Atopic Eczema

Dr Tshegofatso Mabelane
Family Allergist
MBChB; MBA; M. Fam Med; FCFP (SA); Dip Allerg (SA); Cert Allerg (SA); M. Phil Allergy (UCT)
Content

• Introduction
• Epidemiology
• Clinical presentation
• Differential diagnosis
• Management
Introduction

• 1st manifestation in the “atopic march” and often the initial indication that a child may later develop other allergic diseases such as asthma and or allergic rhinitis.

• *Family stress of patients with moderate to severe AE is greater than that of patients with type 1 diabetes* because of sleep deprivation, employment loss, time to care for eczema, and financial costs.
Definition

- **Eczema** also known as dermatitis is a name for group of conditions that result in itchy, red and inflamed skin.
- World Allergy Organisation defines eczema as an **inflammatory, chronically relapsing, non-contagious and extremely itchy skin disease**.
- AE is also referred to as atopic dermatitis and often used interchangeably with eczema.

- “eczema” comes from the Greek “to boil over”, condition periodically flares up (or boils over)
Epidemiology

• AE affects up to 20% of children and up to 3% of adults in the world.
• International Study of Asthma and Allergies (ISAAC) phase III in 2002 showed that the world average prevalence of AE between 6-7yr old was 7.9% with Africa being 9.5%.
• Recent data show that the prevalence of AE is increasing in developing world.
Dermatitis

Eczema

Atopic Eczema

Non-atopic eczema

Contact Dermatitis

Allergic contact dermatitis

Non-allergic contact dermatitis

Other forms of dermatitis
Atopic Eczema

Intrinsic
- SPT
- Specific IgE
- Atopy history

Extrinsic
- + SPT
- + Specific IgE
- + Atopy history
Functions of the skin

• The skin contains cells that **prevent water loss** which can result in dryness and cracks of the skin.

• Serves as a **mechanical defence** and prevents environmental irritants, allergens and microbials from entering the body.

• **Regulates skin surface acidity** because a **high pH favours** the development of skin surface pathogens such as *Staphylococcus aureus* and *Candida albicans*. 
Skin barrier defect

Skin barrier breakdown in atopic dermatitis

decreased protection from the environment
ITCH IMMONOLOGICAL

ENVIRONMENTAL

ITCH

BARRIER DYSFUNCTION
Environmental triggers

• chemical irritants: soap, shampoo, bubble bath, shower gel, soap, detergents.
• mechanical irritants: irritating fabrics, e.g. wool
• inhalant allergens: house dust mite, pet dander, pollen
• contact allergen fragrances and preservatives in cosmetics, nickel in jewellery or belts, latex, component in rubber e.g. shoes.
• food allergen
• skin infections (especially Staphylococcus)
• stress
Clinical presentation

• Onset of AE is usually before the age of 2 years
• Acute symptoms: *itch, redness, swelling, oozing and crusting*
• Chronic symptoms: thickening of skin with exaggerated skin markings - *lichenification*
• Dryness is a consistent complaint
AE distribution

• infancy: on the scalp, face, trunk, extremities and extensors, sparing of the nose and diaper area
AE distribution

• toddler & childhood commonly flexors but extensors may be involved
AE distribution

- adults varies but commonly on hands, neck, face and flexors
Diagnosing eczema (U.K. criteria)

Eczema is an itchy skin condition

plus three or more of the following features

• Personal history of Allergic rhinitis or Asthma (1st degree family history of atopy if child<4yrs)
• History of dry skin in the past year
• History of involvement of skin creases
• Onset below 2 years of age (not used in children <4 years)
• Visual eczema on flexures (or as defined in different age groups)
Differential diagnosis

- Contact dermatitis
- Hand eczema
- Nummular eczema
- Hyper IgE syndrome
- Others
Contact dermatitis

✓ occurs due to either an irritation or allergy but both may co-exist in a patient
✓ acute, chronic or relapsing
✓ Often bilateral
Irritant contact dermatitis

- Irritant CD is a non-immunological condition with sudden onset (within minutes) of erythema and oedema after exposure to substances or stressful environment that damage the skin barrier.
- Patients with irritant CD are predisposed to allergic CD because of the impaired skin barrier that allows penetration of allergens.
- common than allergic CD
Allergic contact dermatitis

• Allergic contact dermatitis is the 2\textsuperscript{nd} most common contact dermatitis type. Unlike, irritant contact dermatitis, the skin can take 48 to 96 hours to develop a reaction.

• Allergic contact dermatitis may also be limited to the site of original contact, but more often spreads.

• The distribution has a defined pattern and is often unilateral but may be bilateral if the allergen exposure is both sides e.g. gloves, shoes

• Common allergen examples include metals such as nickel (e.g. belts, jewellery), components in rubber, perfumes and preservatives in cosmetics
Nickel allergy

Glue in shoes
Hand eczema

- **Irritant contact dermatitis** - prolonged exposure to irritants predispose to allergic contact dermatitis, usually seen in patients using occlusive gloves or wet work (contact with solvents)

- Allergic contact dermatitis

- **Protein contact dermatitis** - subtype of allergic CD occurring in food workers. Latex allergy also forms a part of protein CD

- **Atopic hand dermatitis** - seen mostly in adult patients with other atopic conditions. Approximately one third of patients with atopic hand dermatitis are reported to have associated foot eczema.
Nummular eczema

• Itchy and associated with AE

• *commonly occurs in children* and is characterised by *coin shaped or round, well demarcated lesions that are unsymmetrical*

• occurs on any area of the body but *often on the extremities and rarely on the face*
Hyper IgE syndrome (HIES)

• HIES was described first as “Job syndrome”
• HIES is a rare primary immunodeficiency characterized by recurrent eczema, skin abscesses, lung infections, eosinophilia and high serum levels of IgE.
HIES Autosomal dominant

