopy (Table 46.1). New users should attend a training course for

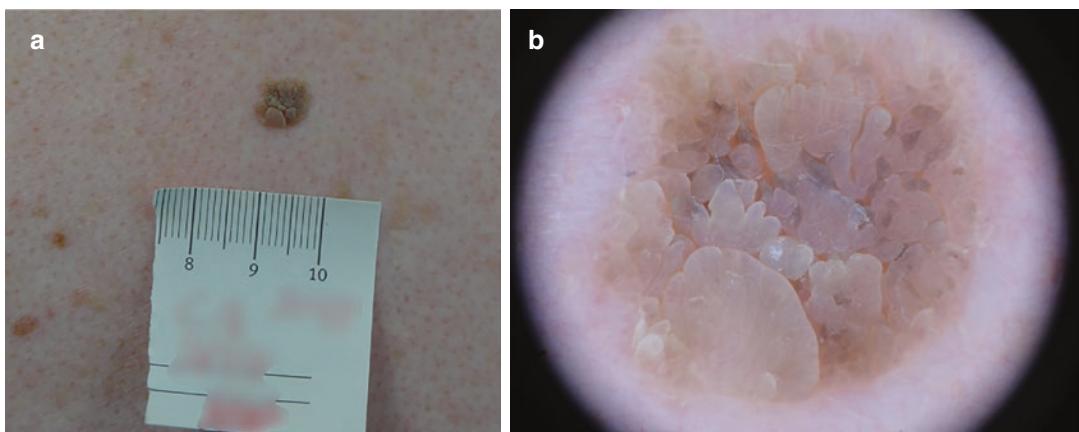
beginners supplemented with online tutorials, suitable text books and CD's with lots of reference images (Table 46.2). The new user should look at as many benign lesions as possible to



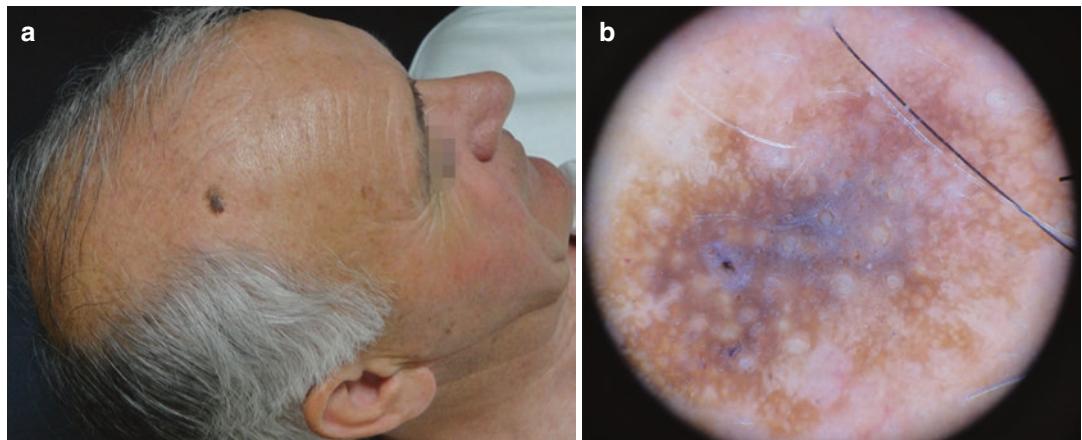
**Fig. 46.4** (a) Superficial spreading melanoma (SSM) in a 45 year old. The mole was present for 4 years but got bigger and itchy in last 10 months. (b) Dermoscopic image of the same SSM



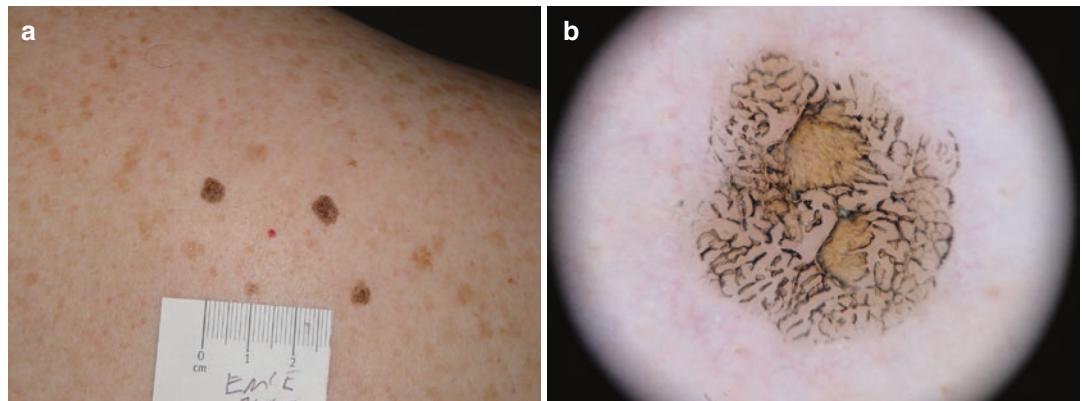
**Fig. 46.5** (a) Seborrheic keratosis on the right and stable mole on the left. (b) Same patient showing the seborrheic keratosis on dermoscopy



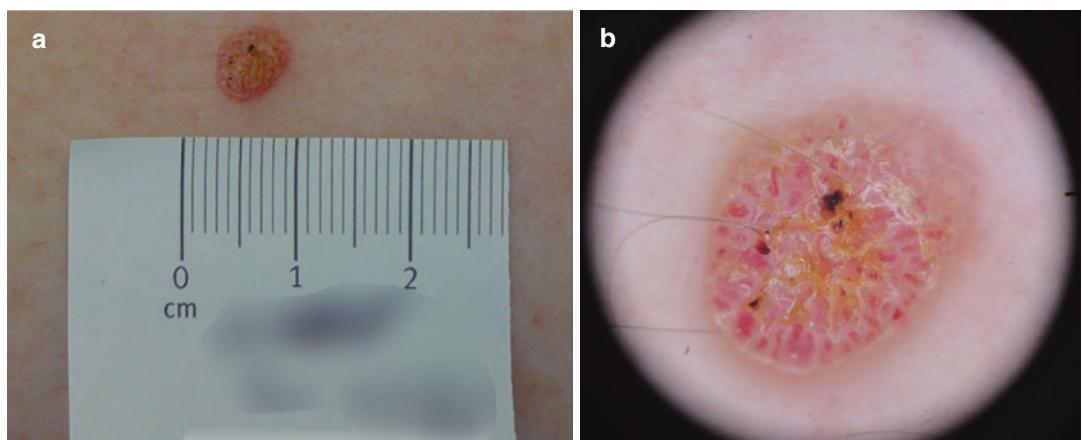
**Fig. 46.6** (a) Seborrheic keratosis. (b) Same patient showing seborrheic keratosis on dermoscopy



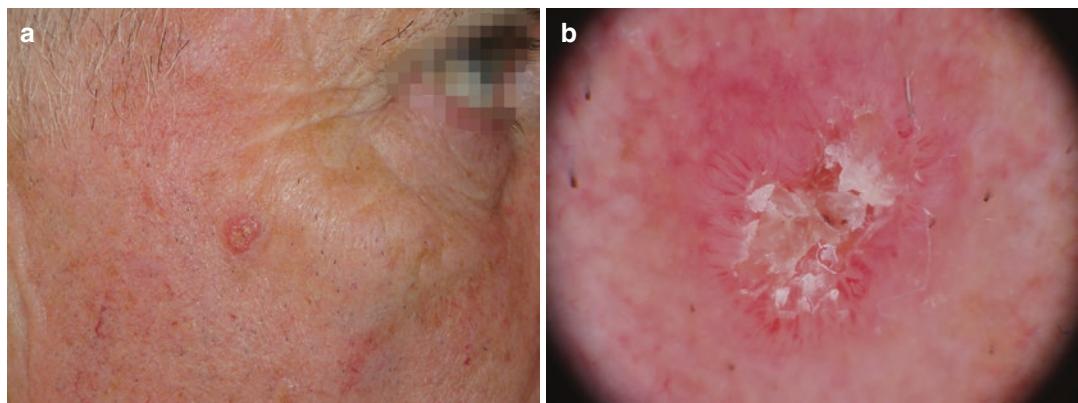
**Fig. 46.7** (a) Seborrheic keratosis. (b) Same lesion on dermoscopy



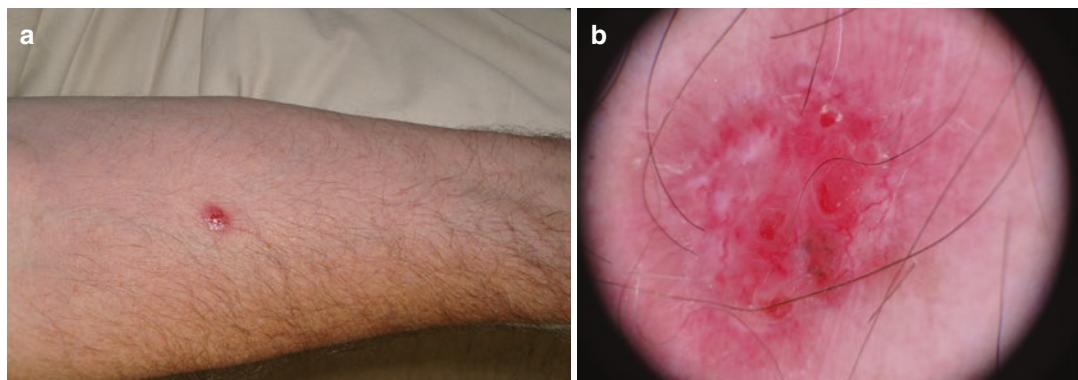
**Fig. 46.8** (a) Three seborrheic keratosis. (b) Same patient showing seborrheic keratosis on dermoscopy



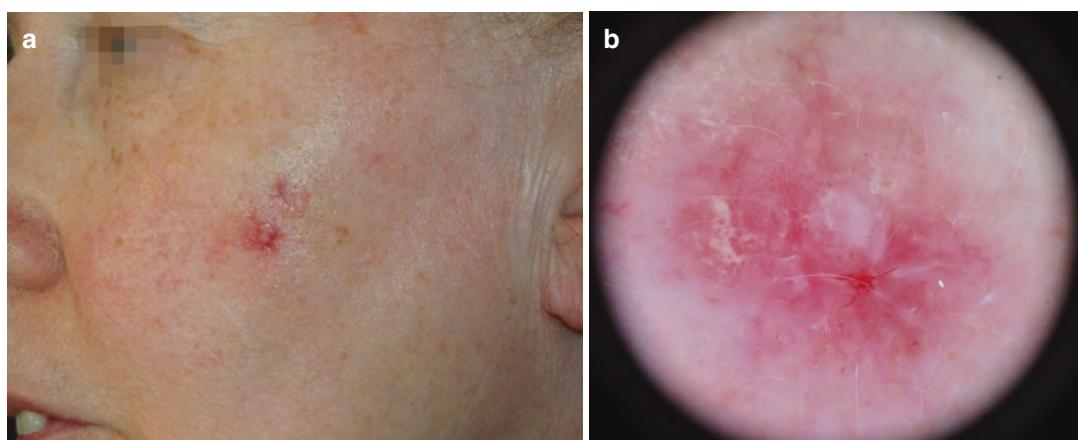
**Fig. 46.9** (a) Seborrheic keratosis. (b) Same patient showing seborrheic keratosis on dermoscopy



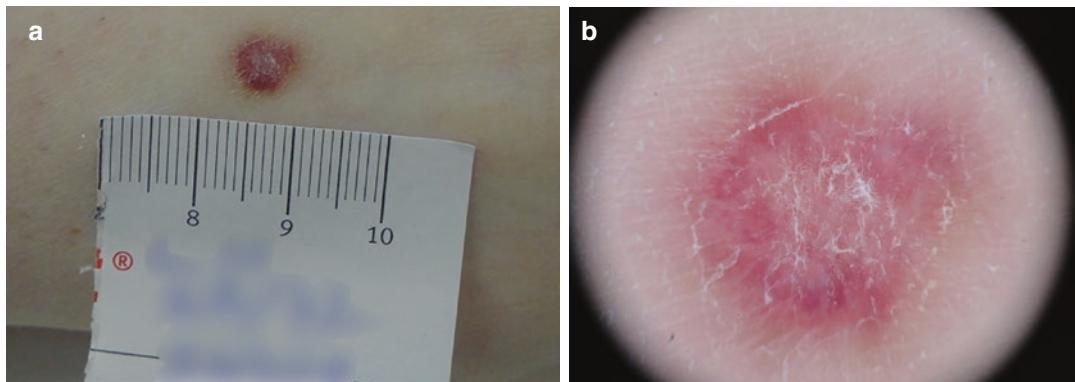
**Fig. 46.10** (a) Bowen's disease. (b) Same patient showing Bowen's disease on dermoscopy



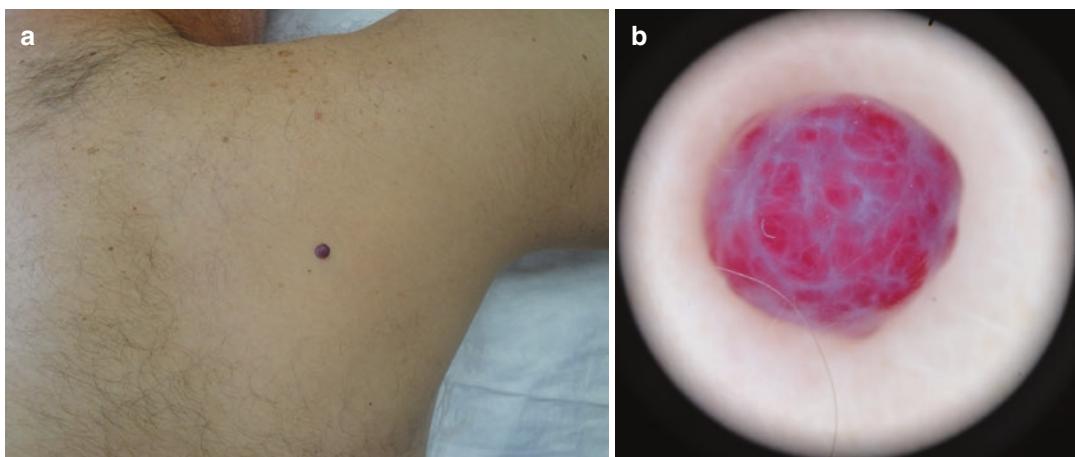
**Fig. 46.11** (a) BCC upper forearm. (b) Same BCC on dermoscopy showing arborising vessels at 3 o'clock, ovoid nests at 6 o'clock and superficial ulceration



**Fig. 46.12** (a) Amelanotic melanoma 1.4mm deep. (b) Same amelanotic melanoma on dermoscopy



**Fig. 46.13** (a) Dermatofibroma on the ankle. (b) Same lesion on dermoscopy



**Fig. 46.14** (a) Haemangioma on the upper back. (b) Same haemangioma on dermoscopy

**Table 46.1** Terminology in dermoscopy

Colours:	Yellow	= Keratin
	Red	= Blood
	White	= Collagen
	Black, brown, grey or blue	= Melanin – colour dependant on depth
Structures:	Pigmented network	
	Globules	
	Streaks	
	Milia-like cysts	
	Comedo-like openings	
	Gyri (ridges) and sulci (fissuring) (cerebriform = brain like)	
	Arborising vessels (like the branches of a tree in winter)	
	Leaf-like structures	
	Ovid nests	

learn to recognise normal colours and structures within benign moles. This will help them to recognise when a lesion shows some abnormal patterns or colours which may indicate that it might need to be biopsied or referred. The “ugly duckling” sign which is useful when examining moles with the naked eye (i.e. looking for the mole that looks completely different from all other adjacent moles and other moles on different parts of the body) will also work with dermoscopy. A melanocytic lesion (i.e. a mole) which has colours and structures on dermoscopy which are completely different from all the other nevi in the adjacent area (“the ugly duckling”) should be viewed with suspicion.

## 46.4 Digital Photography

Another advantage of dermoscopy is that digital photographs can be taken of the lesion through the dermatoscope using a camera or smart phone attachment. The image can be shared with colleagues online on various discussions forums (Table 46.2).

Digital images can also be used to monitor low-risk macular (flat) lesions over the course of 3 months, checking to see if there are any suspicious changes in that period of time. Nodular (rasied) lesions should never be followed up as a nodular melanoma can grow quickly and spread rapidly, so if there are any doubts about a nodular lesion, it should be excised fully or referred immediately.

One difficulty with dermoscopy is its failure to detect very early or featureless melanomas. This is also a problem with naked eye examination or examination with a magnifying lens. All patients should be advised to return for a follow-up if there is a change in size, shape or colour of their mole for re-assessment.

Dermoscopic features of melanoma and pigmented BCCs can sometimes show overlapping features which may lead to confusion when trying to decide if the lesion is a melanoma or a pigmented BCC. Dermoscopy usually displays enough suspicious features in both of these lesions to suspect a cutaneous malignancy and prompt a biopsy which is the definitive diagnostic tool.

Diagnostic accuracy with dermoscopy depends on good training and experience which will allow the user to develop skills in pattern recognition of microscopic structures and colours, which will enhance the clinician's ability to distinguish between melanomas, BCC's and many common benign lesions. If a doctor experienced in dermoscopy is still looking at a suspicious lesion after 1 min he/she should be considering referring the patient or removing the lesion for histology as most benign lesions and obvious melanomas can be diagnosed quickly with dermoscopy (Figs. 46.15a, b, 46.16a, b, 46.17a, b, 46.18a, b, 46.19a, b, 46.20a, b, and 46.21a, b).

**Table 46.2** Further reading

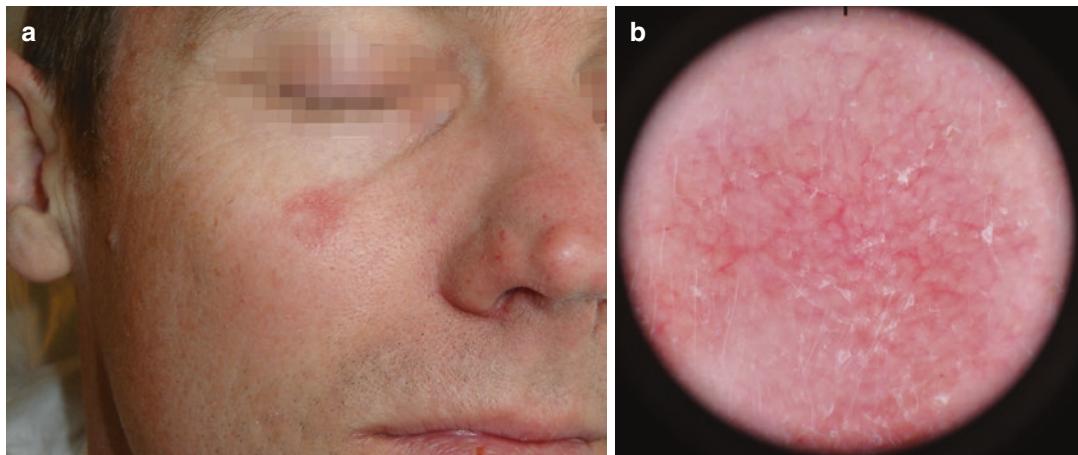
Books:	– “Diagnostic Dermoscopy. The illustrated guide” by Dr. Jonathan Bowring. Atlas of Dermoscopy” by Ashfaq A Marghoob, Josep Malvehy, Ralph P Braun
Websites:	– International Dermoscopy Society (IDS)— <a href="http://www.dermoscopy-ids.org/index.php/education/">www.dermoscopy-ids.org/index.php/education/</a>
Podcasts:	<a href="http://www.genomel.org/dermoscopy">www.genomel.org/dermoscopy</a> <a href="http://www.pcsa.iehas">www.pcsa.iehas</a> a discussion forum for members <a href="http://www.youtube.com/user/dermconsult">www.youtube.com/user/dermconsult</a>

Scientific papers available free on line:

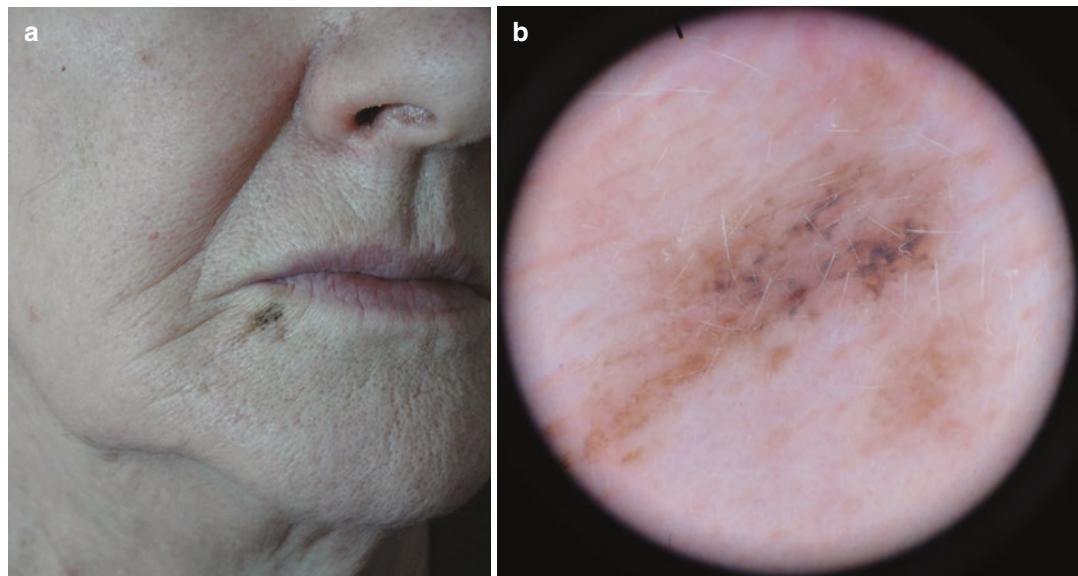
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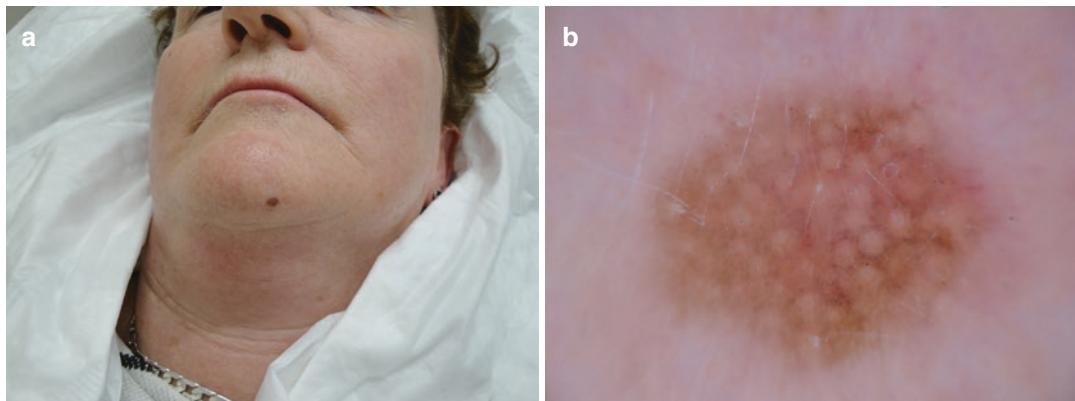
**Fig. 46.15** (a) Ink spot lentigo upper back. (b) Same ink spot lentigo on dermoscopy



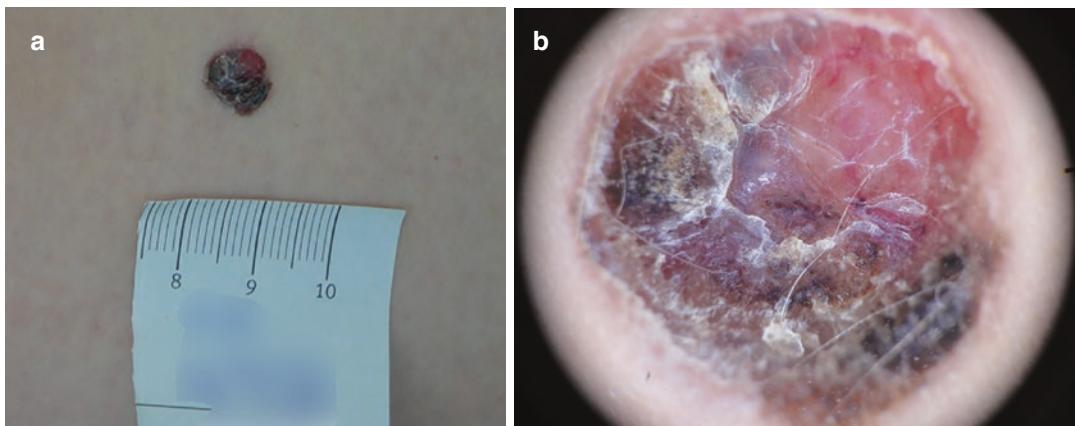
**Fig. 46.16** (a) Actinic keratosis. (b) Same actinic keratosis on dermoscopy



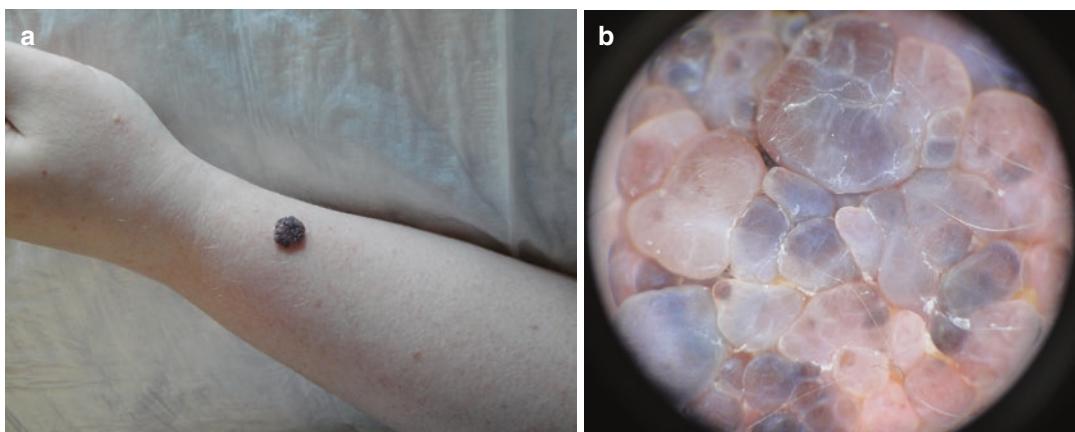
**Fig. 46.17** (a) Lentigo maligna present for 4 years; bigger and darker for the last 6 months. (b) Same lentigo maligna on dermoscopy



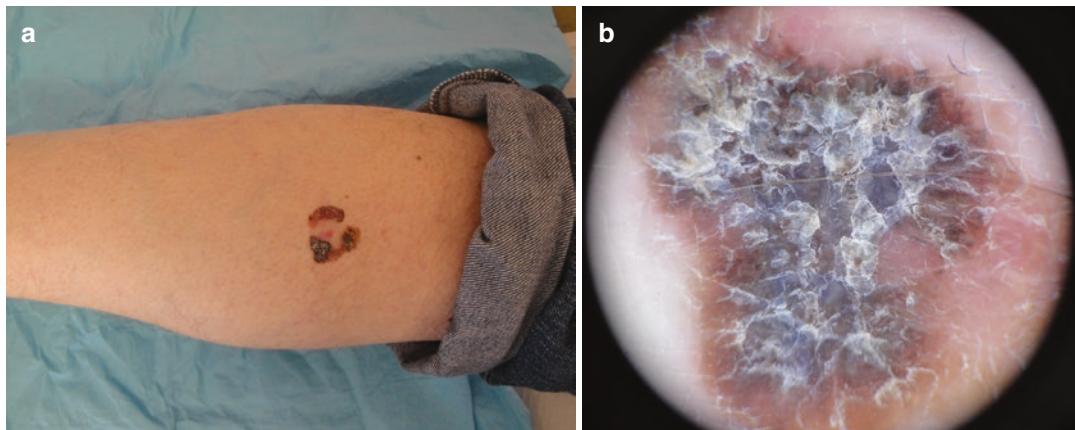
**Fig. 46.18** (a) Melanoma in situ presented as a new mole for the last 8 months. (b) Same melanoma in situ on dermoscopy



**Fig. 46.19** (a) Nodular melanoma L thigh. There for years; more raised for the last 5 months. 4.5 mm deep on histology. (b) Same nodular melanoma on dermoscopy



**Fig. 46.20** (a) Naevoid melanoma 3.9mm deep on histology. (b) Same naevoid melanoma on dermoscopy



**Fig. 46.21** (a) Nodular melanoma 4mm deep. (b) Same nodular melanoma on dermoscopy

## 46.5 Conclusion

The National Cancer Control Programme (NCCP) in Ireland and NICE in the UK currently recommend that GPs refer all lesions suspicious of melanoma to a regional referral pigmented lesion clinic. However, these clinics are often overly busy assessing mostly benign lesions. Studies have shown only a very small proportion of the lesions seen in pigmented lesion clinics turn out to be melanomas (2–8%) [2, 3]. GPs should be encouraged to enhance their diagnostic skills by the use of dermoscopy. If every group practice had at least one doctor trained in dermoscopy and inter-referral among GP was encouraged and supported by

the health services, many unnecessary referrals to pigmented lesion clinics might be avoided.

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## **Part X**

### **Pigment and the Skin**



# Disorders of Pigmentation

47

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## Key Points

- Too much or too little pigmentation can cause problems, particularly if it is blotchy and occurs on exposed areas such as the face and hands.
- Topical treatments of vitiligo include potent topical steroids for 3 months on the body or topical calcineurin inhibitors such as tacrolimus on the face. Surgical treatment, UVL therapy or systemic treatments such as oral steroids, methotrexate or afamelanotide (an analogue of  $\alpha$ -melanocyte-stimulating hormone) may be necessary in more severe cases.

## What to Tell the Patient

- Disorders of pigmentation can cause considerable cosmetic disfigurement with subsequent emotional distress. Most cases can be helped by cosmetic camouflage, a high factor sun block (SPF 30 or greater) and depigmenting creams when necessary.
- Hydroquinone (1, 4-dihydroxybenzene) is the gold standard for treating post inflammatory hyperpigmentation. Side effects are less common if hydroquinone is used under careful medical supervision and limited to 3–12 months in duration.
- Careful sun protection of the skin affected with vitiligo is important as these areas will

burn easily and repeated exposure to UVR could lead to skin cancer.

## 47.1 Introduction

Too much or too little pigmentation can cause problems, particularly if it is blotchy and occurs on exposed areas such as the face and hands. Dark skin types have a higher content of melanin and more effective distribution of melanin for protection against ultra violet radiation (UVR). The black epidermis on average provides the equivalent of sun protection factor (SPF) 13 [1]. This can create problems when people with dark skin move to damp temperate climates, as their ability to produce vitamin D via their skin is severely compromised and many will need to take vitamin D supplements (at least 800iu per day in adults).

Disorders of pigmentation can cause considerable cosmetic disfigurement with subsequent emotional distress. Most cases can be helped by cosmetic camouflage and a high factor sun block [2].

## 47.2 Hyperpigmentation

The higher melanin content in dark skin can make it more prone to **post-inflammatory hyperpigmentation** (PIH) as a result of injury, infection or inflammatory skin diseases such as tinea infection, atopic eczema, or acne. (Fig. 47.1). The post-inflammatory changes can persist

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**Fig. 47.1** Acne patient with postinflammatory hyper-pigmentation

and may be permanent even after the initial inflammatory process has resolved. This can cause considerable cosmetic and emotional problems, especially when it affects the face or hands.

Skin diseases that normally cause hyperpigmentation in white skin such as morphoea and acanthosis nigricans can be more difficult to diagnose in people with dark skin and sometimes require a biopsy to confirm the diagnosis.

**Melasma** can occur in any skin type but is more common in people with Fitzpatrick skin type 4 and 5. It is more obvious in light coloured skin and can cause significant areas of light to dark brown patchiness with irregular borders symmetrically distributed on the face, which can be unsightly (Fig. 47.2). Melasma can be precipitated by pregnancy, oestrogen, progesterone, phenytoin and UVR. It is nine times more common in women.

PIH and melasma can be difficult to treat and the first approach is to deal with any underlying inflammatory process and to protect the skin from ultraviolet light (UVL). Cosmetic camouflage can be very helpful if the patient is self-conscious about their appearance.

Treatment of PIH continues to be a challenge as there is no universally effective treatment and existing agents have varying degrees of success. There are various skin lightening agents available both by prescription and over the counter, particularly in Africa and Asia.



**Fig. 47.2** Melasma on the cheeks

Some of these skin lightening creams and soaps may contain potent topical steroids, hydroquinone, mercury or other agents that may cause skin and internal organ damage if used over a long period of time without medical supervision.

Hydroquinone (1, 4-dihydroxybenzene) is the gold standard for treating PIH and has been available for over 50 years. Adverse reactions such as transient irritation, erythema, peeling, confetti-like depigmentation and exogenous ochronosis (EO) (blueish grey hyperpigmentation with or without papulo-nodular lesions) are dose and duration dependant. These side effects are less common if hydroquinone is used under careful medical supervision and limited to 3–12 months in duration. Because they contain a potent topical steroid they have to be used with great caution on the face and should not be used on the eyelids. On the face they should be only be prescribed by doctors who have experience in using hydroquinone.

Other options include chemical peels and lasers such as the 1064 Q-Switched Nd:YAG laser or Intense Pulse Light (IPL). All these treatments have to be used carefully as they may cause inflammation and possibly aggravate PIH.

Azelaic Acid ("Skinoren®") is anti-proliferative and cytotoxic to melanocytes. Some studies have shown azelaic acid 20% to be equally as effective as 4% hydroquinone on PIH and melasma [3].

Topical retinoids (e.g. tretinoin 0.05–0.1% or tazarotene 0.1%) have also been shown to reduce

hyperpigmentation by a number of mechanisms either as a monotherapy or in combination with other agents such as hydroquinone or topical steroids. Adapalene 0.1% ("Differin®") which is a retinoid like agent used to treat acne, has been found to be effective in melasma and is significantly less irritating than other topical retinoid [4].

Kligman and Willis first proposed a combination of hydroquinone, topical retinoid and topical steroids for PIH and melasma. One of the most successful combinations combined formulations has been hydroquinone 4%, tretinoin 0.05% and fluocinolone acetonide 0.01% which is a moderately potent topical steroid (e.g: "Pigmanorm®" or "Tri-luma®"). This formulation can be expensive to buy or have formulated at the pharmacy and the use should be limited to 6–12 weeks under careful medical supervision [5].

### 47.3 Hypopigmentation

Loss of pigmentation may occur after trauma, infection or inflammation (e.g.: atopic dermatitis, DLE, etc.). It is more difficult to get pigment to come back into the skin than it is to lighten areas of hyperpigmentation. In patients from the tropics or subtropics, hypopigmentation with numbness in an area of skin should raise the possibility of leprosy.

The most common cause of hypopigmentation is **pityriasis alba**. It presents as mild hypopigmented ill-defined patches and is common on the face and forearms in children with Fitzpatrick type 3–6 skin (Fig. 47.3). It can be linked with atopic eczema in some children. It usually resolves spontaneously in time and may be helped with moisturisers, avoiding soaps and other irritants and if inflamed, a weak topical steroid such as 1% hydrocortisone or tacrolimus ("Protopic®"). **Pityriasis versicolour** (PV) can also cause hypopigmentation (Chap. 31). The patches in PV are well defined and scaly; they can be present in the face but are mostly in the upper back and chest.



**Fig. 47.3** Pityriasis alba in a 9 year old with type 5 skin

Another distressing form of hypopigmentation is **vitiligo**. This is a common (0.5–2% of the population) autoimmune condition of unknown aetiology but can be linked with other autoimmune diseases such as insulin-dependent diabetes, thyroid disease, pernicious anaemia, Addison's disease and alopecia areata. Other family members can be affected in up to 25–30% of cases [6]. With vitiligo there is usually complete loss of pigmentation but no signs of inflammation such as erythema or scale. New patches can be slightly pink. (Fig. 47.4). It can be localised to only one area of the body or generalised (Fig. 47.5). The later form usually starts in adults on the hands and feet and can later spread to other areas of the body including the face, axilla, genitalia and groin (Fig. 47.6).

While the incidence of vitiligo is similar in different races and ethnic groups, its impact is far greater in dark skinned individuals as the contrast between the involved and uninvolved



**Fig. 47.4** Vitiligo in a child



**Fig. 47.5** Segmental vitiligo in type 5 skin

skin is far more dramatic and unsightly. Treatment usually starts by reassuring the patient that vitiligo is not dangerous or contagious. Careful sun protection of the affected area is important as these areas will burn easily and repeated exposure to UVR could lead to skin cancer. Cosmetic camouflage, when applied properly, can be extremely helpful and the Red Cross provides free clinics to teach patients how to apply these products.



**Fig. 47.6** Vitiligo on the hands

Medical treatments include topical and surgical treatments. Topical treatments include potent topical steroids for 3 months on the body. Topical calcineurin inhibitors such as tacrolimus can be tried on the face [7]. Phototherapy can help by offering some immunosuppression and by stimulation of cytokines (growth factors). Some patients may respond to oral tetracyclines for a few months as they have been shown to have a strong anti-inflammatory effect.

Surgical treatment involves harvesting healthy melanocytes from pigmented skin and transplanting them to involved skin in the same individual. The cells can be harvested by various techniques including punch biopsy, split thickness skin grafts, or blister grafts. The cells or tissue are then transplanted with or without in vitro cultures. The recipient site is prepared to receive the graft by various techniques such as dermabrasion, laser preparation or cryosurgery [8]. This should only be done in stable vitiligo. The definition of stability has changed in time but is considered no new lesions or no progression of existing lesions in the past 1–2 years [9].

Systemic treatments such as oral steroids, methotrexate or afamelanotide (an analogue of  $\alpha$ -melanocyte-stimulating hormone implant usually used in conjunction with UV therapy) may be necessary in severe, resistant, widespread or rapidly progressive cases.

#### 47.4 Conclusion

Too much or too little pigmentation can cause considerable problems for some people, particularly if it affects exposed sites (e.g. face or hands). Many cases can be treated by dealing with the underlying cause. For those unfortunates who cannot be cured, cosmetic camouflage can be an option.

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# Skin of Colour

48

David Buckley

## Key Points

- Unfortunately, most dermatology textbooks have been written by and for light skin tones. Photographs in the major dermatology resources of darker skin types are limited.
- Patients with dark skin types have a higher incidence of certain skin diseases (fungal infections, keloids, folliculitis keloidalis nuchae, traction alopecia, or pseudofolliculitis barbae) and a lower incidence of other skin problems (skin cancer and actinic damage).
- The classical physical signs of various skin conditions such as rashes can look significantly different in dark skin.
- HIV can occur in any race or ethnic group but the highest known incidence is in people from Africa. As with any patient, those with unusual or difficult to treat skin problems should always be considered for HIV testing.
- Patients with darker skin types are more prone to chronic skin colour changes (hypo or hyperpigmentation) as a result of their underlying skin disease or their treatment.
- Physicians should train their eyes to identify skin conditions in any skin colour

## What to Tell the Patient

- People with post inflammatory hyperpigmentation need strict sun avoidance as ultraviolet light (UVL) will make the hyperpigmentation worse.
- Patients with vitiligo or albinism are at high risk of skin cancer on the hypopigmented skin, especially if they live in a hot climate.
- Tight hair braiding can lead to traction alopecia.
- Razor bumps (pseudofolliculitis barbae) in African men may be helped by letting the beard grow.

## 48.1 Introduction

Ethnic Dermatology deals with skin problems in people with dark coloured skin (Type 4, 5 and 6 Fitzpatrick skin types) (see Table 49.4 on Chap. 49). These are usually people with Mediterranean (type 4) Indian or Asian (type 5), or Afro-Caribbean (type 6) skin types. Some people with dark skin types have a higher incidence of certain skin diseases and a lower incidence of others. This may be partly as a result of their skin colour, ethnic and genetic background but may also be as a result of environmental, cultural, financial, nutritional and linguistic factors. These factors can affect the way patients with dark skin present to the health services and how we manage their skin problems.

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As we live in a globalized world, it is our responsibility to be able to diagnose skin conditions on any skin type and correct a historical error of focusing on just one color of skin patient. A common mistake is to confuse human skin color and ancestry. This chapter will try to focus mostly on the aspects of darker skin types that should be considered to reach the proper diagnosis on all skin types.

## 48.2 Epidemiology

There has been a dramatic increase in the number of people with dark skin migrating to Europe and the USA over the past number of years for economic or political reasons. In the UK the percentage of Black Africans has doubled between 2001 and 2011 [1].

The classical physical signs of various skin conditions such as rashes can look significantly different in dark skin. Doctors have often trained using textbooks where most, if not all, images are from white patients and they are not used to identifying skin diseases in other skin types. For example eczema on white skin usually presents as a red, dry, itchy rash, whereas in dark skin, redness may be less conspicuous or absent and a patient may merely have dry, itchy skin with pigment changes as a result of chronic inflammation (Hypo or hyperpigmentation) (see Chap. 47).

Likewise, childhood viral exanthems that usually cause a rash, look different in tone in a child with darker skin. While vitiligo is equally common in light and dark coloured skin and various races, it has a far more devastating effect on those with dark skin as it is significantly more conspicuous and unsightly. Also, patients with vitiligo or albinism are at high risk of skin cancer on the hypopigmented skin, especially if they live in a hot climate.

Patients with darker skin are less likely to suffer from chronic actinic damage as a result of the photoprotective properties of their skin type. Therefore, actinic keratosis, non-melanoma skin cancer, melanomas and solar damage are all less

common in dark skin races. That is not to say that they cannot get skin cancer. However, the incidence is far less than in light coloured skin types. The only exception is Kaposi sarcoma which is much more common in people from Equatorial Africa who usually have dark skinned races. This type of KS is called Endemic (African) Kaposi sarcoma and it is associated with herpesvirus (KSHV) infection.

In addition, there is the Epidemic (AIDS-associated) Kaposi sarcoma which is more frequent in Africa. People from Africa or of African decent are more likely to suffer from skin infections including bacterial, viral, fungal and protozoal infections.

In regards to melanoma, although it is less frequent, people with dark colored skin are often diagnosed later because of the difficulty in diagnosing changes in moles in dark skin types. Acral lentiginous melanoma is a type of melanoma arising on the palm, sole, or beneath the nail (subungual melanoma) and is the most common subtype of melanoma in people with dark skin. Bob Marley died of this type of melanoma.

People with skin types 5-6 tend to have more skin infections and infestations.

The following skin problems are far more prevalent or problematic in people with dark skin, as the inflammation tends to leave mostly hyperpigmentation, which can last for a long period of time.

## 48.3 Acne in Dark Coloured Skin

Acne is the most common skin problem that causes dark coloured patients to present to their doctor [1]. Acne is more common in women with dark skin colour compared to Caucasians [2]. It can present differently in people with dark coloured skin. Pigment changes as a result of postinflammatory hyperpigmentation (PIH) can be more prominent than comedones or the inflammatory features of acne (papules and pustules). Traumatised (picked) lesions tend to leave hyperpigmentation. Africans in particular are



**Fig. 48.1** Acne in dark skin

more likely to use oily products on their face and scalp which can block pores and aggravate acne (Fig. 48.1). Skin lightening creams are also popular in people with dark skin and have various ingredients such as hydroquinone, mercury or topical steroids that can aggravate acne or rosacea. Picking and scratching can also inflame the skin and cause more PIH. UVL can aggravate PIH and so a high SPF non-comedogenic sun block should be encouraged.

Patients with pigment changes occurring as a result of their acne should be treated aggressively with topical and systemic therapies. Certain anti-acne topical agents such as azelaic acid or adapalene can help fade PIH as well as help clear the acne but they can cause dryness and the patient has to be carefully counselled how to gradually introduce these products to ensure long term compliance. Topical glycolic acid can also be helpful.

Oral therapies should be added in for more troublesome acne. Lymecycline 300 mg daily is usually the first line oral treatment and should be continued for at 3 to 6 months. Doxycycline is also a popular choice but can cause photosensitivity in some patients. For severe, resistant cases, isotretinoin may have to be considered particularly if there is a lot of PIH, scarring or keloid formation.

The classical features of acne that are obvious on Caucasian skin (comedones, papules and pustules) may be less easy to see in dark skin types. Other skin conditions may mimic acne and may be more difficult to diagnose in dark skins (Table 48.1). Acne keloidalis nuchae can respond very well to oral isotretinoin.

**Table 48.1** Differential diagnoses of acne in dark coloured skin

Gram-negative folliculitis
Pityrosporum folliculitis (more common on the trunk)
Rosacea
Pseudofolliculitis barbae
Tinea
Drug induced acneform eruptions (e.g. steroids, isoniazid, lithium, anti-neoplastics, halogens)
Hidradenitis suppurativa
Acne keloidalis nuchae

## 48.4 Hyperpigmentation

Hyperpigmentation can be severe in skin types IV, V and VI. Melasma is also very frequent in this group and, in the case of women, it can be aggravated by oral contraception, pregnancies and UVL. Sun protection and depigmenting treatments are required. With pregnancy or oestrogen, **Linea Nigra** (a dark line that develops across the belly, darkening of areolas) can become very prominent.

Children with dark skin are more likely to have Mongolian spot in the lumbar area and sometimes in other parts of the body. This tends to disappear with time. **Pitiriasis alba** is very common after summer vacations (where there is more sun exposure and chlorinated pool playing) in dark skinned children. It improves with hydrating creams and sunblocks. (Fig. 48.2).

Naevi and haemangioma are darker and more difficult to visualise through a dermatoscope because:

1. physicians are not trained to see darker skins through a dermatoscope;
2. most of the texts have been written by and for white skin. The larger amount of pigment makes visualisation more difficult.

Hispanics have a more common segmental haemangiomas, more complex hemangiomas, and greater morbidity from haemangiomas than all other racial and ethnic groups [3].



**Fig. 48.2** Pityriasis alba in a child with type 5 skin



**Fig. 48.3** Dermatosis papulosa nigra

skin. If considering treatment, always treat 1 or 2 lesions in a non visible site and see the result. You need to make sure that healing does not leave an unpleasant looking hyperpigmentation.

## 48.6 HIV Disease

Although HIV can occur in any race or ethnic group, the highest incidence is reported in the African continent. Anyone, regardless of their ethnic background, who present with unusual or difficult to treat skin problems should always be considered for HIV testing (Fig. 48.4).

HIV infection is associated with a wide range of skin disorders and opportunistic infection as a result of immunosuppression. The treatment of HIV with anti-retroviral drugs can also result in skin side effects in some patients. Mucocutaneous disease is extremely common in people with HIV. They may be the first manifestation of asymptomatic HIV infection. Immunosuppression often results in atypical disease presentation [4]. Recurrent herpes zoster, resistant viral warts, and Kaposi sarcoma are all examples of how cutaneous HIV may present. Widespread molluscum contagiosum, chronic herpes simplex, resistant seborrhoeic dermatitis, psoriasis, puritic papular eruptions and dry itchy skin can also be presenting signs of HIV. Psoriasis, which is uncommon in Africans, occurs more commonly in the presence of HIV infection.

## 48.5 Dermatosis Papulosa Nigra

This is commonly found in patients with skin type 5 and 6. Some people consider it a variant of seborrhoeic keratosis on the face in dark skin. It presents as 1–5 mm asymptomatic pigmented papules distributed symmetrically across the cheeks, forehead and less often on the neck chest and back (Fig. 48.3). Papules start to appear in early adult life and increase in size and number over many years. Differential diagnosis includes seborrhoeic keratosis, naevi, lentigo, warts and other adnexial tumours such as tuberous sclerosis. Treatment is only for cosmetic reasons and options include shave biopsy, cauterity, cryosurgery and laser destruction. Great care needs to be taken when doing these treatments to avoid hypo or hyperpigmentation post-surgery in pigmented



**Fig. 48.4** Difficult to treat tinea pedis in a HIV positive patient

With advanced HIV infection, skin diseases are often atypical, widespread and have more chronic courses. They also tend to be more resistant to treatment. Bacterial, viral, fungal and parasitic infections are all the more common in patients with HIV.

**Syphilis** can co-exist with HIV and there has been a resurgence of syphilis over the past number of years. HIV may alter the typical picture and presentation of syphilis. The primary chancre can be painful, multiple and extragenital. The rash of secondary syphilis may develop quicker in HIV and the cutaneous lesions tend to be more polymorphic. There is a greater risk of neurological involvement and progression to third degree syphilis in HIV. In darkly pigmented skin the rash of secondary syphilis may manifest as a maculopapular pigmented rash rather than a erythematous, psoriasiform rash. Lesions on the palm do not have the coppery hue seen in white skin but instead they are usually pigmented, scaly macules or papules. TB is also more common in HIV infected individuals from sub-Saharan Africa. Most cases of TB with HIV are pulmonary but it may occasionally present with cutaneous signs such as a chronic sinus over an involved lymph-node (scrofula) or a chronic ulcerating lesion.

The prevalence of the human papilloma (wart) virus (HPV) is higher in HIV immunosuppressed patients and this can increase their risk of non-melanoma skin cancer, particularly in the ano-

genital area. Kaposi sarcoma was the first recognized cutaneous marker for HIV infection. Fortunately, with modern anti-viral treatments both the incidence and mortality from Kaposi sarcoma has declined. The lesions of KS usually begin as macules and progress to papules and nodules. The violaceous hue of KS is less apparent in dark coloured skin and they may appear pigmented rather than vascular. As KS is derived from the lymphatic endothelium, there is often associated lymphedema. The incidence of melanoma and non-melanoma skin cancer is far less common in dark skin but is increased in dark skin associated with HIV.

## 48.7 Hair and Scalp Problems in Individuals with Type 6 Skin

There are distinct biological and structural differences in Skin type VI (Afro) textured hair compared to Skin Type 1 (Caucasian) hair. As hair gets curlier, it becomes more difficult to comb and therefore susceptible to breakage and traction. Skin type VI textured hair also grows more slowly [5].

Tight braiding can lead to problems such as **traction alopecia**.

“Hot combing” and chemical relaxers which are used to temporarily straighten Afro textured hair can also lead to damaged hair and possibly irritant or contact allergic dermatitis of the scalp.

Ingrown hairs especially in the beard area are very common in dark skin types because of the shape of the hair. This can cause a foreign body type reaction from ingrown hairs known as **pseudofolliculitis barbae** (shaving rash or razor bumps). This is most commonly seen on the side of the neck below the jaw line. It can be difficult to treat and best results occur by either growing a beard or lasering off the beard which can be difficult in dark skin. Other options include picking out the ingrown hairs with a needle and topical or oral antibiotics such as anti-acne medications which can be given for a few months.

Women from the Indian subcontinent have a lot more dark facial and body hair than Caucasians. Laser and IPL hair removal is more difficult to carry out in dark coloured skin but the 1064 Nd: YAG laser is probably the most useful for dark skin types.

## 48.8 Tinea Infections

Tinea infections are more common in dark skin types. Any adult or child of African descent with an unusual scaly rash on the body or scalp should have skin scrapings and/or plucked hairs sent for fungal stain and culture to rule out an underlying fungal infection. Although animal sources such as tinea canis (from cats or dogs) or tinea verrucosus (from cattle) are the most common causes of ringworm in Ireland, unusual organisms such as *Trichophyton tonsurans* (person-to-person spread) and *Trichophyton violaceum* are often found in Africans.

Tinea capitis is most commonly found in children. Treatment is usually with oral antifungals such as griseofulvin, terbinafine, itraconazole or fluconazole for 4 to 6 weeks (see Chap. 31). Ketoconazole shampoo may help reduce spread of *Trichophyton tonsurans* and may help clear asymptomatic carriers within a household.

## 48.9 Keloids and Hypertrophic Scars

Keloids are benign dermal fibro-proliferative growths that often follow minor deep dermal injuries such as piercings, infections, acne or burns. They are of unknown aetiology. Unlike hypertrophic scars, keloids spread beyond the margins of the original wound site and do not regress spontaneously (Fig. 48.5). Keloids and hypertrophic scars can be itchy or sore but the biggest problem they create is their unsightly appearance. Keloids are unique to humans and are far more common in individuals of darker skin types. There is no cure for keloids and hypertrophic scars, but treatment may be able to improve the signs and symptoms associated with them. People who are prone to keloids and hypertrophic scars should avoid pier-



**Fig. 48.5** Keloid in a 45 year old African man

ings and skin surgery if possible. There is no single universal treatment that always works. Often combination treatments have to be tried and the best that can be offered is to reduce the size and symptoms of the scars but treatment will never eliminate the scar completely. There is a lack of robust randomised evidence based controlled clinical trials evaluating the outcome of various treatments with keloids and hypertrophic scars [6]. The best results are obtained if the keloid or hypertrophic scar is treated early (less than 2 years) in their life span if possible. Hypertrophic scars usually respond better than keloids to treatment. In fact, many improve and even disappear with time. Children of any skin type can get an hypertrophic scar and parents need to be reassured that no treatment is required. Treatment options include intralesional steroids and/or intra-

lesional cryosurgery, and radiotherapy in severe cases [7, 8]. Post treatment hypo or hyperpigmentation may occur.

Surgical excision alone should be avoided as the keloids will invariably return and may grow even larger than the original keloid. Surgery followed by adjuvant treatments such as steroid tape, intralesional steroid, cryosurgery, radiotherapy, imiquimod, silicon sheets or silicon gel may help prevent recurrence. Some anatomical areas (earlobes) respond better to treatment than others (chest and back).

## 48.10 Folliculitis Keloidalis Nuchae

This condition is most commonly found in the back of the neck and posterior scalp in dark skin type men. It causes papules and pustules with multiple tiny keloids and hair thinning (see Chap. 36, Fig. 36.17). There is sometimes secondary infection. Causes are unknown. Treatment is difficult and often unsuccessful. In early cases with fresh lesions potent topical steroids may help

reduce the inflammatory response and prevent keloids. Intralesional steroids may help in more advanced disease. Long courses of oral antibiotics (e.g. clindamycin) or anti-acne treatments (e.g. lymecycline) for 6 to 12 weeks or longer may help if there are a lot of pustules. Laser hair removal may help in early disease. Surgical debridement may help in more advanced disease. Oral isotretinoin can give good results.

## 48.11 Tropical Diseases in the Ethnic Population

Individuals with Afro-Caribbean, Asian or Indian skin types are more at risk from tropical skin diseases as they may have been born in the tropics, they may have visited family in the tropics or they may have close personal contacts with relatives or friends recently returned from the tropics. Common conditions seen in this group include cutaneous larva migrans (creeping eruption), onchocerciasis, tungiasis, cutaneous leishmaniasis, tinea infection, leprosy, TB and tropical ulcers (Table 48.2).

**Table 48.2** Tropical Diseases of the Skin

	Aetiology	Clinical features	Diagnosis	Treatment
Cutaneous larva migrans ("Creeping eruption")	Dog or cat hookworm	Itchy serpiginous slowly advancing lesion—usually feet or buttocks	Clinical	Resolves spontaneously. Potent topical steroids and antihistamines to reduce itch if severe. Topical thiabendazole, oral ivermectin or cryosurgery
Onchocerciasis ("River blindness")	Worm from the bite of a blackfly	Localised or generalised pruritic papular dermatitis and eye problems	Biopsy	Oral ivermectin
Tungiasis ("jiggers")	A burrowing flea	Burrows into feet = pale nodule with central dark dot	Clinical or remove the flea	Remove flea or topical ivermectin or cryosurgery
Cutaneous leishmaniasis	Parasite from bite of a sandfly	Chronic painless ulcer with heaped up margins on exposed surfaces	Biopsy	Can resolve spontaneously or anti-parasitic pentavalent antimonials or liposomal amphotericin B
Leprosy	Mycobacterium leprae	Hypo-pigmented numb plaques	Biopsy	Dapsone, Rifampicin or Clofazimine—long course
Tuberculosis	Mycobacterium tuberculosis	Chancre. Red brown plaques ( <i>lupus vulgaris</i> ). Sinus track over node	Biopsy Culture Mantoux	Triple or quadruple anti TB treatment for nine to twelve months
Tropical ulcer	Mixed infection	Painful circular ulcer with purple edges—usually legs	Swabs Biopsy	Treat underlying cause Ulcer dressings

## 48.12 Conclusion

People with dark coloured skin types (type 5 and 6) can develop a different profile of common skin diseases compared with light skin types. Some skin conditions can be more difficult to identify on darker coloured skin. Patients with darker skin types are more prone to chronic skin colour changes (hypo or hyper-pigmentation) as a result of their underlying skin disease or their treatment.

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## **Part XI**

### **Disorders Due to Physical Agents, Systemic Conditions and the Mind**



# Photobiology and the Skin

49

David Buckley

## Key Points

- Some skin conditions are helped by ultraviolet light (UVL) while others are aggravated by UVL.
- For many women and some men, the fear of developing wrinkles is more of a deterrent than the risk of developing skin cancer when giving health advice regarding excessive UVL.
- People who are particularly careful to protect their skin from UVL should consider taking extra vitamin D as a supplement every day, particularly in the winter and especially for people who are over the age of 50.

