

Management of atopic dermatitis

Learning objectives

At the conclusion of this learning activity, participants should be able to discuss;

- The course and occurrence of atopic dermatitis
- The clinical presentations in different age groups
- The diagnostic criteria for atopic dermatitis
- The skin care and medical treatment recommendations for managing atopic dermatitis

What is atopic dermatitis?

- ⇒ Atopic dermatitis (AD) is a long-lasting, relapsing inflammatory skin disease commonly affecting infants and children that can lead to xerosis, pruritus, and patches of dermatitis.
- ⇒ The word "*Atopic*" refers to diseases that are hereditary, tend to run in families, and often occur together with asthma and allergies (hay fever) and "*dermatitis*" means inflammation of the skin.

Prevalence

- ⇒ Atopic dermatitis is one of the most common skin diseases affecting 5-10% of young children.
- ⇒ It accounts for 4% of acute care paediatric visits.
- ⇒ The onset of disease occurs before age 5 yr in 85% of patients.
- ⇒ The disease persists into adulthood in approximately 60% of children with atopic dermatitis.
- ⇒ This is especially true for patients with early onset, severe disease, associated allergic rhinitis or asthma, and a strong family history of AD.
- ⇒ Although the symptoms of atopic dermatitis resolve by adolescence in 50% of affected children, the condition can persist into adulthood.

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How is atopic dermatitis caused?

The precise pathogenesis of atopic dermatitis is not known, but the disease seems to be the result of genetic susceptibility, immune dysfunction, environmental factors and epidermal barrier dysfunction.

Genetic factors	<ul style="list-style-type: none">• The prevalence of AD in children is about 50% when one parent has AD, while it may be as high as about 80% when both parents have the disease.• Genes play a significant role in IgE production and allergic sensitization.
Immunologic factors	Elevated T-lymphocyte activation, defective cell immunity, and B-cell IgE overproduction may result in increased inflammation of the skin.
Skin barrier dysfunction	<ul style="list-style-type: none">• Filaggrin is a protein essential for normal barrier function of the skin.• Mutations in the gene expressing filaggrin protein may result in decreased production of filaggrin.• Deficiency in this protein may contribute to the physical barrier defects and predispose patients to increased transepidermal water loss, infections, and inflammation associated with exposure of cutaneous immune cells to allergens.• Abnormalities in lipid metabolism with reduced synthesis of ceramide may lead to decreased water-binding capacity of skin. This results in dryness, which causes

	microfissures on the skin surface that promotes entry of irritants, allergens and microbes.
Environmental factors increase the severity of atopic dermatitis	
Allergens	<ul style="list-style-type: none"> • Food: a subset of patients, particularly infants and children, may have hypersensitivity to foods, particularly to milk, peanuts, fruits, fish and soybeans. • Aeroallergens: House dust mites, pollens, and animal danders. • Microbes: Staphylococcal toxins may act as superantigens and stimulate allergic reaction. Other infections like dermatophytes may also act as allergens.
Climatic factors	<ul style="list-style-type: none"> • A dry and cool environment increases xerosis of skin. • Sweating in hot, humid climates may cause irritation, while sunlight tends to improve the symptoms.
Psychological factors	<ul style="list-style-type: none"> • The psychological impact of the disease on the patients leading to depression, anxiety, and frustration may cause reduced itch threshold and increase the inherent irritability of the skin, thus contributing to the pathogenesis of the disease.

What are the clinical manifestations of atopic dermatitis?

Itching and scratching

- ⇒ Itch (pruritus) is the dominant symptom of AD.
- ⇒ It persists throughout the day but increases during evening and night.
- ⇒ It is characteristically paroxysmal and often uncontrollable.
- ⇒ It causes varying degrees of sleep disturbance in affected individuals. There is a reduced threshold for itching to irritants and the itch exacerbates with sweating, and low humidity.
- ⇒ Chronic itching leads to scratching, often violent, which results in secondary changes in the skin like excoriation, infections and thickening, that leads to more itching.
- ⇒ Thus, a vicious cycle (itch-scratch-itch cycle) is established which perpetuates the condition.

