CHAPTER 1

Eczema

- Atopic dermatitis
- Eczema herpeticum
- Pompholyx eczema
- Discoid eczema
- Venous eczema
- Allergic and irritant contact dermatitis
- Seborrhoeic dermatitis
- Chronic actinic dermatitis
**Atopic dermatitis**

In atopic dermatitis (AD) the skin is chronically dry and itchy but also susceptible to intermittent inflammatory exacerbations. An acute flare of AD is characterized by worsening pruritus and active skin inflammation which, if untreated, can involve the whole skin. A number of factors can precipitate an exacerbation of AD, including temperature changes (central heating), strong sunlight (in some individuals, in others summer sunshine is helpful), common allergens (house dust mite, animal dander), and pregnancy. Secondary staphylococcal skin infection commonly complicates acute AD and, indeed, may be the trigger for a sudden deterioration in previously well controlled eczema.

**CLINICAL FEATURES**

Examination of active AD reveals areas of poorly defined erythema, inflammatory papules, and fine scaling (1). The sites of predilection are the face, neck, limb flexures, and hands (2). When very active the erythema of AD is urticated and can rapidly spread to involve the whole skin (3). The intense itch leads to scratching which results in linear scratch marks and excoriated papules (4). White dermographism describes pale streaks from scratching within areas of erythema. Unlike other forms of acute dermatitis, vesicles do not occur in active AD, however a serous exudate and crusting may be present. Secondary bacterial infection with *Staphylococcus aureus* causes a honey-coloured discolouration to the scale (impetiginization) (5). Patients with longstanding AD also display the signs of chronic eczema, such as lichenification.

**DIFFERENTIAL DIAGNOSIS**

- Seborrhoeic dermatitis (p. 20, erythema and scaling of the face, scalp, and chest).
- Contact dermatitis (p. 18, eczema in a localized area or unusual distribution).
- Eczema herpeticum (p. 10, eczema plus small, punched-out erosions of herpes simplex virus (HSV) infection).
- Scabies infestation (p. 162, severe pruritus plus burrows at the wrists and finger webs).
COMPLICATIONS
- Malaise and fatigue (with extensive involvement).
- Regional lymphadenopathy (with secondary bacterial infection).
- Fever (with severe secondary bacterial infection).
- Erythroderma (see p. 51).

INVESTIGATIONS
- The diagnosis is usually made clinically.
- Skin swab for bacteriology (S. aureus infection occurs commonly).
- Skin swab for virology (if secondary infection with HSV is suspected).
- Swab of anterior nares for bacteriology (to exclude nasal carriage of S. aureus).
- Blood count, basic chemistry, liver function tests (if considering systemic immunosuppressant therapy or if the patient is systemically unwell).

IMMEDIATE MANAGEMENT
Topical therapy
- General emollient therapy.
- Corticosteroid ointment twice per day (use for a restricted period):
  - face: mildly potent.
  - trunk and limbs: moderately potent.
- A gauze bodysuit can be used to maximize the effects of topical therapy.

Systemic therapy
- For secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacillin 500 mg four times per day for 7 days).
- A sedating oral antihistamine at bedtime (e.g. hydroxyzine 25 or 50 mg) will relieve itch and improve sleep.
- A short, reducing course of oral corticosteroids can be helpful to induce remission (e.g. prednisolone 20–30 mg/day and reducing by 5 mg every 5th day).

LONG-TERM MANAGEMENT ISSUES
Adherence to appropriate skin care is essential to maintain a remission: regular use of an emollient, bath oil, and soap substitute is essential. Other conservative measures should be considered: cotton clothing, the control of house dust mite in the home, and behaviour modification techniques to prevent scratching. Nasal carriage of S. aureus needs to be treated with fusidic acid or mupiricin ointment or chlorhexidine cream applied to the anterior nares three times per day for 7 days. Intermittent use of topical corticosteroid or immunomodulator (e.g. tacrolimus, pimecrolimus) is usually necessary. Patients with severe, recalcitrant AD need to be under the care of a dermatologist and may require treatment with second-line therapy, such as phototherapy or systemic immunosuppressants (e.g. azathioprine or ciclosporin).
Eczema herpeticum

Eczema herpeticum is the widespread infection of atopic dermatitis by herpes simplex virus (HSV). It can occur in an atopic dermatitis patient who develops a cold sore or who comes into contact with an active herpetic lesion on another individual. This condition, with the alternative name of Kaposi’s varicelliform eruption, can also occur in cutaneous T cell lymphoma, pemphigus foliaceous, and Darier’s disease. Less commonly it can occur following genital HSV infection. Eczema herpeticum is a serious dermatosis which can progress into a disseminated and potentially fatal systemic herpetic infection.