## What to Tell the Patient

- Repeated or severe sunburn, even in early life, is one of the major identifiable risk factors for skin cancer, especially melanoma.
- Low grade chronic exposure to UVL over months and years as a result of outdoor work, hobbies or sunbeds can also predispose to skin cancer and is the major cause of premature aging of the skin causing wrinkles and sagging of the skin.
- People who are particularly careful to protect their skin from UVL should consider taking extra vitamin D as a supplement every day, particularly in the winter and especially for

people who are over the age of 50 with low levels of Vitamin D on blood analysis.

- Clouds only block out about 30% of damaging ultraviolet rays.

## 49.1 Introduction

Everybody loves a sunny day! It can lift our spirits and a tan can make our skin look better and our teeth look whiter. The sun shining on our skin is also our primary source of vitamin D. However, too much ultraviolet light (UVL) from sunlight or artificial light from sunbeds can harm our skin. The most obvious and immediate harmful effect is sunburn. Sudden bursts of sunshine on the skin that is not accustomed to it (e.g. on a sun holiday or starting a session on the sunbed) can cause sunburn. As much as 70% of UVL can penetrate thin clouds, so you can sunburn even on a cloudy day. This is sometimes incorrectly referred to as “windburn”. 10% of UV rays are reflected from sand or grass, 20% from the sea and 80% from snow. The World Health Organization has identified UVL as a proven human carcinogen. Repeated or severe sunburn, even in early life, is one of the major identifiable risk factors for skin cancer, especially melanoma. Low grade chronic exposure to ultraviolet light over months and years as a result of regular tanning, outdoor occu-

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pation, outdoor hobbies or living in a hot climate for a few years can also predispose to skin cancer, especially the non-melanoma skin cancers.

Another common effect of too much UVL over the years is premature ageing of the skin and wrinkles. This is known as solar elastosis and can lead to thinning and sagging of the skin, especially on the exposed area such as the face, neck and the back of the hands. Most dermatologists agree that the best prevention against wrinkles and premature aging of the skin is to be “sun smart” (see Chap 66: “How to be Sun Smart”). Since sun damage is cumulative, it is never too late to start being sun smart! Sun blocks with a high SPF (sun protective factor 30 or greater) will help but have to be applied generously and be reapplied frequently especially if sweating or swimming. SPF is a relative measure of how long a sunscreen will protect a person from UVB rays. Assuming it is used correctly, if a person burns after 20 min in the sun, an SPF 30 sunscreen protects for about 10 h ( $20 \text{ min} \times 30 = 600 \text{ min} = 10 \text{ h}$ ). To get the most protection, use a “broad spectrum sunscreen”—these protect against UVB **and** UVA rays. However, no sunscreen blocks 100% of UVB rays, and ultrahigh SPFs are not much more protective than SPFs of 30. SPF 15 blocks 93% of UVB rays. SPF 30 blocks 97%. The increase in protection is even more gradual after that, 98% for SPF 50. So a SPF of 50 does not give double the protection of an SPF 25. High-SPF chemical sun blocks require higher concentrations of sun-filtering chemicals than low-SPF sunscreens. Some of these ingredients may pose health risks when they penetrate the skin, and have been linked to tissue damage and potential hormone disruption. SPF higher than 50 may be inherently misleading and sunscreen products are capped at SPF 50 in Japan, and “50+” in Europe, Canada and Australia. When used correctly, sunscreen with SPF values in the range of 30 to 50 will offer adequate sunburn protection, even for people most sensitive to sunburn.

There are basically two types of sunblocks—chemical and physical. See Table 49.1 for the difference between the two.

**Table 49.1** Sun Protective Factors (SPF)

	Physical	Chemical
Mode of action	Reflecting or scattering UVR	Absorbs UVR
Contents	Contains minerals (titanium dioxide and/or zinc oxide)	Various chemicals
Stability	Photostable	May be unstable in UVR
Cosmetic acceptability	May be pasty and thick unless formulated as an ultrafine grade	Translucent and thinner
Environmental impact	Chemically inert	May be harmful to marine environment
Allergies	Low allergy potential	Increased allergy potential
Effect on eyes	No stinging	May irritate
Onset of action after applying	Immediate	10-20 min
Effect on rosacea and telangiectasia	Cools the skin by deflecting the heat from the sun	May heat up the skin
Effects on acne prone skin	Non comedogenic	May cause comedones (blackheads)
Shelf life	Long	Short
Penetration	Sits on the skin	Absorbed by the skin
How much protection	Broad spectrum UVA and UVB protection	Varies

Seeking suitable shade from broad brimmed hats, clothing and umbrellas is equally important as an SPF, especially in the middle of the day, even in cloudy days in the summer. Polarised sunglasses should also be encouraged and large ones will not only protect the eyes, but also the skin around the eyes. People who are particularly careful to protect their skin from ultraviolet light should consider taking extra vitamin D as a supplement every day, particularly in the winter and especially for people who are over the age of 50 if blood testing shows them to be Vitamin D deficient.

Options for treatment of photoaged skin include topical retinoids (tretinoin, tazarotene or adapalene), vitamin C and possibly vitamin E.

Too little sun and ultraviolet light can also cause problems. For example, the Asian community who moved to the United Kingdom in the late 50's and early 60's often suffered from osteomalacia and rickets as a result of lack of vitamin D from not getting enough sunlight on their skin. Too little sunlight may also affect some people's mood and may even lead to depression in the winter (seasonal affective disorder).

## 49.2 Skin Conditions that Are Usually Helped by UVL

Certain common skin conditions such as acne, eczema and psoriasis are usually helped by ultraviolet light. Most acne sufferers notice an improvement of their skin in the summer. One exception is "tropical acne". This is where someone develops acne or their existing acne gets worse when they are in a hot climate. This is usually as a result of excessive humidity and the use of too many oily products on the skin such as oily sun protection factors (SPF) and oily after-sun lotions.

Ninety per cent of people with psoriasis and eczema improve with ultraviolet light. In fact, some patients with these conditions and other conditions such as chronic urticaria, cutaneous T-cell lymphoma (mycosis fungoides) and vitiligo are sometimes treated with artificial light therapy such as narrow band UVB or PUVA lamps (Table 49.2). Other light based therapies including lasers, intense pulse light and photodynamic therapy can be used to treat a variety of skin conditions.

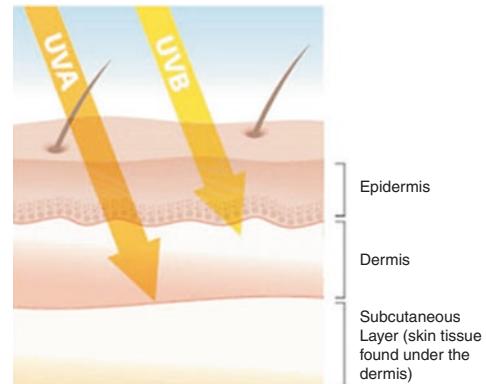
## 49.3 Skin Conditions that May Be Aggravated by UVL

Many skin conditions can be aggravated by ultraviolet light from the sun or artificial sources (Table 49.3). **Rosacea** and **telangiectasia** (broken veins on the face) usually occur in fair skinned people (Type 1 and type 2 skin—Table 49.4) who

**Table 49.2** Types of UVL

	UVA	UVB
Wavelength	Long wave	Short wave
Penetration	Deep	Superficial
Effects on the skin	Aging + tanning	Burning
Can cause skin cancer	Yes	Yes
Therapeutic use	PUVA phototherapy + sunbeds	UVB phototherapy
Season	All year round	Summer
Penetrates glass	Yes	No
Exposure during the day	Constant	Varies

UV Radiation and the skin  
(Copyright: AlexeyKazakov@123RF.com)



**Table 49.3** Skin conditions that may be aggravated by UVL

- Rosacea
- Telangiectasia
- Herpes simplex
- Melasma/Cholasma
- Vitiligo
- Lupus Erythematosus
- Porphyria cutanea Tarda
- 10% of people with psoriasis
- 10% of people with eczema
- Actinic keratosis
- Melanoma
- Non-melanoma skin cancer

have been exposed to excessive sunlight as a result of their lifestyle, work or hobbies (Fig. 49.1). Being sun smart is important, both as part of the treatment and also to prevent relapse.

**Herpes simplex** (cold sores), which most commonly occur on the lips but can occur in any

**Table 49.4** Fitzpatrick's Skin Types

Type 1	Always burns, never tans (pale white skin)
Type 2	Usually burns, may tan (white skin)
Type 3	Usually tans, may burn (light brown skin)
Type 4	Always tans easily, burns minimally (Mediterranean type skin)
Type 5	Tans profusely, rarely burns (Indian type skin)
Type 6	Always dark brown or black skin, never burns (African type skin)

**Fig. 49.1** Telangiectasia in a 48 year old

part of the body, are often triggered by ultraviolet light, especially in the summer. Protecting the area affected by appropriate clothing and high SPF sunblocks will help prevent relapse.

**Melasma** (chloasma) can cause facial hyperpigmentation in women and usually occurs during and after pregnancy, or when a woman is using hormonal contraception. These women are very sensitive to ultraviolet light on the affected areas, which will result in a blotchy tan on the

face. They must be extremely careful to protect their face from ultraviolet light.

**Vitiligo**, on the other hand, is an autoimmune condition where there are patches of skin and/or hair with absolutely no pigmentation. These areas have no protection from the sun as they will never tan and will always burn. Chronic exposure over many years may lead to skin cancer. If patients with vitiligo get sun on their skin it usually only makes the vitiligo more obvious as the non-affected areas will tan and the vitiligo areas will remain white, thus making the condition more conspicuous.

**Lupus Erythematosus** (DLE, sub-acute lupus and SLE) is a rare condition that causes multiple red, scaly plaques that can occur on any part of the body but it is usually worse on the exposed areas of the body (scalp, face, ears, neck and hands) as the rash is aggravated by UVL (Fig. 49.2). Although each type of lupus has its own skin manifestations, they all are photosensitive conditions. The plaques of lupus can leave permanent scars if not treated aggressively with very potent topical steroids. Patients with lupus have to be sun smart both to control the condition and prevent relapse.

**Porphyria Cutanea Tarda (PCT)** is a rare condition that usually presents as a photosensitive rash on the dorsum of the hands, the forearms or the face. It is caused by increased levels of porphyrins in the skin. The patient usually has fragile skin in these exposed areas that can tear easily with minor trauma. They may also develop blisters and scars. This condition is caused by a defective enzyme in the liver known as uroporphyrinogen decarboxylase. This can

**Fig. 49.2** Discoid lupus erythematosus

occur as a result of alcoholic liver disease, iron overload such as haemochromatosis, viral hepatitis or as a reaction to oestrogen containing medications. The condition is diagnosed by a skin biopsy and a 24-hour urine collection for porphyrin profile. Treatment will depend on the underlying cause but the patient will have to protect their skin from ultraviolet light in the wavelength at 400 nm, which is unfortunately not blocked by most ordinary SPF, except those containing dihydroxyacetone, which is capable of blocking this wavelength.(see Chap. 51: Skin and systemic diseases).

#### 49.4 Primary Photodermatoses

There are many skin conditions caused as a direct result of being exposed to ultraviolet light. These usually present as a rash that appears only on the exposed sights such as the face, the “V” of the neck, forearms and the back of the hands. In general practice, probably the most common cause of a photosensitive rash is from cosmetics, toiletries or drugs. In these patients, the topical or systematic agent needs ultraviolet light before an allergic or toxic reaction occurs. Unfortunately, some sunscreens can ironically cause photosensitivity. Identifying the correct suspect can be difficult and may require photopatch testing which can only be carried out in tertiary referral centres with a photobiology department.

#### 49.5 Drug Induced Photosensitivity

Many drugs can cause photosensitivity so a careful drug history has to be taken. Sometimes it may be obvious as the rash may commence immediately after starting the new drug. Other times it can be very difficult as the patient may be on many different drugs which could cause photosensitivity or there may be a delay of weeks or months between starting the drug and developing photosensitivity. (Table 49.5). The reaction can be photoallergic or phototoxic and the rash can take many different forms usually on

**Table 49.5** Common drugs that can cause photosensitivity

*Antibiotics*

- quinolones [for example, ciprofloxacin or levofloxacin, *nalidixic acid*]
- tetracyclines [for example, *tetracycline or doxycycline*]
- sulfonamides [for example, sulfamethoxazole and trimethoprim]

*Antifungal*

- terbinafine, itraconazole, *voriconazole*

*Antihistamines*

- diphenhydramine

*Malaria medications*

- quinine
- chloroquine
- hydroxychloroquine

*Cancer chemotherapy drugs*

- 5-fluorouracil (5-FU, “Efudex®”)

- vinblastine

- dacarbazine

*Cardiac drugs*

- *amiodarone (“Cordarone®”)*

- calcium channel blockers (nifedipine, diltiazem)

- ACE inhibitors

- quinidine

*Diuretics*

- furosemide (“Lasix®”), bumetanide

- *thiazides [hydrochlorothiazide]*

*Diabetic drugs*

- sulfonylureas [chlorpropamide, glyburide]

*Painkillers*

- Nonsteroidal antiinflammatory drugs [*naproxen*], *piroxicam (“Feldene®”)*

*Acne medications*

- isotretinoin (“*Roaccutane®*”)

- acitretin

- adapalene (“*Differin Gel®*”)

*Psychiatric drugs*

- *phenothiazines [chlorpromazine, thioridazine]*

- tricyclic antidepressants [desipramine, imipramine]

*Oral Contraceptive pills*

(Bold-italic indicates the most common drugs to cause photosensitivity)

the exposed skin (eczematous, erythema, lichenoid, erythema multiforme, hyperpigmentation, etc.,) (Fig. 49.3). In phototoxic reactions, the drug becomes activated by exposure to sunlight and cause an acute sunburn type reaction. A phototoxic reaction typically clears up once the drug is stopped.



**Fig. 49.3** Photodermatitis, possibly drug-related

With photoallergic reactions, the ultraviolet exposure changes the structure of the drug (systemic or topical) so that it is seen by the body's immune system as an antigen. The immune system initiates an allergic response and causes a chronic eczematous reaction in the sun-exposed area. The photoallergic reaction may recur after sun exposure even after the drug has cleared from the system.

## 49.6 Phytophotodermatitis

Some plants such as giant hogweed, chrysanthemums, poison ivy, cow parsley, celery and bergamotta essence from citric fruit can cause a phototoxic reaction when the sap, which contains potent furocoumarin derivatives, gets on the skin and it is exposed to ultraviolet light. This can cause blisters and brown streaks that are often linear where the plant has rubbed against the skin. It can appear 2-3 days after exposure and it is recognized by the bizarre shape of the lesions and their presence in sun exposed areas. **Strimmer dermatitis** is a similar condition where the sap from the plants is spread onto the skin of the person using the strimmer to cut grass and weeds. This, combined with ultraviolet light, causes a phototoxic dermatitis with characteristic

linear streaks on the exposed area such as the face, arms and legs if appropriate clothing and masks have not protected them. It usually responds to potent topical steroids and can be prevented by the appropriate protection measurements. In addition, sun protection should be used while the skin is inflamed to reduce further hyperpigmentation.

## 49.7 Polymorphic Light Eruption

Polymorphic light eruption is a common condition that can affect up to 10% of the population. It is most common in adult women between the ages of 20 and 40. The usual history is that the patient develops an itchy, eczematous, urticarial, polymorphic (= many forms) rash, sometimes known as "prickly heat", that begins at the start of the summer, the start of a summer holiday or at the beginning of a course of sunbed sessions. The rash usually occurs on the areas that have not been exposed to ultraviolet light for some time (e.g. upper arms and the trunk) (Fig. 49.4). The face and hands are often spared, as these have been chronically exposed all year round. The rash is usually worse at the start of the summer and eases as the summer progresses, as the skin becomes more accustomed to ultraviolet light. Treatment is with potent topical steroids or occasionally systemic steroids (e.g. 25 mg predniso-



**Fig. 49.4** Polymorphic light eruption in a 37-year-old female on the upper back

lone daily  $\times$  5 days). Being sun smart will prevent further attacks.

## 49.8 Juvenile Spring Eruption

Juvenile spring eruption is probably a localised form of polymorphic light eruption that occurs on the outer rim of the ears, mostly in young boys. It causes an itchy, scaly, eczematous rash on the ears that starts in the spring and can continue throughout the summer unless the ears are protected by high SPF sunblocks, a broad rimmed hat or letting the hair grow over the ears. The rash usually responds to topical steroids and will usually resolve as the child gets older.

## 49.9 Chronic Actinic Dermatitis

Chronic actinic dermatitis is a rare photodermatosis that mostly occurs in older men. It can cause a severe, itching, eczematous rash after being exposed to sunlight or artificial light. It can even be provoked by the light from fluorescent bulbs and daylight through glass. The rash can develop within seconds of being exposed to ultraviolet light and the itch can be very severe. It may be unresponsive to very potent topical steroids and some patients need oral steroids or other systemic treatment such as azathioprine or cyclosporin. Patients need to take extreme precaution to protect their skin from ultraviolet light. They may also have to get special filters for the glass in their home, office and car. Phototesting and photo-patch testing in a specialist photobiology unit may be required to confirm the diagnosis and to establish if there are any provoking factors such as fragrances, sunscreens, plants or drugs.

## 49.10 Solar Urticaria

Solar urticaria is a rare form of physical urticaria where the patient develops an urticarial type rash within minutes of being exposed to ultraviolet light. Some of these patients may have polymorphic light eruption. In solar urticaria the rash usu-

ally resolves quickly without leaving a trace. Most patients respond to the newer non-sedating oral antihistamines. These patients need to be sun smart to prevent relapse.

## 49.11 Actinic Prurigo

Actinic prurigo (Hutchinson prurigo) is a rare intensely itchy papular eruption that may occur all year round but is usually more severe in the summer months and on the exposed parts of the body. It is more common in people of Latin-American and American-Indian decent. The patient may present with a severe eczematous rash mainly on the exposed areas but it can occur on areas covered by clothing (e.g. the buttocks) in long-standing cases. Lip and eyes are commonly affected. Most cases respond by being sun smart, using emollients and potent topical steroids. Severe cases may need systemic treatment such as hydroxychloroquine or thalidomide.

## 49.12 Hydroa Vacciniforme

Hydroa vacciniforme is a very rare blistering photodermatosis that occurs on sun exposed areas in children.

## 49.13 Xeroderma Pigmentosum

Xeroderma pigmentosum is a very rare autosomal recessive condition that causes severe scarring photodermatitis, premature ageing of the skin and can lead to multiple skin cancers at a young age. Twenty per cent of these children may have neurological problems.

## 49.14 "Fake Tan"

Fake tans are considerably safer than getting a tan from ultraviolet light, such as the sun or sun beds but they can cause a streaky, brown or orange colour particularly as they fade.

Some people worry that the chemicals in fake tans may be harmful to humans. Generally, the ingredients are safe in humans when applied to the skin. Inhaling some of these ingredients may be more harmful.

Fake tans do not give any protection against ultraviolet light, so people using fake tan should also use sun blocks. Fake tans may also be more difficult to put on evenly if there is an underlying skin problem such as eczema or psoriasis. The chemicals in fake tans may be more harmful to those with eczema as their skin barrier may be compromised and they are more likely to develop allergic reactions to some of the ingredients in fake tans.

Dihydroxyacetone (DHA) is one of the most potentially dangerous ingredients contained in many fake tans. This appears to be safe on the skin but is dangerous if inhaled or if it gets into the eyes. Therefore, fake tans applied by creams or lotions are safer than spray-on fake tans if they contain DHZ, as the spray may be inhaled or get into the eyes of the user or the beautician.

Some fake tans do not contain DHA such as “He-Shi One Day Bronzer®” ([www.heshi.eu](http://www.heshi.eu)), “Melvita Prosun Gradual Self Tanning Moisturizing Gel®” ([www.melvita.co.uk](http://www.melvita.co.uk)) and

“BeautyLab Peptide Tanning Lotion®” ([www.beautylabshop.com](http://www.beautylabshop.com)).

Organic fake tans may not be inherently safer than any other fake tan. The word “organic” is often promoted in the cosmetic industry as being something safe and “natural”. However, when it comes to fake tans the word “organic” is usually used more as a marketing ploy to try and make people believe that the product is in some way safer than other fake tans, which it probably is not.

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## 49.15 Conclusion

Sunlight is essential for life. It also helps many skin conditions. However, too much sunlight or artificial light on the skin can lead to premature ageing (wrinkles) and UV damage such as actinic keratoses and possible skin cancers. For many, the fear of developing wrinkles is more of a deterrent than the risk of developing skin cancer when giving health advice regarding excessive UVL.

Too little UVL on the skin can result in deficiency in vitamin D. Patients who need to stay out of the sun for health or cosmetic reasons should have their vitamin D levels measured and supplemented if necessary.



# Pruritus (Itch)

50

David Buckley

## Key Points

- Itch is one of the most common presenting symptoms in dermatology. It may be due to an underlying skin or general medical condition or as a side effect of medication. In some situations no obvious cause can be found.
- Many patients can develop secondary psychiatric problems such as anxiety, insomnia or depression as a result of chronic itch. Conversely, a primary psychiatric disorder may present with pruritus because of an illusion, delusion or phobia.
- While every effort should be made to identify and treat the underlying cause, effective anti-pruritus measures may be required while awaiting the results of investigations, a response to treatment or if no cause can be found for the itch.
- Emollients and soap substitutes are important when treating chronic itch in the elderly as dry skin is common in this age group.

## What to Tell the Patient

- Chronic itch can be very distressing and can be as difficult to live with as chronic pain.

- Anti histamines and other anti-itch tablets can cause drowsiness and may impair driving, especially if taken with alcohol or other sedatives or even by themselves.
- Prescribed or over the counter medications are the most common cause of itch in the elderly. Almost every drug on the market has the potential to cause an itch but some are more likely to cause itch than others.

## 50.1 Introduction

Pruritus and itch are synonymous but when used as a diagnostic term, generalised pruritus usually signifies that itching is the primary and presenting complaint without any obvious or visible underlying skin disorder. However, the skin often shows secondary changes as a result of rubbing and scratching and this can lead to excoriation, lichenification and possibly skin infection. Once an “itch-scratch-more-itch” cycle becomes established it can be self perpetuating and difficult to break and may lead to secondary disorders such as neurodermatitis (lichen simplex chronicus), prurigo nodularis (nodular prurigo), pigmentary changes and lichenification.

Pruritus is the most common symptom in dermatology. Chronic pruritus (itch >6 weeks) can be very distressing and can be as difficult to live with as chronic pain. Many patients can develop secondary psychiatric problems such as anxiety,

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insomnia or depression. Conversely, a primary psychiatric disorder may present with pruritus because of an illusion, delusion or phobia.

## 50.2 Aetiology

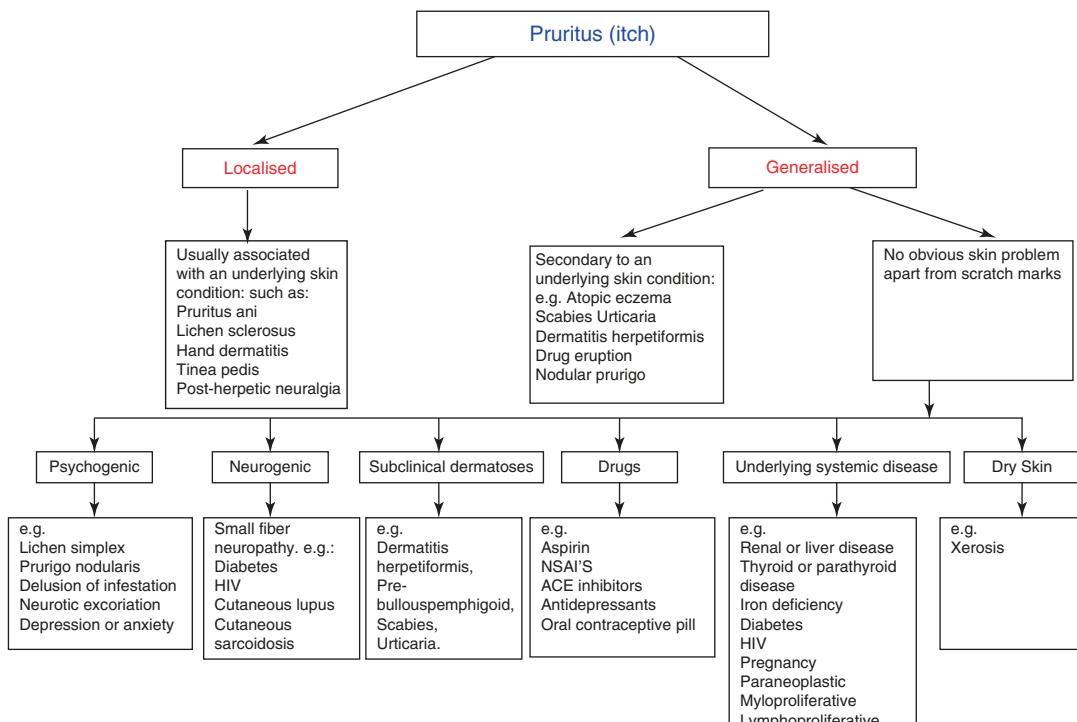
Pruritus can be classified as localised or generalised (Fig. 50.1 and Table 50.1). Most localised itch is due to a localised skin condition such as hand eczema, pruritus ani, post-herpetic neuralgia or lichen sclerosus. Generalised itch can be divided into patients who have an obvious underlying skin condition (eg. Atopic eczema, urticaria, scabies, lichen planus, dermatitis herpetiformis, nodular prurigo) and those patients where there is no obvious skin condition apart from scratch marks (generalised pruritus) (Fig. 50.2).

Generalised itch in a child is most commonly due to atopic eczema, in a young adult is most likely due to scabies or urticaria and in the elderly is most likely due to medication or dry skin (xerosis).

Pruritus (itch) (Table 50.2). In women of childbearing age, generalised pruritus may be associated with pregnancy or the oral contraceptive pill (see Chap. 25). However, there can be many other causes of generalised pruritus and there can be considerable overlap of the causes between the different age groups.

Many systemic diseases may present with generalised pruritus. These include thyroid disease (hypo/hyper), liver disease, renal disease, diabetes, iron deficiency anaemia, HIV, internal malignancy and lymphomata.

Occasionally, an underlying itch dermatosis may be present but difficult to diagnose because the classical clinical signs are masked by scratching. For example, scabies burrows may be hard to find because of scratch marks or vesicles in dermatitis herpetiformis may be ruptured by scratching. Pre-bullous phase of bullous pemphigoid may present as an itch without any blisters and little or no rash. Urticaria causes a transient rash that may not be present at the time the patient visits the doctor.



**Fig. 50.1** Most common causes of Pruritus

**Table 50.1** Causes of generalised pruritus in the absence of obvious skin disease**Underlying systemic diseases:**

Uremic pruritus (in renal failure)

Hepatobiliary disease (e.g. Primary biliary cirrhosis, primary sclerosing cholangitis, drug induced cholestasis, chronic hepatitis C)

**Metabolic disease:**

Endocrine disorders (e.g.: hyperthyroidism, hyperparathyroidism, diabetes)

Malabsorption (e.g. coeliac disease, inflammatory bowel disease, iron deficiency anaemia)

Myeloproliferative disease (e.g. Polycythaemia vera)

Lymphoproliferative disease (e.g.: Non-Hodgkin lymphoma, Hodgkin lymphoma, Mycosis fungoides)

Solid tumours.

**Psychiatric causes:**

Delusional parasitosis,

Depression+ anxiety (cause of pruritus or effect?)

**Drug induced**

Many mechanisms of action (e.g. cholestasis, central acting, histamine release, etc)

**Neurogenic pruritus**

Small fiber neuropathy (e.g.: diabetes, HIV, cutaneous lupus, cutaneous sarcoidosis)

**Subclinical skin disease:**

(e.g: Dermatitis herpetiformis, scabies, pre-bullous phase of bullous pemphigoid, urticaria)

**Dry skin:** (e.g. in the elderly—Senile prurigo)

Blood tests and a skin biopsy may be helpful in difficult cases (see Table 50.3). A careful drug history should always be taken as certain prescribed or over the counter medications may cause generalised pruritus (e.g. antidepressants, cimetidine, chloroquine, etc). However, it can be difficult to identify which if any drug may be responsible for the itch, especially in the elderly with multiple comorbidity and polypharmacy. If the itch started around the time of starting a new drug, if the itch clears on stopping the drug and if it recurs on restarting the drug, then this can be taken as good evidence of that particular drug being responsible. However, the itch may not start immediately after starting a new drug and it can sometimes take weeks or months for the itch to clear after stopping an offending drug. Certain drugs that are more likely to cause itch should be stopped or substituted on a best guess basis (Table 50.4).

**Fig. 50.2** Generalised pruritis possibly from drugs. Excoriation but no rash in a 78 years old**Table 50.2** Common Cause of Generalised Pruritus According to Age

Children:	<ul style="list-style-type: none"> <li>– Atopic eczema</li> <li>– Urticaria</li> <li>– Childhood exanthems (e.g. chickenpox)</li> <li>– Scabies</li> <li>– Dermatitis herpetiformis</li> </ul>
Adults:	<ul style="list-style-type: none"> <li>– Scabies</li> <li>– Urticaria</li> <li>– Dermatitis herpetiformis</li> <li>– Folliculitis (e.g. gram-negative, malassezia, jacuzzi)</li> <li>– Pregnancy rashes</li> </ul>
Elderly:	<ul style="list-style-type: none"> <li>– Xerosis (dry skin)</li> <li>– Drugs</li> <li>– Contact dermatitis</li> <li>– Scabies</li> <li>– Dermatitis herpetiformis</li> <li>– Chronic disease (e.g. renal failure, diabetes, anaemia, hepatobiliary, endocrine, myeloproliferative)</li> <li>– Delusion of infestation</li> </ul>

**Table 50.3** Investigations for generalised pruritus

- Full Blood Count, Erythrocyte Sedimentation Rate, C Reactive Protein, serum ferritin.
- Fasting blood glucose + HbA1C.
- Liver Function tests
- Renal function and electrolytes.
- Calcium, phosphate, alkaline phosphatase.
- Coeliac antibodies
- Thyroid Function Tests
- HIV
- Pregnancy test (in women of child bearing age)
- Chest X-Ray
- Skin biopsy

Generalised pruritus without an obvious rash may be due to psychogenic causes such as anxiety (e.g. pickers nodules), or delusions of infestation. Metabolic causes such as renal, liver or thyroid disease, iron deficiency anaemia, diabetes, HIV or paraneoplastic syndromes may also cause itch without a rash. Xerosis (dry skin) which can occur in the elderly, especially those in hospitals or nursing homes where there is low humidity, can cause an itch with very little visible on examination apart from excoriations.

### 50.3 Management

Management of pruritus is usually by dealing with any underlying cause—moisturisers for dry skin, insecticides for scabies, steroids for lichen sclerosus. However an underlying cause may be difficult to find or treat and so we often have to manage the itch itself. Patients often require symptomatic relief of their itch while undergoing investigations to find an underlying cause.

Distraction may work temporarily especially in children. Behavioral modification with star charts and rewards for not scratching may also help children with chronic itch. Keeping the finger nails short and wearing cotton gloves or mittens at night may help as it will lessen damage to the skin which in turn may cause more itch. Bandaging the affected area or the use of wet wraps can be very helpful as they both cool the skin and act as a barrier against scratching. Rubbing or pinching the skin instead of scratch-

ing with the finger nails sometimes relieves the itch without damaging the skin. Keeping the skin cool by avoiding hot rooms, hot baths and hot beds may help relieve the itch. Ice packs may help but care should be taken not to cause an ice burn.

Dry skin tends to be itchy (e.g. atopic eczema) so constant washing with soap and shampoos may dry out the skin and produce pruritus. Skin gets drier with age and pruritus may develop (senile pruritus). Liberal moisturising with a safe greasy moisturiser such as emulsifying ointment or “Epiderm®” ointment should relieve itch due to these causes. Patients with dry sensitive skin should also avoid soaps and other irritants such as bubble bath, perfumes, etc. Sensitive skin may become itchy after contact with various irritants (e.g. wool or soap) or allergens such as pollen or dust. A careful history can help to identify these factors which, if eliminated, should help resolve the problem. Topical steroids or topical calcineurin inhibitors may help relieve the itch if there is any evidence of an underlying dermatosis.

Oatmeal compresses made with fresh water (even kept in the refrigerator) or hydrating creams also kept in a refrigerator and applied generously, can help in reducing itch.

### 50.4 Drug Therapy for Chronic Itch

If itch is severe enough to cause sleep, work or school disruption, it probably needs systemic treatment with anti-itch medications (Table 50.5). Topical antihistamines should not be used as they have a high potential to cause sensitisation. The new generation, **non-sedating, oral antihistamines** (e.g. fexofenadine (“Telfast®180 mg”), desloratadine (“Neoclarityn®”) levocetirizine (“Xyzal®”), cetirizine (“Zirteck®”)) will only work if there is a histamine mediated reaction (urticaria or atopic eczema). Some patients with urticaria may need double or quadruple the standard dose of a non-sedating, newer generation antihistamine to give good results. This is considered safe, provided the patient does not have any underlying cardiovascular disease and are not on drugs that can interact with these antihistamines

**Table 50.4** Drugs that could induce pruritus. (Almost all drugs have the possibility to cause itch or rash. This is a list of the more common offenders)

Group of drugs	Examples	Possible mechanism of pruritus	Frequency of pruritus
Antihypertensive drugs	Angiotensin-converting enzyme inhibitors	Increase of bradykinin level or cholestatic liver injury or secondary to skin lesions	1–15%
	Angiotensin II antagonists (sartans)	Cholestatic liver injury	Case reports
	Beta-adrenergic blockers	Secondary to skin lesions Cholestatic liver injury	Frequent, if administered transdermally Rare
	Calcium channel blockers	Secondary to skin lesions or unknown Cholestatic liver injury	< 2% Case reports
	Methyldopa	Unknown or secondary to skin lesions	< 2%
	Sildenafil	Cholestatic liver injury	Case report
Anti-arrhythmic drugs	Amiodarone	Cholestatic liver injury	Case reports
Anticoagulants	Ticlopidine	Cholestatic liver injury	Case reports
	Fractionated heparins	Urticular reaction	Case reports
Anti-diabetic drugs	Biguanides	Cholestatic liver injury	Case reports
	Sulfonylurea derivatives	Unknown	< 5%
Hypolipidemic drugs	Statins	Unknown or secondary to skin lesions	16%
Antibiotics and chemotherapeutics	Penicillins	Secondary to skin lesions or cholestatic liver injury	2–20%
	Cephalosporins	Unknown or secondary to skin lesions	< 2%
	Macrolides	Secondary to skin lesions or cholestatic liver injury	< 0.3%
	Carbapenems	Cholestatic liver injury	Rare
	Monobactams	Secondary to skin lesions	Rare
	Quinolones	Unknown or secondary to skin lesions	1–4%
	Tetracyclines	Unknown or cholestatic liver injury	1–2%
	Lincosamides	Secondary to skin lesions or cholestatic liver injury	Rare
	Streptogramins	Secondary to skin lesions	2.5%
	Metronidazole	Unknown or secondary to skin lesions	< 5%
	Rifampin	Unknown	Case report
	Thiamphenicol	Unknown	< 0.1%
	Trimethoprim/sulphamethoxazole	Secondary to skin lesions Cholestatic liver injury	2–10% Rare
	Antimalarials	Unknown, but genetic background is important: Release of histamine or activation of $\mu$ -receptors were postulated	Up to 60–70% of black Africans, uncommon in Caucasians or Asians
Psychotropic drugs	Tricyclic antidepressants	Cholestatic liver injury	Rare
	Selective serotonin re-uptake inhibitors	Activation of peripheral serotonin receptors or secondary to skin lesions	Rare
	Neuroleptics	Cholestatic liver injury	Rare
Anti-epileptics	Carbamazepine, fosphenytoin, oxcarbazepine, phenytoin, topiramate	Secondary to skin lesions, allergic reaction	Rare
Cytostatics	Chlorambucil	Secondary to skin lesions	Case reports
	Paclitaxel	Unknown or secondary to skin lesions	10–14%
	Tamoxifen	Sebostasis/xerosis	3–5%

(continued)

**Table 50.4** (continued)

Cytokines, growth factors and monoclonal antibodies	Granulocyte-macrophage colony-stimulating factor	Unknown	Common
	Interleukin 2	Direct pruritogenic effect of IL-2	Very common
	Matuzumab	Unknown	< 10%
	Lapatinib	Unknown or urticarial reaction	3%
Plasma volume expanders	Hydroxyethyl starch (HES)	Deposition of HES in small peripheral nerves or in Schwann's cells of cutaneous nerves	12.6–54%
Others	Anti-thyroid agents	Cholestatic liver injury	Rare
	Non-steroidal anti-inflammatory drugs	Increased synthesis of leukotrienes Cholestatic liver injury	1–7% Rare
	Corticosteroids	Cholestatic liver injury	Very rare
	Sex hormones	Cholestatic liver injury	Rare
	Opioids	Centrally mediated process via $\mu$ -opioid receptor	2–100%
	Inhibitors of xanthine oxidase	Secondary to skin lesions	0.8–2.1%

Adam Reich<sup>1</sup>, Sonja Ständer<sup>2</sup> and Jacek C. Szepietowski. Drug-induced Pruritus: A Review. Acta Derm Venereol 2009; 89: 236–244.

**Table 50.5** Systemic treatment for pruritus

– Sedating antihistamines
– Non sedating antihistamines
– Amitriptyline
– Hydroxyzine
– Doxepin
– Gabapentin

such as tricyclic antidepressants, oral anti fungals, erythromycin and grapefruit juice. Fexofenadine (“Telfast® 180 mg”) and desloratidine (“Neoclarityn®”) are considered good for chronic idiopathic urticaria. Levocetirizine (“Xyzal®”) is considered to be one of the most potent non-sedating antihistamines. Cetirizine (“Zirteck®”) has a quick onset of action. Desloratidine (“Neclaritin®”) has a long duration of action. Some patients may have to try a few different non-sedating, newer generation antihistamines to see which one suit them best.

For non histamine related itchy skin problems, an older generation, **sedating antihistamine** (e.g. promethazine-“Phenergan®”chlorpheniramine-“Piriton®”) taken at night may be more suitable as they appear to work primarily by virtue of their sedative effect rather than their antihistamine effects which are weak compared to the newer generation of antihistamines. Patients should be warned not to drive or operate dangerous machin-

ery while on these medications if they cause daytime drowsiness and it is safer to write this warning on the prescription. They can interact with alcohol or sedatives causing prolonged drowsiness. This is why they are almost always best taken at night to relieve itch that may be keeping the patient awake, regardless of the cause. Taking a double dose at bedtime is sometimes necessary and seems to be quite safe apart from the risk of drowsiness the next morning. Promethazine (“Phenergan®”) is considered more sedating than chlorpheniramine® (“Piriton®”).

Old fashioned sedating antihistamines such as chlorpheniramine (“Piriton®”) or promethazine hydrochloride (“Phenergan®”) are considered safe in pregnancy but can cause drowsiness and so best used to relieve itch at night and help sleep. They are probably the safest antihistamine in the first trimester. Loratadine or cetirizine are considered safe in the second trimester. All antihistamines should be avoided in the third trimester if possible.

Other antipruritic agents includes **amitriptyline** which is a tricyclic antidepressant that is now most commonly used for chronic pain or chronic itch. It should given an hour or two before bedtime. The dose should be started low (e.g. 10 mg at night) and gradually increased every few days until the patient gets relief from the itch at night

and does not wake up with drowsiness the next morning. Patients should be warned not to drive or operate dangerous machinery if it causes daytime drowsiness. The final dose can be very variable and can range from 10 to 75 mg at night.

**Doxepin**, like amitriptyline, is a tricyclic anti-depressant that also has potent H<sub>1</sub>- and H<sub>2</sub>-blocking properties. It can be used to relieve itch associated with atopic dermatitis and other itchy skin conditions [1]. It acts by depressing cutaneous sensory receptors. The starting dose is 25–50 mg, taken orally at bedtime. Low-dose doxepin (10 mg three times a day) is a potentially effective and well-tolerated alternative in patients who do not respond to conventional antihistamines. Adverse effects (including sedation) and drug interactions may occur.

**Mirtazapine** (“Zispin®”) is a presynaptic α antagonist antidepressant with additional anti-histamine and serotonergic effects. It can be helpful in refractory urticaria and pruritis of unknown origin [2].

**Hydroxyzine** (“Ucerax®”) is an antihistamine with anticholinergic and sedative properties that is used to treat pruritis. Hydroxyzine has its maximal effect about 30 to 60 minutes after it is taken. Its effects last for 4 to 6 h. The recommended adult dose for treating itching (pruritis) is 25 mg given 3 or 4 times daily by mouth. It should be used with caution in the elderly and the maximum dose in this age is 50 mg/day.

Side effects include drowsiness, dizziness and dry mouth. It should be avoided in pregnancy and when breast feeding. Hydroxyzine should be used with caution (if at all) in persons with narrow-angle glaucoma, prostatic hypertrophy, hyperthyroidism, cardiovascular disease, hypertension, and asthma. It can prolong the QT inter-

val and is contraindicated in cardiovascular disease, bradycardia and when there is electrolyte imbalance.

**Gabapentin** (“Neurontin®”) is an anticonvulsant that is also used to treat neuropathic pain and can help with chronic itch, especially itch associated with renal disease, vulvodynia and post-herpetic neuralgia. The starting dose in adults is usually 300 mg TID and increased if necessary. The maximum daily dose is 3600 mg. Side effects include drowsiness, nausea, confusion, headaches, and dizziness.

**Ultraviolet therapy (UVB)** can sometimes help with chronic urticaria. Opiate receptor antagonists such as **naltrexone** (“Naxolone®”) may help with cholestatic pruritus.

## 50.5 Conclusion

In summary, chronic itch can cause severe impairment in a patient’s quality of life. While every effort should be made to identify and treat the underlying cause, effective anti-pruritis measure may be required while awaiting the results of investigations, a response to treatment or if no cause can be found for the itch (Table 50.5).

## References

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# Skin in Systemic Disease

51

David Buckley

## Key Points

- Systemic diseases can have associated skin manifestations which may precede the underlying disease and may be a clue to their diagnosis. Some systemic diseases are treated with drugs which may also cause skin problems.
- Treatment of some skin conditions may precipitate or aggravate underlying systemic diseases (e.g. high dose steroids may aggravate diabetes or methotrexate may cause liver disease).
- Dermatitis herpetiformis is strongly associated with coeliac disease in approximately 80% of cases, so all suspected cases should have blood tests for coeliac antibodies and a jejunal biopsy to look for total or subtotal villous atrophy.
- Urticaria and acne rosacea may occasionally be associated with Helicobacter pylori infestation in the stomach.
- Eating certain foods can cause rashes. One example is shiitake mushroom intake which can cause a distinctive rash (shiitake flagellate dermatitis).

## What to Tell the Patient

- Patients with rheumatoid arthritis have thin skin with easy bruising but this may be partially as a result of chronic systemic steroids.
- Poorly controlled systemic diseases such as diabetes or HIV can result in exacerbation of incidental skin problems such as skin infections or psoriasis and make them more difficult to control.

## 51.1 Introduction

The skin is often a mirror of what is going on underneath. Systemic diseases can have associated skin manifestations which may precede the underlying disease and may be a clue to their diagnosis. Some systemic diseases are treated with drugs which may also cause skin problems. In addition, underlying systemic diseases may alter or aggravate a coincidental skin disease such as acne, psoriasis or seborrhoeic dermatitis (e.g. HIV, Diabetes).

### Diabetes

See Chap. 52.

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## 51.2 Rheumatoid Arthritis (RA)

Patients with RA can develop various skin manifestation of their disease such as rheumatoid nodules over affected joints in 25% of cases. Patients with RA have thin skin with easy bruising but this may be partially as a result of chronic systemic steroids. Palmar erythema is also more common in RA patients. They are also more likely to develop a generalized itch known as neutrophilic dermatosis. Cutaneous vasculitis can occur in RA and may present as a purpuric rash.

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## 51.3 Palmar Erythema

This presents as redness of the skin on the palms, most prominent over the thenar and hypothenar eminence and sometimes the fingers. It can rarely occur on the soles of the feet (plantar erythema). It can occur naturally but may be associated with various underlying diseases (Table 51.1). It usually does not require treatment but should alert the doctor to possible underlying disease [1].

**Table 51.1** Possible causes of Palmar Erythema

Pregnancy (occurs in 30% of cases)
Liver cirrhosis (occurs in 23% of cases)
Rheumatoid arthritis (occurs in 60% of cases)
Systemic lupus erythematosus.
Thyrotoxicosis.
Diabetes mellitus.
Sarcoidosis.
Syphilis.
Neoplastic disease (as a paraneoplastic disorder), particularly primary or metastatic brain neoplasm and ovarian carcinoma.
Drug-induced (amiodarone, gemfibrozil, cholestyramine, topiramate and salbutamol have all been implicated).
Smoking.
Chronic mercury poisoning.
Polycythaemia
Human T-lymphotropic virus type 1 (HTLV-1)-associated myelopathy.

## 51.4 Cutaneous Signs of Anorexia Nervosa (AN)

Patients with AN often develop cutaneous signs either as a result of malnutrition, vitamin deficiency, vomiting, self inflicted injuries (dermatitis artefacta or trichotillomania) or as a consequence of the abuse of drugs such as diuretics or laxatives. These may help in the diagnosis of AN.

Skin signs include xerosis (dry skin), lanugo-like (baby like) body hair, telogen effluvium, carotenoderma, acne, hyperpigmentation, seborrhoeic dermatitis, acrocytosis, brittle nails, petechiae, livedo reticularis, paronychia, acquired striae distensae, and premature aging of the skin [2].

The most characteristic cutaneous sign of vomiting is Russell's sign (calluses on the knuckles from friction against the teeth as a result of repeatedly ramming the fingers down the throat to induce vomiting).

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## 51.5 Vitiligo

Vitiligo is an acquired autoimmune depigmenting disorder in which the melanocytes (pigment producing cells) disappear from certain areas of the skin. It is quite common affecting 0.5% of the population across all races [3].

Vitiligo is an autoimmune disease that can be linked with other autoimmune diseases such as thyroid disease, diabetes, pernicious anaemia or Addison's disease. Most cases of vitiligo are mild and isolated. It usually starts with sharply demarcated patches of white skin with no scale or itch. It is most commonly found on the hands and feet but occasionally can spread to other parts of the body including the face, groin, axillae, nipples, umbilical area and genitalia. The diagnosis is usually clinical and biopsies are not normally required. Treatment is only required for cosmetic reasons and is usually only necessary in people with dark skin. There is also a link between vitiligo and alopecia areata.

Treatment involves strict protection from ultra violet light and the patient should be advised to take extra vitamin D. Cosmetic camouflage can be extremely effective in improving the appearance of the rash. Potent or super potent topical steroids can be tried for up to 3 months on the body. Using them in a cyclical fashion (e.g. 2 weeks on and 2 weeks off per month) may help reduce local side effects such as skin thinning if they are to be used long term. Topical calcineurin inhibitors such as tacrolimus ointment can be tried on the face. UVB phototherapy may help stimulate repigmentation in some patients. Surgical treatments including melanocyte transfer grafting using various techniques such as split skin grafting or blister grafting is popular with the Asian population. Systemic treatment such as oral steroids, methotrexate or afamelanotide may be required in widespread, rapidly progressive treatment resistant cases (see Chap. 47).

## 51.6 Granuloma Annulare

This is a benign condition that usually presents as an annular rash that often affects the hands or feet in children and young adults. The dermal annular plaques which have a thickened, nodular, papular border, most commonly occur over joints, particularly the knuckles. The centre of each ring is often flat and relatively normal. The plaques may be solitary or multiple. The annular rash can be confused with tinea corporis (ringworm) or a BCC, but unlike these conditions, granuloma annulare is a deep dermal lesion with no scaling, bleeding or ulceration and the skin surface is smooth.

Occasionally, granuloma annulare can become more generalised in adults, particularly spreading to the skin folds around the axillae and groin.

The main problem with granuloma annulare is cosmetic as it can cause an unsightly rash. Some cases can be associated with itch. Diagnosis can be confirmed by a biopsy which show necrobiotic degeneration of dermal collagen but despite the name there are no granulomas on histology. Milder cases with little or no symptoms do not necessarily require treatment as the plaques often clear spontaneously after a few months or years.

More troublesome cases may respond to potent topical steroids or intralesional steroid injections. Some cases can respond to cryosurgery, topical imiquimod or topical calcineurin inhibitors (tacrolimus). Widespread cases may require systemic doxycycline [4], steroids or other immunosuppressants.

Most cases of granuloma annulare occur spontaneously but this condition can occasionally be linked with diabetes or thyroid disease. Extensive cases can sometimes be linked with lymphoma, HIV infection and solid tumours [5].

As a curiosity, some cases respond to the inflammation caused at the biopsy site. Once the biopsy is taken, the lesion heals completely.

## 51.7 Lupus Erythematosus

Most autoimmune conditions have some skin involvement. Most tend to worsen with UV light. These are patients that need to be treated by specialists and many times the medication used to control the systemic condition improves the skin lesions also.

### 51.7.1 Systemic Lupus Erythematosus (SLE)

The rash may be the presenting feature in 25% of cases. It consists of erythema and scaling of the upper cheeks and spreading across the nose causing the characteristic butterfly rash which is usually aggravated by ultraviolet light. Ear, scalp and trunk involvement with red scaly plaques may also occur with SLE (Figs. 51.1 and 51.2). It is twice as common in females as in males. Most patients (80%) will have positive anti-nuclear factor (ANF) antibodies. Some patients may have associated problems such as chilblains, scarring alopecia or urticaria. Patients with SLE may have underlying systemic involvement of the joints, kidney, heart, brain, etc. [6]. Certain drugs can cause a lupus type syndrome (Table 51.2).

Treatment of the skin manifestation of SLE usually involves potent or very potent topical steroids (e.g. clobetasol propionate,



**Fig. 51.1** Discoid lupus erythematosus



**Fig. 51.2** Discoid lupus erythematosus involving the external auditory canal

“Dermovate®”) even on the face. Patients should be instructed on protecting the exposed skin from ultra violet light. Topical calcineurin inhibitors such as tacrolimus can sometimes help. The more severe cases, particularly when complicated by underlying systemic involvement may require systemic steroids, antimalarials, methotrexate or other immunosuppressive agents under the care of a dermatologist or rheumatologist.

**Table 51.2** Drugs that can induce lupus erythematosus (SLE)

Terbinafine
Hydralazine
Carbamazepine
Lithium
Phenytoin
Sulphonamides
Minocycline



**Fig. 51.3** Subacute lupus causing a photosensitive rash

### 51.7.2 Subacute Lupus Erythematosus (SLE)

This is a papulo-squamous, annular rash that most commonly occurs on the upper trunk and neck, often following sun exposure. It can resemble psoriasis but there is little or no scaling (Fig. 51.3). Sub acute lupus usually spares the face and hands. 60% of these patients will have a positive anti-nuclear antibody test. 50% will go on to develop classical SLE. Subacute lupus usually does not scar. Most patients will respond to the same treatment as for SLE.

### 51.7.3 Discoid Lupus Erythematosus (DLE)

This is a more benign skin problem. However, it can still cause problems with an unsightly scarring rash, usually affecting the face or scalp. DLE causes persistent scaly discoid plaques which most commonly affect the face, neck, scalp and the external auditory meatus (Figs. 51.1, 51.2, and 51.4). When the scales are carefully removed there are plugs of keratin within the hair follicles (carpet tack sign). The plaques can clear spontaneously or with treatment but often leave permanent scarring. Scarring alopecia can cause permanent hair loss. Diagnosis is confirmed by skin biopsy which shows typical features of lupus.

Most cases are worse in the summer and one-third of patients with DLE can have positive ANF. It is twice as common in females and there is a less than 5% risk of the patient going on to develop SLE. This is usually treated with very potent topical steroids (even on the face) or topi-

cal calcineurin inhibitors. More severe cases usually respond to cryosurgery or intralesional steroids. Some cases resolve spontaneously as the patient gets older. Few cases may need systemic steroids or antimalarials. Camouflage make-up may help improve the appearance of the rash and hats and sun blocks are essential.

### 51.8 Erythema Nodosum

This is caused by panniculitis, characterised by sore, blotchy, hot, erythematous (red) nodules which normally affect both shins but may affect the dorsum of both forearms (Fig. 51.5). The rash appears suddenly and is symmetrical in most cases. It usually lasts a few weeks and then resolves spontaneously. It is a skin manifestation caused by a hypersensitive reaction to various triggers including a streptococcal sore throat, sarcoidosis and drugs including sulphonamides, salicylates, oral contraception pill and HRT. Other possible causes include pregnancy, TB or inflam-



**Fig. 51.4** Discoid Lupus erythematous in the scalp



**Fig. 51.5** Erythema nodosum post sore throat in a 22-year-old female

matory bowel disease. Many cases can be idiopathic with no underlying cause found.

Erythema nodosum is more common in females between the ages of 20 and 40 and the patient may have a low grade fever, myalgia and arthralgia.

The following investigations should be performed:

- Chest x-ray (TB or sarcoid)
- Full blood count
- Urea and electrolytes
- Liver function tests
- Erythrocyte sedimentation rate
- Random glucose
- HbA1c
- Throat swab
- Pregnancy test
- Serum ACE (Angiotensin-converting enzyme)
- ASO Titre (a test for streptococcal infection)

Treatment is symptomatic with rest, elevation, cold compresses, non-steroidal anti-inflammatories and emollients. Compression stockings may help. Oral tetracyclines and colchicine have been reported to help [7].

## **51.9 Dermatitis Herpetiformis (DH)**

This is a rare autoimmune itchy, blistering rash that most commonly occurs in people over the age of 50 and is twice as common in men. Patients usually present with an intensely itchy, eczematous rash with a classical distribution affecting the back of the elbows, the front of the knees, the buttocks and the scalp. They usually have tiny vesicles, less than 3 mm in diameter but most of these are scratched away as soon as they appear as they are intensely itchy. The diagnosis is confirmed by biopsying a blister which should show the classical histological features and positive direct immunofluorescence of uninvolved perilesional skin (see Chap. 23).

Dermatitis herpetiformis is strongly associated with coeliac disease in approximately 80% of cases, so all suspected cases should have blood tests for coeliac antibodies and a jejunal biopsy to look for total or subtotal villous atrophy. If coe-

liac disease is confirmed the rash usually improves with a strict gluten free diet for life. Dermatitis herpetiformis without coeliac disease may respond to emollients, potent topical steroids and antihistamines. More severe cases may require dapsone under careful specialist supervision. Dapsone can cause haemolytic anaemia, agranulocytosis, methaemoglobinæmia, and peripheral neuropathy. Regular FBCs and reticulocyte counts are necessary when taking dapsone [8].

Differential diagnosis for dermatitis herpetiformis includes the following:

- Eczema
- Contact dermatitis
- Scabies
- Bullous pemphigoid
- Linear IGA dermatitis
- Bullous lupus erythematosus

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## **51.10 Dermatomyositis**

This is a rare inflammatory autoimmune condition that can sometimes be a marker for underlying malignancy. It usually presents as photosensitive, reddish-purple patches which can affect the eyelids and face. The rash is described as heliotrope as it resembles the heliotrope flower which has a small purple petal. The rash may also affect the cheeks, nose, shoulders, upper chest and elbows. It is often associated with periorbital oedema. Purple spots, known as Gottron papules, are sometimes found symmetrically over the knuckles or over the extensor surfaces of the interphalangeal joints and there is often periungual telangiectasia and thickened, ragged cuticles [9] (Fig. 51.6). Dermatomyositis in children may be associated with calcification of the skin.

There is usually an associated myositis with proximal muscle weakness. This may be manifested by the patient having difficulty going up or down the stairs or combing their hair. One-third of cases of classical dermatomyositis have an underlying malignancy which may be diagnosed before, during or after the appearance of the rash. Patients should be investigated for adenocarcinoma of the lung, breast and GI tract. Treatment of the underly-



**Fig. 51.6** Gottron papules in dermatomyositis Photo courtesy of Dr. Myriam Raquel González Oviedo



**Fig. 51.7** Acanthosis nigricans in a 24-year-old obese man

ing malignancy may alleviate the symptoms of the dermatomyositis but overall the mortality rate is approximately 25%. Treatment usually involves oral steroids or other immunosuppressive or biologic agents. Patients should be advised on protecting their skin from ultra violet light.

## 51.11 Acanthosis Nigricans

This is a rare condition where the patient presents with thickened, brown, velvet-like hyperkeratotic plaques, usually affecting the neck, axillae and groin, as a result of increased insulin growth factor or tumour growth factors (Fig. 51.7). It can be associated with obesity, diabetes, the metabolic syndrome, autoimmune disease, paraneoplasia or drugs. Extensive cases, particularly if there is a sudden onset and associated thickening of the palms and face, can be linked with an underlying malignancy such as carcinoma of the gastrointestinal tract. Treatment is by dealing with the underlying cause. Topical retinoids, lasers or other surgical methods may help improve the appearance.

## 51.12 Neutrophilic Dermatoses

Neutrophilic dermatoses are autoinflammatory skin conditions which usually arise as a reaction to an underlying systemic diseases.

