

indefinite border with satellite macular lesions
at the edge

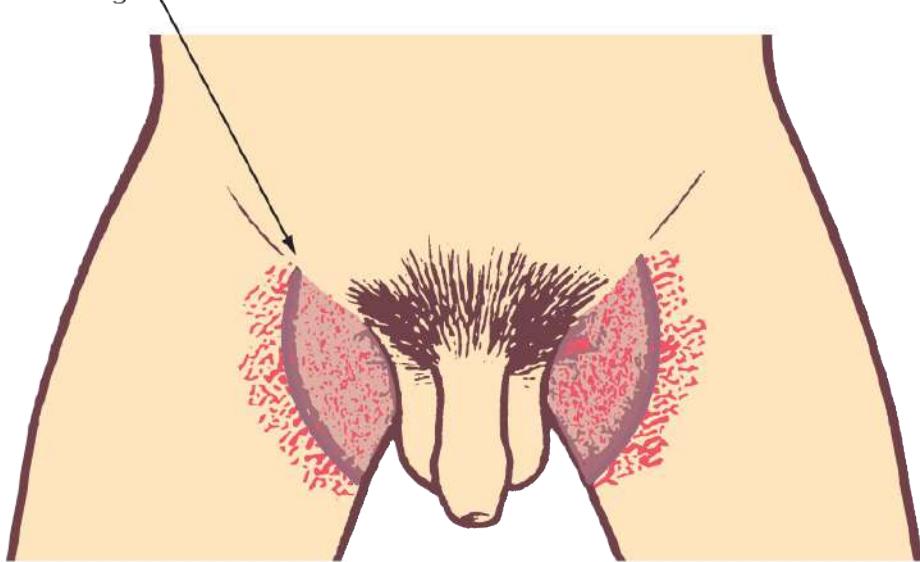


FIGURE 112.8 Dermogram for candidiasis of crural area

Treatment⁸

- Treat predisposing factors where possible.
- Apply an imidazole preparation such as miconazole 2% or clotrimazole 1%, twice daily for 2 weeks.
- Use short-term hydrocortisone cream for itch or inflammation (long-term aggravates the problem).

⌚ Erythrasma

Erythrasma, a common and widespread chronic superficial skin infection typically in a skinfold, is caused by the bacterium *Corynebacterium minutissimum*, which can be diagnosed by coral pink fluorescence on Wood's light examination. Itch is not a feature.

Clinical features

- Superficial reddish-brown scaly patches
- Enlarges peripherally
- Mild infection but tends to chronicity if untreated
- Coral pink fluorescence with Wood's light

- Common sites: groin (especially men), axillae, submammary, toe webs (see FIG. 112.9)

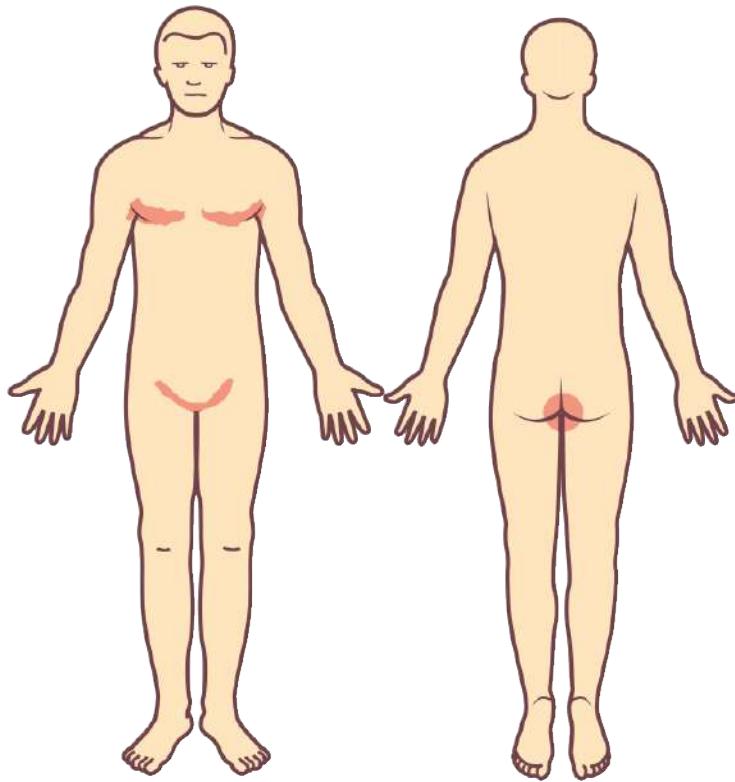


FIGURE 112.9 Typical sites of erythrasma

Treatment

- Topical imidazole, e.g. miconazole or erythromycin 2% gel
- Oral roxithromycin or erythromycin
- Loose-fitting clothing and antibacterial wash may prevent recurrence

⌚ Asteatotic eczema (winter itch)

This often unrecognised problem, which can be very itchy, is a disorder of the elderly. It is a form of eczema that typically occurs on the legs of the elderly (see FIG. 112.10), especially if they are subjected to considerable scrubbing and bathing. Other predisposing factors include low humidity (winter, central heating) and diuretics. The problem may be associated with a malabsorption state, hypothyroidism or drugs, e.g. statins, diuretics.



FIGURE 112.10 Asteatotic eczema (winter itch) showing the 'crazy paving' pattern with a pruritic scaling erythematous eruption on the legs of a 74-year-old man

Clinical features

- Dry skin
- Fine scaling and red superficial cracking
- 'Crazy paving' appearance
- Occurs on legs, especially shins
- Also occurs on thighs, arms and trunk

Treatment

Based on correcting dry skin:⁹

- Quick, cool showers (<2–3 minutes)

- Use soap-free substitutes in the shower
- Liberal use of emollients on damp skin, after the shower, e.g. QV or emulsifying ointment
- Avoid excessive heating in winter
- Avoid use of electric blankets
- Apply topical steroid diluted in white soft paraffin to reddened skin

'Golfer's vasculitis' (summer leg rash)¹⁰

This is a term used to describe an erythematous pruritic rash that appears on the legs after prolonged exercise such as golf or hiking, usually during summer months.

The rash is a red, blotchy, flat to slightly raised eruption on the lower leg. It is more common over age 50. It usually clears spontaneously within 3 days.

Brachioradialis pruritus

In this condition, itch and discomfort are limited to the outer surface of the upper limb above and below the elbow. It is often associated with sun damage, xerosis and nerve entrapment, hence the term 'golfer's itch'.²

Grover disease

Also known as transient acantholytic dermatosis, Grover disease produces small, firm, intensely pruritic, reddish-brown, warty papules with minimal scale, mainly on the upper trunk. It usually occurs in middle-aged to elderly men (typically 70–80 years). Trigger factors include heat, sweating, fever and occlusion, especially on photo-damaged skin. Diagnosis is by biopsy. Treatment is to relieve the itch until spontaneous resolution occurs. Effective treatments include topical (preferable) or oral corticosteroids and ultraviolet light.³

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Cercarial dermatitis

Cercarial dermatitis (swimmer's itch) is an acute allergic dermatitis caused by contact with schistosomes in warm freshwater lakes. Skin under swimwear is often protected.

Nodular prurigo (prurigo nodularis)

These are extremely pruritic firm lumps about 1–3 mm in diameter that erupt on the arms, legs and trunk. They can affect all ages and may manifest in late pregnancy. Definite diagnosis is with skin biopsy. They tend to run a long course and are difficult to treat.

⌚ Lichen simplex

Lichenification is a form of dermatitis caused by repeated scratching or rubbing, which results in epidermal thickening. Lichen simplex is the term used when no primary dermatological cause can be found.

⌚ Urticaria¹¹

Urticaria is a common condition that mainly affects the dermis. It can be classified as acute (minutes to weeks) or chronic (lasting more than 6 weeks). It can also be classified as diffuse wheal-like or papular (hives).

The three characteristic features of diffuse urticaria are:

- transient erythema, often with central pallor
- transient oedema
- transient intense itch

The most common causes are infections, especially viral URTIs, drug allergies and IgE-mediated food reactions.

Urticular vasculitis is a rare subtype of urticaria which causes painful rather than pruritic lesions and may have an autoimmune aetiology.

Classification according to site

1. *Superficial*: affecting superficial dermis = urticaria; occurs anywhere on body, especially the limbs and trunk.
2. *Deep*: affecting subcutaneous tissue = angio-oedema; occurs anywhere but especially peri-orbital region, lips and neck.

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Checklist of causes³

- Infections: viruses (the most common cause in children), bacteria (especially *streptococcus* infection), parasites, protozoa, yeasts
- C₁ esterase inhibitor deficiency (hereditary angio-oedema)—recurrent unexplained episodes of angio-oedema
- Allergies (acute allergic urticaria is dramatic and potentially very serious): azo dyes

drugs: penicillin and other antibiotics

foods: eggs, fish, cheese, tomatoes, others

- Pharmacological:

drugs: penicillin, aspirin, codeine

foods: fish, shellfish, nuts, strawberries, chocolate, artificial food colourings, wheat, soy beans

plants: nettles, others

- Systemic lupus erythematosus (associated with urticarial vasculitis)

- Physical:

- cholinergic: response to sweating induced by exercise and heat (e.g. young athletes)
- heat, cold, sunlight
- Insect stings: bees, wasps, jellyfish, mosquitoes
- Pregnancy (last trimester), other hormonal
- Unknown (idiopathic)—80%; possible psychological factors

Investigations for chronic urticaria

- Full blood examination—look for eosinophilia or parasites
- ANA and DNA binding tests—for urticarial vasculitis
- Skin-prick tests

Treatment¹¹

- Avoid any identifiable causes.
- Consider elimination diets (although evidence is poor).
- Use topical soothing preparation if relatively localised (e.g. crotamiton 10%, or phenol 1% in oily calamine or menthol 1% cream).
- Lukewarm baths with Pinetarsol or similar soothing bath oil.
- Use oral antihistamines (e.g. cyproheptadine) or a less sedating one (e.g. cetirizine, loratadine, fexofenadine).

- Consider adding an H₂ antagonist (e.g. ranitidine 150 mg bd).
- Systemic corticosteroids (e.g. prednisolone 50 mg once daily) can relieve acute urticaria but should be avoided as high doses are required and symptoms usually return once treatment stops.
- For severe urticaria with hypotension and anaphylaxis give IM adrenaline.
- Specialist referral is appropriate if symptoms persist beyond 6 weeks.
- Specialist treatment includes phototherapy, leukotriene receptor antagonists, sulfasalazine, cyclosporin or omalizumab.

Papular urticaria

This is a hypersensitivity to insect bites or insects in the environment, particularly seen in children aged 2–6 years. The lesions are grouped together, often as clusters of very itchy papules.

Common urticaria tends to come and go within hours but the lesions of papular urticaria persist.

The treatment for insect bites includes antipruritics and topical corticosteroids, e.g. betamethasone dipropionate 0.05% ointment or cream tds until resolved.

Flea bites

Fleas (see FIG. 112.11) cause itchy erythematous maculopapular lesions. They are usually multiple or grouped in clusters, occurring typically on the arms, forearms, legs and waist (where clothing is tight). Treat the source of infestation, particularly domestic cats and dogs. The itchy bites can be treated with an application of simple agents such as Stingose, alcohol, damp soap, anti-itch cream or a potent corticosteroid ointment.

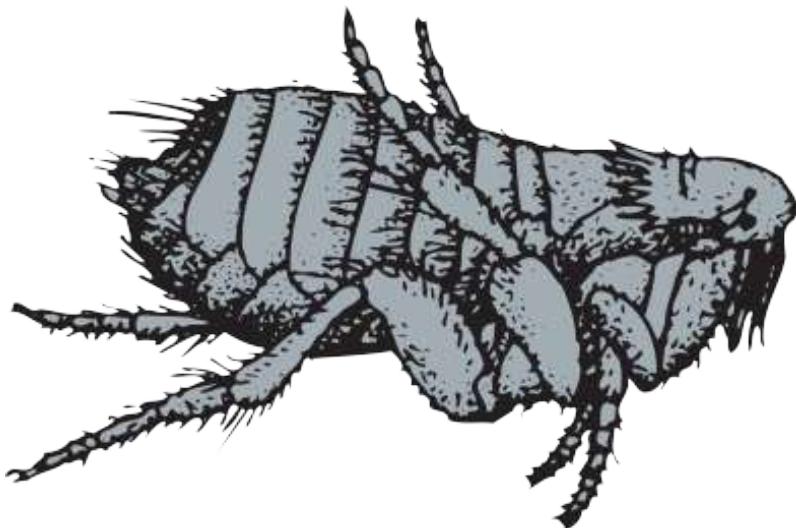


FIGURE 112.11 Flea**Bed bug bites¹²**

The common bed bug (*Cimex lectularius*, see FIG. 112.12) is now a major problem Page 1245 related to international travel. It travels in baggage and is widely distributed in hotels, motels and backpacker accommodation. The bugs hide in bedding and mattresses. Clinically, bites are usually seen in children and teenagers. The presentation is a linear group of three or more bites (along the line of superficial blood vessels), which are extremely itchy. They appear as maculopapular red lesions with possible wheals. The lesions are commonly found on the neck, shoulders, arms, torso and legs. A bed bug infestation can be diagnosed by identification of rust-coloured specimens collected from the infested residence. In hotels and backpacker accommodation look for red specks in mattresses and check luggage.

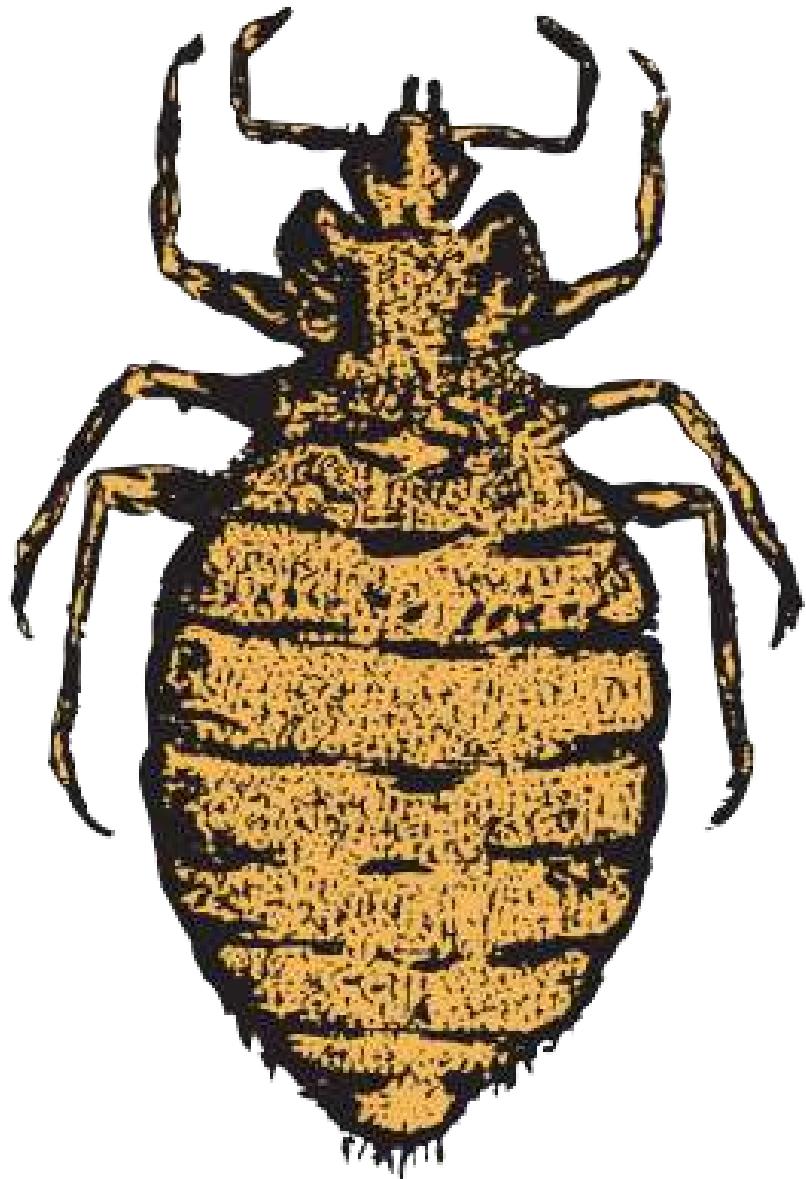


FIGURE 112.12 Bed bug

Management

- Clean the lesions with antiseptic soap.
- Apply a corticosteroid ointment.
- A simple antipruritic agent may suffice, e.g. calamine lotion.
- Call in a licensed pest controller.

