Over the last few decades the prevalence of atopic eczema has increased significantly, with prevalence rates the highest in preschool children (45–64%). Generally, there is little data available on the prevalence of the disease in South Africa. The ISAAC (International Study of Asthma and Allergies in Childhood) study reported a prevalence of 5–10% among Cape Town schoolchildren. Another recent study reported levels of dermatologist-diagnosed eczema among Xhosa children of 0.7%, 1.1% and 3.7% in rural, peri-urban and urban settings, respectively.

Nomenclature and classification
The World Allergy Organisation (WAO) has recommended that the umbrella term for local inflammation of the skin should be “dermatitis”. See Figure 1.

- Atopic refers to an inherited predisposition to produce immunoglobulin E (IgE) antibodies in response to natural exposure to minute quantities of environmental allergens, manifesting clinically with atopic diseases, one of which is eczema. Other atopic diseases include: food allergy, asthma, seasonal and persistent rhinitis and urticaria.
- Eczema, which can be atopic or non-atopic, refers to an aggregation of several skin conditions having common clinical characteristics and which involve a genetically determined skin barrier defect.
- Atopic eczema is the term applied to those cases of eczema in which the inflammatory response is triggered by immunoglobulin E. This accounts for more than 50% of cases of eczema, particularly in children and young adults.

Note: according to the WAO classification, the term “atopic dermatitis” should no longer be used.

Diagnosis
Atopic eczema cannot be diagnosed without an atopic immune response being confirmed; this is done by establishing, via skin prick or IgE specific allergen testing, whether or not the patient has elevated total IgE levels or specific IgE.
antibodies to environmental allergens. Also crucial to making an accurate diagnosis is a thorough history-taking about a family history of atopic diseases, typically food allergy, asthma, seasonal and persistent rhinitis or urticaria. The younger the child, the more likely the eczema is to be atopic. Some children and most adults present with eczema that is not atopic, as evidenced by an absence of a family history of atopic diseases, normal IgE levels and no documented specific IgE sensitivities.

Pathogenesis
The pathogenesis of eczema is complex; immune, genetic, infective and neuroendocrine factors, and their interaction with the environment, are involved. Eczema exacerbations are also associated with a multitude of factors, including:
• bacterial colonisation (*Staphylococcus aureus*)
• stress
• barrier disruption
• environmental exposure
• contact, inhalant or ingestant allergens
• sweating
• pollutants
• sensory irritants (wool)
• chemical irritants
• maternal ingestants (in breast milk)

Prevention
The cornerstone of prevention is the identification and avoidance of environmental factors, as these are known to trigger atopic eczema exacerbations. This would include both specific avoidance strategies for specific identified environmental factors (for e.g. house dust mites), as well as avoidance of non-specific irritants. The latter (for e.g. wool or chemicals in soaps) aggravate both atopic and non-atopic eczema. The recommended avoidance strategies for house dust mites include washing bedding at 60°C and using mite-impenetrable bedding and mattress covers. ²

Evidence suggests that intervention has been proved to be worthwhile in high-risk families. For example, exclusive breast-feeding for at least four months has been shown to prevent the development of atopic eczema. ³

Mothers unable to breast-feed should use a hypoallergenic, extensively hydrolysed formula (e.g. Alfare®, Nutramigen®) or at least a partially hydrolysed formula (e.g. Nan-HA® or Similac® HA). Although soy-protein based formula is indicated for babies who have a confirmed cow’s milk allergy, there is insufficient evidence to suggest that the automatic substitution of breast-milk with a soy-based formula will prevent allergic diseases.

Role of dietary intervention
While diet has no place in the management of non-atopic eczema, besides a general caution to avoid non-specific possible triggers for pruritis (such as preservatives in processed foods), younger eczema patients could benefit from allergic dietary intervention, since their disease is more likely to have an atopic component. Food allergy is much less common in adolescent and adult atopic eczema patients. There is a high prevalence (up to 80%) of food allergy in infants and young children with atopic eczema; in comparison, the prevalence of food allergy in the general paediatric population is only 4–5%.

Testing for food allergy is recommended as routine for infants and young children and can be done via skin-prick tests or IgE testing. Skin-prick tests are inexpensive, highly sensitive and provide quick results, but they are only moderately specific. IgE testing is cost-effective; usually egg, milk, peanut, wheat, fish and soya are screened, followed by specific testing for those allergens that screen positively.