### 51.12.1 Pyoderma Gangrenosum

This is a rare painful ulcerating neutrophilic dermatosis that mostly affects the lower legs in people over the age of 50 but can occur on other sites. It usually occurs as a result of a reaction to an underlying disease. 50% of patients will have inflammatory bowel disease, rheumatoid arthritis, hematological disorders or myeloproliferative disease (e.g. IGA gammopathy). There are four main types of pyoderma gangrenosum.

1. Ulcerative
2. Pustular
3. Bullous
4. Negative (granulomatous)

Pyoderma gangrenosum usually starts after a minor skin trauma (pathergy). This may cause a small pustule or blister which usually breaks down resulting in a painful ulcer with purple undermined edges. The ulcer usually deepens and widens rapidly. Several ulcers may develop simultaneously (Fig. 51.8). The condition can be more troublesome and difficult to treat if there is underlying venous insufficiency in the lower leg.

Pyoderma gangrenosum is usually confirmed by biopsy and this will also rule out other serious conditions such as a malignant ulcer.

Treatment is usually by dealing with the underlying cause, if found. The ulcers may respond to very potent topical steroids or intralesional steroids. Appropriate dressings such as sil-



**Fig. 51.8** Pyoderma gangrenosum

ver impregnated dressings and compression bandaging may help particularly if there is also venous insufficiency. However, the ulcers may be too painful for compression.

Topical calcineurin inhibitors such as tacrolimus may help. More severe cases may require systemic treatment with oral steroids, dapsone, minocycline, immunosuppressants or biological agents such as infliximab under the care of a dermatologist.

### 51.12.2 Acute Febrile Neutrophilic Dermatosis

**Acute febrile neutrophilic dermatosis (Sweet's syndrome)** is another rare neutrophilic dermatoses that presents with a fever and a painful rash sometimes with blisters and mucosal involvement. It is most common in adults. It is usually triggered by an underlying condition such as

infection, inflammatory bowel disease, rheumatoid arthritis, pregnancy, myeloproliferative disorders, cancers or drugs. Sometimes no underlying cause can be found. One attack may occur as an isolated event or it may be recurrent. The rash is usually painful or tender and may cause papules, plaques, nodules, blisters, erosions or ulcers. The patient usually feels unwell with fever, malaise, arthralgia, headaches, mucosal or eye symptoms. Biopsy of the rash shows inflammation that is composed mainly of neutrophils without vasculitis. Most cases respond to systemic steroids. It usually clears without scarring but one third of patients may develop recurrent episodes. In these cases a careful search for underlying disease needs to be carried out.

### 51.12.3 Erythema Annulare Centrifugum

**Erythema annulare centrifugum (also known as annular erythema)** is another neutrophilic dermatoses that presents as an annular, red, non-scaly rash with little or no symptoms. The rash spread centrifugally while the centre clears and can reach 6 to 8 cm in diameter on the limbs, trunk or face. It can resemble tinea but there is no scale and can look like erythema migrans (Lyme disease) but there is no history of a tick bite.

Histology of the rash shows characteristic perivascular lymphocytic infiltration. The cause is usually unknown but it can occasionally be linked with underlying diseases such as infections, drugs, cancers, liver or thyroid disease.

Treatment is symptomatic and most cases clear spontaneously over a few weeks or a few months. Topical steroids can help if there is itch.

## 51.13 Scleroderma

Scleroderma is a rare auto-immune skin disease that can be localised (morphoea) or more generalised (systemic sclerosis).

### 51.13.1 Morphea

This is a localised form of scleroderma with no systemic involvement. It presents as an asymptomatic plaque of hairless hyper-pigmentation or hypo-pigmentation (Figs. 51.9 and 51.10). The plaques are usually found on the trunk, may be single or multiple and can measure from one to twenty centimetres in diameter. The skin has a distinctive leathery feel with the plaques having an ivory white centre, a lilac border and a smooth, shiny surface. The cause is unknown. Most cases improve spontaneously over many years. Rarely morphea can affect the underlying subcutaneous tissues including bone leaving a permanent area of depressed brown skin.

Diagnosis is confirmed by biopsy. Treatments are many and varied and are not always successful. Mild cases may require no treatment. Treatments that have been tried include topical tacrolimus, potent topical steroids, topical calcipotriol, topical imiquimod, phototherapy, methotrexate and systemic steroids in severe, extensive



**Fig. 51.9** Morphea on the back x 10 years



**Fig. 51.10** Morphea of unknown cause

cases [10]. Treatments are more successful if commenced early in the disease process.

### 51.13.2 Systemic Sclerosis

This generalised form of scleroderma can be divided into progressive systemic sclerosis and the CREST syndrome.

**Progressive systemic sclerosis** most commonly appears in women in their 30's and 40's. It is a multisystem autoimmune condition of unknown cause. It often begins with symptoms of Raynaud's disease. Fingers and toes go white in the cold from vasospasm (Fig. 51.11). As the fingers warm up they usually go blue and then red as the blood returns to the fingers. Symptoms are worse in the winter and in cold weather. Patients should be encouraged to wear extremely warm gloves such as ski gloves or motorcycling gloves. Hand warmers may also help. More severe cases often respond to calcium antagonists such as nifedipine, especially in the winter. Patients with systemic sclerosis may also develop Sjögren's syndrome (dry eye, mouth and vagina). Severe cases can be associated with a vasculitis that can affect internal organs including kidneys, lungs and skin. In more advanced cases the skin generally becomes tight (scleroderma is the Greek word for "hard skin") and can affect the face, resulting in expressionless facial features and difficulty opening the mouth.



**Fig. 51.11** Raynaud's and chilblains in a 20-year-old female



**Fig. 51.12** Raynauds in CREST syndrome in a 50-year-old female



**Fig. 51.13** Same patient with Raynauds in CREST syndrome

**CREST syndrome** is a limited form of scleroderma and is again more common in women. Calcium deposits can form in the skin especially on the fingers and hands (calcinoses). Patients may have Raynaud's phenomenon, oesophageal dysmotility, thickening of the skin of the fingers which eventually become thin, scarred and spindle shaped (sclerodactyly) and telangiectasia which may affect the fingers, palms, face, lips, tongue and chest (Figs. 51.12 and 51.13).

Patients with CREST syndrome will often need to be cared for by a multi-disciplinary team especially when there are multiple organs involved. Most patients are treated with systemic steroids, cyclophosphamide, methotrexate or anti-tumour necrosis factor alpha (anti-TNF-alpha) [11].

## 51.14 Porphyria Cutanea Tarda (PCT)

PCT can be an acquired (type 1) or inherited autosomal dominant (type 2) metabolic disorder of hepatic haem biosynthesis. It is caused by a

deficiency in the uroporphyrinogen decarboxylase leading to an accumulation of uroporphyrin in the urine and serum. Precipitating factors include alcohol abuse, oestrogen containing medicine, HIV infection, hemochromatosis, hepatitis C and hepatotoxic drugs including non steroidals anti-inflammatories, furosemide and tetracyclines.

The acquired form usually occurs in older men. It usually starts with a photosensitive rash and fragile skin especially on the dorsum of hands and sometimes on the face. There may be bullae and erosions on the exposed skin and hypertrichosis (excess hair growth) especially on the face. The urine is normally the colour of dark tea and fluoresces a pink coral colour under a Wood's lab.

Diagnosis can be confirmed by biopsy for histology and immunofluorescence. A 24 hour urine collection for total porphyrin levels will also help make the diagnosis. Plasma porphyrin screen is also useful and it can be evaluated using spectrofluorometry. (Table 51.3).

Treatment should include dealing with triggers like excess alcohol and high ferritin intake. Strict photo-protection should be encouraged. Some cases may respond to phlebotomy and anti-malarials such as chloroquine. Differential diagnosis includes bullous SLE and bullous pemphigoid. Patients with mixed porphyrias are at risk of acute neuropsychiatric attacks and abdominal symptoms as well as the skin manifestations [12]. Pseudoporphyria presents similarly to PCT except that porphyrin investigations are normal. It is often drug induced, secondarily to haemodialysis or sunbed use.

**Table 51.3** Investigations for porphyria cutanea tarda.

Full blood count, urea and electrolytes, liver function tests, glucose
Iron studies
Viral screen including Hep C + HIV
Auto antibody screen
Urine porphyrin levels
Plasma porphyrin screen
Abdominal ultrasound
Skin biopsy (histology and immunofluorescence)

## 51.15 Cutaneous Sarcoidosis

Sarcoidosis is a rare condition of unknown ideology that cause non-caseating epithelioid granulomas affecting various parts of the body including the lung, nodes, eyes, liver, skin, heart, neurological system and musculoskeletal system. Sarcoidosis can occur at any age in both sexes and all races. Cutaneous manifestation of sarcoidosis occurs in approximately 25% of patients and skin involvement is usually accompanied with other organ involvement, most commonly the lung and the mediastinal nodes. Cutaneous sarcoid can occur in isolation in approximately one-third of cases.

Granulomatous inflammation is caused by a variety of conditions including infection, autoimmune, toxic, allergic, drug, and neoplastic conditions. The precise type of granulomatous inflammation narrows the pathologic and clinical differential diagnosis and subsequent clinical management [13]. Common reaction patterns include necrotizing granulomas (caseating), non necrotizing granulomas, suppurative granulomas, autoimmune granulomatous inflammation, foreign body giant cell reaction and neoplastic (Table 51.4). Sarcoidosis is one of the most common causes of non-caseating epithelioid granulomas (Table 51.5).

Cutaneous sarcoidosis can cause non-specific signs such as erythema nodosum, erythema multiforme or calcinosis cutis. Specific skin lesions, which show granuloma on histology, are many and varied. Sarcoidosis is called “The Great Imitator” because it can present in so many different ways on the skin. The most well-known skin manifestation of sarcoid infiltration is lupus pernio which usually affects the nose and face. However, sarcoid can present on any part of the skin as non-specific nodules, plaques, annular lesions (Figs. 51.14 and 51.15), psoriatic form, lichenoid or ulcerative lesions or sub-cutaneous nodules. Sarcoid can sometimes infiltrate scars or tattoos.

Lupus pernio causes a red, purple or violaceous indurated plaques and nodules most commonly affecting the nose, cheeks, forehead, lips, ears, the dorsum of the hands, fingers or toes. Lupus pernio involving the central face such as the nose, lips or cheeks should prompt a thorough respiratory tract

**Table 51.4** Types of cutaneous granulomatous reactions

Non necrotizing	Sarcoid
Necrotizing(caseating)	Lupus vulgaris (TB)
	Leprosy
	Late syphilis
	Leishmaniasis
Suppurative	Mycobacterium marinum (fish tank granuloma)
	Aspergillus (fungal)
	Actinomycosis
	Cat scratch disease (gram negative bacteria = Bartonella henselae)
Autoimmune	Granuloma annulare
	Necrobiosis lipoidica
	Rheumatoid nodules
	Crohn's disease
	Rosacea
	Orofacial granuloma
	Systemic lupus erythematosus
	Drugs
Foreign body type reaction	
Neoplastic	Granulomatous mycosis fungoides (T-cell cutaneous lymphoma)
	Hodgkin lymphoma (HL)
	Metastasis

**Table 51.5** Differential diagnosis of sarcoidosis includes

- Cutaneous tuberculosis (mycobacterium tuberculosis)
- Drug eruptions
- Granuloma annulare
- Deep fungal infection
- Atypical mycobacteria
- Granuloma faciale
- Lamellar ichthyosis
- Leprosy (mycobacterium leprae)
- Lichen planus
- Discoid lupus erythematosus
- Subacute cutaneous lupus erythematosus
- Lymphocytoma cutis
- Necrobiosis lipoidica
- Plaque psoriasis
- Syphilis
- Tinea corporis
- B-cell lymphoma
- Foreign body reaction
- Lichen planopilaris



**Fig. 51.14** Cutaneous sarcoidosis in a patient who also had Type 2 diabetes



**Fig. 51.15** Cutaneous sarcoidosis on the back of a 75-year-old female who also had pulmonary sarcoidosis

investigation as it is commonly associated with chronic fibrotic lung disease.

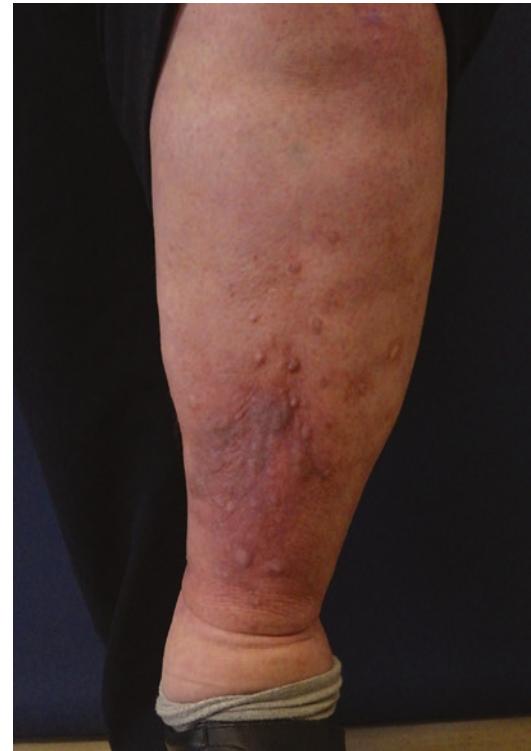
It can be resistant to treatment and it can cause considerable disfigurement. Diagnosis is usually by skin biopsy and tissue culture may have to be performed to rule out an infectious cause such as mycobacteria or deep fungal infection. A chest x-ray and pulmonary function tests should be performed to rule out pulmonary or mediastinal sarcoid. Routine investigation should include serum calcium, liver function tests, Vitamin D levels, thyroid function tests, VDRL, ANF and TB screening including a tuberculin skin test. Blood tests for TB including Interferon Gamma Release Assay (IGRA) such as QuantiFERON Gold which is a marker for active TB that is not influenced by previous BCG vaccination. Serum angiotensin-converting enzyme (ACE) levels may be increased in patients with sarcoidosis.

Treatment of cutaneous sarcoidosis will depend on whether it involves skin only or

involves other organs. The most common treatment for skin involvement would be topical or intralesional steroids. Oral steroids are sometimes required. Other treatments tried include antimalarials, tetracycline-class antibiotics, methotrexate, antimalarials, TNF Alpha inhibitors, PDT, deep x-ray therapy, lasers and surgery. Ironically, TNF Alpha blockers such as infliximab can sometimes cause a granulomatous eruption. Response to treatment is varied but some patients with papules and nodules can have them persist for many months or years.

## 51.16 Conclusion

Many systemic diseases can have skin manifestations. Sometimes the skin signs can predate the diagnosis and may in fact make the doctor suspicious and look more closely for an underlying disease (e.g. necrobiosis lipoidica is linked with diabetes, pretibial myxoedema may be associated with thyroid disease) (Fig. 51.16). Poorly con-



**Fig. 51.16** Pretibial myxoedema in a 53-year-old female with hypothyroid and obesity

trolled systemic diseases such as diabetes or HIV can result in exacerbation of incidental skin problems and make them more difficult to control. Treatment of some skin conditions may precipitate or aggravate underlying systemic diseases (e.g. high dose steroids may aggravate diabetes or methotrexate may cause liver disease).

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# Skin Problems Associated with Diabetes

52

David Buckley

## Key Points

- Systemic diseases such as diabetes can have associated skin manifestations which may precede the underlying disease and may be a clue to their diagnosis. Some systemic diseases are treated with drugs which may also cause skin problems.
- All patients presenting with skin infections should have their blood sugar or HbA1c checked, looking for undiagnosed diabetes or checking how well controlled their existing diabetes is.
- Necrobiosis lipoidica is strongly linked with diabetes and can predate the diagnosis of diabetes by many years. 1% to 2% of patients with diabetes will develop necrobiosis lipoidica.
- Treatment of some skin conditions may precipitate or aggravate underlying systemic diseases (e.g. high dose steroids may aggravate diabetes or methotrexate may cause liver disease).

## What to Tell the Patient

- Diseases such as seborrhoeic dermatitis and viral warts can be more difficult to treat in the presence of poorly controlled diabetes.

- Diabetes mellitus (DM) Type 1 and 2 is very common and the incidence is increasing worldwide. It is commonly associated with skin problems, some of which can predate the development of diabetes or be a clue to its existence for undiagnosed cases.
- Poorly controlled diabetes and patients on multiple medications because of their diabetes are more likely to develop skin problems.

## 52.1 Introduction

Skin conditions associated with diabetes mellitus (DM) can be mainly classified into five main categories [1]:

1. Infections (bacterial, fungal, viral) associated with hyperglycaemia.
2. Cutaneous manifestations of diabetic complications (microangiopathy, macroangiopathy, neuropathy).
3. Skin reactions due to diabetic treatment (sulphonylureas or insulin).
4. Skin lesions with an association with diabetes (necrobiosis lipoidica, diabetic dermopathy, diabetic bullae, diabetic stiff skin, eruptive xanthomas, acanthosis nigricans, granuloma annulare).
5. Skin diseases that are more common in patients with diabetes such as xerosis (dry skin), psoriasis and eczema.

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## 52.2 Infections

At least 30–50% of patients with diabetes have skin problems. The most common are bacterial skin infections such as boils, carbuncles, folliculitis and cellulitis. Yeast and fungal infections such as tinea paedis, tinea unguium, candidiasis, intertrigo and seborrhoeic dermatitis are all more common in those with diabetes. Viral infections such as viral warts, herpes simples and varicella-zoster also occur more often in patients with diabetes. These infectious diseases are more common if the diabetes is poorly controlled. All patients presenting with skin infections should have their blood sugar or HbA1c checked, looking for undiagnosed diabetes or checking if their known diabetes is being well controlled.

## 52.3 Cutaneous Manifestations of Diabetic Complications

Diabetes can often lead to peripheral vascular disease, especially if the patient has hyperlipidaemia or is a smoker. This can predispose to diabetic leg and/or foot ulcers which are mostly arterial. Diabetes can also be associated with neuropathy which can be another risk factor for foot ulcers.

## 52.4 Skin Reactions to Diabetic Treatment

The drugs that are used to treat diabetes can sometimes cause drug rashes and this should always be considered if someone with diabetes presents with a rash or itch of unknown origin. Injection site reactions from insulin can also lead to local skin reactions.

## 52.5 Skin Lesions with an Association with Diabetes

### 52.5.1 Necrobiosis Lipoidica

Necrobiosis lipoidica is a rare granulomatous skin disorder that most commonly occurs on the

shin, often bilaterally, although it can occur in other areas of the body. Necrobiosis lipoidica is also known as necrobiosis lipoidida diabetorum as it is strongly linked with diabetes and can predate the diagnosis of diabetes by many years. More than half of the patients with necrobioses lipoidica will have or will eventually develop diabetes. 1% to 2% of patients with diabetes will develop necrobiosis lipoidica [2]. It is more common in women and in smokers.

It usually starts as an asymptomatic, erythematous, macular patch which grows slowly over months or years. As it matures, the plaques become more atrophic with a yellow-orange appearance and associated telangiectasia (Figs. 52.1 and 52.2). Sometimes it can ulcerate and become painful. As the plaques mature they become shiny, pale, thin and hairless. A minor injury may cause small ulcers which may become infected.

Although necrobiosis lipoidica has a classical clinical appearance, a skin biopsy is usually required to confirm the diagnosis and to exclude more serious conditions such as squamous cell carcinoma or a superficial spreading BCC.

Treatment is symptomatic and will depend on the stage of the disease. Milder cases may respond to potent topical steroids, sometimes with occlusion or intralesional steroid injections.



**Fig. 52.1** Necrobiosis lipoidica that was present for 5 years without being diagnosed with diabetes



**Fig. 52.2** Necrobiosis lopoidica present for the last 13 years and no diabetes diagnosed

Topical tacrolimus may help in milder cases. Systemic treatment such as cyclosporin or biologic agents may be required in severe cases [3].

### 52.5.2 Granuloma Annulare

This is a benign condition that usually presents as an annular rash that often affects the hands or feet in children and young adults. Most cases of granuloma annulare occur spontaneously but this condition can occasionally be linked with diabetes or thyroid disease. Extensive cases can sometimes be linked with lymphoma, HIV infection and solid tumours [4].

The dermal annular plaques which have a thickened, nodular, papular border, most commonly occur over joints, particularly the knuckles. The centre of each ring is often flat and relatively normal. The plaques may be solitary or multiple (see Chap. 5, Fig. 5.1). The annular rash can be confused with tinea corporis (ringworm) or a BCC, but unlike these conditions, granuloma annulare is a deep dermal lesion with no scaling, bleeding or ulceration and the skin surface is smooth.

Occasionally, granuloma annulare can become more generalised in adults, particularly spreading to the skin folds around the axillae and groin.

The main problem with granuloma annulare is cosmetic as it can cause an unsightly rash. Some cases can be associated with itch. Diagnosis can be confirmed by a biopsy which show necrobiotic degeneration of dermal collagen but despite the name there is no granulomas on histology. Milder cases with little or no symptoms do not necessarily require treatment as the plaques often clear spontaneously after a few months or years. More troublesome cases may respond to potent topical steroids or intralesional steroid injections. Some cases can respond to cryosurgery, topical imiquimod or topical calcineurin inhibitors (tacrolimus). Widespread cases may require systemic doxycycline [5], steroids or other immunosuppressants.

### 52.5.3 Acanthosis Nigricans

This is a rare condition where the patient presents with thickened, brown, velvet-like hyperkeratotic plaques, usually affecting the back of the neck, axillae and groin, as a result of increased insulin growth factor or tumour growth factors (Fig. 52.3). It can be associated with obesity, diabetes, the metabolic syndrome, autoimmune disease, drugs and even paraneoplastic syndrome. Extensive cases, particularly if there is a sudden onset and associated thickening of the palms and face, can be linked with an underlying malignancy such as carcinoma of the gastrointestinal tract. Treatment is by dealing with the underlying cause. Topical retinoids, lasers or other surgical methods may help improve the appearance.

### 52.5.4 Diabetic Dermopathy ("Shin Spots")

This usually presents as small reddish-brown, oval or round pigmented patches on the shins or sometimes other areas of the body (front of the thighs, forearm, side of the foot, scalp and trunk). They are most commonly found in patients with DM. They may be traumatic in origin or associ-



**Fig. 52.3** Acanthosis nigricans in the axilla in an obese female with a history of gestational diabetes

ated with neuropathy or vasculopathy. It is more common in the elderly and in those with poor diabetes control. No treatment is usually required as they are usually asymptomatic. Cosmetic camouflage may help hide the appearance of the pigmentation.

### 52.5.5 Diabetic Bullae

This condition causes large spontaneous bullae on any part of the body in patients with DM but most commonly occur on the feet and hands. It is also known as bullosis diabetorum and is of unknown aetiology. The blisters may be intraepidermal (heal with no scarring) or subepidermal

(heal with scars or atrophy) and may be fluid or blood filled. Most cases heal spontaneously.

### 52.5.6 Diabetic Stiff Skin

This most often occurs in patients with long-standing type 1 DM. The skin can become waxy, thickened and yellow and may cause stiffness of the skin especially over the fingers and hands.

### 52.5.7 Eruptive Xanthomas

This is very rare condition where the patient develops crops of tender or itchy, red-yellow papules which most commonly arise over the buttocks, shoulders, arms or legs. It can be associated with diabetes and most cases resolve spontaneously over a few weeks or months.

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## 52.6 Skin diseases that are more common in patients with diabetes

Some common skin conditions such as xerosis (dry skin), psoriasis and eczema are more commonly found in patients with diabetes.

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## 52.7 Conclusion

Diabetes is the most common endocrine disorder found in primary care, affecting 8.3% of the population [6]. Skin disorders are found in 79.2% of people with diabetes and mostly in type 2 diabetes. The most common skin manifestations are cutaneous infections (47.5%), xerosis (26.4%), and inflammatory skin diseases (20.7%) [7]. Cutaneous disease can appear as the first sign of diabetes or may develop at any time in the course of the disease.

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# Skin and the Mind (Psychodermatology)

53

David Buckley

## Key Points

- Skin diseases can have a profound effect on the emotional, social and sexual aspects of a patient's life.
- Skin problems may lead to low self-esteem, depression, insomnia, substance abuse or even suicide.
- Depression and anxiety can result in picking and scratching which may cause skin problems (e.g. neurodermatitis), aggravate an associated skin condition (e.g. acne, eczema) or mask underlying disease making diagnosis of the primary skin problem more difficult.
- Some people may deliberately cause or aggravate their skin problem to adopt "the sick role" in order to seek attention or avoid situations such as work or school.
- Many psychiatric drugs can cause skin problems (lithium can aggravate psoriasis) or photosensitivity (chlorpromazine can cause photosensitivity).
- Some drugs used to treat skin problems may cause or aggravate depression, anxiety or insomnia.

## What to Tell the Patient

- Sometimes itch and other uncomfortable sensations in the skin that the person believes to

be due to insect bites or infestation can originate in the mind rather than the skin.

- Some people may become overly fixated or self-conscious about the appearance of their skin that nobody else can see.
- It is important to explore the patient's ideas, concerns and expectations (ICE) regarding their skin problems.

## 53.1 Introduction

Many skin conditions have a major psychological component, which if not dealt with, will result in poor response to treatment. Conversely, some psychiatric conditions can present with a skin manifestation. In this chapter we will explore the link between the skin and the mind.

## 53.2 Psychological Aspect of Skin Disorders

Some patients' skin conditions may be aggravated if they are anxious or depressed (e.g. acne or eczema may flare up around exam times). This can be as a result of many factors associated with stress such as picking and scratching, lack of sleep, poor diet, lack of exercise and poor compliance with treatment. It is important to assess the impact of a skin condition on the patient's quality of life as this may determine how aggressively the skin condition needs to be treated. [1] (Table 53.1).

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**Table 53.1** Dermatology Life Quality Index (DLQI)**DERMATOLOGY LIFE QUALITY INDEX (DLQI)**

Hospital No: .....

Date: .....

Name: .....

Score: .....

Address: .....

Diagnosis: .....

.....

**The aim of this questionnaire is to measure how much your skin problem has affected your life  
OVER THE LAST WEEK. Please tick (✓) one box for each question.**

- |   |              |                          |
|---|--------------|--------------------------|
| 1. Over the last week, how <b>itchy, sore, painful or stinging</b> has your skin been?  | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
| 2. Over the last week, how <b>embarrassed</b> or <b>self conscious</b> have you been because of your skin?  | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
| 3. Over the last week, how much has your skin interfered with you going <b>shopping</b> or looking after your <b>home</b> or <b>garden</b> ?            | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 4. Over the last week, how much has your skin influenced the <b>clothes</b> you wear?   | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 5. Over the last week, how much has your skin affected any <b>social</b> or <b>leisure</b> activities?  | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 6. Over the last week, how much has your skin made it difficult for you to do any <b>sport</b> ?  | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 7. Over the last week, has your skin prevented you from <b>working</b> or <b>studying</b> ?   | Yes          | <input type="checkbox"/> |
|   | No           | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| If "No", over the last week how much has your skin been a problem at <b>work</b> or <b>studying</b> ?   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
| 8. Over the last week, how much has your skin created problems with your <b>partner</b> or any of your <b>close friends</b> or <b>relatives</b> ?       | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 9. Over the last week, how much has your skin caused any <b>sexual difficulties</b> ?   | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |
| 10. Over the last week, how much of a problem has the <b>treatment</b> for your skin been, for example by making your home messy, or by taking up time? | Very much    | <input type="checkbox"/> |
|   | A lot        | <input type="checkbox"/> |
|   | A little     | <input type="checkbox"/> |
|   | Not at all   | <input type="checkbox"/> |
|   | Not relevant | <input type="checkbox"/> |

**Please check you have answered EVERY question. Thank you.**

**Tab. 53.1** (continued)**DERMATOLOGY LIFE QUALITY INDEX (DLQI) - INSTRUCTIONS FOR USE**

The Dermatology Life Quality Index questionnaire is designed for use in adults, i.e. patients over the age of 16. It is self explanatory and can be simply handed to the patient who is asked to fill it in without the need for detailed explanation. It is usually completed in one or two minutes.

**SCORING**

The scoring of each question is as follows:

Very much	scored 3
A lot	scored 2
A little	scored 1
Not at all	scored 0
Not relevant	scored 0
Question 7, 'prevented work or studying'	scored 3

The DLQI is calculated by summing the score of each question resulting in a maximum of 30 and a minimum of 0. The higher the score, the more quality of life is impaired.

**HOW TO INTERPRET MEANING OF DLQI SCORES**

0 – 1	no effect at all on 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

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*There is more information about the DLQI, including over 85 translations, at [www.dermatology.org.uk](http://www.dermatology.org.uk). The DLQI is copyright but may be used without seeking permission by clinicians for routine clinical purposes. For other purposes, please contact the copyright owners.*

### 53.2.1 Ideas, Concerns and Expectations (I.C.E.)

When treating patients with skin problems it is important to treat them holistically and to explore their *ideas* about their skin problems, their *concerns* regarding the diagnosis and their *expectations* regarding treatment. Some patients have unrealistic ideas about why they have a skin problem (e.g., all eczema is due to drinking milk or acne is caused by greasy foods). These ideas need to be explored so that the doctor can educate the patient about the aetiology of their skin condition. Many patients have unrealistic concerns about their skin problem (i.e. there is no treatment available or they will have the skin problem for the rest of their life). Again, it is important to explore the patients concerns regarding their treatment and to correct any misconceptions. Finally, many patients may have unrealistic expectations about their treatment (e.g. acne will clear up in 2 weeks or laser treatment will clear their extensive psoriasis). If patients are not given realistic expectations about what to expect from their treatment they will become disillusioned, may default or go elsewhere (e.g., to an alternative practitioner). Conversely, if the patient knows what to expect with their treatment, realise what the doctor is trying to achieve (often we will try to improve the appearance and symptoms rather than cure the underlying condition) and have some idea how long it will take to see some improvements, they are more likely to comply and persevere with treatment.

## 53.3 Skin Aspect of Psychiatric Disease

There are some primary psychiatric conditions which manifest themselves in the skin and are usually associated with deep psychological problems such as anxiety, depression or psychosis.

### 53.3.1 Habit Biters and Pickers

Some skin conditions are as a result of patients' bad habits (e.g., picking, biting or scratching

their nails and cuticles can cause acute or chronic paronychia). Unless the habit is broken, successful treatment is unlikely. Likewise, some skin conditions such as **neurodermatitis (lichen simplex chronicus)** can arise primarily as a result of scratching, which may be the patients' way of dealing with stress. The scratched area is typically unilateral and reachable by the dominant hand. The back of the neck, forearms, elbows, scrotal skin, legs, and ankles are common sites. The constant scratching of a small area of the skin will irritate the skin, which will cause more itch and more scratching. This itch-scratch-itch cycle can go on for weeks or months resulting in thickening (lichenification), scaling and pigmented changes in the affected area (Fig. 53.1). The itch-scratch cycle has to be broken in order to clear the rash. This can be helped with the use of potent topical steroids and emollients. The patient may also need help with any possible underlying anxieties or stresses that may have led them to start scratching in the first place.

Some skin conditions such as severe acne, eczema or psoriasis can cause the patient to suffer stress or depression to the extent that some patients may even contemplate suicide. This needs to be explored in each individual patient, as some may need counselling or cognitive behavioural therapy to come to terms with their skin problem and the resulting anxiety or depression.

Acne patients who are very distressed as a result of their appearance may need more potent treatment such as oral isotretinoin to control the disease. This should be prescribed



**Fig. 53.1** Picker's dermatitis for 3 years in a 21 year old

only by doctors with experience in systemic retinoids as there are unconfirmed reports that oral isotretinoin may cause mood disorders including depression in some patients. On the other hand, acne itself can cause depression in some patients and clearing their acne with whatever treatment is necessary (including using oral isotretinoin) will improve not only their acne but also the patient's mood (see Chap. 7).

### **53.3.2 Skin Picking Disorder (SPD) ("Pickers Dermatitis")**

SPD has recently been recognised as an independent condition in the Diagnosis and Statistical Manual of Mental Disorders (DMS-5; 2013), (Table 53.2). It is also known as "Excoriation Disorder" or what I like to call "pickers dermatitis". This is a compulsive and habitual behaviour that is more common in females and may be associated with anxiety, depression, obsessive compulsive disorder, eating disorders, trichotillomania, substance abuse and body dysmorphic disorder. Picking episodes may be triggered by stress, anxiety or boredom and the patient may experience a feeling of relief, pleasure or gratification immediately after picking.

Self induced excoriation is usually obvious on examination with monomorphic, discreet, eroded

**Table 53.2** American Psychiatric Association's diagnostic criteria for skin-picking disorder

1. Recurrent skin picking resulting in skin lesions
2. Repeated attempts to decrease or stop skin picking
3. The skin picking causes clinically significant distress or impairment in social, occupation, or other important areas of functioning
4. The skin picking is not attributable to the psychological effects of a substance (e.g., cocaine) or another medical condition (e.g., scabies)
5. The skin picking is not better explained by symptoms of another mental disorder (e.g., delusions or tactile hallucinations in a psychotic disorder, attempts to improve a perceived defect or flaw in appearance in body-dysmorphic disorder, stereotypes in stereotypic movement disorder, or intention to harm oneself in non-suicidal self-injury)

or ulcerated lesions symmetrically distributed in easily accessible areas of the body. The targets for skin pickers include healthy skin, minor imperfections, pimples (acne excoriate), callouses and old picking scabs, which can be picked at with fingernails (most common) or even tools like tweezers or pins. Right handed people may have more lesions on the left side of their body and vice versa. There may be multiple lesions at various stages of healing with secondary scarring and pigmentation changes.

A careful history and physical examination is required to rule out underlying medical causes such as iron deficiency, diabetes, drug eruptions, scabies or the pre-bullous stage of bullous pemphigoid. Blood tests and a skin biopsy may be required to rule out underlying disease. A careful mental state examination should be carried out to detect underlying psychological factors. Unlike dermatitis artefacta, patients with SPD are usually willing to accept that their problem is self-inflicted.

Management can be difficult but a sympathetic, empathetic approach will help. A thorough history and physical examination will reassure the patient that you are taking their problem seriously. It may take a few consultations to convince the patient to approach the problem from a psychological rather than a dermatological point of view. Cognitive behaviour therapy or habit reversal therapy may help. Underlying psychological problems may have to be treated. Referral to a psychiatrist may be necessary in some patients especially if there is a doubt about the diagnosis or a risk of suicide. Good skin care such as moisturising and using a soap substitute may help.

### **53.3.3 Body Dysmorphic Disorder (BDD) (Delusional Dysmorphophobia)**

This unusual condition is a form of delusion where the patient believes some part of their appearance is grossly and hideously distorted or unsightly (e.g. their nose is too big, their chin is too small or their mild acne is horrendously

severe). These patients are rich in symptoms but poor in signs.

They have a preoccupation with an imagined or exaggerated defect in their physical appearance [2].

Population studies suggest that the general incidence of BDD is around 2% but rises to 4.2% in dermatology clinics and 8.6% in cosmetic dermatology clinics. These patients can be very difficult to reason with. What we perceive as trivial may be a source of great distress for the patient. They have a very fixed idea about their appearance and will not respond to reassurance. Just like patients with anorexia nervosa, they usually have a fixed body image disorder. They often use words like “massive,” “huge,” “horrendous,” yet when their skin is examined only normal anatomy or very mild disease is evident. These patients may often insist on radical treatment such as plastic surgery for what they perceive to be a large nose or oral isotretinoin for very mild acne. Doctors should resist this approach as invariably the patient will be unhappy with anything the doctor does for them and will often come back complaining about the treatment or the problem may shift to another area of the body. If BDD is suspected, photos should be taken to document the condition of the skin before treatment as the patient may attribute minor defects to your treatment although they were present and documented beforehand.

Reassuring the patient is usually ineffective and may even be harmful, triggering feelings of shame, guilt, frustration and anger. If the doctor is too forceful and direct, they may default or seek treatment elsewhere. It is important to focus on their distress rather than the skin complaint. Honesty and empathy are important when dealing with patients with BDD. They need to be confronted gently and offered help in trying to understand that much of their problems are in their mind rather than in their skin and that treatments are available to relieve their distress. It may be better to avoid labelling the patient too early and use terms like “body image concerns” or “poor self image” instead of BDD particularly during the first few visits to the doctor.

These patients usually need a multidisciplinary approach and have to be handled sensitively. They often have deep psychological

problems, sometimes with obsessive-compulsive traits, shame, anxiety, depression or social phobia. Some may even be suicidal. If possible, they should be referred to a psychiatrist and/or a counsellor with experience in body dysmorphic disorder. Some of these patients may respond to high dose SSRI antidepressants and/or cognitive behavioural therapy. It can often be difficult to convince them to go for psychological help because of their lack of insight.

### **53.3.4 Delusional Infestation (Parasitophobia)**

This is a rare condition where patients have a fixed, false belief that they are infected or infested with some form of biting insects or mites that they feel are crawling under their skin or biting them. It is also known as delusional parasitosis or Ekbom’s syndrome. It is more common in older women. They will constantly scratch, pick or bite their skin. This will result in skin trauma, minor bleeding and scabs which the patient will be convinced are tiny insects. Some cases can occur following a real infestation but the patient cannot get the feeling of insects crawling under their skin or biting them out of their mind long after the infestation has been treated. Other cases can arise de novo with no previous infection or infestation. They will invariably try numerous anti-scabies treatments and various insecticides, which may further irritate their skin causing more itching and sometimes a rash, which they will interpret as worsening of the infestation. Patients sometimes present with samples of tiny amounts of dead skin, scabs and dry blood which they will want the doctor to examine with a microscope to prove that they have insects in their skin (the matchbox sign). Invariably, examining the specimens will show only dead skin but no insects. Not only will the patient be convinced that they are infested or infected but family members may also get caught up in the delusion (Folie-à-deux) and may go to enormous lengths to rid the house of the insects such as fumigation. These patients have a fixed mono-delusion of infestation and tactile hallucinations known as formication, a sensation resembling insects crawling on or under the skin.

It is very hard to reason with these patients because of their lack of insight. They will often have seen a number of different doctors who have treated them with various anti-scabies and other anti-infective agents, which will further reinforce their delusion, despite the lack of response to these treatments. Some of these patients may have deep underlying psychological problems such as anxiety, depression, dementia, or schizophrenia. However, many are quite well apart from their isolated mono delusion of infestation.

It is very important to encourage the patients to stop using insecticides or any other anti-infective agents. They should be encouraged to stop picking and scratching. They need to avoid soaps and other irritants and moisturise their skin gently to try and ease any itch.

Patients with delusional infestation will often have to be referred to a colleague with more experience in skin disorders as it can be difficult to prove definitively that the patient is not being bitten or infested. Other causes of generalised pruritus need to be excluded. It is important to empathise with the patient and their family and to get them to consider psychological causes for their symptoms. The emphasis when treating the patient is to focus on relief of the symptoms rather than be fixated on the cause. However, many of these patients will require treatment with anti-psychotics such as pimozide (“Orap®”). The initial doses of 1–2 mg daily should be titrated slowly (1 mg every 5–7 days) to the effective dose that is best tolerated. Typical maintenance doses are between 2 and 4 mg daily [3]. Risperidone 0.5–1 mg BD for a few months is an alternative treatment to break the cycle of delusion, scratching and picking.

### 53.3.5 Dermatitis Artefacta

Deliberate self-harm is obvious and a patient may admit to what they are doing to their skin (e.g. multiple superficial cuts with a blade to the wrist, abdomen or thighs) (Fig. 53.2). Invariably, these patients will have deep psychological problems that will need to be assessed by a psychiatrist and/or a counsellor who should help them with strategies to release stress other than self-harm. These patients are sometimes suicidal and need urgent professional help by a mental health team.



**Fig. 53.2** Dermatitis artefacta. Photo courtesy of Dr Myriam Raquel González Oviedo



**Fig. 53.3** Trichotillomania. Photo courtesy of Dr Myriam Raquel González Oviedo

Some patients may hide the fact that they are self-harming. This is known as **dermatitis artefacta**. This is often a form of Munchausen syndrome (“Factitious Disorder”) where they are using their skin disease to adopt “the sick role” in order to seek attention or avoid situations such as work or school. It is far more common in women (7:1). They may present with bizarre rashes or ulcers that cannot be explained and are unresponsive to all forms of treatment because they are secretly damaging their skin in the privacy of their own bedroom. Some patients may present with alopecia as a result of secretly pulling out their own hair (**trichotillomania**) (Fig. 53.3). These patients almost invariably will have deep psychological problems and may be suicidal. Some patients may be victims of physical or sex-

ual abuse. Others may have features of obsessive-compulsive disorder. They will need referral to a doctor with experience in skin diseases to confirm that there is no underlying skin or scalp pathology and that the clinical findings are as a result of self-inflicted injury. They will need multi-disciplinary management from their GP, skin specialist, psychiatrist and counsellor. Getting them to admit to self-harm can be difficult and referring them to a psychiatrist may prove impossible. Initially, it is probably better to avoid direct confrontation and concentrate on dealing with any underlying psychiatric morbidity.

### 53.3.6 Other psychiatric conditions with skin manifestations

Certain psychiatric conditions can cause skin problems such as patients with obsessive compulsive disorder who repeatedly wash their hands often end up with hand dermatitis. Patients with schizophrenia are more likely to suffer from fungal infections and dermatitis. Many psychiatric drugs can cause skin problems such as itch, rash, acne or photosensitivity (e.g. lithium can aggravate psoriasis, chlorpromazine can cause photosensitivity).

## 53.4 Conclusion

Many diseases can have physical and psychological components, none more so than in dermatology. GPs are well placed to deal holistically with both the skin problem and any associated emo-

tional, psychological or psychiatric issues. There is a lot of stigma attached to skin diseases especially if it affects exposed areas (face or hands). This can be intensified by the mistaken, traditional belief that many skin rashes are infectious and contagious. In more recent times the fear of cancer is foremost in many patients mind when they develop a new lesion or a rash. The anxiety associated with skin diseases can cause stress which can further exacerbate the skin problem. Stress can also cause picking and scratching which may mask the underlying disease and make diagnosis of the primary skin problem difficult.

Skin diseases can have a profound effect on the emotional, social and sexual aspects of a patient's life. This can be measured using various questionnaires such as the Dermatology Life Quality Index (Table 53.1) [1]. These can measure the patients perception of the effects of a treatment on their quality of life, which is equally as important as assessing the effects of the treatment in clearing a rash.

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# Cutaneous Vasculitis

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David Buckley

## Key Points

- The most common causes of vasculitis are infectious disease, autoimmune disease and drug-induced.
- The most common forms of cutaneous vasculitis include Henoch–Schönlein purpura (HSP), meningococcal septicaemia and urticarial vasculitis.
- An urticarial type rash that lasts more than 24 hours, leaves some bruising on the skin and does not respond to antihistamines is probably due to urticarial vasculitis.
- Cutaneous vasculitis usually presents as palpable purpura.

## What to Tell the Patient

- Bruising that appears for no reason, especially in young people and those not on blood thinners may be a sign of a serious underlying disease.
- Bruising with a fever is a medical emergency and the patient should seek immediate medical attention.

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## 54.1 Introduction

Vasculitis is defined as inflammation of the blood vessels. This can affect all vessels from the tiniest capillaries to the largest artery including the aorta, as well as veins. It is a vascular disorder and as such may affect any area of the body including the skin, kidneys, lung, heart, brain or muscles. Severe vasculitis can be life threatening (Table 54.1). Cutaneous vasculitis (also known as cutaneous leukocytoclastic vasculitis) is where the disease process mainly affects the blood vessels of the skin. Many cases of cutaneous vasculitis may have underlying systemic involvement and may affect other areas of the body such as the kidneys, GI tract or the lungs.

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## 54.2 Cutaneous Vasculitis

Cutaneous vasculitis causes the blood vessel in the skin to become fragile which then leak resulting in petechiae (tiny red dots less than 3 mm), palpable purpura (red or purple, raised, discoloured spots 3–10 mm in diameter), ecchymosis (bruising >10 mm), swelling or in severe cases even blisters or ulceration (Fig. 54.1). Healed areas may leave evidence of post-inflammatory hyperpigmentation. The key distinguishing feature and important clinical point is that petechiae and purpura are raised (palpable) and do not

**Table 54.1** List of some rare generalised forms of vasculitis some of which may have skin manifestation

*Cutaneous vasculitis*

Manly affects the skin but other organs may be involved (e.g. Henoch–Schönlein purpura, urticarial vasculitis, meningococcal septicaemia, drug induced vasculitis)

*Behçet's syndrome*

Also known as Behçet's disease, is an autoimmune condition which causes mouth ulcers, genital ulcers, skin problems and eye inflammation

*Giant cell arteritis (temporal arteritis)*

Giant cell arteritis causes severe inflammation in the large blood vessels, and commonly affects the arteries in the head that can cause headaches and rarely blindness

*Polyarteritis nodosa*

Polyarteritis nodosa (PAN) causes inflammation to medium-sized arteries of the GI tract and kidneys

*Takayasu arteritis*

Takayasu arteritis (TA) is a rare inflammatory disease that affects the large artery from the heart and its large branches, usually in younger women

*Kawasaki disease*

Kawasaki disease is quite a very rare condition which affects small and medium-sized arteries, usually in small children (<5 years old)

*Granulomatosis with polyangiitis*

This very rare vasculitis can affect the ear, nose and throat problems but can also involves the skin, lungs, eyes and kidneys

*Eosinophilic granulomatosis with polyangiitis*

This can result in adult onset asthma as a result of vasculitis affecting the lungs

*Microscopic polyangiitis*

This causes vasculitis affecting the kidneys which can lead to high blood pressure and possibly renal failure

*Cryoglobulin-associated vasculitis*

Cryoglobulins which develop as result of cold temperatures can lead to vasculitis and small vessel occlusion

*Immunoglobulin A vasculitis*

IgA vasculitis, affects the small blood vessels mostly in boys from 2 to 10 years

blanch on pressure. The diagnosis can only be confirmed by skin biopsy.

Cutaneous leukocytoclastic vasculitis is a histopathologic term that refers to vasculitis limited to the small vessels in the skin in which the inflammatory infiltrate is composed of neutrophils and accompanied by leukocytoclasis, fibri-



**Fig. 54.1** Cutaneous vasculitis with palpable purpura in a patient with a underlying malignancy

noid necrosis, damage of endothelial cells and extravasation of red blood cells. If cutaneous leukocytoclastic vasculitis is diagnosed on skin biopsy it is important to exclude systemic organ involvement by taking a careful history, carrying out a thorough physical examination and doing some baseline tests such as urinalysis, full blood count, erythrocyte sedimentation rate, urea and electrolytes, liver function tests, thyroid function tests (TFT's), antinuclear factor (ANF), rheumatoid factor (RF), C3, C4, serum protein electrophoresis, cryoglobulins, hepatitis B and C screen and a chest X-Ray. Blood cultures may have to be considered if the patient is sick or has a fever. The second goal when approaching a patient with cutaneous leukocytoclastic vasculitis is to try to establish the cause [1]. Table 54.2 outlines some investigations to consider for vasculitis.

There are many causes of vasculitis but the most common causes are infectious disease, autoimmune disease and drug induced (e.g. ACE inhibitors, angiotensin II receptor antagonist, clarithromycin, NSAIDs). In up to 50% of cases of vasculitis there may be no known cause (Table 54.3).

The most common forms of cutaneous vasculitis include Henoch–Schönlein purpura, meningococcal septicaemia and urticarial vasculitis.

**Table 54.2** Investigations for vasculitis

- Investigations should be tailored to the possible cause. For all patients suspected of having a vasculitic lesion, consider:
- Full blood count
  - Erythrocyte sedimentation rate
  - C reactive protein
  - Urea and electrolytes
  - Liver function tests
  - Thyroid function tests (TFT's)
  - Urine culture, microscopy
  - Urinalysis (glucose, protein and blood, Bence Jones Protein)
  - Hepatitis serology (B and C)
  - VDRL
  - Complement levels (C3,C4, etc.)
  - Rheumatoid factor
  - Autoantibody screen(ANF, etc.)
  - Blood pressure
- Other tests to consider:**
- Chest X-ray
  - Skin biopsy
  - Angiography
  - Antistreptococcal antibody titres (ASOT)
  - Throat Swab
  - Monospot
  - Blood culture
  - Coagulation screen
  - Cryoglobulins

**Table 54.3** Aetiology of vasculitis

- Idiopathic (45–55%)
- Infection (15–20%)—e.g., Henoch-Schönlein purpura, septic vasculitis, upper respiratory tract flares of granulomatosis with polyangiitis (Wegener's granulomatosis), polyarteritis nodosa (PAN)
- Inflammatory disease (15–20%)—e.g., systemic lupus erythematosus (SLE), rheumatoid arthritis, Crohn's disease and ulcerative colitis
- Drug-induced (10–15%)—e.g., sulfonamides, beta-lactams, quinolones, non-steroidal anti-inflammatory drugs (NSAIDs), oral contraceptives, thiazides, anti-influenza vaccines. Chemicals such as insecticides and petroleum products
- Neoplastic (<5%)—e.g., as a result of a paraproteinaemia or lymphoproliferative disorder

### 54.3 Henoch-Schönlein purpura (HSP, also called anaphylactoid purpura)

This is a rare condition and usually occurs in children under the age of ten. They often present with petechiae or palpable purpura on the lower

**Fig. 54.2** Henoch-Schönlein purpura. Photo courtesy of Dr Myriam Raquel González-Oviedo

legs (Fig. 54.2). Seventy-five per cent of cases will have joint involvement and up to three quarters of patients may have abdominal symptoms. Kidney involvement is seen in up to 50% of cases. This is usually mild and self-limiting. Approximately 10% of cases can have serious kidney problems and 1–5% of cases can progress to chronic renal failure. There are often associated systemic symptoms such as fever, joint pains and abdominal pains. The exact aetiology is unknown but many cases start after a viral upper respiratory tract infection (URTI). HSP is more common in the autumn and winter months.

All patients with HSP should have their urine and blood pressure checked. Treatment is usually symptomatic but children should avoid non-steroidal anti-inflammatory drugs if they have renal involvement. The vast majority of cases clear spontaneously over a number of weeks but a small number of patients may relapse a few weeks or few months later. Severe cases with renal involvement may need systemic treatments such as steroids under the care of a nephrologist. Adults who develop HSP may have more severe systemic involvement such as kidney disease.

### 54.4 Meningococcal Septicaemia

The petechial and purpuric rash which is one of the hallmarks of meningococcal septicaemia is caused by a vasculitis as a result of meningococcal endotoxins. Petechiae and purpura, which do not blanch with pressure, occur in 50–75% of cases of meningococcal disease [2]. The rash can progress to large, red or purple lesions and severe cases can cause a haemorrhagic ulcerating necrosis.

By the time the rash appears the patient is usually critically ill and may have signs of shock and meningitis. A delay of even a few hours can be fatal. Early detection and appropriate treatment with intravenous or intramuscular benzypenicillin can be lifesaving and should be carried out even before the patient is transferred to hospital. Meningococcal disease is most prevalent in children from 6 months to 4 years old but can occur at any age group. It can be more common and more severe in patients who are immunosuppressed or who have a non-functioning or non-existing spleen (see Chap. 55).

## 54.5 Urticular Vasculitis

This is more to do with vasculitis and less to do with urticaria. It is a variant of cutaneous small vessel vasculitis. It causes inflamed, red patches or wheals on the skin that can look like urticaria but lasts more than 24 hours in any one area. The rash can be itchy, painful or burning. As the rash fades, it often leaves bruising in the skin which can last a few days or a few weeks (Fig. 54.3). Urticular vasculitis does not usually respond to antihistamines. There may be systemic symptoms such as joint pains in up to 50% of cases. Some patients can have enlarged lymph glands, a fever, abdominal pain or breathlessness which implies systemic involvement of the affected organ with vasculitis.

Diagnosis is usually by skin biopsy which shows evidence of vasculitis. This condition can



**Fig. 54.3** Urticular vasculitis with a rash that lasts more than 24 hours in the one place and causes bruising. Photo courtesy of Dr Myriam Raquel González-Oviedo

**Table 54.4** Causes of urticarial vasculitis

- Autoimmune disease such as SLE
- Viral infections such as hepatitis B, C or infectious mononucleosis (glandular fever)
- Drug induced (e.g. ACE inhibitors, penicillin, sulphonamides, fluoxetine, thiazides,)
- Leukaemia and other internal malignancies
- Idiopathic

be due to a number of factors but in the majority of cases no obvious causes can be found (Table 54.4).

Some patients with urticarial vasculitis have lower levels of complement (hypocomplementemic urticarial vasculitis) and these patients usually have more severe disease that is often resistant to standard treatments. Patients with normal levels of complement often respond to simple treatment such as anti-inflammatories (if there is no renal involvement) or topical steroids. More severe cases of urticarial vasculitis, particularly if it is associated with lower levels of complement may have more systemic involvement such as joint pain, fever, abdominal pain, kidney or lung involvement. These cases may need systemic treatment with various anti-inflammatories such as oral steroids, azathioprine, cyclophosphamide or cyclosporine.

## 54.6 Conclusion

Cutaneous leukocytoclastic vasculitis can present in isolation or be part of a systemic disease. It is very important to exclude internal organ involvement and/or systemic disease in patients with cutaneous vasculitis. If the patient is sick and has a fever they should be assessed urgently.

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# Emergencies in Dermatology

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## Key Points

- When faced with an urgent skin problem in the emergency room or out-of-hours setting the doctor has to decide whether to treat the patient and send them home, refer them back to the GP or the dermatology OPD in the next few days, or arrange urgent admission.
- Patients presenting with an acute generalised rash and systemic symptoms such as a high fever, tachycardia or low blood pressure will need urgent care and attention.
- Other clues that the patient may have a more serious or dangerous skin complaint is the presence of pain rather than itch, blisters, mucosal lesions or a non-blanchable petechial rash.

## What to Tell the Patient

- If you or any of your family develop a rash and feel generally unwell (temperature, rapid heartbeat, weakness, dizziness, nausea, vomiting, off your food, muscle aches and pains, sores in the mouth or nose, etc.) you may need emergency assessment by a doctor.

- Anyone with a fever and a new rash that may resemble bruising on the skin that will not fade on pressure should seek medical attention immediately.

## 55.1 Introduction

True emergencies in dermatology are very rare. Many patients present to their GP, at out-of-hours setting or to the Emergency Department (ED) with rashes or skin problems that they believe require urgent attention. 8% of all ED visits are due to skin complaints [1]. While the majority of these patients do not have genuine emergencies, occasionally a patient is seen in the acute setting with a rash or some skin problem that may progress rapidly and be potentially serious or life threatening (e.g. meningococcal septicaemia, cellulitis or erythema migrans of Lyme disease, anaphylaxis).

## 55.2 Dermatological Emergencies

Some patients present with an acute generalised rash and systemic symptoms such as a high fever, tachycardia or low blood pressure where it is clear that they will require urgent care and attention. Other clues that the patient may have a more serious or dangerous skin complaint are the presence of skin pain rather than an itch, blisters or mucosal lesions. It is more worrying

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**Table 55.1** Distinguishing anaphylaxis from fainting (vasovagal reaction)

	Fainting	Anaphylaxis
Onset	Immediate (1–10 min)	Delayed (5–30 min)
Skin	Pale	Flushed, swelling, urticaria
Respiratory	Normal to deep breaths	Wheeze
Cardiovascular	Bradycardia	Tachycardia
Neurological	Weak, faint or loss of consciousness. Feels better when lying flat	Weak, faint or loss of consciousness. Little response to lying flat.
Gastrointestinal	Nausea/vomiting	Abdominal cramps
Treatment	Lie flat, head down, legs up	Lie flat, head down, legs up <i>Intramuscularly adrenaline 1:1000</i> – Adult = 0.5 ml – Child 6–12 yo = 0.3 ml – Child < 6 yo = 0.15 ml Intravenously fluids—crystalloid

when patients present early with acute symptoms where the potential danger to general health may not be easily recognised (e.g. acute urticaria and bronchospasm which may progress to full blown anaphylaxis) (Table 55.1).

When faced with a skin problem in the emergency setting, the doctor has to decide whether to treat the patient and send them home, refer them to their GP or the dermatology OPD in the next few days, or arrange urgent admission.

Emergencies in dermatology may arise out of an exacerbation of an underlying chronic disease (e.g. erythroderma arising from chronic eczema or psoriasis or eczema herpeticum caused by an infective exacerbation of atopic eczema by the herpes simplex virus). Other skin problems in the emergency setting may develop as a new skin problem that develops rapidly and may be fatal (e.g. an acute drug eruption that may progress to SJS/TEN). Some serious systemic diseases may also present with cutaneous manifestations (e.g. endocarditis presenting with splinter haemorrhages in the nail folds or Henoch–Schönlein purpura (HSP) presenting with a purpuric rash on the legs in children). Trauma from burns, lacerations or bites may also present as an emergency.

Table 55.2 lists some of the more common dermatological presentations to the ED in adults. Many patients present to the out-of-hours setting or the ED with relatively minor or simple skin problems (Table 55.3). These should be triaged by a nurse and referred back to the GP or seen by

**Table 55.2** Potentially life threatening skin problems seen in the ED

- SJS/TEN
- Pemphigus vulgaris
- Severe cutaneous drug eruption
- Anaphylaxis
- Meningococcus septicaemia
- Necrotizing fasciitis
- Eczema herpeticum (disseminated herpes simplex)
- Erythroderma
- DRESS
- Burns

**Table 55.3** More common, less serious skin problems seen in GP out of hours services or the ED

- Eczema/dermatitis
- Scabies
- Contact dermatitis
- Cutaneous drug eruption
- Psoriasis
- BCC
- Erythema multiforme
- Cellulitis
- Shingles
- Urticaria and angioedema
- Lacerations
- Bites

the emergency doctor on call. Table 55.4 lists some of the more common paediatric dermatological emergencies [2].

