How to Treat

**PULL-OUT SECTION**

ATOPIC eczema is the most common inflammatory skin disease in Australia. While eczema most often first appears in early childhood, it can begin at any age. Asthma in the mother is the strongest predictor of eczema in an infant. Dry skin, intense pruritus, erythema, scaling and weeping are prominent features. Eczema usually runs a relapsing and remitting course.

Eighty per cent of cases of atopic dermatitis (ATOD) have elevations of the total serum immunoglobulin E (IgE) concentration. IgE-mediated sensitisation to food and environmental allergens exacerbates eczema and contributes to future development of several allergic mucosal disorders, including food allergy, asthma and allergic rhinitis. This has been called the atopic march.

Atopic eczema can significantly impair the quality of life of the individual and family and is a significant health burden in Australia. Nocturnal itch can lead to sleep disturbance and poor school performance in affected children. This article provides an update on the pathogenesis, clinical features and management of eczema.

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**References:**

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**THE AUTHORS**

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**INSIDE**

Epidemiology
Pathogenesis
Diagnosis
Clinical types of eczema
Management of atopic eczema
Case study

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How To Treat – Atopic eczema

Epidemiology

ATOPIC eczema affects 15-30% of children and 2-10% of adults worldwide. The incidence appears to be rising globally. The reason for this is uncertain; however, a number of hypotheses, including increased urban living and improved hygiene, have been postulated.

In 85% of affected individuals, eczema starts in infancy — generally around three months of age. Atopic eczema may remit in childhood only to reappear in adolescence or early adult life. However, in many patients, atopic eczema continues throughout their lives.

Pathogenesis

ATOPIC dermatitis is a complex polygenic trait. Concordance between identical twins was 86% and 21% in dizygotic twins in a study of the Danish Twin Registry.

Several susceptibility loci for atopic dermatitis and psoriasis have been identified. These gene loci are involved in skin barrier formation, inflammatory pathways, asthma and, interestingly, psoriasis.

The gene association with psoriasis is interesting because, while many people with psoriasis have a past history of childhood atopic dermatitis, it is rare to see atopic dermatitis and psoriasis together in the same patient at the same time.

TRANSDERMAL water loss causes the skin dryness, which is a hallmark of eczema. Loss of filaggrin and epidermal skin barrier function leads to penetration of environmental allergens and microbes and results in activation of the innate and acquired immune systems, production of abnormal cytokines and inflammation.

Acquired immunity to skin allergens contributes to future development of asthma, food allergy and allergic rhinitis, potentially leading to the atopic march.

Although the discovery of the mutation of filaggrin protein is a major breakthrough in the understanding of eczema pathogenesis, about 40% of patients with eczema do not have filaggrin mutations. Filaggrin mutation is the primary cause of eczema. However, other factors also play a role in the pathogenesis of atopic eczema.

Diagnosis

WHILE spongiosis is a pathognomonic histological characteristic of eczema on skin biopsy, the diagnosis of ATOD is usually made on clinical grounds. Skin biopsy is only rarely required. Intense pruritus is the most characteristic feature of eczema. Poorly defined erythema, scaling and exudation are seen in early lesions of eczema. Dry skin is seen in most patients.

Plaque psoriasis is an important differential diagnosis. In contrast to eczema, plaques of psoriasis have well-defined borders, thicker scales and little or no itch.

Golden-yellow crusting is seen when eczema is secondarily infected with Staphylococcus aureus, hence the name golden staph. Lichenification of affected skin manifests as skin thickening with exaggerated normal skin markings, and it occurs as a result of chronic rubbing. Post-inflammatory hypo- or hyperpigmentation is common in chronic eczema.

Patch testing to exclude associated allergic contact dermatitis may be necessary if eczema has sudden and severe exacerbations, occurs in unusual body sites or is made worse by topical steroids and moisturisers. Allergy testing for common environmental allergens — such as house dust mites, moulds and grass pollens — is rarely useful because false positives are common and desensitisation or avoidance of allergens is not usually possible.

The role of food allergy in the pathogenesis of eczema is limited, and testing should be restricted to patients with a clear history of diet-exacerbated eczema. Dietary modifications should only occur after a positive food challenge. Routine avoidance of food is not recommended, and no specific diet that alleviates atopic eczema has been discovered. Unnecessary restrictions of foods can lead to dietary deficiency, especially in children.

