ATOPIC DERMATITIS

Atopic dermatitis (AD) is a chronic inflammatory skin condition, which occurs in allergy-prone (atopic) individuals. This eczematous skin disorder usually begins in early infancy and 40% of cases continue into adulthood.

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Patients with atopic dermatitis often suffer from closely related allergic conditions including asthma and allergic rhinitis. These conditions are often present in several members of the same family; making family history of asthma, eczema or hayfever a useful clue in making a diagnosis.

AD is thought to be a cutaneous manifestation of a systemic disease which gives rise to other atopic conditions; the 'atopic march'.

PATHOPHYSIOLOGY

There is no single cause of atopic dermatitis. Several explanatory theories exist regarding the underlying mechanism of atopic dermatitis development. Structural abnormalities in the skin, immune abnormalities, genetics and environmental factors all play a role in the pathophysiology of the disease.

Skin barrier dysfunction

The epidermis serves as a protective barrier against pathogens and allergens. One or more defects in the epidermal barrier may lead to AD development. Epidermal barrier dysfunction allows entry of allergens and microbes into the skin, which triggers an inflammatory response. Transepidermal water loss is also increased resulting in dehydrated skin.

The skin of patients with AD has been shown to be deficient in ceramides as well as antimicrobial peptides such as cathelicidins, which represent the first-line of defence against many infectious agents. This increases the skins susceptibility to infection by invading pathogens as well as the skins own normal flora.

Filaggrin is a filament-associated protein that contributes to the normal barrier function of the skin by binding to keratin fibres in the epidermis. The Filaggrin gene is responsible for expression of Filaggrin protein in the epidermis. Mutations of the Filaggrin gene, therefore, cause a Filaggrin deficiency in the skin, which results in a 'leaky' skin barrier. Filaggrin deficiency is not a major cause of atopic dermatitis in South Africa.

Immune abnormalities

It has been suggested that a primary immune dysfunction is present in AD patients resulting in IgE sensitisation. Patients with atopic dermatitis have been found to have a predominance of type 2 T helper lymphocytes (TH2) versus type 1, which results in greater cytokine production. The result is an increase in IgE production by plasma cells and a systemic inflammatory response, which leads to pruritic skin inflammation. Langerhans cells are specialised immune cells present in the epidermis, which respond to antigens and induce the TH2 response.

Job syndrome is a rare inherited immune deficiency caused by an abnormality of chromosome 4q. Atopic dermatitis appears soon after birth in affected individuals and puts patients at risk for severe infection of the skin and organs.

CLINICAL PRESENTATION

Atopic dermatitis typically has a chronic and relapsing course. The main symptom is pruritus or itch. Clinical presentation depends on chronicity of lesions. In the acute setting, erythema, oedema and blisters occur followed by oozing, crusting, excoriations and dryness. The skin becomes lichenified or hyperpigmented after persistent scratching. Scratching also causes breaks in the skin and results in increased likelihood of secondary infections.

Distribution of lesions is largely determined by age of the affected individual. The scalp, face, neck and extensor surfaces are commonly affected areas in infants. Anaesthetic mask sign is typical of AD - this is eczema on the face with sparring of the nose and nasolabial folds. The axillae and groins are usually spared. In children over two years of age, the flexural surfaces, neck, wrists and ankles become more prominent site of AD affectation. In adults, the flexor surfaces of the extremities, hands and feet are more commonly affected. Upper lip cheilitis and nipple eczema are quite specific for AD however these are uncommon clinical findings.

The pruritus associated with AD persists throughout the day and worsens at night. This results in sleep disturbance and may affect quality of life. Other signs of atopy may be present in the patient, eg. allergic shiners and allergic salute. Certain conditions are clinically associated with AD. These are not specific for the condition but suggest the diagnosis. Such conditions include pityriasis alba, keratosis pilaris and hyperlinear palms.

AD should be differentiated from other eczematous skin diseases such as seborrheic dermatitis, contact dermatitis and photosensitive dermatitis. It is sometimes difficult to distinguish between AD and seborrheic dermatitis (SD). The two conditions may also overlap in early infancy. It is useful to note that AD usually spares the groins and axillae and is pruritic whereas SD often does affect these areas and is usually not pruritic. Scabies and erythroderma should also be excluded prior to making a diagnosis of Atopic dermatitis.

TRIGGER FACTORS

Certain stimuli have been found to trigger AD flares. These include irritants such as wool or synthetic clothing, soaps, detergents, preservatives, perfume-based

products, metals and latex. Dietary factors may also be associated with exacerbations.