“It is not the eruption that is itchy but the itchiness that is eruptive.”



Fig. 1: Itching and scratching

Patterns of inflammation

Several patterns and types of lesions may be produced by exposure to external stimuli or may be precipitated by scratching.

Acute dermatitis	Erythematous papules and erythema associated with excoriations (superficial loss of skin), erosions, and serous exudates (oozing from crusts).
Subacute dermatitis	Erythematous, excoriated, scaling papules
Chronic dermatitis	Scratching over an extended period results in thickened skin, accentuated skin markings resembling a washboard (lichenification), and fibrotic papules. Inflammation resolves slowly, leaving the skin in a dry, scaly, compromised condition called xerosis.

The morphology and distribution of the lesions evolve in the following phases;

Infantile AD (birth to 2 yrs)	<ul style="list-style-type: none"> ⇒ Usually starts in the first year of life. ⇒ Dry, red scaling, oozing and crusting areas confined to face (cheeks, chin, lips, perioral skin), scalp, forehead and extensor surface of the extremities (Fig. 2). ⇒ Buttocks and diaper area are frequently spared.
Childhood AD (2-12 yrs)	<ul style="list-style-type: none"> ⇒ The most common appearance is inflammation in flexural areas (i.e., antecubital fossae, neck, wrists, and ankles). Facial lesions with eyelid involvement also occur (Fig. 3). ⇒ The lesions tend to be drier and scaly. Lichenification from chronic itching and scratching occur over flexor surfaces.

- ⇒ Psychological effects often are very prominent.
- ⇒ AD begins near the onset of puberty.
- ⇒ Flexural predilection of lesions persists.
- Adult AD (12 yrs to adult)** ⇒ Localized, eczematous or lichenified plaques often predominates the clinical picture.
- ⇒ Prurigo papules and nodules tend to occur. Resolved cases show dryness and irritability of the skin with a tendency to itch with sweating and other triggers.
- ⇒ Recurrent and persistent hand dermatitis is a frequent feature.



Fig. 2: AD in infants



Fig. 3: AD in the neck, eyelids and back of knees in children and adults

Diagnosis

The diagnosis of AD is based on clinical criteria. Itch must be present for the diagnosis of AD. In addition, the patient should have three or more of the following criteria;

- ⇒ Visible rashes on the flexural areas (elbows, back of knees, front of neck, or eyelids); in infants, the rash may be present on the cheeks or extensor areas of the knees or elbows
- ⇒ History of rashes on the flexural areas
- ⇒ Personal or family history of respiratory allergies (asthma or allergic rhinitis)
- ⇒ History of dry skin in the past year
- ⇒ Onset before 2 years of age

There is currently no diagnostic laboratory test for AD. Elevated serum IgE level is a frequent finding, but this is non-specific.

Dermatological diseases	Seborrheic dermatitis, contact dermatitis, psoriasis, nummular pilaris, keratosis pilaris, lichen simplex chronicus, pityriasis rosea, nonbullous congenital ichthyosiform erythroderma
Neoplastic diseases	Cutaneous T cell lymphoma (mycosis fungoides, Sezary syndrome), Letterer-Siwe disease (Langerhans cell histiocytosis), necrolytic migratory erythema associated with pancreatic tumor
Immunodeficiencies	Immune dysregulation, polyendocrinopathy, enteropathy X-linked (IPEX) syndrome, hyper-IgE syndrome, Wiskott-Aldrich syndrome, severe combined immunodeficiency syndrome
Infectious diseases	Human immunodeficiency virus-associated eczema, scabies, candidiasis, Tinea vesicolor
Congenital and metabolic disorders	Netherton's syndrome, phenylketonuria, zinc deficiency, essential fatty acid deficiency, histidine deficiency, infantile-onset multiple carboxylase deficiency

