TEENAGERS (10 - 20 YEARS)

Teenagers have issues of self-esteem and image that have to be taken into consideration when managing their skin problems.

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The principal challenge during the teenage years is acne, but some other important dermatoses occur for the first time during this turbulent stage. Unfortunately, when individuals are trying to look their best the skin does not always play along, and enormous emotional distress can result from inadequate treatment. A tendency to anxiety or depressive disorder can be unmasked by a severe skin condition, and conversely skin lesions can be self-inflicted as a cry for help, or as a result of a severe psychosis. Teenagers are generally modest and self-conscious, and do not enjoy clinical examination of their bodies: a gentle approach is encouraged.

ACNE

With the advent of puberty, androgens cause sebaceous glands in certain hair follicles to become much larger and more active. These so-called sebaceous follicles are concentrated on the face, upper back and chest. The increased oil production overwhelms the small hair follicle, which becomes blocked and distended. This first lesion of acne is the microcomedone, and it is the basis for all subsequent lesions. Excessive cornification at the hair follicle orifice aggravates the problem. The microcomedone is invisible to the naked eye, but the skin looks greasy. Microcomedones can remain unchanged for years, or they can evolve into closed, and then open comedones, the so-called whiteheads and blackheads so typical of acne. A predominance of these lesions causes comedonal acne. Usually, however, the comedone evolves further because of spontaneous rupture within the dermis, to form inflamed lesions of acne, inflamed papules, pustules and cysts (Fig.1).

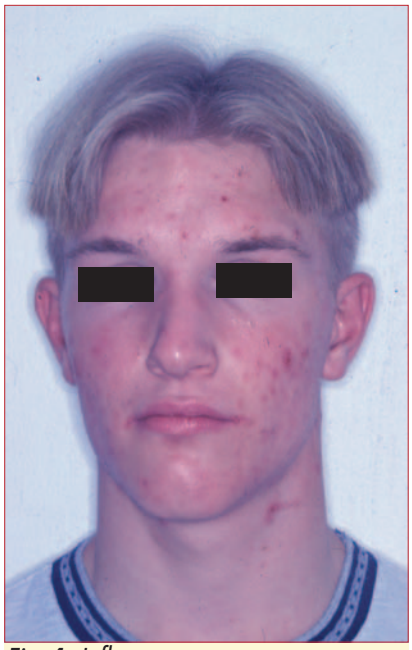

Fig. 1. Inflammatory acne.

Inflammation is due to sebum leaking into the dermis, and is a foreign-body reaction. Commensal bacteria, especially *Propionibacterium acnes* living in the sebum, escape into the dermis and elicit severe inflammation. Acne is *not* an infection. The intensity of the inflammation depends on whether papules, pustules or cysts will be formed. All acne lesions, both comedones and inflamed lesions, will heal with time, but scarring and/or hyperpigmentation can result. This is often more unsightly than the actual acne lesion.

The main focus of therapy is to reduce the formation of microcomedones in the sebaceous follicles. This is best achieved using topical or systemic retinoids. All patients should receive a trial of retinoid cream or gel. However, these agents

tend to cause severe irritant dermatitis and are not always tolerated. If applied infrequently at the beginning of therapy, perhaps twice a week at night, tolerance improves. Applications are gradually increased to nightly. Available topical retinoids include adapalene gel or cream (Differin), tretinoin cream or gel (Retin-A, Ilotycin-A) and isotretinoin gel (Isotrex). These should be applied to the whole face, and not to individual pimples. A moisturiser may be required the following day to prevent excessive dryness.

Comedonal acne is treated with topical retinoids alone: a very gradual response is expected. Inflammatory acne should receive, in addition, either a topical benzoyl peroxide agent or a topical or oral antibiotic. Benzoyl peroxide is also irritant, and can be applied to individual lesions or the whole face, at a different time from the retinoid. There are many benzoyl peroxide agents available, including Benzac-AC 5, Panoxyl 5 or 10, Acnidazil, Brevoxyl, Benoxyl, Acneclear and Benzamycine. Note that these bleach fabrics. An alternative to benzoyl peroxide is azelaic acid cream or gel (Skinoren).