Examples of trigger factors	
Irritants:	Wool, synthetic clothing, soaps, detergents
Foods:	Cow's milk, eggs, peanuts, wheat, fish
Inhalant allergens:	House dust mite, animal dander, grass pollen
Climate:	Extremes of temperature and humidity

SECONDARY INFECTION

Patients with AD are at risk for secondary infection due to epidermal barrier dysfunction and ceramide deficiency within the skin. Superinfection is commonly caused by Staphylococcus aureus or Streptococcus pyogenes. This impetiginised eczema presents with weeping and crusting, preauricular fissuring or small superficial pustules. Folliculitis and abscesses may also occur in patients with atopic eczema.

Eczema herpeticum is the term given to secondary infection of eczematous lesions with Herpes simplex virus. The presentation is that of vesicles and blisters in clusters. These may progress to punched out erosions if not treated. Signs appear 5-12 days after contact with an infected individual, who may or may not have visible 'cold sores'. Eczema herpeticum may affect any site, but is usually seen on the face or neck where there is active or previous atopic dermatitis. Chickenpox may severely exacerbate atopic dermatitis. Molluscum contagiosum is a common complication of AD resulting from the local immunosuppression associated with the use of topical corticosteroids.

INVESTIGATION

The diagnosis of AD is made clinically. Laboratory investigation is not usually necessary, as no reliable biomarker exists to confirm the diagnosis. An IgE level may be done to confirm an atopic pattern. A skin biopsy would show non-specific spongiotic dermatitis.

Patch testing is performed to determine the cause of a suspected allergic contact dermatitis. It is not routinely offered to patients with atopic dermatitis. However, it is possible for atopic individuals to develop an allergic contact dermatitis and where indicated, patch testing may be offered.

A skin prick test may be used to evaluate responses to common trigger materials such as foods or inhalant allergens after direct application to the skin. The response is assessed after 15-20 minutes. A positive result produces a wheal and flare response, signifying the presence of antigen-specific IgE antibodies. RAST (radioallergosorbent test) is a blood test, which measures the serum concentration of IgE antibody. It is more expensive and less sensitive when compared to direct skin testing. These tests should not be performed routinely, but rather when some question about the diagnosis of allergy exists.

Topical treatment forms the basis of therapy

MANAGEMENT

There is no known cure for atopic dermatitis. Management should address itch, dryness, inflammation and superinfection.

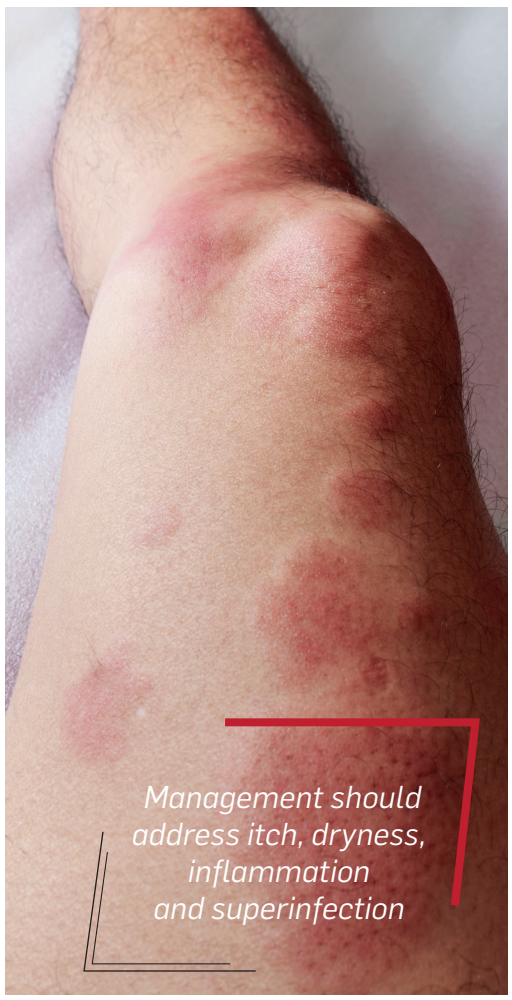
Education about the condition and avoidance of trigger factors is an important first step in successful control of the disorder. Make use of gentle body washes and cleansers instead of regular soaps. Keep water contact as brief as possible.

Avoid:

- Irritants: wool, latex, metal,
- Strong detergents, soap
- Food: Eggs, Fish, wheat, cow's milk
- Extremes in temperature

Use only mild detergents and soft cotton clothes. After bathing, apply lubricating ointments to damp skin. This will help trap moisture in the skin.