Clinical types of eczema

Infantile eczema

INFANTILE eczema generally starts around two months of age. Typical lesions of infantile eczema are scaly, erythematous patches seen on the cheeks. Lesions can later extend to the scalp, neck and limbs. The patches of eczema may become crusted and weepy, indicating that they have become secondarily infected with bacteria. Affected babies are generally irritable and sleep poorly.

Childhood eczema

Classic lesions of childhood eczema are seen on the elbows, knees, wrist flexures, ankles and neck. Itching can be intense, and lichenification is common. Severe itching can disturb sleep, cause growth retardation and lead to poor school performance in affected children (see figure 1).

Discoid or nummular eczema

Discoid or nummular eczema often begins on the lower legs and arms. A single patch of eczema can precede secondary lesions by many weeks. In our experience, active treatment of the initial lesion prevents development of secondary lesions.

The typical lesion of discoid eczema is a discrete, circular and erythematous patch with well-defined borders (see figure 2). This can lead to confusion with psoriasis, tinea corporis; however, the surface of discoid eczema tends to be studded with tiny vesicles. Pruritus is usually intense. If left untreated, discoid eczema can lead to multiple lesions distributed widely over the body. With treatment, the lesions generally heal without trace, but post-inflammatory hypo- or hyperpigmentation is seen in patients with darker skin types.

Gravitational or venous eczema

Venous or gravitational eczema is typically seen in the lower legs of middle-aged or elderly patients with venous insufficiency. A history of DVT, chronic venous insufficiency or chronic leg swelling increases the incidence of gravitational eczema.

Gravitational eczema is characterised by haemosiderin pigmentation of the surrounding skin, lipodermatosclerosis (thickening of skin and narrowing of legs, giving a champagne bottle appearance) and atrophic blanche (see figures 3a and 3b). Gravitational eczema can be complicated by cellulitis of the lower leg and venous ulceration. Contact allergy to topical creams is common in this condition. Compression hosiery and bandaging are critical in the management of gravitational eczema, and resolution rarely occurs with topical steroids alone. Patch testing should be considered in refractory cases to
ECZEMA can be exacerbated by known and unknown triggers. It is desirable to avoid known triggers and, in particular, activities of daily living that are likely to aggravate ATOD.

General measures
- Long, hot showers can paradoxically lead to skin dryness.
- Soak, bubble bath and detergents also lead to dryness.
- Coarse fabrics can irritate the skin and produce itch. Cotton or fine merino wool is favoured.
- Ick is accentuated by heat. Overheating during the day occurs when ductal heating is turned up above 20°C. Doonas may lead to overheating at night, so sheets and blankets are favoured.

Skin infection
Secondary bacterial infection with S. aureus is common. Infected eczema becomes weepy and crusted. Even in the absence of visible weeping, colonisation of the skin with S. aureus is thought to exacerbate atopic eczema. Recurrent boils and folliculitis may also occur.

Eczema herpeticum is caused by secondary infection with HSV and requires urgent treatment. Patients with eczema herpeticum present with fever, malaise and sudden worsening of their eczema, with umbilicated vesicles, blisters and punched-out ulceration.

Bleach baths
Bleach baths (see table 2) are helpful in both clinically infected eczema as well as apparently uninfected eczema. They reduce secondary bacterial infection and staphylococcal colonisation. Bleach baths are recommended in conjunction with topical moisturisers and other forms of eczema treatments, such as topical steroids.