More severe inflammatory acne warrants addition of a systemic antibiotic to the retinoid and benzoyl peroxide. The most widely used antibiotics are tetracyclines in fairly high doses. Options include tetracycline or oxytetracycline 500 mg bd before meals, with a full glass of water to prevent oesophagitis; doxycycline 100 - 200 mg daily; lymecycline 300 - 600 mg daily (Tetralysal) and minocycline 100 - 200 mg daily. Side-effects include photosensitivity and nausea with doxycycline, and vertigo or severe allergy with minocycline. Topical antibiotics include erythromycin solution (Ilotycin TS, Stiemycin, Eryderm) and clindamycin solution or lotion (Dalacin-T). The antibiotic is used primarily for its anti-inflammatory properties in skin, but also to reduce colonisation by *P. acnes*. Courses of 3 - 6 months are usually required, and these can be repeated at intervals, as necessary, until the acne burns out. An alternative regimen in older female teenagers includes one of the antiandrogenic oral contraceptives such as Diane-35, Yasmin or Tricilest. More severe acne, or resistant acne, should be treated with oral isotretinoin, as this offers the best chance of a long-lasting result, and avoidance of severe scarring. A dose of 40 - 60 mg daily for 4 - 6 months is usually adequate, with attention paid to the numerous side-effects and contraindications. Oral isotretinoin agents include Roaccutane, Oratane and Acnetane.

Finally, a word about skin cleansing and cosmetics: these are unfortunately of little value. However, regular cleansing with soap or one of the many acne cleansers may help. Moisturisers, make-up and sunscreens can aggravate acne, but usually do not. Trial and error is the best advice for these products, which are invariably included in a daily ritual of skin care. Regular facials can be beneficial. Diet plays no role in acne despite anecdotal reports to the contrary. Hot weather, sweating, emotional stress, topical and oral corticosteroids, anabolic steroids and excessive intake of iodine can exacerbate acne, and many women experience a premenstrual flare. Most acne will undergo spontaneous resolution in the early twenties, but acne can persist, and can also occur for the first time later in life.

ACANTHOSIS NIGRICANS

Usually found in overweight teenagers, acanthosis nigricans describes brownish-grey, velvety thickening of the flexures, especially the neck, axillae and peri-umbilical area (Fig. 2). Skin tags can occur within the areas of thickening. It is probably due to insulin resistance, and fasting glucose levels should be measured. In older adults the sudden onset of severe and itchy acanthosis nigricans can be paraneoplastic, and a search for malignancy is warranted. The treatment of acanthosis nigricans is difficult: topical steroid creams like prednisolone aceponate (Advantan) can be helpful. Otherwise the best therapy is weight reduction, which often brings about improvement or resolution.

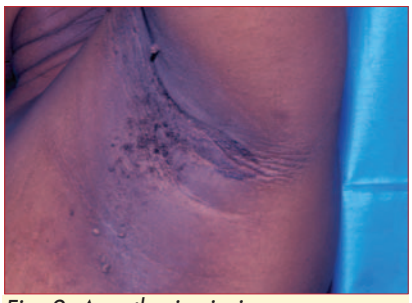

Fig. 2. Acanthosis nigricans.

ALOPECIA AREATA

The sudden onset of perfectly bald round to oval patches anywhere on the scalp is a frightening occurrence. The scalp skin in the bald spot is completely normal, and tiny, short hairs are seen at the periphery of the patch (exclamation mark hairs). This condition is thought to be autoimmune and is often preceded by a period of emotional stress, but may be familial. Fortunately most affected persons regrow their hair within 6 - 12 months, but recurrences are common. A search for other autoimmune conditions is sometimes recommended. Treatment with intralesional steroids like Celestone Soluspan, diluted with saline, into the bald areas is helpful, and should be repeated monthly for several months (Fig. 3).

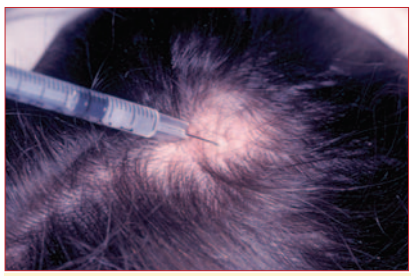

Fig. 3. Alopecia areata — intralesional steroid.

ATOPIC DERMATITIS

Persistence of atopic dermatitis into the teens can portend a chronic, severe course, as most cases will have resolved by now. Involvement tends to be maximal on the neck, scalp, elbow and knee flexures and trunk. Emotional stress is a major trigger fac-

tor, and can cause frequent relapse. Exam times are particularly likely to cause exacerbation. Systemic therapy with oral corticosteroids is often needed, together with multiple courses of antistaphylococcal antibiotics. Resistant cases should be referred for ultraviolet phototherapy, azathioprine or cyclosporin, and psychotherapy for stress management can be helpful.

