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Atopic eczema

Introduction

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 ing 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

tal 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

vidual 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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THE AUTHORS



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impair the quality of life of the indipruritus, erythema, scaling and weepsensitisation to food and environmen-

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TOO MANY PATIENTS ARE FALLING THROUGH THE CRACKS.[™]



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tified.

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 Regis-

atopic dermatitis have been iden-

skin barrier formation, inflam-

matory pathways, asthma and,

The gene association with pso-

riasis is interesting because, while

many people with psoriasis have

a past history of childhood atopic

interestingly, psoriasis.

Several susceptibility loci for

These gene loci are involved in

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.

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

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



Figure 1: Flexural eczema in childhood.



dermatitis, it is rare to see atopic dermatitis and psoriasis together in the same patient at the same time.

The ATOD1 gene codes for the protein filaggrin, and filaggrin mutations are strongly associated with early-onset atopic eczema.

Filaggrin is a structural protein in the corneocytes that is essential for the integrity of epidermis and epidermal barrier function.

The epidermal barrier restricts transepidermal water loss and prevents allergens, microbes and irritants from permeating across the stratum corneum and entering the viable epidermis.

Disruption of epidermal barrier function caused by loss of filaggrin protein results in increased transepidermal water loss.

Transepidermal 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. 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.

Acquired immunity to skin

with *Staphylococcus aureus*, hence the name golden staph. Lichenification of affected skin manifests as skin thickening with exaggeration of 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.





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 welldefined borders (see figure 2). This can lead to confusion with psoriasis or 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



Figure 2: Discoid eczema with well-defined borders .

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 character-

ised by haemosiderin pigmentation

Figure 3b: Lipodermatosclerosis with champagne bottle appearance in varicose eczema.

of the surrounding skin, lipodermatosclerosis (thickening of skin and narrowing of legs, giving a champagne bottle appearance) and atrophie 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

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identify contact allergy to topical medications.

Pompholyx or dyshidrotic eczema

Dyshidrotic eczema presents as very itchy, recurrent vesicles in the palms and soles. The vesicles of pompholyx are clear and deep-seated (see figure 4). Within a few days, the vesicles burst, and the overlying skin peels off. The clear contents of the vesicle distinguish pompholyx from palmer and plantar psoriasis, which produces yellow pustules. Unilateral hand vesicles raise the possibility of tinea manus, and skin scrapings for microbiological examination should be performed.

Asteatotic eczema or eczema craquele

This type of eczema is most common in the elderly and occurs most commonly on the shins on a background of very dry skin. Eczema craquele is known to be associated with ichthyosis, hypothyroidism, malnutrition and retinoid therapy. Excessive soap use and low humidity make eczema craquele worse. The shins have a characteristic crazy-paving appearance (network of diamond-shaped skin plates separated from each other) (see figure 5). Secondary bacterial infection may make eczema craquele weepy.

Avoiding soap, overheating of the skin (which exacerbates itch) and liberal use of moisturisers are the most important steps in the management of eczema craquele. A mild to moderate topical steroid can be used to reduce inflammation and itch.

Regional eczemas

Table 1 summarises the clinical features of different regional eczemas.



Figure 4: Clear vesicles in pompholyx.

The following minor physical fea-

tures are commonly seen in assoc-

iation with eczema. Their presence

• Dennie-Morgan fold (infraorbital

Cutaneous stigmata of eczema

favours a diagnosis of eczema:

fold)

Xerosis

- Pityriasis alba
- Keratosis pilaris
- White dermographism
- Dirty neck
- Hyperlinearity of the palms or soles



Figure 5: Characteristic crazy paving appearance of the shin in eczema craquele.