Control treatment is basically directed towards applying insecticides to the crevices in walls and

furniture. Be careful of used furniture and insist that mattresses are delivered in plastic coverings.

⌚ Sandfly bites

Refer to [CHAPTER 120](#).

Other insect bites

Stinging bites are associated with various insects, including bull ants, jumper ants, bees, wasps and mosquitoes. For treatment, see [CHAPTER 120](#).

When to refer

- Lichen planus
- Dermatitis herpetiformis
- Crusted scabies
- Chronic urticaria

Practice tip

An itchy non-specific rash worse at night? Think scabies.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Bed bug bites
- Pruritus ani
- Scabies
- Urticaria (hives)

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113 Common skin problems

The power of making a correct diagnosis is the key to all success in the treatment of skin diseases; without this faculty, the physician can never be a thorough dermatologist, and therapeutics at once cease to hold their proper position, and become empirical.

LOUIS A DUHRING (1845–1913)

Skin disorders are common. They account for 10.8% of all problems¹ encountered in general practice, the most common being dermatitis/eczema, malignant skin neoplasms, solar keratoses, lacerations, warts and acne.

This chapter focuses on the common dermatoses.

Dermatitis/eczema

Dermatitis is a non-specific inflammatory response of the skin, presenting as an erythematous rash, that is usually itchy, and sometimes scaly.² The terms dermatitis and eczema are often used interchangeably, with eczema referring to the process that causes dermatitis.

Dermatitis can be divided into *exogenous* causes (allergic contact, irritant contact, photo-allergic and phototoxic) and *endogenous*, which implies all forms of dermatitis not directly related to external causative factors. Endogenous types are atopic, nummular (discoid), vesicular hand/foot (pompholyx) and pityriasis alba.

Dermatitis can occur as the result of dry skin, which impairs the barrier function of the skin, making it more susceptible to irritation by soap and other contact irritants, the weather, temperature and non-specific triggers.

The meaning of atopy

The term ‘atopic’ refers to a hereditary background or tendency to develop one or more of a group of conditions, such as allergic rhinitis, asthma and eczema. It is not synonymous with allergy.

An estimated 10% of the population are atopics, with allergic rhinitis being the most common

manifestation.³

Atopic dermatitis

Features of classic atopic dermatitis:^{2,4}

- itch
- usually a family history of atopy
- about 20% of children are affected, signs appearing between 3 months and 2 years
- specific triggers (see TABLE 113.1) may be evident

Table 113.1 Trigger factors for atopic dermatitis

Dust mite (common)
Sweating
Sand (e.g. in sandpits)
Extremes of hot and cold
Rapid temperature changes
Soap, shampoo and water/frequent washing, especially in winter
Chlorinated water
Bubble baths
Infection (viral, bacterial, fungal)
Allergy
Stress/emotional factors
Skin irritants:

- wool (e.g. sheepskin covers)
- rough clothing
- chemical disinfectants
- detergents
- petrochemical products
- pollens

Scratching and rubbing
Perfumes
Poor general health

- food allergies are rarely the main cause of the condition

- lichenification may occur with chronic atopic dermatitis
- flexures are usually involved (see FIG. 113.1)
- dryness is usually a feature



FIGURE 113.1 Atopic dermatitis in the flexures of the knees: a typical location

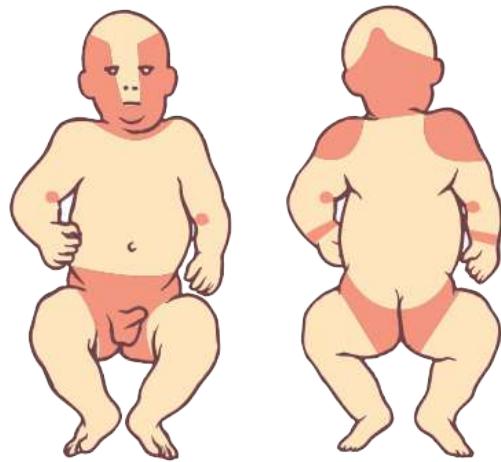
Criteria for diagnosis

- Itch
- Typical morphology and distribution
- Dry skin
- History of atopy
- Chronic, relapsing dermatitis

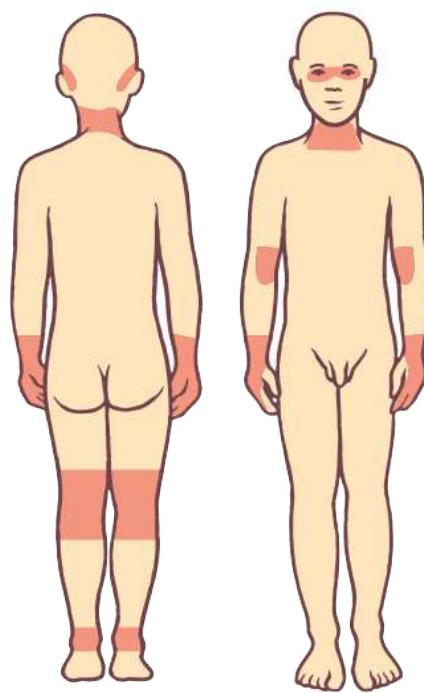
Distribution

The typical distribution of atopic dermatitis changes as the patient grows older. In infants the rash appears typically on the cheeks of the face, the folds of the neck and scalp and extensor surfaces of the limbs. It may then spread to the flexures of limbs and groin (see FIG. 84.9 in CHAPTER 84). The change from infancy through to adulthood is presented in FIGURE 113.2 .

(a)



(b)



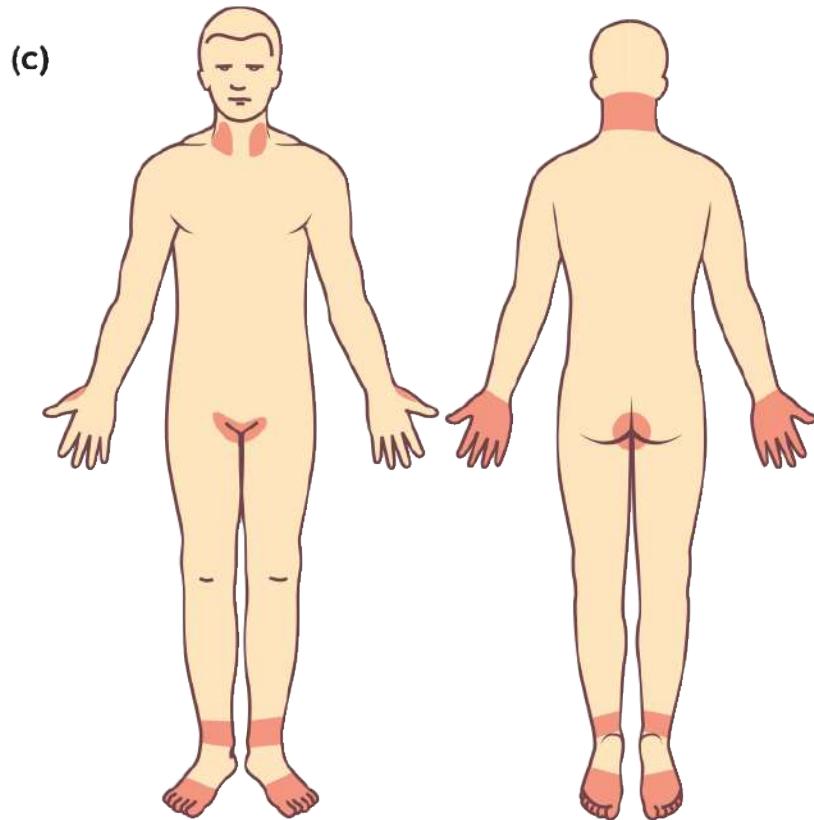


FIGURE 113.2 Relative distribution of atopic dermatitis in (a) infants, (b) children and (c) typical distribution of atopic dermatitis in adults

During childhood a drier and thicker rash tends to develop in the cubital and popliteal fossae and on the hands and feet, which may be dry, itchy, fissured and painful. The face often clears. Refer to [CHAPTER 84](#).

Prognosis

It is generally correct that children tend to ‘grow out of’ the problem as the function of their oil and sweat glands matures. Most children go into remission or substantially improve by the time they enter primary school. A minority still have atopic dermatitis in their teenage years and then into adulthood.²

Management

Education and reassurance

Explanation, reassurance and support are very important. It is common for patients and parents to look for a ‘cure’ (particularly food allergies) and to have misplaced fears of the safety of topical corticosteroids. Emphasise that it is a chronic, relapsing condition for which there is no cure,

address allergy concerns and provide education and reassurance of the safety of topical corticosteroids.⁵ Counselling is indicated where family stress and psychological factors are contributing to the problem.

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Avoid irritants

- Avoid soap and perfumed products.
- Older children should have short, tepid showers.
- Avoid rubbing and scratching—keep fingernails short, consider mittens or splints at night if severe.
- Avoid overheating, particularly at night.
- Avoid sudden changes of temperature, especially those that cause sweating.
- Wear light, soft, loose clothes, preferably made of cotton. Cotton clothing should be worn next to the skin.
- Avoid wool next to the skin.
- Avoid dusty conditions and sand, especially sandpits.
- Consider dust mite strategies:

dust mite covers (premium grade) for bedding

wash linen in hot water >55°C

consider replacing fabric on chairs and changing carpet.

Improve skin condition

- Frequent use of emollients is essential to improve skin barrier function and prevent flares.
- Use a bland bath oil in the bath and a soap substitute (e.g. aqueous cream, soap-free bars, soap-free wash).
- If itch is severe, make an oatmeal bath (put half a cup of oats in a sock or stocking and add this to the bathwater).
- Apply an emollient soon after bathing.
- Apply emollients at least twice/day (choose from):
 - emulsifying ointment

aqueous cream

QV intensive, Dermeze moisturising creams

paraffin creams—good in infants

moisturising lotions in summer

Medication²

Topical corticosteroids are the mainstay of therapy. The current approach is to apply them liberally until the inflamed skin is perfectly clear. Most dermatitis should clear within 7–14 days with use of the appropriate strength steroid but may require longer in severe cases. Emollients are the key to preventing flares, although periodic flares are common. When the condition flares, resume the topical corticosteroid promptly until the skin clears again.

Topical corticosteroids are available in ointments, creams and lotions. Ointments are usually the preferred option as they are more effective for dry skin and sting less. The appropriate strength corticosteroid depends on the site and the severity of the rash (see [CHAPTER 111](#)).

A general rule for recommended strength of topical corticosteroid for site:

- Face, axillae and groin—mild
- Trunk and limbs—moderately potent, potent if severe
- Hands and feet—potent

Topical corticosteroid therapy

For face, axillae and groin:

- 1% hydrocortisone ointment, daily until skin is clear
- if inadequate response, methylprednisolone aceponate 0.1% ointment or fatty ointment, daily for 7–14 days

For trunk and limbs:

- triamcinolone acetonide 0.02% ointment, daily until skin is clear
- if more severe or in the flexures, use:

methylprednisolone aceponate 0.1% ointment or fatty ointment, daily

or

mometasone furoate 0.1% ointment, daily

For hands, feet, lichenified wrists and ankles:

betamethasone dipropionate 0.05% ointment, daily until skin is clear

or

betamethasone valerate 0.1% ointment, daily

or

mometasone furoate 0.1% ointment, daily

A non-steroidal alternative is pimecrolimus 1% (Elidel) cream once to twice daily as a maintenance preparation for sensitive areas, e.g. face, eyelids, axillae, groin.

Oral antihistamines generally have no role in the treatment of atopic dermatitis; however, a sedating antihistamine at night is appropriate if itch prevents sleep.

Secondary infection

Atopic skin is particularly susceptible to secondary bacterial and viral infection. Skin and nasal staphylococcal carriage is also more common and may require eradication measures. Secondary infection often reduces the effectiveness of topical treatments and is a common cause of treatment failure.

Consider taking skin or nasal swabs if:

- crusting or pustules present
- dermatitis fails to respond to appropriate topical therapy.

Mupirocin 2% ointment is appropriate for localised bacterial infection, while a short course (5–10 days) of oral antibiotics (such as flucloxacillin or cephalexin) is appropriate for widespread infection. For recurrent infection, consider bleach baths (sodium hypochlorite 6% solution 60 mL per bath, twice weekly).

Eczema with secondary HSV infection (*eczema herpeticum*) requires prompt antiviral therapy and specialist referral.

Severe dermatitis

- Expert advice usually required
- Consider wet dressings
- Consider hospitalisation

- Phototherapy therapy as back-up
- Systemic immunosuppressants may be used for recalcitrant cases
- Oral corticosteroids are generally not recommended unless advised by a specialist

Other types of atopic dermatitis

⌚ Nummular (discoid) eczema

- Chronic, red, coin-shaped plaques
- Crusted, scaling and intensely itchy
- Secondary infection common due to excoriation
- Mainly on the legs, also buttocks and trunk
- Often symmetrical (no central clearing)
- Common in middle-aged patients
- May be related to stress
- Tends to persist for months, seek expert advice

Treatment as for moderate to severe atopic dermatitis, using a potent topical corticosteroid.

⌚ Pityriasis alba

- These are white, scaly, poorly demarcated patches on the face of children and adolescents
- Very common mild condition
- More common around the mouth and on cheeks
- Can occur on the neck and upper limbs, occasionally on trunk
- It is a subacute form of atopic dermatitis
- Full repigmentation occurs eventually

Treatment

- Reassurance
- Avoid skin irritants

- Simple emollients
- May prescribe hydrocortisone 1% ointment bd for 5–7 days (rarely necessary)

⌚ Lichen simplex chronicus

- Circumscribed thick plaques of lichenification
- Caused by repeated rubbing and scratching of previously normal skin
- Due to chronic itch of unknown cause
- At sites within reach of fingers (e.g. neck, forearms, thighs, vulva, heels, fingers)
- May arise from habit

Treatment

- Explanation
- Refrain from scratching
- Potent topical corticosteroid ointment (with or without occlusion) to break the itch–scratch cycle

⌚ Dyshidrotic dermatitis (pompholyx)

- Typically in patients aged 20–40 years
- Itching vesicles usually on fingers (see FIG. 113.3)



FIGURE 113.3 Pompholyx showing the typical vesicular dermatitis along the borders of the fingers. Look for associated inflammatory tinea of the feet which can precipitate it.

Photo courtesy Robin Marks

- May be larger vesicles on palms and soles
- Commonly affects sides of digits and palms
- Initial phase may be exudative as vesicles burst
- Lasts a few weeks
- Tends to recur
- Associated with atopic dermatitis, although cause is unclear
- Possibly related to stress
- Often triggered by high humidity

Treatment

- Consider infection if exudative and treat
- Potent corticosteroids as for atopic dermatitis

- Use a cream for exudative phase, then ointment as skin dries
- Consider occlusion (e.g. damp cotton gloves)

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Asteatotic dermatitis

This is the common, very itchy dermatitis that occurs in the elderly, especially in the winter, with a dry ‘crazy paving’ pattern, especially on the legs (see [CHAPTER 112](#)). If unresponsive to simple moisturisers, use a topical corticosteroid.

Note: ‘Asteatotic’ means without moisture.

Cracked (fissured) hands/fingers

This common cause of disability is usually due to dermatitis of the hands, irritant contact dermatitis or very dry skin. It is usually part of the atopic dermatitis problem and it is important to consider allergic contact dermatitis.

Management (hand dermatitis)

Hand protection:

- avoid domestic or occupational duties that involve contact with irritants and detergents
- wear protective work gloves; cotton-lined PVC gloves
- avoid perfumed toilet soaps—use a substitute (e.g. Cetaphil lotion)
- apply emollients frequently (e.g. emulsifying ointment, glycerine-rich creams), especially after finishing work duties
- potent topical corticosteroid as for atopic dermatitis
- oral corticosteroids may be required for severe flare
- expert referral for severe cases for patch testing

Cracked heels

Cracked painful heels are a common problem, especially in adult women. It is a manifestation of very dry skin.

Treatment

- Soak the feet for 15 minutes in warm water containing an oil such as Alpha Keri or Derma Oil.

- Pat dry, then apply an emollient foot cream (e.g. Nutraplus 10% urea).
- Emollients, keratolytics and humectants may be helpful such as urea and salicylic acid.
- Can use tissue glue (with care) to hold edges together.
- Wear enclosed footwear.

Contact dermatitis

Acute contact (exogenous) dermatitis can be either *irritant* or *allergic* and it is estimated that at least 70% of patients have an irritant cause. It is difficult to separate these types on clinical or histological grounds. The presence of irritant dermatitis increases the risk of developing a contact allergy.