BOILS

These acutely painful, deep-seated nodules can mimic acne cysts, but are an infection of the terminal hair with *Staphylococcus aureus*, which causes an abscess. These are common on the face, nose, neck, axillae, groins, buttocks and trunk. Individual boils are treated conservatively with hot compresses and antibiotic ointments or creams. Large boils should be lanced under ethyl chloride or EMLA anaesthesia. Systemic antibiotics should only be used if surrounding cellulitis is seen, particularly on the face. Recurrent boils suggest diabetes. Prophylactic treatment with chlorhexidine scrubs of the whole body excluding the face for several weeks (Hibiscrub, Bioscrub), together with mupirocin nasal ointment (Bactroban nasal) is helpful. Recurrent boils in the axillae and groins could suggest hidradenitis suppurativa, an acne-like condition which is not an acute infection, and is treated differently (Fig. 4).

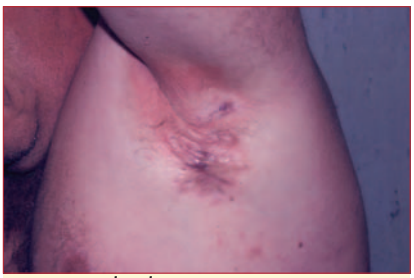

Fig. 4. Hidradenitis suppurativa.

CHILBLAINS

Chilblains are surprisingly common in teenagers living in temperate regions. Itchy or painful reddish papules or nodules occur on the dorsal aspect of the fingers or toes (Fig. 5), and must be distinguished from Raynaud's phenomenon. There may be poor peripheral circulation with acrocyanosis. Treatment is with potent topical steroid creams, and gloves to avoid damp cold. In severe cases, calcium antagonists can be used, but most teenagers outgrow chilblains.

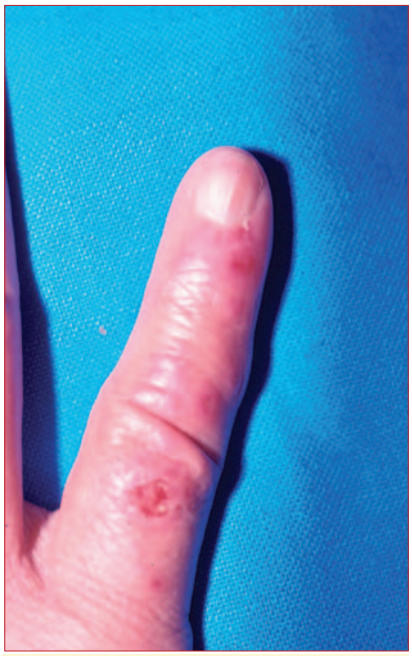

Fig. 5. Chilblains.

CUTANEOUS LARVA MIGRANS

Also known as sandworm, this is caused by larvae of various parasitic worms that normally affect other species, commonly the dog or cat hookworm, and most infections are acquired by walking barefoot on the beach. An intensely itchy lesion develops in 1 - 2 weeks at the site of implantation of the larva, which moves proximally to create a raised, serpiginous, scaly lesion (Fig. 6). The condition is self-limiting but is usually treated with oral albendazole (Zentel) 400 mg daily for 3 days.

DERMATITIS ARTEFACTA

Psychological distress can provoke teenagers (and adults) to wound their skin as a way of attracting attention. These self-inflicted lesions have bizarre morphology, unlike organic disease, and are usually caused by scratching or burning (Fig. 7). Common sites are those easily accessible to the dominant hand, such as the forearms and face. It is important to exclude another cause before diagnosing dermatitis artefacta. Referral for psychotherapy or psychiatric treatment is indicated. A mild form of dermatitis artefacta is acne excoriée, where acne lesions are picked at obsessively.

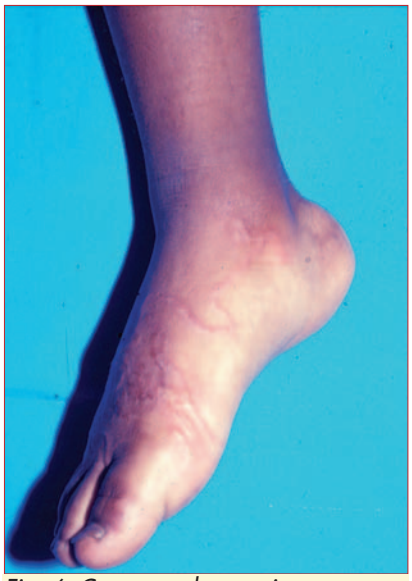

Fig. 6. Cutaneous larva migrans.

