Dermatology. **Lecture: 9**
Dr. Intisar Ahmed **Stage: 5**

Eczema and Dermatitis

*Make up **20%** of all new patients referred to dermatology clinics.

Terminology

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

Classification

1. **Exogenous**:
   *Irritant,
   *Allergic
   *Photodermatitis.
2. **Endogenous**
   *Atopic
   *Seborrhoeic
   *Discoid(nummular)
   *Pompholyx
   *Gravitational(venous,stasis)
   *Asteatotic
   *Neurodermatitis
   *Juvenile plantar dermatosis
   *Napkin(diaper)dermatitis

Histology

*The clinical appearance of different stages of eczema mirrors their histology.

*In acute stage:

-oedema in the epidermis (spongiosis) → to intra-epidermal vesicles → coalesce into larger blisters or rupture.

*In chronic stage:

-less spongiosis and vesication

-thickening of the prickle cell layer(acanthosis) and horny layers(hyperkeratosis and parakeratosis).

*These changes are accompanied by a variable degree of vasodilatation and infiltration with lymphocytes.

Clinical appearance

Acute eczema

*Weeping and crusting.
*Blistering (vesicles or large blisters).
*Redness.
*Papules and swelling.
*Usually with an ill defined border.
*Scaling.

Chronic eczema

*Chronic eczema may show all of the above changes but in general:
*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.

Complications

1.Heavy bacterial colonization.
2.Local superimposed allergic reactions to medicaments.
3.Huge effect on the quality of life.

Common patterns of eczema

Contact Dermatitis

*Is an eczematous dermatitis caused by exposure to substances in the environment.
*These substances act as irritants or allergens and cause acute, subacute, or chronic eczematous inflammation.

Irritant contact dermatitis

*Irritation of the skin is the most common cause of contact dermatitis.
*The epidermis is a thin cellular barrier with an outer layer composed of dead cells.
*Any process that damages any component of the barrier compromises its function, and a non immunologic eczematous response may result.
*Repeated use of strong alkaline, soap, industrial exposure to organic solvents ➡ extracts lipids from the skin.
*Acids may combine with water in the skin and cause dehydration.
*When the skin is compromised, exposure to even a weak irritant sustains the inflammation.

*Intensity of inflammation related to: concentration of the irritant.
length of exposure.
*Patients vary in their ability to withstand irritants.

***Management of irritant contact dermatitis**

1. Avoid exposure to irritants by using protective equipment, such as gloves.
2. Topical steroids are used to initially control inflammation.
3. Moisturizers frequently increase skin hydration. Lipid-rich moisturizers both prevent and treat irritant contact dermatitis.
4. Barrier creams containing dimethicone prevent irritant contact dermatitis.
5. Cool compresses are used for acute inflammation. They suppress vesiculation and decrease inflammation.
6. Repeated low-level UV exposures may be effective for long-term resistant cases.

*Even after the skin appears normal, it takes approximately 4 months or more for barrier function to normalize.

Allergic contact dermatitis

*Is an inflammatory reaction that follows absorption of antigen applied to the skin and recruitment of previously sensitized, antigen-specific T lymphocytes into the skin.
*It affects a limited number of individuals.
*The antigens are usually low-molecular-weight substances that readily penetrate the stratum corneum.
*Most contact allergens are weak and require repeated exposure before sensitization occurs.

Pathogenesis

*Two phases:

1**. Sensitization phase:**

*Antigen is applied to the skin, penetrates stratum corneum and is taken up by Langerhans cells in the basal layer.
*The antigen is "processed" and displayed on the surface of the Langerhans cell.
*This cell migrates to the regional lymph nodes and presents the antigen to T lymphocytes.
*Cytokine induced proliferation and clonal expansion within the lymph nodes results in T lymphocytes bearing receptors that recognize the specific antigen. *These antigen-specific T lymphocytes enter the blood stream and circulate back to the epidermis.