Features:

- itchy, inflamed skin
- red and swollen
- papulovesicular
- may be dry and fissured

Irritant contact dermatitis

This is caused by primary irritants such as acids, alkalis, detergents, soaps, oils, solvents and chronic water exposure. A reaction may result from either a once-only exposure to a very irritant chemical or, more commonly, repeated exposure to weaker irritants. This is irritation, not allergy.

Allergic contact dermatitis

Caused by allergens that provoke an allergic reaction in some individuals only—most people can handle the chemicals without undue effect. It is immunologically mediated. This allergic group also includes photo-contact allergens. Approximately 4.5% of the population is allergic to nickel, which is found in jewellery, studs on jeans, keys and coins (see FIG. 113.4). Contact dermatitis is due to delayed hypersensitivity, sometimes with a long time of days to years. It is common in industrial or occupational situations, where it usually affects the hands and forearms.



FIGURE 113.4 Contact dermatitis due to nickel-containing underwire in brassiere in 17-year-old girl with a history of eczema

Common allergens:

- ingredients/fragrances in cosmetics (e.g. perfumes, preservatives)
- topical antibiotics (e.g. neomycin)
- topical anaesthetics (e.g. benzocaine)
- topical antihistamines
- plants: *Rhus*, *Grevillea*, *Asteraceae*
- metal salts (e.g. nickel sulfate, chromate)
- dyes (especially clothing dyes)
- perfumes, cosmetics
- hairdressing chemicals
- rubber/latex
- epoxy resins and glues/acrylates
- glutaraldehyde (e.g. sterilising agents)
- toluene sulfonamide compound resins: nail polish
- coral

Note: The skin of mangoes cross-reacts with *Rhus* and *Grevillea*.

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Clinical features⁴

- Site and shape suggest contact
- Dermatitis ranges from faint erythema to ‘water melon’ face oedema
- Worse in peri-orbital region, genitalia and hairy skin; least in glabrous skin (e.g. palms and soles)
- Think of *Rhus* or *Grevillea* allergy if linear blisters on forearms and/or puffy eyes
- Improvement when off work or on holiday

Note: Can be delayed onset.

Diagnosis

- Take a thorough history; consider occupation, family history, vacation or travel history and clothes
- Review all topical applications (e.g. medicines, cosmetics) for allergic or irritant potential
- Usage test—apply products used by the patient to the cubital fossa and note development of rash
- Refer to a dermatologist for patch testing

Management

- Determine cause with vigour and remove it
- Topical corticosteroid treatment as for atopic dermatitis
- Oral prednisolone for severe cases² (start with 25–50 mg daily for adults for 5–7 days then gradually reduce over 2 weeks)

Seborrhoeic dermatitis

Seborrhoeic dermatitis is a very common skin inflammation that usually affects areas abundant in sebaceous glands or intertriginous areas. It is therefore common in hair-bearing areas of the body, especially the scalp and eyebrows. It can also affect the scalp, face, neck, axillae and groin, eyelids (blepharitis), external auditory meatus and nasolabial folds. The presternal area is often involved (see FIG. 113.5).



FIGURE 113.5 Seborrhoeic dermatitis in an adult showing a typical position on the chest

There are two distinct clinical forms: seborrhoeic dermatitis of infancy, and the adult form.

Studies have indicated that it may be caused by a reaction to the yeast *Malassezia* sp. It may be associated with HIV infection and Parkinson disease.

A feature of seborrhoeic dermatitis is that, unlike atopic dermatitis, itching is absent or minimal. Seborrhoeic scale is greasy and yellowish, unlike the silvery scale of psoriasis.

Treatment principles for seborrhoeic dermatitis

- Keratolytics such as salicylic acid may be used to lift the scale.
- Anti-yeast treatments reduce the skin's load of *Malassezia* sp., e.g. ketoconazole, miconazole and ciclopirox.
- Topical corticosteroids target inflammation and pruritus and often used in combination.

⌚ Seborrhoeic dermatitis of infancy

Infantile seborrhoeic dermatitis is different from adult seborrhoeic dermatitis. In infancy, it is often known as 'cradle cap' if it affects the scalp, or nappy rash/diaper dermatitis if it involves the napkin area.

It can be difficult to differentiate the rash from that of atopic dermatitis but seborrhoeic dermatitis tends to appear very early (before atopic dermatitis), from 3–12 weeks, when androgen activity is most prevalent. Both atopic dermatitis and seborrhoeic dermatitis can present with red, scaly patches.

The different features are summarised in [TABLE 113.2](#) and the distribution is presented in [FIGURE 113.6](#).

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Table 113.2 Differential diagnosis of seborrhoeic dermatitis and atopic dermatitis in infancy

	Seborrhoeic dermatitis	Atopic dermatitis (eczema)
Age of onset	Mainly within first 3 months	Usually after 2 months
Itchiness	Nil or mild	Usually severe
Distribution	Scalp, cheeks, folds of neck, axillae, folds of elbows and knees	Starts on face Elbow and knee flexures
Typical features	Cradle cap Red and yellow greasy scale	Vesicular and weeping Becomes dry and cracked
Napkin rash	Common Prone to infection with <i>Candida</i>	Less common

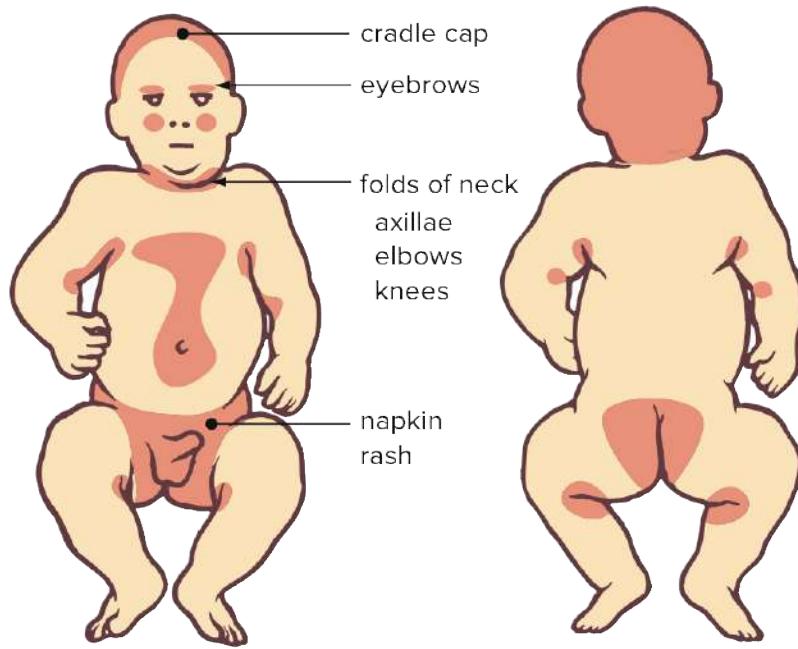


FIGURE 113.6 Seborrhoeic dermatitis: typical distribution in infants

In cradle cap, a flaky, scurf-like dandruff appears first, and then a yellowish, greasy, scaly crust forms. This scurf is usually associated with reddening of the skin. It may resolve spontaneously within a few months (see [CHAPTER 84](#)).

The dermatitis can become infected, especially in the napkin area, and this may be difficult to treat. If untreated, it often spreads to many areas of the body. It is said that cradle cap and nappy rash ‘may meet in the middle’.

Treatment

Simple basic methods are:

- keep areas dry and clean
- keep skin exposed to air as much as possible
- use soap substitute (e.g. Cetaphil lotion)
- rub scales of cradle cap gently with baby oil or white soft paraffin then wash away loose scales

Medication⁶

Scalp

If lesions still persist, use:

- Egozite cradle cap lotion (6% salicylic acid)
- 2% salicylic + 2% sulfur + 2% liquor picis carbonis in aqueous cream:
 - apply overnight to scalp, wash off next day with soap substitute
 - use daily or second daily until it clears

For persistent scalp erythema:

desonide 0.05% lotion, daily after bath

Face, flexures and trunk

desonide 0.05% lotion, bd for up to 7 days

or

hydrocortisone 1% ointment, bd for up to 7 days

Napkin area

- Mix equal parts 1% hydrocortisone with nystatin or miconazole + zinc oxide

Prognosis

Most children are clear by 18 months (rare after 2 years).

§ Adult seborrhoeic dermatitis

Clinical features

- Any age from teenage onwards
- The head is a common area: scalp and ears, face, eyebrows, eyelids (blepharitis), nasolabial folds (see FIG. 113.7)

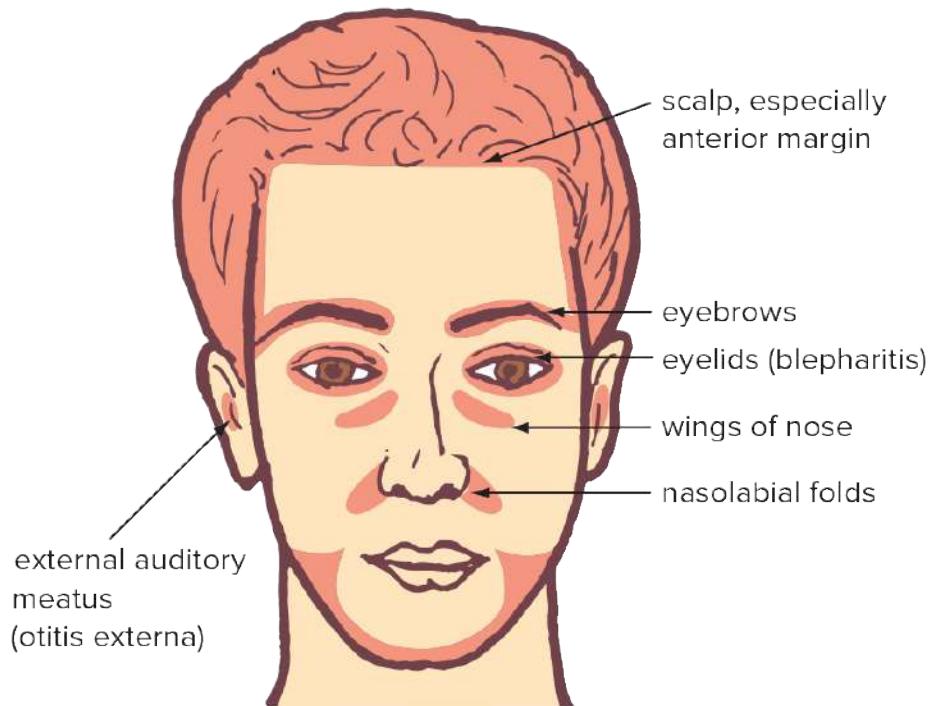


FIGURE 113.7 Seborrheic dermatitis: facial distribution in adults

- Other areas: centre of chest, centre of back, scapular area, intertriginous areas, especially perianal (see FIG. 113.8)

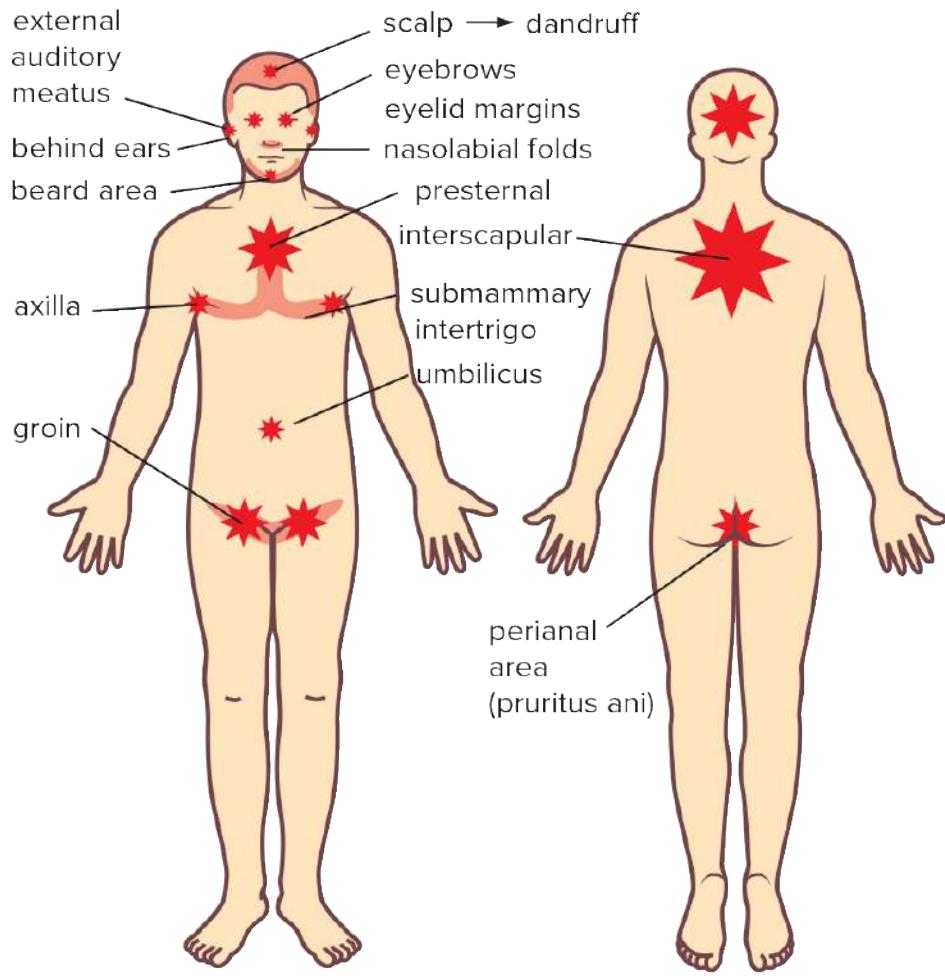


FIGURE 113.8 Seborrhoeic dermatitis: possible distribution in adults

- Pale pink, ill-defined erythematous rash characterised by loose, flaky scale
- Scale may be yellow and greasy
- Dandruff a feature of scalp area
- Worse with stress and fatigue
- It is a chronic, recurring condition

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Treatment²

Scalp

First-line treatment is an anti-yeast shampoo applied often (twice per week to daily). Shampoos often require rotation as response diminishes.

Anti-yeast shampoo options:

- ketoconazole
- ciclopirox olamine
- zinc pyrithione
- selenium sulfide

If inadequate, add a topical corticosteroid lotion for 7 nights. Options include:

- betamethasone dipropionate 0.05%
- methylprednisolone aceponate 0.1%
- mometasone furoate 0.1%

If inadequate response and especially if thick scale, add coal tar (LPC) at night, once or twice weekly and wash off the next morning with anti-yeast shampoo. Recommended topical tar preparations:

- coal tar prepared 1% emulsion or gel
- LPC 3–6% + salicylic acid 3% in aqueous cream

If all above therapies fail, use corticosteroid shampoo, clobetasol propionate 0.05%, twice weekly (use anti-yeast shampoo on the other 5 days).

Face and body

- Wash regularly using bland soap
- Combination antifungal cream and topical corticosteroid is first line:

hydrocortisone 1% + clotrimazole 1% once or twice daily

or

hydrocortisone 1% + miconazole 2% cream, once or twice daily for up to 2 weeks

- If inadequate, use separate topical corticosteroids and antifungal creams:

desonide 0.05% lotion daily for up to 2 weeks

or

methylprednisolone aceponate 0.1% cream daily for up to 2 weeks

in combination with

ketoconazole 2% or bifonazole 2% creams daily for up to 2 weeks

or

clotrimazole 1%, econazole 1% or miconazole 2% creams twice daily for up to 2 weeks

If treatment fails, a tar cream can be used such as LPC 1–2% (for face, flexures and groin) and LPC 3–6% (for trunk and limbs) in aqueous or sorbolene cream, once daily for up to 2 weeks.

Note: Oral antifungals are not recommended at all.

Psoriasis

Psoriasis (see FIG. 113.9) is a chronic, immune-mediated skin disorder of unknown aetiology which affects 2–4% of the population. It appears most often between the ages of 10 and 30 years, although its onset can occur any time from infancy to old age. It has a familial predisposition although its mode of inheritance is debatable. If one parent is affected there is a 25% chance of developing it; this rises to 65% if both parents are affected.³



FIGURE 113.9 Psoriasis showing the typically raised pink plaques surmounted with a silvery scale

Psoriasis is now regarded as a disorder involving activation of helper T cells in the skin. Cytokines are then released and cause skin cells to multiply faster, leading to thickening of the skin and overscaling. The new 'biological agents' intervene in this process.

Capillary dilatation explains the redness.

