Eczema

Younes Malkawi

Diana Suhbat

Eczema

- Introduction
- Endogenous eczemas
- Exogenous eczemas

Introduction

Introduction

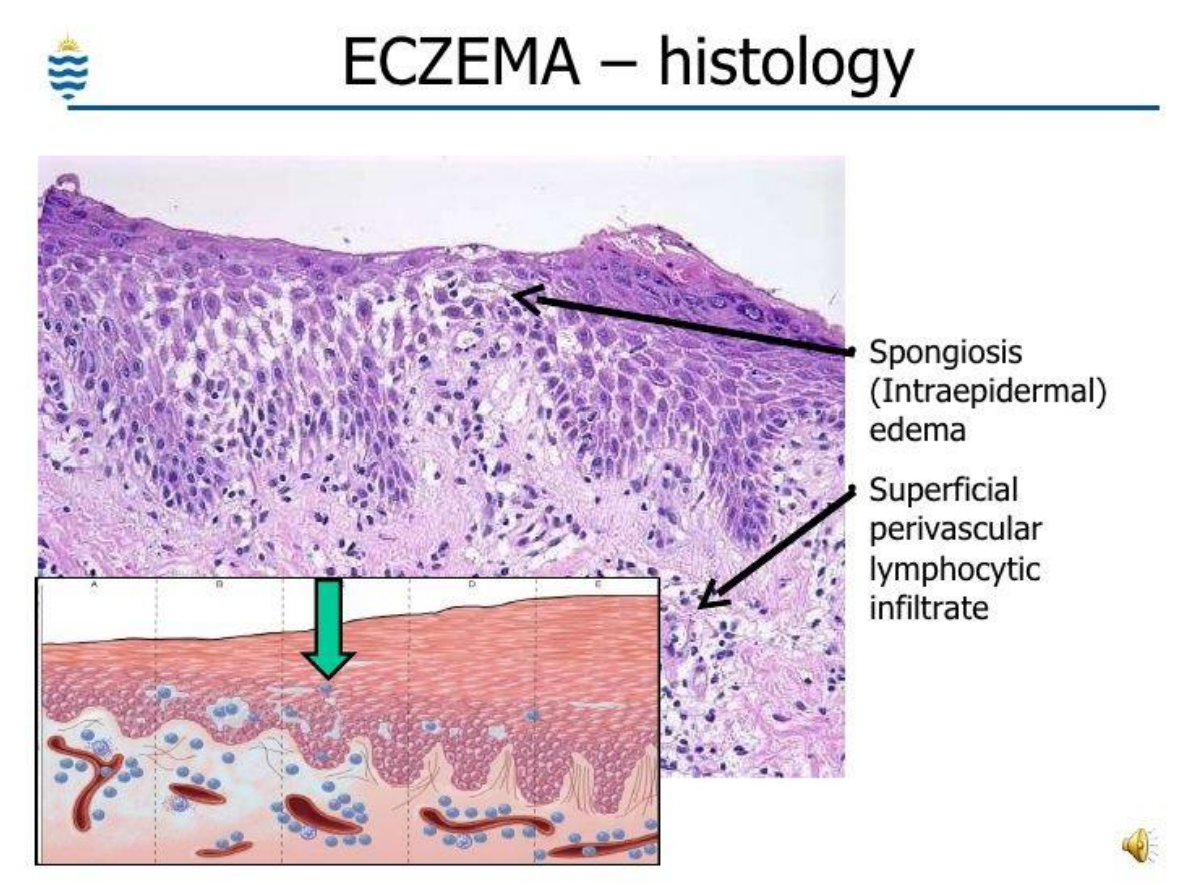
- Definitions:
  - Dermatosis is a condition of the skin
  - Dermatitis is the inflammation of the skin
  - Eczema is a type of dermatitis characterized not just by erythema, but by oozing, crusting, scaling & lichenification depending on its duration and chronicity.
- Key points:
  - “Eczema is dermatitis, but dermatitis is not eczema”
  - Although different, the terms "dermatitis" and "eczema" are frequently used interchangeably in the practical setting, where “eczema” alone is referring to **atopic dermatitis** specifically

Introduction

- General histology:
  - **Spongiotic** tissue reaction pattern → intracellular edema within the epidermis
  - Initially, there is a widening of intercellular spaces between keratinocytes and elongation of the intercellular bridges.
  - Dynamic pathological process → vesicles appear & disappear at different epidermal levels
  - Dermal changes include varying degrees of oedema and a superficial perivascular infiltrate with lymphocytes, histiocytes and occasional neutrophils and eosinophils.
  - Histological features change with **time**. Duration is very important!

Introduction

- General histology:

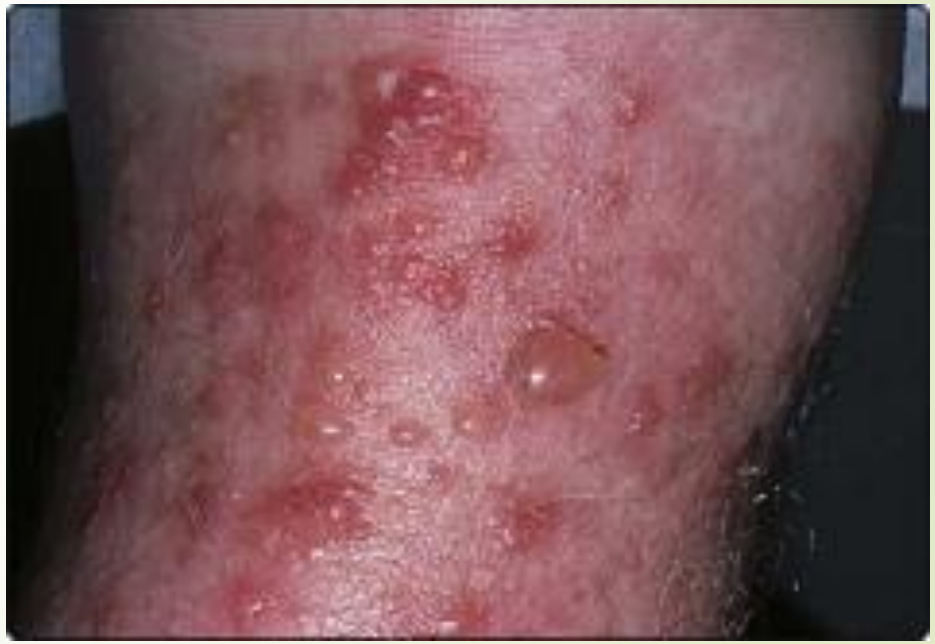


Introduction

- Classification of eczema:
  - According to duration:
    - Acute eczema
    - Sub-acute eczema
    - Chronic eczema
  - According to cause:
    - Endogenous eczema → An internal property of the skin is responsible for the disease
    - Exogenous eczema → An external cause is responsible for the disease
    - Mixed eczema → Both endogenous & exogenous processes precipitate the disease (e.g. Xerotic eczema)

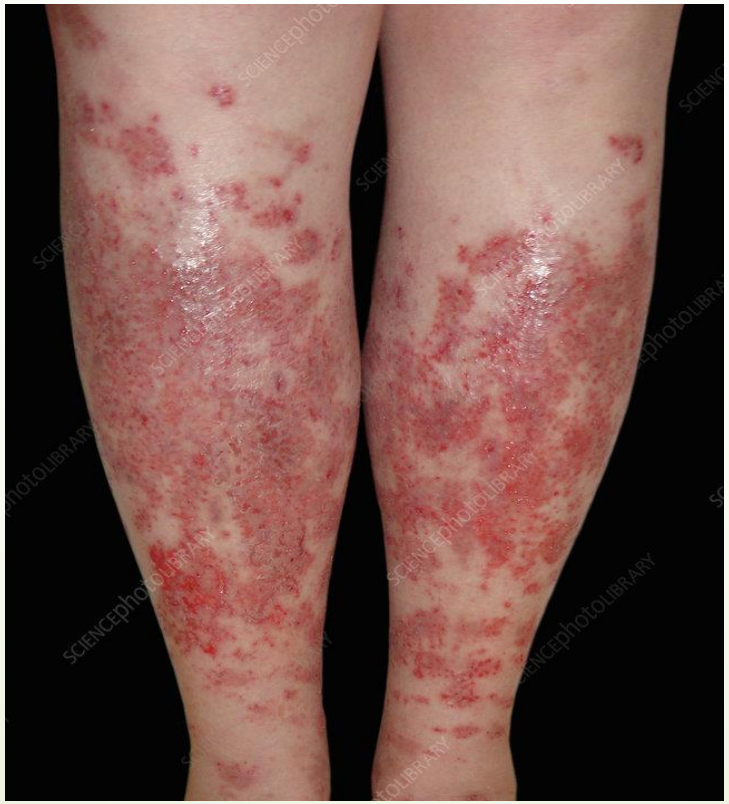
Introduction

- 1. According to duration:
  - 1. Acute eczema: (Clinical features)
    - Severe pruritus (1st sign)
    - Erythema
    - Edema
    - Papulo-vesicles
    - Oozing
    - **No** crusting/scaling