- *Autosomal dominant*: Stat 3 mutation- *newborn rash, eczema, recurrent skin abscesses and ear, sinus and lung* infections resulting in formation of cavitary lesions in the lungs (*pneumatoceles*)

- Typical facial appearance characteristic: *hyper-extensibility of joints, retained primary teeth* and *recurrent bone fractures* secondary to even minimal trauma.
HIES autosomal recessive

• DOCK8 deficiency is particularly common in intermarriage among close relatives
• Presents with new born eczema, skin abscesses, recurrent respiratory infections, candidiasis and other fungal infections
• severe, recurrent viral infections caused by pathogens such as Herpes simplex, Herpes zoster and Molluscum contagiosum
• susceptible to food allergy
• high frequency of neurologic complications, including encephalitis and vascular brain lesions
• No skeletal abnormalities.
Investigations

- Investigations done in AE are not for diagnosis but to assist in identifying triggering factors.
- Skin prick testing or specific IgE testing for allergen is often positive in patients with atopic dermatitis and not relevant without associated clinical symptoms.
- No role of total IgE testing unless Hyper IgE syndrome is suspected.
- Atopy patch testing for contact dermatitis.

AE is a clinical diagnosis.
Every day treatment

• Avoid triggers and aggravators
• Use bath oils and/or wash with cetamecragol
• Moisturize with an emollient minimum twice a day
✓ Creams and lotions may be less effective than ointments due to the lower proportion of oil.
✓ Aqueous cream or any cream containing sodium lauryl sulphate must be avoided as moisturisers
Flaring treatment

- Continue with the every day treatment!
- Steroid ointments/ topical Calcineurin inhibitor
- Wet wraps or dressings
- Antibiotics if infected
TCS

• Potency tailored to severity
• Use early in a flare-up
• **First line treatment**
• Avoid using emollients mixed with TCI for maintenance as long-term use of TCS results in side effects and increased risk of contact hypersensitivity to the steroid.

- **Ointments are more effective than creams because of better penetration.**
<table>
<thead>
<tr>
<th>Age</th>
<th>FACE/NECK FTU</th>
<th>ARM/HAND FTU</th>
<th>LEG/FOOT FTU</th>
<th>TRUNK-FRONT FTU</th>
<th>TRUNK-BACK FTU</th>
</tr>
</thead>
<tbody>
<tr>
<td>3–6 months</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>1–2 years</td>
<td>1.5</td>
<td>1.5</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3–5 years</td>
<td>1.5</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3.5</td>
</tr>
<tr>
<td>6–10 years</td>
<td>2</td>
<td>2.5</td>
<td>4.5</td>
<td>3.5</td>
<td>5</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>2.5</td>
<td>3 arm/1 hand</td>
<td>6leg/2foot</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>
Possible side effects of prolonged topical steroid use

- Skin thinning (atrophy)
- Stretch marks (striae).
- Easy bruising and tearing of the skin.
- Perioral dermatitis
- Enlarged blood vessels (telangiectasia).
- Susceptibility to skin infections.
Recommendations

• Patients over the age of 2 years
• Avoid long-term treatment
• Not for use in the immuno-compromised
• Not for treatment of cancerous lesions
• Once daily application whenever possible
Staphylococcus prevention

• Staphylococcus infection aggravates acute flares and needs to be well managed in atopic dermatitis.

• No role of oral or topical antibiotics, antibacterial soaps or topical antiseptics in the prevention of AE.

• Bleach baths maybe useful directed as a quarter cup of bleach in half tub water or half cup of bleach in full bathtub. Patients soak for 5-10 minutes and apply emollients afterwards.

• Treatment with oral antibiotics is for active bacterial infections.
Controlling flares

- TCS two days a week with continuous emollients is recommended in preventing acute flares. Minimises side effect risk and is cost effective.

- TCI may also be used to control flares.
Management to patient with AE in primary care
(Adapted from European Dermatology Forum, Part I- 2018)

**Baseline treatment:**
- Educational programme
- Skin care strategies
- Emollient therapy
- Allergen avoidance

**Mild (SCORAD <25) or transient AE:**
- Baseline treatment and mild topical steroids PLUS
- Mild topical steroids

**Moderate (SCORAD 25-50) or recurrent AE:**
- Baseline treatment PLUS
- Moderate or potent topical steroids
- Wet wrap therapy
- Psychosomatic counselling

Refer moderate AE in children not controlled optimum treatment

**Severe AE (SCORAD >50):**
Refer to specialist for evaluation

**Treatment recommendation:**
- Consider diagnosis
- Assess adherence
- Optimise management
Food avoidance?

- **avoidance of food is not advised** for management of AE and could affect growth in children and unnecessary diet restrictions.

- **history of immediate symptoms occurring within 2hrs should be advised to avoid the food** and be referred to an allergist for evaluation and oral food challenge procedure if necessary.

- **AE exacerbations from food is not very common and seen in 1/5 of patients with severe AE.**
Prognosis

✓ 50% of children will clear by age 2
✓ 85% of children will clear by age 3
✓ About 5% of children with eczema will continue to have eczema into adulthood
Factors influencing poor prognosis

- Onset after 2 years of age
- Severe eczema in infancy
- Atypical location for age
- Eczema to extensors – wrist and hands are more prone to persistence of eczema
- Bi-parental history of atopy
Take home message

• AE is now thought to be due to a primary defect in the epidermal barrier

• Frequent use of emollients is fundamental in restoring and maintaining skin moisture.

• management of AE involves a multidisciplinary team because of its psychosocial impacts on patients and families
References


• Allsa handbook

Useful websites

http://www.allergysa.org

http://www.scah.uct.ac.za

http://www.allergy.org.au
QUESTIONS