Be aware of association with cardiovascular disease, particularly heart disease, depressive illness, diabetes, arthritis and inflammatory bowel disease.⁷

Factors that may worsen or precipitate psoriasis

- Infection, especially group A *Streptococcus*
- Trauma or other physical stress
- Emotional stress
- Sunburn
- Puberty/menopause
- Drugs:

antimalarials (e.g. chloroquine)

beta blockers

lithium

NSAIDs

oral contraceptives

Types of psoriasis	Differential diagnosis
Infantile	Seborrhoeic dermatitis, atopic dermatitis
Plaque (commonest)	Seborrhoeic dermatitis, discoid eczema, solar keratoses, Bowen disease
Guttate	Pityriasis rosea, secondary syphilis, drug eruption
Flexural	Tinea, candida intertrigo, seborrhoeic dermatitis
Scalp (sebopsoriasis)	Seborrhoeic dermatitis, tinea capitis
Nail	Tinea, idiopathic onycholysis
Pustular (palmoplantar)	Tinea, infected eczema
Exfoliative	Severe seborrhoeic dermatitis

The typical patient

- Older teenager or young adult
- Possible family history
- Onset may follow stress, illness or injury

- Rash may appear on areas of minor trauma—the Koebner phenomenon
- Rash improves on exposure to sun but worse with sunburn
- Worse in winter
- Itching not a feature but is present sometimes

Arthropathy

About 30% can develop a painful arthropathy (fingers, toes or a large joint) or a spondyloarthropathy (sacroiliitis).⁸

The rash

The appearance depends on the site affected. The commonest form is plaque psoriasis, which begins with red lesions that enlarge and develop silvery scaling. The commonest sites are the extensor surfaces of the elbows and knees; then the scalp, sacral areas, genitals and nails are affected (see FIG. 113.10).

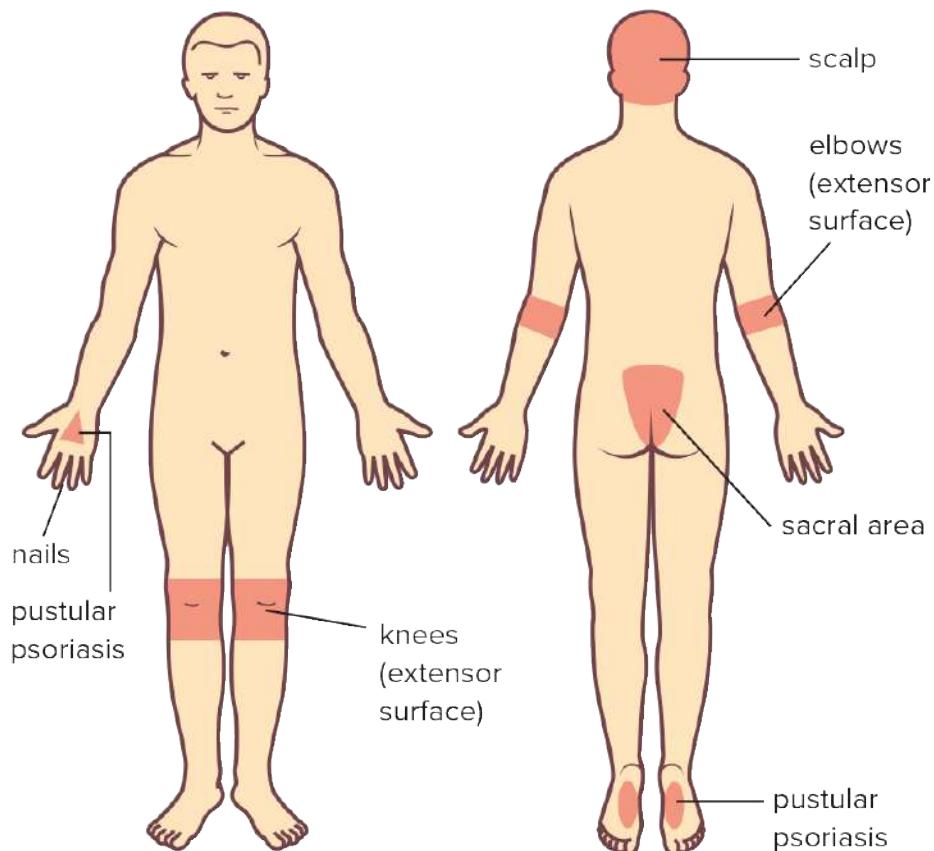


FIGURE 113.10 Psoriasis: typical skin distribution

Diagnosis

Psoriasis is a clinical diagnosis but biopsy may be needed for confirmation. No laboratory test (including blood testing) is available.

Principles of management

While realising there is no cure for psoriasis, the aim of treatment is to relieve discomfort, slow down the rapid skin cell division and work in consultation with a specialist to achieve these aims.⁸

- Provide education, reassurance and support.
- Promote general measures such as rest, and holidays, preferably in the sun.
- Promote healthy lifestyle, including balanced diet, exercise, reduced alcohol and smoking cessation.
- Advise prevention, including avoidance of skin damage and stress if possible.
- Avoid irritants and use a soap substitute.
- Recommend regular use of emollients.
- Tailor treatment (including referral) according to the degree of severity and extent of the disease.
- Shared care with a consultant is advisable.

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Topical treatment options⁹

Topical agents useful for treatment of psoriasis in primary care:

- Tar preparations
 - anti-inflammatory, anti-pruritic
 - often used in combination with keratolytics (e.g. salicylic acid) to soften and lift scale
 - stinging can be avoided by using with topical corticosteroid or when symptoms mild
 - colour and odour limit concordance, apply at night
 - common preparation is liquor picis carbonis (LPC)
- Topical corticosteroids
 - most common treatment for psoriasis

anti-inflammatory, reduces rate of skin turnover

useful for flares; add tar or calcipotriol if ineffective or step down to tar alone once controlled

- Calcipotriol

vitamin D derivative, regulates proliferation and differentiation of keratinocytes

takes up to 6 weeks to take effect

stinging is common, reduced when used in combination with topical corticosteroid

Therapy can be monotherapy or combined therapy, e.g. tar and corticosteroids or corticosteroids and calcipotriol. Rotational therapy is often required as patients become less sensitive to one therapy over time, especially topical corticosteroids. Adding another treatment can extend disease control.

Recommended topical regimens⁹

Chronic stable plaque psoriasis

For trunk and limb psoriasis, apply:

coal tar prepared 1% emulsion or gel, once or twice daily, for 1 month

or

LPC 6% + salicylic acid 3% cream or ointment, twice daily for 1 month

if insufficient or flare, add

moderately potent to potent topical corticosteroid ointment, daily until skin is clear (usually 2 to 6 weeks)

or if inadequate response

calcipotriol + betamethasone dipropionate 50 + 500 mcg/g ointment, daily until skin is clear (usually about 6 weeks)

Once psoriasis controlled, reduce the potency of steroid and withdraw if possible. Continue tar as maintenance therapy.

Palmoplantar psoriasis

Treat as for trunk or limb psoriasis, but higher dose of salicylic acid is required if hyperkeratotic, i.e. LPC 6% + salicylic acid 6%. Also consider earlier use of calcipotriol, given common resistance to topical therapy.

Scalp psoriasis

potent corticosteroid lotion or shampoo (as for [seborrhoeic dermatitis](#)), daily until skin is clear (usually 2 to 6 weeks)

then

coal-tar shampoo (over the counter) for maintenance

or if thickened scale, add

LPC 6% + salicylic acid 3% in aqueous cream, twice daily

if inadequate response

calcipotriol + betamethasone dipropionate 50 + 500 mcg/g gel, once daily until skin is clear (expect some response within 2 weeks)

Facial psoriasis

methylprednisolone aceponate 0.1% ointment or fatty ointment, daily for several weeks (usually 2 to 6 weeks) or 1% hydrocortisone for children

once controlled

LPC 2% + salicylic acid 2% in aqueous cream at night

Flexural (inverse) and genital psoriasis

Note that fissuring (e.g. inframammary, natal cleft) is a feature. There is little or no scale.

methylprednisolone aceponate 0.1% ointment or fatty ointment, once daily for several weeks (up to 2 weeks in children)

once controlled

LPC 2% in emulsifying ointment at night

Nail psoriasis

If patient motivated:

calcipotriol + betamethasone dipropionate 50 + 500 mcg/g ointment, in proximal nail fold and under nail, at night for up to 3 months

If onycholysis or subungual hyperkeratosis:

betamethasone dipropionate 0.05% lotion topically, twice daily under nail for up to 3 months

Expert treatments

- Dithranol topical therapy is rarely used, can stain clothes and burn unaffected skin
- Narrowband ultraviolet B phototherapy (UV-B)
 - treatment 2–3 times a week for a few months
 - often used in combination with other therapies
- Methotrexate
 - dramatic results in severe cases
- Cyclosporin
 - symptoms quickly return when treatment stops and not recommended long term
- Acitretin is a vitamin A derivative and effective in severe intractable psoriasis (never use in females of child-bearing age)
- Biological agents
 - directed at the T cell dysfunction (i.e. immune response modifiers), e.g. infliximab, ustekinumab, adalimumab and secukinumab

Nappy rash⁶

Nappy rash (or diaper dermatitis) is an inflammatory contact dermatitis occurring in the napkin area and can be a common presentation of mild or moderate underlying skin disease. It is found in children up to 2 years old and has a peak incidence from 9–12 months.¹⁰

Most children will develop nappy rash at some stage of infancy, with an estimated 50% having it to a significant extent.

Causes of nappy rash

The commonest type is *irritant dermatitis* due to dampness from urine and faeces, but consider also:

- *Candida albicans* (invariably present although often not obvious)
- seborrhoeic dermatitis
- atopic dermatitis

- psoriasis

Irritant dermatitis

This is a type of contact dermatitis with the erythema and scaling conforming to the napkin area. The flexures are usually spared. It is related to the activity of faecal proteases and lipases and probably not to the activity of ammonia (from urea) as previously promoted. The problem can vary from mild erythema to a severe blistering eruption with ulceration. Ultrabsorbent disposable nappies appear to be better than cloth nappies.¹¹ Diarrhoea is a causative factor of irritant dermatitis. If the eruption extends further than the points of contact with the nappy, an underlying skin disease such as seborrhoeic or atopic dermatitis must be suspected. Psoriasis always involves the skinfolds.

Other irritants include:

- rough-textured nappies
- chemicals in some baby wipes
- plastic pants (aggravates wetness)
- excessive washing of the skin with soap
- too much powder over the nappy area (avoid talcum powders)

Seborrhoeic dermatitis

This affects mainly the flexures of the natal cleft and groin. It is important to look for evidence of seborrhoeic dermatitis elsewhere, such as cradle cap and lesions on the face and axillae.

Atopic dermatitis

Atopic dermatitis can involve the napkin area. Pruritus is a feature and the child may be observed scratching the area. There may be evidence of atopic dermatitis elsewhere, such as on the face.

Candidiasis (monilia nappy rash)

Superinfection of intertrigo or napkin dermatitis will result in a diffuse, red, raw, shiny rash that will involve the flexures and extend beyond the napkin area as ‘satellite lesions’. Candida tends to invade the skin folds and the foreskin of male babies.

Uncommon causes

Psoriatic nappy rash

This presents as a non-scaling eruption, primarily on the napkin area, but can extend to the

flexures, trunk and limbs. The edge of the rash is sharply demarcated. The typical psoriatic scale is absent. It tends to occur in the first weeks of life. There is usually a family history.

Infections

Bacterial infections to consider include staphylococcal folliculitis, impetigo and perianal streptococcal dermatitis. Culture of the lesion will reveal the cause.

Impetigo

If there is *Staphylococcus* superinfection, bullae and pus-filled blisters will be present.

Langerhans cell histiocytosis (histiocytosis X)

There is a similar rash to seborrhoeic dermatitis but the lesions are purpuric. In this syndrome the child may be ill and lymphadenopathy and hepatosplenomegaly may be found.

Zinc deficiency

May be more common than realised.

Management

Basic care (instructions to parents/carers):

1. Keep the area dry. Change wet or soiled nappies frequently and as soon as you notice them.
Use highly absorbent disposable nappies.
2. After removing nappy, gently remove any urine or moisture with a soap substitute or warm water.
3. Wash gently with warm water, pat dry (do not rub) and then apply a barrier preparation to help protect the area. Vaseline or zinc cream applied lightly will do.
4. Expose the bare skin to fresh air wherever possible.
5. Use a soap substitute and bath oil for bathing.
6. Avoid powder and plastic pants.

Medical treatment

Some principles follow:

- The cornerstone of treatment is prevention.
- Barrier preparations should be used to protect the skin, e.g. zinc oxide (best) or white soft

paraffin or a mixture of zinc oxide and castor oil or Vaseline.

- A mild topical corticosteroid is the treatment of choice.
- It is useful to add an antifungal agent.
- If infection is suspected, confirm by swab or skin scraping.
- Consider a combined steroid and antifungal agent, e.g. Hydrozole.
- When nappy rash is severe, apply a stronger topical steroid for up to 7 days, e.g. methylprednisolone aceponate 0.1% ointment or fatty ointment, once daily.

Treatment:

Atopic dermatitis	1% hydrocortisone
Seborrhoeic dermatitis	1% hydrocortisone + ketoconazole cream
Widespread (<i>Candida</i> present)	1% hydrocortisone cream mixed in equal quantities with nystatin (apply bd) or miconazole + zinc oxide (apply after nappy changes)
Psoriatic dermatitis	tar preparation, or 1% hydrocortisone
Impetigo	topical mupirocin; oral antibiotics if severe

Facial rashes

Common facial skin disorders include acne, rosacea, peri-oral dermatitis and seborrhoeic dermatitis. These conditions must be distinguished from lupus erythematosus (discoid LE is more common).

Acne

Acne is inflammation of the pilosebaceous (oil) glands of the skin (see FIG. 113.11). At first there is excessive sebum production due to the action of androgen. These glands become blocked (blackheads and whiteheads) due to increased keratinisation of the sebaceous duct. A secondary bacterial infection due to *Propionibacterium acnes* in the sebum produces lipases with the resultant free fatty acids, thus provoking inflammation characterised by red papules, pustules, nodules and cysts.

Acne usually peaks early in puberty and resolves in males during the early twenties.¹² Females

are more likely to suffer ongoing acne. Consider PCOS as a causative condition.



FIGURE 113.11 Facial acne showing typical nodulocystic acne, a distressing problem which has been improved greatly by the development of isotretinoin

History

In women, enquire about hirsutism and menstrual irregularity. Consider the patient's occupation (e.g. chef, exposure to grease and oil). Enquire about use of skin preparations—therapeutic or cosmetic, exposure to oils and drug intake.

Drugs that aggravate acne:¹³

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- corticosteroids
- chloral hydrate
- iodides or bromides

- lithium
- anti-epileptics (e.g. phenytoin)
- quinine
- hormonal contraceptives (consider switching to an anti-androgenic combined oral contraceptive)

Management

Support and counselling

Adolescents hate acne; they find it embarrassing and require the sympathetic care and support not only of their doctor but also of their family. It should not be dismissed as a minor problem.

Education

People with acne should understand its pathogenesis and be given leaflets with appropriate explanations. Myths must be dispelled.

- It is not a dietary or infectious disorder.
- It is not caused by oily hair or hair touching the forehead.
- Blackheads are not dirt, and will not dissolve in hot, soapy water.

Reassure the patient that acne usually becomes less of a problem after the age of 25 years, although 15% of women and 5% of men continue to have acne as adults.

General factors

- Diet is not considered to be a causative factor but a healthy diet is encouraged.
- Special soaps and overscrubbing are unhelpful. Use a normal soap and wash gently.
- Avoid oily or creamy cosmetics and all moisturisers. Use cosmetics sparingly.
- Avoid picking and squeezing blackheads.
- Exercise, hair washing and shampoos are not of proven value.
- Ultraviolet light such as sunlight may help improve acne but avoid overexposure to the sun.

Principles of treatment¹²

1. Comedolysis: unblock the pores (follicular ducts) with keratolytics such as salicylic acid (5–10%) or benzoyl peroxide (2.5%, 5% or 10%) which also has antibacterial properties. Topical

retinoids are the most effective comedolytics, e.g. tretinoin 0.025% cream or adapalene 0.1% cream or gel.

1. Anti-inflammatory: tetracyclines or erythromycin—or with a topical antibiotic such as clindamycin.
2. Decrease sebaceous gland activity with oestrogens, spironolactone, cyproterone acetate or isotretinoin.

Note: Oral isotretinoin is teratogenic.

Avoid concomitant use of oral and topical antibiotics.

Recommended treatment regimens

Topical regimens

Suitable for mild to moderate acne.