2. **Elicitation phase:**

*Occurs in sensitized patients with re exposure to the antigen.
*Langerhans cells bearing the antigen interact with antigen-specific T lymphocytes that are circulating in the skin.
*This interaction results in cytokine induced activation and proliferation of the antigen-specific T lymphocytes and the release of inflammatory mediators.
*Allergic contact dermatitis develops within 12 to 48 hours of antigen exposure and persists for 3 or 4 weeks.

Patch tests

*Used to detects allergic contact dermatitis.
*Involves applying a chemical to the skin and watching for dermatitis to develop 48–96 hours later.
*Either suspected antigens, or a battery of antigens that are common culprits, can be tested.
*The test materials are applied to the back under aluminium discs or patches.
*The patches are left in place for 48 hours and then, after careful marking, are removed.
*The sites are inspected 10 minutes later, again 2 days later, and some times even later if doubtful reactions require further assessment.
*The test detects type IV delayed hypersensitivity reactions. The readings are scored according to the reaction seen:

NT Not tested.
− No reaction.
± Doubtful reaction (minimal erythema).
+ Weak positive reaction (erythema and may be papules).
++ Strong reaction (palpable erythema and/or vesicles).
+++ Extreme reaction (intense palpable erythema, coalescing vesicles and/or bullae).
IR Irritant reaction (variable, but often sharply circumscribed, with a glazed appearance and increased skin markings).

***Management of allergic contact dermatitis**

1. Minimize products for topical use.
2. Use ointments instead of creams (creams contain preservatives and are complex mixtures of chemicals).
3. Botanical extracts may be used in “fragrance-free” products.
4. When patch testing, also test the patient’s consumer products.

5. Read product labels carefully. Many "dermatologist recommended" products contain sensitizers (e.g., lanolin, fragrance, quaternium-15, parabens, methylchloroisothiazolinone/methylisothiazolinone).

*__**The common allergens**__

1. Metals:
   Chrome: Cement, common in men.
   Nickel: Nickel-plated objects, especially cheap jewellery, in women.
2. Cosmetics:
   Fragrance mix: variety of cosmetics, sprays, toiletries and perfume.
   Para phenylene diamine(PPD): Dark dyes for hair and clothing.
3. Preservatives:
   Formaldehyde: shampoos, cosmetics, and pathology laboratories.
   Parabens-mix: in a wide variety of creams and lotions, both medical and cosmetic.
   Quaternium15
4. Medicaments:
   Neomycin: topical antibiotic, used (e.g.for impetigo).
   Benzocaine: local anaesthetic used in some topical applications (e.g.for piles and sunburn).
5. Rubber:
   Rubber it self is often not the problem, but it has to be converted from soft latex to usable rubber by adding additives to make it harder. These additives are allergens.
   Mercapto-mix: Shoe soles, rubber bands and golf club grips.
6. Plants: poison ivy and poison oak.

Irritant versus allergic dermatitis

	Irritant	Allergic
People at risk	Everyone	Genetically predisposed
Mechanism of response	Non immunologic; a physical and chemical alteration of epidermis	Delayed hypersensitivity reaction
Number of exposures	Few to many; depends on individual's ability to maintain an effective epidermal barrier	One or several to cause sensitization
Nature of substance	Organic solvent, soaps	Low molecular weight hapten (e.g., metals, formalin, epoxy)
Concentration of substance required	Usually high	May be very low
Mode of onset	Usually gradual as epidermal barrier becomes compromised	Once sensitized, usually rapid; 12 to 48 hours after exposure
Distribution	Borders usually indistinct	May correspond exactly to contactant (e.g., watchband, elastic waistband)
Investigative procedure	Trial of avoidance	Trial of avoidance, patch testing, or both
Management	Protection and reduced incidence of exposure	Complete avoidance

Atopic Dermatitis

*The term atopy designate a group of patients who had a personal or family history of one or more of the following diseases: hay fever, asthma, very dry skin, and eczema.

*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 relationship of environmental, immunologic, genetic, and pharmacologic factors.

Prevalence.

*15% to 30% of children, 2% to 10% of adults.

Genetics.

*Concordance rate is higher among monozygotic twins 77% than dizygotic twins 15%.

Pathogenesis and immunology

1. Elevated IgE and the inflammatory response.

*20% of AD patients have normal serum IgE levels, therefore elevated serum IgE levels can support diagnosis.