Xerosis and skin dryness
Transdermal water loss is increased in eczema and leads to dryness of skin. Skin dryness predisposes to itch. Scratching further damages the epidermis and exacerbates atopic dermatitis. Moisturisers/ointments play an important part of eczema management. Emollients reduce transdermal water loss, hydrate the stratum corneum

<table>
<thead>
<tr>
<th>Table 1: Clinical features of different regional eczemas</th>
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<tbody>
<tr>
<td><strong>Regional eczema</strong></td>
</tr>
<tr>
<td>Eyelid eczema</td>
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<td>Nipple eczema</td>
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<tr>
<td>Hand eczema</td>
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<table>
<thead>
<tr>
<th>Differential diagnoses</th>
<th>Management</th>
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<tbody>
<tr>
<td>Contact dermatitis</td>
<td>Patch test and avoidance of possible allergens; topical steroids; ophthalmology referral if there is eyelid involvement</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>Topical steroids are usually effective; secondary bacterial infections should be treated on the basis of culture and sensitivities</td>
</tr>
<tr>
<td>Seborrhoeic dermatitis</td>
<td></td>
</tr>
<tr>
<td>Psoriasis</td>
<td></td>
</tr>
<tr>
<td>Occupational contact dermatitis</td>
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<tr>
<th>Table 2: Instructions for bleach bath</th>
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<tbody>
<tr>
<td>• Add 100mL of bleach (8% sodium hypochlorite) to a full tub (80L) of warm water; use household bleach and not concentrated bleach; two tablespoons of bleach (10mL) should be added to a baby bath (10L)</td>
</tr>
<tr>
<td>• Soak from the neck down for about 10 minutes</td>
</tr>
<tr>
<td>• Gently pat dry with a towel; avoid excessive rubbing</td>
</tr>
<tr>
<td>• Immediately apply moisturiser/steroid ointment as directed</td>
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Even in the absence of visible weeping, colonisation of the skin with S. aureus is thought to exacerbate atopic eczema.

**Figure 4:** Clear vesicles in pompholyx.

**Figure 5:** Characteristic crazy paving appearance of the shin in eczema craquele.
**How To Treat – Atopic eczema**

and improve skin barrier function. They are recommended for both induction of remission and maintenance therapy.

Patients are advised to use the thickest moisturiser they can tolerate. Daily emollient use reduces the requirement for topical steroids. The optimal time to apply a moisturiser is immediately after bathing, petrolatum and ceramide-dominant lipids in the emollient reduce transdermal water loss and affect skin barrier repair. Emollients are available as creams, ointments, lotions, soap substitutes and bath additives (see table 3).

**Wet wraps**

Wet wraps are helpful in the most severe cases of eczema and used only for a few days until the redness and swelling settle. They are applied over emollients and/or topical steroids. Wet wraps act by cooling the skin and relieving itching, enhancing the moisturising effects of emollients and increasing absorption of topical steroids. The bandaging also prevents itching and breaks the itch-scratch cycle. The basic steps in wet wrapping include the following:

- The patient may first soak in a bath with bath oil or emollient solution.
- An emollient and/or steroid cream (as prescribed by the doctor) is liberally applied to the area.
- Bandages soaked in warm water are wrapped over the top of the creams.
- Dry bandages are placed over the top of the wet bandages to protect clothing.

Wet wraps are time-consuming and may be uncomfortable for the patient and are generally reserved for use in the hospital setting in cases of severe eczema.

**Topical corticosteroids**

Topical corticosteroids are the most commonly used topical treatment in eczema. Steroid phobia and steroid underuse are bigger risks than overuse. Pharmacists have been encouraged to desist from automatic labelling of topical steroids with the words ‘use sparingly’.

Steroid phobia and steroid underuse are bigger risks than overuse. Pharmacists have been encouraged to desist from automatic labelling of topical steroids with the words ‘use sparingly’.

**Preparation for wet wrap treatment.** Source: NAPRA bit.ly/1YL94AD

Table 3: Different emollient preparations available in Australia

<table>
<thead>
<tr>
<th>Options</th>
<th>Examples of products</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lotions</strong></td>
<td>Aveeno Cetaphil Dermal 500 (with antimicrobials) E45 Eucerin lotion (3% and 10% urea) Keri Oilatum QV Vaseline Dermacare lotion Aveeno Cetaphil Eucerin (5% urea) Neutrogena dermatological cream QV 50/50 white soft paraffin/liquid Emulsifying ointment</td>
</tr>
<tr>
<td><strong>Creams</strong></td>
<td>Aveeno Colloidal Aveeno bath oil Eucerin Bath Therapy Emulsiderm Emollient (with antimicrobials) Oilatum fragrance-free Oilatum Plus (with antimicrobials) QV Dove soap Simple soap Aveeno cleansing bar Cetaphil wash Cetaphil cleansing bar Emulsifying ointment Eucerin Dry Skin Relief Wash (5% urea) Oilatum soap bar</td>
</tr>
</tbody>
</table>

Soap substitutes

Soap is alkaline and very drying and irritating to eczematous skin; soap substitutes and bath additives (see table 4).