CLINICAL FEATURES

Secondary herpetic infection of eczema can be difficult to identify and should therefore be considered in all patients with an acute flare of atopic dermatitis. The eruption of eczema herpeticum is painful and usually involves the face, neck, and upper chest, but can become widely disseminated. It is characterized by discrete vesicles and vesico-pustules occurring on red, eczematous skin which burst to leave punctate, haemorrhagic erosions (6). Coalescence of individual lesions produces large eroded areas (7). During the acute phase there may be significant local oedema. Lesional skin may become additionally infected with S. aureus or streptococci. The associated impetiginized crust may obscure the signs of herpes infection (8).

DIFFERENTIAL DIAGNOSIS

- Impetiginized atopic dermatitis (p. 8, eczema with overlying honey-coloured crust).
- Impetigo (p. 118, areas of erythema with superficial blisters and overlying honey-coloured crust).
- Primary HSV infection (p. 142, cluster of vesicles on a red base).
- Primary varicella zoster virus (VZV) infection (chicken pox) (p. 146, disseminated vesicles, each on a red macule).

6 Eczema herpeticum. Numerous vesicles have ruptured to produce circular erosions. Eczema herpeticum usually occurs on the face or neck.

7 Eczema herpeticum. Confluence of numerous individual herpetic lesions can result in a large eroded area often with straight or angulated margins.

8 Eczema herpeticum. Individual lesions on the temple and lower eyelid can be seen but extensive impetiginized crust on the upper eyelid and eyebrow has obscured signs of herpetic infection.
COMPLICATIONS

- Fever (if infection is severe).
- Malaise and fatigue (with extensive involvement).
- Regional lymphadenopathy.
- Hepatitis.
- Pneumonitis.
- Encephalitis.

INVESTIGATIONS

- Skin swab of vesicle base for viral culture (culture of HSV from vesicle fluid takes 1–5 days).
- Collection of vesicle fluid and scrapings of ulcer base for immunofluorescence, electron microscopy, and polymerase chain reaction (PCR) (electron microscopy can identify viruses of the herpes family rapidly; immunofluorescence of vesicle scrapings can differentiate HSV1 and HSV2; HSV PCR is the most sensitive identification method).
- Tzanck smear: scrapings of the ulcer base for cytology (a nonspecific test demonstrating multinucleated giant cells and intranuclear inclusion bodies in epithelial cells).
- Skin swab for bacteriology (if secondary bacterial infection is suspected).
- Blood count, basic chemistry, liver function tests (if patient is systemically unwell).
- Chest radiograph (to exclude herpes pneumonitis).

IMMEDIATE MANAGEMENT

Topical therapy

- General emollient therapy.
- Potassium permanganate soaks once per day.

Systemic therapy

- For modest involvement, with no systemic features: oral aciclovir 200 mg five times per day for 5 days. An alternative to aciclovir is valaciclovir 500 mg twice per day for 5 days.
- For moderate or severe involvement, or any case with systemic features: intravenous aciclovir 5 mg/kg three times per day (10 mg/kg three times per day in the immunocompromised) for 5 days.
- With concomitant staphylococcal or streptococcal infection give oral or intravenous antibiotics.

Supportive therapy

Patients with moderate or severe eczema herpeticum should be admitted to hospital for the following:

- Bed rest and intensive topical therapy (see above).
- Monitoring of vital signs (pulse rate, blood pressure, temperature).
- Monitoring of fluid balance and administration of intravenous fluids, if required.
- Initiation of systemic therapy (see above).
- Analgesia.