**Table 55.4** Common paediatric dermatological problems seen in the emergency department.

– Viral exanthema
– Urticaria
– Atopic eczema
– Varicella
– Napkin dermatitis
– Herpetic gingivostomatitis
– Burns
– Lacerations
– Bites

More serious skin problems may need immediate attention or urgent admission. Below are a few conditions, some frequent (burns and bites) and some less common, which most doctors may encounter only once or twice in their career. Nonetheless, it is important to recognise the clinical features of these rare conditions and how to manage these patients appropriately.

### 55.3 Burns

Minor burns to the skin can be managed by immediately placing the burned area under a cold running tap for 15–30 min. Analgesia should be given and medical advice sought. If there are tense fluid filled blisters in a small area, it is best to clean the skin with an antiseptic and puncture the blister with a sterile pin. Empty out the fluid by pressing down on the blister and leave the top of the blister over the burn which will act as a biological dressing. Application of a potent or very potent topical steroid may help reduce the inflammatory response and reduces the chance of more blistering. The wound should be dressed with a non-adherent gauze dressing such as “Bactragras®” (a tulle gras presented as a gauze of leno weave impregnated with white soft paraffin BP containing 0.5% chlorhexidine acetate BP) and covered with sterile gauze. The area should be rested and elevated.

Depending on depth, site and size of the burn further specialist advice or admission may be required. Careful follow up is required for more dressings and to insure the area does not get

infected or develop other complications such as scarring or contractures (see Chap. 38).

### 55.4 Bites

**Insect bites** are usually harmless and self-limiting but can be quite uncomfortable and persistent in some individuals. Symptomatic treatment can be achieved with new generation antihistamine and the application of a soothing insect bite cream from the pharmacist. A potent topical steroid applied at the earliest possible stage may help if the person is prone to more persistent, severe allergic reactions to insect bites. Medical advice may be required if there is a risk of anaphylaxis, Lyme's disease, or wound infection after the bite (see Chap. 35). Insect bites while travelling overseas may cause various tropical illnesses such as malaria, yellow fever or dengue fever. Insect repellents and appropriate clothing may help prevent bites in those who react more severely.

**Animal bites** may look relatively harmless, but often can become infected. Minor bites should be cleaned thoroughly under local anaesthetic if necessary and should not be sutured if possible. Large deep bites should be referred for a plastic surgery opinion. Prophylactic antibiotic may be required such as Amoxicillin/clavulanic acid (co-amoxiclav) or for those who are allergic to penicillin consider doxycycline plus metronidazole, oxytetracycline plus metronidazole, or clindamycin plus ciprofloxacin (Table 55.5). Specialist opinion may be required for bites on the hands, face or scalp and in patients who are

**Table 55.5** Prophylactic antibiotics should be considered in the following situations [3]

- All cat bites
- All human bites under 72 hours old
- All dog bites to the face, hand or foot, genitals or those that require surgical debridement
- All bites which affect underlying structures such as tendons, ligaments, joints or bones
- Wounds that have undergone primary closure
- People at increased risk of infection. (e.g. diabetes, immunosuppression, prosthetic joints, etc.)

immunosuppressed. It is important to know what type of animal has bitten the patient as different species pose different problems (venom injection from snakes and spiders, bite from a domestic or wild dog). If the bite is deep, there is the risk of overgrowth of anaerobic bacteria originally present in the mouth of the animal and the wound may need surgical debridement. Tetanus prophylaxis should also be considered. If the person has been bitten in a country where rabies is endemic, rabies postexposure prophylaxis (PEP) consisting of a dose of human rabies immune globulin (HRIG) and rabies vaccine may have to be given on the day of the rabies exposure, and then a dose of vaccine given again on days 3, 7, and 14.

**Human bites** usually arise as a result of fighting, sports or from small children. The management is similar to animal bites (Table 55.5) except blood born infections such as HIV, hepatitis B and C should be considered. Post-exposure prophylaxis may need to be considered with hepatitis B immunoglobulin, hepatitis B vaccine and antiretroviral drugs if there is a risk of HIV infection.

## 55.5 Meningococcal Disease

Meningococcal meningitis and septicaemia usually presents as an acutely sick child with a high fever. Some children may have little or no fever especially if they have been given antipyretics recently, if they are seen very early in the course of the disease or if they are in shock. 50–70% of patients will develop a petechial rash (1–2 mm red or purple spots from leaking capillaries) that will not blanch on pressure. The rash may progress to bruising or even frank necrosis in extreme cases. Meningococcal disease can progress rapidly and be fatal so early recognition, diagnosis and treatment is paramount (see Chap. 27).

A rapidly evolving petechial or purpuric rash (haemorrhage into the skin causing purple or brownish-red spots which can resemble bruising) is a marker of very severe disease requiring urgent assessment and treatment. A non-blanching, haemorrhagic rash is characteristic of meningococcal disease but the rash is seldom an early sign,

and the underlying disease may be advanced by the time a rash appears. In meningococcal disease, the rash may be absent, scanty, or it may be blanching in the early stages and can sometimes be confused with insect bites but it nearly always develops into a non-blanching red, purple or brownish petechial rash or purpura eventually [4].

Isolated pin-prick spots may appear where the rash is mainly maculopapular, so it is important to search the whole body for small petechiae, especially in a febrile child with no focal cause. The rash can be more difficult to see on dark skin, but may be visible in paler areas, especially the soles of the feet, palms of the hands, abdomen, or on the conjunctivae or palate. Purpuric areas that look like bruises can be confused with injury or abuse.

It is very important to examine children for the signs of meningitis or septicaemia and investigate and treat if necessary based on those findings even in the absence of a rash. Although some of the causes of petechial rashes are self-limiting conditions, many others, including meningococcal disease can be life-threatening and a non-blanching rash should be treated as an emergency. Treatment of a suspected case of meningococcal disease should be initiated at the earliest opportunity and prior of hospital transfer if possible.

GPs should carry benzoylpenicillin and inject it unless there is a history of anaphylaxis after previous penicillin administration. It should be administered intravenously, where possible. Otherwise, it can be given intramuscularly but as proximally as possible, into a part of the limb which is still warm (the cold area being more poorly perfused) (Table 55.6) [5].

## 55.6 Acute Generalized Pustular Psoriasis (AGPP)

The patient is usually unwell with chills, aches and pains and nausea. They can develop a sudden worsening of their chronic psoriasis over a few

**Table 55.6** Benzoylpenicillin dose for emergency treatment of suspected cases of meningococcal disease (IM or IV)

Adult and child aged 10 or older	1200 mg
Child 1–9 years	600 mg
Infant	300 mg

**Table 55.7** Triggers for acute generalized pustular psoriasis

- Irritation from topical treatments such as tar or dithranol
- Infection
- Pregnancy
- Hypocalcemia
- Drugs—e.g. salicylates, lithium, terbinafine, etc.
- Sudden withdrawal of potent topical or oral steroids

days or weeks with an extensive rash and pustules. Various factors can trigger AGPP (Table 55.7). The patient will usually require admission for stabilization. Treatment is usually initially with bland emollients and supportive care. Most patients will respond to gradual introduction of acitretin (an oral retinoid), cyclosporine, methotrexate or some of the newer biological agents.

## 55.7 Erythroderma

This can be caused by a number of conditions that result in extensive redness and scaling covering 90% of the skin surface area. It can occur as a primary skin disease or secondary to an underlying skin problem such as atopic eczema, psoriasis, contact dermatitis, pityriases rubra pectoris, cutaneous T cell lymphoma (Mycosis fungoides), leukaemia, or from various medications. It is potentially life threatening and requires admission for correction of any electrolyte imbalances, temperature control, and investigation and treatment of the underlying cause.

## 55.8 Erythema Multiforme and Stevens–Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN)

These are often triggered by drugs or infection (Table 55.8). **Erythema multiforme minor** presents with classical target lesions in a patient who is systemically quite well with minimal or no mucosal involvement. Prodromal symptoms are usually absent or mild in persons with erythema multiforme minor, consisting of a mild,

**Table 55.8** Drugs that can cause SJS/TEN include the following

- Allopurinol
- Abacavir (for HIV treatment)
- Sulfa drugs (e.g. sulphonamides)
- Aromatic anti-epileptics
- NSAIDs
- Sulphonamides
- Trimethoprim
- Terbinafine
- Antibiotics including cephalosporins, quinolones
- Aminopenicillins
- Tetracyclines
- Macrolides
- Imidazole antifungal (e.g. fluconazole, itraconazole)
- Anticonvulsants (e.g. Carbamazepine)

nonspecific upper respiratory tract infection. The non itchy papules evolve into pathognomonic target or iris lesions (see Chap. 23, Photo 23.5) that appear within a 72-hour period and begin on the extremities. Lesions remain in a fixed location for at least 7 days and then begin to heal. Recent or recurrent herpes simplex infection has been reported as the principle risk factor for erythema multiforme minor. Erythema multiforme is rare in children younger than 3 years and adults older than 50 years, and is most common in males age 20–49 years.

**Erythema multiforme major** is a more severe, potentially life-threatening disorder with mucous membrane involvement (eyes, mouth and genitalia) and up to 10% of body surface area may have epidermal detachment but most cases heal within 10–14 days. There is usually a negative Nikolsky's sign. In erythema multiforme major, 50% of patients have an influenza-like illness for 1–14 days before the rash appears, including fever, general aches and pains, cough, sore throat, vomiting, diarrhoea or chest pain. More than 50% of all cases of **Erythema multiforme major** are caused by medications (Table 55.8).

Systemic corticosteroids should be avoided in minor cases. In severe cases, their use is controversial, because these agents do not improve prognosis and may increase risk for complications. Suppression of HSV can prevent HSV-associated erythema multiforme, but antiviral

treatment started after the eruption of erythema multiforme has no effect on the course of erythema multiforme [6].

**Steven-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN)** are considered severity variants of a single entity.

The clinical descriptions are as follows:

- **SJS/TEN**—Widespread blisters predominant on the trunk and face, presenting with erythematous or pruritic macules and one or more mucous membrane erosions; epidermal detachment is less than 10% TBSA for Steven-Johnson syndrome and 30% or more for toxic epidermal necrolysis [6].

More severe forms of SJS/TEN present with a generalised rash in a very sick patient. Approximately 50% of cases are idiopathic, with no precipitating factor identified. There is often a positive Nikolsky's sign (skin that tears and blisters easily on light lateral pressure). Table 55.9 shows the differential diagnosis of SJS/TEN (see also Chap. 23).

The treatment of SJS/TEN involves withdrawing the suspected drug that may be causing the rash and/or dealing with any underlying infections. Patients are usually admitted for supportive care in ICU or a burns unit. Some patients may require intravenous immunoglobulins. Most authors believe that systemic steroids are of unproven benefit in early forms of SJS/TEN.

**Table 55.9** Differential diagnosis for SJS/TEN

– Acute graft versus host disease
– Linear IgA
– Paraneoplastic disease
– Pemphigus
– Pemphigoid
– AGEP—Acute Generalised Exanthematous Pustulosis (usually drug induced)

TEN and are harmful in advanced disease (see Chap. 23).

## 55.9 Drug Rash with Eosinophilia and Systemic Symptoms Syndrome (DRESS)

**DRESS** is a severe drug reaction that causes a wide variety of clinical symptoms, anywhere from 2 to 8 weeks after initiating the offending drug. It usually presents with a fever, rash, lymphadenopathy, eosinophilia and abnormal liver function tests. The cutaneous manifestations can include urticarial, maculopapular eruption and, in some instances, vesicles, bullae, pustules, purpura, target lesions, facial edema, cheilitis, and erythroderma. Visceral involvement (hepatitis, pneumonitis, myocarditis, pericarditis, nephritis, and colitis) is the major cause of morbidity and mortality in this syndrome. Mortality is estimated to be around 10%. Antiepileptic medications such as phenytoin and phenobarbitone, antibiotics (particularly beta-lactams), allopurinol, non-steroidal anti-inflammatory drugs, captopril, mood stabilisers, and antiretrovirals are some of the most common cause of DRESS syndrome. This syndrome must be recognized promptly and the causative drug withdrawn as the earlier the drug withdrawal, the better the prognosis. Treatment is largely supportive and symptomatic; corticosteroids are often used, but the evidence regarding their effectiveness is poor [7].

## 55.10 Necrotizing Fasciitis (NF)

This is a life threatening, rapidly progressing soft tissue infection primarily involving the superficial fascia. The infection tracks along the fascial planes (sheaths of tissue covering muscles) caus-

ing thrombosis and necrosis of the skin, subcutaneous fat and even muscle ("the flesh eating disease"). Early diagnosis and aggressive surgical debridement are essential to save lives. Mortality rate can run between 20 and 40% in adults and even higher in neonates [8].

NF usually starts with some minor trauma such as a needle puncture, insect bite, laceration or surgical wound. Some patients may have underlying conditions that make them more susceptible to infection such as advanced age, chronic renal failure, peripheral vascular disease, diabetes, myxoedema or immunosuppression (e.g. HIV, patients on chemotherapy or with leukaemia). NF is also more common in intravenous drug abusers.

Many organisms can be implicated but group A streptococcus is the most common. A smaller number of cases can be caused by *Staphylococcus aureus* and some cases can be caused by community associated methicillin-resistant *Staphylococcus aureus* (MRSA).

Most patients present with features of cellulitis, fever and severe pain. The infection spreads rapidly and is most commonly found in the extremities, perineum or trunk. In many cases, the pain and fever are severe and disproportionate to the local findings on the skin. As the infection progresses, the skin colour changes from a red-purple to a dusky blue. Bullae may develop and frank cutaneous gangrene may occur. By this stage the rash may no longer be tender owing to destruction of superficial nerves and crepitus may be felt in the area.

Treatment is with broad spectrum intravenous antibiotics until the causative organism is isolated by culture and sensitivity of the blood, swabs and tissue. Surgical debridement and supportive care is paramount. Amputation is occasionally required. Intravenous immunoglobulins and hyperbaric oxygen may benefit some patients. Even in the best of centres, mortality can be up to 40% and is higher in those aged over 50 years, patients with diabetes, malnutrition, hypertension, intravenous drug abusers and neonates. Death is usually caused

by sepsis, multi-system organ failure or invasion of major vessels. Early operative debridement is associated with lower mortality.

## 55.11 Conclusion

Most emergencies in dermatology are due to trauma, infectious diseases (bacterial or viral), an exacerbation of an underlying inflammatory skin disease or drug eruptions. There may be a history of pre-existing skin disease that has suddenly flared up (e.g. psoriasis or atopic eczema) or the emergency may be as a result of a brand new skin problem. If the patient has an extensive rash and systemic symptoms (fever, tachycardia, low blood pressure, nausea, anorexia, arthralgia, mucosal involvement, etc.) they may need emergency admission for investigation, stabilization with intravenous fluids, temperature control and bland emollients. Specific treatment may be required for the underlying cause.

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**Part XII**

**Surgical Therapies**



# Local Anaesthesia for Skin Surgery

56

David Buckley

## Key Points

- Always lie the patient down flat before and after giving local anaesthesia (LA).
- Use the smallest needle possible (30G) when giving LA.
- Inject very slowly and subcutaneously, rather than intradermally, to reduce pain.
- Leave sufficient time for the LA to act before starting your procedure.
- Although allergic reactions to LA are very rare, always have resuscitation equipment available.

## What to Tell the Patient

- LA will make your procedure painless and when given with a 30G needle it causes very little discomfort.
- When the LA wears off you may need to take a pain killer by mouth, such as paracetamol or ibuprofen.

## 56.1 Introduction

Adequate **analgesia** is required for most surgical procedures including cryosurgery and punch

biopsy. This will make the procedure more successful, more comfortable for the patient and less stressful for the doctor [1, 2]. It is very difficult to operate on a patient who is wriggling in pain.

## 56.2 Topical Anaesthetic

While topical anaesthetic such as “EMLA<sup>®</sup>”, “Amitop<sup>®</sup>”, “LMX4<sup>®</sup>” or “Pigalis<sup>®</sup>” can be helpful for small procedures on their own, they are most useful in children and needle phobics, to allow painless insertion of a local anaesthetic needle. They need to be applied under occlusion for 30 min to 2 hours before the procedure. (Table 56.1). A “Dermajet<sup>®</sup>” is another way of instantly anaesthetising a small area, approximately 5 mm in diameter, by injecting a local anaesthetic via a high pressure air-gun without the need for needles. This area can then be used for painless needle insertion to deliver local anaesthetic. A “Dermajet<sup>®</sup>” can also be used for intradermal injection of steroids in conditions such as alopecia areata.

## 56.3 Local Infiltration of Anaesthesia

When injecting local anaesthetic, the smallest needle that is practical should be used. A 30-gauge needle is relatively painless when it pene-

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**Table 56.1** Topical anaesthesia

Name	Constituents	Indications	Special precautions	Onset (minutes)	Maximum dose
“EMLA®” 5% cream	Lidocaine 2.5% and Prilocaine 2.5%	Injections and dermatology procedures	Can be used on genital mucosa and leg ulcers	60–120	Infants < 2 months: 1 g 3–11 months old: 2 g 1–5 years old: 10 g 6–11 years old: 20 g >12 years + adults: 60 g
“LMX4®”	Lidocaine 4%	Injections and dermatology procedures	Not for mucous membranes or broken skin	30–60	Infants < 1 months: <b>do not use</b> 1–12 months old: 1 g 1–11 years old: 10–20 g >12 years + adults: 60 g
“Ametop®” 4% cream	Tetracaine 4%	For venepuncture + IV cannulation	Not for mucous membranes	30–45	30 g in adults + children > 5 years
“Pliaglis®”	Lidocaine 7% Tetracaine 7%	Injections + dermatology procedures	Not for mucous membranes or children	30–60	60 g in adults

brates the skin, particularly if pressure is applied with a thumbnail for 5 s before inserting the needle. This is the concept behind the Melzack and Wall's gate control theory of pain. This theory asserts that activation of large diameter nerve fibres which transmit touch, pressure and vibration can interfere with signals from the smaller diameter pain fibres, so that the sensation of pressure can reduce pain. If necessary, the initial local anaesthetic infiltration can be carried out using a 30-gauge needle and then a longer, large-bore needle (27- or 25-gauge) can be painlessly inserted into the same site as the initial injection, for deeper infiltration (Table 56.2).

While there are many types of LA available, for a vast majority of situations, 1% lidocaine (“Xylocaine®”), with or without adrenaline is safe and effective. The maximum dose allowed without adrenaline is 3 mg/kg and with adrenaline it is 7 mg/kg (1% “Xylocaine®” contains 10 mg/1 ml) and so 20 ml of 1% “Xylocaine®”

**Table 56.2** Ways of reducing pain when giving local anaesthetic Local anaesthesia (LA), for skin surgery

- Relax and reassure patient (Pre med)
- Lie down
- Consider topical anaesthesia
- Consider a “Dermajet®” to numb the skin before inserting the needle
- LA at room temp + without Adrenaline
- If using adrenaline = buffer with sodium bicarbonate
- Start with the smallest needle possible (e.g. 30 g)
- Pressure on puncture site before inserting the needle
- Ethyl chloride or light Cryo × 3 seconds.
- Distract the patient
- Inject slowly + subcutaneously + start proximally
- Consider regional anaesthesia (ring block, post tib. block)
- Wait till fully anaesthetized (3–5 min)

plain or 50 ml of 1% “Xylocaine®” with adrenaline can be used in a 70 kg adult (Table 56.3). There is no great advantage to stocking 2% or 0.5% lidocaine and it is safer and easier to calcu-

**Table 56.3** Local anaesthetic for local infiltration or peripheral nerve block

Name	Description	Onset (min)	Duration (h)	Strength (mg/ml)	Max dose (mg/kg)	Max in 70 kg child (approx. 3 year old) (ml)	Max in 14 kg child (approx. 6 year old) (ml)	Max in 22 kg child (approx. 6 year old) (ml)
Lidocaine 1% ("Xylocaine ®")	Amide	2–10	0.5–2	10	3	21	4	7
Lidocaine 1% with adrenaline (epinephrine) 1:200,000	Amide	2–10	2–4	10	7	49	10	15
Bupivacaine <sup>a</sup> (0.25%) ("Marcaine ®")	Amide for local infiltration	10–15	3–12	2.5	2	56	a	a
Bupivacaine <sup>a</sup> (0.5%)	Amide for peripheral nerve block	10–30	5–15	5	1	14	a	a

<sup>a</sup>Generally not recommended in children less than 12 years old

late the safe dose if only 1% is stocked. It is advisable to buy the lidocaine with adrenaline and lidocaine without adrenaline in different types of vials and store them in different locations to avoid errors. It is also safer to draw up LA in only 2 or 5 ml syringes to ensure you do not give too much, especially when injecting children.

Local anaesthetic solution injected at body temperature produces significantly less pain than local anaesthetic injected which has been cooled in the fridge.

Injecting very slowly and subcutaneously rather than intradermally will also reduce pain. Start injecting the area of the lesion that is closest to the origin of the sensory nerve supplying that site (e.g. proximal before distal injection). Regional nerve blocks such as a ring block, a posterior tibial block or an infraorbital block are often less painful than local infiltration in certain areas.

When adrenaline is used in an LA, sodium metabisulphite or bisulphite is added by the manufacturers to prevent adrenaline oxidation. These chemicals increase the acidity of the LA and make the injection more painful. Adding 1 ml of 8.4% sodium bicarbonate for every 10 ml of lidocaine with adrenaline immediately prior to use

**Table 56.4** Alkalisation of local anaesthetic solutions

Anaesthetic solution	Volume of 8.4% sodium bicarbonate to be added to 20 mL
Lidocaine 1 or 2% + adrenaline	2 mL
Bupivacaine 0.25 or 0.5% + Adrenaline	0.1 mL <sup>a</sup>

<sup>a</sup>The small volume of 8.4% sodium bicarbonate to be added requires great care as adding too much will cause precipitation. Discard any unused buffered LA immediately after use as it will not store

will raise the pH and make the administration of the local anaesthetic less painful. This is called 'alkalinisation' or 'buffering' of the solution. Sodium bicarbonate also shortens the onset and prolongs the intensity and the duration of the block. It also shortens the shelf life of the LA and should only be added immediately prior to use and any unused mixed solution should be discarded immediately (Table 56.4).

The maximum amount of lidocaine with adrenaline should be reduced in patients with severe ischaemic heart disease, arrhythmias, thyrotoxicosis, unstable hypertension, uncontrolled diabetes, epilepsy and porphyria. According to the Summary of Product Characteristics, lidocaine should be used with caution with certain drugs

such as cimetidine, betablockers, monoamine-oxidase inhibitors, tricyclic antidepressants, phenothiazines and butyrophenones.

The onset of action from 1% lidocaine with or without adrenaline is usually approximately 3–5 min and is up to 10 min when using a ring block anaesthetic. Adding adrenaline considerably prolongs the duration of action (up to 7 h) and will reduce bleeding. Local anaesthetic with adrenaline should be used with caution in fingers, toes, the penis, nasal tip and ear lobes and for ring block anaesthesia, especially in patients with PVD or diabetes. The effectiveness of LA may be reduced and the absorption increased when injecting into infected or inflamed areas. Although injecting intradermally is more painful, the onset of action is far more rapid.

LA can be used to hydrodissect the skin from the cyst wall when preparing to remove a sebaceous cyst or to raise the skin off superficial fascia to protect vital structures in facial work.

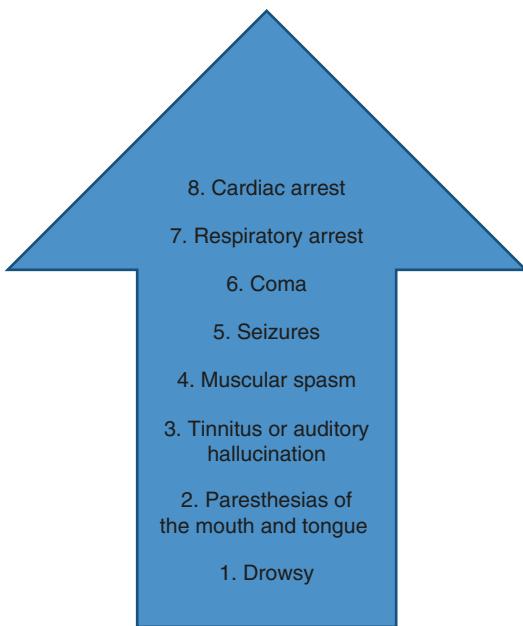
Vasoconstriction after infiltration of the local anaesthetic is common as a result of the pressure effect on the adjacent blood vessels and the vasoconstriction effect of adrenaline. This may make the outline of the lesion or rash to be biopsied or removed more difficult to see. For this reason, it is advisable to mark out the outline of the lesion and the safe margins for excision or biopsy after cleaning the skin thoroughly and before infiltrating with local anaesthetic.

#### 56.4 Toxicity, Anaphylaxis and Fainting

Local anaesthetic is very safe. However, there is a very small risk of toxicity (Table 56.5 and Fig. 56.1) or anaphylaxis to amide anaesthetics or some of the additives, so full resuscitation equipment should be on hand at all times. Always aspirate before injecting to ensure the LA is not injected into a vein or artery. A more likely reaction is a vasovagal syncope (fainting) episode as a result of the injection, especially in children

**Table 56.5** Signs and symptoms of systemic lidocaine toxicity include

- Severe numbness or tingling especially around the mouth
- Dizziness and drowsiness
- Tinnitus (ringing in the ears)
- Slurred speech
- Metallic taste in mouth
- Mental status change or loss of consciousness
- Muscle twitching
- Seizures
- Arrhythmia (tachycardia or bradycardia)
- Coma
- Respiratory arrest
- Cardiac arrest



**Fig. 56.1** Progression of local anesthetic toxicity

and young adults. Always lie the patient down before infiltrating with local anaesthetic and performing surgery and if a patient feels faint they should be placed on a couch with their head down and feet up for at least 10 min. Always consider LA toxicity if the patient becomes unwell following procedures under LA (Table 56.6).

**Table 56.6** Distinguishing anaphylaxis from fainting (vasovagal reaction) and lidocaine (“Xylocaine®”) toxicity

	Fainting	Anaphylaxis	Lidocaine toxicity
Onset	Immediate (1–10 min)	Delayed (5–30 min)	Delayed (10 min–2 h)
Skin	Pale	Flushed, swelling urticaria	—
Respiratory	Normal to deep breaths	Wheeze	Slow respirations
Cardiovascular	Bradycardia	Tachycardia	Arrhythmias
Neurological	Weak, faint or loss of consciousness. Feels better when lying flat	Weak, faint or loss of consciousness. Little response to lying flat.	<ul style="list-style-type: none"> <li>— Tingling lips</li> <li>— Tinnitus</li> <li>— Blurred vision</li> <li>— Slurred speech</li> <li>— Muscle twitching</li> <li>— Seizures</li> </ul>
Treatment	Lie flat, head down, legs up	Lie flat, head down, legs up IV fluids—crystalloid <i>IM adrenaline 1:1000</i> — Adult = 0.5 ml — Child 6–12 yo = 0.3 ml — Child < 6 yo = 0.15 ml	<ul style="list-style-type: none"> <li>— Lie flat</li> <li>— I.V. fluids—crystalloids</li> <li>— I.V. Diazepam for seizures</li> <li>— Consider I.V. lipid emulsion 20%</li> </ul>

## 56.5 Conclusion

Topical or local anaesthetic are vital when investigating and/or treating some skin conditions. It ensures the patient is comfortable and relaxed which will make the procedure more tolerable and successful for everyone concerned. However, giving an injection of local anaesthetic can itself be painful and the doctor or nurse should use the correct product, the smallest needle possible and a perfect technique when injection local anaes-

thetic. In addition, great care needs to be taken to avoid complications from giving a local anaesthetic, from fainting to an allergic reaction.

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# Simple Skin Surgery

57

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## 57.1 Key Points

- Some conditions may need surgical intervention for either diagnosis, treatment or both.
- Most skin surgery errors are made with the pen, not the knife. Carefully patient and lesion selection plus marking the lesion and borders with ink before surgery is very important.
- Simple techniques such as shave, punch or snip biopsy and curettage are easy to carry out in general practice.
- All tissue removed from the skin should be sent for histology.
- Disposable instruments are the easiest way to ensure a safe supply of instruments.

## 57.2 What to Tell the Patient

- Simple surgical procedures in primary care are safe, convenient and a lot less expensive than when carried out in hospital.
- Side effects are rare but bleeding and infection can sometimes occur.
- Please do not drive immediately after a surgical procedure even if performed under local anaesthetic.
- You will need to sign a consent form.

## 57.3 Introduction

While many skin problems seen in primary care can be easily diagnosed clinically and managed with simple topical or oral therapies, some conditions may need surgical intervention for either diagnosis, treatment or both.

Important attributes for any surgeon are competent clinical judgement as well as the technical skills for operating. Once you have started an operation and made your first incision there is no going back. So give careful consideration to the following points:

- Do I know what the lesion is?
- Do I need to operate?
- Am I experienced enough to do it?
- Do I have the right equipment?
- Have I got enough help?
- Have I got enough time?

Some surgical techniques such as punch biopsy or cryosurgery can be carried out easily in primary care. Patients and staff safety is paramount and a doctor must have a way of insuring that all required instruments are properly decontaminated either by only having disposable

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instruments or having an effective decontamination protocol.

## 57.4 Instruments

The easiest way to ensure a reliable source of safe, sound instruments is by using disposable instruments. While these might seem expensive initially, they are probably more cost effective, given the time and equipment that is required to decontaminate reusable surgical instruments in primary care. The quality and range of instruments available when using disposables is not as good as re-usable instruments but disposables are adequate for small procedures such as biopsies. Another option for decontamination of reusable surgical instruments is to get them decontaminated in a *Central Sterile Supply Department* (CSSD) in the local hospital if that facility is available. For in-house decontamination in a GP surgery the minimum standard is a safe method of cleansing the instruments (e.g. a washer/disinfector) and a class B autoclave.

It is vital to ensure that all doctors, nurses, administration staff, and cleaners are vaccinated against **Hepatitis B** and are aware of the practice guidelines for needle stick injuries, if surgery is to be carried out in the practice (Table 57.1).

Other pre-requisites for simple skin surgery is a clean procedure room, good lighting and magnification, protected time and an assistant, if possible. Consent and proper surgical record keeping is vital for medico-legal reasons (See sample consent form, Chap. 66).

**Table 57.1** Essential documents required if carrying out surgery in a practice

- Health and Safety Statement
- Hepatitis B vaccination status of all personnel working in the surgery
- Guidelines for needle stick injuries
- Guidelines for decontamination of reusable surgical instruments
- Hand hygiene protocol
- Significant event analysis record
- Infection control policy
- Guidelines on anticoagulants

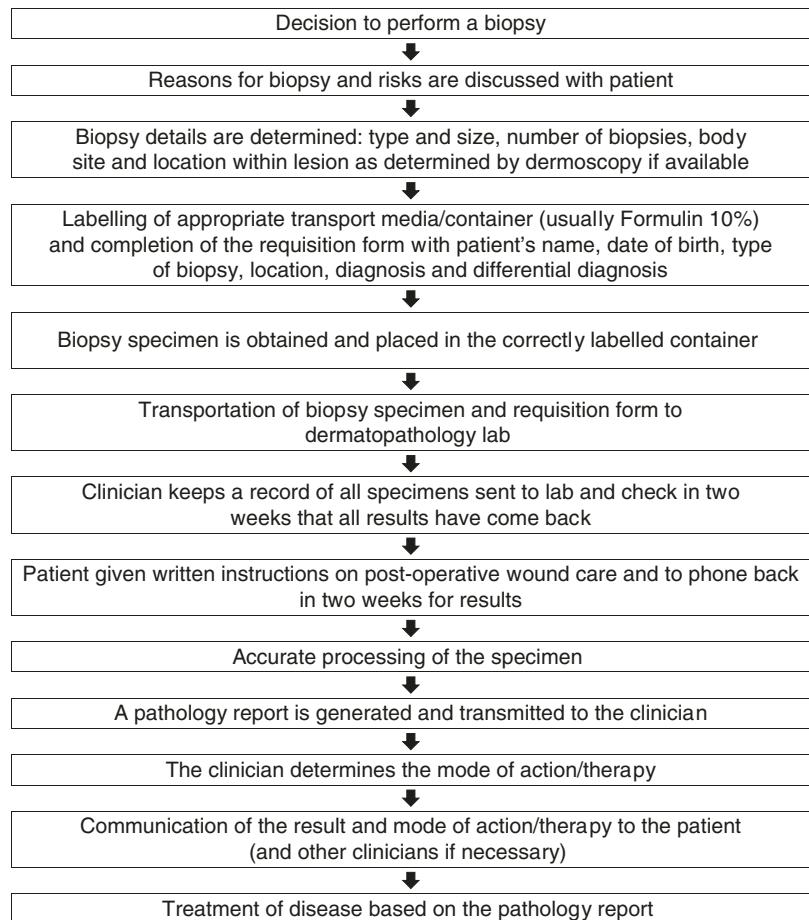
## 57.5 Histology

All tissue removed from the skin should be sent for histology. Specimen should be placed in 10% formalin and the bottle should be labelled in handwriting with the patient's name, date of birth, location of the specimen and type of biopsy taken.

It is important to give the pathologist as much information as possible when sending histology. The minimum required is the name, address, date of birth and sex of the patient. The size of the lesion, the location of the biopsy site and the type of biopsy taken (punch, shave, incision, excision, curettage, etc.) are also important to include in the form. A brief history of the lesion or rash with a clinical diagnosis and differential diagnosis is important to convey to the pathologist. If you cannot make a reasonable assessment of what the rash or lesion might be on clinical grounds alone, it may be better to refer the patient to a colleague with more experience in dermatology who can give the pathologist a reasonable differential diagnosis to work from. Relevant past medical history (e.g. past history of skin cancer) and previous histology reports should be included if available.

If possible all skin specimens should be sent to a pathologist with an interest in dermatopathology. Tumors are easier to interpret than rashes. Vague, non specific rashes may not be the best ones to biopsy as the histological report may also be vague and non-specific. On the other hand, certain rashes have quite characteristic histological features and the pathologist should be able to confirm the diagnosis if the clinician asks if the biopsy shows features of a specific condition (e.g. lupus erythematosus, lichen planus, dermatitis herpetiformis, vasculitis, fungal infection of the skin, etc.). Histopathology may help make a diagnosis such as eczema or dermatitis but may not be able to distinguish the exact type (atopic, allergic contact, irritant, etc.) or the cause. Histopathologists may sometimes find it difficult to differentiate between different skin tumours such as Bowen's disease from SCC or a dysplastic nevus from a melanoma in situ. Getting a second histological opinion can be useful in this situation.

There must be a robust method of tracking histology to ensure that the results come back in a

**Table 57.2** The skin biopsy pathway

\*Ref: Adapted from JAAD 2016, 74: 19-25

timely fashion and the patient is informed of the results and advised if any further treatment is necessary (Table 57.2).

Precise measurements of this size and location of the lesion, with drawings or diagrams if possible, should be taken prior to surgery. A photo is even better at recording the location and size of the lesion in case further treatment or referral is required.

## 57.6 Bleeding and Infection

All surgery carries risks. The most common is bleeding and infection. Bleeding is more common if the patient has a coagulopathy (rare) or is on anticoagulants (common) (Fig. 57.1). If possible, anticoagulants should be stopped before



**Fig. 57.1** Large haematoma in the breast post punch biopsy in a patient on aspirin and dipyridamole

surgery but the surgeon may have consulted with the doctor who prescribed the anticoagulants to ensure that it is safe to stop (Table 57.3 and Chap. 66 PIL on “Anticoagulants and surgery”). Patients on warfarin can have low risk cutaneous

**Table 57.3** PCSA Guidelines on anticoagulants and cutaneous surgery in the community

Risks to patient from stopping anticoagulation or antiplatelet drugs include stroke, DVT/PE, MI, or even death. The risk of skin surgery bleeding may be inconvenient, can result in a haematoma or an abscess and may jeopardise a flap or graft but is rarely serious or life threatening

#### **Stopping anticoagulants that have been prescribed for strong clinical indications is therefore rarely justifiable**

Meticulous operative technique is always required to minimise the risk of bleeding with cutaneous surgery but, even so, bleeding problems can occur. Excessive bleeding during surgery usually responds to electrosurgery or vessel tying, followed by a pressure dressing and patient rest and elevation of the operative site where possible. However, some agents can cause prolonged oozing after the local anaesthetic (LA) wears off, or for several days post-op, even if excellent haemostasis is achieved intra-operatively. For this reason, reducing this risk by postponing surgery, altering the choice of procedure or repair, or sometimes withholding medications may be prudent. The use of LA without adrenaline may be considered so as to avoid delayed bleeding problems

#### **General tips to reduce risk of bleeding**

- Consider postponing surgery until risk factor(s) can be removed or optimised
- Choose safer procedure type if possible or alternative if sufficiently effective e.g. radiotherapy, cryosurgery or non-surgical options such as Aldara or Efudix.
- Use meticulous surgical techniques and consider electrical surgery, radiosurgery, aluminium chloride or tranexamic acid for injection (“Cyklokapron®” applied topically on a gauze to control bleeding).
- For open wounds consider an alginate dressing to adsorb exudates which may aid haemostasis
- Elevate and compress post-op
- Consider referral to a more experienced operator
- Consider admitting the patient overnight post-surgery

#### **Risk factors for significant post-operative bleeding events**

- Previous post-op bleeding episode
- Unable or unwilling to rest post-op
- Bleeding tendency (anticoagulants, Haemophilia, low platelets  $<50 \times 10^9/l$ , herbal medicines)
- Age  $>65$

#### **Risk Stratification of Community Surgery Procedures**

##### *Low Risk Procedures*

- Curettage
- Punch biopsy
- Incisional biopsy – scar length  $<10$  cm
- Excision and direct closure on trunk, limbs or compressible head and neck sites (scar length  $<10$  cm)
- Joint injection
- Cryosurgery

##### *Moderate Risk Procedures*

- Excision and direct closure on non-compressible areas (neck, lip, genitals)
- Wide excision and direct closure on trunk and limbs
- Secondary intention wounds on compressible sites
- Grafts on compressible sites (and split thickness graft donor sites)
- Small local flaps(e.g. rhombic on nose, or wedge or helical rim advancement on ear)
- Ingrown toenails

##### *High Risk Procedures*

- Secondary intention wounds on non-compressible sites
- Excision within the orbit (e.g. eyelids)
- Where bone is involved
- Local flaps on head and neck with wide undermining (e.g. forehead, periocular – especially orbital, cheek, large nose flaps, neck)
- Local interpolated flaps (e.g. paramedian forehead flap)
- Wide excision and direct closure on non-compressible sites (e.g. neck)
- Grafts on non-compressible sites
- Vasectomy

Some high bleeding risk procedures could potentially be avoided altogether by choosing alternative methods of treatment such as topical therapies (e.g. Aldara, Efudix etc.), cryosurgery or deep X-ray therapy (DXT) in patients who are at increased risk of bleeding

**Table 57.3** (continued)**It is recommended that most anti-thrombotic agents are continued for most skin surgery procedures**

This is based on the evidence of a very low risk of morbidity and mortality from peri-operative bleeding, versus a variable risk of highly morbid or fatal thrombotic events associated with cessation of anticoagulants

**Specific points about bleeding agents or tendencies:****Aspirin**

**Aspirin** (ASA) is often taken by patients without clear indication (e.g. post-MI, post-CVA or post-stenting or CABG) in which case it can be stopped 10–14 days before surgery. Otherwise unlikely to cause significant bleeding problems in isolation at 75–300 mg od. If considering medium or high risk skin surgery stop 10–14 days pre-op for full reversal, 5 days for 50% efficacy. Restart 7 days post-op.

**Clopidogrel (“Plavix®”) and prasugrel (“Efient®”)**

These are antiplatelet drugs that can cause prolonged oozing. For clopidogrel, postpone surgery until off drug if possible e.g. sometimes used for 1 year post-percutaneous coronary intervention (PCI). If high bleeding risk procedure ask the prescriber's advice regarding the risk of stopping clopidogrel for 7 days pre-op and if a substitute alternative drug should be used while off the drug. Restart 7 days post-op. Similar rules for prasugrel (“Efient®”)—stop 7 days pre op (stop 9 days if you want complete reversal)

**Warfarin**

Stopping or avoiding not usually justified. Check INR 72 hours pre-op. The bleeding risk is very small if the INR is <3.5 for low risk procedures. For medium or high risk procedures aim for an INR of 2–2.5 if the therapeutic range allows for the indication the patient is taking warfarin. Take advice from the patients GP or haematology if INR reduction needed

**NSAIDs:**

Stop 3 days pre-op and for 7 days post-op if possible for moderate and high risk procedures

**Dipyridamole (“Persantin®”)**

No need to stop for cutaneous surgery if used alone. If taken with other anticoagulants stop 48 hours before medium or high risk surgery. Restart 7 days post op.

**Novel oral anticoagulants / Direct acting oral anticoagulants (NOACs/DOACS):** E.g.: apixaban (“Eliquis®”, 2.5–5 mg BD), dabigatran (“Pradaxa®”, 15 mg BD), edoxaban (“Lixiana®”, 60 mg OD), rivaroxaban (“Xarelto®”, 15–20 mg OD)

**For low risk cutaneous surgery procedures:**

Perform the procedure *just before the next dose is due*

Or

Perform the procedure approximately 18–24 hours after the last dose. The drug can be restarted 6 hours post surgery. This means one dose of a drug taken BD (e.g. “Eliquis®” or “Pradaxa®”) may be missed on the evening before the surgery and take the morning dose six hours post-surgery on the day of the surgery. Take the evening dose as late as possible. For once a day drugs (“Lixiana®” or “Xarelto®”) take the daily dose six hours post surgery on the day of the surgery. Schedule the surgery for early in the morning

**For moderate risk cutaneous surgery procedures:**

Check with the prescriber (e.g. cardiologist) and consider stopping the drug *24 hours before the procedure*

(If the creatinine clearance (CrCl) is between 15 and 30 ml/min apixaban (“Eliquis®”) or edoxaban (“Lixiana®”) or rivaroxaban (“Xarelto®”) should be stopped 36 hours before the procedure)

If the creatinine clearance (CrCl) is between 50 and 80 ml/min dabigatran (“Pradaxa®”) should be stopped 48 hours before the procedure and if it is between 30 and 50 ml/min it should be stopped 72 hours before the procedure

**For high risk cutaneous surgery procedures:**

Check with the prescriber (e.g. cardiologist) and consider stopping the drug *48 hours before the procedure*

(If the creatinine clearance (CrCl) is between 50 and 80 ml/min dabigatran (“Pradaxa”) should be stopped 72 hours before the procedure and if it is between 30 and 50 ml/min it should be stopped 96 hours before the procedure)

**Antidotes**

A specific reversal agent for dabigatran (“Pradaxa”) is available: idarucizumab (“**Praxbind®**”). This is licensed for use in adults treated with dabigatran etexilate when rapid reversal of its anticoagulant effects is required for emergency surgery or urgent procedures, or in life-threatening or uncontrolled bleeding (see the data sheet—SPC)

There are currently *no* other licensed agents to reverse the anticoagulant effect of dabigatran etexilate or any other NOAC

To reverse **warfarin** in an emergency situation consider fresh frozen plasma (FFP), Vitamin K, recombinant Factor VIIa (rFVIIa) and/or Three Factor PCC which can all be given iv usually in the A+E department

**Combinations of multiple drugs**

Shown to increase the risk of bleeding significantly so consider taking advice on modification of regimen if high bleeding risk procedure.

(continued)

**Table 57.3** (continued)**Herbal remedies and supplements**

Examples include (but are not restricted to): Garlic, Ginger, Ginkgo, Ginseng, Saw Palmetto, Fish Oil (e.g. cod liver), Camomile, Feverfew. Many can promote bleeding (and other relevant effects). Only likely to be significant for patients on other antithrombotic agents, or those at high risk from post-operative bleeding. Most bodies advise discontinuation of all supplements (including herbal teas) at least 2 weeks pre-operatively, although this takes in all risks including for general anaesthesia interactions, not just bleeding.

Adapted from: BSDS Guidance on antithrombotics and skin surgery. Aug 2016

surgical procedures carried out once the INR is less than 3.5 (72 hours before surgery). For medium or high risk procedures aim for an INR of 2–2.5 if the therapeutic range allows for the indication the patient is taking warfarin. Patients on aspirin and the newer novel anticoagulants may be unable to stop their medication prior to surgery. Provided the lesion or rash to be operated on is on an area of the body that can be dressed with a pressure dressing and can be elevated post operatively, then it is probably safe to go ahead with low risk cutaneous procedures while still on the anticoagulant. However, it is probably better to use local anaesthetic without adrenaline as this will ensure the operator has adequate haemostasis during and immediately after surgery.

The risk of infection is reduced by cleaning the skin with mentholated spirits and/or chlorhexidine solution immediately before surgery and by applying a sterile dressing to the wound post surgery. Patients should be given verbal and written instructions on how to care for the wound post surgery (Chap. 66. PIL: Care of your wound after surgery). Patients with prosthetic valves and prosthetic joint implants may need subacute bacterial endocarditis (SBE) prophylaxis. Prophylactic antibiotics should be considered when operating on the nostril, mouth and lower legs.

## 57.7 Sutures

Use the smallest suture possible. A 3/8 reverse cutting needle is useful for most skin surgery. On the face + hands us a small suture such as 6/0 or 5/0 nylon (e.g. “Proline®”). Small absorbable sutures such as “Monocryl®” or “Vicral Rapide®” can also be used on the face. On the back, scalp

**Table 57.4** Timing of suture removal

Lips and eyelids	3–5 days
Face	5–7 days
Scalp	7 days
Trunk and arms	10–14 days
Lower legs	14 days

and lower legs use a larger, stronger suture such as 3/0 “Proline®” or if you need a deep absorbable suture use 3/0 “PDS®”. For most other areas on the body use a 4/0 suture such as nylon (e.g. “Proline®”) or 4/0 “Monocryl®” (absorbable).

The optimal time for suture removal depends upon both the location of the wound and how much stress is placed on the wound (Table 57.4). For example, an posterior elbow laceration will require the suture to remain in place longer than on the forearm, since the skin will be stretched each time the elbow flexed.

Sutures on the face are usually removed within five days since there is such good blood supply in this region and healing occurs more quickly. The goal is to remove the sutures as early as possible to minimise scarring; the risk of the sutures causing a scar has to be balanced against the strength and potential weakness of the healing wound (Table 57.4). Sutures may be removed earlier in children and may have to be left in longer if there is a risk of poor wound healing (e.g. elderly, diabetics, on high dose steroids, etc.) [1].

## 57.8 Punch Biopsy

Punch biopsy is a quick way of obtaining tissue for histology, particularly for larger lesions where the pathology is unclear. Having a defini-



**Fig. 57.2** Punch biopsy of suspected non-melanoma skin cancer

tive histological diagnosis will allow better planning for further management such as subsequent excision, topical treatments or referral.

Punch biopsy needles come in various sizes from 1 up to 8 mm in diameter. By far the most common are 3 and 4 mm (Fig. 57.2). Some have an open window near the tip of the blade which allows adequate placement of the biopsy needle over the lesion to be biopsied. Others have plungers which make it easier to remove the specimen from the barrel of the needle. Elliptical shaped biopsy needles of various sizes are also available.

When biopsying a lesion, it is important to take a representative sample of the most suspicious or clinically classical area. Old scales and scabs should be removed prior to biopsy. Ulcerating lesions should be biopsied at the edge and a large lesion may need two or more biopsies from different areas of the lesion to ensure the most serious pathology in a lesion is not missed (e.g. an SCC within a background of an AK).

Punch Biopsy is not suitable for assessing suspicious pigmented lesions where there is a risk that it may be a melanoma. In these situations it is far safer to remove the whole lesion with a 2 mm border of uninvolved skin for histological examination of the whole specimen.

A large pigmented lesions, especially those seen on the face in the elderly, where the differential might vary from a solar lentigo, actinic keratosis or a seborrhoeic keratosis and where the possibly a lentigo maligna is low, a series of punch biopsies from the most suspicious areas

guided by dermoscopy may be sufficient in experienced hands.

Legions suspicious of being a keratoacanthoma (KA) are probably best removed intact as the differential between a KA and a squamous cell carcinoma can be difficult and the pathologist normally needs the complete lesion to make an accurate diagnosis. In fact, nowadays KA's are considered a subtype of squamous cell carcinoma and should probably be treated as such.

Vague rashes are not easy to diagnose histologically and the pathologist, like the clinician, may struggle to make a specific diagnosis. An elliptical biopsy which includes some of the involved and uninvolving skin will give the pathologist the best chance of making an accurate histological diagnosis but the clinician should give the pathologist a reasonable differential diagnosis.

Punch biopsy can also be useful for therapeutic purposes such as removing a 4 mm hairy intradermal naevus or a small dermatofibroma for cosmetic reasons ("punch excision"). In addition, using larger punches (e.g. 4–6 mm), an elliptical punch or taking a number of punches will also debulk and shrink the tumour which can make subsequent excision easier, especially on critical sites such as the face.

It is important to have proper technique when taking a punch biopsy. The doctor should be aware that a punch biopsy needle is as sharp as a scalpel blade and can damage underlying structures, especially if the biopsy needle is pushed too deep into the subcutaneous tissues or when working on higher risk sites such as the face. While inadvertently damaging an underlying blood vessel can be messy, bleeding can usually be controlled by pressure, elevation or suture. It is far more serious if an underlying nerve, duct or tendon is damaged. Nerves that are more likely to be damaged in cutaneous surgery include the temporal branch of the facial nerve after it leaves the parotid gland and crosses the zygoma and the marginal mandibular branch of the facial nerve as it crosses the lower jaw line (Fig. 57.4). Other nerves to be aware of include the spinal accessory nerve in the posterior tri-



**Fig. 57.3** Three danger zones for motor nerve damage in cutaneous surgery. **Oval:** The temporal branch of the facial nerve innervates the frontalis and the orbicularis oculi muscles. It runs very close to the surface near the zygomatic arch and lateral to the eyebrow. Damage to the nerve results in the inability to raise the eyebrow. **Star:** The marginal mandibular branch of the facial nerve innervates the depressor anguli oris and runs very superficially here. Damage to this nerve will lead to inability to smile on this side or depress the lip. **Rectangle:** Erb's point. This is the approximate location of the spinal accessory nerve which emerges very superficially just behind the sternocleidomastoid muscle in the posterior triangle of the neck. Damage to this nerve will result in paralysis of the trapezius muscle resulting in a dropped shoulder

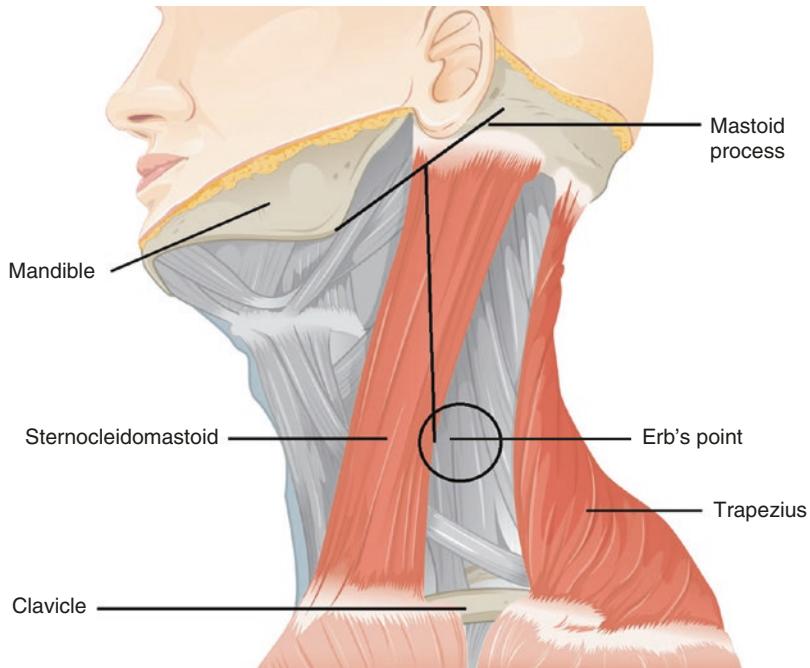
angle of the neck (Figs. 57.3 and 57.4) and the common peroneal nerve as it crosses the upper end of the fibula just below and lateral to the knee joint (Fig. 57.5).

Fortunately nerves that are cut may regenerate but this can take 6 or 12 months, giving the patient plenty of time to initiate a law suit. Care should also be taken when using a punch biopsy needle over tendons or ducts such as lacrimal apparatus or the parotid duct.

When taking a punch biopsy the skin should be stretched perpendicular to the lines of skin tension so when the biopsy is taken and the tension released the round hole will end up in the shape of an ellipse running in the lines of skin tension, which is easier to close (see Fig. 57.6). Marking the area to be operated and its borders with a skin marker before putting in the local anaesthetic is very helpful as the anaesthetic may blanch the skin and make the lesion more difficult to identify (Fig. 57.2). Most skin surgery errors are made with the pen, not the knife. Carefully patient and lesion selection plus marking the lesion and borders with ink before surgery is very important.

The punch biopsy needle needs to be rotated while exerting very light downward vertical pressure which will ensure the round blades will cut cleanly into the skin and the needle should be advanced so that the specimen includes a good cuff of subcutaneous fat. On thin skin such as the face or over a tendon, the needle might only have to be advanced 50% down the hilt, which will allow an adequate biopsy without endangering underlying structures. There are various ways of getting the specimen out of the skin. The operator can angle the punch biopsy to 45° after the needle enters the subcutaneous fat. The tip of the biopsy is then pushed against the gloved finger on the operator's other hand while further angulating the biopsy needle and lifting it out of the skin. This will ensure that the specimen will stay in the barrel of the needle rather than in the skin. It can usually be pinched out of the barrel with your fingers or if necessary with a small needle. Punch biopsy needles with plungers are another easy way to remove specimens from the barrel.

Sometimes the specimen stays in the skin after punching the hole. If this happens it is important to handle the tissue gently so as to avoid crushed artefact for the pathologist. The simplest way to remove the specimen is to pierce the subcutaneous fat with a small 30g needle and then carefully lift out the specimen while cutting the underlying deep fat with a scalpel or scissors.



**Fig. 57.4** Erb's point: Where a vertical line dropped from the midpoint of a line connecting the angle of the jaw (mandible) and the mastoid process crosses the posterior border of the sternocleidomastoid muscle (SCM) marks Erb's point. This is the point at which the spinal accessory nerve exits superficially from behind SCM at its

midpoint. Damage to this nerve results in weakness of the trapezius muscle and a dropped shoulder (Ref. Image by OpenStax College—Anatomy & Physiology, Connexions Web site. <http://cnx.org/content/col114961.6>, Jun 19, 2013, CC BY 3.0, <https://commons.wikimedia.org/w/index.php?curid=3510323>)

A 3 mm punch biopsy of the trunk or arms does not always have to be sutured. A firm pressure bandage and elevation should be sufficient provided the patient is not on anticoagulants. For biopsies on the face, lower legs or larger biopsies, one or two suture should be inserted. A 4/0 “Proline®” is usually sufficient on the body and 6/0 on the face. This suture can usually be removed after 5 or 7 days [2].

## 57.9 Shave Biopsy

These are very useful for removing raised up, nodular lesions, either for histological diagnosis or cosmetic reasons or both. Shave biopsies also debulk and shrink tumours and can make subsequent treatment such as excision or cryosurgery more effective.

The shave biopsy blade is extremely sharp and can be curved to varying degrees which will allow taking a flat superficial biopsy (no curvature of the blade) or a deep shave biopsy (deep curvature of the blade). For suspected malignancies (e.g. a cutaneous horn) a deep shave biopsy should always be taken to ensure the pathologist does not miss an underlying neoplasm and can assess the depth of the tumour. If the surgery is for cosmetic reasons (e.g. an intradermal naevus on the face), a superficial biopsy is desirable as a patient does not want to be left with an unsightly, depressed scar.

Careful hand hygiene and gloves are necessary before any procedure. Disposable surgical gloves are sufficient for most small procedures provided sutures are not being inserted.

After cleaning the skin, marking the outlines of the lesion and applying a local anaesthetic, the

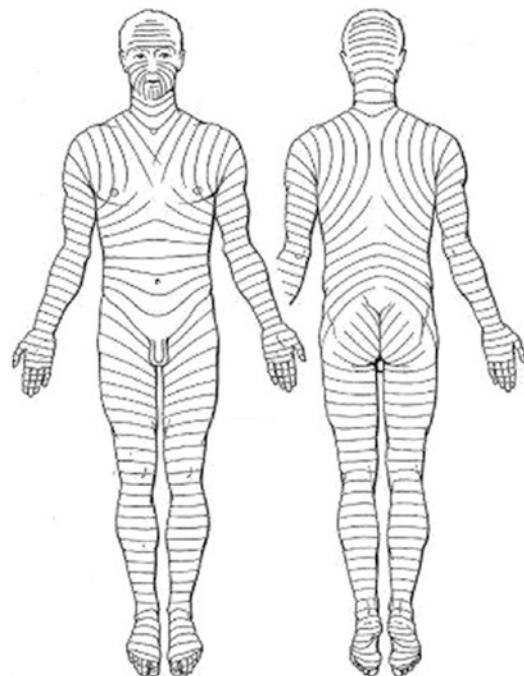


**Fig. 57.5** Location of the common peroneal nerve

biopsy is taken by using a gentle side to side sawing motion while advancing the blade staying within the marked borders (Fig. 57.7).