- Basic starting regimen is a topical retinoid in combination with benzoyl peroxide:
 - use tretinoin 0.025% cream or adapalene 0.1% cream or gel, apply each night
 - if slow response after 6 weeks, add benzoyl peroxide 2.5% or 5% gel or cream once daily (in the morning)
 - maintain for 3 months and review.
- Alternative or add-on regimens, if persistent: clindamycin HCl 600 mg in 60 mL of 70% isopropyl alcohol (e.g. ClindaTech). Apply with fingertips twice daily.

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Oral antibiotics

Use for inflammatory acne: (moderate to severe papulopustular) ± trunk involvement

- doxycycline or minocycline, 50 to 100 mg per day
- erythromycin 250 to 500 mg twice daily is an alternative, especially for pregnant women
- give a minimum 12-week trial; 6 months is standard
- avoid using antibiotics alone

Other therapies

Severe cystic or recalcitrant acne (specialist care):

- spironolactone
- isotretinoin (Roaccutane): outstanding agent

Females not responding to first-line treatment:

- combined oral contraceptive pill with anti-androgenic effect (e.g. ethinyloestradiol with cyproterone acetate, drospirenone or gestodene)
- oral spironolactone may be used in combination with an oral contraceptive pill as an anti-androgen (single use should be avoided due to risk of menstrual irregularity and contraindication in pregnancy)

Note: Response to any acne treatment occurs in about 8 weeks or longer.

Common mistakes with acne

- Not treating comedones with a comedolytic
- Monotherapy (e.g. antibiotics only)
- Not using recommended combinations
- Not using isotretinoin for cystic acne

Rosacea

Rosacea is a common persistent eruption of unknown aetiology. It is typically chronic and persistent with a fluctuant course.

Clinical features³

- Mainly 30–50 years
- Usually females of Celtic origin: ‘the curse of the Celts’
- On forehead, cheeks, nose and chin (see FIGS 113.12 and 113.13)



FIGURE 113.12 Rosacea: typical appearance with erythema, papules and pustules

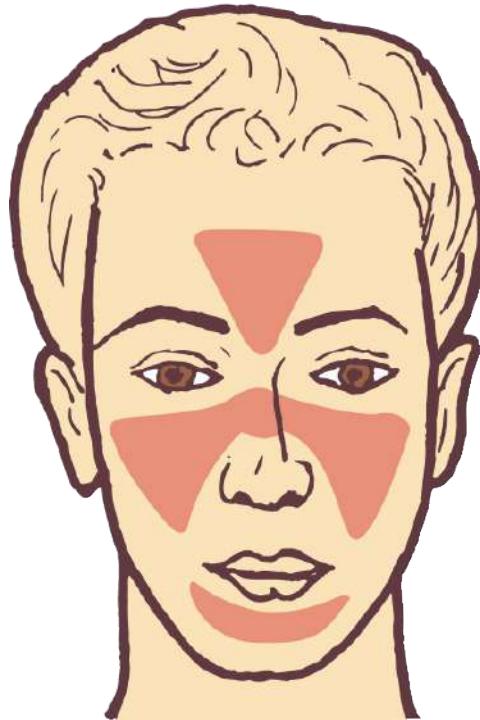


FIGURE 113.13 Rosacea: typical facial distribution

- ‘Flushing and blushing’ (often precedes the rash)
- Fluctuates from day to day

- Peri-orbital and peri-oral areas spared
- Vascular changes—erythema and telangiectasia
- Inflammation—papules and pustules (sterile)

Complications

- Blepharitis
- Conjunctivitis, rarely keratitis and corneal ulcer
- Associated rhinophyma in some cases

Management¹⁴

General measures

- Avoid factors that cause facial flushings (e.g. excessive sun exposure, heat, wind, alcohol, excessive exercise, hot baths, spicy foods, hot drinks—tea and coffee)
- Sun protection
- Use a gentle soap-free cleanser
- Apply green-tinted foundation to mask redness
- Avoid topical corticosteroids which can cause rebound vascular changes on cessation

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Topical agents

For mild erythema and inflammatory lesions:

metronidazole 0.75% gel or cream, bd

or

azelaic acid 15% gel, once daily

or

ivermectin 1% cream, once daily

Apply for 6 to 12 weeks for maximal response. Long-term treatment is usually required to prolong remission. If topical therapy is unsuccessful, add or replace with oral therapy.

For erythematotelangiectatic skin, applying a topical vasoconstrictor improves its cosmetic

appearance, but the effect is temporary (about 12 hours):

brimonidine tartrate 5 mg/g gel, once daily in the morning

Systemic antibiotics

Antibiotics are used for their anti-inflammatory effect:

- Doxycycline 50 to 100 mg daily for a total of 8–10 weeks.
- If inadequate after 4 weeks, consider minocycline 50 to 100 mg daily for up to 8 weeks. Repeat for recurrences.
- Erythromycin 250 to 500 mg twice daily is an alternative for pregnant women or if doxycycline or minocycline are not tolerated.
- Low dose doxycycline or minocycline, 50 mg daily, is appropriate for longer use if symptoms recur within a month.

Laser therapy

Telangiectasia, erythema and rhinophymas respond to laser therapy.

Rhinophyma

This is due to hypertrophy of the nasal sebaceous glands and is associated with older men with rosacea. Treatment options include laser ablation, shave excision or isotretinoin (see CHAPTER 48).

¶ Peri-oral dermatitis

Clinical features

- Papular and pustular, acne-like dermatitis of lower face
- Usually young women 20–50 years
- May be seen in children and adolescents
- ‘Muzzle area’ distribution around mouth and on chin, sparing adjacent peri-oral area (see FIGS 113.14 and 113.15)
- Peri-orbital skin involvement is common in men
- Rash may be unilateral

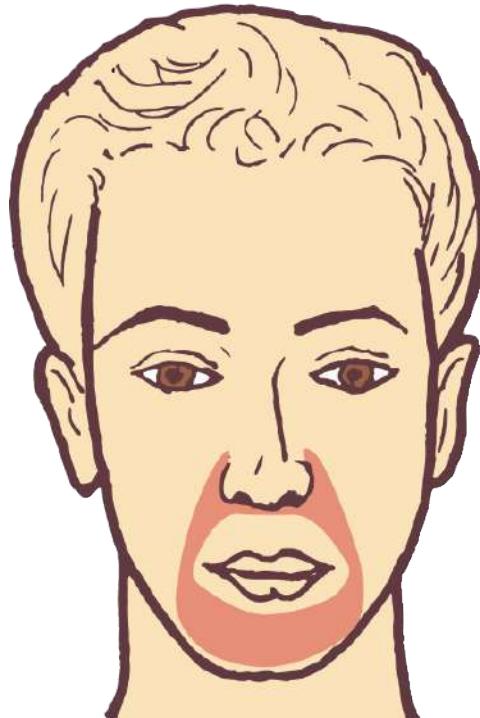


FIGURE 113.14 Peri-oral dermatitis: typical distribution



FIGURE 113.15 Peri-oral dermatitis: this eruption (which has been linked with the use of fluorinated steroid creams on the face) frequently begins in the nasolabial fold. Papules and pustules occur on a background of erythema and scaling.

- Frequently begins at the nasolabial folds
- Multiple small red macules and papules

- On a background of erythema and scaling
- Burning and irritation
- May be associated with seborrhoeic dermatitis on scalp and head
- May be related to pregnancy and oral contraception
- Related to the use of creamy cosmetic products
- May be related to repeated topical corticosteroid use (may be a rebound on ceasing it)
- Peri-oral dermatitis belongs to the rosacea spectrum of disease

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Treatment

- Discontinue any topical corticosteroid therapy (tends to flare initially) and all ‘creamy’ preparations including cleansers, moisturisers and make-up.
- Simple treatments include cleansing with a gentle soap substitute, cold compress for irritation and use of a light emollient.
- Treatment is with oral antibiotics for their anti-inflammatory properties, as for rosacea.
- Topical treatments are not as effective and may irritate: metronidazole 0.75% gel or clindamycin 1% lotion twice daily.

Tinea

Tinea (ringworm infections) is caused mainly by three major classes of dermatophytic organisms that have the ability to invade and proliferate in keratin of the skin, nails and hair.

It is most useful to perform skin scrapings and microscopy to look for encroaching septate hyphae. Confirm the diagnosis by fungal culture. Tinea cruris is presented in [CHAPTER 112](#).

Tinea pedis (athlete's foot)

Tinea pedis is usually caused by *Trichophyton rubrum* and is the commonest type of fungal infection in humans. Candida intertrigo and interdigital maceration (alone without secondary tinea) in particular, and also erythrasma, eczema and psoriasis, are important differential diagnoses.

Symptoms

The commonest symptoms are itchiness and foot odour. Sweat and water make the top layer of

skin white and soggy. There is scaling, maceration and fissuring of the skin between the fourth and fifth toes and also third and fourth toes.

Advice to patients

- Keep your feet as clean and dry as possible.
- Carefully dry your feet after bathing and showering.
- After drying your feet, use an antifungal powder, especially between the toes.
- Remove flaky skin from beneath the toes each day with dry tissue paper or gauze.
- Wear light socks made of natural absorbent fibres, such as cotton and wool, to allow better circulation of air and to reduce sweating. Avoid synthetic socks.
- Change your shoes and socks daily. Spray shoes with an antifungal agent.
- If possible, wear open sandals or shoes with porous soles and uppers.
- Go barefoot whenever possible.
- Use thongs in public showers such as at swimming pools (rather than bare feet).

Treatment

Several topical antifungals can be used. Preferable to use terbinafine 1% once or twice daily for 1–2 weeks and review. If severe and spreading, prescribe oral griseofulvin (see *Tinea corporis*) or terbinafine for up to 6 weeks after confirming the diagnosis by fungal culture.

Castellani paint may be helpful for macerated areas.

Tinea corporis¹⁵

Tinea corporis (ringworm infection of the body) is usually caused by *Trichophyton rubrum* (60%) or *Microsporum canis*.¹⁶ Strongly related to exposure to cats and dogs, while the guinea pig is a potent source of facial tinea (can present as pustular folliculitis).

Clinical features

- Spreading circular erythematous lesions (see FIG. 113.16)
- Slight scaling or vesicles at the advancing edge
- Central areas usually normal
- Mild itch

- May involve hair, feet and nails



FIGURE 113.16 Multiple ringworm (tinea corporis): this 12-year-old boy presented with a two-week history of an increasing number of pruritic scaling erythematous lesions on his face, neck and upper chest. Fungal scrapings confirmed the cause as *Microsporum canis*.

Photo courtesy Robin Marks

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Treatment

- First choice is terbinafine 1% cream or gel, once or twice daily for 1–2 weeks
- Other options include clotrimazole 1%, miconazole 2% cream, bifonazole 1% cream, ketoconazole 2% cream, applied bd for 2–4 weeks
- Oral terbinafine 250 mg or griseofulvin 500 mg daily, for up to 6 weeks if no response or widespread

⌚ Tinea capitis¹⁷

In Australia, tinea capitis is usually due to *M. canis* acquired from cats and dogs.

Clinical features

- Usually in children (rare after puberty)
- Patches of partial alopecia
- Scaly patches

- Small broken-off hair shafts
- Hairs fluoresce yellow-green with Wood's light (not invariably, e.g. with *Trichophyton tonsurans* infection)

Treatment

terbinafine (o): adults 250 mg daily for 4 weeks; children 62.5–125 mg

or

griseofulvin (o): adults 500 mg daily; children 10 mg/kg/day (max. 500 mg) 6–12 week course

Take hair plucking and scale for culture. Treat *Trichophyton tonsurans* with terbinafine and *Microsporum canis* with griseofulvin.

⌚ Kerion

Kerion of the scalp and beard area may present like an abscess—tender and fluctuant. Usually occurs on the scalp, face or limbs. A fungal cause is possible if the hairs are plucked out easily and without pain (if painful and stuck, bacterial infection is likely).

⌚ Tinea incognito

This is the term used for unrecognised tinea infection due to modification with corticosteroid treatment. The lesions are enlarging and persistent, especially on the groin, hands and face.

The sequence is initial symptomatic relief of itching, stopping the ointment or cream and then relapse.

⌚ Tinea cruris

Refer to [CHAPTER 112](#).

⌚ Tinea unguium (toenails and fingernails)

Refer to [CHAPTER 119](#).

⌚ Pityriasis versicolor (tinea versicolor)¹⁶

Pityriasis versicolor is a superficial yeast infection of the skin (usually on the trunk) caused by *Malassezia* sp.). The old name, tinea versicolor, is inappropriate because the problem is not a dermatophyte infection. It occurs worldwide but is more common in tropical and subtropical climates.

There are two distinct presentations:

- 1. reddish brown, slightly scaly patches on upper trunk
- 2. hypopigmented area that will not tan, especially in suntanned skin

Note: The term ‘versicolor’ means variable colours.

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Clinical features

- Mainly young and middle-aged adults
- Brown on pale skin or white on tanned skin (see FIG. 113.17)



FIGURE 113.17 Pityriasis versicolor showing the hypopigmented scaly patches on a suntanned skin mainly affecting the trunk

- Trunk distribution (see FIG. 113.18)

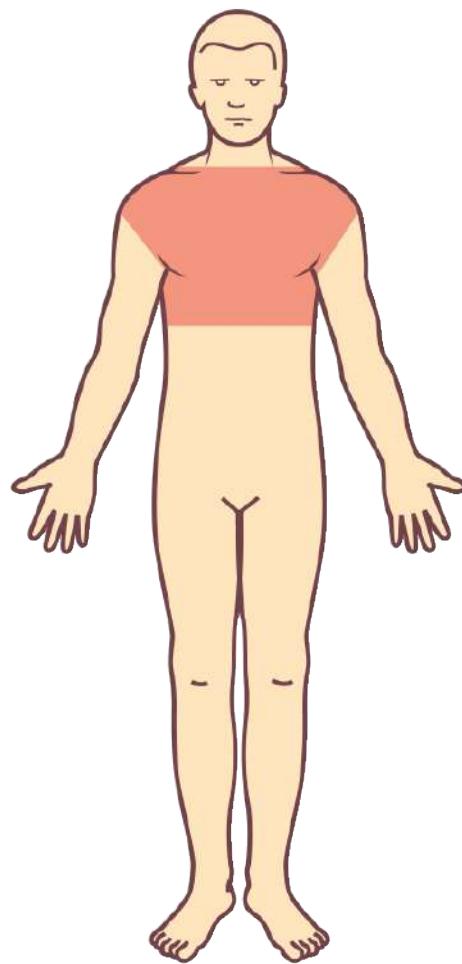


FIGURE 113.18 Pityriasis versicolor: typical truncal distribution
(corresponding area on back)

- Patches may coalesce
- May involve neck, upper arms, face and groin
- Slight scaling when scratched indicates active infection
- Scales removed by scraping show characteristic short stunted hyphae with spores on microscopy
- Often recurrent, especially in summer

Differential diagnosis

Seborrhoeic dermatitis of trunk (more erythematous), pityriasis rosea, vitiligo, pityriasis alba (affects face).

Treatment²

econazole 1% solution to wet skin, leave overnight, for 3 nights

or

ketoconazole shampoo, once daily for 3–5 minutes and wash off, for 5 days

or

selenium sulfide 2.5% shampoo, once daily, leave on for at least 10 minutes or overnight, for 7–10 days

or

(for persistent or recurrent problems) fluconazole 400 mg orally, as a single dose

or

(for prophylaxis) itraconazole 200 mg (o) twice daily for 1 day per month for 6 months²

Note:

- Griseofulvin and terbinafine are inappropriate because the rash is not a fungal infection.
- Warn patients that the white patches will take a long time to disappear and that cure does not equate to disappearance. Pigment will return once there is sufficient sun to repigment the white patches.

Dry skin

Disorders associated with scaling and roughness of the skin include:

- atopic dermatitis—all types (e.g. pityriasis alba, nummular eczema, asteatotic dermatitis)
- ageing skin
- psoriasis
- ichthyotic disorders
- keratosis pilaris

Itching may be a feature of dry skin (but is not inevitable).

Aggravating factors:

- low humidity (e.g. heaters, air-conditioners)

- frequent immersion in water
- heat/hot water
- toilet soaps
- swimming in chlorinated pools

Management

- Avoid a dry home environment if possible.
- Reduce bathing.
- Bathe or shower in tepid water.
- Use a soap substitute (e.g. Dove or Neutrogena/Cetaphil lotion).
- Pat dry—avoid vigorous towelling.
- Rub in baby oil after bathing (better than adding oil to the bath).
- Avoid wool next to the skin (wear cotton).
- Use emollients/moisturisers (e.g. Alpha Keri lotion, QV skin lotion, Nutraplus).