LONG-TERM MANAGEMENT ISSUES

Following treatment, the erosions of eczema herpeticum generally heal within 1–2 weeks. Once the herpetic infection has been treated, manage the underlying atopic dermatitis with topical therapy (see Atopic dermatitis, p. 8). Ensuring the dermatitis is well controlled is essential in minimizing any future risk of reinfection with HSV. Patients with atopic dermatitis should have cold sores treated aggressively and should avoid close contact with an individual who has an open cold sore. In recurrent HSV infections consider giving prophylactic oral aciclovir 400 mg twice per day for 6 months.
Pompholyx eczema

Pompholyx or dyshidrotic eczema is characterized by vesicles on the palms and soles. The hands are more commonly affected than the feet but both sites may be involved concurrently. Although pompholyx eczema appears to be a distinct entity it may represent a manifestation of atopy or a presentation of contact dermatitis. Palmar pompholyx also occurs as a response to dermatophyte infection of the feet (tinea pedis). The vesiculation and blistering of pompholyx eczema often develops suddenly and, because of the sites of involvement, can be functionally disabling.

**CLINICAL FEATURES**
Deep-seated, itchy vesicles develop on the palms and lateral borders of the fingers and on the soles of the feet (9, 10). The signs are usually symmetrical. Unlike other forms of eczema there is little associated dryness in the acute phase. Coalescence of individual vesicles can produce large tense blisters, especially on the feet. Rupture of the blisters will lead to weeping and sometimes secondary staphylococcal or streptococcal infection. Attacks usually subside spontaneously after a few weeks with desquamation.

**DIFFERENTIAL DIAGNOSIS**
- Contact dermatitis (p. 18, eczema in localized area or unusual distribution).
- Palmo-plantar pustulosis (p. 30, pustules on palms and soles with scaling).
- Bullous tinea manum/pedis (p. 154, unilateral itchy blisters with erythema on palms or soles).

**SYSTEMIC COMPLICATIONS AND ASSOCIATIONS**
**Complications**
- Regional lymphadenopathy (with secondary bacterial infection).
- Erysipelas/cellulitis.

**Associations**
- Atopy.
- Tinea pedis.

**INVESTIGATIONS**
- The diagnosis is usually made clinically.
- Swabs for bacteriology (if secondary infection is suspected).
- Skin scrapings for mycology (to exclude dermatophyte infection).
- Patch testing (if allergic contact dermatitis is suspected).

**IMMEDIATE MANAGEMENT**
**Topical therapy**
- General emollient therapy.
- Potassium permanganate soaks once per day after rupturing blisters.
- Corticosteroid ointment twice per day (use for a restricted period):
  - palms and soles: potent.
  - (Corticosteroid efficacy can be enhanced by occlusion using polythene gloves for the hands and plastic kitchen wrap for the feet.)

**Systemic therapy**
For secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacinil 500 mg four times per day for 7 days). In severe involvement a short course of oral corticosteroids can be helpful to induce remission (e.g. prednisolone 20–30 mg/day and reducing by 5 mg every 5th day).

**LONG-TERM MANAGEMENT ISSUES**
Adherence to appropriate skin care is essential to maintain a remission: regular use of an emollient and soap substitute is essential. Hyperhidrosis can be a contributory factor in plantar pompholyx eczema, therefore careful foot hygiene and iontophoresis may be helpful. Patch testing must be performed in patients with chronic pompholyx eczema to exclude an allergic contact dermatitis. Severe, recalcitrant pompholyx eczema may respond to hand–foot phototherapy, systemic retinoid (e.g. acitretin), or systemic immunosuppressants (e.g. azathioprine or ciclosporin).
Pompholyx eczema. There are numerous tense vesicles occurring on
the skin of the palms, worst on the left. In places the vesicles have
become confluent.

Pompholyx eczema. There is extensive vesiculation
on the soles of the feet. Some lesions have become
pustular indicating secondary infection with \textit{S. aureus}. 
Discoid eczema

Discoid (or nummular) eczema is characterized by discrete, round patches of dermatitis. Sometimes a solitary lesion will occur, more commonly they are multiple. The eruption is very itchy and often develops suddenly, precipitating an acute presentation.

CLINICAL FEATURES
In discoid eczema a localized area of dermatitis initially develops on the lower aspect of one leg (11). The lesion starts as a cluster of tiny papules which become confluent and evolve into an intensely itchy, disc-shaped patch or plaque (12). Thereafter several similar, discoid lesions may appear elsewhere on the legs and arms. In severe cases, patches of eczema can also develop on the torso (13). Initially the lesions are weepy but become dry with time, often covered by an impetiginized crust.