2. Blood eosinophilia.

3. Reduced cell-mediated immunity.

*AD patients have disordered cell-mediated immunity. So develop severe diffuse cutaneous infection with the herpes simplex virus (eczema herpeticum) .

4. Aeroallergens.

Clinical aspects

1. Major and minor diagnostic features:

*Criteria for diagnoses of AD:

Major Features (Must Have Three or More)

*Pruritus.

*Typical morphology and distribution:

-Flexural lichenification in adults.

-Facial and extensor involvement in infants and children.

*Dermatitis—chronic or chronically relapsing.

*Personal or family history of atopy—asthma, allergic rhinitis, atopic dermatitis

Minor Features (Must Have Three or More)

*Cataracts (anterior-subcapsular).

*Cheilitis.

*Conjunctivitis—recurrent.

*Eczema—perifollicular accentuation.

*Facial pallor/facial erythema.
*Food intolerance.
*Hand dermatitis—nonallergic, irritant.
*Ichthyosis.
*IgE—elevated.
*Immediate (type 1) skin test reactivity.
*Infections (cutaneous)— Staphylococcus aureus, herpes simplex.
*Infraorbital fold (Dennie-Morgan lines).
*Itching when sweating.
*Keratoconus.
*Keratosis pilaris.
*Nipple dermatitis.
*Orbital darkening.
*Palmar hyperlinearity.
*Pityriasis alba.
*White dermographism.
*Wool intolerance Xerosis.

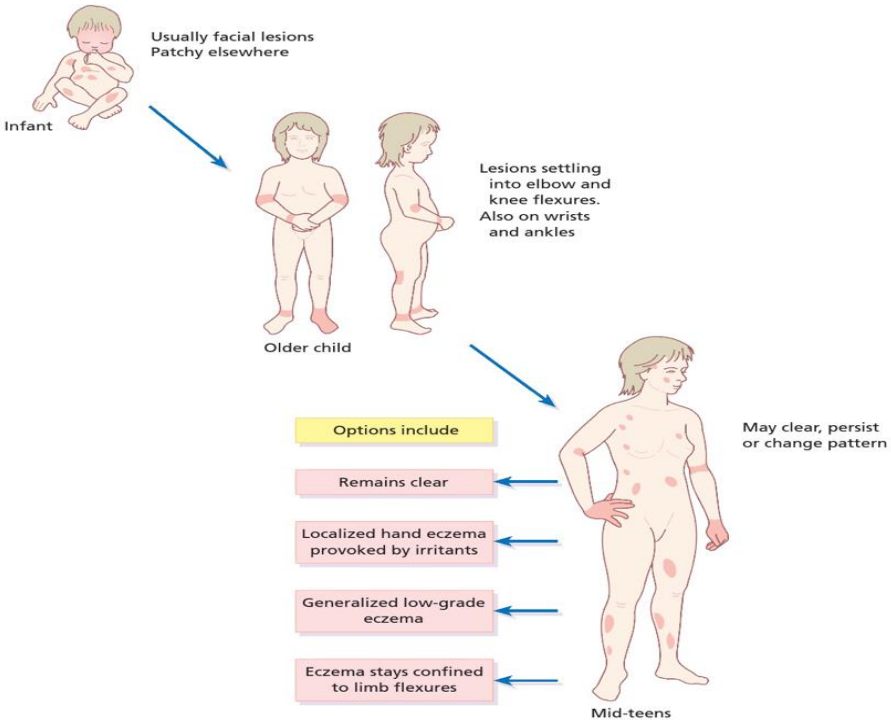
2. **Patterns of inflammation.**
*Several types of lesions present:
Acute inflammation: erythematous papules, erythema, excoriations, erosions, and serous exudate.
Subacute: erythematous, excoriated, scaling papules.
Chronic: lichenification and fibrotic papules.
*All three types of reactions can coexist in the same individual.
*58% of infants with AD were found to have persistent inflammation 15 to 17 years later.
*AD is divided into three phases.

