



Clinical Case Study Of Atopic Dermatitis

1Dr. Gurpreet Kaur Gill, 2Dr. Roopinder Kaur

1Associate Professor and HOD, 2Associate Professor and HOD

1Homoeopathic Medical College and Hospital, Chandigarh.,

2Homoeopathic Medical College and Hospital, Chandigarh

Abstract

Atopic dermatitis (AD) is a chronic, relapsing, inflammatory skin disorder characterized predominantly by pruritus and xerosis. It commonly presents in early childhood and is frequently associated with other atopic conditions such as asthma and allergic rhinitis. This case study highlights the clinical presentation, diagnostic criteria, pathophysiology, complications, and management considerations of atopic dermatitis, emphasizing its multifactorial etiology and significant psychosocial impact.

Introduction

Atopic dermatitis is a non-contagious, pruritic dermatosis arising from a complex interaction between genetic predisposition and environmental factors. Individuals with AD often exhibit an “atopic tendency,” manifesting as a triad of atopic dermatitis, asthma, and allergic rhinitis. A positive family history of atopy plays a pivotal role in clinical diagnosis and disease prognosis.

associated with other atopic diseases (e.g. asthma, allergic rhinitis, urticaria, acute allergic reaction to foods.)

Prevalance

It affects 10-20% of children and 1-3% adults in industrialized countries.

Sex

Male:female- 1:1.4

Age

In 85% cases, AD occurs in first year of life

In 95% cases, AD occurs before 5 years of age.

The incidence is highest in early infancy and childhood. The disease may have periods of complete remission, particularly in adolescence, and may then recur in early adult life.

Causes

1. Genetics- genetic history of atopy is common
2. Infection- skin colonized by *S aureus*, clinically it causes flare up in AD patients and it is also proposed as cause of AD by acting as a superantigen
3. Hygiene-hygiene hypothesis is touted as a cause, as this causes decreased exposure to childhood infections and bacterial endotoxins.
4. Climate and environmental factors-AD flares up in extreme climates. Heat is poorly tolerated as well as cold climate. Sun exposure improves the lesions but perspiration aggravates. AD is more common in urban as compared to rural areas probably because of industrialization and changed lifestyle.
5. Role of food is controversial, both in prevention of AD and by the withdrawal of foods in persons with established AD, because of the controversy many physicians don't withdraw food from diet. But, acute anaphylactic reactions are seen from certain food reactions
6. Probiotics- role of probiotics in AD patients remains controversial
7. Aeroallergens- role of aeroallergens and house dust mites has been proposed but further corroboration is further awaited

Morality and Morbidities

In children the disease causes enormous psychological burden to families and loss of school days. Mortality is rare due to AD.

Bacterial infection of *Staph aureus* or *Staphylococcus pyogenes* is not infrequent in the setting of AD. The skin is colonized by *S aureus* but colonization does not imply clinical infection and the physicians should only treat clinical infection.

Eczematous and bullous lesions on the palms and soles are often infected with beta-hemolytic group A streptococcus.

Kaposi varicelliform eruption (eczema herpeticum) is a well-recognised complication of AD. It usually occurs with a primary herpes simplex infection, but it may also be seen with recurrent infection. Vesicular lesions usually begin in areas of eczema and spread rapidly involving eczematous and healthy skin. The lesions may be infected secondarily as well.

Urticaria and acute anaphylactic reactions to food occur with increased frequency in patients with AD. The food group includes- peanuts, eggs, milk, soya, fish and sea-food. In studies in peanut allergic children, a large cross-section was atopic.

In AD- 30% develop asthma and 35% develop nasal allergies

Pathophysiology

Significant evidence indicates the role of genetics in atopic dermatitis, but the pathophysiology is still poorly understood.

Two hypothesis were proposed for the inflammatory process.

1. Immune dysfunction resulting in IgE sensitization and a secondary epithelial barrier disturbance.
2. Defect in epithelial cells leading to the defective barrier problem, with immunological aspects being epiphenomena.

Inherited barrier defect- role of filaggrin

Inflammation in AD results from inherited abnormalities in skin- the skin “barrier defect.” Barrier defect causes increased permeability of the skin and reduces its antimicrobial function. An inherited abnormality in filaggrin expression (filaggrin are filament-associated proteins which bind keratin fibres in the epidermal cells. The gene for filaggrin resides on chromosome 1(q21.3)). This gene was first identified as the gene involved with ichthyosis vulgaris.