Management

Treatment aims at an attempt to--

- ⇒ Preserve and restore the stratum corneum barrier

- ⇒ Eliminate inflammation and infection
- ⇒ Control pruritus to reduce the self-inflicted damage to the involved skin
- ⇒ Control exacerbating factors

1. Skin care

General measures

- ⇒ Clip nails to decrease abrasion of skin
- ⇒ Rather than soaps, use cleansers with minimal defatting activity and a neutral pH.
- ⇒ Avoid alcohol and astringents in skin care products.
- ⇒ Avoid wool clothing or other materials that may be irritating to the skin; cotton or cotton blends are generally preferred.
- ⇒ Launder clothing to remove formaldehyde and other chemicals.
- ⇒ Use liquid detergents, which are easier to rinse out than powder detergents.
- ⇒ Add a second rinse cycle to facilitate further removal of detergents.
- ⇒ Avoid extremes of environmental temperatures or humidity; prolonged exposure to sun may lead to overheating and evaporation, as well as perspiration, all of which can be irritating.
- ⇒ Use sunscreens with low irritancy potential, such as those made specifically for the face.
- ⇒ After swimming, shower with a cleanser to remove chlorine or bromine.

Skin hydration and moisturizers (emollients)

- ⇒ Daily soaking baths for 10 to 20 minutes is recommended.
- ⇒ Emollients should be applied immediately after a soaking bath.
- ⇒ This attempt seals the water that has been absorbed into the skin and prevents drying of the skin.
- ⇒ Moisturizers are available as lotions, creams, and ointments.

2. Control of inflammation and infection

Infants—Localized inflammation

- ⇒ Infants with dry, red, scaling plaques and flares on the cheeks respond to group V or VI topical steroids (Hydrocortisone valerate 0.2% ointment, Flucinolone acetonide 0.01% cream) applied twice a day for 7 to 14 days.

Topical steroids should be used to treat dermatitis until the skin clears; then steroid application should be discontinued.

- ⇒ Pimecrolimus cream 1% or Tacrolimus 0.03% ointment may be used for long-term stability and control.
- ⇒ Parents are instructed to decrease the frequency of washing, to start lubrication with a bland lubricant during the initial phase of the treatment period, and to continue lubrication long after topical steroids have been discontinued.
- ⇒ Cephalexin 250 mg four times daily can be given only if there is moderate serum production and crusting.

- ⇒ Cracking on and around the lips is controlled in a similar fashion, but heavy lubricants (such as petroleum jelly, Aquaphor ointment, or Eucerin) are used after the inflammation has cleared.

Infants—Generalized inflammation

- ⇒ Group V to VI topical steroid cream or ointment applied two to three times a day for 10 to 21 days can be beneficial.
- ⇒ Secondary infection often accompanies generalized inflammation, and a 3- to 7-day course of antistaphylococcal antibiotics, such as cephalexin suspension, is helpful.
- ⇒ Start oral antibiotics 2 days before initiating topical steroid treatment.
- ⇒ Sedation with hydroxyzine (10 mg/5 mL) is useful during the initial treatment period. The bedtime dose gives the child a good night's sleep and seems to suppress the unconscious scratching that occurs during disturbed sleep.
- ⇒ Potent topical or systemic steroids are potentially hazardous and may be associated with relapse after therapy has been discontinued.
- ⇒ Avoidance of triggering factors is recommended.

Antibiotics used to suppress *Staphylococcus aureus* infection

- ⇒ Cephalexin 250 mg four times daily
- ⇒ Cefadroxil 500 mg twice daily
- ⇒ Dicloxacillin 250 mg four times daily

Children and adults—Lichenified plaques

- ⇒ Mid-potency topical steroids are used with occlusive dressings.
- ⇒ Occlusive therapy for 10 to 14 days is preferred as soon as the infection is controlled if the plaques are resistant to treatment or are very thick.
- ⇒ Adults may be treated with high-potency topical steroids for 1 to 4 weeks.