Sunburn

Sunburn is normally caused by UV-B radiation, which penetrates the epidermis and superficial dermis, releasing substances such as leukotrienes and histamines, which cause redness and pain. Severe sunburn may develop on relatively dull days because thin clouds filter UV-B poorly. Beware of solariums and the midday sun.

Clinical presentations:

[Page 1264](#)

Minor sunburn:	Mild erythema with minimal discomfort for about 3 days.
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Moderate:	Moderate to severe erythema within a few hours; worse the following day—red, hot and moderately painful. Settles in 3–4 days with some desquamation.
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Severe:	Classic signs of inflammation—redness, heat, pain and swelling. Skin develops vesicles and bullae. Systemic features develop with very severe burns (e.g. fever, headache, nausea, delirium, hypotension). May require IV fluids.
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Differential diagnosis

- General photosensitivity: consider drugs (e.g. thiazide diuretics, tetracyclines, sulfonamides, phenothiazines, griseofulvin, NSAIDs, isotretinoin)
- Acute systemic lupus erythematosus may present as unexpectedly severe sunburn
- Photo-contact dermatitis

Treatment

Oral aspirin or ibuprofen ease pain. Oil in water baths or bicarbonate of soda paste may help, and wet applications such as oily calamine lotions or simply cool compresses may give relief. Solugel is effective. Topical corticosteroids (e.g. betamethasone dipropionate 0.05%) may be applied to unblistered erythematous skin, twice daily for the first 2 or 3 days.

Prevention

Avoid direct exposure to summer sunlight during peak UV periods (10 am to 3 pm). Use natural shade—beware of reflected light from sand or water and light cloud. Use a sunscreen with a minimum of SPF 50. Wear broad-brimmed hats and protective clothing.

⌚ Photo-ageing/wrinkles

Prevention

- Stop smoking.
- Avoid cold, dry and windy conditions.
- Avoid exposure to the sun.
- Use an SPF 50 or more sunscreen during the day.
- Avoid soaps with perfume and alcohol.
- Wash with a ‘neutral’ mild soap (e.g. Neutrogena, max. twice daily) and pat dry.
- Apply a simple moisturiser immediately after the bath.

Treatment (options)

- Optimal nutrition—diet rich in fruit and vegetables
- Alpha hydroxy acid preparations (e.g. Elucent cream)
- Tretinoin (Retin-A) cream: apply once daily at bedtime (on dry skin); test for skin irritation by

gradual exposure (e.g. 5 minutes at first, wash off, then 15 minutes until it can be left on overnight)

- Injectable treatments such as botulinum toxin and fillers
- Laser treatments

Sweating and odour disorders

Primary hyperhidrosis (excessive sweating)¹⁸

This problem is usually idiopathic and prolonged.

Clinical features

- Male = female incidence
- Affects axilla, groin, soles and palms
- Onset usually around puberty
- Tends to improve after age 25 years
- Family history
- ± Bromhidrosis (malodorous perspiration)
- Usually independent of climate
- Exacerbated by stress and heat; stops during sleep

Causes (secondary/pathological)

- Fever/sepsis
- Thyrotoxicosis
- Acromegaly
- Diabetes mellitus
- Phaeochromocytoma
- Drugs: alcohol/narcotics/antidepressants
- Some neurological conditions (e.g. Parkinson)

- Malignancies especially lymphomas

Treatment^{2,18}

- Avoid known aggravating factors
- First use:
an aluminium chlorohydrate-containing antiperspirant deodorant (spray or roll on each morning) in axilla (suitable for palms and soles)

or

aluminium chloride hexahydrate 20% solution or spray—apply to affected areas at night when the area is dry (best for palms and soles, although irritation is a problem).

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Additional treatments

- Oral anticholinergics (e.g. oxybutynin, propantheline) may give some benefit.
- Iontophoresis (in specialist centres) to hands and feet.
- Injection of botulinum toxin into dermis of affected area. Proven effectiveness for axillae and palms with up to 6 months of anhydrosis.
- Surgical sympathectomy is an option for severe, recalcitrant cases.

Body odour/bromhidrosis

Cause: poor hygiene, excessive sweating, active skin bacteria, metabolic disorders, e.g. trimethylaminuria (rare)—causes fishy odour

Main focus: axillae and groin

Precautions: consider uraemia, vaginitis

Treatment

- Scrub body, especially groin and axillae, with deodorant soap at least morning and night
- Try an antibacterial surgical scrub
- Keep clothes clean, launder regularly
- Choose suitable clothes—natural fibres (e.g. cotton), not synthetics
- Use an antiperspirant deodorant

- Alternative soap—pine soap
- Diet: avoid garlic, fish, asparagus, onions, curry
- Reduce caffeine (coffee, tea and cola drinks), which stimulates sweat activity
- Consider a sugar-free diet
- Remove axillary hair
- Consider injections of botulinum toxin for excessive perspiration

Foot odour (smelly and sweaty feet)

Includes pitted keratolysis secondary to hyperhidrosis (common in teenagers).

Treatment (with options)

- Education and reassurance
- Wear cotton or woollen socks
- Aluminium chloride hexahydrate 20% solution (Driclor) or Neat Feet—apply nocte for 1 week, then 1–2 times weekly as necessary
- Shoe liners (e.g. ‘Odor eaters’), charcoal inner soles
- Apply undiluted Burow’s solution after a shower or bath
- Formalin 1–5% soaks every second night
- Iontophoresis
- The teabag treatment (if desperate):

prepare 600 mL of strong hot tea (from two teabags left in water for 15 minutes)

pour the hot tea into a basin with 2 L of cool water

soak the feet in this for 20–30 minutes daily for 10 days, then as often as required

Skin disorders of feet

Two conditions commonly seen in teenagers are pitted keratolysis and juvenile plantar dermatosis.

Pitted keratolysis

This common malodorous condition known as ‘stinky feet’ or ‘sneakers feet’, usually seen in 10–14-year-olds, is related to sweaty feet. It is caused by an overgrowth of bacteria that digest keratin on the feet (*Corynebacterium sp.* most common). It has a ‘honeycomb’ pitted appearance with maceration between the toes, resembling tinea. For mild cases no specific treatment is needed other than attending to hyperhidrosis. Otherwise, treatment includes keeping the feet dry and using clindamycin 1% lotion bd for 10 days, to remove the responsible organism. Try oral roxithromycin if topical therapy fails. Consider changing to all-leather shoes with charcoal liners. Use a drying agent to decrease sweating.

Juvenile plantar dermatosis

‘Sweaty sock dermatitis’ is a painful condition of weight-bearing areas of the feet. The affected skin is red, shiny, smooth and often cracked. It is rare in adults. The treatment is to avoid friction as much as possible with a barrier cream and suitable footwear, preferably with leather or open shoes and cotton socks. A simple emollient such as urea cream gives relief. Cracks can be treated with medicinal glues.

Prickly heat (miliaria/heat rash)

- Avoid sweating as much as possible.
- Keep the skin dry and cool (e.g. fan, air-conditioner).
- Dress in loose-fitting cotton clothing.
- Attend to bedding—cotton sheets, no plastic underlay.
- Reduce activity.
- Avoid frequent bathing and overuse of soap.
- Dilute topical steroid cream bd.

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Treatment

lotion: salicylic acid 2%, menthol 1%, chlorhexidine 0.5% in alcohol or calamine lotion

or

Egozite (infants), Isophyl (adults)

If severe: hydrocortisone 1% + clotrimazole 1% cream (brief spells only).

Prevention

- Zinc oxide powder

Chilblains and frostbite

⌚ Chilblains (perniosis)

These are localised inflammatory reactions, caused by prolonged exposure to cold, usually on toes and fingers (see FIG. 113.19) but also on heels, nose, ears and thighs (horse riding).



FIGURE 113.19 Chilblains (perniosis) showing erythematous purplish swellings on fingers

Precautions

- Think Raynaud phenomenon—exclude connective tissue disease with ANA, ENA and dsDNA.
- Protect from trauma and secondary infection.
- Do not rub or massage injured tissues.
- Do not apply heat or ice.
- Wear warm gloves and socks.

Differences between chilblains and Raynaud phenomenon:

- chilblains are intermittent without a pattern
- itchy at the onset
- patchy appearance (can be more generalised)

- Raynaud has two or three phases including a ‘dead white’ phase with line of demarcation
- it is significant if it extends to the MCP joints

Treatment

- Elevate affected part
- Warm gradually to room temperature

Drug treatment

- Potent topical corticosteroid (see TABLE 111.9 in CHAPTER 111) twice daily
- If severe and recurrent, apply glyceryl trinitrate vasodilator spray or 0.2% ointment or patch sparingly once daily (use plastic gloves and wash hands for ointment), although weak supportive evidence

Other treatment

- Nifedipine CR 30 mg (o) once daily (if very severe)
- UVB therapy weekly for 4–6 weeks prior to cold weather

Frostbite

Treatment depends on severity.

Precautions

- Watch for secondary infection, tetanus, gangrene.

Treatment

- Elevate affected limb
- Rewarm in water just above body temperature 40°C (104°F) or use body heat (e.g. in axillae)
- Avoid thawing or refreezing
- Surgical debridement
- Don’t debride early (wait until dead tissue dried)
- Don’t drink alcohol or smoke
- For blistering, apply warm water compresses for 15 minutes every 2 hours

Drug treatment

- Analgesics

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

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- Atopic dermatitis (eczema)
- Atopic eczema in children
- Body odour
- Contact dermatitis
- Cradle cap
- Dry skin
- House dust mite management
- Nappy rash
- Pityriasis rosea
- Psoriasis
- Ringworm (tinea)
- Rosacea and perioral dermatitis
- Roseola
- Seborrhoea in infants
- Sunburn
- Sweaty and smelly feet
- Tinea pedis
- Urticaria (hives)

Resources

DermNet NZ: the dermatology resource: www.dermnetnz.org

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114 Acute skin eruptions

They say love's like the measles—all the worse when it comes late in life.

DOUGLAS JERROLD (1803–1857)

The sudden appearance of a rash, which is a common presentation in children (see [CHAPTER 86](#)), usually provokes patients and doctors alike to consider an infectious aetiology, commonly of viral origin. However, an important cause to consider is a reaction to a drug.

A knowledge of the relative distribution of the various causes of rashes helps with the diagnostic methodology. Many of the eruptions are relatively benign and undergo spontaneous remission. Fortunately, the potentially deadly rash of smallpox is no longer encountered.

The diagnostic model is outlined in [TABLE 114.1](#) .

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Table 114.1 Acute skin eruptions: diagnostic strategy model

Probability diagnosis

Hand, foot and mouth disease

Erythema infectiosum (slapped cheek disease or fifth disease)

Roseola infantum (sixth disease)

Other viral exanthema (e.g. enterovirus)

Pityriasis rosea

Impetigo

Folliculitis (e.g. pseudomonas, staphylococcus)

Varicella (chickenpox)

Herpes zoster (shingles)

Herpes simplex

Allergic rash/urticaria/contact dermatitis

Drug reaction (see TABLE 114.4)

Serious disorders not to be missed

Vascular/vasculitis:

- Henoch–Schönlein purpura
- Stevens–Johnson syndrome/toxic epidermal necrolysis
- other vasculitides, e.g. polyarteritis nodosa

Infection:

- purpura of meningococcus, typhoid, other sepsis
- scarlet fever
- primary HIV infection
- secondary syphilis
- measles
- rubella (in pregnancy)
- coronavirus, including COVID-19

Other:

- Kawasaki disease

Pitfalls (often missed)

Guttate psoriasis

Epstein–Barr virus (EBV) mononucleosis

Arbovirus infection (e.g. dengue, Ross River fever, Barmah Forest virus, Japanese encephalitis)

Scabies

Kawasaki disease

Eczema herpeticum

Zoonoses (e.g. listeriosis, Q fever)

Rarities:

- Filovirus diseases (e.g. Ebola, Marburg virus)
- erythema multiforme

A diagnostic approach

The diagnostic approach to skin eruptions presupposes a basic knowledge of the causes; key features of the history and physical examination should logically follow.

The history should include:

- site and mode of onset of the rash
- mode of progression
- drug history
- constitutional disturbance (e.g. pyrexia, pruritus)
- respiratory symptoms
- ?herald patch
- diet—unaccustomed food
- exposure to irritants
- contacts with infectious disease
- bleeding or bruising tendency

The examination should include:

- skin of whole body
- nature and distribution of rash, including lesion characteristics
- soles of feet
- nails
- scalp
- mucous membranes, including oropharynx
- conjunctivae and lymphopoietic system (?lymphadenopathy, ?splenomegaly)

Laboratory investigations may include:

- FBE, ESR, CRP, U&Es
- specific serology (as clinically indicated) for syphilis, parvovirus, rubella, measles, coronavirus
- Epstein–Barr mononucleosis test
- HIV test
- viral and bacterial cultures

- biopsy

Dermatological manifestations of systemic disease

- Painful red nodules
 - erythema nodosum
- Photosensitive rash
 - dermatomyositis (inflamed muscles + rash)—cause unknown, malignancy known association
 - systemic lupus erythematosus
- Palpable purpura
 - vasculitis—primary or secondary (e.g. sepsis), various causes
- Painful ulceration
 - pyoderma granulosum—causes include inflammatory bowel disease, rheumatoid arthritis, haematological malignancies

Acute skin eruptions

The following skin eruptions in children (some of which may also occur in adults) are outlined in [CHAPTER 86](#).

- Measles
- Rubella
- Viral exanthem (fourth disease)
- Erythema infectiosum (fifth disease)
- Roseola infantum (sixth disease)
- Kawasaki disease
- Varicella
- Impetigo

- Hand, foot and mouth disease

§ **Pityriasis rosea**

Pityriasis rosea is a common but mild acute inflammatory skin disorder. The cause is thought to be a viral agent (possibly human herpes virus 6 or 7).

Clinical features

- Any age, mainly older children and young adults
- Preceding oval or round herald patch (1–2 weeks); can have 2–3, but none in 20% (can be mistaken for ringworm)
- Oval, salmon-pink or copper-coloured eruption 0.5–2 cm
- Coin-shaped patches with scaly margins 1–2 cm
- Follows cleavage lines of skin (see FIGS 114.1 and 114.2) with ‘Christmas tree’ pattern on back



FIGURE 114.1 Pityriasis rosea in a 10-year-old child showing the salmon-pink scaly eruption following skin cleavage lines—the ‘Christmas tree’ pattern

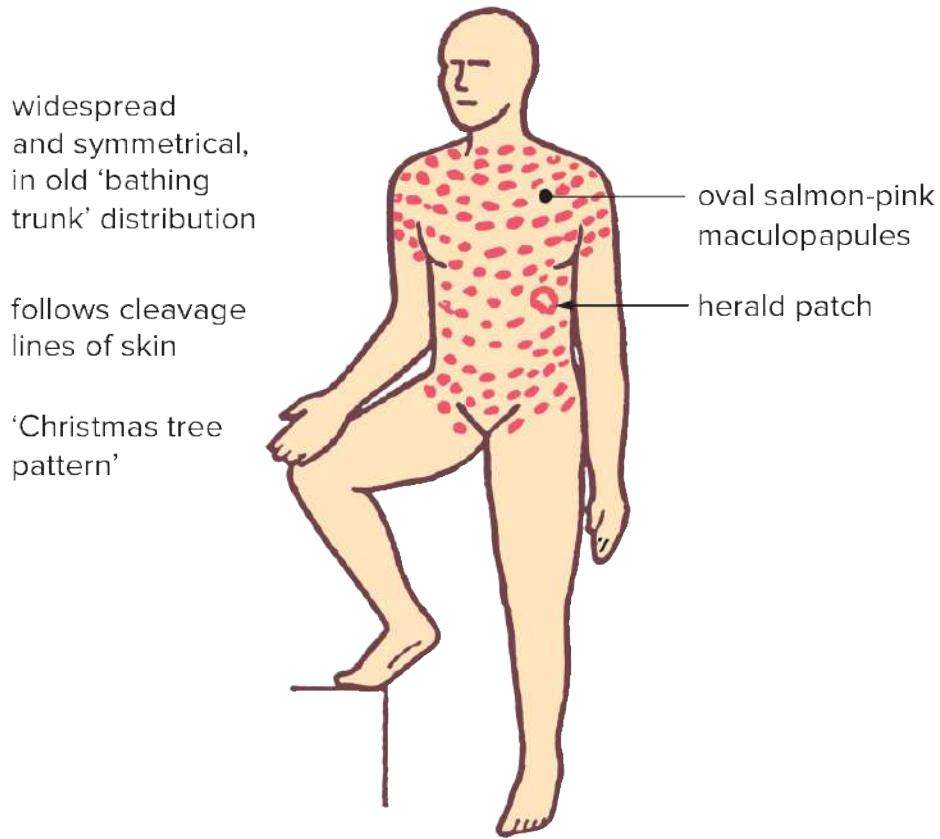


FIGURE 114.2 Pityriasis rosea: typical distribution

- On trunk ('T-shirt' distribution)
- Occurs also on upper arms, upper legs, lower neck, face (rare) and axillae
- Patients not ill
- Itch varies from nil to severe (typically minor itching)
- Scale is on inner aspect of active border

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Differential diagnosis

Herald patch: tinea corporis/discoid eczema.

