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Terminology

Eczema

The word ‘eczema’ comes from the Greek for ‘boiling’ a reference to the tiny vesicles (bubbles) that are often seen in the early acute stages of the disorder.

Dermatitis

means inflammation of the skin and is therefore, strictly speaking, a broader term than eczema which is just one of several possible types of skin inflammation.

Atopy

In 1925 Coca define the atopy as the inherited tendency to develop allergies to food and inhalants substances manifested as eczema , asthma and hay fever.

Classification of eczema and dermatitis:

A-Exogenous eczema includes:

Contact dermatitis which include (irritant and allergic) dermatitis.

Photodermatitis (photoallergic and phototoxic) dermatitis .

Infective dermatitis.

B-Endogenous eczema includes:

1-Atopic dermatitis 2-Seborrhoeic dermatitis.

3 -Discoid or nummular eczema. 4-Asteotic eczema

5-Gravitational eczema. 6-Pompholyx or Dyshidrosis

7- Juvenile plantar dermatosis 8- Napkin (diaper) dermatitis

9-Lichen simplex chronicus (neurodermatitis)

Other classification

Acute dermatitis

- Weeping and crusting;
- blistering usually with vesicles but, in severe cases, with large blisters.

• Redness, papules and swelling usually with an ill-defined border.

• scaling.

Chronic dermatitis

• Less vesicular and exudative;

• More scaly, pigmented and thickened;

• More likely to show lichenification: a dry leathery thickened state, with increased skin markings, secondary to repeated scratching or rubbing.

• More likely to fissure

Subacute dermatitis

Showed both feature of acute and chronic dermatitis.

Atopic dermatitis (AD)

Is a chronic, pruritic eczematous disease that nearly always begins in childhood and follows a remitting/flaring course that may continue throughout life. It develops as a result of a complex interrelationship of environmental, immunologic, genetic, and pharmacologic factors. It may be exacerbated by infection, psychologic stress, seasonal/climate changes, irritants, and allergens. The disease often moderates with age, but patients carry a life-long skin sensitivity to irritants, and this atopy predisposes them to occupational skin disease.

Seventy-five per cent of cases of atopic eczema begin before the age of 6 months, and 80–90% before the age of 5 years. It affects at least 3% of infants, but the onset may be delayed until childhood or adult life.

The distribution and character of the lesions vary with age but a general dryness of the skin may persist throughout life.

Pathogenesis and Immunology:-

Elevated IgE and the inflammatory response.

The role of IgE in AD is unknown. IgE is increased in the serum of many patients with AD, but 20% of AD patients have normal serum IgE and no allergen reactivity. The levels of IgE do not necessarily correlate with the activity of the disease; therefore elevated serum IgE levels can only be considered supporting evidence for the disease.

Blood eosinophilia.

Eosinophils may be major effector cells in AD. Blood eosinophil counts roughly correlated with disease severity, although many patients with severe disease show normal peripheral blood eosinophil counts. Patients with normal eosinophil counts mainly are those with atopic dermatitis alone; patients with severe atopic dermatitis and concomitant respiratory allergies commonly have increased peripheral blood eosinophils.

Reduced cell-mediated immunity.

Several facts suggest that AD patients have disordered cell-mediated immunity. Patients may develop severe diffuse cutaneous infection with the herpes simplex virus (eczema herpeticum). Humoral immunity seems to be normal.

Aeroallergens.

Aeroallergens may play an important role in causing eczematous lesions. About 70% of patients with atopic dermatitis have positive patch test, but avoidance of these antigens rarely improves the dermatitis.

Types of atopic dermatitis

Infentile type (birth to 2 years)

Infants are rarely born with atopic eczema, but they typically develop the first signs of inflammation during the third month of life. The most common occurrence is that of a baby who during the winter months develops dry, red, scaling areas confined to the cheeks, but

sparing the perioral and paranasal areas . This is the same area that becomes flushed with exposure to cold. The chin is often involved and initially may be more inflamed than the cheeks because of the irritation of drooling and subsequent repeated washing.

Many infants do not excoriate during these early stages, and the rash remains localized and chronic. Repeated scratching or washing creates red, scaling, oozing plaques on the cheeks, a classic presentation of infantile eczema. At this stage the infant is uncomfortable and becomes restless and agitated during sleep. A small number of infants have a generalized eruption consisting of papules, redness, scaling, and areas of lichenification. The scalp may be involved, and differentiation from seborrheic dermatitis is sometimes difficult.

Approximately 50% of infants by age of 18-24 months will resolve ; other cases progress to the childhood phase, and a different pattern evolves.