Infant phase (birth to 2 years)
*Typically develop during the third month of life.
*Develops dry, red, scaling areas confined to the cheeks, sparing the perioral and paranasal areas, involving the chin.
*Habitual lip licking by an atopic child results in oozing, crusting, and scaling on the lips and perioral skin.
*Infants do not excoriate during these early stages → rash remains localized and chronic.
*So infant is uncomfortable and becomes restless and agitated during sleep.
*Generalized eruption consisting of papules, redness, scaling, and areas of lichenifi cation.

*The scalp may be involved, the diaper area is often spared.
*Lichenification may occur in the fossae and crease areas, or it may be confined to easily reached spot, such as directly below the diaper, the back of the hand, or the extensor forearm.

Childhood phase (2 to 12 years)
*Inflammation in flexural areas (antecubital, neck, wrists, and ankles).
*The eruption begins with papules that rapidly coalesce into plaques, which become lichenified when scratched.
*The border may be sharply defined or poorly defined.
*Constant scratching → destruction of melanocytes, resulting in areas of hypopigmentation.
*Sleep, school, work, or job performance deteriorates.

Adult phase (12 years to adult)
*Begins near the onset of puberty.
*May be related to hormonal changes or to the stress of early adolescence.
*Localized inflammation with lichenification is the most common pattern.
*One or several areas may be involved, and there are several characteristic patterns.
*-Inflammation in flexural areas.
-Hand dermatitis.
-Inflammation around eyes.
-Lichenification of the anogenital area.



Associated features

***Keratosis pilaris**

*Very common, occur in patients with AD.

*Small (1 to 2 mm), rough, follicular papules or pustules may appear at any age and are common in young children

*The incidence peaks during adolescence, and the problem tends to improve thereafter.

*The posterolateral aspects of the upper arms and anterior thighs are frequently involved, but any area, with the exception of the palms and soles, may be involved.

*The eruption may be generalized.

*Some time red halo appears at the periphery of the keratotic papule.

*Treatment

-Topical retinoids (tretinoin or tazarotine cream)

-Short courses of topical steroids.

-12% ammonium lactate lotion.

-urea cream (10-40%).

-salicylic acid lotion 6% reduces the roughness

-Abrasive washing techniques cause further drying.

*__Pityriasis alba__
*Common asymptomatic, hypopigmented, slightly elevated, fine, scaling plaque with indistinct borders.
*Affects the face, lateral upper arms, and thighs.
*Appears in young children and usually disappears by early adulthood.
*The white, round-to-oval areas vary in size, but generally average 2 to 4 cm in diameter.
*Lesions become obvious in the summer months when the areas do not tan.
*The loss of pigment is not permanent, as it is in vitiligo.
*No treatment other than lubrication should be attempted unless the patches become eczematous, Tacrolimus ointment 0.1% applied two times a day for a few weeks may be effective.

Triggering factors

1. Temperature change and sweating.
2. Decreased humidity.
3. Excessive washing.
4. Contact with irritating substances.
5. Contact allergy.
6. Aeroallergens.
7. Microbic agents: staphylococcus aureus, is the predominant skin microorganism in AD lesions.
8. Food.
9. Emotional stress.

Treatment of atopic dermatitis

*Goals:
1.Eliminate inflammation and infection,
2.Preserving and restoring the stratum corneum barrier by using emollients,
3.Using antipruritic agents to reduce the self-inflicted damage to the involved skin,
4.Controlling exacerbating factors

*Controlling atopic dermatitis
-Avoid frequent, lengthy bathing and Use tepid water, Use soaps only in axilla, groin, feet.
-Avoid wool use 100% cotton.
-Avoid perfumes or makeup that burns or itches.
-Apply soothing lubricants.
-Maintain cool, stable temperatures, do not overdress, avoid sweating.

-Humidify the house in winter.
-Avoid cigarettes.
-Avoid cats, dogs, rodents, or birds.
*Treating atopic dermatitis
1. **Topical Therapy:**
*Topical steroids to treat dermatitis until the skin clears; then discontinued.
*Topical nonsteroidal anti inflammatory agents initial therapy or following treatment with topical steroids (Pimecrolimus, Tacrolimus).
*Tar Creams.
*Moisturizers should be applied after showers and after hand washing.
*Lipid-free lotion cleansers.