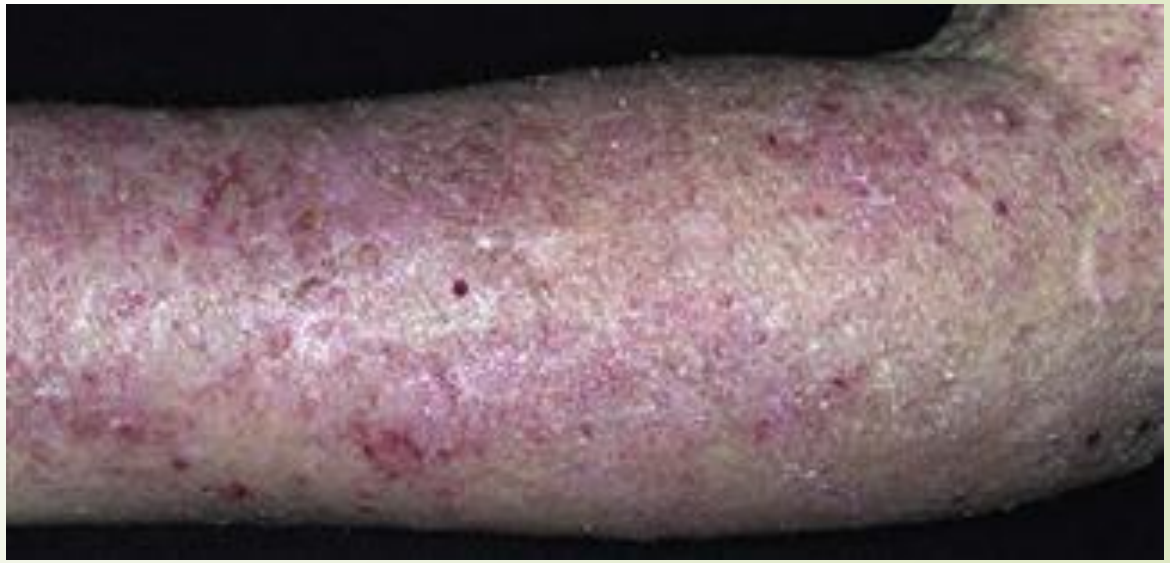
Introduction

- 1. According to duration:
  - 1. Acute eczema:



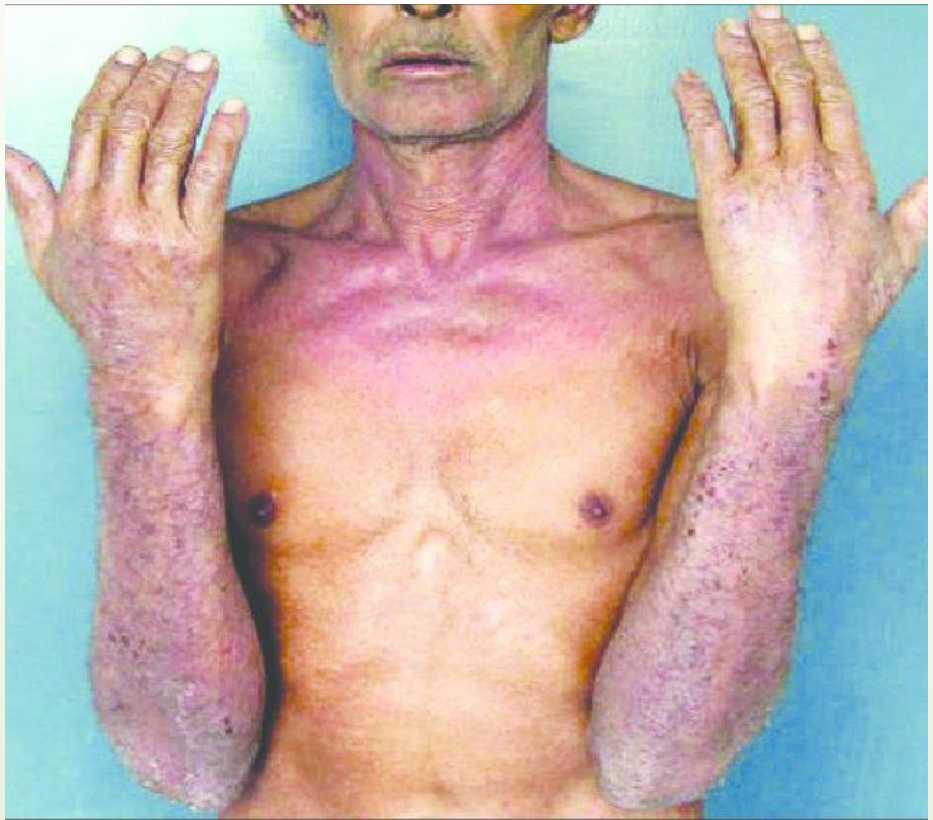
Introduction

- 1. According to duration:
  - 2. Sub-acute eczema: (Clinical features)
    - Pruritus (less pronounced than the acute stage)
    - Erythema (less pronounced than the acute stage)
    - Fissuring
    - Crusting/scaling **present**



Introduction

- 1. According to duration:
  - 2. Sub-acute eczema:

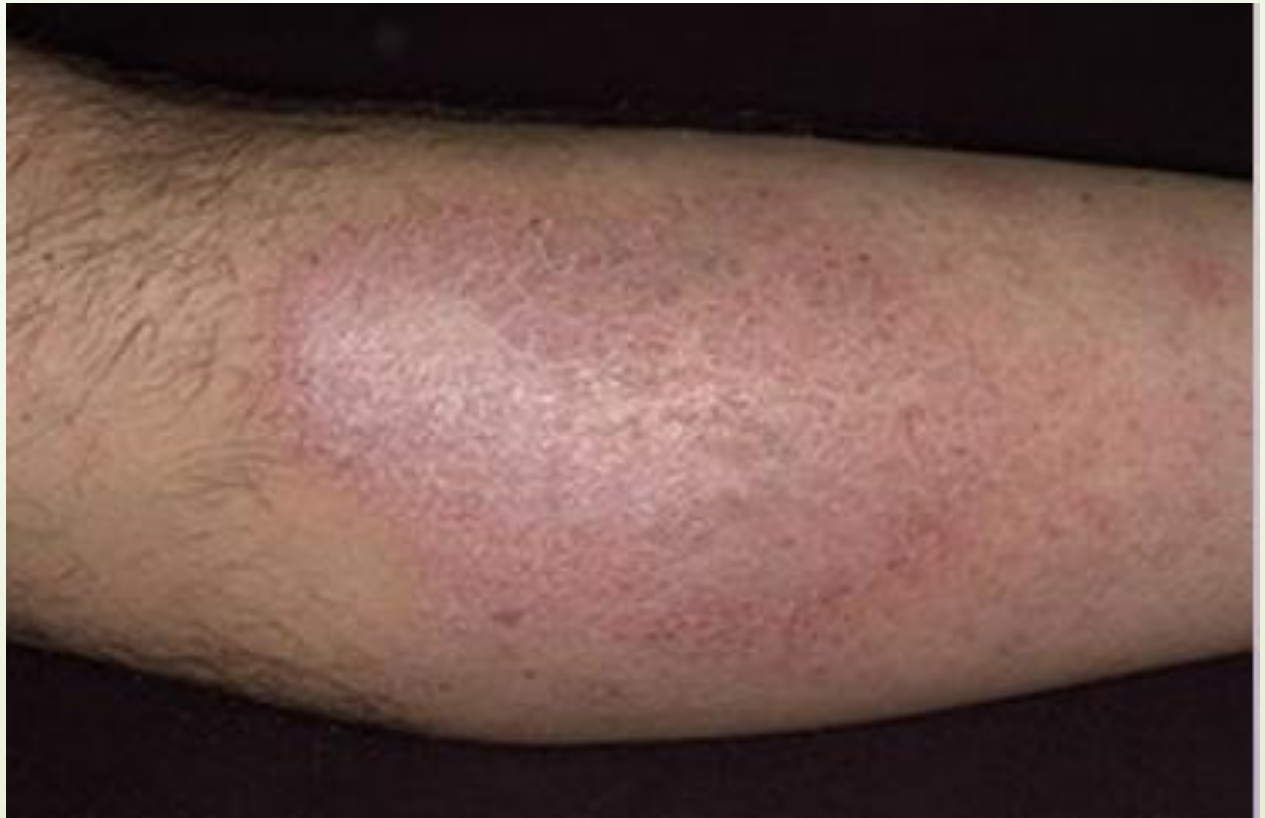


Introduction

- 1. According to duration:
  - 3. Chronic eczema: (Clinical features)
    - Skin dryness
    - Excoriation
    - Fissuring
    - **Lichenification** – thickening, hyperpigmentation & increased skin markings