DIFFERENTIAL DIAGNOSIS
- Tinea corporis (p. 154, annular lesions with inflammatory margins).
- Plaque psoriasis (p. 28, scaly plaques distributed symmetrically, typical nail changes).
- Bowen’s disease (intraepidermal carcinoma: red patch with mild scale and distinct margin).
- Mycosis fungoides (p. 198, pink or red patches with superficial atrophy, distributed asymmetrically).

COMPLICATIONS
There are usually none.
INVESTIGATIONS
• The diagnosis is usually made clinically.
• Skin swab for bacteriology (if secondary infection is suspected).
• Skin scrapings for mycology (to exclude dermatophyte infection).
• Skin biopsy for histopathology if the diagnosis is in doubt (epidermal spongiosis, vesiculation, acanthosis, and exocytosis of lymphocytes and neutrophils).

IMMEDIATE MANAGEMENT
Topical therapy
• General emollient therapy.
• Topical corticosteroid ointment twice per day (use for a restricted period):
  trunk and limbs: potent.
  (Corticosteroid efficacy can be enhanced by occlusion using a dressing or a paste bandage.)

Systemic therapy
For extensive secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacillin 500 mg four times per day for 7 days).

LONG-TERM MANAGEMENT ISSUES
Discoid eczema tends to run a chronic course and consequently prolonged adherence to appropriate skin care is essential. Relapses can be treated with topical therapy, as above. Severe recalcitrant discoid eczema may respond to phototherapy or systemic immunosuppressants (e.g. azathioprine, ciclosporin). In some cases chronic discoid eczema may be associated with alcohol misuse and, therefore, advice on alcohol restriction can be helpful. Patients with chronic discoid eczema or who suffer frequent relapses should undergo patch tests to exclude an allergic contact dermatitis.

13 Discoid eczema. In severe discoid eczema there may be involvement of the arms and torso, as with this patient. In areas there is an extensive impetiginized crust, indicating heavy secondary infection with S. aureus.
Venous eczema

Venous eczema involves the skin of the lower legs and develops as a result of venous insufficiency. It tends to occur in elderly patients with venous incompetence arising from varicose veins or previous deep venous thrombosis (DVT). The eruption may develop acutely and, if unilateral, can be mistaken for cellulitis.

CLINICAL FEATURES
Initially there is erythema involving the medial aspect of the lower leg (14). The eruption is very itchy and usually becomes excoriated (15). With time venous eczema extends to involve the whole of the lower leg and foot. Scaling and crusting is often prominent. There are usually other signs of venous insufficiency, e.g. varicose veins, oedema, haemosiderin deposition, lipodermatosclerosis, and ulceration. Autosensitization can occur in which papular eczema appears on the arms and torso as a reactive phenomenon to eczema on the legs. As with other forms of dermatitis, venous eczema can be complicated by secondary bacterial infection.

DIFFERENTIAL DIAGNOSIS
- Allergic contact dermatitis (p. 18, eczema in a localized area or unusual distribution).
- Dependency syndrome (p. 96, lymphoedema and venous congestion in poorly mobile chair-bound individuals).
- Discoid eczema (p. 14, discrete, oval patches of eczema, usually on lower legs).
- Psoriasis (p. 25, scaly plaques distributed symmetrically, typical nail changes).
- Erysipelas/cellulitis (p. 126, zone of painful, spreading erythema with fever).

COMPLICATIONS
- Erysipelas/cellulitis.

INVESTIGATIONS
- The diagnosis is usually made clinically.
- Skin swabs for bacteriology (if secondary infection is suspected).
- Venous studies (may identify a cause for venous insufficiency, e.g. perforator incompetence, past DVT).
- Ankle–brachial pressure index (ABPI) of affected leg (ABPI <0.8 indicates significant arterial disease and compression should be avoided).
- Thrombophilia screen with history of DVT (to exclude clotting diathesis).

IMMEDIATE MANAGEMENT
Topical therapy
- General emollient therapy.
- Topical corticosteroid ointment twice per day (use for a restricted period):
  - legs: potent.
- Leg elevation.

Systemic therapy
For secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacillin 500 mg four times per day for 7 days).