| Table 1: Clinical features of different regional eczemas | | | |
|--|---|--|--|
| Regional eczema | Features | Differential diagnoses | Management |
| Eyelid eczema (see figure 6) | Can have associated allergic contact dermatitis; this should be suspected if no atopic eczema elsewhere; causes rubbing and scratching of the eyelids; can cause blepharitis | Contact dermatitis Psoriasis Seborrhoeic dermatitis | Patch test and avoidance of possible allergens; topical steroids; ophthalmology referral if there is eyelid involvement |
| Nipple eczema (see figure 7) | Common in nursing mothers; nipple may be the only area affected; can present with fissuring, oozing and crusting; it usually involves areolar and surrounding skin, and the base of the nipple is usually less affected; secondary infection with Candida albicans is common in breastfeeding mothers; if unilateral and poor response to topical steroid, a biopsy should be done to exclude Paget's disease | Joggers nipple Paget's disease | Topical steroids are usually effective; secondary bacterial infections should be treated on the basis of culture and sensitivities |
| Hand eczema | May be vesicular, bullous or hyperkeratotic; vesicular eczema is intensely pruritic; single or multilocular vesicles develop on the sides of fingers and palms; lesions are bilateral and symmetrical Hyperkeratotic eczema may produce painful fissures; eczema involving the proximal nail folds can lead to dystrophy of nails and nail pitting | Pompholyx Ide reaction Tinea Allergic contact dermatitis Occupational contact dermatitis Psoriasis | |

Management of atopic eczema

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.
- Itch is accentuated by heat. Overheating during the day occurs when ductal heating is turned up above 20°C. Doonas may lead to



Table 2: Instructions for bleach bath

- Add 100mL of bleach (6% 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)
- Soak from the neck down for about 10 minutes
- Gently pat dry with a towel; avoid excessive rubbing
- Immediately apply moisturiser/steroid ointment as directed

Even in the absence of visible weeping, colonisation of the skin with *S. aureus* is thought to exacerbate atopic eczema. 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 staphylococcus colonisation. Bleach baths are recommended in conjunction with topical moisturisers and other forms of eczema treatments, such as topical steroids.

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

Xerosis and skin dryness

Transepidermal 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/emollients play an important part of eczema management. Emollients reduce transepidermal water loss, hydrate the stratum corneum *cont'd page 39*

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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 transepidermal 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 itchscratch 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'. Patients require specific instruction on the dose of topical steroid to apply. The fingertip unit (FTU) is one method of prescribing dose.

One FTU is the amount of topical steroid that is squeezed out from a standard tube along an adult's fingertip. One FTU is about 1g and is enough to treat an area of skin twice the size of the flat of an adult's hand with the fingers together.

The risk of potential unwanted side effects from topical steroid use

| Louons | Avee |
|---|--------|
| Lotions contain mainly water mixed with a low concentration of oil; they are | Ceta |
| easy to spread and most useful in hairy areas; lotions are least effective at | Derm |
| moisturising dry skin. | E45 |
| | Euce |
| | Keri |
| | Oilatu |
| | QV |
| | Vasel |
| Creams | Avee |
| Creams contain a mixture of water with a higher concentration of oil than | Ceta |
| lotions: the concentration of oil is generally between 10% and 30%; creams | Euce |
| are loss greasy than gintmenter proper stives in greams can consition and | Noutr |

Table 3: Different emollient preparations available in Australia

are less greasy than ointments; preservatives in creams can sensitise and induce contact allergic dermatitis in some individuals.

Ointments

Options

Lation

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 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.



Aveeno Colloidal Aveeno (bath oil) Eucerin Bath Therapy Emulsiderm Emollient (with antimicrobials) Oilatum fragrance-free Oilatum Plus (with antimicrobials) QV Dove soap Simple soap

Avène cleansing bar Cetaphil wash Cetaphil cleansing bar Emulsifying ointment Eucerin Dry Skin Relief Wash (5% urea) Oilatum soap bar



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'.

Ointments are preferred to creams in dry hyperkeratotic eczema but are avoided in the flexures and on hairy skin. Creams are preferable for weeping and infected eczema. Lotions are best used in hairy areas.

Potential side effects from topical corticosteroid use include skin thinning (atrophy) and stretch marks, purpura, ecchymosis and increased susceptibility to skin infection. If eczema worsens or fails to respond to topical steroids, the possibility of contact allergy should be suspected. Contact allergy to preservatives or the corticosteroid itself can be diagnosed by patch testing.

Non-steroidal topical immunomodulators

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.

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can be minimised by prescribing the lowest strength steroid in adequate quantity for the required time duration. Once eczema is under control, intermittent use can be recommended or a less potent topical steroid can be prescribed.