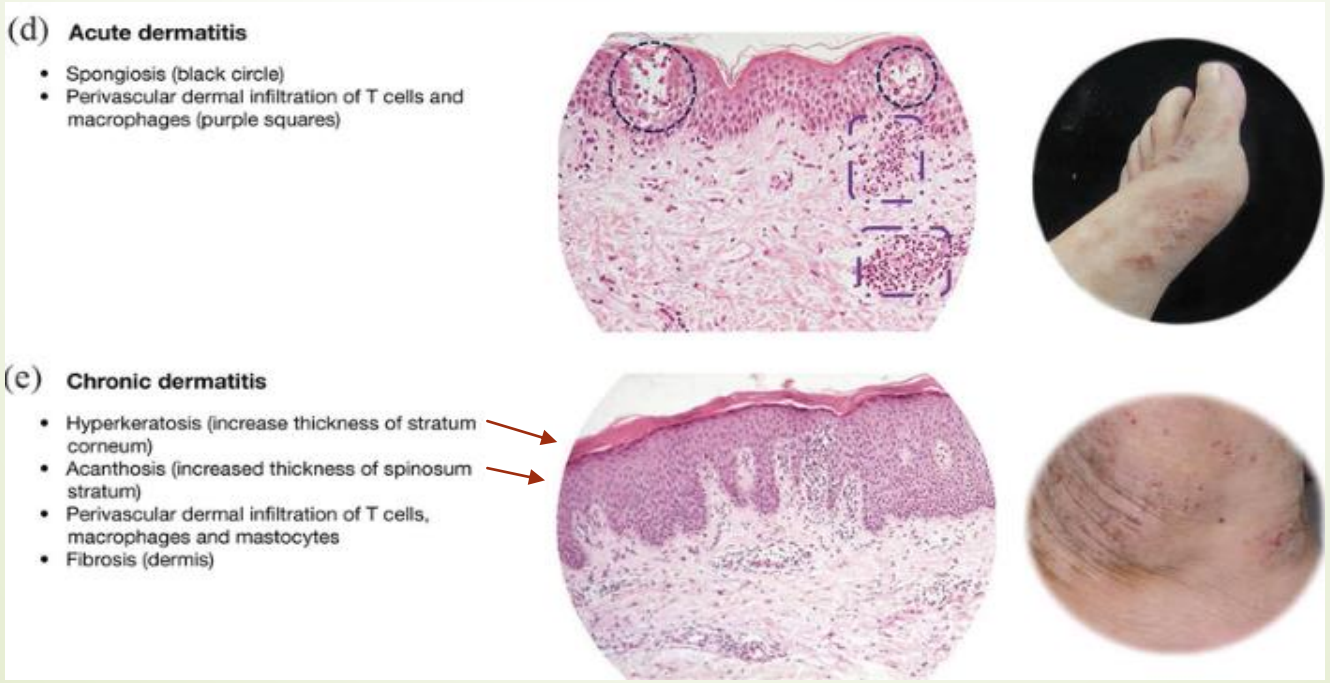
Introduction

- 1. According to duration:
  - 3. Chronic eczema:



Introduction

- 1. According to duration:
  - The histopathological hallmark of ***acute*** dermatitis is **spongiosis** (intra-epidermal vesicles). As eczema becomes more ***chronic***, there is tendency for it to become more **acanthotic** (thickened epidermis) and **less spongiotic**.



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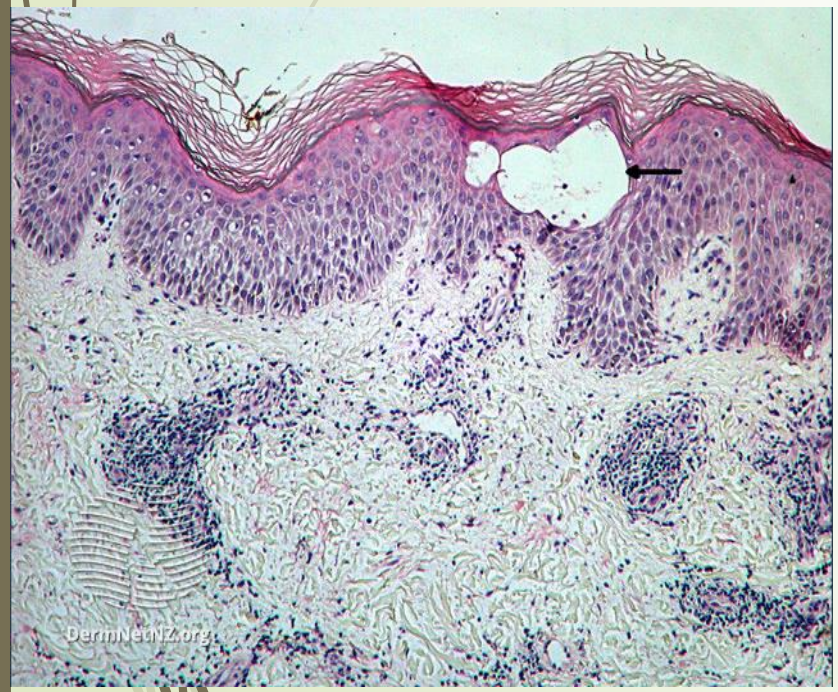
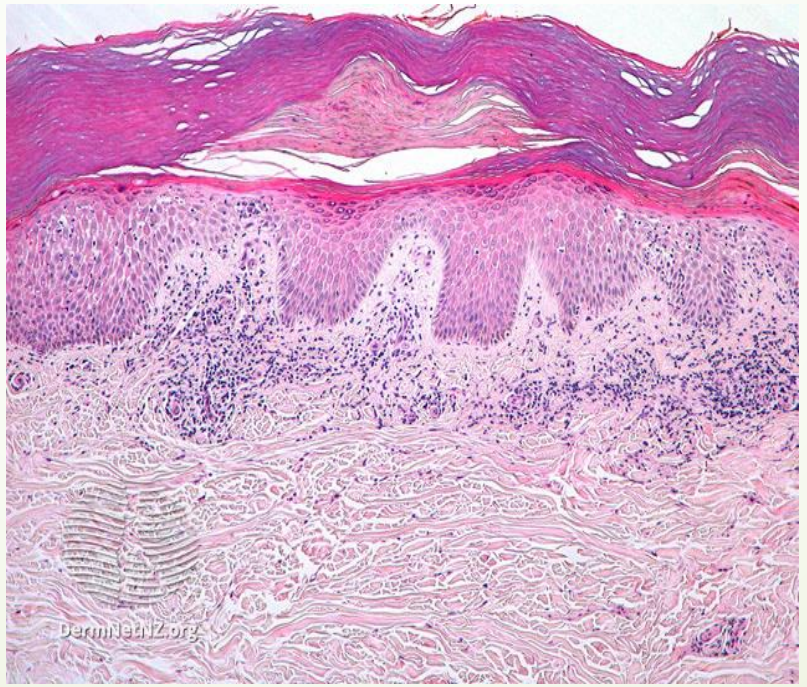
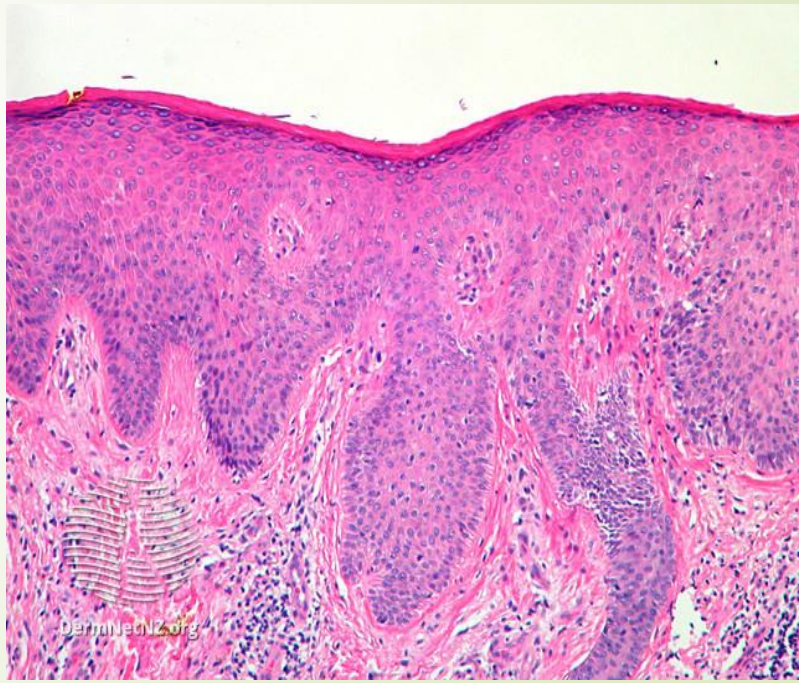
- 1. According to duration:



Acute



Sub-acute



Chronic

Introduction

- 2. According to cause:
  - 1. Endogenous eczemas:
    - Atopic dermatitis → **Most common**
    - Seborrheic dermatitis
    - Nummular (discoid) eczema
    - Stasis (varicose) eczema
    - Asteatotic eczema
    - Dyshidrotic eczema (pompholyx)
    - Neurodermatitis → lichen simplex chronicus

Introduction

- 2. According to cause:
  - 2. Exogenous eczemas:
    - Irritant contact dermatitis
    - Allergic contact dermatitis
    - Photosensitive dermatitis

Endogenous eczemas

Atopic dermatitis

- A chronic, pruritic, inflammatory skin disease that occurs most frequently in children but also affects adults A chronic, pruritic, inflammatory skin disease that occurs most frequently in children but also affects adults.
- Often associated with:
  - An elevated serum **Immunoglobulin E (IgE)** level.
  - Personal or family history of **atopy** → A genetically mediated predisposition to an excessive (IgE) reaction encompassing a triad of : Eczema, Asthma & Allergic rhinitis.

Atopic dermatitis

- Epidemiology:
  - US Prevalence: **8-12%** of children, **6-9%** of adults.
  - An increasing trend in incidence and prevalence of atopic eczema has been reported in the last few decades.
  - Africa, Oceania, and the Asia-Pacific region have higher rates of atopic dermatitis.
  - Age of onset: Most cases occur before the age of 5 (85%), with disease progression fading out as the child reaches puberty → *Note: Some cases progress into adult atopic dermatitis and others even start there.
  - Gender: Slight female predominance

Atopic dermatitis

- Risk factors:
  - Genetic factors:
    - A family history of **atopy** (eczema, asthma, or allergic rhinitis) is the **strongest risk factor** for atopic dermatitis.
    - Loss-of-function variants in the ***FLG* gene**, resulting in defective epidermal barrier, are a **major risk factor** for atopic dermatitis and other skin and allergic diseases
  - Environmental factors:
    - Climate
    - Air pollution
    - Urban vs. rural
    - Water hardness ???

Atopic dermatitis

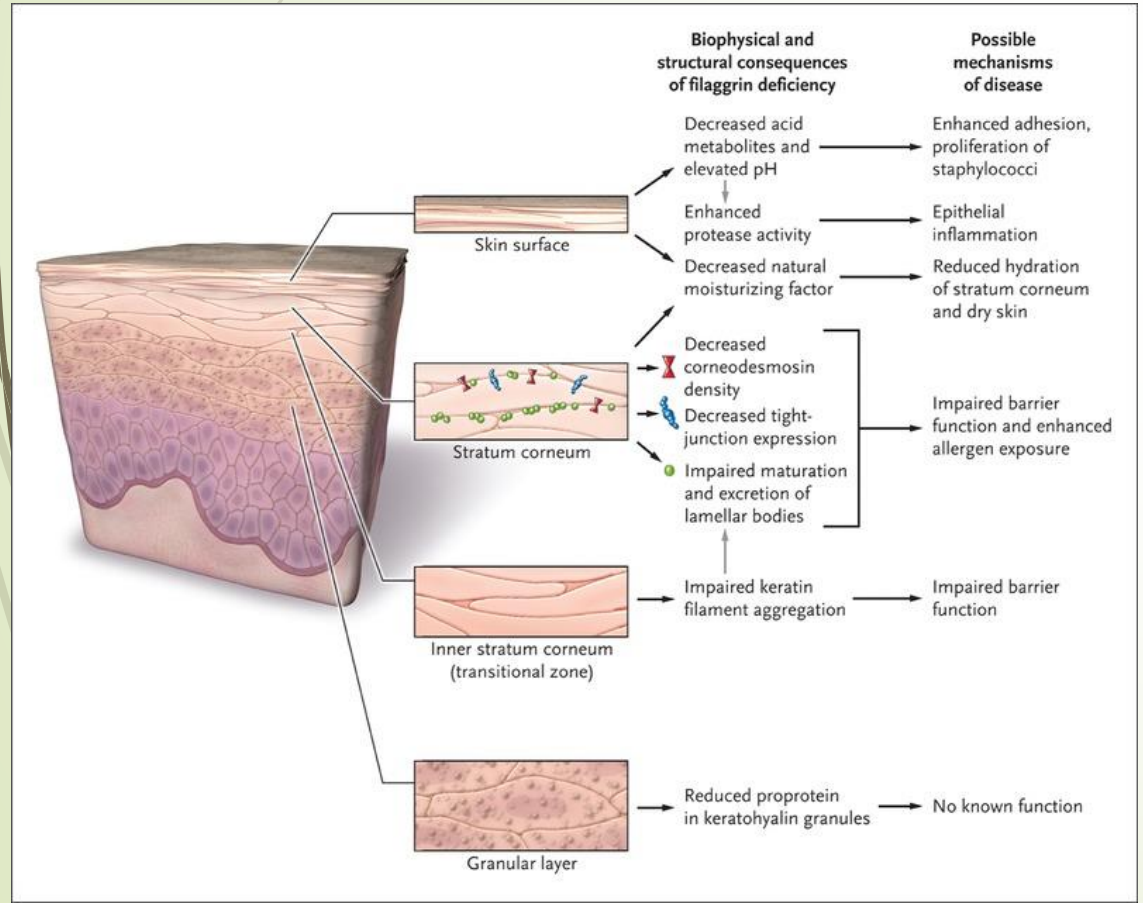
- Pathophysiology:
  - A multiplicity of mechanisms are involved in the pathogenesis of atopic dermatitis, including:
    - Epidermal barrier dysfunction
    - Genetic factors
    - Immune dysregulation and inflammation
    - Neuro-immune interactions
    - Alteration of cutaneous microbiome
  - Outside-in vs. Inside-out hypotheses:
    - Whether skin inflammation is initiated by skin barrier dysfunction ("outside-in" hypothesis) or by immune dysregulation ("inside-out" hypothesis) is still debated.

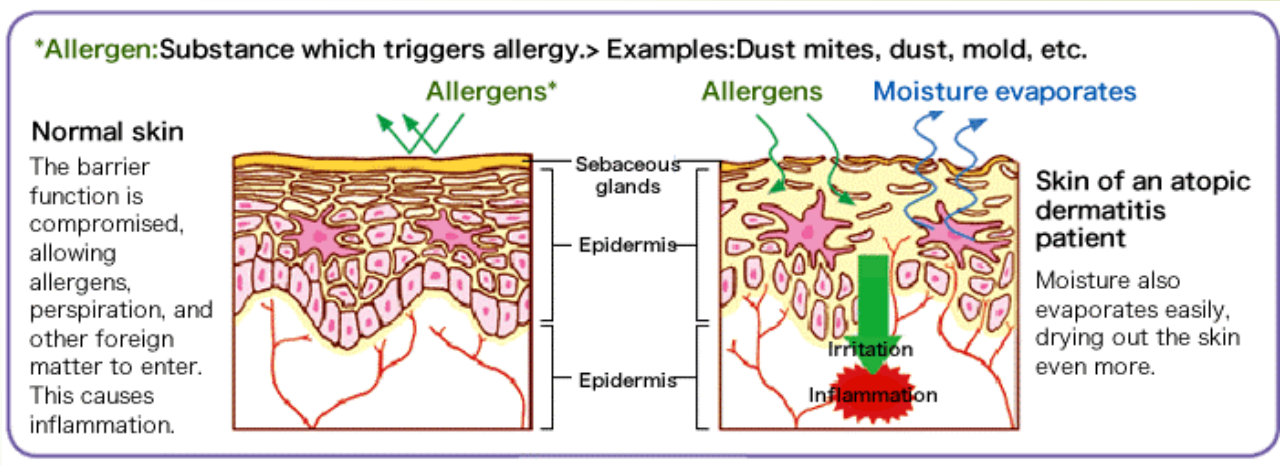
Atopic dermatitis

- Pathophysiology:
  - 1. Epidermal barrier dysfunction:
    - The epidermal barrier function primarily resides in the **stratum corneum.**
    - **Key abnormality** in the pathophysiology of atopic dermatitis → Basis of using moisturizes and emollients in management
    - Multiple factors contribute:
      - Reduced **Filaggrin** production → The filaggrin precursor profilaggrin is encoded by the ***FLG*** gene, located in the epidermal differentiation complex on chromosome **1q23.3** → Associated with disruption of keratinocyte differentiation, impaired corneocyte integrity and cohesion, impaired tight junction formation.
      - Imbalance between stratum corneum proteases (e.g. kallikrein) & anti-proteases (e.g. LEKTI)
      - Abnormalities of the tight junction function in stratum granulosum (e.g. claudin, occluding ..)
      - Microbial colonization and release of pro-inflammatory & inflammatory cytokines.