LONG-TERM MANAGEMENT ISSUES
Venous eczema tends to run a chronic course and therefore adherence to appropriate skin care is essential. Compression bandaging can be helpful to induce remission. Long-term use of support hosiery can prevent relapses. A vascular surgical opinion may be helpful to identify any correctible venous disorder. If the eczema fails to remit with appropriate treatment then consider a secondary allergic contact dermatitis to medicaments in topical agents or to rubber in elasticated bandages or hosiery.
Venous eczema. This patient with varicose veins, worse on the right, has venous eczema involving the right lower leg.

Venous eczema involves the gaiter area of the lower leg. It often gets excoriated.
**Allergic and irritant contact dermatitis**

Contact dermatitis is an eczema caused by skin contact with an exogenous substance. It is divided into two groups: (1) allergic contact dermatitis (ACD), which is a delayed hypersensitivity reaction to an exogenous allergen, and (2) irritant contact dermatitis (ICD), which is induced by direct inflammatory pathways without prior sensitization. Certain occupations are considered more likely to predispose individuals to develop contact dermatitis, e.g. hairdressing, cleaning, catering, nursing, child care, and construction. Common sensitizers include nickel in jewellery, rubber in gloves and shoes, and preservatives in cosmetics. Identification of the relevant allergen requires information concerning the relation of the dermatitis with work, periods away from work (e.g. holidays), hobbies, and household chores. Severe ACD can occur in patients allergic to certain plants, e.g. poison ivy. Some substances are photoallergens and need both sunlight and the chemical to induce dermatitis. In ICD the common irritants include detergents, water, organic solvents, acidic and alkaline chemicals. Contact dermatitis can present acutely, often in an individual with no previous history of eczema.

**CLINICAL FEATURES**

Acute contact dermatitis of both types is characterized by erythema, oedema, vesicles, and exudation. The precise distribution of the eruption may provide a valuable diagnostic clue to the likely allergen or irritant (16–19). For example, involvement of ear lobes may suggest ACD to nickel in jewellery, involvement of the face may suggest ACD to preservatives or fragrances in skin-care products. With prolonged exposure to an allergen the dermatitis will extend beyond the zone of contact. In ICD the hands are the sites most usually affected, both dorsal and palmar surfaces, often secondary to prolonged contact with irritants such as detergent and water. Mild irritants cause redness, dryness, and scaling, whereas potent irritants induce fiery erythema, oedema, vesicles, and, in some cases, ulceration.
DIFFERENTIAL DIAGNOSIS

Face
- Atopic dermatitis (p. 8, eczema as part of atopic diathesis).
- Seborrhoeic dermatitis (p. 20, erythema and scaling of the face, scalp, and chest).
- Psoriasis (p. 25, scaly plaques distributed symmetrically, typical nail changes).
- Tinea faciei (p. 154, annular lesion(s) with inflammatory margin).

Hands
- Atopic dermatitis (p. 8, eczema as part of atopic diathesis).
- Psoriasis (p. 25, scaly plaques distributed symmetrically, typical nail changes).
- Pompholyx eczema (p. 12, vesicular eczema of palmo-plantar skin).
- Tinea manum (p. 154, erythema and scaling, usually affecting one hand only).

Feet and lower legs
- Venous eczema (p. 16, eczema on lower legs caused by venous insufficiency).
- Psoriasis (p. 25, scaly plaques distributed symmetrically, typical nail changes).
- Pompholyx eczema (p. 12, vesicular eczema of palmo-plantar skin).
- Tinea pedis (p. 154, erythema and scaling of foot (feet), usually involving toe web(s)).

COMPLICATIONS
- Regional lymphadenopathy (with secondary bacterial infection).
- Erysipelas/cellulitis.

INVESTIGATIONS
- Patch tests (multiple, standardized allergens are applied to the upper back: a positive reaction confirms an allergic contact sensitivity; multiple allergens in a single patient is common).
- Skin swabs for bacteriology (if secondary infection is suspected).

18 Allergic contact dermatitis. This patient has acute dermatitis, worse on the right leg, with erythema, vesiculation, swelling, and impetiginization. This was triggered by contact with poison ivy.