2. **Antibiotics**:
*To suppress Staphylococcus aureus; they may be administered on a short- or long-term basis:
*Cephalexin 250 mg four times daily.
*Cefadroxil 500 mg twice daily.
*Dicloxacillin 250 mg four times daily.

3. **Antihistamines**:
*Antihistamines control pruritus and induce sedation and sleep.
*Hydroxyzine.
*Doxepin.

4. **Treating Severe Cases:**
*Corticosteroids.
*Oral prednisone.
*Intramuscular triamcinolone.
*Cyclosporine.
*Mycophenate mofetil.
*Azathioprine.

5. **Light Therapy:**
*Combined UVA-UVB.
*UVA.
*UVB.
*UVA1.
*Narrow-band 311-nm UVB.
*PUVA.

Seborrhoeic eczema

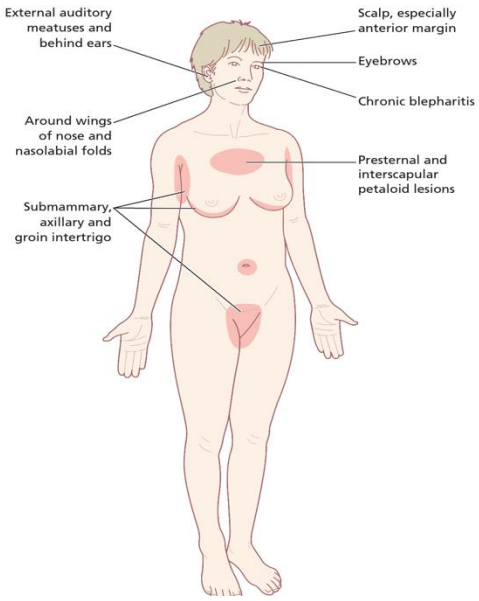
*Affect infants but is most common in adult males.

*In infants it clears quickly but in adults its course is unpredictable and may be chronic or recurrent.

Presentation and course:

*Three common patterns of eczema, affecting hairy areas, and showing characteristic greasy yellowish scales, these patterns may merge together:

1. A red scaly or exudative eruption of the scalp, ears, face and eyebrows. May be associated with chronic blepharitis and otitis externa.
2. Dry scaly petaloid lesions of the presternal and interscapular areas. There may also be extensive follicular papules or pustules on the trunk (seborrhoeic folliculitis or Malassezia folliculitis).
3. Intertriginous lesions of the armpits, umbilicus or groins.



Cause:

* Not obviously related to seborrhoea.
* Familial.
* Overgrowth of the Malassezia yeast skin commensals.
* Early sign of AIDS.

Complications:

-Furunculosis.

-In the intertriginous type, superadded Candida infection is common.

Investigations:

-None.

-Bear possible HIV infection and Parkinson's disease in mind.

Treatment:

*Therapy is suppressive rather than curative and patients should be told this.

1. Topical imidazoles.
2. 2% sulfur and 2% salicylic acid in aqueous cream.
3. Medicated shampoo, contain ketoconazole, tar, salicylic acid, sulfur, zinc or selenium sulfide.
4. Topical lithium preparation.
5. For intertriginous lesions a weak steroid –antiseptic or steroid –antifungal combination is often effective.
6. For severe and unresponsive cases a short course of oral itraconazole may be helpful.

Discoid (nummular) eczema

Cause:

-No cause has been established.
-Chronic stress is often present.
-Reaction to bacterial antigens.

Presentation and course:

*Classically affects the limbs of middle-aged males.
*Lesions are multiple, coin-shaped, vesicular or crusted, highly itchy plaques, usually less than 5 cm across.
*Persist for many months, and recurrences often appear at the site of previous plaques.

Investigations:

-None are usually needed.

Treatment:

-Topical steroid –antiseptic combinations.
-Topical steroid –antibiotic combinations

Pompholyx

Cause:

-Unknown,
-Heat or emotional upsets.

Presentation and course:

*Unpleasant form of eczema.