Children and adults—Diffuse inflammation

- ⇒ Group V topical steroids (Hydrocortisone valerate 0.2% ointment, betamethasone valerate 0.1% ointment) can be applied two to four times a day.
- ⇒ Pimecrolimus cream 1% or Tacrolimus 0.03% ointment can be used as initial treatment for mild to moderate inflammation and for maintenance.
- ⇒ A 3- to 7-day course of systemic antistaphylococcal antibiotics is almost invariably required that should be started oral antibiotics 2 days before initiating topical steroid treatment.
- ⇒ Exudative areas with serum and crust are treated with a Burow's solution compress for 20 minutes three times a day for 2 to 3 days.

Children and adults—Severe AD

- ⇒ Prednisone should be administered at a dose of 20 mg twice each day for at least 7 days and later, the dose should be tapered over the next 2 or 3 weeks.
- ⇒ Patients who may have trouble adhering to the medication schedule may be treated with triamcinolone acetonide 40 mg suspension given intramuscularly.

3. Antihistamines

- ⇒ The conventional antihistamines like diphenhydramine or hydroxyzine provide better results for their additional actions as sedative or anxiolytic.
- ⇒ Topical antihistamines should be avoided for their sensitizing potential.

4. Tar

- ⇒ Tar in a lubricating base such as T-Derm or Fototar applied twice daily is an effective alternative to topical steroids. Intensely inflamed areas should first be controlled with topical steroids.
- ⇒ Tar ointments can then be used to complete therapy as well as an initial therapy for chronic, superficial plaques.

5. Phototherapy

- ⇒ Phototherapy is effective in treating refractory atopic dermatitis.
- ⇒ This treatment may be administered as ultraviolet A (UVA), ultraviolet B (UVB) or combined UVA and UVB.

6. Cyclosporine

- ⇒ By virtue of its immunomodulating action, cyclosporine has a limited role in controlling atopic dermatitis in recalcitrant adult cases.
- ⇒ Cyclosporine is not approved by FDA to treat AD.

Suggested Readings

- Atopic Dermatitis – Clinical Aspects. *Habif: Clinical Dermatology*, 2009; 5th edition.
- Atopic Dermatitis – Treatment of atopic dermatitis. *Habif: Clinical Dermatology*, 2009; 5th edition.
- Atopic Dermatitis. *Ferri: Ferri's Clinical Advisor* 2010, 1st ed.
- Atopic Dermatitis. MedicineNet. Available from: http://www.medicinenet.com/atopic_dermatitis/article.htm
- Bandyopadhyay D. Atopic Dermatitis. Available from: <http://dermind.tripod.com/atopicderm.htm>
- Buys LM. Treatment Options for Atopic Dermatitis. *Am Fam Physician* 2007;75(4):523-28.
- Correale CE, et al. Atopic Dermatitis. A Review of Diagnosis and Treatment. *Am Fam Physician* 1999;60:1191-210.
- Hanna DM, Moennich J, Jacob SE. A Practical Management of Atopic Dermatitis—Palliative Care to Contact Dermatitis. *Journal of Dermatology Nurses Association*. 2009;1(2):97-105.
- Ong PY, et al. atopic Dermatitis. *Primary Care: Clinics in Office Practice*. 2008;35(1).

TEST QUESTIONS

1. AD in infants is typically confined to all except;

- a. Neck
- b. Cheeks
- c. Lips
- d. Chin

2. The typical hallmark manifestation of AD is.

- a. Erythema
- b. Xerosis
- c. Pruritus

d. All of the above

3. Subacute AD is characterized by;

- a. Erythema
- b. Excoriations
- c. Lichenification
- d. Both A and B**

4. Topical steroids should be discontinued after resolution of skin rashes

- a. True**
- b. False

5. Antibiotic treatment should be usually started _____ initiating topical steroids.

- a. A day before
- b. 2 days after
- c. 3 days after
- d. 2 days before**