19 Allergic contact dermatitis. ACD to a topical analgesic applied to the lower leg under a bandage. There has been a blistering reaction with an area of epidermal loss over the calf. The sharp cut-off denotes the zone of analgesic application and the limits of the bandage.
IMMEDIATE MANAGEMENT

Topical therapy

- ICD: following recent contact with a potent chemical irritant, lavage immediately with water.
- ACD: identify potential allergen and avoid further contact.
- General emollient therapy.
- Corticosteroid ointment twice per day (use for a restricted period):
  - face: mildly potent.
  - trunk and limbs: moderately potent.
- If vesiculation and weeping are prominent use potassium permanganate soaks once per day.

Systemic therapy

For secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacillin 500 mg four times per day for 7 days). With severe contact dermatitis give a short course of oral corticosteroids (e.g. prednisolone 20–30 mg/day and reducing by 5 mg every 5th day).

LONG-TERM MANAGEMENT ISSUES

Careful interpretation of patch tests by a dermatologist is central to the correct management of a patient with ACD. Once initial sensitization to an allergen has occurred the potential to react persists indefinitely and continued exposure to the allergen will result in chronic ACD. It is therefore important that information concerning sources of allergen exposure is given to the patient. Long-term skin care with the regular use of an emollient is helpful in prolonging remission. The use of topical tacrolimus ointment or pimecrolimus cream will reduce requirements for topical corticosteroid. In ICD persistence of contact with irritant will induce dryness, scaling, and lichenification. Avoidance of the irritant(s) will eventually facilitate resolution of the dermatitis. In ICD the use of relevant protective clothing and barrier creams can be helpful. Patients with irritant hand dermatitis must be encouraged to wear vinyl gloves when undertaking wet work.

Seborrhoeic dermatitis

Seborrhoeic dermatitis occurs at sites rich in sebaceous glands and is caused by an excess of Malassezia yeasts in these areas. Seborrhoeic dermatitis tends to run a relapsing–remitting course, however acute exacerbations are common.

CLINICAL FEATURES

The scalp is almost always involved with itchy erythema and scaling causing dandruff. On the face, areas of predilection include the hairline, eyebrows, glabella, and nasolabial folds (20, 21). Blepharitis is common. Involvement of the presternal and flexural skin is characterized by well demarcated patches of erythema with scaling. An acute, severe flare can result in widespread eczema with minimal scaling involving the head, face, neck, torso, and flexures (22).

DIFFERENTIAL DIAGNOSIS

- Psoriasis (p. 27, scaly plaques distributed symmetrically, typical nail changes).
- Contact dermatitis (p. 18, eczema in a localized area or unusual distribution).
- Atopic dermatitis (p. 8, eczema as part of the atopic diathesis).
- Tinea faciale/corporis (p. 154, annular lesions with inflammatory margins).
- Subacute cutaneous lupus erythematosus (p. 110, annular and polycyclic rash on torso).

COMPLICATIONS

There are usually none.

INVESTIGATIONS

- The diagnosis is usually made clinically.
IMMEDIATE MANAGEMENT
Topical therapy
• General emollient therapy.
• Treat scalp involvement with ketoconazole shampoo.
• Corticosteroid ointment twice per day (use for a restricted period):
  face and flexures: mildly potent containing azole antifungal.
  torso and limbs: moderately potent.

LONG-TERM MANAGEMENT ISSUES
Seborrhoeic dermatitis tends to relapse and so active therapy may need to be reinstated intermittently. Long-term skin care with the regular use of an emollient is helpful in prolonging remission. The use of topical tacrolimus ointment or pimecrolimus cream will reduce requirements for topical corticosteroid. For unresponsive cases a course of oral itraconazole (200 mg once daily for 14 days) can be useful. Ultraviolet B (UVB) phototherapy may also be helpful. In some cases seborrhoeic dermatitis can be associated with alcohol misuse and, therefore, advice on alcohol restriction may be useful. HIV infection should be considered in patients with poorly controlled, active seborrhoeic dermatitis.