There are many topical steroid preparations available in Australia, and they differ in potency and formulation (see table 4). Depending on the type, site and severity of eczema, different potency formulations are used. Mild-potency preparations are preferably used on the face and body flexures. The legs, palms and soles usually need moderate-to-strong-potency steroids.



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.

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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 TGAapproved 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 curr-

| Table 4: Common corticosteroid preparation available in Australia | | |
|---|--|--|
| Class 1 | Very potent or super potent (up to 600 times as potent as hydrocortisone) Clobetasol dipropionate (needs to be compounded) Betamethasone dipropionate (Diprosone OV cream/ointment) | |
| Class 2 | Potent (100-150 times as potent as hydrocortisone) Betamethasone valerate (Betnovate lotion/C cream/C ointment) Betamethasone dipropionate (Diprosone cream/ointment) Hydrocortisone 17 Mometasone furoate (Mometasone cream/ointment, Elocon cream/lotion/ointment) Methylprednisolone aceponate (Advantan cream/ointment) | |
| Class 3 | Moderate (2-25 times as potent as hydrocortisone) Clobetasone butyrate (Eumovate cream) Triamcinolone acetonide (Aristocort cream/ointment, Kenacomb) | |
| Class 4 | Mild • Hydrocortisone (DermAid cream/soft cream, Sigmacort) | |
| | | |

Table 5: Factors to consider in the management of unresponsive eczema before commencing systemic therapy

 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

2. Is there evidence of secondary bacterial or viral infections?

3. Are there any environmental factors that need to be addressed, such as overheating; long, hot showers; or use of soap and bubble bath?

4. Is there associated contact allergy to topical steroids, preservatives, cosmetics or emollients?

5. Has the patient been adhering to the treatment instructions provided or underusing topical corticosteroids?

ently being evaluated in phase III clinical trials.

Over the past 10 years, biologic treatments have proven to be very effective in the management of moderate to severe psoriasis for patients who have previously failed to respond to standard oral immunosuppressive medications. The recent emergence of new biologic drugs for severe atopic eczema is potentially a major therapeutic advance in eczema management.

Elevated levels of leukocyte phosphodiesterase type 4 (PGE4), 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.



Figure 6: Scaling and erythema around the eye in eyelid eczema.



Figure 7: Fissuring and scaling in nipple eczema in a man.

Tinea incognito. Source: The lotus carroll bit. ly/1WP1EBX

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Online resources

www.nlm.nih.gov/medlineplus

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www.dermnetnz.org



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Case study

TINA, aged 28, presents with a very itchy and extensive erythematous, scaly rash that has photoexacerbated 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 NBUVB three times a week with a short course of oral prednisolone. Initially, she responds positively to the treat-



ment. However, she complains of increased itching and erythema after NBUVB once the tailing dose of prednisolone is over. Therefore, NBUVB 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 NBUVB — 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.



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 the most common inflammatory skin disease in Australia.
- 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

- 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

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.

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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

treatment in venous/gravitational eczema.

8. Which THREE features are associated with eczema craquele?

- a) Ichthyosis
- 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
- a) Long, hot showers can paradoxically lead to skin dryness.
 b) Itab is soothed by heat
- b) Itch is soothed by heat.
- c) Bleach 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-potency steroid preparations are preferably used on the face and body flexures.

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corneocytes 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. be avoided.

c) Dietary modifications should be instituted early, based on patient/carer reports of foodstuffs exacerbating eczema.d) Routine avoidance of food is not recommended, and no specific diet 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

c) Creams are preferable for weeping and infected eczema.

d) Ointments are best used in hairy areas.



CPD QUIZ UPDATE

The RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2014-16 triennium. You can complete this online along with the quiz at **www.australiandoctor.com.au**. Because this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

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Next week's How to Treat discusses the diagnosis and management of septic shock. The systematic approach used is provided by the Emergency Life Support Course and may be adapted for use in any medical emergency. The author is **Adjunct Associate Professor Andrew Singer**, Australian National University Medical School; senior specialist in emergency and retrieval medicine, Canberra Hospital and Health Services; principal medical adviser, Australian Government Department of Health; Instructor, Emergency Life Support Course.