- An emollient and/or steroid cream (as prescribed by the doctor) is liberally applied to the area.
- Bandages soaked in warm water are wrapped over the top of the creams.
- Bath oils

Bath oils and warm water clean and hydrate the skin; the preferred bath oil for individual patients needs to be found by trial and error.

**Soap substitutes**

Soap is alkaline and very drying and irritating to eczematous skin; soap substitutes are very effective at cleaning the skin and are less irritating.

**Ointments**

Ointments contain more oil than water; as bacteria do not grow as well in oil as in water, most ointments do not require the addition of preservatives; ointments are suitable for people with very dry skin and for people who react to preservatives; some patients may find them too greasy for everyday use; they are effective at holding water in the skin and are useful for very dry and thickened skin; ointments should be avoided on weeping eczema because occlusion increases bacterial colonisation.

**Bath oils**

Bath oils are warm water clean and hydrate the skin; the preferred bath oil for individual patients needs to be found by trial and error.

**Topical calcineurin inhibitors**

Topical calcineurin inhibitors, such as pimecrolimus cream (Elidel) and tacrolimus ointment, are useful in mild to moderate eczema. They have anti-inflammatory properties. They do not thin the skin, produce telangiectasia or aggravate rosacea and are most useful in eczema on facial and flexural areas. They are also used intermittently as maintenance therapy to prevent eczema relapse. A burning sensation, feeling of warmth and itching are the limiting factors in using these creams on sensitive area like the face and flexures.

**UV therapy**

Narrowband UVB phototherapy (NBUVB) is used by dermatologists for eczema not controlled by topical therapy alone. NBUVB generally requires three treatments a week. Twenty-five to 30 treatments are generally required before improvement is seen, and atopic eczema may be exacerbated on initiation of therapy.

**Preparation for wet wrap treatment.** Source: NAPRA bit.ly/1YL94AD

**Wet wrap head.** Source: NAPRA bit.ly/1YL94AD

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11 September 2015 | Australian Doctor | 39
Phototherapy can be used in combination with topical therapies but is only reluctantly used in combination with methotrexate or cyclosporine because of the increased risk of skin cancers.

**Oral medications**

Patients not controlled by topical therapy or UVB may require oral immunosuppressive medications. Fewer than 10% of patients with atopic eczema should ever require systemic therapy.

Before commencing oral therapy, doctors should consider the factors listed in table 5. Short courses of oral steroids may be required in acute flare-ups. Long-term steroid therapy should be avoided because of cumulative toxicity.

Azathioprine, cyclosporine, methotrexate and mycophenolate mofetil can all be used. These medications all require regular monitoring and blood tests.

**Emerging treatments**

Omalizumab is a monoclonal anti-IgE antibody currently TGA-approved for the treatment of asthma. The usefulness of omalizumab in the treatment of atopic eczema in the context of high IgE is still being evaluated.

Biologic drugs targeting IL-4 and IL-13 have shown promising results in severe eczema in phase I and phase II clinical trials and are currently being evaluated in phase III clinical trials.

Over the past 10 years, biologic treatments have proven to be very effective in the management of severe eczema, which increases proinflammatory cytokines, have been found in inflammatory disorders like atopic eczema and asthma. Apremilast, an oral phosphodiesterase type 4 (PDE4) inhibitor, has been shown to improve atopic eczema — measured by improvement of Dermatology Life Quality Index (DLQI), Eczema Area and Severity Index (EASI) and improvement of pruritus in small group studies.