Bleeding is invariable and sometimes heavy. Pressure, elevation or a tight haemostatic dressing such as an alginate dressing maybe sufficient to control bleeding. Alternatively light cautery (Fig. 57.8) or aluminium chloride solution 20% applied to the bleeding area using cotton buds is also effective. The most convenient source of aluminium chloride is a commercial antiperspirant known as "Anhydrol Forte®" or "Driclor®" which are available without prescription in the local pharmacy. It can be applied via a cotton bud dipped into the solution and then held against the bleeding points for 30–60 s. Used buds should be discarded and not re-dipped (Table 57.6). If using cautery, please ensure there is no mentholated spirits on the skin or soaked into the drapes, as it is highly flammable.

**Cryobiopsy** is an alternative for sensitive areas and needle phobic patients. It can be used



**Fig. 57.6** Langer's lines of skin tension



**Fig. 57.7** Shave biopsy using a disposable razor blade

any time one needs to do a shave biopsy. The area should be frozen lightly and before it is completely thawed, shave the tissue and applied aluminium chloride. No injection of local anaesthesia is required.

## 57.10 Snip Biopsy

This is a useful technique for removing small filiform or pedunculated lesions with a narrow base such as skin tags or fibroepithelia polyp. Under



**Fig. 57.8** Electrocautery with a ballpoint cautery tip immediately post shave biopsy

**Table 57.6** Methods of haemostasis

Pressure × 15 min
Compression bandaging
Elevation >45°
Cold compress
Haemostatic dressings (Alginate dressings)
Aluminium Chloride 20%
Cautery
Suturing
Tranexamic acid for injection ("Cyklokapron®" applied topically on a gauze)

local anaesthetic and aseptic technique, the lesion is usually held up with the non-dominant hand or a tissue forceps while the base is snipped off with a sterile scissors, scalp blade or flat cautery tip. Bleeding can be controlled as outlined above for shave biopsies. The specimen should always be sent for histology. Grabbing the lesion with a cryotweezer is another option: freezing is done just to the base of the lesion where the cutting has been planned. No need to use local anesthesia (see Chap. 58).

## 57.11 Curettage

Curettage is useful for paring down or de-bulking friable tissue such as viral warts or seborrhoeic keratosis. Most doctors now use disposable



**Fig. 57.9** Curettage of a seborrhoeic keratosis with a disposable 7mm cutette

curettes which are as sharp as a scalp blade on one side and blunt on the other (Fig. 57.9). Great care should be taken to identify the sharp side as this can easily gorge out a large chunk of skin.

Curettage is usually carried out under local anaesthetic. The curette is held like a pen and the lesion is gently scraped using the sharp side, either towards or away from the operator while steadyng the skin with the other hand. The fragments should be sent for histology but it can be more difficult to interpret by the pathologist than a solid piece of intact skin. Haemostasis is as outlined above for shave biopsy.

Certain lesions such as warts and seborrhoeic keratosis may need cryosurgery post curettage to ensure there is no recurrence.

In the USA deep curettage and cautery is still quite a popular method for treatment for nodular basal cell carcinoma but this should only be carried out by those with extensive experience in this technique.

nurses doing even simple skin surgery in the community should ensure they have adequate malpractice insurance for the procedures they are performing and should have a friendly surgeon and histopathologist who they can lease with for difficult cases.

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## 57.12 Conclusion

Skin surgery is a valuable service to offer patients and can be enjoyable and lucrative for the practitioner. With careful attention to patient selection, anatomy, lesion recognition and techniques, many small skin lesions can be safely and effectively removed in primary care. Doctors and

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# Cryosurgery in Primary Care

58

David Buckley

## Key Points.

- Never freeze any lesion unless you are completely satisfied about the diagnosis on clinical grounds—if in doubt biopsy or refer the patient—don't freeze!.
- Pare down or debulk nodular lesions before cryosurgery.
- Most lesions will require one or two freeze-thaw cycles to obtain a high cure rate.
- Local anaesthetic using a 30 g needle makes cryosurgery more successful and less painful for the patient when treating lesions larger than 5 mm or a cluster of lesions.
- Cryosurgery is not a panacea for all surgical problems in primary care. Not all patients and not all lesions are suitable for cryosurgery.

## What to Tell the Patient

- There may be some pain, swelling and possibly blistering post cryosurgery and the wound may take 2–6 weeks to heal, depending on which area is treated and the size of the lesion.
- You should delay cryosurgery if you have a major social or work event coming up as the wound may be unsightly and uncomfortable for a number of weeks post cryosurgery.
- Cryosurgery usually does not leave scarring but there may be loss of pigment which is usually

ally transient but may be permanent after a deep freeze.

## 58.1 Introduction

Cryosurgery has a number of **unique advantages** that make it ideally suitable as a surgical modality in primary care.

- Suitable for a wide range of indications in primary care.
- Short treatment times.
- Little or no scarring.
- Low set up and running costs.
- Techniques are relatively easy to learn.
- Low incidence of side effects.
- Low risk of cross infection.
- Immunostimulation (cryo-immunization)
- Equipment highly portable.
- Suitable for use on patients on anti-coagulants

**Common indications for cryosurgery include:**

- Viral warts
- Plantar warts
- Molluscum contagiosum
- Seborrhoeic keratosis
- Actinic keratosis
- Bowens disease

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- Selected cases of non melanoma skin cancer
- Solar lentigo
- Cherry angioma
- Spider naevi
- Venous lake on the lip
- Mucocoeal in the mouth
- Keloids
- Xanthelasma
- Self-made tattoos

Success in cutaneous cryosurgery is dependent on four main factors;

- Cryogen (this chapter will deal only with liquid nitrogen delivered via a hand-held cryogun)
- Equipment
- Technique
- Patient selection (see Chap. 59 on Cryosurgery for warts).

Many group practices now have liquid nitrogen filled hand held cryoguns (Chap. 59, Fig. 59.9) which is the most cost effective and versatile method of delivering cryosurgery in primary care (Fig. 58.1). “Hydrozid®” is an alternative, cheaper option with a long shelf life. It is a hand held CFC (norflurane) containing spray that is usually sprayed on the lesion via a template that limits the extent of the spray. However, it only reaches  $-50^{\circ}\text{C}$  at the surface and will not penetrate deeply, so it is not suitable for deep or bulky lesions and is *not* suitable for superficial skin cancers.

Not all patients and not all lesions are suitable for cryosurgery. Many lesions are better dealt with using different surgical techniques such as elliptical excision, cauterisation, curettage or radiosurgery. Cryosurgery is sometimes used in conjunction with some of these other surgical modalities to give higher success rates and better cosmetic results, for example, by surgical debulking immediately prior to cryosurgery or combining cryosurgery with imiquimod.



**Fig. 58.1** Pouring liquid nitrogen into a hand-held cryogun

## 58.2 Indications

Cryosurgery is the surgical treatment of choice for all types of viral warts in adults and children over the age of 12 years which are causing pain or are unsightly and have failed to respond to simple over-the-counter topical treatments (see Chap. 59). Cryosurgery can also be useful for treating selected cases of non-melanoma skin cancers but this should only be carried out by the most experienced cryosurgeons [1–3]; biopsies need to be taken to confirm the diagnosis histologically prior to or during a cryosurgery treatment. Thermocouple needles, which can measure the temperature under the tumour being frozen, should be available for beginners in skin cancer cryosurgery to ensure adequate depth of freeze ( $>-50^{\circ}\text{C}$ ) when treating

selected cases of non melanoma skin cancer [4]. They are not routinely used by experienced cryosurgeons. These needles make cryosurgery invasive and they may soon be substituted by surface temperature measurement using infrared light.

Cryosurgery is also very useful for treating other common skin complaints such as actinic keratosis, seborrhoeic keratosis, self-made tattoos, mucocles, chondrodermatitis, spider naevi, keloids and xanthelasma.

### 58.3 Safety

The most important rule in cryosurgery is that one should never treat any lesion unless completely satisfied about the diagnosis on clinical and/or dermoscopic grounds. If there is any doubt about the diagnosis, do not freeze the lesion. A biopsy should be taken or the patient should be referred to a colleague for a second opinion (Table 58.1)

**Table 58.1** Rules for safer more effective cryosurgery

1.	Never treat any lesion unless you can make a confident, <b>named, clinical diagnosis</b> on visual assessment. If there is any doubt as to the diagnosis, take a biopsy or refer the patient on to a colleague for a second opinion
2.	Cryosurgery is unsuitable for most children under the age of 6. One exception is molluscum contagiosum. These can be treated with a 2–3 seconds freeze which is practically painless. Some children between the ages of 6 and 12 may be unable to tolerate cryosurgery
3.	The treatment of choice for warts or verrucae in children is either to do nothing or use topical treatments. Cryosurgery should be used only in children with difficult or complicated warts or verrucae and only if they can tolerate a local anaesthetic
4.	Always lie the patients down when performing cryosurgery, as fainting could occur.
5.	Always pare down horny or keratotic lesions before cryosurgery, as keratin is a very good thermal insulator and will prevent from getting a sufficient depth of freeze
6.	Bulky tumours such as large warts and verrucae should be <b>debulked by paring, curetting or doing a deep shave biopsy</b> under local anaesthetic before cryosurgery
7.	When treating large lesions such as large warts and verrucae <b>&gt;5 mm it is advisable to infiltrate with local anaesthetic</b> using a 30G needle under the lesion before paring and freezing. However, avoid a local anaesthetic with adrenaline on or near the fingers, toes, the tops of the nose and earlobe, as it can cause vaso-constriction
8.	Always warn patients about post-operative swelling, pain and possible serous or haemorrhagic blistering. Instruct them to burst the blister with a sterile pin and squeeze out the fluid. They should bathe the area in some dilute “Dettol” and apply an antiseptic cream such as “Savlon” cream and dress with a dry dressing
9.	Oral analgesics such as paracetamol or ibuprofen may be given before or immediately after cryosurgery and continued as required
10.	Treat just one hand per session when treating bilateral hand warts. If there are numerous hand warts, treat only half of one hand or less per session as postoperative pain and swelling can be troublesome. As a general rule, treat a maximum of 4–8 warts per session
11.	When treating verrucae treat one foot per session. In addition, treat only the fore foot or the hind foot (not both) per treatment session, as this will allow the patient to walk on either the heel or the toe post-operatively. Spraying down a suitably sized auroscope cone can improve the success rate and reduce the complications when treating verrucae. Avoid using the auroscope cones on the face or hands, as scarring may be a problem
12.	Any cryosurgery probes or tips that touch the patients skin should be sterilised between patients. If you are using the cotton bud or dip probe technique, decant a small amount of liquid nitrogen into a polystyrene cup and dip into this. Discard the cotton bud, the liquid nitrogen and the polystyrene cup after treating each patient, as viruses can survive in liquid nitrogen
13.	If you are a beginner in cryosurgery, confine your treatments to warts and verrucae for the first year until you get used to handling the equipment, the patient and the tissues. It is advisable to get training and supervision from an experienced cryosurgeon before treating patients alone in your surgery

Children under the age of 6 years are not usually good candidates for cryosurgery with one exception and that is treatment of molluscum contagiosum which requires only a 3 seconds spot freeze which is relatively painless. Children between the ages of 6–12 years old can sometimes tolerate the discomfort of cryosurgery particularly when topical, local and/or regional anaesthesia is used.

Liquid nitrogen can cause serious burns (frostbite) if not handled safely. Protective clothing, gloves and eyewear should be used when filling a cryogun or transporting liquid nitrogen. It should be stored in specially designed storage flasks (Dewars Flasks) and should never be placed in a sealed container without a vent as it is explosive if pressure is left to build up in a sealed container.

It should *not* be stored in small rooms, cars or elevators as it can displace oxygen from the air if it leaks or spills which can lead to asphyxiation. There have been reports of deaths of laboratory technicians dying from asphyxiation as a result of leakage of liquid nitrogen in confined spaces. Liquid nitrogen should always be stored in a large well ventilated room and the room should be vacated immediately if there are any serious leaks or spills as liquid nitrogen is odourless and colourless [5].

## 58.4 Freeze-Thaw Cycle

Maximum cell destruction is achieved by a rapid freeze followed by a slow thaw. The length of time it takes to freeze a lesion will vary depending on the size of the aperture or the probe on the cryogun and the size of the lesion being treated. A 20 seconds freeze with a small D spray tip on an actinic keratosis greater than 10 mm in diameter will be inadequate. On the other hand, a 20 seconds freeze with an A or B spray tip on a small actinic keratosis, less than 5 mm, may be too much. Most lesions up to 10 mm in diameter can be frozen with a C spray tip (Brymill Corp.©).

It is important to define what “a freeze time” and “a freeze thaw cycle” really means, so that results can be compared, adequacy of treatment assessed and cryosurgery techniques can be properly taught to both doctors and nurses.

The following definition has been proposed when using liquid nitrogen via a cryogun [6]:

- Start freezing at a fast rate using an appropriate size spray tip or probe for the size of the lesion (C or E tip for a small lesion <10 mm and a B tip for lesions between 10 and 20 mm in diameter)
- Continue freezing until the complete lesion (e.g. actinic keratosis or wart) is frozen and continue until a halo of normal uninvolved skin around the lesion is also frozen (2–3 mm margins for hand warts and actinic keratosis, >3 mm margins for plantar warts and 4–5 mm margins for small non-melanoma skin cancers)
- Continue to freeze (spraying intermittently to avoid excessive lateral spread of the ice front) for the desired length of time (“freeze time”) e.g. 10 seconds for warts or actinic keratosis, 15 seconds for plantar warts, 30 seconds for MMSC (Table 58.2)
- Let the lesion thaw out completely without external warming.

**Table 58.2** The freeze-thaw cycle using the open spray technique

Diagnosis	Freeze time (Seconds) <sup>a</sup>	Number of FTCS <sup>b</sup>	Margin (MM)
Molluscum	3	1	<1
Actinic keratosis	10	1	2–3
Seborrhoeic keratosis without paring	10	1	2
Seborrhoeic keratosis post paring	5	1	2
Common warts	10	2	2–3
Periungual warts	7	1	1–2
Plantar warts	10–15	2	3
Bowen’s disease <sup>c</sup>	30	1	>3
BCC/SCC <sup>c</sup> <10 mm	30	2	>3
BCC/SCC <sup>c</sup> 10–20 mm	30	2	>6

<sup>a</sup>Interval after the complete lesion is frozen and a margin of normal uninvolved skin is also frozen (not the total spray time). C spray tip is suitable for tumours up to 10 mm in diameter and a B spray tip should be used for larger tumours up to 20 mm

<sup>b</sup>FTCS freeze-thaw cycle

<sup>c</sup>Not all non melanoma skin cancers are suitable for cryosurgery

- If a second freeze-thaw cycle is required (e.g. for warts, plantar warts or NMSCs) it can be started using the exact same technique as described above once the complete thaw has occurred from the first freeze. A second freezing will always be faster because there will be ice crystals in the tissue.

Bulky lesions should be pared down or debulked prior to cryosurgery. Different techniques may be required for treating larger lesions (greater than 2 cm), when spraying down an oto-scope cone, using a cryochamber or freezing in conjunction with imiquimod (i.e. immunocryosurgery). Sometimes, temperature monitoring at the base and periphery of a tumour may be more appropriate than measuring time when treating non-melanoma skin cancers.

## 58.5 Immunocryosurgery

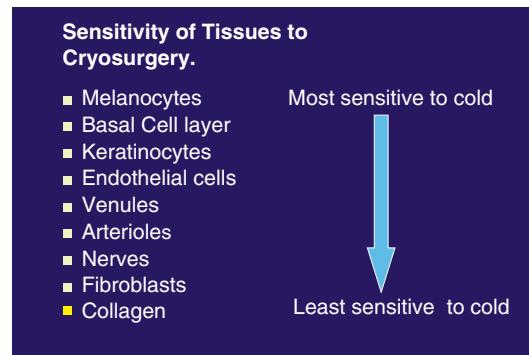
Combining imiquimod 5% “(Aldara®)” with cryosurgery can enhance the success of both techniques compared to when they are used individually (immunocryosurgery). This combination can be used for genital warts, non genital warts (see Chap. 59) and selected cases of non melanoma skin cancers (NMSCs). For NMSCs, have the patient pre-treat the tumour daily for 8 hours for 2 weeks with imiquimod 5% cream. Then freeze the tumour, including a 4 mm border of surrounding uninvolved skin, for 15–20 seconds and with a double freeze-thaw cycle. Have the patient continue with imiquimod 5% topically immediately after the session of cryosurgery for another 3 weeks starting on the same day as the freezing. This cycle can be repeated if necessary [7]. Warn the patient that local side effects such as pain, swelling, crusting and inflammation can be unpredictable and sometimes severe. There may even be systemic symptoms such as a flu like illness during the treatment with imiquimod (see Chap. 45).

## 58.6 Side Effects

Serious side effects with cryosurgery are rare. Different tissues have different sensitivities to cryosurgery (Table 58.3). Melanocytes, for instance, are most sensitive to cold; this is why hypo or hyper-pigmentation post-cryosurgery can happen although this is temporary in most cases. As fibroblasts and collagen are less sensitive to cold, little or no scarring post-cryosurgery occurs. The periungual skin and cuticle are very sensitive to cold and permanent scarring can occur if this area is frozen too hard (Fig. 58.2).

Pain, swelling and serious or haemorrhaging blistering can occur post-cryosurgery and patients should be warned about these side effects and instructed on how to manage them (Fig. 58.3). It

**Table 58.3** Sensitivity of tissues to cryosurgery



**Fig. 58.2** Nail damage from previous cryosurgery for periungual warts



**Fig. 58.3** Haemorrhagic blisters 2 days post cryosurgery for warts

is probably better to delay cryosurgery if the patient has any major social or work engagements coming up in the following 2 week as weeping and blistering can be unsightly and uncomfortable. There have been rare reports about tendon rupture with cryosurgery but with proper techniques, this can almost always be avoided [8]. Temporary anaesthesia post cryosurgery over a nerve is not uncommon and the sensation usually returns after 3–6 months as the nerve regenerates.

## 58.7 Conclusion

Techniques in cutaneous cryosurgery are best learned by seeing patients being treated by an experienced cryosurgeon. Articles in scientific journals, books, internet videos and CD's are also available [9, 10]. All doctors and nurses should have adequate training prior to carrying out cryosurgery in their own surgery to ensure high success rate and a low incidence of side effects.

Perfect techniques on carefully selected patients with good follow up and record keeping should ensure doctors do not freeze now and fry later in the hands of the lawyers!

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# Cryosurgery for Warts in General Practice

59

David Buckley

## Key Points

- For warts that require treatment, the first line of treatment is usually with topical creams, gels or solutions which the patient can use at home.
- Cryosurgery is safe and successful when treating warts but the technique has to be perfect to get high cure rates.
- Never freeze any lesion unless you are certain of the diagnosis.
- Cryosurgery is painful and most warts >5 mm or cluster of warts are best treated under local anaesthetic using a 30 G needle.
- Avoid using cryosurgery for warts in children under the age of 12 years and almost never in children under 6 years old.

## What to Tell the Patient

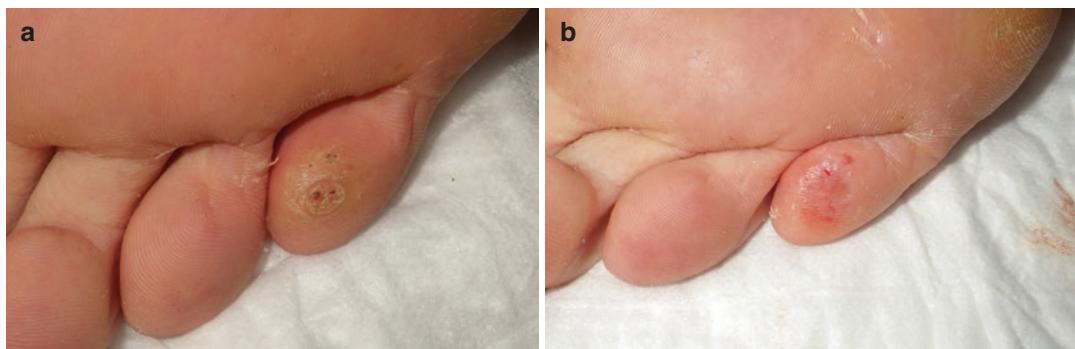
- Most warts do not require treatment as many will clear spontaneously in time especially in children.
- Cryosurgery for treating warts is highly successful but can be painful and may require local anaesthesia.
- There may be some pain, swelling and sometimes blistering post cryosurgery and healing may take 2–3 weeks.

- If you have cryosurgery for plantar warts you may be limping for a few days or weeks post-treatment.

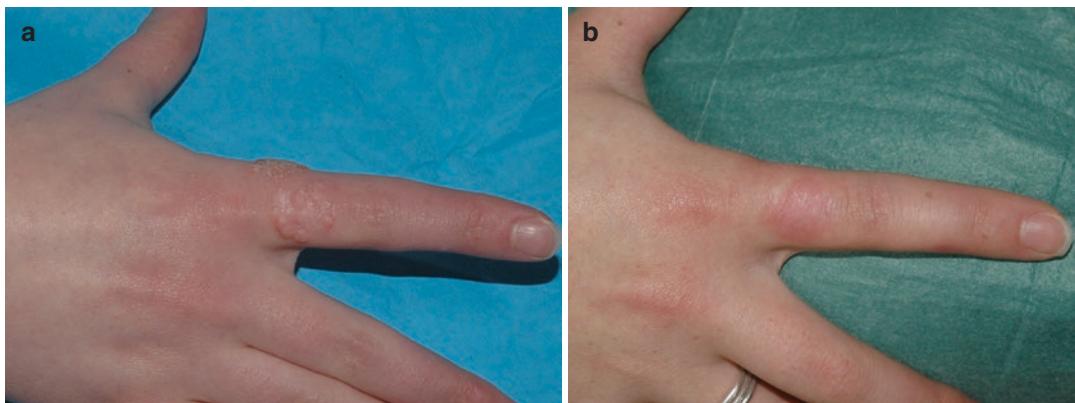
## 59.1 Introduction

Cryosurgery is a method of selectively destroying unwanted tissue using cold liquids or gasses. The aim is to cause maximum tissue destruction in the target lesion with minimal collateral damage to the surrounding healthy structures. Cryosurgery does not kill the human papilloma virus (HPV); in fact, viruses can survive and be preserved in liquid nitrogen. What we are trying to achieve with cryosurgery is to destroy the cells that are infected with the HPV by creating intracellular ice crystal formation that ruptures the cell, thus allowing clean, healthy, uninfected cells to take their place. Cryosurgery causes local swelling which blocks the small feeding vessels resulting in ischaemic necrosis of the frozen area, further enhancing cell death. Furthermore, it has the unique action of cryoimmunostimulation, whereby some of the wart virus is released from the frozen tissue after cryosurgery, presenting wart antigen to the immune system [1]. This acts like a vaccine, helping the body to fight off the HPV in the treated and sometimes even distant untreated warts (Fig. 59.1a, b). Patients who have a suppressed immune system are less likely to benefit from cryoimmunestimulation. Combining

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**Fig. 59.1** (a) Plantar wart in a 13 year old (b) Same patient 1 month later with spontaneous resolution of the plantar wart on her 5th toe after treating a larger one elsewhere on her foot 1 month earlier



**Fig. 59.2** (a) Recalcitrant hand warts in a 22 year old immunosuppressed woman. (b) Same patient cleared with cryosurgery and imiquimod 5%

imiquimod (“Aldara®”) with cryosurgery may enhance this response and this is known as immunocryosurgery [2] (Fig. 59.2a, b).

There is only one important rule in cryosurgery: never freeze any lesion unless you are 100% sure of the diagnosis. If you cannot make a confident named clinical diagnosis, do not freeze—take a biopsy or refer the patient for another opinion.

Success in cryosurgery is dependent on four main factors (Table 59.1):

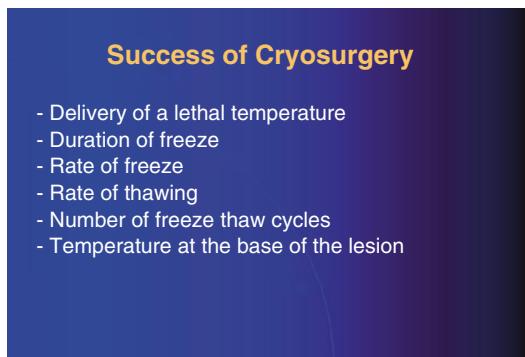
- Cryogen
- Delivery system
- Patient selection
- Technique

## 59.2 Cryogen

Maximum cell destruction is achieved by applying temperatures of less than 40 degrees Celsius ( $-40^{\circ}\text{C}$ ) at the base of the lesion (rapid freezing, due to temperature difference with human skin), followed by a slow thawing and carrying out at least two freeze-thaw cycles [3] (Fig. 59.3). This is best achieved by using cryogens with a very low boiling point such as liquid nitrogen ( $-196^{\circ}\text{C}$ ), which is one of the coldest most versatile and cheapest cryogen available (Tables 59.2, 59.3, 59.4 and Fig. 59.4). Over the counter cryogens such as home freezers like “Wartner®”, which contain a mixture of dimethyl ether and propane (DMEP), are much less effective, as

most only get down to  $-29^{\circ}\text{C}$  and have very poor penetration of tissues (see Fig. 59.5). Hand held medical devices such as the “Histofreezer®” or the “Dermafreeze®” also contain DMEP and are only suitable for very small, superficial lesions as the cryogen does not penetrate deeply. “Hydrozid®”, which is an aerosol can that contains a hydrofluorocarbon (norflurane), can reach

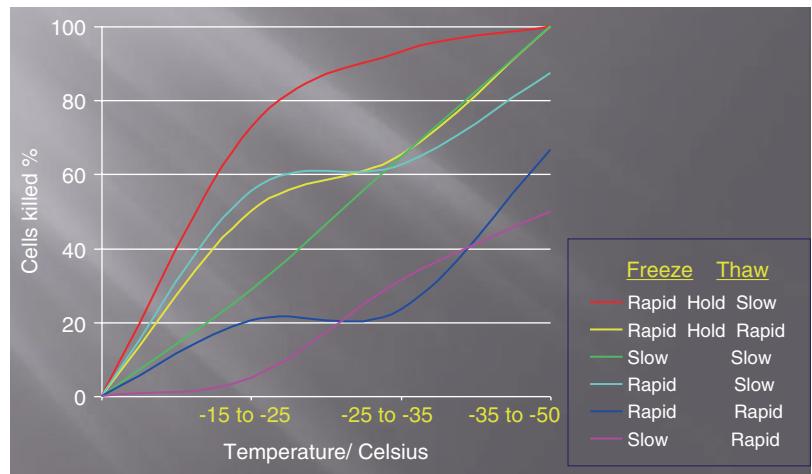
**Table 59.1** Success in cryosurgery is dependent on a number of factors



temperatures of  $-60^{\circ}\text{C}$  at the surface but will not penetrate deeply (Fig. 59.6). It has a long shelf life and is portable. It is a reasonable alternative for those who do not have access to liquid nitrogen but it is not cold enough to treat deep or bulky tumours or skin cancers.

Nitrous oxide gas, while not as cold as liquid nitrogen, can reach  $-89^{\circ}\text{C}$  and can give reasonably good results provided there is meticulous attention to technique (Figs. 59.4 and 59.7). Large devices with a handle and various contact probes and tips which are linked to a large tank of nitrous oxide are now much less popular as they are expensive to run and are bulky to store. The “Freezezen®” and the “Dermagen Cryo®” are small hand held units with disposable capsules filled with nitrous oxide gas and have a long shelf life (Fig. 59.8). However, they are relatively expensive and have very poor penetration of the tissues. They are only effective for small, superficial lesions. Their advantage is the small size of the unit and the use of capsuled

**Fig. 59.3** Percentage of cells killed with various freezing and thawing rates and temperatures. (courtesy of Mr Omar Maiwand)



**Table 59.2** Boiling point of the most common cryogens

Ice (water)	$-0^{\circ}\text{C}$
Ice (Saturate salt and water mix)	$-21^{\circ}\text{C}$
Dimethyl ether and propane in a cotton bud (“Histofreezer®”)	$-29^{\circ}\text{C}$
Norflurane (HFC) (“Hydrozid®”)	$-60^{\circ}\text{C}$
Nitrous oxide	$-89^{\circ}\text{C}$
Liquid nitrogen	$-196^{\circ}\text{C}$

nitrous oxide gas as a cryogen which has a long shelf life. These hand held nitrous oxide devices also avoid having to buy, store and refill a dewar flask with liquid nitrogen (Table 59.4).

**Table 59.3** There are four main cryogens used in clinical practice

Which Cryogen?	
Main cryogens:	
Dimethyl ether, propane+ isobutene (DMEP) ("Histofreezer®", home freezing kits = "Wartner")	
Hydrofluorocarbon (HFC) (e.g. Norflurane) (" Hydrozid®")	
Nitrous oxide (N <sub>2</sub> O). ("Welch Allen", "Freeseopen", "Dermpen", "Cryo-omega", "CryoSuccess")	
Liquid nitrogen (LN) Cryogun	

### 59.3 Equipment

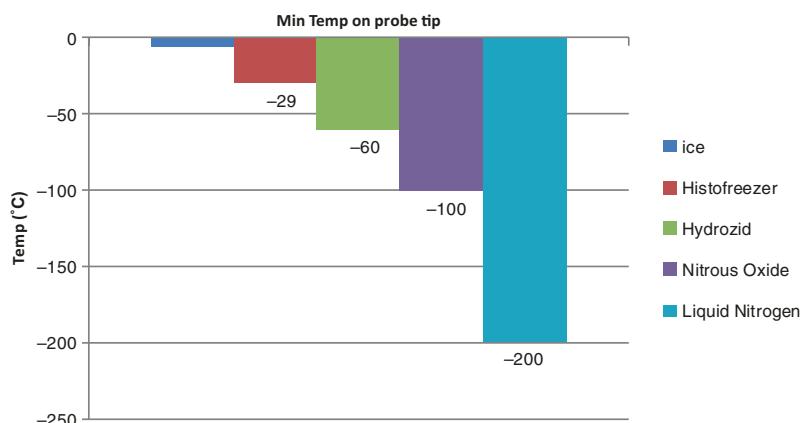
Liquid nitrogen cryosurgery via a closed hand-held cryogun is the safest, most effective and most versatile method to deliver a freeze (Fig. 59.9). Applying liquid nitrogen with a cotton bud is not as effective as a liquid nitrogen cryogen but is still more effective than nitrous oxide gas, "Hidrozid®", "Histofreezer®" or the "Dermafreeze®".

### 59.4 Patient Selection

Patient selection is crucial in delivering effective cryosurgery. Children under the age of six do not make good candidates for cryosurgery for warts and doctors should resist parental pressure to freeze warts in this age group. The only excep-

**Table 59.4** Advantages and disadvantages of various cryogens

	DMEP ("Histofreezer®")	HFC ("Hydrozid®")	N2O Handheld ("Freeze Pen®")	N2O Tank ("Welch Allyn®")	Liquid nitrogen Cryogun
Safety	+	Global warming	Explosive	Explosive	O <sub>2</sub> ↓ Burns
Portability	+++	+++	+++	+	++
Shelf life	+++	+++	++	+++	+
Pain score (out of 10)	1/10	3/10	5/10	5/10	8/10
Cure rate	-	+	++	++	+++
Cost per RX	Expensive	Expensive	Expensive	Cheap	Very cheap
Various tips	-	-	+	++	+++



**Fig. 59.4** Minimum temperature reached when the cryogen was places on the tip of the thermocouple (©David Buckley)

tion is freezing *molluscum contagiosum*, which usually clear up with a tiny, almost painless, 3 seconds freeze. Children between the ages of 6 and 12 years old are generally poor candidates for cryosurgery unless the child (and not the parent) is highly motivated, can understand what is involved and is very eager for treatment. Avoid treating children with cryosurgery for warts greater than 4 or 5 mm in diameter or a cluster of warts together, unless the child can tolerate local anaesthetic. Other poor candidates for cryosurgery are needle phobics and immunosuppressed patients (diabetes, transplant patients).

## 59.5 Technique

There is a limit to the depth of freeze one can achieve with cryosurgery. As you freeze from the surface down, the isotherms get progressively warmer until equilibrium is reached between the cold of the cryogen at the surface and the heat of the skin from the underlying circulation (Figs. 59.10 and 59.11). Hypertrophic warts are often covered with thick keratin, which acts as a thermal insulator. This has to be removed with a blade to allow the freeze to penetrate to the base of the wart. Even after removing keratin, many

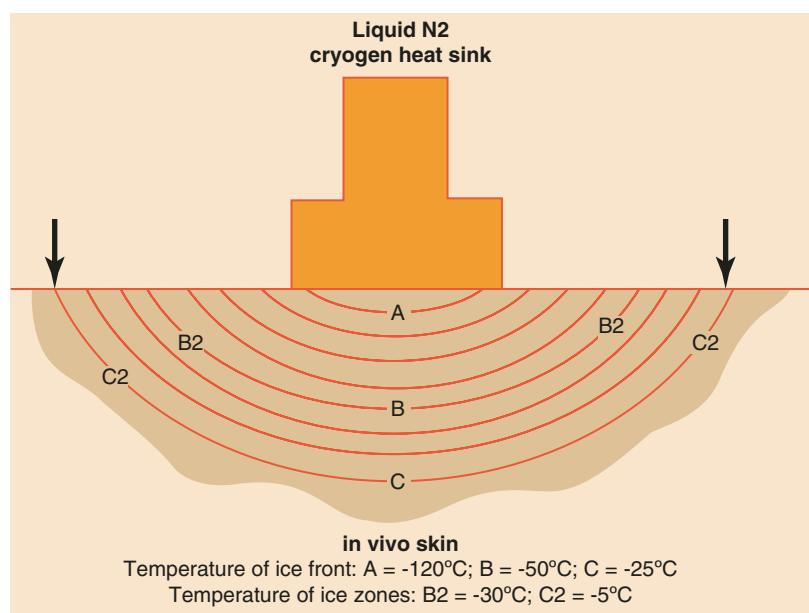
large warts can be 4 or 5 mm deep. Success with cryosurgery is increased dramatically when a wart is de-bulked (Fig. 59.12a, b, c). Generally, this can only be achieved by applying local anaesthetic and surgically paring down the wart. Bleeding can be controlled with 20% aluminium chloride applied with a cotton bud, pressure and elevation.

For most warts greater than 4 mm in diameter, or for a cluster of warts together, the discomfort of a prick with a 30 G needle with local anaesthetic is generally a lot less painful than trying to treat warts with cryosurgery without local anaes-

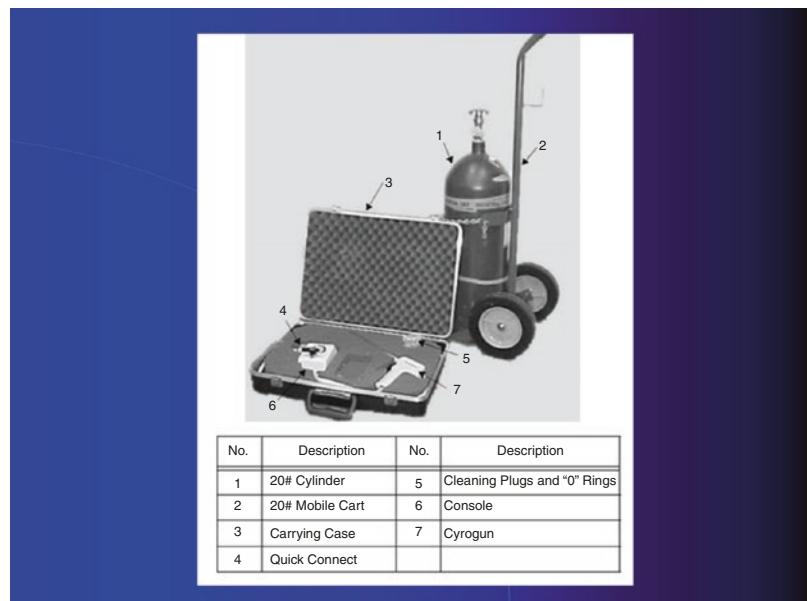


**Fig. 59.6** “Hydrozid®” (norflurane) can reach  $-60^{\circ}\text{C}$ .

**Fig. 59.5** Isotherms created at different distances radiating from the heat sink source



**Fig. 59.7** Nitrous oxide cryosurgery kit and tank



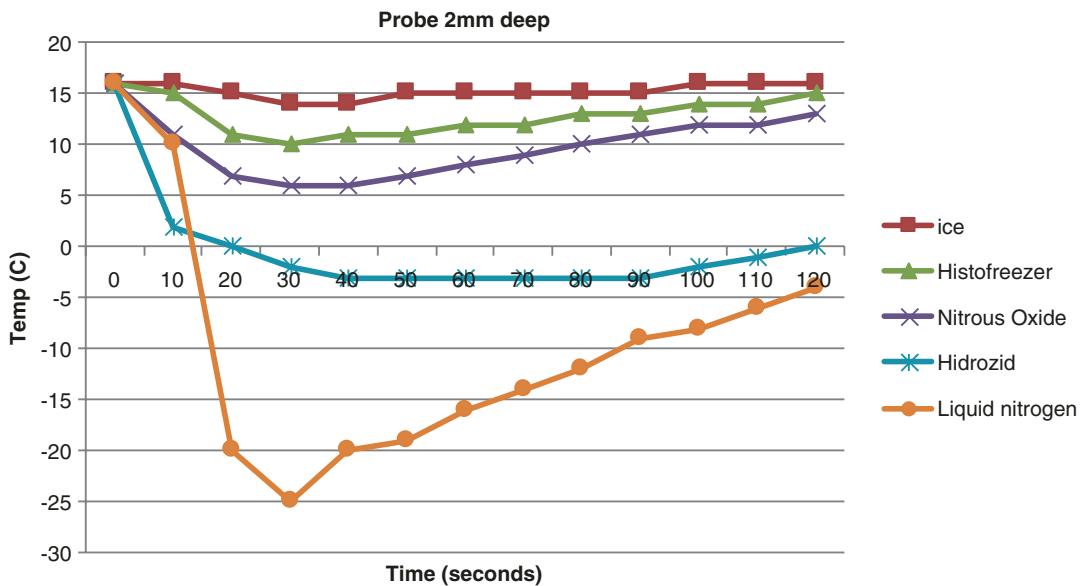
**Fig. 59.8** Nitrous oxide handheld cryosurgery device



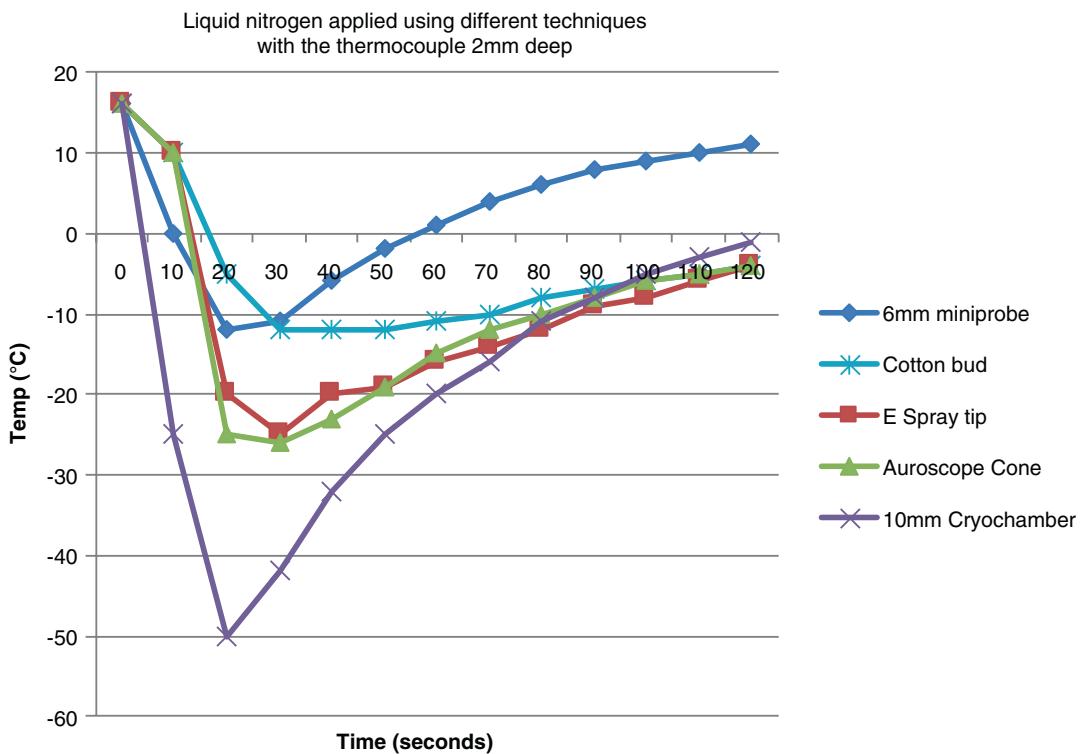
**Fig. 59.9** Liquid nitrogen cryogun and various attachments

thetic. A recent study by this author showed that the average pain score of cryosurgery without local anaesthetic was more than twice the pain score of administering the local anaesthetic with a 30-gauge needle [4] (Fig. 59.13). See Table 56.2 in Chap 56 on local anaesthetics for some techniques for minimizing the pain when giving local anaesthetic.

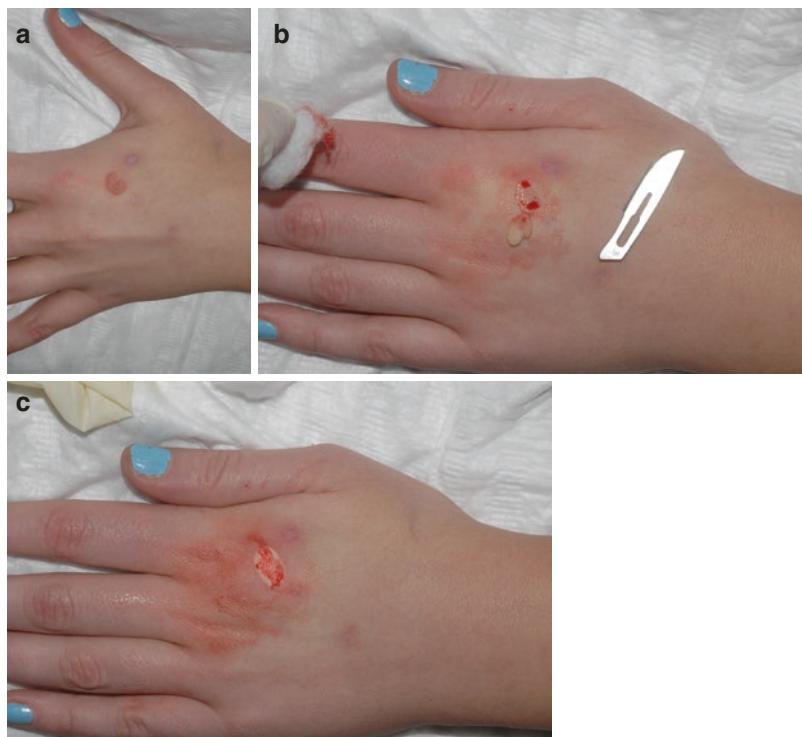
For hand warts, have them flush with the surrounding skin by peeling them down with a scalpel blade before beginning cryosurgery and for plantar warts, scoop them out with a number 10



**Fig. 59.10** Temperature reached with various cryogens with termocouple 2 mm deep in an ultrasound gel pad

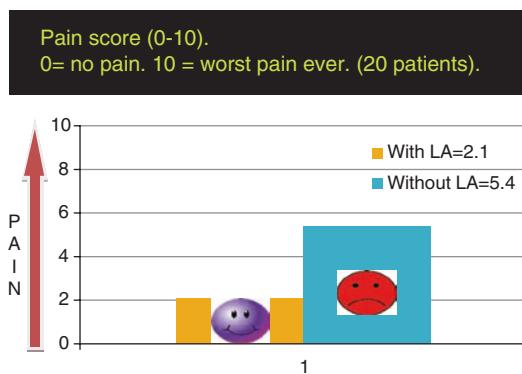


**Fig. 59.11** Temperature reached with various probes and tips using liquid nitrogen with termocouple 2 mm deep



**Fig. 59.12** (a) Hand wart in a 18 year old (b) Hand wart post debulking under local anaesthetic (c) Same wart immediately post-cryosurgery with a C spray tip on a

hand-healed liquid nitrogen cryogun frozen for 10 seconds and 2 freeze-thaw cycles



**Fig. 59.13** The difference between the pain of cryosurgery with and without local anaesthetic (ref. [3])

scalpel blade or a curette leaving a crater to freeze into (Fig. 59.14a-d). In this way, 75–90% of the wart is removed before starting the freeze. Another bonus is that the local anaesthetic makes the whole procedure far more tolerable for the patients and more enjoyable for the doctor.

Using this technique, success rate can be as high as 90% with one single treatment for hand warts and verrucas [4, 5] (Fig. 59.15a, b). Another advantage to de-bulking is that there is less necrotic tissue to die off post cryosurgery. The only disadvantage is that there can be a lot of bleeding during and after the treatment. Care should be taken not to contaminate the cryosurgical unit with blood. Put on a fresh glove just before picking it up for the freeze to avoid contaminating it with blood. Post-operative bleeding can be controlled by a pressure dressing and elevation. Heavy bleeding can usually be controlled with 20% aluminium chloride and/or alginate dressings combined with pressure dressing and elevation. In addition, a post-treatment bullae filled with blood or serous fluid is possible. Let the patient know about this possibility in advance. Post-operative pain can usually be controlled by giving paracetamol or ibuprofen immediately after the session of cryosurgery, before the local



**Fig. 59.14** (a) Plantar wart (b) Same patient post paring under local anaesthetic (c) More paring. (d) Immediately post cryo

aesthetic has had time to wear off. Most warts will heal in 3–6 weeks.

Most bulky warts require two freeze-thaw cycles (Table 59.5). There is some controversy, even amongst expert cryosurgeons, as to what constitutes a freeze-thaw cycle. However, most now agree that the following definition is correct [6]:

Freezing down the auroscope cone is a useful technique for plantar warts, which allows deeper penetration of the freeze, without too much lateral spread. This leads to a higher success rate with lower morbidity, such as blisters. Spray down the auroscope scope cone with a C or E spray tip for 10 second only, as this is a much more concentrated form of cryosurgery than using the open spray technique. Auroscope cones should be avoided on the dorsum of the hands and face as it can leave an unsightly round patch of hypo or hyper pigmentation.

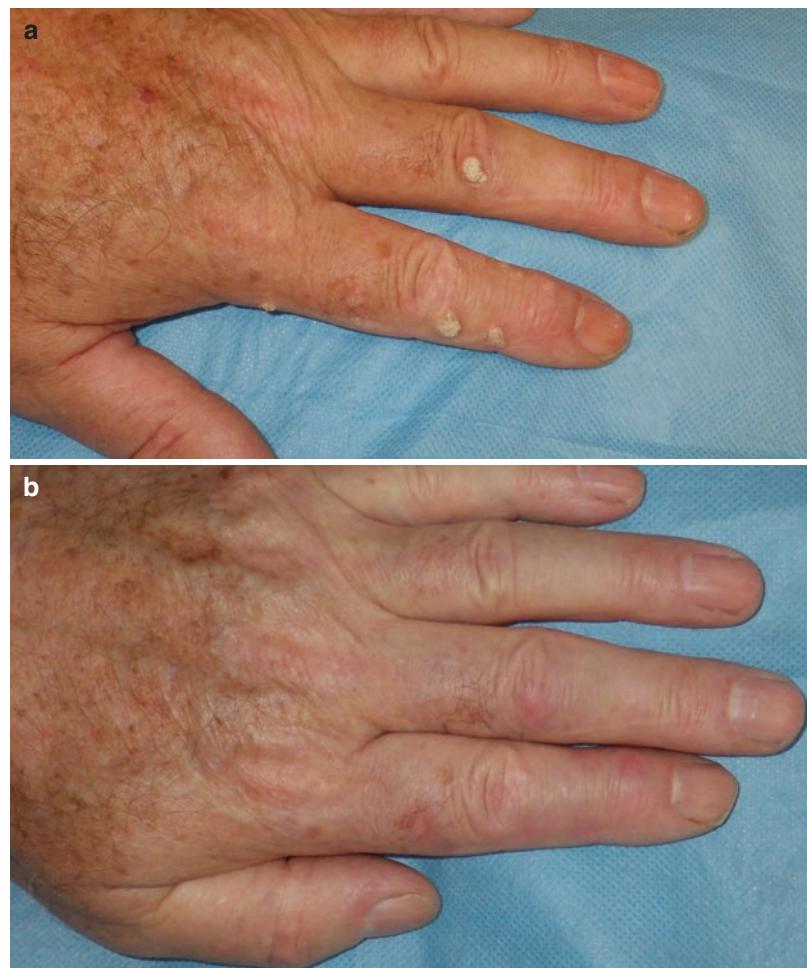
Flat warts (plain warts) are usually quite superficial and so are much easier to treat. They do not usually require paring or de-bulking because they are usually small and can often be treated without local anaesthetic. Ano-genital and mucous membrane warts are usually soft with no keratin and again do not usually require paring or de-bulking before cryosurgery. Topical anaesthetic (e.g. “Amitop®”) works well and quickly (~15 minutes) on mucous membranes.

Periungual warts usually occur as a result of damage to the cuticle, which is normally self

## 59.6 What is a 10 second Freeze-Thaw Cycle?

- Start freezing at a fast pace using a B or C spray tip until the whole wart is frozen (Fig. 59.14d).
- Continue freezing until a halo of clinically non affected skin, 1 or 2 mm, around the wart is also frozen.
- Continue to freeze (at a slower rate by using intermittent bursts of liquid nitrogen spray to avoid excessive lateral spread) for 10 seconds.
- Then let the wart thaw out completely without heating, before starting a second freeze thaw cycle exactly the same way, if required.

**Fig. 59.15** (a) Hand warts before treatment with an open spray technique using liquid nitrogen via a hand held cryogun (b) Same patient a few months post cryosurgery



**Table 59.5** Freeze-thaw cycles

Type of wart	Freeze times (s)	Freeze-thaw cycles
Molluscum contagiosum	3	1
Periungual warts	7	1
Filiform wart	10	1
Ano-genital warts	3–10	1
Common wart	10	2
Plantar wart	10–15	2
Mosaic plantar wart	15	2

inflicted from biting or picking (Fig. 59.16). They can be very difficult to manage, as the periungual skin is very delicate. A good technique is to pare them down and freeze them gently with a 5–7 seconds freeze and only one freeze thaw-cycle to avoid damaging the germinal matrix of the nail plate.

## 59.7 Immunocryosurgery

Imiquimod 5% (“Aldara®”) is a Toll-like receptor (TLR) 7 agonist. that induces production of inflammatory cytokines including interferon-alpha, tumour necrosis factor alpha, and interleukin-12, and also enhances antigen presentation to T-cells. The overall effect is an enhanced immune response to viral infection. It is licensed for genital warts but there are a number of studies using imiquimod for non-genital warts. Best results seem to be achieved when imiquimod is combined with cryosurgery. One technique is to pare down the thick keratin over the wart and apply imiquimod to the wart alternate days for 3 weeks. Cryosurgery (10 second freeze and one freeze thaw cycle) should be applied after the second



**Fig. 59.16** Periungual warts

week of imiquimod and imiquimod should then be continued immediately after the cryosurgical session daily for one more week. [7] (Fig. 59.2a, b).

There is some, weak evidence that oral zinc sulphate, 10 mg/kg/day for 2 months (max 600 mg/day) in divided doses with food may boost the immune system and help the body fight off the wart virus. It can be used it in conjunction with cryosurgery for resistant warts [8, 9].

## 59.8 Side Effects

Serous or haemorrhagic blisters may occur within a few days of cryosurgery. Pigmented changes (hypo-pigmentation or hyper-pigmentation) can sometimes occur, particularly in dark skinned patients, but the pigment usually comes back after a few months, especially when the freeze thaw cycle is not more than 10 seconds. Nerve damage is very rare when treating warts. If it does occur, for instance on the digital nerve, it may result in a temporary numbness of the side of the finger that will resolve after a few months (Table 59.6).

**Table 59.6** Side effects of cryosurgery

<b>Side Effects of cryosurgery:</b>	
<b>Immediate:</b>	
Pain	
Swelling	
<b>Delayed:</b>	
Blister formation (serous or haemorrhagic)	
Ulceration	
Secondary infection	
<b>Side Effects</b>	
<b>Prolonged:</b>	
- Hypo or hyperpigmentation	
- Contracted scar	
- Hypertrophic scar	
- Skin atrophy	
- Paraesthesia	
- Hair follicle loss	

## 59.9 Conclusion

The maximum number of warts treated in any one session of cryosurgery should be limited to approximately six to ten. If a wart dose not clear after three separate sessions of cryosurgery using perfect technique via a liquid nitrogen cryogun, there is little point in persisting with cryosurgery. It may be better to revert to other techniques or perhaps encourage the patient to simply live with the wart and keep it under control by paring it at home, which makes it look and feel better.

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# A Practice Nurse Led Cryosurgery Clinic

60

David Buckley

## Key Points

- Techniques in cryosurgery are relatively simple to learn. The real skill is knowing which patient to treat and with which method.
- A named diagnosis should be confirmed clinically or histologically by a doctor experienced in lesion recognition before a nurse starts treating a lesion with cryosurgery.
- Nurses should not treat any lesion unless they are absolutely sure of the diagnosis.
- Adequate analgesia and debulking prior to cryosurgery are important in order to achieve a high cure rate and satisfied patients.
- Children are not good candidates for cryosurgery.

## 60.1 Introduction

Nurses have been carrying out cryosurgery in dermatology outpatients for many years. The biggest growth area in the use of cryosurgery in the past 20 years has been in primary care. Many practices now have excellent cryosurgical equipment. For those practices fortunate enough (or wise enough) to have a practice nurse, it may be more appropriate to delegate some of the cryosurgical work to the nurse. The technique of

cryosurgery is relatively easy to learn and provided the nurse confines her treatment to certain areas, (e.g. warts, verrucae and molluscum contagiosum, in patients over the age of 12 years old), it is unlikely that there would be any serious complications to the treatment. The only really important safety rule in cryosurgery is that no lesion should be treated unless there is absolutely certainty of the diagnosis. For example, a warty lesion on the back of a hand of a 60 year old male may not be a viral wart. Skin cancers sometimes present like this. Corns and verruca can sometimes be difficult to distinguish even by experienced dermatologists. Great care should be taken when treating pigmented lesions as a suspected seborrhoeic keratosis may turn out to a malignant melanoma. It can sometimes be impossible to distinguish an actinic keratosis from a squamous cell carcinoma on clinical grounds alone. Therefore, it is probably safer that a named clinical diagnosis be confirmed by the doctor (and noted in the chart) prior to the nurse carrying out the cryosurgery (see Chap. 58).

## 60.2 Techniques

Cryosurgery is a painful procedure. Always lie the patient down before cryosurgery, especially in younger patients, as fainting is relatively common. For larger warts or verrucae greater than 4 mm in diameter or for a cluster of warts

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together, it is much less painful to inject a small amount of local anaesthetic under the wart using a 30 gauge needle rather than trying to treat the wart without a local anaesthetic [1]. Local anaesthetic will also allow paring down the wart or verrucae more deeply prior to cryosurgery and this should ensure higher success rates. Topical anaesthetics (e.g. "EMLA®" or "AMITOP®") have not been shown to significantly reduce the pain of cryosurgery for warts but they will ease the discomfort of a local anaesthetic injection if applied 30–60 min beforehand. Tetracaine gel ("AMITOP Gel®") alone may provide sufficient anaesthesia for treating warts on mucus membranes (e.g. genital warts) and usually works within 10–15 min in these areas.

Treating children under 6 years old with cryosurgery is usually difficult and unrewarding and other methods should be applied if at all possible. The one exception would be molluscum contagiosum. They respond extremely well to cryosurgery and require a very light freeze (2–3 seconds) since they are extremely cryosensitive. Children may tolerate this level of discomfort without an anaesthetic if they are well motivated. However, most molluscum clear spontaneously within 6–12 months especially if the area is treated with a thick greasy ointment like emulsifying ointment and they do not usually require cryosurgery.

Warn all patients about the possibility of pain, swelling, blistering and hypopigmentation. All patients should be given verbal and written instructions on how to manage post-operative blisters and pain (see Chap. 66).