Management
The management of atopic eczema includes three stages, viz general measures – avoidance of trigger factors, adjuvant measures and anti-inflammatory therapy (as summarised in Table 2).

General measures
The following may be beneficial:
• avoiding overheating and external irritants
• keeping the skin covered with clothing to reduce exposure to irritants and trauma from scratching
• avoiding skin care products that cause irritation, e.g. astringents and soaps
• avoiding wool clothing and rough or occlusive fabrics; choosing cotton preferably
• avoiding irritants associated with hobbies or occupational exposure
• avoiding potentially harmful habits, for e.g. excessive hand washing

Adjuvant measures
Although the role of bathing still has not been fully investigated, the current recommendations regarding bathing are as follows:¹
• Bath regularly to hydrate the skin and debride crust.
• Take once daily baths for several minutes in warm (not hot) water.
• Use a moisturising cleanser.

| Table 1: Assessment and stepwise diagnosis of atopic eczema¹ |
|-----------------|-----------------|-----------------|-----------------|
| Step 1          | Step 2          | Step 3          | Step 4          |
| Must have:      | Need 3 or more: | Can do as necessary (not essential) | Classify:         |
| Pruritis        | Flexural eczema | Evaluate and manage: | Mild Moderate Severe |
| Previous flexural eczema | Rhinitis | Asthma | Food allergy | Environmental allergy |
Avoid antibacterial cleansers which could lead to bacterial resistance.

- Pat body dry after bathing. Do not rub the body with the towel.
- Apply an emollient immediately after bathing.

Emollients are recommended as first-line therapy since skin dryness is common and one of the diagnostic criteria of atopic eczema. Dry skin leads to inflammation, loss of suppleness leading to fissuring, impaired barrier function, and increased adherence of Staphylococcus aureus. Emollients may be steroid-sparing and have few or no adverse effects, besides some mild transient burning with oil-in-water or urea-containing emollients. The current recommendations regarding emollient use are as follows:

- Select ointments or creams rather than lotions as these provide better barrier protection.
- Allow patients to select preparations that are cosmetically acceptable; oilier preparations, while providing a better barrier function, could be too messy for use on the face.
- Apply frequently, at least twice daily, even if no symptoms are experienced.
- Apply after swimming and bathing, within three minutes to retain hydration.
- Prescribe sufficient quantity, viz 250 g/week for children and 500 g/week for adults.

Anti-inflammatory therapy: Topical steroids

Despite topical steroid therapy having been the cornerstone of atopic eczema treatment for at least 40 years, distinct “steroid phobia”, particularly in affluent countries, is recognised, where there is an unwillingness to use these preparations on account of a falsely exaggerated perception of side-effects. Health care practitioners should definitely reinforce the positive benefits of topical steroids on the basis of the evidence from an extensive systematic review of randomised controlled trials, which confirmed that topical steroids were significantly more beneficial than placebo.

Although there are many preparations and strengths of topical steroids on the market (see Table 3), some general claims can be made:

- There is no evidence that antibiotic/steroid combinations are more effective than topical steroids alone.
- The type of vehicle influences efficacy.
- Patients prefer cosmetically-acceptable preparations.
- There is no evidence to support once vs twice daily use, therefore manufacturer’s recommendations should be followed.
- There is no evidence that skin-thinning is a problem with the correct use of topical steroids.
- Dilution does not necessarily reduce adverse effects; rather stability, compatibility and microbial purity could be compromised.

The standard disease-management approach is the use of mild- to moderate-potency steroid plus emollient. Acute flares should be treated reactively with more potent steroids as research has shown that short-burst treatment with a potent steroid (0.1% betamethasone valerate) achieved