*Recurrent bouts of vesicles or larger blisters appear on the palms, fingers and/or the soles of adults.
*Bouts lasting a few weeks recur at irregular intervals.
*Secondary infection and lymphangitis are a recurrent problem for some patients.

Investigations:

-None are usually needed.

Treatment:

-Appropriate antibiotics for bacterial infections.
-Aluminium acetate or potassium permanganate soaks, followed by applications of:
-a very potent corticosteroid cream.
-If fails to respond, alitretinoin should be prescribed.

Gravitational (stasis) eczema

Cause:

-Accompanied by obvious venous insufficiency.

Presentation and course:

*Chronic patchy eczematous condition of the lower legs, sometimes accompanied by varicose veins, oedema and haemosiderin deposition.
*When severe it may spread to the other leg or even become generalized.

Complications:

-Sensitization to local antibiotic applications or to the preservatives in medicated bandages.
-Excoriations may lead to ulcer formation.

Treatment:

-Elimination of oedema by elevation, pressure bandages or diuretics.
-A moderately potent topical steroid.
-Medicated bandages are useful but stasis eczema is liable to persist, despite surgery to the underlying veins.

Asteatotic eczema

Cause:

-Develop in old age who have a dry skin and a tendency to chap.
-Removal of surface lipids by over-washing.
-Low humidity of winter and central heating.
-Use of diuretics and hypothyroidism.

Presentation and course:

*Often unrecognized.
*Usually on the legs of elderly patients.

*Against a background of dry skin, a network of fine red superficial fissures creates a ‘crazy paving’ appearance.

Investigations:

-None are usually needed.

-Very extensive cases may be part of malabsorption syndromes, zinc deficiency or internal malignancy.

Treatment:

-Mild or moderately potent topical steroid in a greasy base.

-Baths should be restricted until clearance.

-Daily use of unmedicated emollients usually prevents recurrence.

Localized neurodermatitis (lichen simplex)

Cause:

*The skin is damaged as a result of repeated rubbing or scratching, as a habit or in response to stress, but there is no underlying skin disorder.

Presentation and course:

*Usually as a single fixed itchy lichenified plaque.

*Favourite areas are the nape of the neck in women, the legs in men and the anogenital area in both sexes.

*Lesions may resolve with treatment but tend to recur either in the same place or elsewhere.

Investigations:

-None are usually needed.

Treatment:

-Potent topical steroids or occlusive bandaging, where feasible, help to break the scratch–itch cycle.

-Tranquillizers are often disappointing.

Juvenile plantar dermatosis

Cause:

-Impermeability of modern socks and shoe linings with subsequent sweat gland blockage.

-Manifestation of atopy.

Presentation and course:

*The skin of the weight-bearing areas of the feet, particularly the forefeet and undersides of the toes, becomes dry and shiny with deep painful fissures that make walking difficult.

*The toe webs are spared.

*Onset can be at any time after shoes are first worn.

*Even if untreated, clears in the early teens.

Treatment:

-Topical steroid.
-Emulsifying ointment or
-1% ichthammol paste, or
-Emollient containing lactic acid.

Napkin (diaper) dermatitis

Cause:

*The most common type of napkin eruption is irritantin origin, is aggravated by the use of waterproof plastic pants.
*The mixture of faecal enzymes and ammonia produced by urea-splitting bacteria, and prolonged contact with the skin, leads to a severe reaction.
*The overgrowth of yeasts is another aggravating factor.
*The introduction of modern disposable napkins has helped to reduce the number of cases sent to dermatology clinics.

Presentation:

*The moist, glazed and sore erythema affects the napkin area with the exception of the skin folds, which tend to be spared.

Complications:

-Superinfection with Candida albicans → small erythematous papules or vesicopustules appearing on periphery of the main eruption.

Treatment:

-The child should be allowed to be free of napkins as much as possible
-Disposable diapers (superabsorbent) type is best and should be changed regularly, especially in the middle of thenight.
-The area should be cleaned at each nappy change with aqueous cream and water. -Protective ointments (e.g. zinc and castor oil ointment).
-Combinations of hydrocortisone with anti fungals or antiseptics.