Most warts and verrucae can be frozen using the open spray technique after paring off the hard keratin. The nozzle of the cryogun is held about 10 mm away from the skin. The lesion is frozen until it goes completely white. The freezing is continued until a halo of 1 or 2 mm of surrounding normal skin is also frozen. The freeze is then maintained at this level for 10 seconds by short rapid pulses of liquid nitrogen spray, being care-

ful not to let the ice ball extend more than 1–2 mm beyond the wart. The lesion is allowed to thaw out completely without external heating. If a second freeze-thaw cycle is required, the procedure is repeated immediately once the wart has thawed out after the first freeze. The technique of cryosurgery is best learnt from expert cryosurgeons and by practical demonstrations on patients (see Chap. 59).

Some viral warts such as periungual warts and mosaic verrucae can be very difficult to treat and are probably best left to expert cryosurgeons. Larger warts (greater than 5 mm) may require local anaesthetic and unless a practice nurse is capable of administering it, these warts are best left to the doctor in the practice who is most experienced in cryosurgery.

### 60.3 Conclusion

Good record keeping is important. Written informed consent is recommended when freezing simple warts or verruca. Patients should be warned about the possibility of post-operative pain, bleeding, blistering and hypo or hyper pigmentation. The size, position and number of lesions and the exact type of treatment given should be recorded (record freeze times, number of freeze thaw cycles and type of tip used on the cryogun). A note should also be kept as to whether topical or local anaesthetic was used and whether oral analgesics were administered. Good records will be of great help should there be any problems that might lead to a medico legal case. Detailed surgical notes of the procedure will also facilitate audit and research.

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## **Part XIII**

### **Pharmacology and the Skin**



# Pharmacists and Skin Disease

61

David Buckley

## Key Points

- Pharmacists can diagnose and treat many minor skin complaints using simple over the counter non prescription items.
- Pharmacists can play a role in the primary care team working alongside doctors and nurses to help patients manage acute and chronic skin problems.
- Pharmacists can advise patients on the correct method of applying medications such as creams and gels and warn them about possible side effects and what to look out for.
- Pharmacist also monitor drug treatments for skin diseases checking for interactions with other medication and for drug allergies.

## What to Tell the Patient

- For minor skin ailments such as sun burn, mild acne, mild eczema or athletes foot your pharmacist may be able to diagnose and treat your skin problem.
- One of the key measures in skin cancer prevention is the careful use of high potency sun protection factors which your pharmacist should be able to advise you on. These are also one of the best anti-aging measures you can adopt.

## 61.1 Introduction

Skin care products account for 17% of pharmacy over-the-counter (non-prescription) sales [1]. Symptomatic skin problems make up to 12–23% of all symptom based requests for advice from pharmacists. Pharmacies see a wide variety of skin complaints that can vary according to the seasons (Table 61.1). Simple advice from the community pharmacists can enhance the effectiveness of over-the-counter and prescription skin care products. The community pharmacist can also facilitate effective self care for patients with dermatology problems who wish to treat themselves. Patients often present to their pharmacist before their doctor with skin problems because of ease of access to the pharmacist and it may be less expensive.

**Table 61.1** Common ailments seen by the community pharmacist

Spring/summer	Autumn/winter
Insect bites and stings	Acne
Sun burn	Eczema/dermatitis
Cuts and scrapes	Dry skin
Cold sores (Herpes simplex)	Psoriasis
Warts, verrucas and corns	Dandruff
Hives (urticaria)	Head lice
Thrush (candidiasis)	Athletes foot (Tinea pedis)
	Impetigo

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## 61.2 Diagnosis

When a patient presents to the pharmacist with an undiagnosed skin condition, the pharmacist will have to assess the patient and decide whether to refer them on to their GP or treat them with simple over the counter products. As in general practice, the range of diagnostic skills in dermatology amongst pharmacists is very variable. Further training in dermatology for pharmacists could be beneficial.

## 61.3 Treatment of Skin Diseases

The pharmacist's ability to manage skin problems is limited as they cannot at present dispense certain common prescription topical and systemic treatments such as potent topical steroids or antibiotics without a doctor's prescription. However, when treating common skin conditions such as eczema, over-the-counter products such as emollients, soap substitutes and the careful use of gloves are often as important, if not more important, than the use of prescription items such as potent topical steroids.

## 61.4 Health Promotion

Health promotion and disease prevention is also an important part of the pharmacist's role. In skin care, this primarily centres on advice regarding protection of the skin from excessive ultraviolet light to prevent skin aging and skin cancers. Sunblocks play an important role in this respect. Pharmacies should have a basic knowledge about the early signs of skin cancer especially melanomas. Patients who must avoid sun exposure completely such as those with photosensitivity or those who have previous skin cancer, will need advice on vitamin D supplementation.

## 61.5 Chronic Disease Management

Pharmacists play an important role in chronic disease management of common skin illness such as acne, eczema and psoriasis. The first line treatment for many common skin diseases such

as eczema and acne is simple over-the-counter topical treatments that can be initiated by the pharmacist.

The pharmacist can reinforce the advice given by the GP, dermatologist or practice nurse about how to apply various topical treatments, how much to use, on which particular area of the body and how long it should take before improvements are seen. He/she should be able to discuss possible side effects of medication for dermatology problems such as those from potent topical steroids and be able to reassure the patient that when used correctly under careful medical supervision, side effects are very rare. Many topical acne treatments such as benzoyl peroxide and adapalene are potentially drying and irritating when first used. The pharmacist can advise the patients on how to apply these products sparingly initially all over the acne affected areas and not just onto spots. Pharmacist can also monitor drug treatments for skin diseases by looking for interactions with other medication and checking for drug allergies. In the UK, pharmacists are now included in primary care teams to enhance compliance and safety of treatments resulting in better outcomes.

Pharmacists need to be more precise when advising on topical treatments. For instance, it is common practice for pharmacists is to write "use sparingly" when prescribing potent topical steroid. This is meaningless as "sparingly" can be interpreted differently by different patients. Doctors and pharmacists should be encouraged to advise the patient how much of a particular topical steroid they can safely use on certain parts of the body per month (e.g. adults can use 120 g of a potent topical steroid per month for extensive eczema on the body but not on the face).

Another area of chronic disease management where the pharmacist can play a part is in the treatment and prevention of varicose eczema and varicose ulcers. Provided there is no evidence of peripheral vascular disease, pharmacists can measure and fit patients for high compression hosiery which can help heal small ulcers and prevent the recurrence of new varicose ulcers. Proper compression is more important than expensive wound dressings when managing varicose ulcers and varicose eczema.

**Hair and nail problems** are also commonly seen by community pharmacists. They have a

**Table 61.2** Range of conditions and over-the-counter treatments available from the community pharmacist in Ireland

Eczema/dermatitis/dry skin (see Chaps. 13, 14 and 62)	<ul style="list-style-type: none"> <li>– Emollients</li> <li>– Soap substitute and bath oils</li> <li>– 1% Hydrocortisone cream</li> <li>– Gloves</li> <li>– Wet wrap garments</li> </ul>
Acne (see Chap. 7)	<ul style="list-style-type: none"> <li>– Salicylic acid washes</li> <li>– Benzoyl Peroxide</li> <li>– Nicotinamide gel</li> </ul>
Fungal infection (see Chap. 31)	<ul style="list-style-type: none"> <li>– Imidazole creams (e.g. “Canestan®”)</li> <li>– Allylamine creams (e.g. “Lamisil cream®”)</li> <li>– Amorolfine nail lacquer</li> </ul>
Dandruff/Seborrhoeic Dermatitis/Pityriasis versicolour (see Chaps. 16 and 31)	<ul style="list-style-type: none"> <li>– Ketoconazole shampoo (“Nizoral®”)</li> <li>– Selenium Sulphide (“Selsun®”) (“Head &amp; Shoulders®”)</li> </ul>
Hyperhidrosis (see Chap. 12)	<ul style="list-style-type: none"> <li>– Simple underarm deodorants</li> <li>– Aluminium Chloride (“Anhydrol Forte®” or “Driclor®”)</li> </ul>
Psoriasis (see Chap. 15)	<ul style="list-style-type: none"> <li>– Coal tar e.g. “Exorex lotion®”, “Cocois ointment®”, Calcipotriol (“Dovonex®”)</li> </ul>
Warts/Verrucas/Corns (see Chap. 34)	<ul style="list-style-type: none"> <li>– Salicylic acid paints</li> <li>– Corn plasters</li> <li>– Files and blades</li> </ul>
Cold sores (see Chap. 32)	<ul style="list-style-type: none"> <li>– Acyclovir cream</li> </ul>
Scabies/Pubic and head lice (see Chap. 35)	<ul style="list-style-type: none"> <li>– Malathion</li> <li>– Permethrin</li> <li>– Dimethazone</li> </ul>
Urticaria hives and allergic reactions (see Chap. 20)	<ul style="list-style-type: none"> <li>– Non-sedating antihistamines</li> </ul>
Hair loss (see Chap. 40)	<ul style="list-style-type: none"> <li>– Volumizing shampoos</li> <li>– Minoxidil (“Regaien®” or “Rogaine®”)</li> </ul>
Varicose eczema	<ul style="list-style-type: none"> <li>– Compression stockings</li> </ul>
Varicose ulcers (see Chap. 37)	<ul style="list-style-type: none"> <li>– Ulcer dressings</li> </ul>
Skin cancer, photo protection and skin aging (see Chaps. 45 and 49)	<ul style="list-style-type: none"> <li>– Sunblocks (SPF)</li> <li>– Vitamin D</li> </ul>

wide range of over the counter products to help deal with these problems including dandruff, hair loss and fungal nail infections.

Table 61.2 outlines the range of conditions and the treatments available for use by community pharmacists in Ireland.

## 61.6 Conclusion

Pharmacists and doctors should work together as a team to provide the patient with skin disease the

best treatments available and to ensure they are used safely and effectively. The pharmacist can help the doctor source difficult to get or off-licensed drugs at a competitive price for their patients.

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# Emollients and Moisturisers

62

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## Key Points

- Most dry skin conditions should benefit from a moisturiser. The best moisturiser is the greasiest one the patient will use.
- The primary aim in the management of eczema is to reverse the dryness and reduce the itch by the appropriate use of emollients and the avoidance of soaps, irritants and allergens which should help restore the skin barrier function.
- Eczema sufferers should use hypo-allergic, oil based moisturisers that come in sufficiently large quantities and are relatively inexpensive, as they will have to be used in high quantities over a long period of time.
- Ointment based moisturisers are more effective but also more greasy to use.
- Sometimes giving the patients a few different moisturisers to use at different times of the day can help; for example a thick greasy moisturiser at night and a lighter less greasy moisturiser for work or school.
- Moisturising is steroid sparing.

## 62.1 What to Tell the Patient

- When using a greasy moisturiser, always rub it downwards as rubbing it up and down may cause irritation or infection to the hair follicles.

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- cles. If applied in excess, wait a few minutes and then rub down again.
- Sufficient moisturisers are necessary to cover the affected areas at least twice a day.
- Avoid moisturisers with perfumes, colourings and sulphates.
- Use approximately ten times more moisturiser than topical steroids with treating eczema.
- Keep away from fire or flames and do not smoke when using paraffin based moisturisers.
- Moisturisers are one of the best ways to relieve itch in dry skin conditions.

## 62.2 Introduction

The drug of first choice for most dry skin conditions such as eczema, psoriasis, or ichthyosis is a safe greasy moisturiser. The basic underlying problem in most forms of eczema is dry, itchy skin that leads to a defective skin barrier function, making the patient susceptible to infection, irritants and allergens. Scratching further compromises the skin barrier function. The primary aim in the management of eczema is to reverse the dryness, reduce the itch by the appropriate use of emollients and the avoidance of soap, irritants and allergens which should help restore the skin barrier function. Topical steroids (TS) and topical immunomodulators (TIM) such as tacrolimus, may ease itch and inflammation but they will do nothing for dry skin.

A recent Cochrane review showed that moisturisers showed some beneficial effects; prolonging time to flare, reducing the number of flares and the amount of topical corticosteroids needed to achieve similar reductions in eczema severity. Moisturisers combined with active treatment gave better results than active treatment alone. They did not find reliable evidence that one moisturiser is better than another [1].

It is important to show and demonstrate to the patient how to apply greasy moisturisers. They should be rubbed downwards like stroking a cat, especially on the hairy areas of the body, as rubbing up and down will irritate the skin and may cause folliculitis. It is useful to keep a wide range of various emollients and soap substitutes in the doctor's office to show the patients or parents what they look like, their consistency and to give them some idea how long a tub or tube should last (Fig. 62.1). If the patient wants to sample an emollient in a tub, remove some with a tongue depressor to give to the patient so as to avoid contaminating the tub with fingers.

The best moisturiser is the greasiest one the patient will use. They should be hypo-allergic and fragrance, preservative and SLS free. Cosmetic type moisturisers are unsuitable for eczema sufferers as they are usually too light, the quantities too small and they tend to be too expensive. In addition, they often contain many colourings, preservative and perfumes which the eczema sufferer should avoid. Eczema sufferers should only use hypo-allergic, oil based moisturisers that come in sufficiently large quantities and are relatively inexpensive, as they will have to be used in high quantities over a long period of time.



**Fig. 62.1** Emollient tray

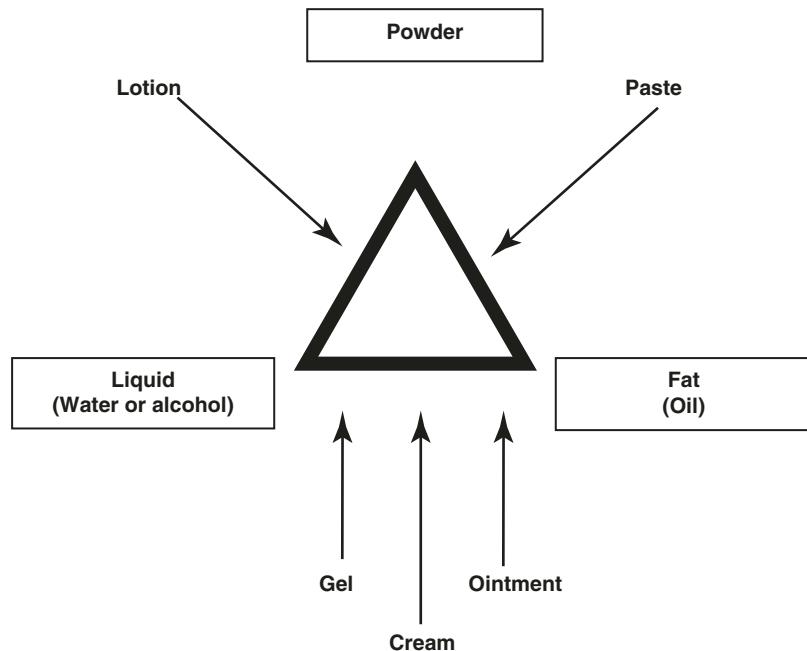
## 62.3 Ointments Versus Creams

Many years ago, the advice given to medical students was if a rash is wet (weepy), you should dry it with creams and if is dry you should wet it with ointments. Although there is still some truth in this, there are now a very wide range of topical formulations available for various situations (Table 62.1).

**Emulsifying ointment** is cheap, safe, effective water-in-oil emollient ointment. Because it is so greasy it only has to be applied twice a day, unlike some moisturising creams which may have to be applied every few hours. However, a lot of patients will not use emulsifying ointment as they find it too thick and greasy and it may sting the skin as it contains sodium lauryl sulfate (SLS) which is a surfactant used in many cleaning products and detergents.

It is important to give the patient a choice of different moisturisers—a greasy water-in-oil one such as “**Paraffin Gel**”, “**Epiderm ointment®**”, “**Hydromol ointment®**” or “**Diprobase ointment®**” (Table 62.2) and a less greasy oil-in-water emollient cream such as “**Aveno Dermexa®**”, “**Epiderm Cream®**” or “**Oilatum Cream®**” (Table 62.3). It is kinder and more realistic to get the patient to use the greasier but more effective moisturiser at home at night and to use the less greasy but cosmetically more acceptable moisturising cream during the day, especially if is needed on their face or hands at work or at school. “**Doublebase Emollient Gel®**” is half way between an ointment and a cream. It is highly moisturising yet cosmetically acceptable. It contains liquid paraffin, isopropyl myristate and glycerol which is a humectants which helps retain moisture in the skin (Table 62.4).

Sufficient quantities of moisturisers need to be given (Table 62.5). Adults with dry skin all over may need 1000 g/month if they are to apply their moisturiser all over twice a day. Children may need approximately half this amount. Emollients are steroid sparing. As a general rule of thumb, the patient needs ten times more moisturiser than topical steroid or TIM such as tacrolimus. So, if prescribing 100 g of a potent topical steroid every

**Table 62.1** Topical formulations**Cream:**

- Oil in water (mostly water) (e.g. Aqueous cream\*)
- Light and creams
- Cosmetically acceptable
- Contain preservatives which may cause allergies

\*Aqueous cream contains Sodium Lauryl Sulfate (SLS) which is a surfactant and detergent which should not be used in sensitive skin such as atopic eczema

**Ointment:**

- Water in oil (mostly oil) e.g. emulsifying ointment
- Thick and greasy – useful for dry skin
- Can be messy and sticky
- Occlusive effects enhances skin barrier function
- No preservatives
- Not suitable for hairy areas
- May cause folliculitis (“rub downwards”)

**Paste:**

- Powder and ointment (e.g. zinc oxide paste or Lassar's paste)
- Does not spread
- More sticky and more difficult to rub off

**Lotion:**

- Liquid preparation – usually has to be shaken to mix the content (e.g. “Betnovate Scalp Application®”, “Calamine lotion®”)
- The solvent evaporates to leave the active ingredient in contact with the skin and a cooling sensation
- Not sticky and dry fast (e.g. acne lotions)
- Suitable for hairy areas and acne prone skin

**Gel:**

- A water-alcohol mix (e.g. “Dovobet gel®”)
- Liquefy on contact with the skin leaving a thin film of active medication
- Drying, cosmetically acceptable
- Useful on hairy areas and acne prone skin

**Table 62.2** Common emollient ointments (water-in-oil)

Emulsifying ointment 500 g (contains SLS)
"Hydrous ointment®" 500 g
"Diprobase ointment®" 500 g
"Paraffin gel®" (50; 50 liquid paraffin/white soft paraffin) 500 g
"Hydromol ointment®" 500 g
"Epiderm ointment®" 500 g

**Table 62.3** Common Emollient creams (oil-in-water)

"E45®" 500 g (contains lanolin)
"Oilatum cream®" 500 g
"Epiderm cream®" 500 ml
"Diprobase cream®" 500 g
"Aveno Daily Moisturising Lotion®" 200 ml + 354 ml
"Aveno Dermexa cream®" 200 ml

**Table 62.4** Emollient Gels

"Doublebase emollient Gel®" 100 g/500 g
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**Table 62.5** Amount of cream or ointment required to treat a skin condition twice daily for a month

Age	Whole body	Both arms and legs	Trunk
6 months	144 g	80 g	64 g
1 year	184 g	104 g	64 g
4 years	240 g	144 g	80 g
8 years	320 g	200 g	144 g
12 years	480 g	264 g	184 g
16 years	624 g	344 g	224 g
Adult <sup>a</sup>	680 g	360 g	240 g

Adapted from Maurice PDL, Sainan EM. Br J Clin Pract 1985; **39:** 441–2

<sup>a</sup>70 kg male (All other figures given are a means of the values for male and female subjects)

month, 1000 g of moisturiser should be prescribed monthly.

## 62.4 Active Ingredients

Urea based moisturisers (e.g. "Calmurid®", "Eucerin®" or "Relief U=Life®") are less greasy but can sting if applied to broken skin. They act as a humectant which works by attracting water from the dermis below and helping to keep that water bound in the stratum corneum. 10%–30% urea based emollients can be useful in ichthyosis

and keratosis pilaris. It is also a good foot moisturiser. 3% urea is more suitable for the face and for children. Urea should be avoided in children under the age of 3 years old. Glycerol which is in "Doublebase Emollient Gel®" also acts as a humectant.

Glycyrrhetic acid (in Bioderma "Sensibo Rich Cream®") is a major metabolite of glycyrrhizin, one of the main constituents of licorice. Both glycyrrhetic acid and glycyrrhizin have been shown to exhibit anti-inflammatory, and immunomodulatory properties.

The active ingredients in all "Aveeno®" products are colloidal oats and/or oat extracts—avenanthramides, which have the most anti-irritant properties of all oat extracts. The "Aveeno Dermexa®" range is enriched with avenanthramides to help soothe and relieve itchy, dry skin conditions such as eczema. "Aveeno Dermexa®" is a good cooling cream, especially if it is stored in the fridge, and can be helpful with rosacea, flushing or sunburn.

"E45®" is a cheap moisturising cream but it contains lanolin (wool alcohol) to which some people may be allergic, especially if they have atopic eczema. "Oilatum cream®" contains light Liquid Paraffin and White Soft Paraffin which exert an emollient effect by forming an occlusive film which reduces trans-epidermal water loss, thus helping to maintain normal skin humidity levels. Oilatum cream also contains polyvinyl pyrrolidone which enhances the strength and longevity of the occlusive film on the skin.

Greasy moisturisers should be avoided in acne prone skin. Oil free, non comedogenic moisturising creams should be used in these areas, if required, because of the overuse of anti-acne topical therapies or from oral isotretinoin. Most patients with acne have oily skin and do not need moisturising.

Paraffin based moisturisers are **flammable**. The risk is greater when these preparations are applied to large areas of the body and clothing or dressings become soaked with the emollient. Patients should be told to keep away from fire or flames and not to smoke when using these preparations.

**Zinc and castor oil** is a good lubricant and acts as a barrier cream. It is most commonly used for babies bottoms to treat and prevent nappy rash. It is also useful in adults who have problems with skin friction and irritation such as pruritus ani, pruritus vulva, or intertrigo.

Tar based products such as 10% coal tar, 10% tar and 10% urea, or “**Exorex Lotion®**” (5% coal tar solution) can be helpful for dry skin conditions especially psoriasis and it offers some mild anti inflammatory effects.

Topical salicylic acid is sometimes added to a moisturiser or tar (e.g. “**Cocois®**”) as it acts as a keratolytic agent, de-scaling thick, scaly skin conditions such as scalp psoriasis.

Non-paraffin based moisturisers such as coconut oil, bees wax or olive oil may suit some patients who are intolerant to paraffin based products which are made from crude coal tar. It may be helpful to give the patient a small amount of two or three different types of moisturisers to try out to see which one suit them best and which is most cosmetically acceptable for them. They could rub one sample downwards on one arm and another on the opposite arm for a few days. It should soon become apparent which one is most effective and acceptable.

**Wet wraps** are another way of locking moisturisers into the skin and can prevent skin damage created by scratching. Wet wrap garments (tight cotton tops and pull ups) are more popular and more practical than tubular dressings that were used in the past. A damp garment is put on after moisturising the skin and this is covered with a second dry garment. These can be left on overnight or 24 hours and usually ease itch considerably in children with severe atopic eczema. Topical steroids may be put on the badly affected areas under the wet garments.

Some light moisturisers come in pump dispensers which are handy for measuring out a fixed amount and stops dirty fingers going into the pot contaminating the contents. Thick, greasy moisturisers will not work in a pump dispenser. When taking ointments out of the pot please instruct the patient or parent to use a spoon so as to avoid contaminating the pot.

## 62.5 Soap Substitutes and Bath Oils

It is very important to encourage the patient with dry skin to avoid soaps and other irritants such as shampoo, shower gels, bubble baths, detergent and washing up liquids. All of these agents will break down what little natural oils the eczema sufferer will have left in their skin and they will make their eczema worse. Safe alternatives such as “**Elave Wash®**” and “**Elave Shampoo®**” or “**Aveno Wash®**” should be encouraged. People whose job involves getting their hands wet a lot such as homemakers, hairdressers, kitchen workers, plasterers and dairy farmers should wear gloves for all wet work, not only when their hand rash is troublesome, but also as they improve to prevent relapse. They should also use safe soap substitutes and use a soap free shampoo or else wear surgical gloves when washing the hair using their ordinary shampoo.

Emulsifying ointment and “**Epiderm Ointment®**” can also be used as soap substitutes but they are not as comfortable to use as the proprietary soap substitutes such as “**Elave Wash®**” or “**Aveno Body Wash®**”. “**Dove soap®**” and “**Simple soap®**” should be avoided in patients with dry skin. “**Aqueous Cream BP**” and “**Silcocks Base BP**” can be used as a soap substitute. Despite claims from the manufacturers, these should not be used as a moisturiser as they may contain sodium lauryl sulfate (SLS) which is a surfactant used in many cleaning products and detergents. Leaving “**Aqueous cream BP**” or “**Silcock’s Base BP**” on the skin is akin to leaving shampoo on the skin which is obviously not a good idea for eczema sufferers.

Bath oils may help to get moisturisers into the skin, especially in children, although recent evidence for their effectiveness is poor [2]. Emulsifying ointment or “**Epiderm Ointment®**” can be dissolved in a small amount of boiling water and added to the bath but they usually float as globules on the top of the bath water. It is much better to use special bath oils such as “**Oilatum®**” or “**Aveno Bath Emollients®**” which will dissolve easily in the bath water and soak into the skin. Great care should be taken as bath oils make the bath very slippery. Bath mats should be

encouraged to avoid falls and baths should be limited to 5–10 min. After getting out of the bath the skin should be patted dry with a soft cotton towel and immediately covered in a greasy moisturiser to “lock in” the bath oils. For infective exacerbations of eczema “**Oilatum Plus Bath Emollient®**” (or “**Oilatum Junior Flare Up®**” for children) can be used daily for a week as they contains antiseptics which help clear infections.

An alternative for recurrent skin infection is “**Milton® baths**” twice a week but this may have to be combined with a bath emollient (see patient info leaflet on Milton baths in Chap. 66). However, there are very few good clinical studies published in the literature on the use of bleach baths for the adjunctive treatment of patients with infected atopic eczema [3]. **Potassium permanganate soaks** are also good for skin infection.

## 62.6 Conclusion

When treating dry skin conditions, it is often more important to advise the frequent use of an appropriate moisturised (Table 62.5) and soap substitute than prescribing topical steroids.

The management of dry skin requires a lot of hard work by the patient since they will constantly have to moisturise their skin and change most of their toiletries and cosmetics not just when their skin is irritated, but for the long term. Patients or parents should be given written information on the choices of emollients, bath oils and soap substitutes available over the counter in the pharmacy and how to use these products. (See Chap. 66 PIL. “Management of dry sensitive skin conditions”).

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## References

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# Steroids in Dermatology

63

David Buckley

## Key Points

- Used properly and appropriately, under careful medical supervision, topical steroids (TS) are very safe and effective.
- Underuse of topical steroids is now a more common problem than overuse and abuse.
- Compliance with treatment will be improved if a frank and honest discussion is carried out with the patient about the risk and benefits of TS especially if the patient or parent is a steroid phobic.
- The safe use of topical steroids involves knowing how much and which potency of topical steroids to put on which part of the body and for how long.
- Ointments have a greater penetration than creams so TS that have an ointment base are considered more potent than the same TS in a cream base.
- Wrapping an area where TS has been applied with wet wraps or kitchen cling wrap will increase its potency, reduce chances of losing the cream on clothes or sheets and reduce scratching. However, it can increase the risk of skin thinning if continued too long.

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## 63.1 What to Tell the Patient

- When used under careful medical supervision, topical steroids (TS) are safe and effective for a wide range of inflammatory skin disease.
- As a general rule nothing stronger than a weekly potent TS (e.g. 1% hydrocortisone) should be used on the face in adults and children and nothing stronger than a moderately potent TS should be used on the body in children or potent TS on the body in adults.
- TS should always be reduced or stopped gradually if they have been used for more than a few weeks, as suddenly stopping them may result in a rebound flare of the underlying conditions (e.g. Psoriasis).

## 63.2 Introduction

The introduction of topical steroids (TS) in the 1950s is still one of the greatest advances in dermatology as they provide substantial relief in a wide range of dermatoses in a safe, cosmetically acceptable form (Table 63.1). Overuse of very potent topical steroids in the 1970s caused local adverse effects such as skin atrophy, striae, telangiectasia or systemic side effects such as hypothalamic–pituitary–adrenal axis (HPA axis) suppression and Cushing's disease, and gave steroids a bad name generally (Fig. 63.1). Now most doctors understand that, used properly and appro-

**Table 63.1** Steroid responsive dermatoses

<b>Eczema/dermatitis</b>
Atopic eczema
Contact dermatitis
Pompholyx
Seborrhoeic dermatitis
Varicose eczema
Discoid (nummular) eczema
Neurodermatitis (lichen simplex chronicus)
<b>Papulo squamous</b>
Lichen planus
Psoriasis
<b>Autoimmune</b>
Lupus (DLE,SLE)
Alopecia areata
Morphea
<b>Vesicular/bullous</b>
Dermatitis herpetiformis
Bullous pemphigoid
Pemphigoid vulgaris
<b>Miscellaneous:</b>
Keloids + hypertrophic scars
Papular urticaria
Lichen sclerosis
Pruritus ani
Pruritus vulva
Pyoderma gangrenosum
Polymorphic eruption in pregnancy

priately, under careful medical supervision, topical steroids are very safe and effective. However, the general public still needs a lot of reassurance and education about their benefits. In one recent Italian study, 81% of parents of children with atopic eczema reported to have a certain amount of fear of TS [1]. Underuse of topical steroids is now a more common problem than overuse and abuse.

TS have anti inflammatory and immunosuppressive effects. They also have anti-mitotic effects which can cause steroid atrophy. In addition they have a vasoconstriction effect which is sometimes used as a grading scale to judge their potency. They can also cause skin whitening: this is the reason why they are included in cosmetic products used to reduce hyperpigmentation.

The safe use of topical steroids involves knowing how much and which potency of topical ste-

**Fig. 63.1** Echymosis and skin thinning from very potent topical steroids

roids to put on which part of the body and for how long. While every effort should be made to make an accurate diagnosis before commencing topical steroids this is not always possible or practical. Once skin infection, infestation, malignancy, rosacea and perioral dermatitis can rule out, it is usually quite safe to give a trial of topical steroids. Topical steroids should not be put in or near varicose ulcers. Table 63.2 shows possible reasons why topical steroids are not working (Fig. 63.2).

### 63.3 Potency

Mild and moderately potent steroids (Table 63.3) are considered safe on the body, even on children. 1% Hydrocortisone cream is considered so safe it can be purchased over the counter in the pharmacy without a prescription from a doctor. 1%

**Table 63.2** Possible reasons why apparent steroid responsive dermatoses fail to respond to topical steroids include

- Too little or too weak (e.g. parental or medic anxiety about steroids)
- Too strong or too much (= telangiectasia or atrophy)
- Emollients = not enough or not greasy enough
- Irritants = too much
- Disease is too severe (may need UVB or systemic treatment)
- Infected: e.g. bacterial (impetigo), viral (herpes or varicella-zoster), fungal (tinea)
- Infestation (e.g. scabies)
- Allergies (Contact allergic dermatitis, foods, drugs, gluten, HDM, to steroid base, etc)
- Underlying diseases: Varicose veins, renal or liver failure, thyroid disease, iron deficiency, diabetes, HIV, pregnancy, para-neoplastic, lupus, etc
- Incorrect diagnosis (e.g. neoplasm such as Bowen's disease, urticaria, pre-bullous pemphigoid etc)
- Psychogenic causes: delusion of infestation, dermatitis artefacta
- Used on a steroid aggravated skin condition such as peri-oral dermatitis or rosacea



**Fig. 63.2** Worsening of tinea paedis (tinea incognita) from applying a very potent topical steroid

hydrocortisone is considered so weak (600 times weaker than clobetasone) that it is of little help on the body, hands or feet in adults. Potent TS should be avoided in children and very potent TS should never be used in children. Potent TS should be avoided on the face and flexures in children and adults. Very potent steroids are safe on the body in adults who may require them for troublesome corticosteroid-responsive dermatoses, provided they are used in relatively small quantities (e.g. 60 g/month for chronic use; larger amounts can be used for short term use) (Table 63.4). Many patients get a weak TS for the face (e.g. 1% Hydrocortisone) and a moderately potent (in children) or potent (in adults) TS for the body.

Patients often get confused with the percentages of the various TS commonly available. For instance, clobetasol propionate **0.05%** ("Dermovate Ointment®") is a lot more potent than betamethasone valerate **0.1%** ("Betnovate ointment®") which in turn is a lot more potent than **1%** hydrocortisone ointment.

Stronger steroids will usually induce a rapid remission which can then be maintained with the less potent varieties (the step down approach). Alternatively, reducing the potent topical steroid to twice a week on the areas that tend to flare up during exacerbations (e.g. the backs of the knee and front of the elbows) may help maintain the improvement long term. Sometimes potent or super potent topical steroids may have to be continued for a few months in conditions like vitiligo. In this situation, it might be safer to use them

**Table 63.3** Topical steroid potency

Potency	Example	Trade Names	Potency ratio
Super potent	Clobetasol propionate	"Dermovate®"	600
Potent	Betamethasone(as valerate) Betamethasone dipropionate Hydrocortisone butyrate Mometasone furoate	"Betnovate®". "Diprosone®". "Locoid®". "Elocon®".	100
Moderately potent	Hydrocortisone butyrate Alclometasone dipropionate	"Eumovate®". "Modrasone®".	25
Weak	Hydrocortisone 0.1–2.5%	1% hydrocortisone. "Diaderm®"	1

**Table 63.4** Maximum amount of topical steroids per month for chronic use<sup>a</sup>

Potency	Age adult	12 years	3 years	Infant < 12/12 months old
Mild	No max	No max	200 g	100 g
Moderate	200 g	100 g	60 g	30 g
Potent <sup>b</sup>	90 g	30 g	15 g. For acute use only	Avoid
Very potent	30–60 g	Avoid	Avoid	Avoid

Greater than 2 months duration. Higher amounts can be used for a short period of time in *acute* flare ups

<sup>a</sup>Adapted from: Position paper on diagnosis and treatment of AE. EADV (2005)19, 286–295

<sup>b</sup>Four times this amount can be prescribed if using “Betnovate RD®”

in cyclical fashion (e.g. 2 weeks on and 2 weeks off every month) and this may reduce the risk of local side effects such as skin thinning, striae or telangiectasia. Some of the newer potent topical steroids such as mometasone furoate (“Elocon®”) may be safer for chronic use. TS should always be reduced or stopped gradually if they have been used for more than a few weeks, as suddenly stopping them may result in a rebound flare of the underlying conditions (e.g. psoriasis).

Sometimes combining topical steroids with other topical agents such as an antibiotic (“Fucidin H®” or “Fucibet®”), urea (“Calmurid HC®”), tar (“Alphosyl HC®”), antifungals (“Daktacort®” or “Canesten HC®”) or salicylic acid (“Diprosalic®”) may have a steroid sparing effect.

As a general rule, nothing stronger than 1% hydrocortisone should be used on the face or in flexures. One exception is in DLE (lupus) on the face where potent or very potent steroids may sometimes be required. A potent topical steroid (e.g. “Elocon®”) is sometimes necessary for severe dermatosis on the face in adults but it should be stopped after five days, as prolonged use may result in side effects such as skin thinning, telangiectasia (broken veins), steroid rosacea (Fig. 63.3) or perioral dermatitis. Nothing stronger than 1% hydrocortisone cream should be used on the delicate eyelid and genital skin, especially in children, and they should only be used short term in this area. Prolonged use on the eyelids increases the risk of skin thinning, cataracts and glaucoma. Topical immune modulators such as tacrolimus (“Protopic®”) are safe on the delicate eyelid skin for more resistant eczema/dermatitis.

A potent TS should be avoided in the genitalia and perianal areas except when treating resistant vulval or perianal rashes such as lichen sclerosus



**Fig. 63.3** Steroid rosacea from using a potent topical steroid on the face for 3 years

but a once a day application is usually sufficient in these areas. The potency of the steroid should be reduced as soon as the symptoms improve as the occlusive effects of the folds of skin in the flexures increase the steroid effects but also the risk of skin atrophy and striae. Very potent topical steroids (e.g. “Dermovate®”) are usually reserved for very resistant dermatosis such as severe DLE, alopecia areata and keloid scars. The

long term management of very potent steroids is best given under specialist supervision.

A short course of a potent topical steroid may sometimes be required in children with a severe flare up of their eczema but the potency should be reduced after a maximum of 7 days. This is similar to using a short course of oral steroids in children with an acute exacerbation of asthma. But topical steroids are more effective and safer than oral steroids for most skin conditions since the topical steroid goes straight to the target organ. While **oral steroids** are very rarely required for skin problems in primary care, they may sometimes be necessary for severe, extensive skin conditions such as uncontrolled atopic eczema, bullous pemphigoid, pemphigus vulgaris, SJS/TEN or lichen planus and in these conditions they should be used under the supervision of a doctor with allot of experience in dermatology.

### 63.4 The Fingertip Unit (FTU)

It is important to prescribe enough TS to cover all the affected areas with a thin film of the cream or ointment. Pharmacists sometimes put the

comment “use sparingly” on the tube. This is unhelpful and misleading. It can give the impression that the steroid is very dangerous and “sparingly” does not give the patient any indication of how much to use. A useful way of knowing the correct amount to use is the fingertip unit (FTU) method of measurement. A FTU is the amount of cream or ointment that comes out of the opening of a standard tube with a 5 mm nozzle that extending from the tip of an adult index finger to the first finger crease. This amount of cream or ointment should be enough to cover an area of skin the size of two flat adult palms of the hand including fingers. This is approximately 0.5 g if measured using an adult male finger and 0.4 g in an adult female finger. It is more helpful for the patient if you give them an estimate of how long a particular sized tube of a specific potency topical steroid should last if applied to the affected area once daily. An adult with eczema affecting the front of the trunk may need 7 FTU/day which is equivalent to 3.5 g/day or 95 g/month if applied once daily. Therefore the patient should be given a 100 g tube of the TS and ten times this amount of moisturiser (1000 g) per month (Fig. 63.4).

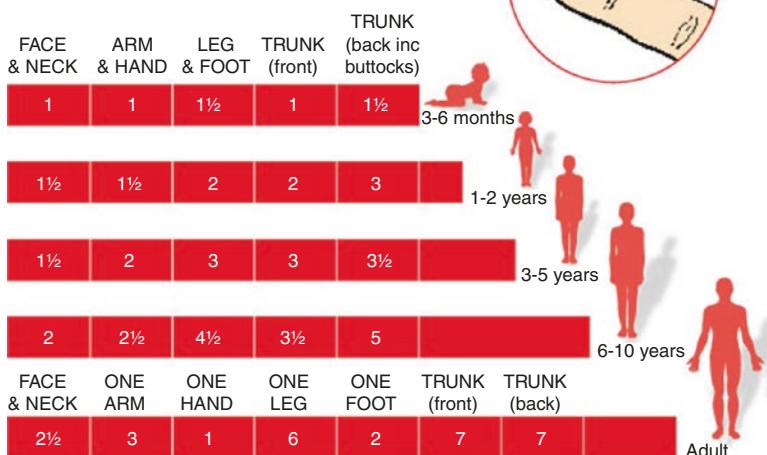
**Fig. 63.4** Finger tip unit measurement

#### The fingertip unit method\*

FTU = Fingertip unit(adult)

1 FTU = 1/2 g of cream or ointment.

Measurement based on 5mm nozzle.



### 63.5 Topical Steroid Vehicle (Base)

**Ointments** contain less potential sensitisers and will help moisturise dry skin. They should be used in preference to **creams** unless the eczema is weepy in which case a cream base may be required. Ointments have a greater penetration than creams so TS that have an ointment base are considered more potent than the same TS in a cream base. Greasy ointments may be unacceptable on the face and hands for some patients. Be careful with steroid/moisturiser mixtures (e.g. "BetnovateRD 1:4<sup>®</sup>") since diluting the steroid will not make them less potent and patients often use these mixtures to moisturise the skin when a simple emollient would have been sufficient. **Gels** (e.g. "Dovobet Gel<sup>®</sup>") and lipocream (e.g. "Locoid Lipocream<sup>®</sup>") are not as messy as ointments but not as drying as creams.

**Lotions** (e.g. "Betnovate Scalp Application<sup>®</sup>"), foams (e.g. "Bettamousse<sup>®</sup>") and gels (e.g. "Dovobet Gel<sup>®</sup>") are convenient and less messy for hairy areas such as the scalp. Steroid based shampoos such as "Etrivex Shampoo<sup>®</sup>" which contains a very potent TS (clobetasol propionate) can be very effective and convenient for severe scalp dermatoses (e.g. scalp eczema or dermatitis). "Etrivex shampoo<sup>®</sup>" should be applied directly to the dry scalp once daily to the affected areas taking care to avoid getting any of the shampoo in the face. An amount equivalent to a half tablespoon approximately (~7.5 ml) per application is sufficient to cover the entire scalp. "Etrivex shampoo<sup>®</sup>" should be kept in place for 15 min before wetting the scalp which forms a lather which can then be rinsed out and the hair dried in the usual way. Hands should be washed carefully after application. The shampoo can be used daily initially if necessary and then reduced as the symptoms improve. The treatment duration should be limited to a maximum of 4 weeks.

Steroid ointments and creams can be used under wet wraps or with **occlusion** such as cling film. It can also be applied with a tape impregnated with a moderately potent topical steroid (fludrocytide) that can be cut to the appropriate size and stuck onto the affected area for

24 hours or longer (e.g. "Haelin Tape<sup>®</sup>"). This can be useful for overgranulation (over healed ulcers), keloid scars, small patches of eczema, fissuring (fingers, hands, heels and soles), lichen simplex, lichen planus, nodular prurigo and stoma sites. Occlusion makes the steroid more potent and effective but also increases the risk of local or systemic side effects.

**Intralesional** steroids injections such as methylprednisolone ("Depomedrone<sup>®</sup>") can be injected with a needle of an air gun (e.g. "Dermajet<sup>®</sup>") and can be useful for deep inflammatory conditions such as alopecia areata, keloids, acne cysts, DLE, necrobiosis lipoidica and lichen simplex chronicus (neurodermatitis). The injections may have to be repeated every 1–3 months. Injections can be painful and may cause local side effects such as skin atrophy, telangiectasie, pigment changes and infections. Systemic absorption is rare when treating a keloid.

Systemic absorption may also cause problems such as HPA axis suppression and diabetes especially if high doses are used over a long period of time in children.

### 63.6 Compliance

Compliance with treatment will be improved if a frank and honest discussion is carried out with the patient about the risk and benefits of TS especially if the patient or parent is steroid phobic [1]. Patient information leaflets on the safe use of topical steroids can help compliance and reduce the risk of side effects (see Chap 66. PIL, "Safe use of topical steroids").

Compliance can also be improved if the patient is given a reasonable treatment schedule for applying their topical treatments especially for busy people. TS can be applied to the badly affected areas once daily at bedtime and a suitable moisturiser can be applied more generously to the affected areas in the mornings and after showers. If a TS has to be applied around the same time as a moisturiser, the moisturiser should be applied generously to the general area first and the TS should be applied on top of the moisturiser to the badly affected areas only.

### 63.7 Conclusion

When used under careful medical supervision, TS are safe and effective for a wide range of inflammatory skin disease. Emollients are often more important than TS when managing dry skin conditions. As a general rule nothing stronger than a weekly potens TS (e.g. 1% hydrocortisone) should be used on the face in adults and children and nothing stronger than a moderately potent TS (e.g. 0.5 mg clobetasone butyrate, "Eumovate®") should be used on the body in

children. Greater caution should be taken when using high potency TS, especially in pregnancy (see Chap. 25), when using TS in large areas of the body and when using TS on delicate skin (e.g. eyelids, genitalia, perianally) or under occlusion.

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### Reference

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# Topical Immunomodulators (TIMs)

64

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## Key Points

- Topical immunomodulators (TIMs) like tacrolimus (“Protopic®”) or pimecrolimus (“Elidel®”) are a safe alternative to potent topical steroids especially on the face and in children.
- TIMs are safe to use on the body, face, genitalia and flexures.
- TIMs should not be used if there is skin infection.
- Although only licenced for atopic eczema, TIMs can be helpful in a wide range of inflammatory dermatoses.
- TIMs are not licenced for children under the age of 2 years old but are sometimes used by skin specialists in this age group off-licence as they are considered safer than potent topical steroids in children.
- Tacrolimus is a large molecule and will not penetrate dry, thickened, scaly skin such as psoriasis, hand dermatitis or neurodermatitis.
- TIMs can cause a transient worsening of eczema in the first week of use in up to 50% of patients but the majority of these will start seeing improvements in their eczema in the second and subsequent weeks.
- Other possible side effects include facial flushing with alcohol, headache and a flu-like illness.
- TIMs should always be used in conjunction with generous applications of an appropriate emollient and the avoidance of soaps, detergents, irritants and allergens.
- TIMs are considerably more expensive than topical steroids and should not be used in pregnancy, when breast feeding or in immunosuppressed patients.

## 64.1 What to Tell the Patient

- TIMs are as potent as a potent topical steroid without the steroid side effects.
- TIMs are slow to work and often take 2 or 3 weeks to have a good effect.

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## 64.2 Introduction

TIMs are potent anti-inflammatories that are licenced for the treatment of atopic eczema in adults and children over the age of 2 years old which is resistant to or unsuitable for treatment with conventional topical steroids. TIMs are calcineurin inhibitors which are as potent as some potent topical steroids without the inherent steroid side effects such as skin thinning, telangiectasia, striae or HPA Axis suppression from systemic absorption. This is why they are considered safe on the face, in flexures and in children where potent topical steroids would

normally be avoided. According to the Summary of Product Characteristics (SPC), TIMs should be initiated by a physician with experience in the diagnosis and treatment of atopic eczema. This should include almost all general practitioners.

Although TIMs are very effective for more resistant atopic eczema, they should still be reserved for second line treatment where topical steroids are ineffective or unsuitable because of side effects. The main disadvantage of TIM's is that they are much slower than topical steroids to exert their anti-inflammatory effect and up to 50% in patients will develop the transient irritation and apparent worsening of their eczema in the first week of use until the drug begins to work. If irritation occurs it normally settles in the second and subsequent weeks of use. As small proportion of patients cannot tolerate TIM's even after the first week of use. TIMs are also considerably more expensive than topical steroids. They are not licensed for use in pregnancy, breast feeding, in children under the age of 2 years or in infected skin.

TIMs are considered a monotherapy which means that the one treatment can be used in all areas of the body including the face and flexures. In clinical practice TIMs may be used in difficult to treat areas such as the face and flexures and topical steroids may be used on the body since they are considerably less expensive and the response is more predictable. They should always be used in conjunction with generous applications of an appropriate emollient and the avoidance of soaps, detergents, irritants and allergens.

TIMs should not be used if there is skin infection (bacterial, viral or fungal). There is also a theoretical risk that TIMs may predispose to skin cancer because of their immunosuppressant effect. It is recommended to avoid excessive ultra violet light on areas where TIMs are being applied.

The two main TIMs available are tacrolimus ("Protopic®") ointment and pimecrolimus ("Elidel®") cream.

### 64.3 Tacrolimus ("Protopic®") Ointment

Tacrolimus belongs to a group of drugs called macrolide lactones. It works by reducing the activity of the lymphocytes and reduces the number of IL8 cytokine receptors on the keratinocytes which reduces inflammation. The oral version of tacrolimus is known as "Prograf®" which is used to prevent rejection in solid organ transplant recipients. Tacrolimus ointment comes in two strengths; 0.1% for adults and 0.03% for children aged from 2 years old to 16 years old. It comes in two sizes; 30 and 60 g. It is licenced for use in moderately to severe atopic eczema which is resistant to or unsuitable for treatment with topical steroids.

Unfortunately tacrolimus is not licenced for children under the age of 2 years. Ironically, this is the group of patients where we are most in need of a topical steroid alternative when dealing with severe, resistant atopic eczema. Many pediatric dermatologists use tacrolimus "off-licence" in this age group since tacrolimus is almost certainly safer than a potent topical steroid in small children. Paediatric dermatologists sometimes use the higher strength (0.1%) of tacrolimus off-licence in children aged 2–16 years of age with more severe resistant atopic eczema.

While tacrolimus is only licenced for atopic eczema, it is sometimes used "off-licenced" for a number of other common dermatosis (Table 64.1).

Tacrolimus is particularly useful on thin skin such as the face, eyelids, flexures, genitalia and perianal areas. It is a relatively large molecule and will not penetrate dry, thickened, scaly skin such as psoriasis, hand dermatitis or neurodermatitis. Tacrolimus should not be used in patients with congenital or acquired immune-deficiency. It should not be used in pregnancy and should be avoided in breast-feeding mothers. Tacrolimus is a useful alternative in patients or parents who are "steroid phobic".

Tacrolimus is licenced both for acute flare-up of atopic eczema and also as a maintenance treat-

**Table 64.1** Non-licensed users of tacrolimus

- Facial psoriasis
- Flexural psoriasis
- Lichen planus
- Seborrhoeic dermatitis
- Pruritus ani
- Pruritus vulvae
- Discoid eczema
- Vitiligo
- Vasculitis
- Pyoderma gangrenosum
- Lichen sclerosus

ment. For flare-up it should be applied twice a day for 3–6 weeks. If no improvement is seen after 2 weeks of treatment, further treatment options should be considered. For maintenance treatment, tacrolimus should be applied once daily, twice weekly (e.g. Monday and Thursday) to the areas commonly affected by atopic dermatitis to prevent flare-ups. Maintenance treatment should be reviewed every 12 months. If signs of a flare-up occur, twice daily treatment should be reinitiated for up to 3–6 weeks.

The most common side effect with tacrolimus ointment is a transient irritation and apparent worsening of the atopic eczema in the first week of use. This occurs in about 50% of patients and it is impossible to predict who will get this reaction. All patients should be warned of the possibility of a transient irritation in the first week and should be reassured that most patients will improve in the second and subsequent weeks of treatment. If there is no improvement after 2 weeks of treatment, the treatment should be stopped as some patients (~10%) can be intolerant of tacrolimus on an ongoing basis. Other side effects include facial flushing with alcohol, headache and a flu-like illness may occur. Tacrolimus should not be used under occlusion.

Concern for potential systemic absorption resulting in possible immune suppression has led to caution with regard to the administration of live vaccines like Measles, Mumps and Rubella (MMR), oral polio and BCG in patients using TIMs, with some avoiding live vaccines for up to 28 days before initiation and after cessation of topical tacrolimus [1]. However, a study in 2006

showed that the immune response to vaccination against meningococcal serogroup C in children 2–11 years old with atopic dermatitis applying either 0.03% tacrolimus or a potent topical steroid did not affect the immediate response to vaccination, generation of immune memory or humoral and cell-mediated immunity [2]. In another US study, the serological response to pneumococcal polysaccharide vaccine was unaffected by 7 weeks of treatment with 0.03% tacrolimus in children 2–12 years old [3]. The Immunisation Guidelines for Ireland recently confirmed that live vaccines can be given to those receiving topical calcineurin inhibitors [4].

“Protopic®” ointment should not be used during pregnancy. Although clinical data have shown that systemic exposure from application of tacrolimus ointment is low, breast-feeding during treatment with tacrolimus (“Protopic®”) ointment is not recommended.

#### 64.4 Pimecrolimus (“Edelel®”) Cream

This is another TIM and a calcineurin inhibitor. It is an ascomycin macrolactam inhibitor which comes in 1% cream and in a 15 g tube. Its mode of action similar to tacrolimus but it is considered less potent and is indicated for mild to moderate atopic eczema which is not responding to or unsuitable for topical steroids. It is licenced for adults and children over the age of 2 years old, although in some countries it can be used in children down to the age of 3 months old. It can be used twice a day for at least 3–6 weeks in all areas of the body including the face, flexures and eyelids. A short course of topical steroids may be necessary to control an acute flare-up before using pimecrolimus for managing milder eczema and to prevent relapse. Like tacrolimus, pimecrolimus should not be used if there is a skin infection, in pregnancy, in breastfeeding or under occlusion. It may cause transient irritation in the first week of use in some patients. If there is no improvement after 2 weeks, an alternative treatment should be considered. Some patients may get facial flushing with alcohol or may develop

other less common side effects such as headaches or a flu-like illness when using pimecrolimus.

## 64.5 Conclusion

TIM's are a safe, effective alternative to potent topical steroids and are particularly useful for more severe, resistant dermatitis in children and on the face and flexures in adults and children. TIM's are slower to work than potent topical steroids and they can cause a transient irritation and apparent worsen of the eczema in up to 50% of patients in the first week of use (See Chap. 66. PIL. "Tacrolimus and pimecrolimus"). It is sometimes used off-label (unlicensed) for seborrhoeic dermatitis and psoriasis on the face, flexures and genitalia.

## References

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4. <https://www.hse.ie/eng/health/immunisation/hcpinfo/guidelines/>

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## **Part XIV**

### **Nurses, Patients, Courses and Websites**



# Nursing Care of the Dermatology Patient

65

David Buckley

## Key Points

- The practice nurse is a key player in community based dermatology and with appropriate training can help with wound care, leg ulcer dressings, allergy testing and wart treatments.
- Nurses may have more time and skills to educate the patients on the correct application of various topical medications such as emollients, anti-acne gels and psoriasis treatments.
- Nurses can be very helpful on chronic disease management such as monitoring isotretinoin or methotrexate treatment.
- Community based surgery would be very difficult without the assistance of a practice nurse or health care assistant.

## What to Tell the Patient

- The practice nurse may be able to monitor and treat many minor skin complaints such as cuts and wounds, ulcers, warts, and milder forms of eczema, acne and psoriasis.

## 65.1 Introduction

The best approach to prevent and treat various skin problems in the community is by team work: doctor, nurse, pharmacist and the patient. Nurses

have an important role to play in the care of patients with skin problems, both in primary and secondary care. In some situations the nurse may be the primary carer such as wound care for pressure sores, leg ulcers, diabetic ulcers, burns, traumatic wounds or post-operative wounds (see Chaps. 37 and 38). Nurses may also run wart clinics, psoriasis clinics or eczema clinics, independent of the doctor.

## 65.2 Topical Therapies

Nurses can be very helpful in the education of patients about the management of chronic diseases such as atopic eczema (see Chaps. 13 and 14) or psoriasis (see Chap. 15). They may have more time and skills to demonstrate how to apply appropriate emollients in sufficient quantities. They can also educate the patient or the parent on the appropriate use of topical steroids, topical calcineurin inhibitors and topical calcipotriol in adults and children. Techniques such as “wet wraps” and “Milton®” baths are best demonstrated to the patient or the family and the nurse might be the most appropriate person to do this.

Instructing patients on how to apply dithranol in gradually increasing strengths is complicated and takes time. It may be more appropriate to delegate this task to the nurse.

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Topical acne therapies are often drying and irritating. If the patient is not properly instructed on how to apply these products, they may get flaky or sore skin and stop their treatment prematurely. Careful counselling on the correct skin care and use of topical agents for acne is crucial and may be carried out by a nurse specially trained in acne care (see Chap. 7).

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### 65.3 Dermatological Surgery

Nurses are invaluable in the organisation, preparation and assistance in dermatological surgery (see Chap. 57). They play a vital role in ensuring there are adequate supplies of appropriate sterilised instruments, suture material, drapes, gloves and making sure liquid nitrogen is always available. If a practice is using reusable surgical instruments, the practice nurse is usually responsible for decontamination of these instruments including sterilising and packing them for use again. They can arrange for the transport of histological specimens to the laboratory and ensure the results come back and the patients are notified in a timely fashion. In some practices, a specially trained **Health Care Assistant** can take on some or all of these roles.

Nurses play a vital role in monitoring post-operative wounds to minimise the risk of infections. Removal of sutures is usually carried out by the nurse. Ideally the nurse who assists in inserting the sutures is the best person to remove the sutures 1 or 2 weeks later. Most sutures on the face can be removed after 7 days and on the body after 10 days. Sutures on the legs are normally left in for up to 2 weeks. Interrupted and mattress suture is relatively simple to remove. Running sutures and subcuticular sutures are sometimes more complex and the doctor who inserts the suture should instruct the nurse on how to remove them as there are often slight variations on the insertion techniques.

Nurses who remove sutures should be trained on the clinical features for post-operative wound infections and alert the doctor should they feel topical or systemic antibiotics may be required.

### 65.4 Allergy Testing

Nurses who are specially trained are probably the best healthcare professional to perform routine allergy testing such as skin prick testing and skin patch testing (see Chap. 21). Interpreting the results is the most difficult part of these allergy tests and results should be assessed in association with a detailed allergy history and thorough allergy physical examination by the treating doctor.

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### 65.5 Skin Cancer Screening and Treatment

Nurses can be trained on how to screen patients for signs of pre-cancer and cancer of the skin (see Chap. 45). They can also help in educating patients on how to minimise their exposure to ultra violet light and why they should take extra vitamin D. Nurses can educate patients on the early signs of skin cancer. Some nurses are involved in mole screening using techniques such as dermoscopy, digital photography and mole mapping. **Photodynamic therapy** treatments can be performed by a trained nurse who prepares the patient and stays with the patient during the procedure; post-treatment instructions are best handled by them too.

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### 65.6 Nurse Lead Wart Clinics

Nurses often lead up wart clinics, both in primary and secondary care. These nurses require extra training in lesion recognition to ensure that they are treating a wart and not a premalignant or malignant skin condition such as a keratoacanthoma, squamous cell carcinoma or a melanoma (see Chaps. 58 and 59) [1]. Nurses leading up wart clinics need extra training in cryosurgery (see Chap. 59). They should be encouraged to apply local anaesthetic for any warts greater than 5 mm as research has shown that the discomfort from a 30-gauge needle is at least half that of freezing a wart greater than 5 mm without local anaesthetic [2].