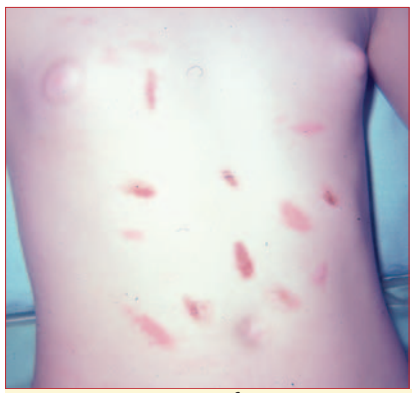

Fig. 7. Dermatitis artefacta.

HALO NAEVUS

Halo naevus occurs when an acquired melanocytic naevus develops a white area around it, with subsequent disappearance of the naevus (Fig. 8). The white area can remain unchanged for life, or can repigment normally. The condition is usually not sinister, although a similar change can occur in melanomas, and many authorities would recommend biopsy or excision.

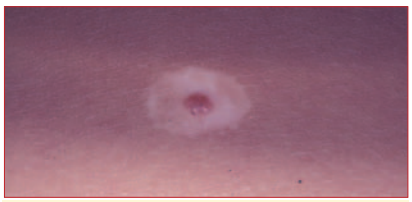

Fig. 8. Halo naevus.

HENOCH-SCHÖNLEIN PURPURA

This syndrome is a leukocytoclastic vasculitis that affects the skin and internal organs. Onset is sudden, with fever, arthralgia, abdominal pain and a rash on the legs, thighs and buttocks. The rash consists of palpable purpura that does not blanch on pressure (Fig. 9). A necrotising neutrophilic vasculitis affects small vessels in the skin, joints and bowel, and can lead to acute renal failure. Skin biopsy can diagnose the condition, which can mimic an acute abdomen. Henoch-Schönlein purpura is probably triggered by a variety of viral upper respiratory tract infections and is self-limiting, with a good prognosis in most cases. It is often treated with a course of oral corticosteroids.

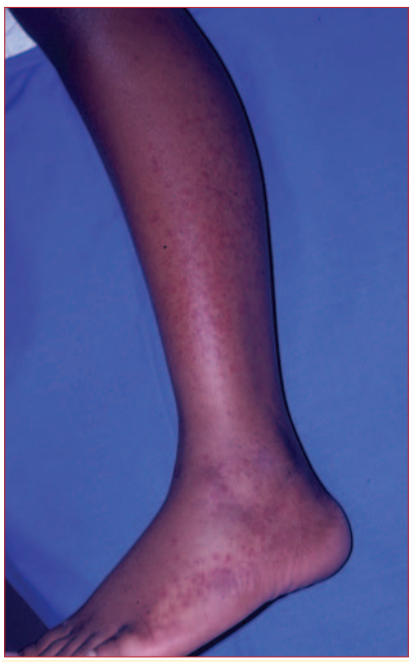

Fig. 9. Henoch-Schönlein purpura.

PAPULONECROTIC TUBERCULIDE

This common condition is usually missed because it mimics so many common skin conditions. Correct diagnosis can be lifesaving, as papulonecrotic tuberculide is caused by an underlying, often silent, focus of tuberculosis, usually in lymph nodes. Crops of non-itchy papules and pustules occur on the elbows, face, ears, knees, palms and soles, leaving pigmented blemishes and deep, punched-out scars (Fig. 10). The symmetry, chronicity and asymptomatic nature of the rash can help to distinguish it from scabies, bites, impetigo, folliculitis, erythema multiforme and other papular conditions. Biopsy is helpful but not essential to the diagnosis, and a search for tuberculosis is mandatory. Even if no obvious focus of tuberculosis is found, empirical antituberculosis treatment for 6 months is warranted. New skin lesions disappear almost instantly.