<table>
<thead>
<tr>
<th>Table 4: Common corticosteroid preparation available in Australia</th>
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<tbody>
<tr>
<td><strong>Class 1</strong> Very potent or super potent (up to 600 times as potent as hydrocortisone)</td>
</tr>
<tr>
<td>• Clobetasol dipropionate (needs to be compounded)</td>
</tr>
<tr>
<td>• Betamethasone dipropionate (Diprosone OV cream/ointment)</td>
</tr>
<tr>
<td><strong>Class 2</strong> Potent (100-150 times as potent as hydrocortisone)</td>
</tr>
<tr>
<td>• Betamethasone valerate (Betnovate lotion/C cream/C ointment)</td>
</tr>
<tr>
<td>• Betamethasone dipropionate (Diprosone cream/ointment)</td>
</tr>
<tr>
<td>• Hydrocortisone 17</td>
</tr>
<tr>
<td>• Mometasone furoate (Mometasone cream/ointment, Eloson cream/lotion/ointment)</td>
</tr>
<tr>
<td><strong>Class 3</strong> Moderate (2-25 times as potent as hydrocortisone)</td>
</tr>
<tr>
<td>• Clobetasone butyrate (Eumovate cream)</td>
</tr>
<tr>
<td>• Triamcinolone acetonide (Aristocort cream/ointment, Kenacomb)</td>
</tr>
<tr>
<td><strong>Class 4</strong> Mild</td>
</tr>
<tr>
<td>• Hydrocortisone (DermAid cream/soft cream, Sigmacort)</td>
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<tr>
<th>Table 5: Factors to consider in the management of unresponsive eczema before commencing systemic therapy</th>
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</thead>
<tbody>
<tr>
<td>1. Is the diagnosis correct? Fungal skin scrapings may be required to exclude tinea incognito; examination of the finger webs should be performed to look for scabetic burrows; a skin biopsy may be required to exclude a drug eruption, psoriasis, cutaneous lymphoma or other clinical mimics of ATOD</td>
</tr>
<tr>
<td>2. Is there evidence of secondary bacterial or viral infections?</td>
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<tr>
<td>3. Are there any environmental factors that need to be addressed, such as overheating; long, hot showers; or use of soap and bubble bath?</td>
</tr>
<tr>
<td>4. Is there associated contact allergy to topical steroids, preservatives, cosmetics or emollients?</td>
</tr>
<tr>
<td>5. Has the patient been adhering to the treatment instructions provided or underusing topical corticosteroids?</td>
</tr>
</tbody>
</table>

References

How To Treat – Atopic eczema

Case study

TINA, aged 28, presents with a very itchy and extensive erythematous, scaly rash that has photo-exacerbated over the past two weeks. She has had a recurrent itchy skin rash over the past five years. She has a severe generalised eczematous rash on presentation. The eczema has also affected her face, with a significant impact on her everyday life: she has experienced poor sleep and lack of enthusiasm for work. Use of soap and exposure to heat have worsened the eczema. However, there is no evidence of contact dermatitis or food allergy.

In order to exclude photosensitive dermatitis, a biopsy and screening blood tests are done at the initial consultation. Pending histology and blood results, she is prescribed topical triamcinolone ointment daily for a week, after a bleach bath. After one week on the above medications, her eczema has only partly improved.

The biopsy confirms eczema, and the blood results are normal. Tina commences NBUBV three times a week with a short course of oral prednisolone. Initially, she responds positively to the treatment. However, she complains of increased itching and erythema after NBUBV once the tailing dose of prednisolone is over. Therefore, NBUBV therapy is discontinued. Tina does not want to take oral prednisolone and topical steroids because they cause purpura on her skin. She commences azathioprine after screening blood tests. Tina shows significant improvement at the six-week review, and the dose of azathioprine is increased to 100mg daily. Pimecrolimus 0.1% cream is prescribed for facial eczema. Tina starts responding to azathioprine, and the treatments are continued until her next review.

Conclusion

MANAGEMENT of ATOD is a challenge for both GPs and dermatologists. The treatment of moderate to severe atopic eczema has not changed over the past 10 years, even though safe and effective biologic drugs have been discovered for the treatment of psoriasis.

Topical therapies — such as topical steroids, calcineurin inhibitors, moisturisers and NBUBV — are safe and effective treatment modalities for mild to moderate eczema.

However, in moderate to severe eczema, systemic medications — like oral steroids, cyclosporine, methotrexate and mycophenolate mofetil — may be required to control the disease.