See also: “Nurse Led Cryosurgery Clinic” (Chap. 60) and “Ten rules for safer more effective cryosurgery” (Chap. 58, Table 58.1).

## 65.7 Professional Nursing Associations

There are very useful professional associations for nurses interested in dermatology such as the Irish Dermatology Nurses Association and the British Dermatology Nursing Group. These groups provide education and training for nurses in the community and in the hospital setting on skin care.

Public health nurses often work alone and are commonly left to deal with complicated wounds in the community. Basic skills such as measuring the ankle brachial pressure index using a vascular doppler probe is vital if the nurse is considering any type of compression bandaging or stocking for lower leg ulcers and wounds (see Chap. 37).

Practice nurses working in primary care with general practitioners should have good basic knowledge of wound care (see Chap. 38), chronic disease management and be able to detect the early warning signs of skin cancer.

Dermatology clinical nurse specialists (CNS) have extra training in specific areas such as phototherapy, patch testing, tissue viability or chronic disease management.

Advancer nurse practitioners (ANP) may specialise in areas such as skin surgery and may provide basic surgical procedures, usually in a

hospital setting, for benign and low grade malignant skin conditions that can be easily carried out under local anaesthetic such as ingrown toenails, sebaceous cysts, lipomas, actinic keratosis, Bowen’s disease and small BCCs.

## 65.8 Conclusion

Nurses have a vital role in working together with doctors and other members of the primary health care team and the patient in the care of skin disease. Nurses often have more time and patience when instructing patients on various techniques in skin care. Specially trained nurse practitioners are an invaluable resource when dealing with specific areas such as wound care and allergy testing. A properly trained nurse can manage diverse aspects of care like a cryosurgery or a photodynamic therapy clinic as long as they have the proper training. However, some practice nurses are expected to manage cryosurgery clinics in primary care with little or no supervision or training and this can lead to poor results, unnecessary pain and possible missed diagnosis of potentially serious skin conditions including melanoma.

## References

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2. Buckley D. Evaluation of local anaesthetic infiltration for cryosurgery of hand warts: a prospective comparative study. Ir J Med Sci. 2015;185:561–4.



# Patient Information Leaflets (PIL)

66

David Buckley

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## 66.1 Acne

### 66.1.1 Acne—how to treat it

While most teenagers will develop some degree of acne it is usually quite mild with occasional spots and pimples that clear up quickly. Unfortunately a small number of people can get quite troublesome acne which can have a major impact on their life. Many of these people become extremely self-conscious, shy and withdrawn. Fortunately all forms of acne, even the most severe, can now be cleared up with safe effective treatments.

Acne is most common in teenagers and usually clears up within a few years. In a small number of people their acne can persist into their 20s, 30s or even into their 40s. In addition, some people do not start to develop acne until they are well into their 20s (“adult onset acne”). Comments such as “don’t worry, you’ll grow out of it” are unhelpful. While it is true that acne will eventually settle down this may take months or years during which time the person may suffer from permanent physical or psychological scars.

There are many misconceptions about acne. Many young people and their parents believe it is due to a food allergy or lack of hygiene. This is totally untrue. Acne is primarily a hormonal disorder. In the teens and early 20s there is a surge of hormones associated with puberty. In people with acne their skin is hypersensitive to these normal fluctuations in hormonal levels. This causes their skin to over-produce oil, which becomes blocked in the pores. Once the pores are blocked the oil becomes trapped and swells up to cause black heads and white heads (comedones). These eventually become infected with bacteria to form pimples and spots. Squeezing, scratching or picking spots usually causes them to last twice as long, so try to leave them alone. Picking may also cause the spots to spread and may scar the skin. Acne will eventually clear up but scars won’t! Other factors that aggravate acne are stress, as experienced before exams. Moisturisers and oily cosmetics should be avoided in the acne-affected areas since the skin is already too oily. A lightweight non-greasy make-up (e.g. a “non-comedogenic” make-up) will do no harm and will help cover up the spots for special occasions. Green concealers are best for the red spots. Once the green is applied, cover with skin colour oil free make up for best results. Many women with acne get worse prior to a period. Certain forms of contracep-

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tives such as progesterone only pills, implants or coils may aggravate acne. Junk food may also make acne worse. Taking too much dairy produce may aggravate acne but there is no need to give it up completely. A glass of milk a day is good for your bones. Avoid foods rich in sugar or fats. If you eat chicken or pork, try to ensure it is free range. Eat plenty of fruit and vegetables, lean meat and fish.

Treatment of acne will depend on its severity. Most mild cases can be managed by the person themselves with simple products that can be bought from the chemist. Washing the face twice a day with soap and water is helpful since this will remove some of the excess oil. However, excessive washing will not clear acne. Various acne lotions, creams or gels, which are recommended by your chemist or doctor, should be applied all over the acne-affected areas and not just onto individual spots. They will help clear up existing spots and will prevent new ones developing. They all help reduce the oiliness of the skin and help clear blackheads and whiteheads. These products often cause some transient drying of the skin for a few hours after application.

It may take 6–12 weeks before one starts to see an improvement with these acne creams so patience and perseverance is crucial in order to see results.

More resistant or severe cases of acne may need more potent topical cream or gel or tablet treatments, which can only be prescribed by your doctor. The most common tablet treatment is Tetracycline tablets, which have antibacterial and anti-inflammatory properties. These are extremely safe and effective. In women a hormonal tablet treatment is sometimes used. Tablet treatments are usually combined with topical treatments for 6 months until the acne is fully under control. The improvement is then maintained with topical agents alone to prevent your acne from returning. The topical agents can be continued for months or years until you are sure you have “grown out of your acne”.

People with a lot of red spots confined to their face with not too many blackheads or whiteheads might benefit from a course of pulse dye laser treatment (Regenlite). This has an anti-inflammatory effect similar to standard acne tablets and can give similar results. The laser light is passed over the skin, which can cause minor discomfort only. Most people need one laser treatment per month for 3 months.

Very occasionally acne can be extremely severe and aggressive with extensive pimples, spots and even lumps on the face, back or chest, which can leave permanent scars (nodular, cystic or scarring acne). This rare severe type of acne may not respond to the standard treatments. These people usually need “Roaccutane®” tablets, which are a very potent vitamin A treatment, which will clear even the most severe resistant cases. However, this vitamin A tablet treatment has some uncomfortable but reversible side effects and so is reserved for only the most severe, resistant cases. People taking Roaccutane® need monthly blood tests and females should not become pregnant when on this drug.

Acne is not the only condition that causes spots and pimples on the face. If your skin is not clearing up with simple acne treatments, check with your doctor as you may be suffering from something other than acne.

In summary, acne is a common condition which unfortunately occurs in a very vulnerable age-group and can cause physical and psychological scarring. Nowadays all types of acne, no matter how severe or extensive, can be managed successfully with safe effective treatments.

### 66.1.2 Isotretinoin (“Roaccutance®”): What patients need to know

Oral isotretinoin is an extremely effective medication for the treatment of severe or resistant acne. It is derived from vitamin A. Doctors have gained a lot of experience with this drug, which was first launched in 1982. Most patients need between 4 and 9 months of treatment depending on the severity and extent of their acne and the daily dose. Almost all patients respond to oral isotretinoin, even those with severe, resistant acne. Most patients, who clear with acne, will remain clear for a long period of time and many will be permanently cured of their acne. However, about one in three patients may relapse a few months or a few years after completing a course of oral isotretinoin. These patients may respond to less potent forms of acne treatments or may require another course of oral isotretinoin.

For female patients, it is imperative that they **do not become pregnant** while taking **oral isotretinoin** as this can have severe damaging effects on an unborn child. For this reason, all women of childbearing age, whether they are sexually active or not, have to use an effective method of contraception (e.g. “the pill”) for 1 month before, throughout the course of treatment and for 1 month after completing the course of **oral isotretinoin**. For women who are sexually active, they need to use two effective methods of contraception (e.g. the oral contraceptive pill and condoms) for the same period of time. The pill can sometimes make you moody—if this happens please let your doctor know. If a woman becomes pregnant or thinks she might be pregnant while taking **oral isotretinoin** she should stop the drug immediately and report her concerns to her doctor.

Although oral isotretinoin has been available since 1982, there is still no clear-cut evidence that it can cause **depression**. However, if you have persistent tiredness, irritability, poor concentration, sleep disruption, sadness, crying spells, loss of motivation or forgetfulness while on oral isotretinoin and if these persists most days for more than 2 weeks, please let your doctor or the nurse know. You may require reduction in the dose of your treatment or you may have to stop your medication while these symptoms are being assessed. Some studies have shown that acne itself can cause mood disorders or depression and treatment with oral isotretinoin can improve mood in these people. Please let your doctor know if you ever suffered from depression in the past.

Please inform your family doctor that you are taking a course of oral isotretinoin. If you are attending a counsellor or a psychiatrist, please inform your doctor before starting this medication and please inform your counsellor that you are taking oral isotretinoin. Please read all the printed information supplied by your doctor and the product information contained in your box of medication. Oral isotretinoin does not mix well with **alcohol or illegal drugs**. Please try to abstain from these while on this medication and for 1 month after completing your course.

You will need to be seen monthly for fasting blood tests and a urine test for the duration of the course of your treatment and one final visit 3 months after completing your treatment. Female patients need one extra visit 5 weeks after finishing **oral isotretinoin**. Please discuss any problems you may be experiencing with your doctor or the nurse on each of these visits.

**Other possible Side Effects:** **Oral isotretinoin** will cause **dryness** of the mucous membranes, (lips, nose, eyes, genitalia and the skin around the anus). These symptoms are often dose related and so if you are having a lot of trouble with dryness, your doctor may reduce the dose you are taking.

Moisturiser your lips frequently with a greasy lip moisturiser such as “**Vaseline Lips®**”, “**La Roche-Pasay Cicaplast Lip Balm®**” or “**Carmex Lip Balm®**”. You may also have to moisturise your nose, genitalia and peri anal skin. If you suffer dry eyes your doctor may recommend artificial tears. Please inform your doctor or nurse if you are still having trouble despite moisturising.

Your doctor may prescribe 1% Hydrocotisyl ointment, which can be applied to the lips twice a day throughout the treatment, if dry lips are a problem and not responding to a lip moisturiser. This can also be applied to the genitalia and the peri anal skin, twice a day, if dryness is not resolved by simple moisturisers such as “**Vaseline®**” or “**Emulsifying ointment**”. Sometimes the skin in these areas can get cracked, sore and scabby. If this occurs, you may require topical or oral antibiotics. Please discuss with your doctor. If you have suffered from eczema in the past, you may experience a relapse of your eczema while on **oral isotretinoin**. This usually responds to greasy moisturisers, avoiding soaps and other irritants and eczema treatments such as steroid ointments. Some people may have to lower the dose of **oral isotretinoin** they are on.

Some people will get a temporary flare up of their acne in the first month of treatment. **Oral isotretinoin** can make your skin more sensitive in the sun, so be careful in the sunny weather by wearing appropriate clothing, a broad brimmed hat and high factor sun blocks (SPF 30). As your skin is sensitive while on **oral isotretinoin**, do not have any cosmetic skin treatments (waxing, dermabrasion, laser) while on this treatment and for 6 months after completing the course.

Some people may experience lower back pain, muscle aches or joint pains while on **oral isotretinoin**, particularly if they are training hard. You may have to reduce your level of physical activity or your doctor might reduce the daily dose. If this does not help, please discuss your problems with your doctor. Other rare side effects include temporary hair thinning, headaches, tiredness, diarrhoea and difficulty with night vision or colour vision. If you have any of these problems, please discuss them with your doctor or the nurse.

**Oral isotretinoin** cannot be used if a woman is breastfeeding. Do not donate blood at the blood bank while on this medication and for 1 month after finishing. Do not share your tablets with anyone else, particularly other women. Do not take vitamin A supplements while on **oral isotretinoin**. Please do not use other acne therapies while on **oral isotretinoin**.

## Dosage

The usual starting dose is 0.5 mg per kilogram of your body weight per day. **Oral isotretinoin** should be taken once or twice a day with food or with a glass of milk as this helps its absorption. It is easier to remember to take your tablets if you take them at the same time every day. If you are under 18 years old, you will need to be accompanied by an adult for every visit.

Please do **not** Google the word “**Roaccutane®**” or “**oral isotretinoin**” as you will get a lot of false information regarding this medication which may confuse you. Do not buy **oral isotretinoin** on the internet—it needs to be prescribed by a doctor with experience in its use and it needs to be dispensed by your local pharmacist. For more information also see: [www.aad.org/isotretinoin](http://www.aad.org/isotretinoin)

If you have any problems while on **oral isotretinoin** please contact your doctor or nurse immediately.

## 66.2 Consent Forms

### 66.2.1 CONSENT FORM

**NAME OF PATIENT:** \_\_\_\_\_

**DATE OF BIRTH:** \_\_\_\_\_

**ADDRESS:** \_\_\_\_\_

I hereby consent to undergo the operation/treatment of:

The procedure, alternatives and risks have been explained to me in clear terms by Dr. ....

I also consent to such further or alternative operative measures or treatment as may be found necessary during the course of the operation or treatment and to the administration of local or other anaesthetics for any of these purposes.

I understand that the treatment itself may not be 100% safe or successful and that there may be unexpected complications following the treatment particularly relating to pain, bleeding, blistering, infection, scarring, pigment changes, delayed wound healing or recurrence. The doctor has answered all my questions relating to this surgery.

- |   | YES                      | NO                       |
|---|--------------------------|--------------------------|
| 1. Do you have a pacemaker?.....  | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. Have you had any blood borne infectious diseases in the past? .....<br>(i.e. Hepatitis B or C or HIV)                | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. Do you suffer from diabetes?.....  | <input type="checkbox"/> | <input type="checkbox"/> |
| 4. Are you on Aspirin or blood thinner medication.....<br>(e.g. Warfarin, Persantin, Plavix, Eliquis, Pradaxa, Xarelto) | <input type="checkbox"/> | <input type="checkbox"/> |
| 5. Are you allergic to local anaesthetic or natural rubber (latex)  | <input type="checkbox"/> | <input type="checkbox"/> |
| 6. For women only-are you pregnant?   | <input type="checkbox"/> | <input type="checkbox"/> |

**PATIENT SIGNATURE:** \_\_\_\_\_ **DATE:** \_\_\_\_\_

I confirm that I have explained the nature and effect of this operation/treatment to the person who signed the above form of consent.

**SIGNATURE OF DOCTOR:** \_\_\_\_\_ **DATE:** \_\_\_\_\_

**Patient Information Leaflet Issued:** Yes  No

**Copy of this consent form given to patient:** Yes  No

### **66.2.2 Photography Consent**

I give my consent for Dr. ..... to take medical photographs of me or of my child (or person for whom I am legal guardian) for record keeping, education, publication or research, for example:

- Use in lectures, reports, research articles, scientific posters, textbooks, etc.
- Publication in professional journals or other print or electronic media including appropriate websites.

We do not ordinarily include photographs of full face or identifiable tattoos unless you have given express permission for this. Your eyes or other identifiable features are usually blurred out in facial photographs. However, complete anonymity cannot be guaranteed.

- I have the right to withdraw consent at any time by writing.
- Images displayed on websites will not include my name or address.
- I understand that on request, my doctor may agree to supply a copy of the images to another person or suitable organisation. My doctor may charge a fee to a 3rd party to offset the time, effort and materials required. I understand I will not receive any payment should this occur.
- I understand that this will not affect my treatment in any way.

Name of patient \_\_\_\_\_ DOB \_\_\_\_\_

Your name (if parent/caregiver) \_\_\_\_\_

Signature \_\_\_\_\_ Date \_\_\_\_\_

Witness \_\_\_\_\_ Date \_\_\_\_\_

## 66.3 Eczema/Dermatitis

### 66.3.1 Cow's Milk (Dairy) Allergy

#### Cow's Milk

Cow's milk tends to be a very important element in the nutrition of all children, especially the younger ones. Its importance lies in its richness in two things: protein and calcium (vital for normal growth of bones and teeth). In order for children to obtain adequate amounts of these nutrients on an egg and milk-free diet, usually they must be given a milk substitute. These can be subdivided into four categories: (1) soya "milk", (2) casein hydrolysate formula, (3) goat's milk, and (4) calcium tablets. It is worth considering each of them in turn.

#### Soya "Milk"

Soya "milk" is based on protein from soya beans and contains no cow's milk protein. Therefore, it is often suggested for a milk-free diet. However, soya milk has a different smell from cow's milk, and children may take some time to accept it. This can be overcome to a great extent if the child drinks from a nursing bottle or trainer cup rather than from an open cup. Soya protein itself can also cause allergic reactions in some children, in which case another alternative should be tried.

Two types of soya milk are available. The first type, soya formula milk, is made for babies and younger children. Like cow's milk formula, soya formula milk is "modified", a treatment process by which the balance of protein, fat, carbohydrate and salt is altered to resemble more closely that found in human milk. It is also nutritionally complete, with a wide range of added vitamins and minerals (including calcium) which is important for good growth and development. Several brands are widely available from chemists' shops, including "ProSobee®" (Mead Johnson), "Formula S®" (Cow and Gate) and "Wysoy®" (Wyeth).

The second type, appropriate only for older children, is ready to drink liquid soya milk. Several brands are available in health food shops and some supermarkets. Please note that these ready to drink soya milks **DO NOT** usually contain adequate calcium (although they are nutritious in other ways) and are **NOT**, by themselves, appropriate substitutes for cow's milk in a child's diet. However, regular calcium supplements **can** make these acceptable for milk-free diets.

#### Extensively Hydrolysate Formulas

These are formulas where the protein source has been broken down to peptides. This removes allergenic proteins, making the formula extremely unlikely to cause allergic reactions. All other components of ordinary cow's milk formula (fats, carbohydrates, vitamins and minerals) are then added and processed to make a modified feed resembling human milk in its nutritional value. Three such formulas are currently available through chemist shops. They are "Pregestimil®", "Nutramigen 1®" and "Nutramigen 2®" (both Mead Johnson), "**Aptamil Pepti 1®**" (for children less than 6 months old) and "**Aptamil Pepti 2®**". These are suitable milk substitutes for infants and children, but must be fed strictly in accordance with instructions from your doctor and/or dietician. Like soya milk, these are unappealing to the adult palate, but most infants accept them as the innate drive of thirst and hunger will overcome the bitter taste. Unlike soya milk, these products very rarely cause an allergic response. For the extremely cow's milk allergic child **elemental formulas** such as "Neocate LCP®", "Neocate Active 1®" or "Neocate Advance®" should be tried. These formulas are more expensive than soya formula, but may be prescribable by your GP.

### Goat's Milk

Goat's milk has been used as a cow's milk replacement with some success. However, we normally recommend soya milk or casein hydrolysate formula in preference to goat's milk, for the following reasons.

1. Many of the proteins contained in goat's milk are virtually indistinguishable from those in cow's milk. Therefore, a change to goat's milk may make little difference in a child's symptoms. However, boiling goat's milk may make the protein more acceptable to some allergic children.
2. Goat's milk is not subject to the strict hygienic controls enforced in the production of milk from dairy cattle to guard against harmful bacteria. Goats are normally hand milked instead of machine milked, adding to the chances of contamination. Furthermore, goat's milk is rarely pasteurised, while cow's milk is always pasteurised. As a result when we have checked in our hospital the bacterial content of goat's milk obtained from various sources, we have almost invariably found potentially harmful bacteria. If you do choose goat's milk, we recommend that you *boil it for 2 minutes*. The milk can then be cooled and drunk cold. Unfortunately, boiling does make the flavour worse and reduces the content of some of the vitamins, especially folic acid, which is already low in goat's milk.
3. Goat's milk is entirely *unsuitable for infants under 6 months of age*. Un-boiled, it is positively dangerous, for the bacteria it often contains may cause a lethal infection in infants. Even when boiled, however it is hazardous for this age group because it is not "modified" and therefore lacks a normal balance of nutrients.
4. Goat's milk may be difficult to find in retail stores, although availability has improved in recent years. It may be particularly scarce during the kidding season, when it is fed to baby goats. Goat's milk is regularly stocked by health food shops and some supermarket chains and can be obtained directly from producers in some rural areas. It is available refrigerated or frozen in cartons or, more rarely, dried in tins. If you choose goats milk, we recommend refrigerated goat's milk because it tends to be fresher than frozen milk and you can freeze it at home yourself for later use.

### Calcium Tablets

If your child cannot or will not take sufficient amounts of one of these milk substitutes (more than  $\frac{1}{2}$  pint or 300 ml daily) it is important to provide adequate amounts of calcium in tablet form. Perhaps the most suitable are "Sandocal tablets®", which contain very small amounts of E102 and E110 artificial colourings. One tablet should be taken daily, dissolved in water or orange juice. This provides more than enough calcium for most children. An alternative is Cox effervescent calcium gluconate tablets, which contain no added colourings, though up to six tablets daily may be required.

The elimination of cow's milk also means strict avoidance of: milk powders (full fat and semi-skimmed), cheese, cream, butter, yoghurt, non-fat milk solids, caseinates, lactalbumin, lactose, whey, margarines and shortening containing whey, whey syrup sweeteners.

Suitable alternatives to butter and whey-based margarines are available from some supermarkets and health food shops. A cow's-milk-free soya ice cream is also available from many health food shops.

Food labels should be checked routinely as milk and its derivatives are used fairly frequently in processed foods. Even the labels of tried and trusted milk-free products should be regularly checked as manufacturers may change ingredients from time to time.

## Hidden Sources of Milk

Remember to read the ingredients list before buying packets, tins or jars of baby food, in order to watch out for the “Hidden sources of milk” in manufactured foods.

In ingredient listing and on food labels

Casein	Sodium Caseinate
Caseinate	Lactose
Calcium Caseinate	Whey
Skimmed Milk	Milk Protein
Milk Sugar	Whey Protein

In foods

Cheese	Custards
Cottage Cheese	Some baked products (e.g. biscuits, bread rolls and cakes)
Cream Cheese	Certain cereals
Butter	Crackers
Margarine	Pancakes
Cream	Pasta
Ice Cream	Curds
Yoghurt	Gravy
Creamed Soups and other Foods	White Sauces
Luncheon Meats and other processed meats, (e.g. hot dogs, hamburgers)	Confectionary (especially chocolate)
Puddings	Crisps

Note: this list highlights only a few of the foods to be avoided in a milk-free diet. It should *not* be used as a diet sheet and fully qualified dietary advice should be obtained

### 66.3.2 Hand care tips for hand eczema and dermatitis

1. Constantly moisturise your hands at least five or ten times a day with a “safe” greasy moisturiser as recommended by your doctor or nurse (e.g. Emulsifying ointment, “Epiderm ointment®”, “Aveno Dermexa®” cream or “Neutrogena Hand Cream Unscented®”). Avoid cosmetic type moisturisers containing colours and perfumes on sensitive hands.
2. Keep your hands dry at all times by the careful use of gloves. Before wet work moisturise your hands with your recommended moisturiser and put on a pair of light cotton gloves available from pharmacies (e.g. “Seton Gloves”®) or First Communion gloves) followed by a pair of domestic rubber or PVC gloves for housework or industrial gloves for heavy work. Take a break every 15 min, remove the gloves and moisturise again. Cotton gloves by themselves are suitable for “dry” work around the house such as hoovering, polishing etc. Surgical latex gloves may be more suitable for delicate wet work.
3. Avoid direct hand contact with soaps, shampoos, washing up liquids and detergents. Use a washing machine and dishwasher if at all possible.
4. Avoid hand washing. Get your gloves dirty, not your hands. If you have to wash your hands (e.g. after going to the toilet) never use soap. Use a recommended soap substitute such as “Elave Wash®” or aqueous cream with lukewarm water. Then dry your hands carefully and moisturise immediately with your recommended moisturiser. These soap substitutes can also be used in the bath or shower by placing a small amount on a sponge to clear the skin. Use an electric razor instead of wet shaving.
5. Use a detergent free shampoo such as “Elave shampoo®” or else wear surgical gloves when washing your hair.
6. Rings should only be worn for special occasions, never during work. Avoid cheap jewellery (18 carat gold, solid silver or stainless steel are safe).
7. Buy plenty of gloves and hand moisturisers and place them in strategic places around the home and at work so that you don’t have to go looking for them every time you need them e.g. in the bathroom, kitchen, garage, car and at work. Keep small quantities of your hand moisturiser in a container in your hand bag or pocket to use throughout the day.
8. It may take many months for your hands to recover. Even after recovery you will still have to take great care of your hands to prevent relapse.
9. Pain, oozing, crusting or a sudden deterioration of your hand eczema/dermatitis may indicate that your skin has become infected. Check with your doctor, as you may need antibiotic tablets.
10. If your hands do not improve with these measures you may need to see your doctor to add in a topical steroid cream and perhaps to have some allergy testing such as a skin patch allergy test.

### 66.3.3 Ketoconazole ("Nizoral") Shampoo

#### Instructions for Use for Severe Dandruff, Seborrhoeic Dermatitis and/or Psoriasis

1. Use ketoconazole ("Nizoral") shampoo, .....times a week for 4 weeks.  
Then use once a week to prevent relapse.
2. Use the pink ketoconazole ("Nizoral") shampoo in the following way:
  - a. Wash hair with your regular shampoo. Rinse out well and towel dry the hair.
  - b. Apply one teaspoon full (two teaspoons full for long hair) of ketoconazole ("Nizoral") shampoo to the scalp and work up into a rich lather. You may also rub some of the lather onto your face, ears and body if necessary.
  - c. Leave it soak in for 3 min.
  - d. Rinse out.
  - e. If you have to use a conditioner, use it only on the ends of the hair; not in the scalp.
  - f. Dry hair in the usual way.
3. For a more potent effect, rub in 5 ml (one teaspoon) of the shampoo into the dry scalp, leave soak in for 10 min, then wet the hair, work up into a lather and rinse out.
4. If you need to wash your hair between treatments, use your regular shampoo.

If you have seborrhoeic dermatitis on the face, ears or groin, you may also apply 1% hydrocortisone cream mixed with miconazole nitrate antifungal, (e.g. "Daktakort Cream") or tacrolimus ointment ("Protopic") ointment sparingly to these areas, once or twice daily, till cleared.

### 66.3.4 Management of Dry Sensitive Skin Conditions

Many skin problems such as Eczema, Dermatitis and Psoriasis cause excessively dry skin, which sometimes become scaly and itchy. Almost all dry skin conditions will benefit from the careful use of moisturisers (emollients), which can be applied directly to the skin or mixed in bath water. The greasier the moisturiser the better for most dry skin conditions. However, some people find very greasy moisturisers messy. Some people prefer to use a greaser moisturiser at home and over the weekend and use a lighter moisturiser, which may be more cosmetically acceptable, at work and when out socially.

#### Moisturisers

For moisturisers to work properly and relieve itch, they need to be applied generously and frequently. For people suffering from hand eczema or dermatitis, moisturiser should be applied every hour to the hands and also after washing. If you suffer from generalised dry skin all over your body, you need to moisturise at least twice a day and also after baths or showers.

Adequate quantities of moisturisers need to be prescribed. If you are applying a moisturiser all over the body twice a day then you would need approximately 1000 g of moisturisers in a month for an adult, or 500 gm of moisturiser every month for a child. Moisturisers should be fragrance and perfume free, hypo-allergic and cheap so that they can be used frequently and generously. Moisturisers should **never** be put on acne prone skin. Paraffin based moisturisers may be **flammable** so stay away from naked flames when applying these products. Beware that clothes stained with a paraffin-based moisturiser may catch fire easily with matches, lighters, cigarettes or from a fireplace.

Below is a list of recommended moisturisers graded according to how greasy they are.

Some people like to get small samples (100 g) of various different moisturisers to try them out and see how they feel. By and large the greasiest moisturiser that can be tolerated is the best.

<b>Thick and Greasy:</b>	<p>“Epaderm Ointment®” 125 g, 500 g</p> <p>Liquid paraffin/white soft paraffin in equal parts (“Paraffin Gel®”) (500 g)</p> <p>“Diprobase Ointment®” (50 g)</p> <p>“Hydromol ointment®” (Alliance Pharma Ltd) 125 g, 500 g</p> <p>“Hydrous ointment®” (500 g)</p> <p>“Double base Emollient Gel®” 100 g, 500 g</p> <p>Organic extra virgin coconut oil. 500 ml (from supermarkets)</p> <p>“Bioderma Atoderm Intensive®” 500 ml</p> <p>“La Roche Posay Lipikar Baum AP+®” (200/400 ml pump) for atopic dermatitis</p> <p>“La Roche Posay Cicaplast Mains®” (hand moisturiser) 50 ml</p> <p>“Eucerin Lotion®” 10% (250 ml)</p> <p>“Relife U-Life Cream®” (5%, 10%, 20%, 30%, 40% and 50%) (A Menarini)</p> <p>“Childs Farm Moisturiser®” (unfragranced) (250 ml)</p> <p>“Moogoo Natural Full Cream Moisturiser®” (200 g)</p> <p>“Epaderm Cream®” (500 g)</p> <p>“Neutrogena dermatological hand cream®” (unscented)</p> <p>“Oilatum Cream Pump®” (500 ml)</p> <p>“Aveeno Dermexa cream®” 200 ml</p> <p>“Aveeno Daily moisturising lotion with colloidal oatmeal®” (200 ml, 354 ml)</p> <p>“La Roche Posay Toleriane Ultra®” (40 ml) for extremely sensitive skin</p> <p>“La Roche Posay Effaclar H®” (40 ml) for dry acne prone skin</p> <p>“Cicabio Cream®” (40 + 100 ml) and “Cicabio SPF 50+®” (30 ml) (Bioderma)</p>
<b>Light and Creamy:</b>	“Diprobase cream®” (50 g or 500 g pump)

Greasy moisturisers (e.g. emulsifying ointment) are generally better tolerated on the body but sometimes people prefer to use less greasy moisturiser on the face such as "Diprobase®". This is also good for babies' bottoms during nappy changes.

**Silcock's Base** (100 g + 500 g) is often promoted as a moisturiser but it is more of a cooling cream used to cool down the skin in conditions such as sunburn or facial flushing. **Zinc and castor oil** (100 g + 500 g) is a good lubricant and barrier for nappy rash and itchy bottoms. For dry lips "**Carmex Lips®**", "**Nutragena Lips®**" or "**Atoderm Stick®**" (Bioderma) are good.

When applying moisturisers to the body, the ointment should be always rubbed downwards gently in the direction of the hairs, as you would stroke a cat, going with the fur. Rubbing upwards against the hairs can irritate the skin and cause low-grade infection in the hair roots (folliculitis). Some people find that placing a thick moisturiser such as emulsifying ointment on a radiator, windowsill or in the hot press keeps it softer and easier to apply. When moisturisers are used liberally, people with dry skin conditions often need less prescription medication such as anti-itch tablets or topical steroids. As a rule, you should use 10 times more moisturiser as topical steroid.

Emollients with the same generic name can be manufactured by different companies. You may find a variation from one company's ointment to another's.

## Bath Emollients

"Oilatum®" do a range of bath emollients which easily dissolve in water and are a simple way of moisturising the skin. **Nothing** else should be added to the bath water, such as bubble baths, "Baby Bath", "T-tree oil" etc. Below is a list of bath emollients with the usual indications.

- "**Oilatum bath emollient®**": 20 ml in a bath is suitable for adults (comes in bottles of 100, 250 and 500 ml).
- "**Oilatum Plus Bath Emollient®**": 20 ml in a bath is suitable for adults when their skin is infected.
- "**Oilatum Junior Bath Emollient®**": 10 ml in a bath is suitable for children.
- "**Oilatum Junior Flare-up Bath Emollient®**": 5 ml in a bath is suitable for children greater than 6 months old with infected eczema or
- "**Emulsiderm Emollient® (Dermal)**": (300 ml bottle)—30 ml in a bath for an adult, 10–20 ml in a child's bath.
- "**Aveeno Colloidal Bath Powder®**" with colloidal Oatmeal. Dissolve in bath water as instructed on package.
- "**Hydromol Bath and Shower Emollient®**": 1–3 capful in an 8-inch bath. Infants = Half to 2 cap.
- "**Milton Sterilizing Fluid®**": 120 ml (4 capfuls) in 100 l bath (= a half full adult bath): or half capful in a half full baby bath = Max 2 baths/week (1 ml of "Milton®" per litre of bathwater).
- Water temperature = 28–30 °C. Do not stay in bath more than 3–5 min.
- Alternatively, two dessertspoons full of **Emulsifying ointment** can be added to two pints of boiling water and beaten with a fork or whisk until all lumpiness has gone and a creamy liquid remains. This can be poured into a warm bath and mixed thoroughly to provide a cheap bath emollient that is available on the medical card. However, this does not mix through the bath water as good as the special bath oils mentioned above.
- Bath mats should be used to avoid slipping in the bath when using bath emollients.
- "**Psoriderm Bath Emulsion®**" 200 ml useful for psoriasis.

## Soap Substitutes

It is important that people with dry skin conditions do **not** use soaps or shampoos, as this will dry out the skin even further. "**Elave Wash®**", "**Elave Shower Gel®**", "**La Roche Posay Lipikar Syndet Wash®**" (200 ml/400 ml), "**Hydromol Bath and Shower Emollient®**", "**Aveeno Dermexa Body Wash®**" for very dry skin or "**Aveeno Daily Moisturising Body Wash®**" or "**Bioderma**

**Atoderm Shower Oil®** (1 l) are good soap free washes that can be used for hand and body washing in the bath or shower. Other options include “**Cetaphil Gentle Skin Cleaners®**”, “**Cetaphil Skin Restoring Body Wash®**” (Galderma) or “**Sanex Hypoallergenic Shower Gel®**”. “**Elave Shampoo®**” or “**Bioderma NODE Shampoo®**” is safe for hair washing. Alternatively, **Aqueous Cream (100 g or 500 g)** can be used as a soap substitute by gently massaging a small amount of it on to the skin and washing off with warm water. This can be used for hand washing or for showering. This is a cheap alternative as a soap substitute and is available on the medical card. Do **not** leave Aqueous Cream on the skin as a moisturiser.

### 66.3.5 Milton Baths

Some parents notice that their child's eczema may benefit from being in chlorinated swimming-pool water due to its antiseptic effects. These instructions make an antiseptic bath at home with a similar concentration to a chlorinated swimming pool:

1. Add Milton Sterilising Fluid® (MSF) followed by lukewarm water into the bath tub.
2. **125 ml of Milton® (MSF) in 60 l of water (roughly ½ bath full)**  
or  
**62 ml of Milton® Sterilising Fluid in 30 l of water (roughly ¼ bath full)**
3. Stir the mixture with the jug to make sure that the bleach is completely diluted in the bath water.
4. The patient should soak up to their neck in the chlorinated water for approximately 5 min. Be careful not to get it in your eyes.
5. Thoroughly rinse skin with lukewarm clean water at the end of the bleach bath to prevent dryness and irritation.
6. As soon as the bath is over, pat dry. Do not rub dry as this has the same effect as scratching.
7. Immediately apply any prescribed moisturiser and other treatments.
8. Repeat bleach baths twice weekly or as prescribed by your doctor.

#### Cautions

- Do not use undiluted bleach directly on the skin. Even diluted bleach can potentially cause dryness and irritation.
- Do not use bleach baths with a known contact allergy to chlorine.
- Do not use bleach baths without first taking medical advice from your doctor.
- Before the first bath test the MSF, diluted as above, on a small area of non-eczematous skin. Rinse off after 10 min and wait for 24 h to make sure you don't have a reaction to it.

**Only use "Milton Sterilising Fluid®". DO NOT use any other type of household bleach.**

### **66.3.6 Tacrolimus (“Protopic®”) + pimecrolimus (Elidel®)**

Tacrolimus (“Protopic®” ointment) and pimecrolimus (“Elidel® cream”) are topical *immunomodulating agent* of the *calcineurin inhibitor* class used in the treatment of *atopic dermatitis* (eczema) in adults and children over the age of two, when they are not responding to topical steroids or not able to tolerate topical steroids (cortisone creams).

Tacrolimus and pimecrolimus have a number of advantages over topical steroids in the treatment of atopic eczema and other skin conditions.

#### **Advantages**

- They are as potent as a potent topical steroid in the treatment of atopic eczema but has none of the steroid side effects such as skin thinning, skin redness, or growth retardation.
- They are effective at both settling down a flare of atopic eczema and also for preventing relapse.
- They can be used on the face and body including the flexures (folds of skin such as the groin and under the arms). They are available for both children over the age of two and adults.
- Tacrolimus and pimecrolimus may be used off-licence for a number of other skin conditions such as facial and flexural psoriasis, seborrhoeic dermatitis, vitiligo and erosive lichen planus.

Despite all these advantages with tacrolimus and, pimecrolimus, topical steroids are still the first line of treatment for most adults and children with atopic eczema and topical steroids are considered safe and effective when used under careful medical supervision (see our separate sheet on safe use of topical steroids).

#### **Tacrolimus and Pimecrolimus Can Have a Number of Disadvantages**

- They are considerably more expensive than topical steroids.
- They can cause temporary redness and irritation at the site of application in up to 50% of patients during the first week of use. Most of these flare-ups will settle in the second or subsequent weeks (i.e. “It can get worse before it gets better”).
- Ten per cent of patients cannot tolerate tacrolimus or pimecrolimus, even after the first week of use and they will have to use a different form of treatment.
- Tacrolimus cannot be used on infected skin (i.e. if the skin is sore rather than itchy or if it is weepy or crusty instead of being dry, these are signs of infection.) Infection needs to be managed with appropriate antibiotic or anti-viral agent either topically or orally before starting these drugs.
- Tacrolimus and pimecrolimus can cause facial flushing after consuming alcohol in some patients.
- Tacrolimus and pimecrolimus are not licenced for children under the age of two in most countries, although some skin specialists use them in this age group for severe eczema, as it is probably safer than using potent topical steroids in this age group.
- Tacrolimus and pimecrolimus may interfere with live vaccines such as the MMR or the BCG. Special precautions may be required before giving these vaccines.
- There is a possibility that tacrolimus and pimecrolimus might make a patient more at risk of cancer such as skin cancer. This can be minimised by protecting the skin from ultra violet light by the appropriate use of clothing, hats and high factor sunblocks when using these agents on exposed areas such as the face and hands. Never use tanning beds when using tacrolimus or pimecrolimus. However, a direct link with these drugs and cancer has not been proven.

## How Is Tacrolimus and Pimecrolimus Used?

Tacrolimus comes in two strengths (0.1% for adults and children over the age of sixteen and 0.03% for children from 2 years old to 16 years old) and in two sizes (30 g and 60 g).

Pimecrolimus comes as a cream and in only one strength (1 g of cream contains 10 mg of pimecrolimus which can be used in children over the age of 2 years and in adults).

They should be applied to all the areas affected with atopic eczema, twice a day for 3 weeks and then once a day for a further 3 weeks.

For preventative use, once the atopic eczema has settled down, apply tacrolimus or pimecrolimus to the areas where you previously had atopic eczema twice a week to prevent relapse. Preventative treatment needs to be reviewed every 6–12 months by your doctor.

When using tacrolimus or pimecrolimus, you should avoid soaps, bubble baths, shower gels, washing up liquids and detergents. Use a suitable soap-free wash and soap-free shampoos.

You should moisturise regularly with a good greasy moisturiser as recommended by your doctor. Leave 2 h after applying tacrolimus or pimecrolimus before applying a moisturiser.

For more information on tacrolimus or pimecrolimus, read the information leaflet that is included in the box that comes with your cream.

Please store the tube in a cool, dry place with the cap on and away from direct heat and sunlight. Please keep all medicine out of reach and sight of children. Do not share your prescription treatment with others.

### 66.3.7 Topical Steroids; There safe use

- Topical steroids are very effective anti-inflammatories that will reduce itch and inflammation in a wide range of skin conditions. They have been on the market since the 1950s and there is nothing we don't know about them.
- Used properly, under careful medical supervision, topical steroids are very safe and effective in adults and children. Topical steroids are also known as topical corticosteroids but they are completely different than anabolic steroids used and abused by body builders and athletes. Topical steroids much safer than oral steroid tablets or steroid injections.
- Like all medications, topical steroids can cause side effects. Topical side effects which rarely occur include skin thinning or broken veins. Topical steroids may trigger or worsen acne, rosacea or peri-oral dermatitis. Generalised side effects can occasionally occur if very strong topical steroids are applied to large parts of the body in high quantities over a prolonged period of time. These generalised side effects might include raised blood pressure, diabetes or stunted growth. Your doctor will instruct you on which topical steroid to use on which particular area of the body and how much can be safely used for an adult or a child.
- Topical steroids come in different strengths or potencies (see Table 66.1). We usually start with a stronger strength topical steroid and reduce the frequency of application or the potency of the topical steroid as the underlying condition improves.
- Topical steroids will do nothing for dry skin. If your skin is dry you should moisturise regularly with a safe, greasy moisturiser as recommended by your doctor. By and large, you should use ten times more moisturisers compared to the amount of topical steroids you use per month. You should avoid soaps, shampoos, bubble baths, washing up liquids, etc. Wear gloves to protect your hands and use a suitable soap free wash and shampoo.
- *Mildly potent topical steroids* such as 1% Hydrocortisone is safe on the face and in the folds of skin (e.g. under the armpit or in the groin) even in children.
- *Moderately potent topical steroids* such as hydrocortisone butyrate ("Eumovate®") can be safely used on the body in children and adults but is best avoided on the face.
- *Potent topical steroids* such as betamethasone dipropionate ("Betnovate®" or "Diprosone®"), hydrocortisone butyrate ("Locoid®") or mometasone furoate ("Elocon®") can be safely used on the body in adults but should not be used on the face and should only be used on the body in children for a short period of time (e.g. 1 to 2 weeks) if there is a severe skin problem.
- *Very potent topical steroids* like clobetasol propionate (e.g. "Dermovate®") are rarely necessary in adults. They should never be put on the face and are unsuitable for children on any part of the body (Table 66.2).
- A fingertip unit is a convenient way of measuring the amount of topical steroid required to cover an area the equivalent of two adult palms, including the fingers. A fingertip unit is the amount of cream or ointment that comes out of the opening of a standard tube with a 5 mm nozzle that extends from the tip of an adult index finger to the first finger crease. This is approximately 0.5 g.

**Table 66.1** Topical steroid potency

Potency	Example	Trade names	Potency ratio
Super potent	Clobetasol propionate	"Dermovate®"	600
Potent	Betamethasone dipropionate	"Betnovate®," Diprosone®"	100
	Mometasone furoate	"Elocon®"	
	Hydrocortisone butyrate	"Locoid®"	
Moderately potent	Hydrocortisone butyrate	"Eumovate®"	25
	Alclometasone dipropionate	"Modrasone®"	
Weak	Hydrocortisone 0.5–2.5%	1% Hydrocortisone	1
		"Diaderm®"	

**Table 66.2** Maximum amount of topical steroids per month for chronic use<sup>a</sup> (Greater than 2 months duration) (Higher amounts can be used for a short period of time in acute flare ups)

Potency	Age Adult	12 years	3 years	Infant < 12/12 months old
Mild	No max	No max	200 g	100 g
Moderate	200 g	100 g	60 g	30 g
Potent <sup>b</sup>	90 g	30 g	15 g for acute use only	Avoid
Very potent	30–60 g	Avoid	Avoid	Avoid

<sup>a</sup>Adapted from: Position paper on diagnosis and treatment of AE. EADV (2005)19,286–295

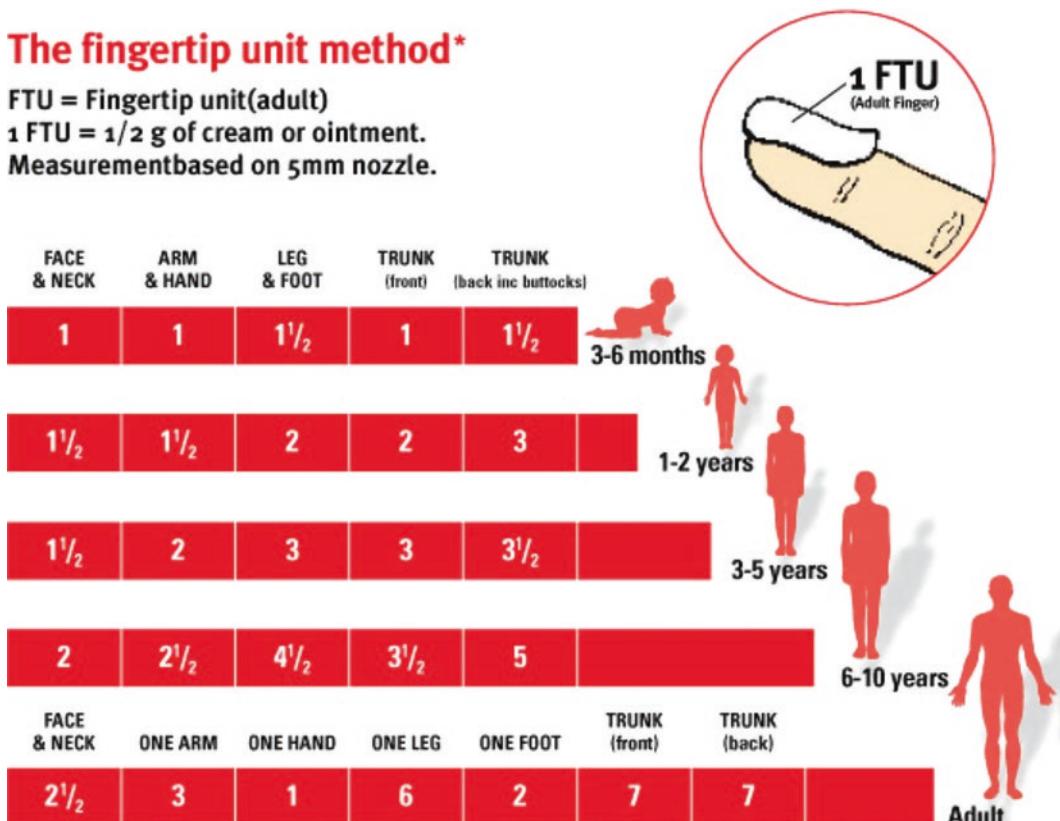
<sup>b</sup>4 times this amount can be prescribed if using “Betnovate RD”

## The fingertip unit method\*

**FTU = Fingertip unit(adult)**

**1 FTU = 1/2 g of cream or ointment.**

**Measurement based on 5mm nozzle.**



**Fig. 66.1** Finger tip unit measurement

Figure 66.1 shows how much topical steroid is safe to use on different parts of the body and in different age groups. These amounts are safe when used over weeks or months. Higher amounts can be safely used for 1–2 weeks for a severe flare up of eczema or dermatitis.

- Generally speaking, topical steroids with an ointment base are safer and more effective than the same potency topical steroid in a cream base.
- Please do not stop your topical steroids suddenly. Try to wean off it slowly over a few weeks by gradually reducing the amount, the frequency of application (e.g. alternate days) and/or the potency of the topical steroid as the underlying skin condition improves.
- Topical steroids are suitable for dry, itchy eczema. They should not be put on infected skin which is usually suspected if the skin condition becomes sore, weepy or crusty. If you think your skin is infected please contact your doctor immediately as you may need an antibiotic.

### 66.3.8 Wet wraps

1. Gather supplies.
  - a. Topical steroid as prescribed by your doctor.
  - b. Emollient of choice (e.g., Emulsifying ointment or “Epiderm ointment®”)
  - c. Wraps consisting of an inner wet layer and a top, dry layer to go over the wet layer. Gauze wraps such as “Tubifast tubular dressings®”, “Tubifast Garments®” or the “Comfi range®”, (all available from Amazon, E Bay or your local pharmacy) or tightly fitting cotton T shirt, tights, sleepers, or long johns may be used. It is necessary to have two sets of the chosen material on hand (a wet layer to be in contact with the skin and a dry layer to be placed over the wet layer). Remove labels and wear the garment inside out if they have rough seems.
  - d. Warm water in a sink or a basin.
2. Apply the moisturiser generously to the child’s dry skin rubbing downwards.
3. Immediately after applying the moisturiser, apply the topical steroid to itchy areas of child’s skin placing it on top of the moisturiser in the badly affected areas. Use the “fingertip units” to calculate how much topical steroid to apply (one “fingertip unit” is the amount of ointment squeezed out form a tube to spread form the last finger crease to the tip of an adults finger; this amount of ointment is enough to covers an area equivalent to the palms of 2 adults hands)
4. Soak a single layer of tubular dressing or garment in warm water.
5. Wring out excess water so that the dressing or garment is only slightly damp.
6. Cover the affected area of skin with a damp layer of the tubular dressing or garment, making sure not to wrap too tightly.
7. Immediately place a dry layer of material over the damp layer.
8. Keep child in a warm, humid environment to ensure that the child does not grow cold as the evaporation process occurs. Leave wraps in place overnight.
9. Change wraps once daily for up to 14 days in a row or as instructed by your doctor.
10. Maintain close contact with your doctor while using wet wraps technique. Report any suspected side effects or concerns immediately. Wet wraps cannot be used if the skin is infected (i.e. weepy, crusty or sore)
11. See YouTube video entitled: “[Wet Wrapping Technique](https://www.youtube.com/watch?v=mVgQ7eLjJcY)”  
<https://www.youtube.com/watch?v=mVgQ7eLjJcY>

## 66.4 Hair Loss

### 66.4.1 Hair Loss in Men

For most men with male pattern hair loss, trying to accept the problem is probably the best approach. Some men will be happy to grow and fashion their hair to cover the affected areas. Others may choose to shave their scalp or wear a hair piece or wig.

**Volumising shampoo** and conditioners may help. (e.g. “Nanogen Thickening Treatment Shampoo®”, available in Boots or try your local pharmacy).

Another cosmetic option is **hair fibres** (e.g. “Nanogen Hair Thickening Fibres®”). This releases thousands of microscopic colour-matched hair fibres, which bind electrostatically individual hair giving the appearance of a fuller head of hair. These can be made wind and waterproof with various locking mists (e.g. “Nanogen Fibre Locking Spray®”).

Lightning the **hair colour** to bend in with the white scalp may help camouflage the hair loss process. Alternatively, temporary scalp dies can safely blends scalp and hair colour together to conceal areas or thinning hair (e.g. “Nanogen Aquamatch Waterproof Concealer®”).

**Vitamin supplements** promoted for hair and nail growth such as *biotin* 2.5 mg/day (a *B complex vitamins* also known as vitamin H that may improve the keratin infrastructure such as “Viviscal®” and “Pantogar®”), may help some men. There are thousands of herbal and homeopathic products available on the internet and in retail outlets that are promoted for hair loss but the vast majority have little or no benefits and have not gone through any serious clinical trials. However, people with hair loss are often desperate, gullible and naive and may try anything that has glossy marketing and convincing websites, despite lack of scientific evidence that they actually work.

Minoxidil (**Regain®**) is the only topical treatment approved by the US Food and Drug Administration (FDA) for use in male pattern hair loss. Although its exact mechanism of action is unclear, minoxidil opens potassium channels and was originally approved as an oral treatment for hypertension. However, results to date with topical minoxidil is not great. Significant regrowth, which is clinically apparent, is rare. In the majority of patients the most that one can expect is a slowing down of the rate of hair loss but some patients are quite happy to accept even this limited response. The 5% foam is probably more effective but may cause scalp irritation. Topical minoxidil should not be used if there is any underlying scaly disease or irritation. It should be continued twice a day for at least 4–6 months before deciding if there is any improvement. There may be a temporary shedding of hair in the first 4–6 weeks of use before re-growth starts. If there is some improvement, minoxidil can be continued once a day or three times per week for months or years. Best results occur if it is started early in the hair loss process. If the treatment is stopped any hair that has been ‘saved’ will gradually fall out.

Oral drug such as **finasteride or dutasteride** may be used for hair loss but these are only available on prescription from a doctor. **Platelet rich plasma (PRP)** and **low-level laser light therapy (LLLT)** have also been tried with varying degrees of success.

**Hair transplantation** using follicular unit transfer, when performed by an experienced surgeon, can give excellent results but is expensive. **Hair piece, hair extensions or a wig** are cheaper and more acceptable approaches for some men with severe hair loss.

### 66.4.2 Hair Loss in Women

Hair thinning in women can be very upsetting and worrying. However, most women with hair thinning will never progress to the stage where they may need a wig. Many women will be happy to grow and fashion their hair to cover the thinned out areas.

**Volumising shampoo** and conditioners may help. (e.g. “Nanogen Thickening Treatment Shampoo”; available in Boots or try your local pharmacy).

Another cosmetic option is **hair fibres** (e.g. “Nanogen Hair Thickening Fibres”). This releases thousands of microscopic colour-matched hair fibres, which bind electrostatically individual hair giving the appearance of a fuller head of hair. These can be made wind and waterproof with various locking mists (e.g. “Nanogen Fibre Locking Spray”).

Lightning the **hair colour** to blend in with the white scalp may help camouflage the hair loss process. Alternatively, temporary scalp dyes can safely blends scalp and hair colour together to conceal areas or thinning hair. (e.g. “Nanogen Aquamatch Waterproof Concealer”).

Some women may need investigations to rule out an underlying hormonal problem (e.g. polycystic ovarian syndrome or thyroid diseases) or nutritional deficiency (e.g. iron deficiency). A scalp biopsy may be necessary in severe cases or hair loss or if there is scaring in the scalp. Alopecia areata (round patches of hair loss on any part of the scalp) may respond to strong steroid scalp lotions or injections into the affected areas of the scalp.

**Vitamin supplements** promoted for hair and nail growth such as biotin 2.5 mg/day (a B complex vitamins also known as vitamin H that may improve the keratin infrastructure such as “Viviscal” and “Pantogar”, may help some women but should be avoided in pregnancy. There are thousands of herbal and homeopathic products available on the internet and in retail outlets that are promoted for hair loss but the vast majority have little or no benefits and have not gone through any serious clinical trials. However, people with hair loss are often desperate, gullible and naive and may try anything that has glossy marketing and convincing websites, despite lack of scientific evidence that they actually work.

Minoxidil (**Regain**) is the only topical treatment approved by the US Food and Drug Administration (FDA) for use in pattern hair loss in both sexes. Although its exact mechanism of action is unclear, 3% minoxidil opens potassium channels and was originally approved as an oral treatment for hypertension. However, results to date with topical minoxidil is not great. Significant regrowth, which is clinically apparent, is rare. In the majority of patients the most that one can expect is a slowing down of the rate of hair loss but some patients are quite happy to accept even this limited response. The 5% foam is probably more effective but may cause scalp irritation. Topical minoxidil should not be used if there is any underlying scaly disease or irritation. It should be continued twice a day for at least 4–6 months before deciding if there is any improvement.

There may be a temporary shedding of hair in the first 4–6 weeks of use before re-growth starts. If there is some improvement, minoxidil can be continued once a day or three times per week for months or years. Best results occur if it is started early in the hair loss process. If the treatment is stopped any hair that has been ‘saved’ will gradually fall out.

Occasionally oral drug such as **finasteride or dutasteride** may be used for hair loss in post menopausal women but they cannot be used in women of childbearing age. **Platelet rich plasma** (PRP) and **low-level laser light therapy** (LLLT) have also been tried with varying success.

**Hair transplantation** using follicular unit transfer, when performed by an experienced surgeon, can give excellent results but is expensive. **Hair piece, hair extensions or wigs** are cheaper and more acceptable approaches for many women with severe hair loss.

## 66.5 Infections and Infestations

### 66.5.1 All about Warts

Although now we know that warts and verrucae are caused by a virus and are therefore contagious, there is still much to learn about the nature of the condition. Why is it, for example, that some people tend to contract warts more readily than others? And why do some warts disappear overnight for no apparent reason? Whatever the answer to these questions, it is clear than many people suffer pain and embarrassment because of their warts. Modern treatment can, when properly applied, go a long way towards alleviating the problem.

There are numerous traditional “cures” for warts such as “the first spit in the morning”, banana skins, duct tape, etc. One cure is “The Crossroad Remedy”. The wart sufferer should rub the warts with stones, which is then wrapped in a brown paper bag and left at a local crossroad. Some inquisitive person picking them up would, by handling the stones, heal the patient and acquire warts himself over the following 6–12 months.

Another popular “cure” is to visit a holy well that has the cure for warts. The person with the warts has to go to the holy well and wash the affected areas in water from the well while saying a prayer.

Verrucae (Plantar Warts) are ingrown warts on the foot and they are contagious. They are caused by a virus entering the foot via a slight area of damage. Swimming pools and changing rooms are common sources of infection and hence children are frequent verrucae sufferers.

Genital warts (on the penis or the vagina) are usually sexually transmitted and always need investigation and treatment by a doctor.

Modern treatment for warts is usually achieved by the use of topical creams or gels from the chemist or the doctor. If this fails then your doctor might consider the use of cryosurgery in people over the age of 12 years and occasionally in children 6 to 12 years old.