Generalised rash: seborrhoeic dermatitis (slower onset), guttate psoriasis, drug eruption (see TABLE 114.2), secondary syphilis.

Table 114.2 Drugs that cause eruptions suggestive of pityriasis rosea¹

Main drugs:

- captopril
- gold salts
- penicillamine

Others:

- arsenicals
- barbiturates
- bismuth
- clonidine
- metoprolol
- metronidazole

Prognosis

A mild, self-limiting disorder with spontaneous remission in 2–10 weeks (average 2–5). It does not appear to be contagious. Recurrence is rare.

Management

- Explain and reassure with patient education hand-out.²
- Bathe and shower as usual, using a neutral soap (e.g. QV wash).
- Use a soothing bath oil (e.g. QV Bath Oil).
- For a bothersome itch, apply moderately potent topical corticosteroid daily, e.g. methylprednisolone aceponate 0.1% ointment.
- For a severe itch, use a potent topical corticosteroid daily, e.g. betamethasone dipropionate 0.05% ointment.
- UV therapy is good but, like psoriasis, sunburn must be avoided. Expose the rash to sunlight or UV therapy (if florid) three times a week, with care.

⌚ Secondary syphilis

The generalised skin eruption of secondary syphilis varies and may resemble any type of eruption from psoriasiform to rubelliform to roseoliform. The rash usually appears 6–8 weeks after the primary chancre.

Clinical features (rash)

- Initially faint pink macules
- Then becomes maculopapular
- Can involve whole body (see FIG. 114.3)

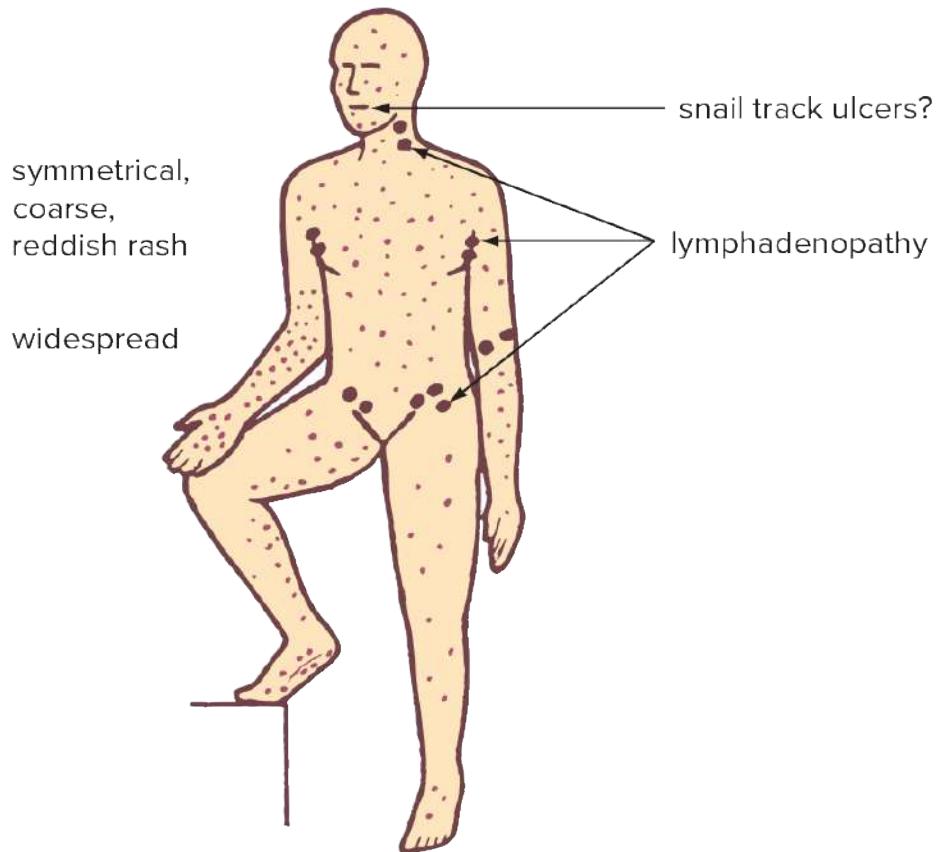


FIGURE 114.3 Secondary syphilis: typical features

- Palms and soles involved
- Dull red in colour and round
- More prolific on flexor surfaces
- Symmetrical and relatively coarse
- Asymptomatic

Associations (possible)

- Mucosal ulcers: ‘snail track’
- Lymphadenopathy
- Patchy hair loss
- Condylomata lata

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Treatment

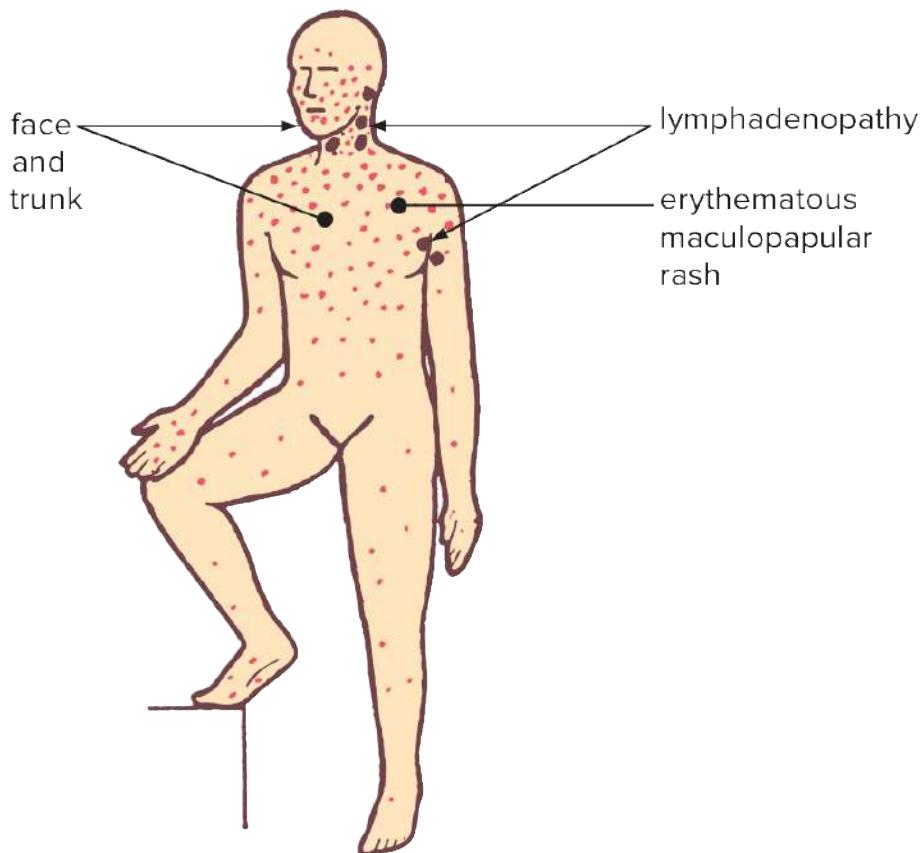
- As for primary syphilis (see [CHAPTER 109](#))

§ Primary HIV infection

A common manifestation of the primary HIV infection is an erythematous, maculopapular rash, although other skin manifestations such as a roseola-like rash and urticaria can occur. See [CHAPTER 18](#).

Clinical features

- Symmetrical
- May be generalised
- Lesions 5–10 mm in diameter
- Common on face and/or trunk
- Can occur on extremities including palms and soles (see [FIG. 114.4](#))
- Non-pruritic



Note: mimics glandular fever

FIGURE 114.4 Primary HIV infection: typical features

Note: Mimics glandular fever

If such a rash accompanied by an illness like glandular fever occurs, HIV infection should be suspected and specific tests ordered.

Guttate psoriasis

Guttate psoriasis is the sudden eruption of small (2–10 mm in diameter), very dense, round, red papules of psoriasis on the trunk (see FIGS 114.5 and 114.6). The rash may extend to the proximal limbs.

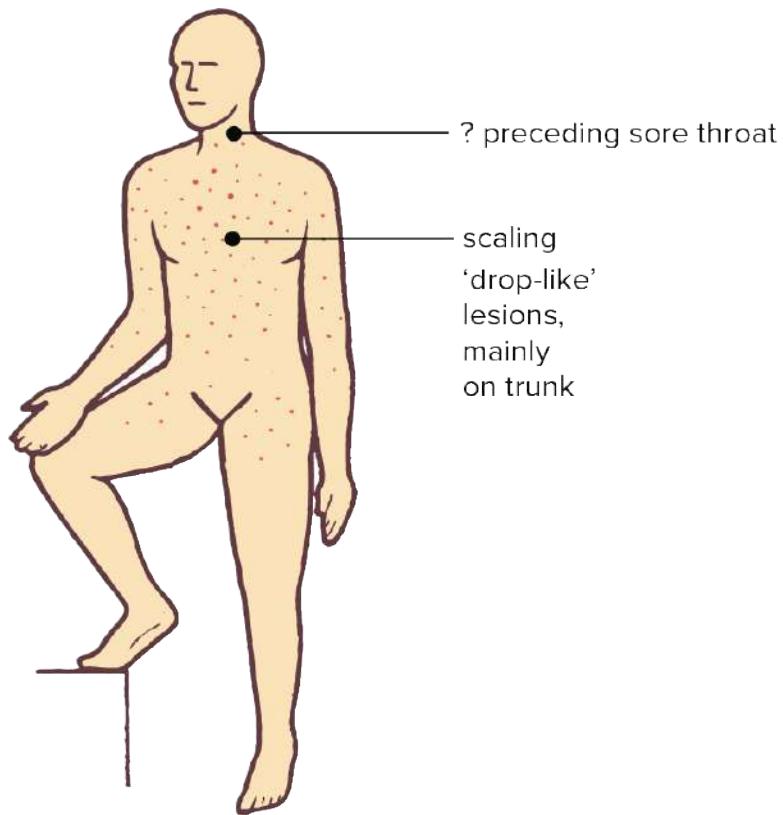


FIGURE 114.5 Guttate psoriasis: small, drop-like lesions, mainly on trunk



FIGURE 114.6 Guttate psoriasis in a child showing the small dense, round erythematous papules of psoriasis on the trunk

It is usually seen in children and adolescents and often precipitated by a *Streptococcus* throat infection. The rash soon develops a white silvery scale. It can persist for up to 6 months. It may undergo spontaneous resolution or enlarge to form plaques, which may become chronic.

Plaques are likely to respond to milder topical treatments. Treatment options are as for [Page 1272](#) psoriasis of the trunk and limbs (see [CHAPTER 113](#)).

Epstein–Barr virus (infectious mononucleosis)

The rash of EBV may be primary or secondary to antibiotics given for tonsillitis (see FIG. 18.3 in CHAPTER 18). The primary rash, most often non-specific, pinkish and maculopapular (similar to that of rubella), occurs in about 5% of cases only. The secondary rash, which can be extensive and sometimes has a purplish-brown tinge, is most often precipitated by one of the penicillins (see FIG. 114.7):

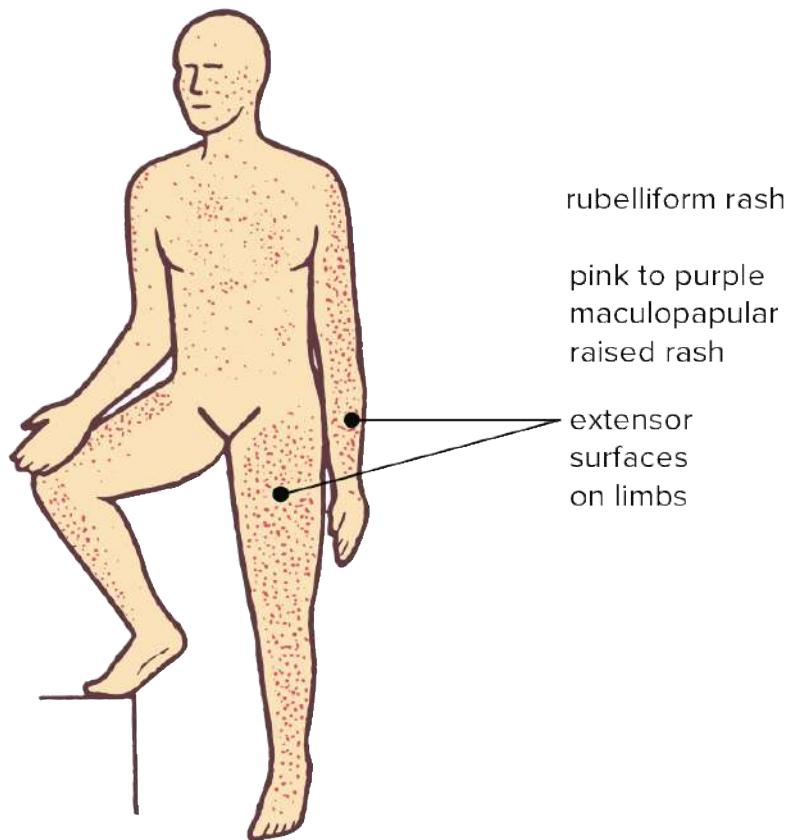


FIGURE 114.7 Epstein–Barr mononucleosis: typical rash induced by penicillin, amoxicillin or ampicillin

- ampicillin 90–100% association
- amoxicillin 90–100% association
- penicillin up to 50%

Drug eruptions

A rash is one of the most common side effects of drug therapy, which can precipitate many different types of rash; the most common is exanthematous, also described as toxic erythema (see TABLE 114.3). Most drug-evoked dermatoses have an allergic basis with the eruption appearing approximately 10 days after administration, though much sooner if previously

sensitised.³ The most common drugs that cause skin eruptions are summarised in TABLE 114.4 .

Table 114.3 Most common types of drug eruptions^{3,4}

Toxic erythema/exanthematous

Urticaria/angioedema

Erythema multiforme

Eczematous dermatitis

Fixed drug reaction

Photosensitivity

Others:

- acneform
- psoriasiform
- lichenoid
- pigmentation
- erythema nodosum
- Stevens–Johnson syndrome/toxic epidermal necrolysis
- vasculitis/purpuric
- pigmentary
- exfoliative

Table 114.4 The most common drugs that cause skin eruptions

Antimicrobials:

Penicillin/cephalosporins*

Sulfonamides*

Tetracyclines

Nitrofurantoin

Streptomycin

Griseofulvin

Metronidazole

Antiretroviral agents

Trimethoprim

Dapsone

Diuretics:

Thiazides

Furosemide

Anti-epileptics:	Carbamazepine*
	Phenytoin
	Lamotrigine
	Phenobarbitone
Tranquillisers:	Phenothiazines
	Barbiturates
	Chlordiazepoxide
Anti-inflammatory and analgesics:	Gold salts
	Aspirin/salicylates
	Codeine/morphine
	Pyrazalones (e.g. BTZ)
	Other NSAIDs*
Hormones:	Combined oral contraceptive
	Stilboestrol
	Testosterone
Others:	Phenolphthalein
	Immunoglobulins
	Amiodarone
	Cytotoxic drugs
	Quinidine/quinine
	Bromides and iodides
	Sulfonylureas
	Allopurinol*
	Warfarin
	Amphetamines

*Often severe

The most important fact to realise about drug reactions is that their appearances are so variable—they may mimic almost any cutaneous disease and, in addition, create unique appearances of their own. Genetic factors appear to be implicated in predisposition to drug reactions, e.g. HLA phenotype.

When taking a history it is appropriate to enquire about medications or chemicals that may be overlooked such as aspirin, vitamins, supplements, laxatives, medicated toothpaste and illicit drugs.

Toxic erythema

The maculopapular erythematous eruption is either morbilliform or scarlatiniform. It is more pronounced on the trunk than on the limbs and face but may become confluent over the whole body (see FIG. 114.8).



FIGURE 114.8 Toxic erythema: maculopapular erythematous scarlatiniform eruption caused by amoxicillin

Drugs that typically cause toxic erythema include:

- antibiotics:
penicillin/cephalosporins

sulfonamides

- thiazides
- carbamazepine
- barbiturates
- allopurinol
- gold salts

⌚ Photosensitivity

Several antibiotics increase the sensitivity of the skin to UV light and may lead to a rash with a distribution according to sunlight exposure. The photosensitive rash may be erythematous, resembling sunburn; eczematous; or vesiculobullous.