Moisturise the skin two to three times a day using ointments such as petroleum jelly and emollients. Moisturisers should be free of alcohol,



scents, dyes, fragrances, and other skin-irritating chemicals. Ointment bases are preferred, particularly in dry environments.

Topical treatment forms the basis of therapy. Emollients maintain barrier integrity and provide much needed moisture to the skin.

Topical corticosteroids provide anti-inflammatory action and are the mainstay of treatment for mild to moderate eczema. The strength of topical steroid used should be determined by the site of the body affected as well as age of the patient. On the face, a low potency topical steroid is preferred, e.g.: 1% hydrocortisone. On the trunk and limbs, higher potency topical steroids may be used, e.g: betamethasone dipropionate.

Calcineurin inhibitors are immunomodulating agents, which are considered second line therapy. Topical calcineurin inhibitors include Tacrolimus and Pimecrolimus. These agents block calcineurin, which activates inflammation within the skin causing redness and itching. These agents are typically used as steroid sparing agents and are applied during initiation of flares.

Oral antihistamines aid in the management of sleep disturbances as well as itch. Wet wrap therapy offers symptomatic relief. This includes three lukewarm baths a day, each followed by an application of topical medicines and moisturiser that is sealed in by a wrap of wet gauze.

Ultraviolet light therapy has proven to be effective in AD management. Narrow band UVB (311nm) is now considered to be the phototherapeutic modality of choice for extensive atopic dermatitis. For localised areas of eczema, the excimer laser or excimer light (308nm) has shown superior efficacy. Home-based light devices are now available and make light therapy accessible to those living far from a phototherapy facility.

Systemic agents including methotrexate, azathioprine and cyclosporine are reserved for severe disease.

Management of secondary infection in atopic dermatitis

A topical antibiotic (such as fusidic acid) is indicated until infection has cleared. Concomitant topical steroid use (e.g.: 1% hydrocortisone or betamethasone, depending on severity) is also recommended. Even once-daily treatment with topical steroid - antibiotic combination therapy for two weeks substantially decreases the density of organisms and offers significant clinical improvement. In the case of folliculitis or abscess formation, Flucloxicillin or a similar oral antibiotic should be used to manage this complication in adults, and Cephalexin in children.

Molluscum contagiosum lesions may be manually removed or treated with imiquimod cream. MC

Classification of topical steroids

Potency	Example
Low potency	Hydrocortisone acetate 1%
Moderate potency	Mometasone furoate
High potency	Bethamethasone diproprionate
Superpotent	Clobetasol propionate

ATOPIC DERMATITIS

3 CPD POINTS

SURNAME **INITIALS** **YOUR HPCSA REGISTRATION NO.** MP

Address:

Telephone: Email: Fax:

1. Atopic Dermatitis most commonly present in:
A. Adulthood.
B. Infancy.
C. Teenagers.

2. Complications of atopic dermatitis include:
A. Pityriasis alba.
B. Eczema herpeticum.
C. Keratosis pilaris.

3. The mainstay of AD therapy is:
A. Topical calcineurin inhibitors.
B. Topical steroids.
C. Methotrexate.

4. Second line therapy for AD is:
A. Phototherapy.
B. Excimer light.
C. Topical calcineurin inhibitors.

5. Clinical presentation of AD includes:
A. Anaesthetic mask sign.
B. Salmon pink plaques with silvery scales.
C. Depigmented patches.

6. Diagnosis is made via:
A. Performing a laboratory biomarker test.
B. RAST test.
C. Clinical judgement.

7. What maintains barrier integrity and provides much needed moisture to the skin?
A. Steroids.
B. aqueous cream.
C. emolients.
D. histamine response.

8. AD may be triggered by:
A. Sunlight.
B. Cotton clothing.
C. Fragrance.

9. Lymphocyte predominance in AD is:
A. Type 1 T Helper cell.
B. Type 2 T helper cell.
C. Type 3 T helper cell

10. In children over 2 years of age, the most predominant sites of AD affectation are:
A. The axillae and groins.
B. Flexural surfaces, neck, wrists and ankles.
C. Palms of the hands and soles of the feet.

This is to state that I have participated in the CPD-approved programme and that these are my own answers.

Signature **Date**

INSTRUCTIONS: **1.** Go to **www.medicalchronicle.co.za 2.** Click the tab labelled 'CPD Portal' on the far right tab near the top of the page. **3.** Select the relevant questionnaire from the list and complete the form at **http://www.medicalchronicle.co.za/dermatology-cpd-atopic-dermatitis-questions/ 4.** If you do not wish to complete this form online, fill in the answers and fax it to: **+27 086-534-1922.**