20 Seborrhoeic dermatitis. Involvement of the nasolabial folds with erythema and scaling is typical.

21 Seborrhoeic dermatitis. The forehead and eyebrows are common sites of involvement.

22 Seborrhoeic dermatitis. In severe, acute seborrhoeic dermatitis widespread involvement can occur on the torso. This patient was found to have HIV infection.
Chronic actinic dermatitis

Chronic actinic dermatitis (CAD) is a rare form of eczema induced by sunlight and is seen most commonly in middle-aged or elderly men. Although affected individuals have abnormal photosensitivity the causative role of sunlight is sometimes not immediately apparent to the patient. The dermatitis develops on exposed skin, occurring maximally during the spring and summer months when the ultraviolet component of sunshine is high. Most patients are sensitive to UVB radiation but also to ultraviolet A (UVA) and, in some cases, visible light. Despite its name, CAD can present acutely as a clinically striking and highly symptomatic dermatosis.

CLINICAL FEATURES
CAD is characterized by eczema involving the light-exposed sites: face, neck, upper chest, and back of hands (23, 24, 25). Usually the limits of clothing (collar and cuffs) produce a clear cut-off with the covered skin being unaffected (24, 25). Involved skin shows confluent eczema although there will be sparing in light-protected areas such as finger webs, upper eyelids, and the submental zone. Although the signs may suggest a chronic form of eczema (lichenification, induration, and scaling), CAD can present as a subacute or acute dermatitis with erythema, swelling, weeping, and secondary infection. In severe cases the eczema may spread from exposed skin to covered sites and lead to erythroderma (see Erythroderma, p. xx).

DIFFERENTIAL DIAGNOSIS
- Allergic contact dermatitis (p. 18, eczema in a localized area or unusual distribution).
- Airborne allergic contact dermatitis (p. 18, eczema localized to the exposed skin).
- Photosensitive drug eruption (p. 224, erythema on exposed skin triggered by a photosensitizing drug).
- Photoaggravated AD (p. 8, atopic dermatitis exacerbated by sunlight).
- Photoaggravated seborrhoeic dermatitis (p. 20, seborrhoeic dermatitis exacerbated by sunlight).

SYSTEMIC COMPLICATIONS AND ASSOCIATIONS
Complications
- Regional lymphadenopathy.
- Erythroderma (see p. 51).

Associations
- HIV infection (rare).

INVESTIGATIONS
- The diagnosis is usually made clinically.
- Skin swab for bacteriology (if secondary bacterial infection is suspected).
- Blood count, basic chemistry, liver function tests (needed if considering systemic immunosuppressant therapy or if the patient is systemically unwell).
- Antinuclear antibody (ANA), -Ro and -La antibodies (to exclude lupus-related photosensitivity).
- Diagnostic light tests: monochromator irradiation testing and solar simulator testing (these tests, performed in a specialist photobiology unit, will confirm the diagnosis and identify the provoking wavelengths of light).

23 Chronic actinic dermatitis. There is eczema involving all the exposed skin of the face and scalp with a cut-off at the neck, caused by the patient’s collar.
• Patch tests (many patients with CAD also have positive patch tests, especially to plant allergens, suggesting concomitant allergic contact dermatitis).

IMMEDIATE MANAGEMENT
Topical therapy
• Sun avoidance and use of photoprotective clothing, including a broad-brimmed hat.
• Use of high protection factor topical sunscreen containing effective UVA and UVB filters.
• General emollient therapy.
• Corticosteroid ointment twice per day (use for a restricted period):
  - face: moderately potent.
  - neck, arms, hands: potent.

Systemic therapy
• For secondary bacterial infection give antistaphylococcal oral antibiotics (e.g. flucloxacillin 500 mg four times per day for 7 days).
• A sedating oral antihistamine at bedtime (e.g. hydroxyzine 25 or 50 mg) will relieve itch and improve sleep.

• A short, reducing course of oral corticosteroids can be helpful to induce remission (e.g. prednisolone 20–30 mg/day and reducing by 5 mg every 5th day).

LONG-TERM MANAGEMENT ISSUES
Ongoing attention to photoprotection is essential and CAD patients usually need long-term follow-up by a dermatologist. Patients who are sensitive to the longer wavelengths of UVA and visible light may require special filters on the glass of windows at home and in their car. CAD tends to relapse each spring and summer and therefore active therapy may need to be reinstated seasonally. Topical tacrolimus ointment or pimecrolimus cream may reduce requirements for topical or oral corticosteroid. Patients with severe CAD or those who are extremely photosensitive will require long-term systemic immunosuppression with azathioprine or ciclosporin. If patch tests are positive patients should be encouraged to avoid relevant allergens. There is a rare association between CAD and HIV infection.