Typical drugs:

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- tetracyclines
- sulfonamides/sulfonylureas
- thiazides and frusemide
- phenothiazines
- retinoids
- amiodarone
- griseofulvin
- antiretrovirals
- antihistamines, especially promethazine
- antimalarials
- psoralens

⌚ Fixed drug eruption

The mechanism of fixed drug eruption is unknown. The most commonly affected areas are the face, hands and genitalia. The lesions, which are usually bright red but can have other characteristics, are fixed in site and appear within hours of the drug's administration.

Typical drugs:

- NSAIDs
- paracetamol
- sulfonamides
- phenolphthalein
- tetracyclines
- penicillins
- salicylates
- combined oral contraceptive pill
- barbiturates
- quinine

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Treatment of any drug reaction

The important aspect of management is to recognise the offending agent and withdraw it. The rash should be treated according to its nature.

There is a therapeutic impulse to prescribe antihistamines but they should be reserved for the treatment of urticarial drug eruptions. They may actually delay healing in purpuric, erythematous and vesiculobullous reactions. Antihistamines may act as allergens and show cross-sensitivity with phenothiazines, sulfonamides and topical antihistamines.

TABLE 114.5 lists drugs with the highest skin reaction rates.

Table 114.5 Drugs with the highest skin reaction rates⁴

Penicillin and derivatives

Sulfonamides*

Trimethoprim*

Thiazide diuretics

Allopurinol*

Dapsone*

NSAIDs, esp. piroxicam*

Nevirapine*, abacavir*

Barbiturates

Quinidine

Anti-epileptics (phenytoin, lamotrigine*)

Blood products

Gold salts

*Severe reactions

Erythema

⌚ Erythema multiforme

Erythema multiforme is a hypersensitivity reaction affecting the skin and mucosal surfaces. It is usually triggered by viral infection, most commonly herpes simplex virus (HSV).

Clinical features

- Mainly in children, adolescents, young adults
- Symmetric
- Erythematous papules
- Mainly backs of hands, palms, forearms and face (see FIG. 114.9)

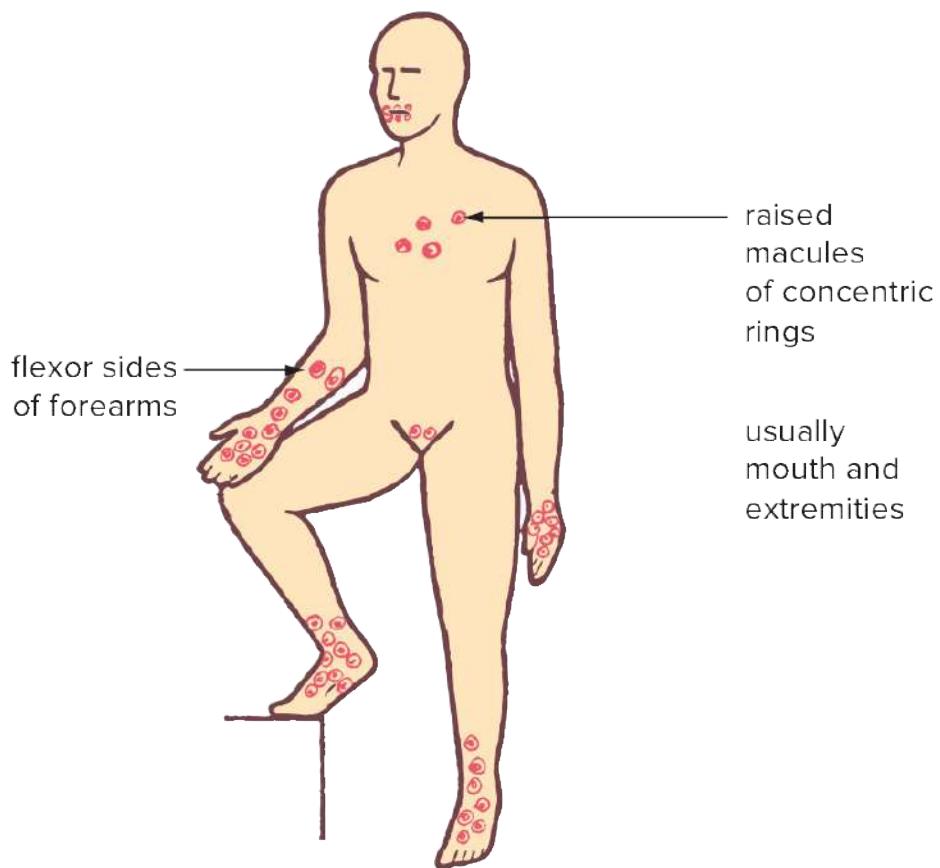


FIGURE 114.9 Erythema multiforme: typical distribution

- Also on feet, toes, mouth
- Occasionally on trunk and genitalia
- Polymorphic
- Vesicles and bullae may develop
- Self-limiting (up to 2 weeks)

Causes and associations

Associations include:

- unknown 50%
- herpes simplex virus 33%
- other infections: *Mycoplasma pneumonia*, tuberculosis, *Streptococcus*, HIV, adenovirus

- drugs 10%:
 - barbiturates
 - penicillin
 - sulfonamides
 - phenothiazines
 - anti-epileptics, e.g. phenytoin

⌚ Stevens–Johnson syndrome/toxic epidermal necrolysis

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are now believed to be variants of the same condition, distinct from erythema multiforme. It is a rare and unpredictable reaction to medication which is severe and often fatal. Onset is usually sudden, with fever, sheet-like skin, mucosal loss and constitutional symptoms. Inpatient treatment and cessation of the causative drug are required.

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Causes

- Beta lactams (penicillins, cephalosporins)
- Sulfonamides
- Anti-epileptics, e.g. phenytoin (known genetic factors)
- NSAIDs
- Allopurinol

⌚ Erythema nodosum

Erythema nodosum is characterised by the relatively sudden onset of bright red, raised, tender nodules on the shins, knees and ankles (see FIG. 19.1 , CHAPTER 19). It is an acute, inflammatory, immunological reaction in the subcutaneous fat. The nodules may also appear on the thighs and the arms. Adult females are typically affected. An arthritic reaction can affect the ankles and knees. It is often associated with recent infection and illness.

Causes and associations

- Idiopathic (most common)
- Sarcoidosis

- Infections:
 - streptococcal infections, e.g. strep throat
 - viral infections (e.g. hepatitis B)
 - tuberculosis
 - leprosy
 - chlamydia infection
 - fungal infections
 - bacterial gastroenteritis
- Inflammatory bowel disorders (e.g. Crohn)
- Drugs:
 - sulfonamides
 - tetracyclines
 - oral contraceptives
 - bromides and iodides
- Malignancy (e.g. lymphoma, leukaemia)
- Pregnancy
- Unknown (about 40%), perhaps autoimmune

Investigations

Tests include FBE, ESR/CRP, chest X-ray (the most important), streptococcal serology (e.g. ASOT) and Mantoux test.

Treatment

Identify the cause if possible. Rest, leg elevation and NSAIDs (e.g. ibuprofen 400 mg (o) bd) for the acute stage. Systemic corticosteroids speed resolution if severe episodes.

- prednisolone 0.75 mg/kg (o) up to 25 mg once daily for 2 weeks, then reduce

Prognosis

There is a tendency to settle spontaneously over 3–8 weeks. The lesions may recur.

Herpes zoster (shingles)

Herpes zoster (shingles) is caused by reactivation of varicella zoster virus (acquired from the primary infection of chickenpox) in the dorsal root ganglion. The term comes from the Greek *herpes* (to creep) and *zoster* (a belt or girdle). Shingles is from the Latin *cingere* (to gird) or *cingulum* (a belt). In most instances the reason for reactivation is unknown, but occasionally it is related to an underlying malignancy, usually leukaemia or a lymphoma, to immunosuppression, or to a local disease or disturbance of the spine or spinal cord, such as a tumour or radiotherapy.

The incidence is 3.4 cases per 1000 population per year. A person of any age can get herpes zoster but it is more common in people over 50 years.

Clinical features

The main features are:

- the condition is preceded by several days of radicular pain with hyperaesthesia
- unilateral patchy rash in one or two contiguous dermatomes (see FIG. 114.10)



FIGURE 114.10 Herpes zoster (shingles) involving the L2 nerve root in a 63-year-old woman presenting with low back and groin pain. Calamine lotion has been applied to soothe the discomfort.

- intense erythema with papules in affected skin
- later crusting and separation of scabs after 10–14 days, often with depigmentation
- regional lymphadenopathy

Distribution

Any part of the body may be affected, but thoracic and trigeminal dermatomes are the most common. It follows the distribution of the original varicella rash (worse on the face and trunk).

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Cranial nerve involvement

The trigeminal nerve—15% of all cases:

- ophthalmic branch—50% affects nasociliary branch with lesions on tip of nose and eyes (conjunctivae and cornea)
- maxillary and mandibular—oral, palatal and pharyngeal lesions

The facial nerve: lower motor neurone facial nerve palsy with vesicles in and around external auditory meatus (notably posterior wall)—the Ramsay–Hunt syndrome.

Complications

- Rare: meningoencephalitis
- Uncommon: motor paralysis
- Common:
 - postherpetic neuralgia; increased incidence with age and debility, with duration greater than 6 months:

less than 50 years	1%
50–59 years	7%
60–69 years	21%
70–79 years	30–50%

the neuralgia resolves within 1 year in 70–80% of these patients but in others it may persist for years

eye complications of ophthalmic zoster including keratitis, uveitis and eyelid damage

Management

- Provide appropriate detailed explanation and reassurance. Dispel myths: namely, explain that it is not a dangerous disease; the patient will not go insane or die if the rash spreads from both sides and meets in the middle.
- Explain that herpes zoster is only mildly contagious; however, people can acquire chickenpox after exposure to a person with the disorder. It is advisable to avoid contact with those who

have never had chickenpox, are not immunised and people who are immunocompromised or undergoing chemotherapy. Consider giving varicella zoster immunoglobulin to immunocompromised contacts who have no history of varicella.

- *Treating the rash:* Instruct the patient to avoid overtreating the rash, which may become infected. Calamine lotion may be soothing but removal of the calamine can be painful. For a hot, painful rash, to remove crusts and exudate, bathe the lesions with saline three times daily. A drying lotion (e.g. menthol in flexible collodion) is most soothing and suitable. Cover the lesions in a light, non-adherent padded dressing.

Oral medication⁵

If the rash has been present for less than 72 hours, antiviral therapy reduces acute pain, duration of the rash, viral shredding and ocular complications. In children, shingles is generally less painful and most children do not require treatment.

Antiviral therapy is indicated in the following patients:

- adults and adolescents who present within 72 hours of onset of vesicles
- who are immunocompromised
- with acute severe pain
- with involvement of special areas (e.g. eye, perineum)

Use:

famciclovir 500 mg 8 hourly (o) for 7 days (10 days if immunocompromised)

or

valaciclovir 1 g 8 hourly (o) for 7 days

or

aciclovir 800 mg (child: 20 mg/kg) (o) 5 times daily for 7 days

Mild pain can be treated with oral paracetamol or NSAIDs.

Corticosteroids⁶

For severe pain, consider prednisolone 50 mg (o) mane for 7 days then taper.

Prevention

- Single varicella zoster vaccine (Zostavax).

Postherpetic neuralgia⁷

Postherpetic neuralgia is pain persisting for at least 3 months after shingles (herpes zoster) infection. It occurs in about 10% of all patients with shingles, and in over 70% of patients older than 50 years.

The pain is usually severe neuropathic pain, varying in quality from paroxysmal stabbing pain to burning or aching. Spasms of pain upon light brushing of the skin are a feature.

Treatment is difficult and a careful ‘trial and error’ approach can be used. Antiviral agents have no significant preventative effect. Pre-emptive treatment with a tricyclic antidepressant may reduce the incidence.

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Treatment options⁶

- Lidocaine 5% patch (to intact skin), up to 3 patches applied at the same time to the painful area. Wear for up to 12 hours, followed by a patch-free interval.
- Oral medication:

tricyclic antidepressants, for example:
amitriptyline 10–25 mg (o) nocte, increasing to a maximum 75–100 mg nocte

or

pregabalin (for lancinating pain) 75 mg (o) nocte initially, increasing the dose gradually to maximum tolerated dose (up to 300 mg bd)

or

gabapentin 100–300 mg (o) daily (nocte) initially, increasing as tolerated to maximum 2400 mg

Management aims to maintain physical function and quality of life. It is more likely to be successful if commenced early.

⌚ Herpes simplex⁷

Herpes simplex is a common infection caused by the large DNA herpes simplex virus (HSV), which can cause a vesicular rash anywhere on the skin or mucous membranes (see FIG. 114.11). There are two major antigenic strains of HSV:



FIGURE 114.11 Acute eruption of herpes simplex on the face: a recurrent problem in this person

- HSV I, which commonly involves the lips and oral mucosa
- HSV II, which basically affects the genitalia (common in adolescents and young adults)

Epidemiology

HSV has a worldwide distribution and is spread orally or genitally by infected secretions. Primary HSV infection is usually a disease of childhood, characteristically causing acute gingivostomatitis in a preschool child (see [CHAPTER 61](#)).

[TABLE 114.6](#) summarises the major manifestations of HSV and the possible complications.

Table 114.6 Herpes simplex virus: manifestations and complications

Examples of manifestations

- Herpes labialis (synonyms: fever blisters, cold sores)
- Keratoconjunctivitis, including dendritic ulcer
- Genital infection
- Other areas of skin such as buttocks

Complications

- Eczema herpeticum
- Erythema multiforme (3–14 days postinfection), often recurrent

Myeloradiculopathy with genital herpes
Pneumonia
Encephalitis

Recurrent infection

Recurrences range from weeks to months and appear due to reactivation rather than re-infection. The cause is not clear but there are several known precipitating factors. These are fever, sunlight, respiratory infections, menstruation, emotional stress, local trauma and, with genital lesions, sexual intercourse.

Fatalities

HSV infections can be potentially fatal. Reactivated HSV can cause a focal destruction encephalitis. The untreated case fatality rate is as high as 70%, but this can be greatly reduced with the use of aciclovir. Neonates exposed to HSV can develop fatal disseminated infection. In compromised patients the infection can be severe.

Diagnosis

If the clinical picture is uncertain, swab of vesicle fluid for PCR can aid diagnosis.

⌚ Genital herpes

See [CHAPTER 109](#) .

⌚ Herpes labialis (classic cold sores)

The objective is to limit the size and intensity of the lesions.

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Treatment⁸

At the first sensation of the development of a cold sore:

aciclovir 5% cream 5 times daily (every 4 hours while awake) for 5 days

or

famciclovir 1500 mg (o) as a single dose

Note: Corticosteroids are contraindicated.

Oral medication

For a severe primary attack:

aciclovir 400 mg (child: 10 mg/kg up to 400 mg)

5 times daily for 5 days

or

famciclovir 1500 mg or valaciclovir 2 g as a single dose

Prevention

If exposure to the sun precipitates the cold sore, use an SPF 50 or more sun protection lip balm, ointment or Solastick. Zinc sulphate solution can be applied once a week for recurrences. Oral aciclovir 400 mg bd or valaciclovir 500 mg daily for 6 months (then review) can be used for severe and frequent recurrences.⁸

Advice to the patient

Herpes simplex is contagious. It is present in saliva and can be spread in a family by the sharing of drinking and eating utensils and toothbrushes, or by kissing. It is most important not to kiss an infant if you have an active cold sore.

Folliculitis⁸

Folliculitis, which is infection in and around hair follicles, can be superficial or deep.

Responsible organisms include bacteria (most common) but consider dermatophytes and yeasts (e.g. *Candida albicans*, *Malassezia*).

⌚ Superficial folliculitis

This usually presents as mild itchy pustules on an erythematous base on any part of hair-bearing skin, particularly in hot weather in a patient who is often a chronic carrier of *S. aureus*. A swab supports diagnosis. Management involves removal of the cause and the application of an antiseptic wash, such as triclosan 1%, chlorhexidine or povidone-iodine. If *S. aureus* is identified consider mupirocin 2% ointment or cream topically, twice daily for 5 days. Occasionally oral flucloxacillin may also be required.

Bacterial folliculitis³

A generalised acute erythematous maculopapular rash can be a manifestation of bacterial folliculitis, typically caused by *S. aureus* or *Pseudomonas aeruginosa*.

Pseudomonas folliculitis can cause confusion, the typical features being:

- rapidly spreading rash