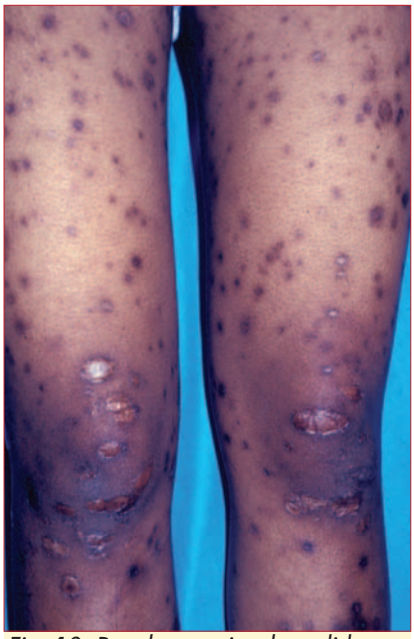

Fig. 10. Papulonecrotic tuberculide.

PLANTAR WARTS

These painful tumours on the soles are caused by infection with certain types of human papillomavirus. They must be distinguished from corns. Corns are usually maximally painful on direct pressure, whereas warts are often more painful on pinching. Corns do not have black dots within them, as these represent dilated thrombosed capillaries in warts. Corns do not disrupt dermatoglyphics (footprints), whereas these creases do not go through warts. Finally corns tend to occur over pressure points, while warts do not always occur there (Fig. 11). Plantar warts should probably only be treated if they are symptomatic as treatment is difficult and dermal scarring on the sole can be painful in itself. Various techniques can be tried; each has a low success rate. Regular application of lactic and salicylic acid in collodion (Duofilm) under plaster occlusion at night over several weeks may help, with the wart pared down with an emery board before each application. Liquid nitrogen cryotherapy is very painful, but can be effective if repeated. Curettage and/or electrodesiccation under local anaesthesia may help, but can cause scarring. Application of a supersaturated solution of monochloracetic acid causes a very painful haemorrhagic blister in a few days, but the success rate is quite good. Surgical excision should be avoided. Corns are best evaluated by a podiatrist, who can also help in the treatment of plantar warts.

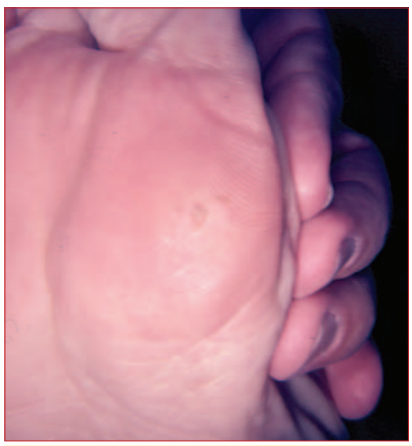

Fig. 11. Plantar wart.

PSORIASIS

Psoriasis presents in many different ways and at any age, and is quite common for the first time in the teens. The cause of psoriasis is not known, but genetic factors are probably involved. Climatic changes, emotional stress, general disability and streptococcal infection can act as trigger factors. There are 7 main types, and individuals may have one or several types together, or at different times of life. Most are chronic, but prone to remissions and relapses.

Guttate psoriasis is classic in children and teenagers and presents as a sudden eruption of numerous, small, thick, waxy, scaly, erythematous papules on the scalp and trunk (Fig. 12),

with minimal itch. A preceding upper respiratory tract infection with *Streptococcus pyogenes* is invariably present, although this might not have been noticed. The rash peaks in several weeks to months, and often resolves spontaneously. Treatment includes a 10-day course of an antibiotic, together with diluted, moderately potent steroid creams or ointments like betamethasone valerate 1:4. Severe cases can be referred for UVB phototherapy, which works very well. Guttate psoriasis can turn into plaque psoriasis, but usually does not.

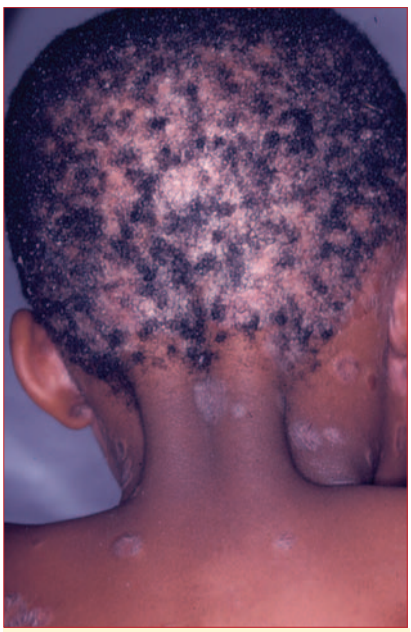

Fig. 12. Guttate psoriasis.