Childhood type (2-12 years)

The most common and characteristic appearance of AD is inflammation in flexural areas (i.e., the antecubital fossae, neck, wrists, and ankles). Tight clothing that traps heat about the neck or extremities further aggravates the problem. Inflammation typically begins in one of the fossae or about the neck. The rash may remain localized to one or two areas or progress to involve the neck, antecubital and popliteal fossae, wrists, and ankles. The eruption begins with papules that rapidly coalesce into plaques, which become lichenified when scratched. The border usually poorly defined but rarely well defined border can occur. Constant scratching may lead to destruction of melanocytes, resulting in areas of hypopigmentation that become more obvious when the inflammation subsides, these hypopigmented areas usually fade with time. Fifty percent of ttose patients will improved and the others will run to the adulthood type.

Adult type (more than 12 years)

The adult phase of AD begins near the onset of puberty or extended from the childhood type. The reason for the resurgence of inflammation at this time is not understood, but it may be related to hormonal changes or to the stress of early adolescence. Adults may have no history of dermatitis in earlier years, but this is unusual. As in the childhood phase, localized

inflammation with lichenification is the most common pattern. One area or several areas may be involved.

Note:
Hand dermatitis may be the most common expression of the atopic diathesis in the adult.
Many adults with AD have inflammation localized to the upper lids.

The diagnostic criteria of Hanifin and Rajika of atopic dermatitis include **three major features** and **three minor features.**

• **Major features:**
  • Pruritus.
  • Typical distribution
  • Chronic or chronic, recurrent course .
  • Positive personal or family history for atopy.

• **Minor features:**
• *Dry skin* with ichthyosis vulgaris, hyperlinear palms, keratosis pilaris.
• Thick, fine dry hair.
• Elevated serum IgE; IgE-mediated skin reactions.
• Predisposition to skin infections (*Staphylococcus aureus*, herpes simplex virus, human papilloma virus, molluscum contagiosum).
• Dermatitis on palms and soles (juvenile plantar dermatosis).
• Nipple dermatitis.
• Cheilitis (dry, inflamed lips).
• Lateral thinning of the eyebrows (Hertoghe sign).
• Double fold of lower lid (Dennie–Morgan fold or line).
• Periorbital hyperpigmentation, obvious facial paleness, or erythema.
• Pityriasis alba.
• White dermatographism.
• Increased pruritus with sweating.
• Diseases flares with emotional changes.
• Unable to tolerate wools or fat solvents.
• Food allergies.
• Recurrent conjunctivitis, keratoconus, anterior and/or posterior subcapsular cataracts

Triggering Factors

Temperature change and sweatin

Atopic patients do not tolerate sudden changes in temperature.

Decreased humidity

The beginning of winter the onset of a difficult period for atopic patients. Cold air cannot support much humidity. Dry skin is less supple, more fragile, and more easily irritated.

Excessive washing

Repeated washing and drying removes water-binding lipids from the first layer of the skin.

Contact with irritating substances

Wool, household and industrial chemicals, cosmetics, and some soaps and detergents promote irritation and inflammation in the atopic patient. Cigarette smoke may provoke eczematous lesions on the eyelids.

Contact allergic substances

Contact allergic reactions to topical preparations, including corticosteroids, should be considered in patients who do not respond to therapy.

Microbic agents

S. aureus is the predominant skin microorganism in AD lesions(found in 90% of eczematous skin). Antibiotics given systemically or topically may dramatically improve atopic dermatitis.

Food

Certain foods can provoke exacerbations of AD. Many patients who react to food are not aware of their hypersensitivity. Foods can provoke allergic and nonallergic reactions. The most common offenders are eggs, peanuts, milk, fish, soy, and wheat.

Emotional stress

Stressful situations can have a profound effect on the course of AD. A stable course can quickly degenerate, and localized inflammation may become extensive almost overnight.

Special investigations:

• Prick testing with common food and inhalant allergens.

• Allergen-specific IgE determinations.

• Atopy patches testing. Common aeroallergens are applied and interpret as in a routine patch test.

Bad prognostic factors of atopic dermatitis

Persistent dry or itchy skin in adult life

Widespread dermatitis in childhood

Associated allergic rhinitis

Family history of atopic dermatitis

Associated bronchial asthma

Early age at onset

Female sex

Treatment

General measurements

Explanation, reassurance and encouragement.

The avoidance of exacerbating factors such as irritants (e.g. woollen clothing next to the skin) and others triggering factors.

Topical therapy

Judicious use of topical steroids .

Topical salicylic acid, tar, urea (10-20%).

Topical potassium permanganate (1:5000-1:10000) in acute weeping dermatitis.

Topical plan emollients (Vaseline).

Topical calcinurin inhibitors (tacrolimus, pimecrolimus)

Topical antihistamine doxipen.

Topical antibiotics (antistaph)

Sysemic therapy

Antihistamines (sedative type) .

Short course of systemic steroid .

Systemic antibiotics (antistaph).

Other immunomodulators (cyclosporine, mycophenolate mofetil, azathioprine).

Phototherapy

As UVA, UVB, PUVA