Loss of filaggrin leads to-

- a) Corneocyte deformation- flattening of skin surface cells)
- b) Reduction in natural moisturizing factors, which include metabolites of pro-filaggrin.
- c) Increase in pH level of skin encouraging serine protease activity, which in turn generate active cytokines like IL1a and IL-1beta and promote skin inflammation.

The immune system

The immune system develops in the first six months of life. There is generally an equilibrium of the two main types of T helper lymphocytes- TH-1 and TH-2. In atopic dermatitis there is often an imbalance, with far more TH-2 cells and their associated chemical messengers (cytokines). In some children there are also high levels of the antibody IgE antibodies and eosinophils.

The loss of the skin barrier function means that

- Water is lost
- Irritants may penetrate (soap, detergents, solvents, dirt etc)
- Allergens may penetrate (pollen, dust mite, antigens, microbes)

Langerhan cells (specialized immune cells of epidermis in atopic dermatitis have an increased response to these antigens and interact with dermal T cells to produce TH2 response.

The inflammation induced by this TH2 exacerbates the barrier defect.

Case Study

A Male child aged 9.5 years presented in clinic with persistent itching with oozing (aggravated for the past 2 weeks) since early infancy, associated with dry, rough skin and recurrent eczematous lesions. The pruritus was severe, leading to frequent scratching and sleep disturbances. Symptoms showed a relapsing-remitting course, with exacerbations triggered by climatic changes, sweating, irritants, and emotional stress.

There was a family history of allergic rhinitis and asthma. The patient also had episodic urticarial reactions following exposure to certain foods.

History of Present Illness

The patient was apparently well 2-3 years back when he developed itchy red patches over the forearm . Over time , Lesions became dry , thickened and hyperpigmented . Scratching leads to burning and slight oozing . conditions worsens with warmth and in the evening , relieved by cold application .

Past History

Recurrent acidity and bloating since childhood

No major illness like tuberculosis or diabetes

Family History

Father suffers from chronic digestive complaints.

Mother has allergic rhinitis and her father(maternal grandfather of the child) had Asthma.

Personal History

Appetite: Good, takes all 3 meals, but gets full quickly.

Desires: Warm food, sweets++

Aversions: Cold food

Thirst: Moderate

Bowels: Constipated, unsatisfactory stool

Sleep: Disturbed due to itching

Mental Generals

Lack of self-confidence but appears confident outwardly.

Fear of failure, avoids taking up task owing to this

Irritable +, wants things his way.

Intellectual, analytical nature

Physical General Examination

Lean built

Dry skin with tendency for chronic eruptions

Local Examination (Skin)

Ill-defined erythematous plaques

Dry, scaly surface. Oozing clear fluid at the lesion

Lichenification indicating chronicity

Post-inflammatory hyperpigmentation

Miasmatic Analysis

Predominant miasm: Psora

Totality of Symptoms

Chronic eczema with dryness and itching

Complaints worse evening

Digestive disturbances with bloating

Mentally lacks confidence but pretends boldness

Remedy Selection

Based on mental generals, physical generals, and particulars, *Lycopodium clavatum* was selected.

Prescription

Lycopodium clavatum 200C

One dose

Followed by placebo for 15 days

Follow-Up & Outcome

- After 15 days:

Itching reduced significantly

Oozing stopped

Skin dryness improving

- After 1 month:

Marked reduction in lesions

Better digestion

Improved sleep

- After 3 months:

Only mild hyperpigmentation left

No recurrence of itching

Repertorial approach**Mental Generals**

Mind – Confidence – want of self-confidence

Mind – Fear – failure, of

Mind – Irritability – contradiction, from

Mind – Intellectual, analytical disposition

Physical Generals

Generalities – Time – evening – aggravation (4–8 pm)

Stool – Constipation – unsatisfactory

Abdomen – Distension – after eating

Particulars (Skin)

Skin – Itching – evening – aggravation

Skin – Eruptions – oozing – scratching, after

Skin – Dryness – general

Skin – Thickening – lichenification

Skin – Discoloration – hyperpigmentation – after eruptions

Extremities – Eruptions – forearm – flexor surface

Generalities – Drug abuse – medicinal drugs – abuse of





Before



During



After

Conclusion

This case demonstrates the effectiveness of *Lycopodium clavatum* in treating chronic eczematous conditions associated with characteristic mental traits, emphasizing the holistic approach of Homoeopathy.