Use of these medications is limited by their immunosuppressive effects and systemic side effects, such as renal toxicity with cyclosporine, hepatic toxicity with methotrexate and bone marrow toxicity with azathioprine and mycophenolate mofetil.

Therefore, finding a safe and effective targeted therapy for eczema will be a welcome development.

INSTRUCTIONS

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points.

We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

GO ONLINE TO COMPLETE THE QUIZ


How to Treat Quiz

Atopic eczema — 11 September 2015

1. Which TWO statements regarding eczema are correct? a) Eczema in the mother is the strongest predictor of eczema in an infant.
   b) Atopic eczema is a complex polygenic trait.
   c) Eczema always first appears in early childhood.
   d) Eczema usually runs a relapsing and remitting course.

2. Which THREE are prominent features of eczema? a) Intense pruritus
   b) Erythema
   c) Blistering
   d) Scaling

3. Which THREE statements regarding the pathogenesis of eczema are correct? a) Atopic dermatitis (ATOD) is a complex polygenic trait.
   b) ATOD and psoriasis commonly occur together in the same patient at the same time.
   c) Filaggrin is a structural protein in the cornocytes that is essential for the integrity of epidermis and epidermal barrier function.
   d) The epidermal barrier restricts transepidermal water loss and prevents allergens, microbes and irritants from entering the viable epidermis.

4. Which TWO statements regarding the diagnosis of eczema are correct? a) Plaque psoriasis — with well-defined borders, thicker scales and significant itch — is an important differential diagnosis.
   b) Post-inflammatory hypo- or hyperpigmentation is uncommon in chronic eczema.
   c) The diagnosis of ATOD is usually made on clinical grounds, and skin biopsy is only rarely required.
   d) Lichenification of affected skin manifests as skin thickening with exaggeration of normal skin markings, and it occurs as a result of chronic rubbing.

5. Which TWO statements regarding allergy testing in eczema are correct? a) Patch testing to exclude associated allergic contact dermatitis may be necessary if eczema has sudden and severe exacerbations.
   b) Allergy testing for house dust mites, moulds and grass pollens is the first-line investigation, usually yielding information on the allergen to be avoided.
   c) Dietary modifications should be instituted early, based on patient/carer reports of foods stuffs exacerbating eczema.
   d) Routine avoidance of food is not recommended, and no specific diet that alleviates atopic eczema has been discovered.

6. Which THREE statements regarding the clinical types of eczema are correct? a) Typical lesions of infantile eczema are scaly, erythematous patches seen on the cheeks, which may later extend to the scalp, neck and limbs.
   b) Classic lesions of childhood eczema are seen on the cheeks, which may later extend to the scalp, neck and limbs.
   c) Discoid or nummular eczema often begins on the lower legs and arms.
   d) The typical lesion of discoid eczema is a discrete, circular and erythematous patch with well-defined borders.

7. Which TWO statements regarding the clinical types of eczema are correct? a) A history of DVT, chronic venous insufficiency or chronic leg swelling increases the incidence of gravitational eczema.
   b) Venous or gravitational eczema is typically seen in the lower legs of middle-aged or elderly patients with arterial insufficiency.
   c) Gravitational eczema is characterised by haemosiderin pigmentation of the surrounding skin, lipodermatosclerosis and atrophie blanche.
   d) Long-term topical steroids are the mainstay of treatment in venous/gravitational eczema.

8. Which THREE features are associated with eczema crurisae? a) Intertrigo
   b) Hyperthyroidism
   c) Malnutrition
   d) Retinoid therapy

9. Which TWO statements regarding the management of eczema are correct? a) Long, hot showers can paradoxically lead to skin dryness.
   b) Itch is soothed by heat.
   c) Blistered baths are recommended in conjunction with topical moisturisers and other forms of eczema treatments, such as topical steroids.
   d) The optimal time to apply a moisturiser is immediately prior to dressing in the morning.

10. Which THREE statements regarding the management of eczema are correct? a) Topical corticosteroids are the most commonly used topical treatment in eczema.
    b) Mild-to-potency steroid preparations are preferably used on the face and body flexures.
    c) Creams are preferable for weeping and infected eczema.
    d) Ointments are best used in hairy areas.