### Table 2: Treatment and management of atopic eczema

<table>
<thead>
<tr>
<th></th>
<th>Clear</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic treatment</td>
<td>Emollients</td>
<td>Emollients</td>
<td>Emollients</td>
<td>Emollients</td>
</tr>
<tr>
<td>Maintenance treatment (Tx)</td>
<td>TCI* Mild TCS**</td>
<td>TCI* Mild/moderate TCS**</td>
<td>Consider referral Moderate/potent TCS** Consider Tx as for flare</td>
<td></td>
</tr>
<tr>
<td>Flare</td>
<td>As above</td>
<td>Temporarily replace maintenance Tx with moderate/potent TCS</td>
<td>Consider referral Ciclosporin Azathioprine Phototherapy Oral steroids Methotrexate</td>
<td></td>
</tr>
<tr>
<td>Adjuvant treatment (optional, not routinely needed)</td>
<td>Antibiotics Search for and avoid trigger factors</td>
<td>Antibiotics Antihistamines Search for and avoid trigger factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Always treat</td>
<td>Herpetic infections</td>
<td>Bacterial infections</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*TCI: topical calcineurin inhibitors (pimecrolimus)

**TCS: topical cortisone preparations

### Table 3: Potency of topical cortisone preparations (TCS)

<table>
<thead>
<tr>
<th>Mild strength</th>
<th>Moderate strength</th>
<th>Potent strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procutan®</td>
<td>Advantan®</td>
<td>Dermovate®</td>
</tr>
<tr>
<td>Mylocort®</td>
<td>Elocon®</td>
<td>Diprolene®</td>
</tr>
<tr>
<td>Stopitch®</td>
<td>Locoid®</td>
<td>Nerisone® Forte</td>
</tr>
</tbody>
</table>
better control than long-term use of 1% hydrocortisone (mild potency) over an 18-week trial.\\(^10\\)

Recommendations for the use of topical corticosteroids:\\(^11\\)

1. Treatment should be initiated with moderate-strength steroids. Children with mild to moderate atopic eczema, unless under specialist care, should use 1% hydrocortisone.
2. The least potent preparation that controls the disease should be selected.
3. Should be applied once or twice daily (as recommended by the manufacturer) until remission achieved. Thereafter, intermittent dosing is recommended (twice weekly).
4. High-potency steroids are indicated for short-term use in areas of lichenification or thick skin.
5. The vehicle base influences potency; ointments are more potent than creams. Wet wraps and occlusive dressings increase potency; only less potent steroids should be used in this fashion.
6. As the inflammation subsides, less steroid and more moisturiser should be used.
7. Moderate strength steroids used immediately after bathing can be used intermittently in conjunction with emollients.

Cutaneous adverse effects of topical steroids are well-documented (skin atrophy, telangiectasia, hypopigmentation, steroid acne, increased hair growth and rosacea-like reactions), especially when applied to delicate skin areas (face, neck, skin folds) on a continuous basis. Systemic adverse effects are uncommon.

Anti-inflammatory therapy: Non-steroidal topical immunomodulators

The introduction of the topical calcineurin inhibitor, pimecrolimus (Elidel\\(^R\\)) represents a major advance in the management of atopic eczema. The specific mechanism of action is the targeting of the inflammatory cells involved in atopic eczema, without suppression of the Langerhans cells. As a result, the therapy is extremely safe and can be recommended for prevention and long-term treatment. Pimecrolimus can even be safely used on sensitive skin areas and in children two years and older. There is concern however about the potential for the development of cutaneous malignancies and patients should be counselled about the concomitant use of sunscreen preparations.

Other therapies

The value of using antihistamines is often disputed and the evidence amassed to date is conflicting. The current recommendation is that they could be used as adjunct therapy during flare-ups; sedating antihistamines at bedtime could be beneficial as pruritis tends to be worse at night. Topical antihistamines, while possibly giving relief for short periods, should not be used chronically to avoid sensitisation.

Phototherapy has been used in the management of atopic eczema, particularly high-dose UVA1, which has significant immunomodulating activity, but the risk of carcinogenesis with long-term use has not been clarified.

Tar preparations, administered topically or in the bathtub, may be beneficial in some patients; cosmetic unacceptability is associated with non-adherence to this form of therapy.

Systemic immunomodulators administered orally are only indicated for severe atopic eczema, preferably only under specialist care, as they are associated with side effects and toxicities. Examples include ciclosporin, azathioprine and mycophenolic acid.

Complementary therapies, including Chinese herbal medicines, herbal medicines, acupuncture, homeopathy, massage therapy, climatotherapy and African traditional medicine, have all failed to produce demonstrable beneficial effects.\\(^\square\\)

References:


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