#### Cryosurgery

Cryosurgery is a method of freezing the wart using very cold liquids such as liquid nitrogen, which is usually sprayed onto the wart using special hand-held cryoguns. This creates a localised frostbite or cold burn, which selectively destroys the wart without damaging underlying structures. Freezing small warts using cryosurgery causes minimal discomfort and can usually be tolerated by most adults without local anaesthetic. Larger warts (greater than 5 mm) can be more painful to treat and therefore your doctor may suggest using local anaesthetic prior to treatment to numb the area much the same as the dentist would numb a tooth before a filling. 75–90% of warts on the hands and feet should clear with one single treatment using cryosurgery when the procedure is properly carried out an experienced Cryosurgeon. However if someone has numerous warts on their hands or feet, the doctor may not treat all the warts in one session.

After the treatment the frozen area will swell up and may blister like a burn. It usually heals within 2–6 weeks without scarring.

### 66.5.2 Patients instructions on detection and treatment of head lice

Confirm the diagnosis by fine combing wet hair, with conditioner looking for a **living, moving louse** under good light.

- Start with the teeth of the detection comb touching the skin of the scalp at the top of the head. Draw the comb carefully towards the edge of the hair.
- Look carefully at the teeth of the comb in good light.
- Do this over and over again from the top of the head to the edge of the hair in all directions, working around the head.
- Head lice are little insects with moving legs. They are often not much bigger than a pinhead, but may be as big as a sesame seed (the seed on a burger bun).

Use a head lice lotion, cream or foam (not a shampoo) if you find a living, moving louse. If you find “nits” (empty egg shells stuck to the hair shafts) and the person has an itchy scalp and has not been recently treated for head lice, it is reasonable to assume they have head lice and should be treated.

- Part the hair near the top of the head, put a few drops on to the scalp and rub it into the scalp and 3 cm down the hair shaft. Part the hair a bit further down the scalp and do the same again. Do this over and over again until the whole scalp is wet.
- Read the manufacturer’s instructions as to how long the lotion, cream or foam should be left on, before washing out. (Lyclear cream rinse—10 min, Derbac M—12 h).
- Check all household contacts and all best friends for a living, moving louse.
- Anybody with a living, moving louse should be treated with a head lice lotion, cream or foam (not a shampoo).
- Re-treat after seven days to clear any new lice that have hatched out from eggs that survived the first treatment.
- Re-check all treated scalps two days after the second treatment, to confirm there is no living, moving lice.
- The itch may take 2 weeks to subside and the “nits” (empty egg shells) can last a lot longer. Do not retreat unless you find a living, moving louse.
- In resistant cases, fine comb for 30 min, once every 4 days, for 2 weeks, to physically remove the lice.

We recommend the following head lice treatment \_\_\_\_\_

### **66.5.3 Treating Fungal Nail Infections with terbinafine (“Lamisil®”) Anti-fungal Tablets**

Before taking a course of tablet treatment for fungal nail infection, diagnosis has to be confirmed by the doctor taking nail clippings from one or more of the affected nails and sending it to the hospital lab for fungal, stain and culture. It usually takes a month to get the full results back as fungus grows very slowly in the lab.

If the nail clippings confirm that there is a fungal nail infection which is sensitive to terbinafine tablets, the doctor will arrange to take routine bloods from you before starting a 3 month course of terbinafine tablets. It is important that you have your bloods checked before starting the course of treatment and have the bloods repeated 1 month into the course of treatment to ensure the tablets are not causing any side effects. Side effects from terbinafine tablets are rare but occasionally they can cause nausea, a generalised rash or liver problems. Should you develop any signs or symptoms suggestive of liver disease such as itch, unexplained persistent nausea, decreased appetite, jaundice (yellow eyes and skin), vomiting, tiredness, right upper tummy pain, dark urine, or pale stools please stop the tablets immediately and let the doctor who prescribed the tablets know.

The doctor will usually give you a prescription for the first month of the tablets and will not give you the prescription for the last 2 months until your bloods have been checked after completing the first month of tablet treatment.

On completion of the 3 month course of treatment your nails will still look unsightly and perhaps crumbly. It will take a further 9 months for the nails to grow out clear.

Terbinafine tablets are usually about 85% effective at clearing Trichophyton rubrum which is the most common fungus to infect toenails. Some patients may get re-infected by the fungus a few years later. The chances of this can be reduced by wearing leather shoes in winter and open toe sandals in the summer to let the air at the feet. You should also be careful not to walk around barefoot anywhere. Wear shoes, slippers or flip-flops in the family home, in public places such as swimming pools, changing rooms, gymnasiums and hotels.

If you are worried that there may be fungal residue in your footwear, please sprinkle miconazole nitrate 2% w/w powder (“Daktarin®” powder) into all your shoes once at night for 7 days at the start of the 3 month course of terbinafine tablets.

#### **In Summary, This is the Normal Schedule for Treating Fungal Nail Infections**

On your first visit, the doctor will take nail clippings for fungal, stain and culture and will also take routine blood tests.

After 1 month, you can phone for the results. If the fungal clippings grow fungus which is sensitive to terbinafine tablets and if your routine blood tests are normal, the doctor will post you out a prescription for 1 month of terbinafine tablets, 250 mg once daily. You should also sprinkle your shoes with miconazole nitrate 2% w/w powder (“Daktarin Powder®”) daily for 1 week.

After completing 1 month of the tablets, you return to the nurse for routine blood tests. The nurse will organise for the doctor to issue another 2 months supply of terbinafine tablets on the same day (i.e. 3 months in total).

It will take 9–12 months **after finishing** the tablets before you notice the nails growing clear.

Should you require any further information please talk to your doctors or nurse.

## 66.5.4 Treatment of Scabies

We recommend the following treatment: Permethrin 5% w/w Cream (“Lyclear Dermal Cream®”).

### 66.5.4.1 Instructions for Use

1. The person with scabies and all their household contacts and close personal contacts (e.g. girl-friend, boyfriend, and child minder) need to be treated.
2. Apply the cream or lotion **from your neck downwards** covering every square inch of your body. Pay particular attention to applying the lotion all over the hands, wrists, feet, ankles, armpits, buttocks and groin. The lotion can be applied with your hand or with a 2" paint brush.
3. After the lotion or cream has been in contact with the skin for 12 h (e.g. overnight) you may have a bath or shower and wash it all off. Reapply the lotion or cream in the same way 1 week later. Change your bed linen, underwear and towels and launder them at 60 degrees centigrade after the first treatment.
4. Reapply cream or lotion to the hands if you wash them during the 12-h treatment period.
5. Two applications of the lotion is sufficient. **The itch may persist for up to a week or two after the treatment** and may require a steroid cream to the itchy areas for a few days after the scabies treatment. Do *not* apply the treatment more than two times without consulting with your doctor. If the itch hasn't settled 2 weeks after the second treatment please return to your doctor. Nodules in the genital areas may last a number of weeks after treatment and a steroid cream may be required to treat these itchy bumps.
6. Small children (<2 years old), elderly patients and anyone with a weakened immune system should also have their face and scalp treated. Otherwise, these people should be treated the same as adults, as outlined above. Pregnant women should use the same treatment as for babies under 6 months of age.
7. Remember, scabies is common and harmless. However, it can cause a very uncomfortable itch. It is contagious and all close personal contacts and household contacts need to be treated **whether they are itching or not**.
8. Make sure you have enough liquid or cream to treat everyone who needs it. Adults need 100 ml of lotion or 30 g tube of cream per treatment (i.e. 200 ml bottle of lotion or two 30 g tubes of cream for the full treatment). Children 5–12 years of age will need half this amount; children 1–5 years of age will need quarter of adult amount, children 2 months to 1 year will need one-eighth of the adult amount. Do not use in children less than 2 months old.

## 66.6 Skin Cancer

### 66.6.1 5 Fluorouracil ("Effudix®")

1. Rub a thin smear of the ointment into the "sun spots" once or twice a day. Use very little ointment but rub it in well.
2. After 4 weeks, the part you have been treating will be red and sore. Stop using the ointment for the next 4 weeks, so that the inflammation can settle down. Then start using the ointment again as before, for another 4 weeks.
3. Continue using the ointment for 4-week periods and resting for 4-week periods until the spot has completely disappeared. Then continue a little longer to destroy any seeds.
4. If possible, avoid using the ointment for 1 week before you are due to be examined at the clinic, as the doctor cannot see if the spot is cured when the area is inflamed.
5. Sunlight will make the reaction much worse. Shield the "sun spots" from the hot sun. If necessary you can cover it.
6. If the inflammation should become very severe or painful, contact your doctor.
7. Do not use this ointment for any other purpose, nor apply it to other spots or rashes, nor give it to anyone else, as it can be harmful if used in the wrong circumstances. Keep away from the eyes, nose, mouth, genitals and anus.
8. Do not treat an area of skin larger than  $23 \times 23$  cm ( $9 \times 9$  inches) at any one time (approximately the size of a dinner plate).

## 66.6.2 Primary Melanomas

You recently had a small operation on your skin and examination of the sample under the microscope showed that the problem was a melanoma, a type of skin cancer. You may have a lot of questions and worries, and the purpose of this leaflet is to help with some of them, but not to be a substitute for a good conversation with your doctor. It is a good idea to:

- Write down all the questions you would like answered and bring them to your next visit.
- Bring another family member or close friend so that you can both discuss the information after your visit and be sure that you both have the same memory of what was said. This will help your family understand what has happened which makes it in turn easier for you to cope with a worrying situation.

### How Will I Be Followed Up?

Your melanoma was in the skin and has been removed by either one or two operations. After these the majority of people who have had melanomas of a similar thickness as yours remain well with no further problems, but because just over one in ten do develop signs of melanoma spread, we plan to see you back at the clinic for regular check-ups. At these visits, we will:

- Look at and feel your scar.
- Check the lymph glands in your groin or armpit to be sure none are bigger than normal.
- Give you a total body skin examination to be sure that you do not have another small early treatable melanoma (once you have had one melanoma you are at increased risk of developing a second so we aim to identify and treat this as soon as possible.)

Some people with melanoma have a very large number of moles. If you are in this group we may take photographs of some of these moles, and compare your skin with these photos at your next clinic visits. Not everyone needs these photographs. You should have a full skin check up every 6–12 months for the first 5 years after your diagnose and then yearly for the rest of your life.

If you have any new or changing skin lesions you are concerned about between visits we will always see you early so do not hesitate to phone and ask for your appointment to be brought forward.

### Why Did I Get a Melanoma?

We also want to know the answer to this question so that we can try to prevent other people developing melanoma in the future. In about two thirds of people with melanoma, too much sun exposure is important. This may be a childhood spent in a sunny country, or a history of many sunny summer holidays, particularly if you remember severe sunburn with blistering or peeling. People with melanoma are usually white skinned and have pale skin which does not tan easily but goes red in the sun. They are often fair or red haired, have blue eyes and may have a lot of both moles and freckles. However, about one third of people with melanoma do not fit into the group described above and may have inherited genes which makes them more likely to develop melanoma. Research in this area is still ongoing.

### Are My Children More Likely to Get Melanoma?

One melanoma patient in 50 has a history of melanoma in a close relative. If you are in this group your children could be at increased risk. In these families we offer regular skin examinations to all family members. If you do not have a close relative who has also had melanoma your children are not

at greater risk, but most families that have had a person with melanoma become very careful and sensible about avoiding sun burning. If any of your family members have unusual moles on their skin, which you would like us to check, we would be happy to see them.

### **What Do I Do Now?**

Most people with melanoma do not tolerate sunbathing well and sunburn could increase your risk of a second melanoma. We therefore suggest that you become very sensible over sun exposure. This does not mean never having a holiday in a sunny country but it does mean avoiding strong noonday sun. Comfortable cotton clothing is an excellent sunscreen, so plan your holiday wardrobe around long cotton trousers or skirts, long sleeved cotton tops and a broad-rimmed hat.

Sunscreen creams, even those called total sunblock, have not yet been shown to protect against melanoma. They do prevent sunburn, so use them as part of your skin protection routine, but not in place of clothing. Oral sun protection is also available in some countries and can be used in addition to the topical sun creams.

### **Further Information**

Please ask us any other questions that are important to you. Women may want to ask about future pregnancies, use of the oral contraceptive or hormone replacement therapy. Advice is best tailored to you personally so do tell us your personal worries. Although there is a large amount of information on the Internet, most of it is aimed at the minority of patients whose melanoma has spread beyond their skin. It therefore does not apply to you, and you may find it unnecessarily alarming.

### **Warning Signs to Look out for in a Mole**

Melanomas usually arise from freckles or moles. The warning signs to look out for is *any one* of the following three major signs:

- A mole or freckle that is getting bigger (**change in size**).
- **Or** A mole or freckle that is **changing in shape**:
- Most moles are round or oval with a symmetrical shape.
- When a mole develops an irregular border, it is a bad sign.
- **Or** A mole or freckle that **changes colour**:
- Most moles are an even shade of light or dark brown.
- When a mole develops irregular shades of colour it is a bad sign.

If any of your moles develop **any one** of the above signs you should contact your doctor immediately as early detection and removal of a melanoma can be life saving.

Other minor warning signs to look out for in a mole or a freckle are as follows:

- Itch
- Size greater than the head of a pencil (i.e. >7 mm).
- Bleeding or crusting
- Inflammation

### 66.6.3 Skin cancer screening

These are the warning signs that a lesion (a growth, a sore a freckle or a mole) that is present for more than 6–12 weeks may be turning cancerous in adults:

“New Cancers Do Show”

- **New**—A new growth, sore, freckle or mole in the last 6–12 months.
- **Changing**—A growth, sore, freckle or mole that is changing in size, shape or color over the past 6–12 months.
- **Different**—A growth, sore, freckle or mole that looks, feels or behaves differently from any other growth, sore or mole on the body (the “ugly duckling”).
- **Sore**—A growth, sore, freckle or mole that is sore, tender to touch, bleeding or itchy and will not heal after 6–12 weeks.

If a growth, sore, freckle or mole shows one or more of these warning signs it should be checked by your doctor. The more warning signs, the greater the risk of skin cancer.

Early detection saves lives!

#### 66.6.4 Skin cancer. What to look out for

There are two main types of skin cancer, melanomas and non-melanoma skin cancers.

**Melanomas**—This is the most serious and dangerous type of skin cancer. They are also called “malignant melanoma”. They grow rapidly, spread early and can be fatal. They usually arise from freckles or moles. The warning signs to look out for are **any one of the following three major signs**:

- A mole or freckle that is getting bigger (**changing in size**).
- **Or** A mole or freckle that is **changing in shape**: most moles are round or oval with a symmetrical shape. When a mole develops an irregular border it is a bad sign.
- **Or** A mole or freckle that is **changing in colour**: most moles are an even shade of light or dark brown. When a mole develops irregular shades of colour it is a bad sign. Some melanomas may be red or flesh coloured.

If any of your moles develop **any one** of the above signs you should contact your doctor immediately as early detection and removal of a melanoma can be life saving.

Other **minor warning signs** to look out for in a mole or a freckle are as follows:

- Itch
- Size greater than the head of a pencil (i.e. >7 mm)
- Bleeding or crusting
- Inflammation.

**Non-melanoma skin cancers**—These include basal cell carcinomas (BCC) and squamous cell carcinoma (SCC). SCCs grow slowly and rarely spread beyond the skin unless they are neglected for a long time. BCC's almost never spread beyond the skin so they are not usually fatal. However, they can spread locally within the skin and cause troublesome ulcers or damage local structures such as the eyes, ears or lips.

The warning signs to look out for are as follows:

- A new growth on the skin, which appears for no apparent reason.
- A sore or an ulcer that will not heal after 2–4 weeks.
- A persistent isolated scaly patch on the skin that does not clear up with topical creams.

Many people just notice a bump that looks like a mosquito bite but does not heal. It grows slowly and sometimes bleeds when scrubbing with the towel.

If you have any of these warning signs please get your doctor to check your skin.

### 66.6.5 Warning signs for melanoma

- A mole that suddenly gets bigger in size (diameter) or you develop a brand new mole on your skin that continues to grow. (**Change in size**)
- The mole develops a ragged or uneven outline (moles are normally round or oval and have a smooth edge). (**Change in shape**)
- The mole develops a mixture of different shades of brown, black or other colours through it (normal moles usually have only one colour and the colour does not change over time) (**Change in colour**)
- The mole **looks completely different** than the rest. (“the ugly duckling sign”)
- The mole becomes **red or inflamed around the edges**.
- The mole starts **bleeding, oozing or crusting**.
- The mole starts to feel different, for example, slightly **itchy or painful**.
- Not all melanomas are brown or black—they may be flesh coloured, red or pink. Any new or changing lesion on the skin that you cannot explain needs to be checked by a doctor with experience in dermatology.

## 66.7 Skin Surgery

### 66.7.1 Anticoagulants and surgery-patient information

If you are taking blood thinners prior to surgery please discuss whether you need to stop, reduce or delay your dose with your surgeon. We normally recommend the following:

**Aspirin** (ASA) is often taken by patients *without* clear indication (e.g. post heart attack or stroke or post-stenting or bypass surgery) in which case it can be stopped 10–14 days before surgery. Otherwise it is unlikely to cause significant bleeding problems in isolation at 75–300 mg daily. If you are having *medium* or *high risk* skin surgery discuss with your GP if it is safe to stop your aspirin 10–14 days before your surgery for full reversal or 5 days for 50% efficacy. Restart 7 days post-surgery.

**Clopidogrel** ("Plavix"®) cause prolonged oozing. Postpone surgery until off drug if possible (e.g. sometimes used for 1 year post cardiac stenting or bypass). If you are having a *high bleeding risk* procedure ask the cardiologist's or GP's advice regarding the risk of stopping clopidogrel for 7 days before your surgery and if a substitute alternative drug should be used while off the drug. Restart 7 days after your surgery.

**Warfarin:** Stopping or avoiding warfarin is not usually justified. Check INR bloods 72 h before your surgery. The bleeding risk is very small if the INR is <3.5 for *low risk* procedures. For *medium* or *high risk* procedures aim for an INR of 2–2.5 if the therapeutic range allows for the reason you are taking warfarin. Take advice from your GP or haematology if INR reduction needed.

#### Novel Oral Anticoagulants/Direct Acting Oral Anticoagulants (NOACs/DOACs)

- E.g.: apixaban ("Eliquis"® 2.5–5 mg BD), dabigatran ("Pradaxa"® 15 mg BD), edoxaban ("Lixiana"® 60 mg OD), rivaroxaban ("Xarelto"® 15–20 mg OD)
- *For low risk cutaneous surgery procedures\**:
- Perform the procedure just before the next dose is due  
**or**
- Perform the procedure approximately 18–24 h after the last dose. The drug can be restarted 6 h post-surgery. This means one dose of a drug taken twice a day (e.g. "Eliquis"® or "Pradaxa"®) may be missed. For once a day drugs (e.g. Xarelto® or Lixinana®) take the daily dose 6 h post-surgery on the day of the surgery.
- *For moderate risk cutaneous surgery procedures\**:
- Check with the prescriber (e.g. cardiologist) and consider stopping the drug *24 h before the procedure*.
- *For high risk cutaneous surgery procedures\**:
- Check with the prescriber (e.g. cardiologist) and consider stopping the drug *48 h before the procedure*.

\* Further dose adjustments may be required for patients with poor kidney function.

#### NSAIDs

Stop three days before your surgery and for 7 days post-surgery if possible for moderate and high risk procedures.

## Herbal Remedies and Supplements

Examples include (but are not restricted to): Garlic, Ginger, Ginkgo, Ginseng, Saw Palmetto, Fish Oil (e.g. cod liver), Camomile or Feverfew. Many can promote bleeding (and other relevant effects). This is only likely to be significant for patients who are on other blood thinners or those at high risk from post-operative bleeding. Discontinuation of all supplements (including herbal teas) at least 2 weeks before your surgery.

## Risk Stratification for Skin Surgery Procedures

### *Low risk procedures*

- Curettage
- Punch biopsy
- Incisional biopsy—scar length <10 cm
- Excision and direct closure on trunk, limbs or compressible head and neck sites (scar length <10 cm)
- Joint injection
- Cryosurgery

### *Moderate risk procedures*

- Excision and direct closure on non-compressible areas (neck, lip, genitals)
- Wide excision and direct closure on trunk and limbs
- Secondary intention wounds on compressible sites
- Grafts on compressible sites (and split thickness graft donor sites)
- Small local flaps (e.g. rhombic on nose, or wedge or helical rim advancement on ear)
- Ingrown toenails

### *High risk procedures*

- Secondary intention wounds on non-compressible sites
- Excision within the orbit (e.g. eyelids)
- Where bone is involved
- Local flaps on head and neck with wide undermining (e.g. forehead, periocular—especially orbital, cheek, large nose flaps, neck)
- Local interpolated flaps (e.g. paramedian forehead flap)
- Wide excision and direct closure on non-compressible sites (e.g. neck)
- Grafts on non-compressible sites

## 66.7.2 Care of your wound after surgery

- Wounds on the face are usually left open. Other wounds may be covered with a light dressing or band-aid for the first few days. Provided they are dry, they can then be left open, which makes it easier to inspect them.
- You can bath or shower after surgery and get the wound wet. However do not rub or scrub over the wound until it is fully healed. If you have had steri-strips (paper strips) placed on the wound, you must keep them dry for at least 7 days.
- Your doctor or practice nurses will tell you when the stitches need to be removed. Stitches on the face are left in for approximately 5–7 days. Stitches on the body and scalp are usually left in for 7–10 days. Stitches on the leg are usually left in for up to 2 weeks.
- Sometimes wounds will **bleed** after surgery. If this happens after going home please put firm pressure on the wound for ten consecutive minutes using some kitchen towel, tissue paper or clean cloth. Bleeding from the arms or legs can be further controlled by elevating the limb as high as possible. If this does not control the bleeding you should contact your doctor or nurse immediately.
- Surgical wounds sometimes become **infected**. This is usually signified by pain, redness around the wound, a smelly discharge, a pussy discharge and a wound that is getting worse rather than better as the days go by. If you have any of these suspicious signs and you are worried please contact your doctor or practice nurses immediately as antibiotics by mouth at any early stage can prevent more serious infection. Infections may be prevented by bathing the wound in an antiseptic solution daily till the wound heals. After drying the wound, apply some antiseptic cream which can help keep the wound free of infection.
- **Scars** can take 9 to 18 months to fully blend into the skin. Daily massaging with "Bio Oil" can help fade a scar. However, scars on the upper chest, back and upper arms tend to become stretched after 9 or 18 months and can sometimes leave a rather unsightly scar. This stretching of scars is unusual in other sites. Very occasionally a person may develop a "keloid scar" which is an unsightly, thickened, lumpy, deep red scar. Keloid scars are a sign of excessive wound healing and happens in a small percentage of patients. Although difficult to manage, there are treatments that can improve the cosmetic appearance of keloid scars.
- If you have had surgery on your foot or leg you should go home immediately after the operation and lie out on the couch with your foot up on the armrest at the far end of the couch and keep it as elevated as possible (at least 30 degrees above horizontal) for the remainder of the day. Do not swim in a pool or partake in any strenuous exercise until the wound is fully healed (usually 2–4 weeks).
- If you have had surgery on the face or around the eyes, you can expect considerable **swelling or bruising** which can take 1–2 weeks to subside. Sometimes an eye can become swollen and closed after surgery but this usually resolves after 5 or 7 days. If you think the swelling is due to infection, please contact your doctor or nurse.
- If you have had some tissue sent to the lab for histology, please phone back in 2–3 weeks' time for the results.

### 66.7.3 Cryosurgery clinic

1. Liquid Nitrogen Cryosurgery is an extremely effective treatment for the destruction of unwanted tissues like verrucae, warts, certain types of skin cancers and other skin growths, with minimal scar formation. Occasionally, there may be residual discolouration in the treated area. This is usually temporary, but it can take several months for the normal skin colour to return. Occasionally you may be left with permanent light or dark discolouration of the skin.
2. Liquid Nitrogen is extremely cold ( $-196^{\circ}$  centigrade). Its application causes rapid freezing to produce an instant and intense frostbite, the extent of which can be accurately controlled. For larger lesions your doctor may use a topical or local anaesthetic (like a dental anaesthetic) to numb the area before treatment.
3. There may be some **pain or discomfort** at the time of treatment and for a few hours afterwards. This seldom needs more than Paracetamol or ibuprofen. Paracetamol can be taken every 4 h but not more than four doses in 24 h. For severe pain take the maximum dose according to your age and alternate Paracetamol with Ibuprofen every 3 h in the first 18 h post cryosurgery. If necessary Paracetamol and ibuprofen can be taken at the same time.
4. The treated site and surrounding skin may **swell** within a few minutes and a **blister** may develop within a few hours or a day or two later. The blister may be filled with clear or blood stained fluid. Burst the blister with a sterile pin and squeeze out the fluid. The swelling will then get smaller and a scab will form.
5. For as long as the wound is **oozing**, clean it daily with an antiseptic wash. Wash gently, pat dry and apply a dry dressing or an antiseptic cream. **Bleeding** occasionally occurs and can usually be controlled by elevating the bleeding site and applying firm pressure with some padding and a firm bandage.
6. Once a scab has formed, leave open to the air. Wait for it to fall off, which it will do when the underlying skin has healed.
7. Areas frozen on the face may result in swelling of the eyelids and/or cheeks. No special treatment is required, as it always goes down in a few days.
8. Healing time after liquid nitrogen treatment varies from 2 to 8 weeks and may be longer on some parts of the body, such as the legs and feet. Wounds occasionally get **infected** and this may cause heat, redness, swelling and a smelly, pussy discharge, with pain that gets worse rather than better as the days post treatment pass by. If you think the wound is infected, please contact your doctor or nurse immediately as you may need an antibiotic.
9. Areas frozen on the scalp, forehead and temples may occasionally produce a dull headache. Paracetamol is usually all that is required.
10. If you have any worries or problems following the freezing treatment, do not hesitate to contact your doctors or nurse.

## 66.8 Sun Protection

### 66.8.1 Be safe in the sun

#### Ten Rules for People with Fair or Sensitive Skin and Those with Sun Damage

1. Cover up in the sunny weather—wear a broad rimmed cotton or felt (not a loosely woven straw) hat that covers your face, ears and the back of your neck. Wear a long sleeved shirt and long trousers or a long dress.
2. Put a total sun block on the exposed skin (e.g. face, ears, neck, hands and feet). Choose a *sun protection factor (SPF) of 30 or greater* (i.e. the amount of UVB blockage). UVA protection factor must be at least one-third of the labelled SPF so choosing a sunscreen with a higher SPF will also mean high UVA protection.
3. Avoid going out in the middle of the day (11 a.m.–3 p.m.) when the sun is most dangerous.
4. Remember that you can get sun damage even on cloudy days (70% of the damaging sun rays will penetrate through clouds, through glass and water).
5. Take care to protect your skin from the sun all year round, not just the summer months. Be extra careful when overseas in sunny countries.
6. Never use a sun bed.
7. Remember there is no such thing as a “safe tan”. Tanning is a sign of skin damage. However, fake tans are safe but you still have to use all the above precautions (i.e. total sun blocks and protective clothing) when in the sun.
8. Too much sun (or sun beds) can cause skin cancer especially in people who have sensitive skin. Excessive sun exposure (or sun beds) will also prematurely age the skin and cause wrinkles.
9. Beware of a cooling breeze, especially on a cloudy day when out walking, golfing, gardening or on a boat as it may temporarily mask the burning effects of the sun rays which may be hidden by the clouds.
10. If you are being sun smart to protect your skin from ultra violet light you should consider taking a vitamin D supplement especially in the winter months (approximately 800 IU or 20 µg per day) as the sun shining on our skin is our principal source of vitamin D.

## 66.8.2 The Sun and Vitamin D

### Advice for People Using Sun and Ultraviolet Light Protection to Guard Against Sunburn, Redness, Wrinkles and Skin Cancer

**Vitamin D** is essential for healthy bones as it allows calcium to be absorbed in the gut. The recommended daily intake of Vitamin D for adults is 400 IU (10 mg). However, some doctors recommend twice this amount (i.e.: 400–800 IU or 10–20 ug per day). People with kidney disease, kidney stones or sarcoidosis should have their vitamin D and calcium levels monitored regularly.

Sunlight is a good source of Vitamin D. Using sun protection may result in Vitamin D deficiency unless you maximise your source of Vitamin D from your diet and you take Vitamin D supplements.

Rich sources of Vitamin D include the following:

- Oily fish, such as herrings, kippers, sardines, mackerel, salmon and tuna (at least once or twice a week).
- Fortified foods such as HiLo Milk or Avonmore Supermilk
- Fortified margarines
- Eggs (seven per week or four per week if you have high cholesterol)

However, it is difficult to consume sufficient Vitamin D from diet alone. Other good sources of Vitamin D are Vitamin D supplements such as “**Adult D 1000®**” by Shield Health, “**BabyVitD3®**” by KoRa (0.4 ml/day for adults = 400 IU/day), “**D Lux spray®**” by BetterYou (1000 IU/spray), “**D-Pearls®**” by Pharma Nord (1520 IU/capsule), “**Desunin®**” 800 IU and **Boots® VitD 25 mg**.

You can also get a once monthly oral dose (“**Thorens 25,000 IU®**” once per month).

By taking sufficient dietary Vitamin D, there is no need to rely on sunlight or daylight as a source of Vitamin D, so it is safe to use total sunblocks, hats and appropriate clothing to protect your skin from damaging ultraviolet light rays.

In addition to taking extra dietary vitamin D, people who need to protect their skin from ultraviolet light should also take plenty of **calcium** to maintain good bones and teeth. Adults need at least 700 mg/day. The best source of calcium is from dairy products (milk, cheese, yoghurt).

Other sources of calcium include the following:

- Sardines
- Green leafy vegetables including spinach and broccoli, baked beans and red kidney beans
- Nuts and seeds
- Soya bean curd

People who do not take regular dairy produce may need to take a calcium supplement every day. Some supplements have both calcium and vitamin D (e.g.: “**Calcichew D3 Forte®**”, “**Caltrate®**”, “**Ideos®**” and “**Osteofos D3®**”)

To maintain healthy bones you should also do some weight-bearing exercises (e.g. brisk walking, jogging, dancing, tennis, golf etc.) for 40 min at least three times a week.

## 66.9 Miscellaneous

### 66.9.1 Advice for patients with varicose ulcers

- Don't stand for long periods
- Whenever possible, sit or lie down with your leg elevated
- Take exercise regularly. Walking is good, standing is bad.
- If unable to walk, exercise your calf muscles by bending your ankle up and down for 5 min every hour while your leg is elevated
- Don't wear garters or stockings which have tight elasticised tops
- If overweight, lose some pounds
- Don't smoke
- You will need high compression stockings or compression bandaging applied regularly provided you have good circulation to your feet.
- When your ulcer is healed, wear medical compression stockings which have been recommended by your doctor for the rest of your life.
- Put these stockings on before getting out of bed in the morning and only take them off last thing at night when you are getting into bed
- Do no rub anything onto the skin on your legs or ulcers unless it has been recommended by your doctor. When showering, use a soap free wash or syndets.
- Emulsifying ointment or Paraffin gel rubbed downwards is a safe moisturiser and can be used as a soap substitute
- Eat a healthy balanced diet with plenty of lean red meat, oily fish, green leafy vegetables, and fresh fruit.
- Have a blood test with your doctor to ensure you have no underlying problems that may slow up the healing of your ulcer.

### 66.9.2 Hyperhydrosis

- Keep cool, avoid hot fires, overheated cars, too much clothes, hot spicy foods, alcohol or anything else that makes you sweat. For more information see: [www.hyperhidrosisuk.org](http://www.hyperhidrosisuk.org) (a UK website) or [www.sweathelp.org](http://www.sweathelp.org) (a USA website).
- Keep calm, avoid stress (if possible) and try not to rush.
- Wear loose fitting cotton clothing. White and black shirts are better at hiding sweat stains. Wear leather soled shoes or open sandals. Consider absorbent insoles for your shoes. ([www.simplyfeet.co.uk](http://www.simplyfeet.co.uk)) Consider disposable axillary soak pants ([www.extremeclothingprotectors.co.uk](http://www.extremeclothingprotectors.co.uk)). Consider clothing impregnated with silver or copper which reduces sweat and odour ([www.buyinconfidence.com](http://www.buyinconfidence.com) or [www.silversocks.co.uk](http://www.silversocks.co.uk) or [www.ccuxsongerrard.com](http://www.ccuxsongerrard.com) or [www.esteemclothingprotectors.co.uk](http://www.esteemclothingprotectors.co.uk)). For sports, wear fabrics that take moisture away from the skin.
- Shower regularly and avoid soaps. Use a soap free wash such as aqueous cream, Elave wash, Aveeno wash, etc.
- Use antiperspirants combined with deodorants twice a day.
- If regular antiperspirants from the supermarket do not work try special ones containing aluminium chloride which can be bought without prescription from your pharmacist or on line (“Dricle®”, “Anhydrol Forte®”, “Sweat Stop®”). Follow the instructions carefully on the box.
- If aluminium chloride antiperspirants are irritating the skin try a weaker version (e.g. [www.sweatstop.co.uk](http://www.sweatstop.co.uk)) or apply a topical steroid to the area in the morning after washing off the antiperspirant for the first 2 weeks.
- For more severe sweating of the hands or feet consider buying a home iontophoresis machine which is made up of two trays containing water and a light electrical current. You place your hands or feet in the trays for 20–30 min every other day initially and less frequently as the sweating reduces. ([www.iontophoresis.info](http://www.iontophoresis.info)).
- For resistant hyperhidrosis in the armpits consider “Botox®” which is extremely effective but expensive. It reduces sweating under the armpits by 70–100% and can last from 4 to 8 months but can be expensive. For excessive sweating on the face consider weaker versions of aluminum chloride ([www.sweatstop.co.uk](http://www.sweatstop.co.uk))
- For very severe sweating, especially if generalised or not responding to the above methods, talk to your doctor about oral prescription medication such as anticholinergics, beta blockers, or calcium channel blockers which can be extremely effective.

### 66.9.3 Keratosis Pilaris

Keratosis pilaris is a very common finding on the outer aspect of the upper arms and cheeks in children and teenagers. It may run in families.

Sometimes keratosis pilaris occurs on the thighs too, rarely elsewhere. It may occur in babies where it tends to be most obvious on the cheeks. It may remain for years but generally becomes less obvious in adult life; keratosis pilaris is uncommon in elderly people.

Keratosis pilaris tends to be more severe during the winter months or other times of low humidity when the skin dries out.

There are numerous tiny rough spots in the affected area. Each one is a horny plug, sometimes rather red but only rarely itchy and never sore. When keratosis pilaris occurs on the cheeks, very often the affected areas are red as well as feeling rough. When the outer eyebrows are affected the condition is known as “ulerythema oophryogenes”.

Keratosis pilaris is completely harmless but sometimes unsightly. It occurs because as the skin renews itself, old skin cells in the hair follicles get stuck, forming a scaly plug.

Treatment:

Some people find the following useful:

- Moisturising cream applied twice a day—try those containing urea (e.g. “Eucerin®” or “Calmurid®”—10% on the body and 3% on the face or “Uriage Keratosane 30®” = 30% urea for arms or legs)
- Alphahydroxy acids.
- Rubbing with a pumice stone or a “Buf-Puf®” in the shower or bath
- Vitamin A derivatives such as tretinoin (“Isotrex®”) or adapalene (“Differin®”), which are prescription gel or cream. For the first few weeks of treatment, redness and peeling of the treated areas can be expected. Vitamin A derivatives must not be used in pregnancy.

#### **66.9.4 Methotrexate Information**

- MTX is used to treat different types of psoriasis and should be used with care as one of its actions is to reduce the activity of the immune system. It may take several weeks before the skin improves.
- MTX is usually taken once a week with food, starting with a low dose such a 5 mg or 7.5 mg, which may be increased if necessary.
- As MTX can affect the blood count and sometimes causes liver damage, patients should have regular blood tests while on treatment.
- Side-effects of MTX include a feeling of nausea, rashes and mouth ulcers. It can cause a reduction in the number of blood cells that are made, which can make the patient more liable to infection. Your doctor should be informed of a sore throat or other infection, a fever, unexplained bleeding or bruising or breathlessness.
- Patients who have not had chickenpox and come into contact with either chickenpox or shingles should inform your doctor immediately, as special treatment may be indicated. If the patient develops chickenpox or shingles, your doctor should be informed immediately and MTX stopped until the spots have scabbed over when it can be restarted.
- Some drugs can interact with MTX to cause problems. Special care is needed with NSAID's which are used to treat arthritis. These and other over-the-counter medication should not be purchased without discussion with the physician.
- Alcohol can increase the risk of liver damage with MTX and should be avoided.
- MTX should not be taken during pregnancy. Both men and women must take contraceptive precautions while taking MTX and for 6 months after treatment is stopped.
- Immunisation with live vaccines such as polio and rubella (German measles) should be avoided while taking MTX.

### 66.9.5 Pruritis Ani

Puritus Ani is itching around the opening of the back passage or anus.

It is a common problem. Although it is seldom caused by serious disease, it can be very troublesome and frustrating. Most cases are caused by irritation from hairy buttocks rubbing against each other, which is further aggravated by the irritating effect of sweat and leakage of faecal material. This irritation must be reduced to allow the problem to settle.

#### General Advice

- Clean the anus carefully after bowel movements—use unbleached or minimally processed toilet paper or a wet flannel. Pat gently. Avoid baby wipes.
- Use a soap free wash or aqueous cream in place of soap for washing.
- This leaves a protective layer on the skin and avoids the irritant effect of soap. Use your fingers—not a flannel.
- Wear loose, soft cotton underwear. Avoid any tight clothing that keeps the buttocks pressed together, such as tight jeans. Free circulation of air is important. Do not use softeners when washing your underwear.
- Wear pyjamas.
- Eat a high fibre diet with plenty of cereals, fruit and vegetables. This avoids straining when passing a bowel motion, which can lead to haemorrhoids (piles) and constipation. These can lead to cracks in the anus.
- Lose weight, if appropriate, as obesity reduces ventilation of the perianal skin.
- Apply any prescribed medication carefully at bedtime (e.g. “Daktacort®” or “Protopic®”)
- Apply Zinc and Castor Oil or “Vaseline®” regularly during the day and before exercise as this acts as a lubricant and barrier cream.

#### Avoid Aggravating Factors

- Do *not* apply any other anti itch, pain relieving, or numbing creams, ointments or suppositories to your anus unless recommended by your doctors as it may cause an allergic rash. Avoid potent topical steroids.
- Do *not* use any bath additives such as bubble bath, “Radox®” or aromatherapy oils as these can irritate the delicate skin around the anus.
- Do *not* apply talcum powder, as this tends to coalesce into small lumps in the crevices.
- Do *not* eat spicy foods, such as chillies, or irritant foods, such as nuts and popcorn. Other foods that may cause problems include citrus fruit, tomatoes, pork, and excess beer or caffeine (coffee and tea).
- Try *not* to open your bowels more than once or twice per day. Frequent bowel motions are usually soft or fluid and cause more irritation.

#### Remember:

Puritus Ani can be a stubborn condition that often needs months of careful treatment. Try *not* to scratch!

### 66.9.6 Tinea Versicolor Treatment

- Apply KETACONAZOL lotion (“Nizoral Shampoo<sup>®</sup>”) to the dry skin at night and wash it out after 15 min.
- Apply the lotion from your neck down to your upper thighs and down as far as your wrists being careful to cover ALL areas in between, front and back.
- Apply the lotion for 15 min every night for 1 week. This will get rid of any itch or scaliness.
- After this treatment you may be left with a faint rash. This will gradually fade away over a few weeks after finishing the treatment especially after getting some sun.
- Do *NOT* continue the treatment indefinitely.
- The rash may recur during the summer or after a “sun holiday”. If this occurs simply treat yourself again as outlined above. Some people need to treat themselves every spring to prevent getting a “blotchy” tan in the summer.
- Remember, it is a safe and effective treatment.
- If you have any problems please contact your doctor.

### **66.9.7 Top ten self-care tips for people with psoriasis**

1. Moisturise liberally with a safe, greasy moisturiser at least twice a day.
2. Avoid dark clothes as they will show off dandruff and silvery scale.
3. Avoid excessive alcohol (keep to less than 14 units per week) and do not smoke.
4. If you are overweight, try to lose some.
5. Try a “Mediterranean diet” for at least 3 months. Take oral Omega 3 capsules daily
6. Get plenty aerobic exercise.
7. Ask your doctor to check your cholesterol, blood sugar and blood pressure annually if you are over 45 years old.
8. Remind yourself and everyone else that psoriasis is not contagious.
9. Try to stay positive; psoriasis is not usually life-threatening.
10. Carefully follow the instructions about your treatment from your doctor or nurse. If you are unsure, ask questions.



# Useful Websites, Courses, Bibliography and Patient Support Groups

67

David Buckley

The following is a list of useful websites and resources:

## 67.1 General Dermatology Resources for Patients

### Irish Skin Foundation:

Information on common skin problems such as eczema, psoriasis, rosacea, hidradenitis suppurativa, melanoma and other types of skin cancer and for patients;

[www.irishskinfoundation.ie](http://www.irishskinfoundation.ie)

### British Skin Foundation:

Get people sharing their experience with one another so they don't have to suffer in silence;

[www.britishskinfoundation.org.uk](http://www.britishskinfoundation.org.uk)

### The British Association of Dermatologists:

British Association of Dermatologists has comprehensive list of patient support groups;

[www.bad.org.uk/for-the-public/patient-supportgroups](http://www.bad.org.uk/for-the-public/patient-supportgroups)

### Skin Support:

British Association of Dermatologists website to support people in psychological distress due to

skin conditions, provides patient information, self help materials and support services;

[www.skinsupport.org.uk](http://www.skinsupport.org.uk)

### Changing Faces:

Changing Faces supports adults, children and families living with disfigurement;

[www.changingfaces.org.uk](http://www.changingfaces.org.uk)

### NHS Dermatology Patient Information:

[www.uhb.nhs.uk/pi-dermatology.htm](http://www.uhb.nhs.uk/pi-dermatology.htm)

### Patient Info:

[www.patient.info](http://www.patient.info)—a UK bases site on information on numerous health-related topics including various skin problems.

### Psoriasis:

Adults:

- Psoriasis Association: [www.psoriasis-association.org.uk](http://www.psoriasis-association.org.uk)
- Psoriasis and Psoriatic Arthritis Alliance: [www.papaa.org/resources](http://www.papaa.org/resources)
- Irish Skin Foundation/Psoriasis: [www.irishskinfoundation.ie](http://www.irishskinfoundation.ie)

Teenagers:

- Psoteen: [www.psoteen.org.uk](http://www.psoteen.org.uk)

### Eczema:

National Eczema Society (UK) Providing information, advice and support for people

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The Ashe Street Clinic, Tralee, Co. Kerry, Ireland

with eczema and those who care for them;  
[www.eczema.org](http://www.eczema.org)

Irish Skin Foundation/Eczema:  
[www.irishskinfoundation.ie](http://www.irishskinfoundation.ie)

Atopic skin disease:

[www.atopicskindisease.com](http://www.atopicskindisease.com) is a self-funded membership website for patients and practitioners. This site explains a combined approach to managing atopic eczema, combining optimal conventional topical treatment with the behaviour modification technique, habit reversal, to eliminate habitual scratching.

#### **Acne:**

- Talk Acne:  
[www.talkhealthpartnership.com/talkacne/](http://www.talkhealthpartnership.com/talkacne/)
- Acne Support is brought to you by the British Association of Dermatologists (BAD) to offer you expert, impartial advice on acne;  
[www.acnesupport.org.uk](http://www.acnesupport.org.uk)

#### **Skin Cancer:**

- Cancer research UK:  
[www.cancerresearchuk.org/about-cancer/skin-cancer](http://www.cancerresearchuk.org/about-cancer/skin-cancer)
- Macmillan Cancer support:  
[www.macmillan.org.uk/information-and-support/skin-cancer](http://www.macmillan.org.uk/information-and-support/skin-cancer)
- Irish Cancer Society:  
[www.cancer.ie/cancer-information/skin-cancer](http://www.cancer.ie/cancer-information/skin-cancer)  
[www.cancer.ie/cancer-information/melanoma](http://www.cancer.ie/cancer-information/melanoma)
- Irish Skin Foundation/Skin Cancer:  
[www.irishskinfoundation.ie/skincancer](http://www.irishskinfoundation.ie/skincancer)

#### **Alopecia UK:**

Provides information, support and advice for people with experience of alopecia areata, alopecia totalis and alopecia universalis;

[www.alopecia.org.uk](http://www.alopecia.org.uk)

#### **Vitiligo Society (UK):**

Offer support and understanding to people with vitiligo and to their families;

[www.vitiligosociety.org.uk](http://www.vitiligosociety.org.uk)

#### **Birthmarks:**

[www.birthmarkssupportgroup.org.uk](http://www.birthmarkssupportgroup.org.uk)

#### **Skin Camouflage:**

- Irish Red Cross:  
[www.redcross.ie/programmes-and-services-in-ireland/in-the-community](http://www.redcross.ie/programmes-and-services-in-ireland/in-the-community)
- The British Association of Skin Camouflage:  
[www.skin-camouflage.net](http://www.skin-camouflage.net)

#### **Allergies:**

- Allergy UK are the leading national patient charity for people living with all types of allergy;  
[www.allergyuk.org](http://www.allergyuk.org)
- Anaphylaxis Campaign (UK) supports people at risk from severe allergic reactions (anaphylaxis);  
[www.anaphylaxis.org.uk](http://www.anaphylaxis.org.uk)
- Irish Food Allergy Network:  
[www.ifan.ie](http://www.ifan.ie)

#### **Body Dysmorphic Disorder:**

- Body Dysmorphic Disorder Foundation (UK):  
[www.bddfoundation.org](http://www.bddfoundation.org)

#### **Hidradenitis suppurativa:**

- Hidradenitis suppurativa trust:  
[www.hstrust.org](http://www.hstrust.org)

#### **Irish Skin Foundation. Hidradenitis Suppurativa:**

[www.irishskinfoundation.ie](http://www.irishskinfoundation.ie)

#### **Herpes Viruses Association:**

<https://herpes.org.uk/>

#### **Hyperhidrosis Support Group: (USA + UK).**

To give advice online to those suffering from Hyperhidrosis, including basic treatment options.

[www.sweathelp.org](http://www.sweathelp.org) (USA site).

[www.hyperhidrosisuk.org](http://www.hyperhidrosisuk.org) (UK site).

#### **Lichen sclerosus:**

[www.lichensclerosus.org](http://www.lichensclerosus.org)

#### **Lupus:**

[www.lupusuk.org.uk](http://www.lupusuk.org.uk)

#### **Pemphigoid and Pemphigus:**

[www.pemphigus.org](http://www.pemphigus.org)

**Shingles:**

[www.shinglessupport.org/](http://www.shinglessupport.org/)

**UK Lichen planus:**

[www.uklp.org.uk](http://www.uklp.org.uk)

**Vulval Pain Society (VPS):** is a confidential service for women who suffer from vulval pain due to vestibulodynia and vulvodynia and associated conditions such as lichen sclerosus, thrush, interstitial cystitis, vulval eczema and vaginismus;

[www.vulvalpainsociety.org](http://www.vulvalpainsociety.org)

## 67.2 Websites for Healthcare Professionals

- **Dermnet NZ:** Facts about skin, hair and nails from the New Zealand Dermatological Society for doctors and the general public.  
[www.dermnetnz.org/](http://www.dermnetnz.org/)
- **Primary Care Dermatology Society UK:** A-Z of Clinical Guidance aimed at GPs, educational courses.  
[www.pcds.org.uk](http://www.pcds.org.uk)
- **Primary Care Dermatology Society of Ireland:** Runs an annual scientific meeting.  
[www.pcpsi.com](http://www.pcpsi.com)
- **British Association of Dermatologists:** Patient information leaflets + clinical guidelines (for dermatologists but some relevant to GP).  
[www.bad.org.uk](http://www.bad.org.uk)
- **The American Academy of Dermatology:**  
[www.aad.org](http://www.aad.org)
- **American Academy of Dermatology:** Curriculum—PowerPoint presentations providing a basic overview of most dermatology topics. Each topic is followed by a quiz.  
[www.aad.org/education/basic-dermatology-curriculum](http://www.aad.org/education/basic-dermatology-curriculum)
- **Primary Care Surgical Association:** Useful website with guidelines and discussion forum for GPs.  
[www.pcsa.ie](http://www.pcsa.ie)
- **Association of Surgeons in Primary Care:** Provides support, training and professional

development for providers of Primary Care Surgery in the UK.

[www.aspc-uk.net](http://www.aspc-uk.net)

- **GREAT Database:** Evidence-based database of systematic reviews and randomised controlled trials of eczema treatments.  
[www.greatdatabase.org.uk/GD4/Home/Index.php](http://www.greatdatabase.org.uk/GD4/Home/Index.php)
- **Cochrane Library/Skin disorders:** Databases containing high quality independent systematic reviews on skin disorders with detailed and plain language summaries.  
[www.cochranelibrary.com/topic/Skin%20disorders/](http://www.cochranelibrary.com/topic/Skin%20disorders/)
- **eIntegrity:** healthcare e-learning developed by the British Association of Dermatologists for trainee dermatologists and GPs.  
[www.eintegrity.org/e-learning-healthcare-course/dermatology.html](http://www.eintegrity.org/e-learning-healthcare-course/dermatology.html)
- An online **dermatology atlas:**  
[www.dermatlas.net](http://www.dermatlas.net)
- **Derm101:** Clinical Case Challenges.  
[www.derm101.com](http://www.derm101.com)
- **Youtube “Basic Dermoscopy” Channel:** by the International Dermoscopy Society (ISD).  
[www.youtube.com/user/dermoscopy](http://www.youtube.com/user/dermoscopy)
- **GP Notebook:** Useful for general medical and dermatology information.  
[www.gpnotebook.co.uk](http://www.gpnotebook.co.uk)
- **Medicine.ie:** for all information about prescription drugs in Ireland.  
[www.medicines.ie](http://www.medicines.ie)
- **British National Formulary:** For all information about prescription drugs in the UK.  
[www.bnf.org](http://www.bnf.org)
- **Antibiotic Prescribing in Ireland:**  
[www.hse.ie/eng/services/list/2/gp/antibiotic-prescribing/](http://www.hse.ie/eng/services/list/2/gp/antibiotic-prescribing/)
- **NICE Antibiotic Guidance (UK):**  
[www.nice.org.uk/Media/Default/About/what-we-do/NICE-guidance/antimicrobial%20guidance/summary-antimicrobial-prescribing-guidance.pdf](http://www.nice.org.uk/Media/Default/About/what-we-do/NICE-guidance/antimicrobial%20guidance/summary-antimicrobial-prescribing-guidance.pdf)
- **The National Institute for Health and Care Excellence (NICE):** provides national guidance and advice to improve health and social care.  
[www.nice.org.uk](http://www.nice.org.uk)

- A **surface anatomy mapper** that describes a location on the body can be very useful.  
<http://anatomymapper.com>
- **Atopic Skin Disease:** is a self-funded membership website for patients and practitioners. This site explains a combined approach to managing atopic eczema, combining optimal conventional topical treatment with the behaviour modification technique, habit reversal, to eliminate habitual scratching.  
[www.atopicskindisease.com](http://www.atopicskindisease.com)
- **Google images** is a very quick way to find pictures of various skin rashes or lesions to compare to what the patient has or to explain to the patient what the rash or lesion can look like on other patients. However, searching for a skin condition using Google images is not always accurate and some of the images you find may not be of the condition you are looking for.  
[www.google.com/imghp](http://www.google.com/imghp)
- **British Association for Sexual Health and HIV:** This is a very useful website with up to date protocols for the diagnosis and treatment of sexually transmitted diseases and other sexual health and HIV issues.  
[www.bashh.org](http://www.bashh.org)
- **VisualDx** is a diagnostic clinical decision support system. Their large collection of dermatologic images are used by means of algorithms to enhance diagnostic accuracy, aiding physicians to make therapeutic decisions.  
<https://www.visualdx.com/>

### 67.3 Patient Information Videos

#### British Association of Dermatologists:

- How to use emollients
- How to use topical steroids
- Treating scalp psoriasis  
[www.bad.org.uk/for-the-public/patient-information-videos](http://www.bad.org.uk/for-the-public/patient-information-videos)

#### Sheffield Childrens Hospital:

Emollients and eczema: what you need to know

[www.youtube.com/watch?v=kPBN4\\_oATEo](https://www.youtube.com/watch?v=kPBN4_oATEo)

How to apply emollients properly

[www.youtube.com/watch?v=Kp4wnfIsots](https://www.youtube.com/watch?v=Kp4wnfIsots)

#### Irish Skin Foundation Videos:

[www.youtubecom/channel/UCAFVEvXeCKK6gWXJUW-HiAQ/videos](http://www.youtubecom/channel/UCAFVEvXeCKK6gWXJUW-HiAQ/videos)

#### University of Bristol:

Useful videos for eczema treatment

<http://www.bristol.ac.uk/primaryhealthcare/researchthemes/apache/ewap//videos/>

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### 67.4 Courses and Diplomas

- **Primary Care Dermatology Society of Ireland.** They run regular courses and educational meetings.  
[www.pcysi.com](http://www.pcysi.com)
- **Primary Care Dermatology Society UK:** regular courses on various dermatology problems including general dermatology, cutaneous surgery and dermoscopy.  
[www.pcds.org.uk](http://www.pcds.org.uk)
- **Primary Care Surgical Association** run an annual scientific meeting in Ireland and have useful guidelines and a discussion forum for doctors and nurses on the site.  
[www.pcsa.ie](http://www.pcsa.ie)
- **Association of Surgeons in Primary Care** run an annual scientific meeting and various surgical courses in the UK.  
[www.aspc-uk.net](http://www.aspc-uk.net)
- **European Academy of Dermatology and Venereology** run regular courses for members and non-members around Europe.  
[www.eadv.org](http://www.eadv.org)
- **Diploma in Practical Dermatology**, Cardiff University.  
[www.dermatology.org.uk](http://www.dermatology.org.uk)  
or see [www.cardiff.ac.uk/medicine/courses/postgraduate-taught](http://www.cardiff.ac.uk/medicine/courses/postgraduate-taught)
- **Certificate/Diploma/Masters in Clinical Dermatology**, University of Hertfordshire, UK.  
[www.herts.ac.uk/courses/msc-clinical-dermatology](http://www.herts.ac.uk/courses/msc-clinical-dermatology)
- **UCD Professional Certificate in Clinical Dermatology:**  
[www.ucd.ie/medicine/studywithus/graduatesStudies/medicineMedicalSpecialties/graduatediplomadermatology/](http://www.ucd.ie/medicine/studywithus/graduatesStudies/medicineMedicalSpecialties/graduatediplomadermatology/)

- **Postgraduate Diploma in Clinical Dermatology**, Barts Diploma London. [www.qmul.ac.uk/postgraduate/taught/course-finder/courses/clinical-dermatology-online-pgdip/](http://www.qmul.ac.uk/postgraduate/taught/course-finder/courses/clinical-dermatology-online-pgdip/)

## 67.5 Textbooks

### 67.5.1 Diagnosis

- **Differential Diagnosis in Dermatology**. Richard Ashton et al. 4th edition. 2014.
- **Dermatology: An illustrated colour text**. 6th edition. 2016. David Gawkrodger, Michael Ardern-Jones.
- **Dermatology Made Easy**. Amanda Oakley. Scion Publishing Ltd. 2017. ISBN: 978 1 907904 82 0.
- **ABC of Dermatology**. 6th edition. 2014, Rachael Morris-Jones.
- **Atlas of Dermatology**. 5th Edition 2005, Lional Fry.
- **Atlas of Clinical Dermatology**. 4th edition. 2012. Anthony du Vivier.

### 67.5.2 Dermoscopy

- **Diagnostic Dermoscopy**. Paperback. 2012. by Jonathan [Bowling](#).
- **Dermoscopy: The Essentials**. 2nd edition 2011. Peter Soyer, Giuseppe Argenziano, Rainer Hofmann-Wellenhof, and Iris Zalaudek.

### 67.5.3 Treatment

- **Treatment of Skin Disease. Comprehensive Therapeutic Strategies**. 5th Edition 2017 by

Mark G Lebwohl and Warren R Heymann. (Excellent evidence based book).

- **Hanbdbook of Systemic Drug Treatment in Dermatology**. 2nd Edition 2015 by [Sarah H. Wakelin, Howard I. Maibach, Clive B. Archer](#).
- **Atopic skin disease, A Manual for Practitioners**. Christopher Bridgett Paperback – December 1, 1996. Also available on line at [www.atopicskindisease.com](http://www.atopicskindisease.com)

### 67.5.4 Dermatological Surgery

- **An Introduction to Dermatological Surgery**. Clifford Laurence. 2nd Edition. 2002.
- **Brown's Skin and Minor Surgery: A Text & Colour Atlas**, 5th Edition. 2014. Edited by [Jonathan Botting](#), Edited by [Julia Schofield](#).

### 67.5.5 Cryosurgery Books

- **Modern Cryosurgery for Cancer**. Xu K, Korpan NN, Niu L (ed), World Scientific, (2012)
- **Cryosurgery. A Practical Manual**, Pasquali P (ed). 1st edition. Springer; (2015). ISBN 978-3-662-43938-8
- **Cutaneous Cryosurgery: Principles and Clinical Practice**. 2005 by [Graham B. Colver](#) (Author), [Arthur Jackson](#) (Editor), [Rodney P R Dawber](#) (Editor).

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