Plaque psoriasis is the commonest chronic form of psoriasis, and presents insidiously with scaly, red plaques spreading on the glabrous (non-hairy) skin, especially the elbows, knees and back (Fig. 13). Itch is variable and can be severe, and the lesions can become very thick. It is treated according to severity and extent of involvement. Mild cases are treated topically with a variety of agents, often used in combination. These include moderate or potent topical steroids, e.g. betamethasone valerate, mometasone, clobetasol propionate and betamethasone dipropionate. These are often combined with tar ointments like 1 - 5% liquor picis carbonis (LPC) (coal tar solution) in emulsifying ointment or Vaseline, or tar gels such as Linotar. Alternatives to tar include the vitamin D ointment or cream, calcipotriol (Dovonex), or vitamin A gel (Zorak). Salicylic acid ointment (1 - 5%) is a useful adjuct for severe scaling. Plaque psoriasis is very slow to respond to therapy, and considerable patience is required. A whole-body moisturiser like emulsifying ointment or mineral oil after bathing is beneficial, as the skin is generally very dry. Extensive plaque psoriasis is treated with one of a variety of systemic therapies, sometimes in combination. These include UVB, PUVA (psoralen and ultraviolet A), acitretin (Neotigason), methotrexate, hydroxyurea and cyclosporin. New biological agents like alefacept are very promising but extremely expensive.

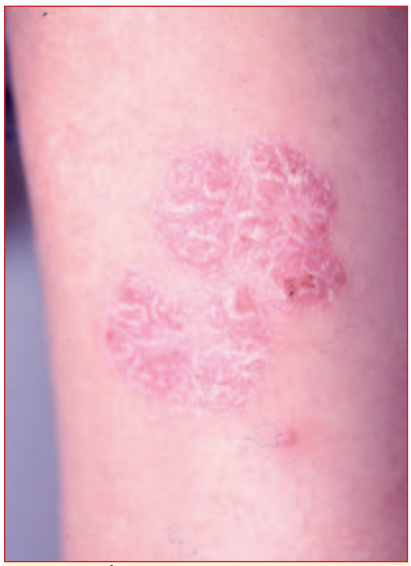

Fig. 13. Plaque psoriasis.

Inverse psoriasis often accompanies plaque psoriasis and signifies involvement of moist flexures as well as the face, scalp, ears and genitalia. Scalp psoriasis mimics severe dandruff, but there is more inflammation, the scale is thicker, and there is patchy rather than diffuse involvement. Scaly, red areas can spread anywhere on the face and ears, simulating seborrhoeic dermatitis. Axillae, groins, and umbilical, perineal and genital areas present as shiny, well-circumscribed erythematous plaques with little or no scale. Inverse psoriasis is generally treated with moderate steroid creams like betamethasone valerate or prednisolone aceponate.

Scalp psoriasis is particularly stubborn, and physical removal of scale is useful, followed by application of a potent steroid cream, lotion or gel such as clobetasol propionate scalp application (Dermovate scalp), mometasone lotion (Elocon lotion), fluocinolone gel (Synalar gel) and betamethasone dipropionate scalp lotion (Diprolene scalp lotion). Prior loosening of scalp scale with 1 - 5% salicylic acid in emulsifying ointment or aqueous cream is helpful, as is the regular use of a tar-based shampoo.

Palmoplantar psoriasis often occurs on its own, and presents with well-circumscribed itchy, erythematous, scaly plaques on the palms and/or soles. The condition is sometimes difficult to distinguish from chronic keratotic hand and foot eczema, but the latter tends to be less well circumscribed and more fissured. Palmoplantar psoriasis is very resistant to therapy, but potent topical steroid ointments can be tried. Systemic therapy with PUVA, acitretin (Neotigason) or methotrexate is often needed.

Pustular psoriasis can be localised or generalised, and presents with sterile pustules on an erythematous base. Localised pustular psoriasis is commonest on the palms and soles, and often occurs in isolation. Treatment is very difficult and along the same lines as palmoplantar psoriasis.
Generalised pustular psoriasis is a medical emergency, requiring admission, supportive care, antibiotics and systemic therapy with cyclosporin, acitretin or methotrexate. Excessive use of potent topical or oral steroids in psoriasis can trigger generalised pustular psoriasis.

Erythrodermic psoriasis is a further severe complication of psoriasis that can evolve from another type of psoriasis or occur *de novo*. Systemic treatment is required, often with hospitalisation.

Psoriasis can also affect the nails and joints, causing one of several patterns of seronegative arthritis.