Atopic dermatitis

- Pathophysiology:





Atopic dermatitis

- Pathophyisology:
  - 2. Genetic factors:
    - Concordance rates of **80 percent** for monozygotic twins compared with **20 percent** for dizygotic twins
    - Loss-of-function variants in ***FLG*** → Encode for profilaggrin
  - 3. Immune dysregulation & inflammation:
    - Both the innate and acquired immune responses have a role in the pathogenesis of **Type 2 inflammation**
  - 4. Neuro-immune interactions:
    - Complex interactions between peripheral C-nerve fibers (itching) & Th2 cells
  - 5. Alteration of cutaneous microbiome → Overgrowth of *S. aureus*

Atopic dermatitis

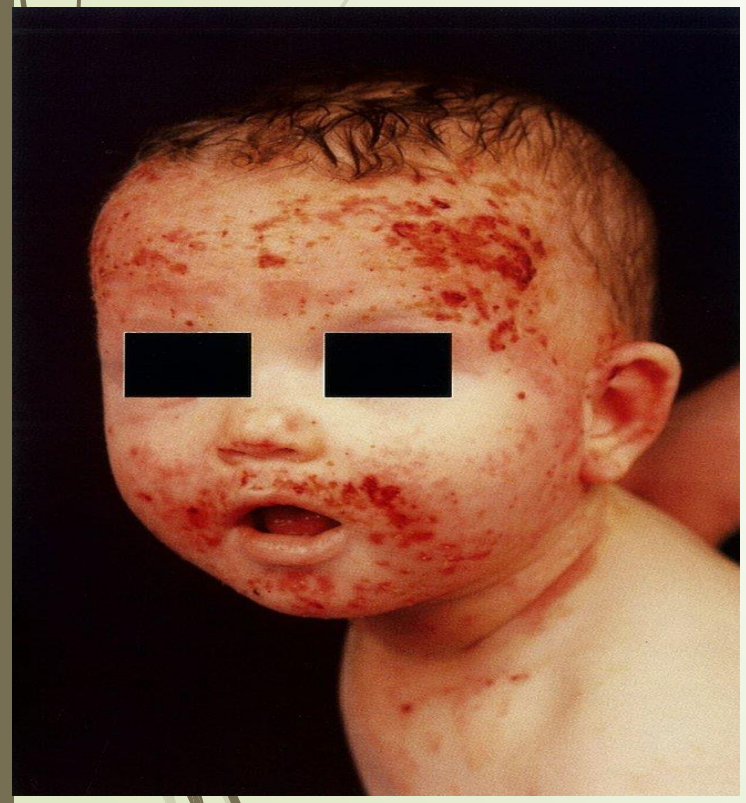
- Clinical manifestations:
  - Cardinal signs:
    - **Dry skin**
    - **Severe pruritus**
  - Acute vs. chronic presentation:
    - Acute → Intensely pruritic, erythematous papules and vesicles with exudation and crusting
    - Subacute/chronic → Dry, scaly, or excoriated, erythematous papules with progression to lichenification (Due to chronic scratching)

Atopic dermatitis

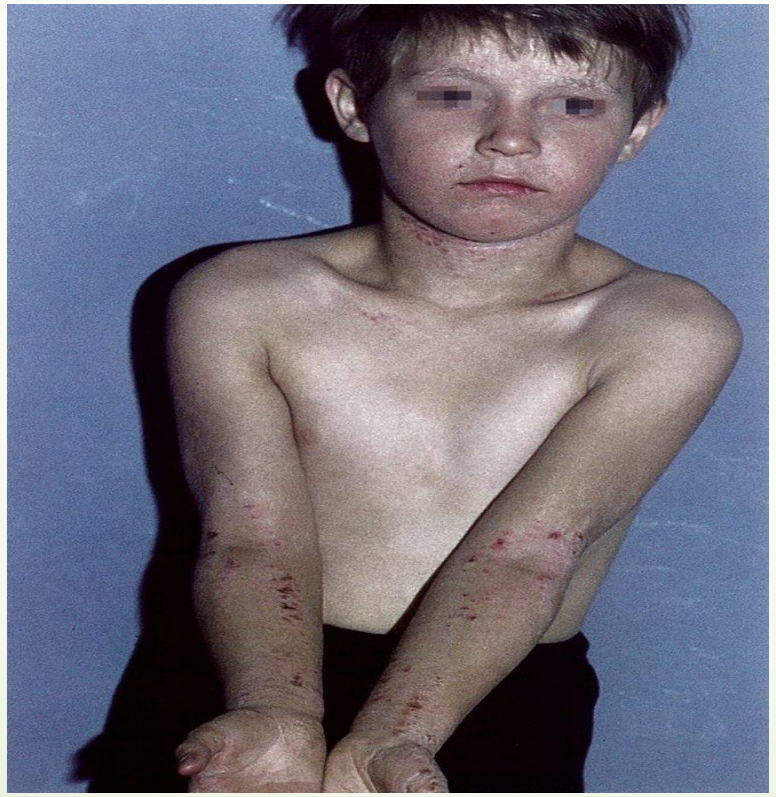
- Clinical manifestations:
  - Sites of eczema vary with age:
    - Infantile AD (0-2 yrs.) → face, head, and **extensor surfaces** of the extremities that usually spares the diaper area.
    - Childhood AD (2-12 yrs.) → **flexural creases** (antecubital fossa and popliteal fossa), skin folds, extensor surface of hands → Usually become **lichenified**
    - Adolescent/adult AD (>12 yrs) → Lichenified lesions and pruritus of **flexor surfaces** of the extremities → Antecubital fossae frequently involved → Adult AD may present as nummular eczema

Atopic dermatitis

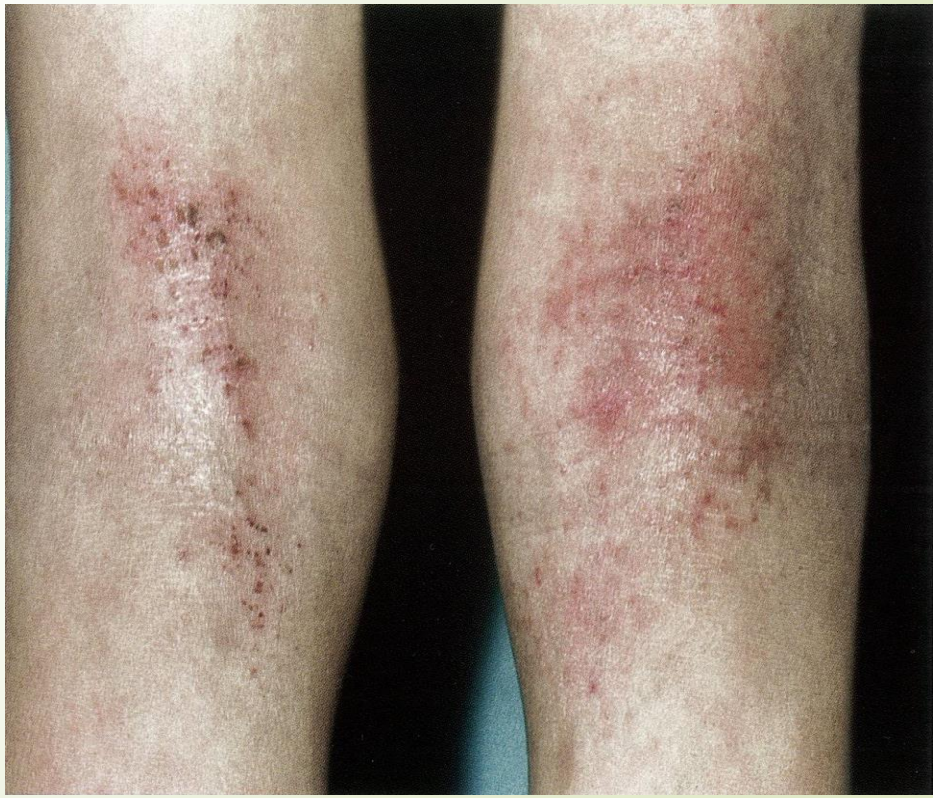
- Clinical manifestations:



Infant AD



Child AD



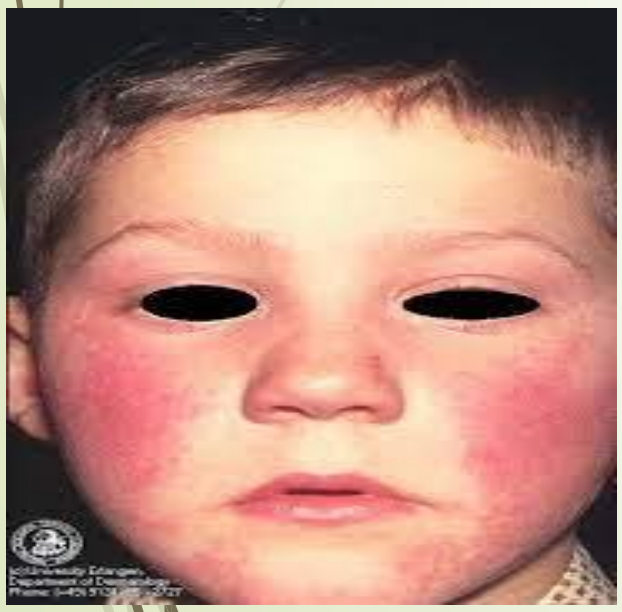
Adult AD

Atopic dermatitis

- Associated features:
  - Centrofacial pallor
  - White dermographism
  - Keratosis pilaris
  - Palmar hyperlinearity
  - Pityriasis alba
  - Dennie-Morgan infraorbital folds
  - Thinning or absence of the lateral portion of the eyebrows (Hertoghe's sign)
  - Juvenile plantar dermatosis
  - Nipple eczema

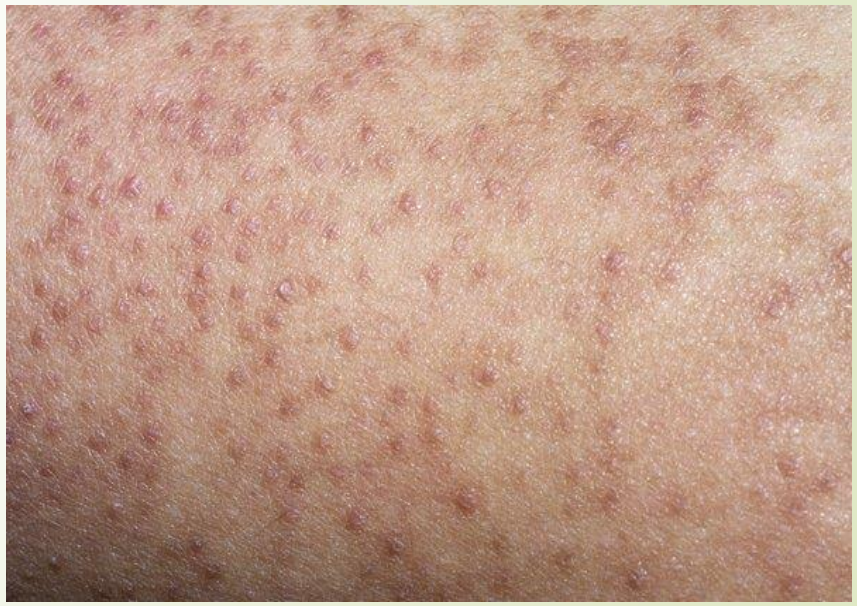
Atopic dermatitis

- Associated features:



Centrofacial pallor

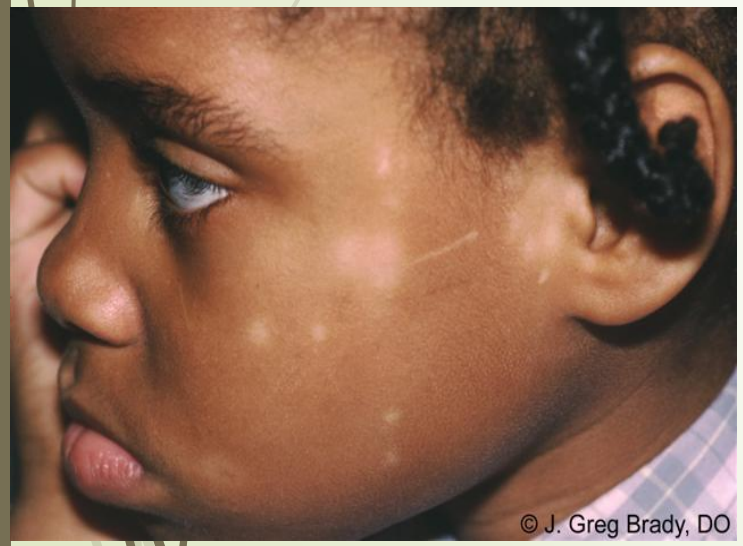
White dermographism



Keratosis pilaris

Atopic dermatitis

- Associated features:



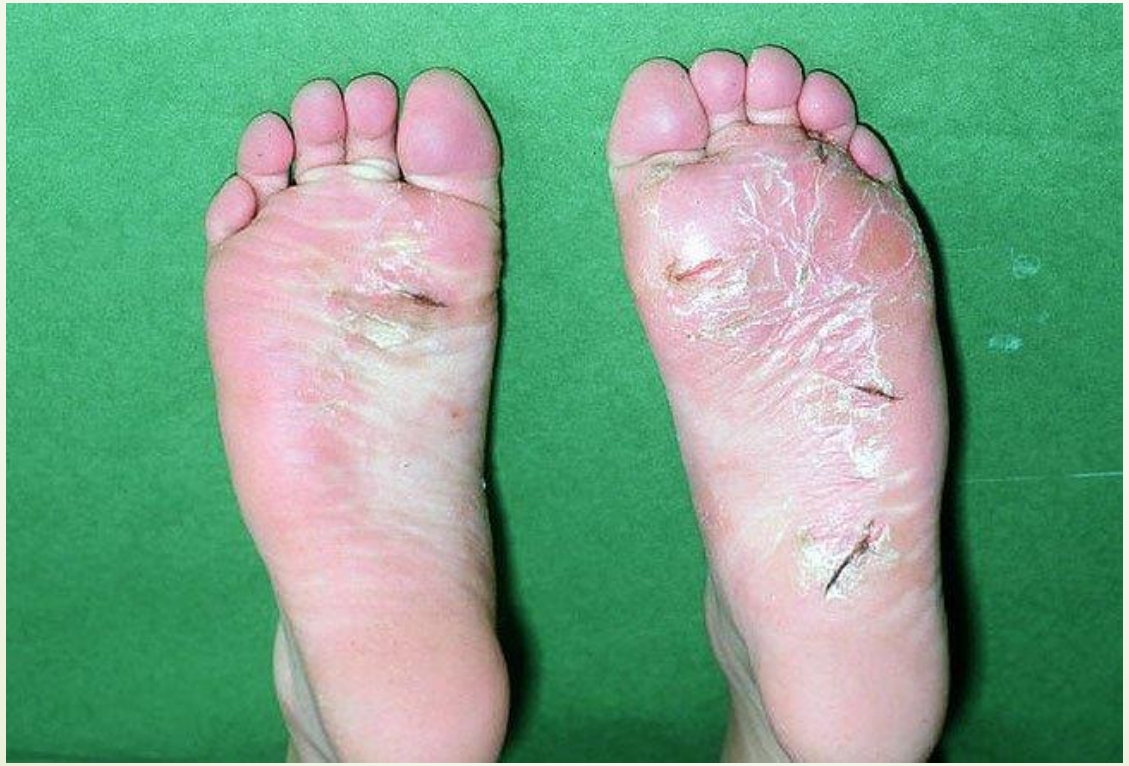
Pityriasis alba

Hertoghe's sign

Dennie-Morgan folds

Atopic dermatitis

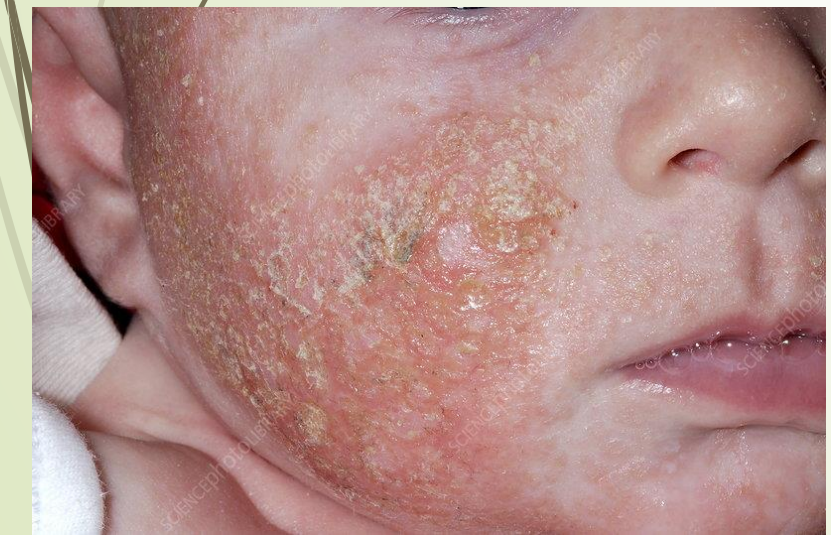
- Associated features:

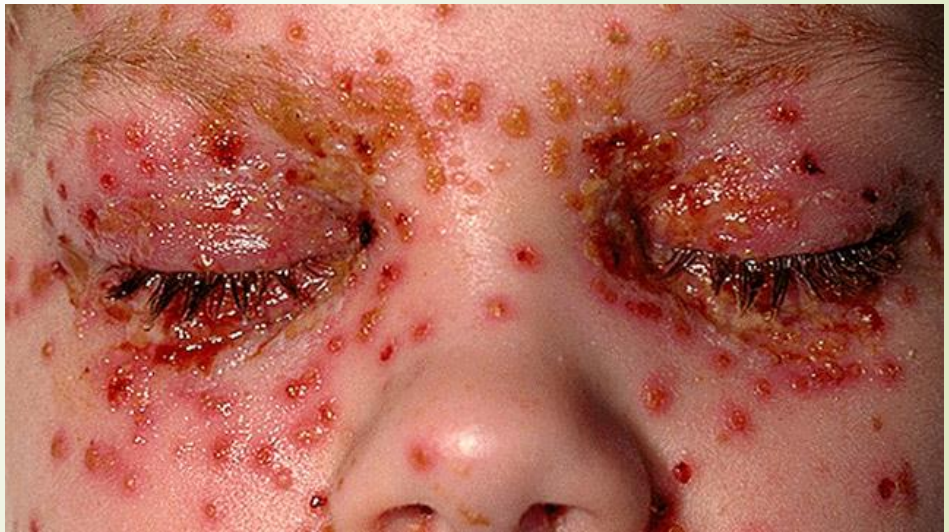


Juvenile plantar dermatosis

Atopic dermatitis

- Complications:
  - Secondary infections:
    - Bacterial → Impetigo (GAS or S. aureus)
    - Viral → Eczema herpeticum (HSV-1) → **Life-threatening** (Start Acyclovir)
    - Fungal → Tinea (*Trichophyton rubrum*)





Atopic dermatitis

- General management:
  - Identify & avoid irritants
  - Use emollients after baths
  - Treat co-morbidities
  - Treat secondary infections
- Treatment approach:
  - Very mild AD → Emollient monotherapy
  - Mild-to-moderate AD → Emollient + topical steroid/topical tacrolimus → First-line for acute flare-ups
  - Moderate-to-severe AD → Emollient + more potent topical steroid/calcineurin inhibitor + systemic therapy (Phototherapy, systemic steroids, systemic immunmodulation)

Seborrheic dermatitis

- Papulosquamous disease, which characteristically involves areas rich in sebaceous glands with high sebum production and large body folds.
- Chronic, relapsing, and usually mild form of dermatitis that occurs in infants and in adults.
- The severity may vary from minimal, asymptomatic scaliness of the scalp (dandruff) to more widespread involvement.
- Biphasic incidence with a **male** predominance in adults:
  - Infants between the ages of 2 weeks and 12 months → Commonly 1st 3 months
  - Adolescence and adulthood
- Occasionally associated with HIV infections, Parkinson's disease & other neurological diseases.

Seborrheic dermatitis

- Pathogenesis:
  - Not entirely known
  - Occurs in body sites with increased number of sebaceous glands
  - May involve the lipid-dependent fungus *Malassezia furfur* which thrives on sebum thus causing dermatitis

Seborrheic dermatitis

- Clinical manifestations:
  - In infants:
    - Scalp (vertex and frontal areas; the **'cradle-cap'** area)
    - Face (forehead, eyebrows, eyelids, nasolabial folds, temple)
    - Diaper area
    - Retroauricular folds
    - Neck
    - Axillae

Seborrheic dermatitis

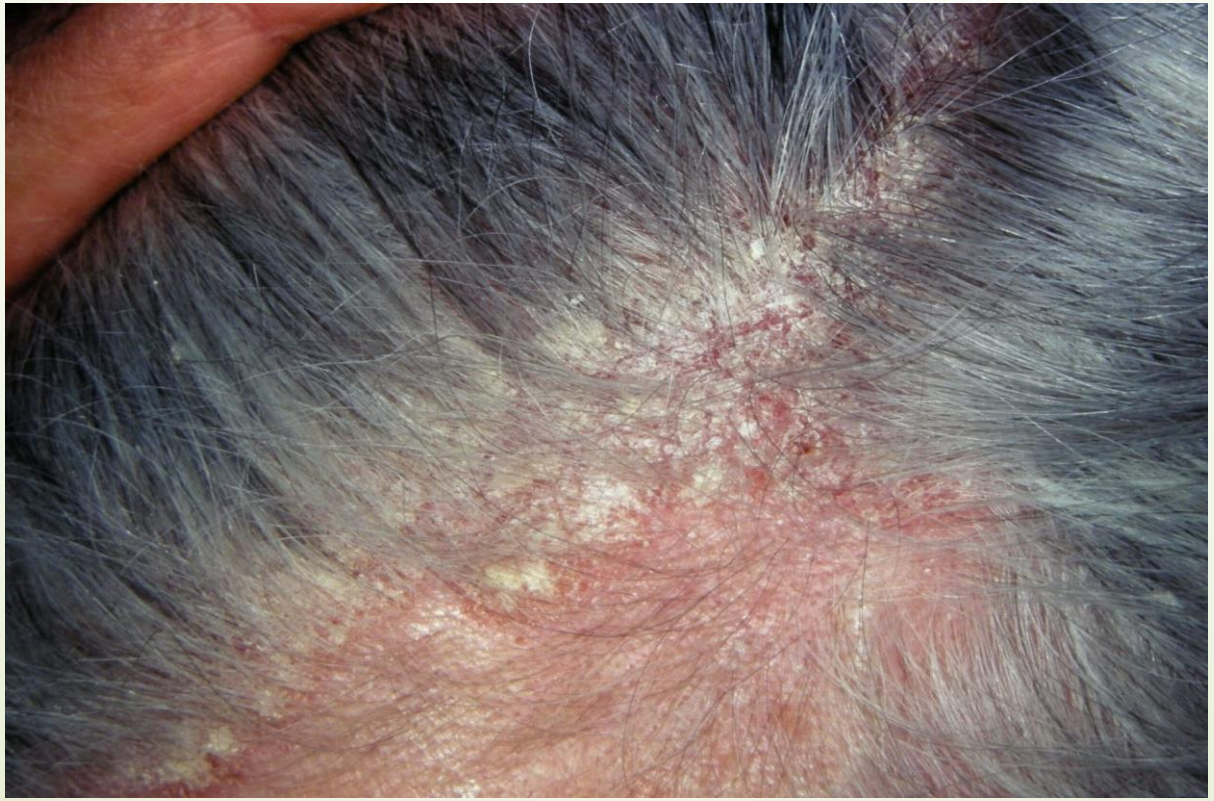
- Clinical manifestations:
  - In infants:

Seborrheic dermatitis

- Clinical manifestations:
  - In adults:
    - Scalp → Earliest sign is **dandruff** → Accompanied later by itching & inflammation → retro-auricular fissuring
    - Face → Scaling & erythema of forehead, medial portion of eyebrows, eyelids, nasolabial folds, lateral part of nose and retro-auricular region
    - Trunk → Papules, greasy scales
    - Flexural areas → Erythema, greasy scaling and secondary infection

Seborrheic dermatitis

- Clinical manifestations
  - In adults:



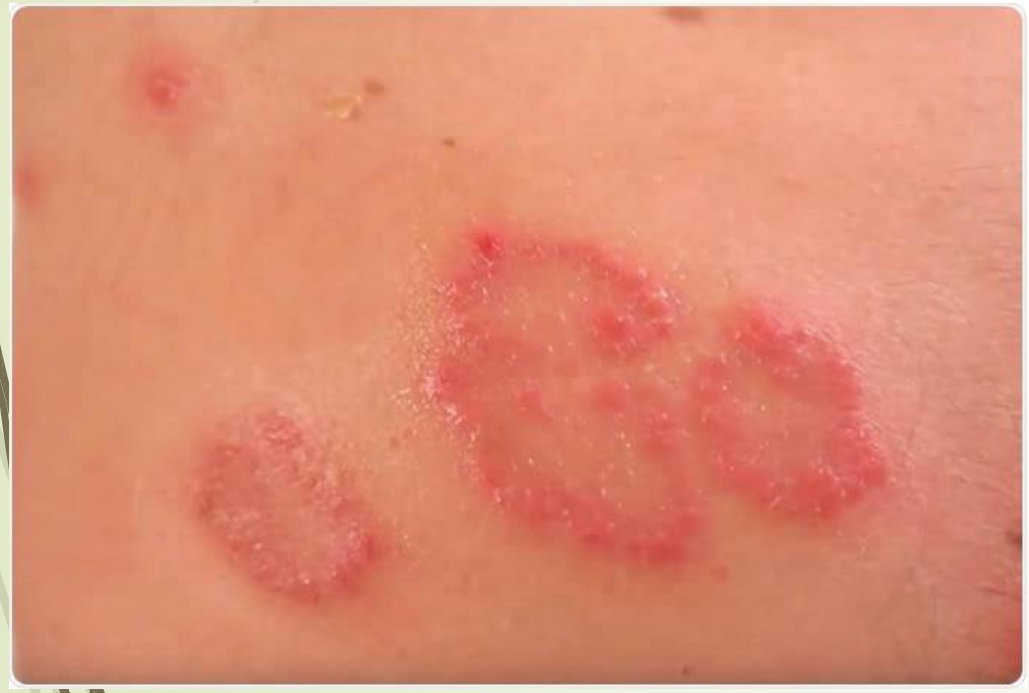
Seborrheic dermatitis

- Management:
  - Treatment over many years with no definitive cure
  - Topical hydrocortisone is effective → Recurrence after discontinuation
  - Steroid lotions/tar shampoos for the scalp
  - Ketoconazole shampoo/cream
  - Imidazole/hydrocortisone combinations

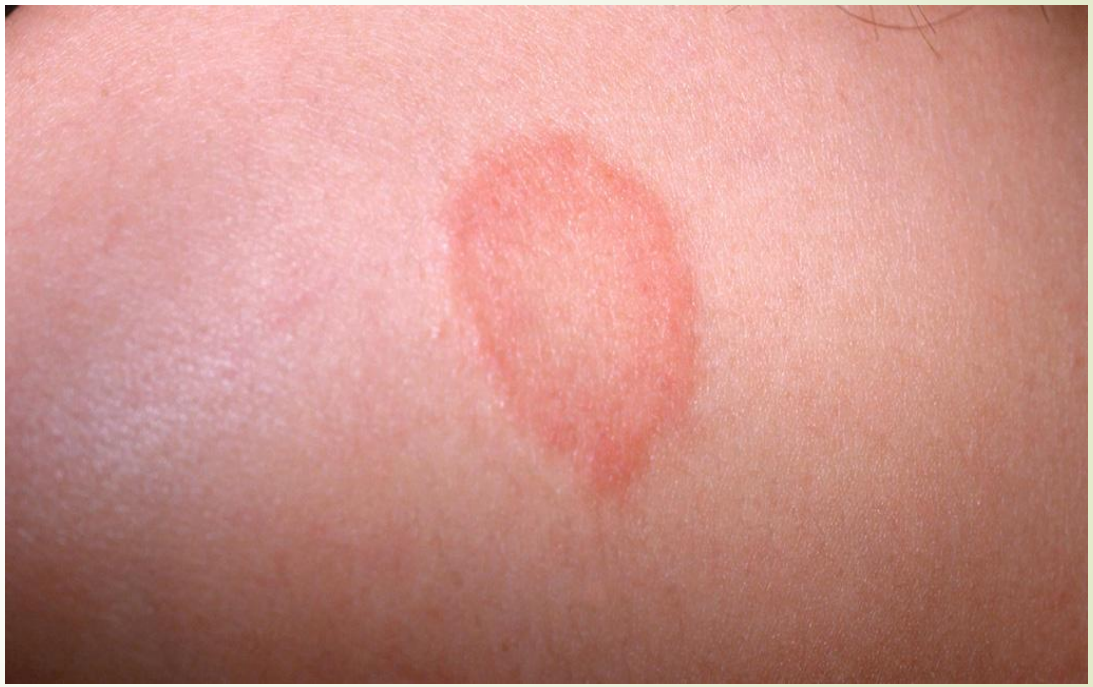
Nummular (discoid) eczema

- Chronic, inflammatory skin disease characterized by multiple pruritic, coin-shaped, eczematous lesions involving the extremities and, less commonly, the trunk.
- More in patients above 50.
- Male predominance.
- Pathogenesis is incompletely understood
- Treatment is with emollients & topical steroids
- Sometimes mistaken with **Ringworm** infections → More symptoms & multiple lesions favour **Nummular eczema**

Nummular (discoid) eczema



Nummular eczema

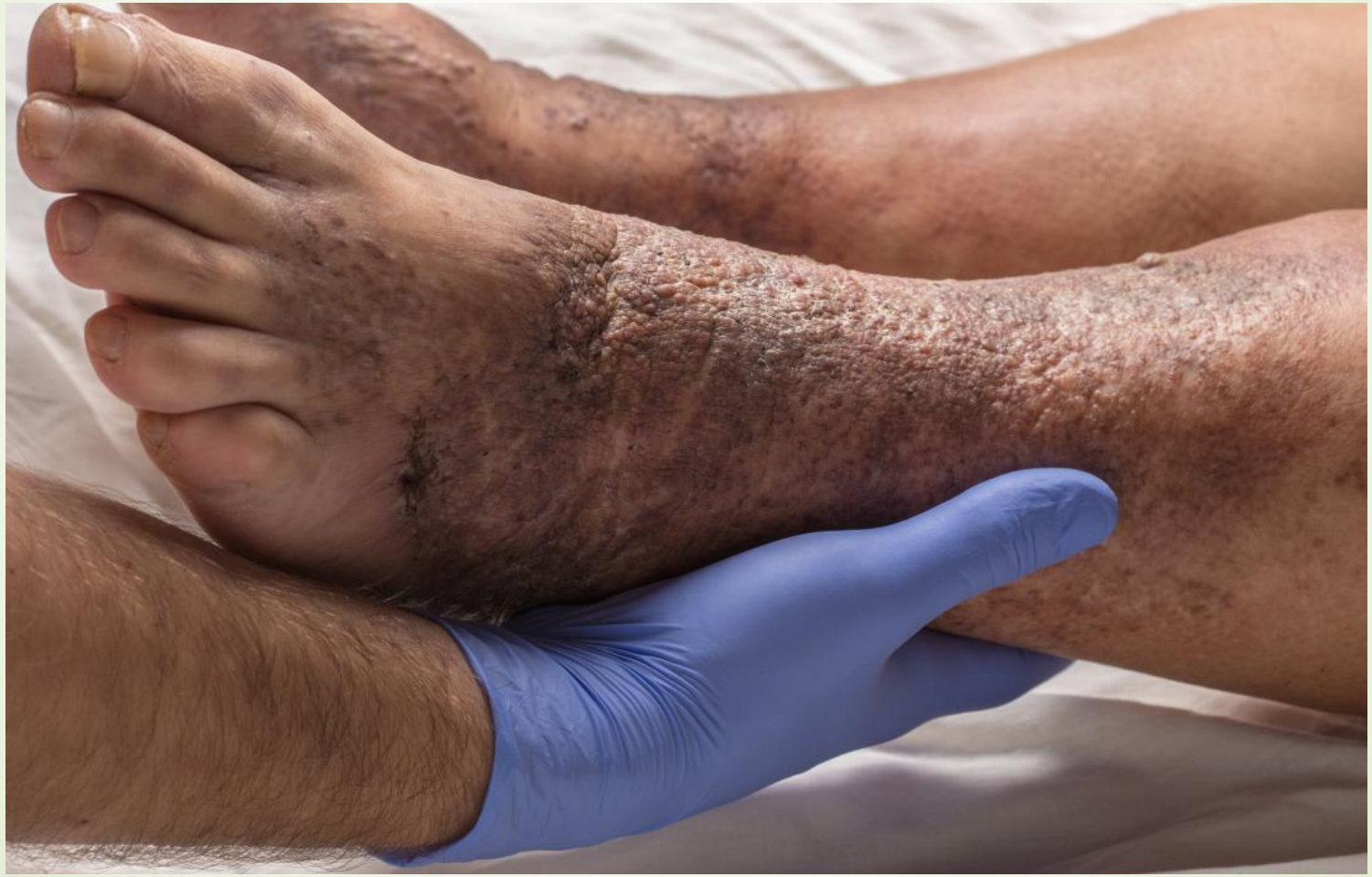


Ringworm

Stasis (varicose) dermatitis

- Inflammatory dermatosis of the **lower extremities** occurring in patients with **chronic venous insufficiency**, often in association with varicose veins, dependent chronic edema, hyperpigmentation, lipodermatosclerosis, and ulcerations.
- May rarely involve the **upper limbs** in patients with artificial arteriovenous (AV) fistulas for hemodialysis, or congenital AV malformations.
- Characterized by diffuse erythema, scaling, crusting & itching over the insufficient areas.
- Treatment → Treat initial cause → Apply emollients & moderately-potent topical steroids

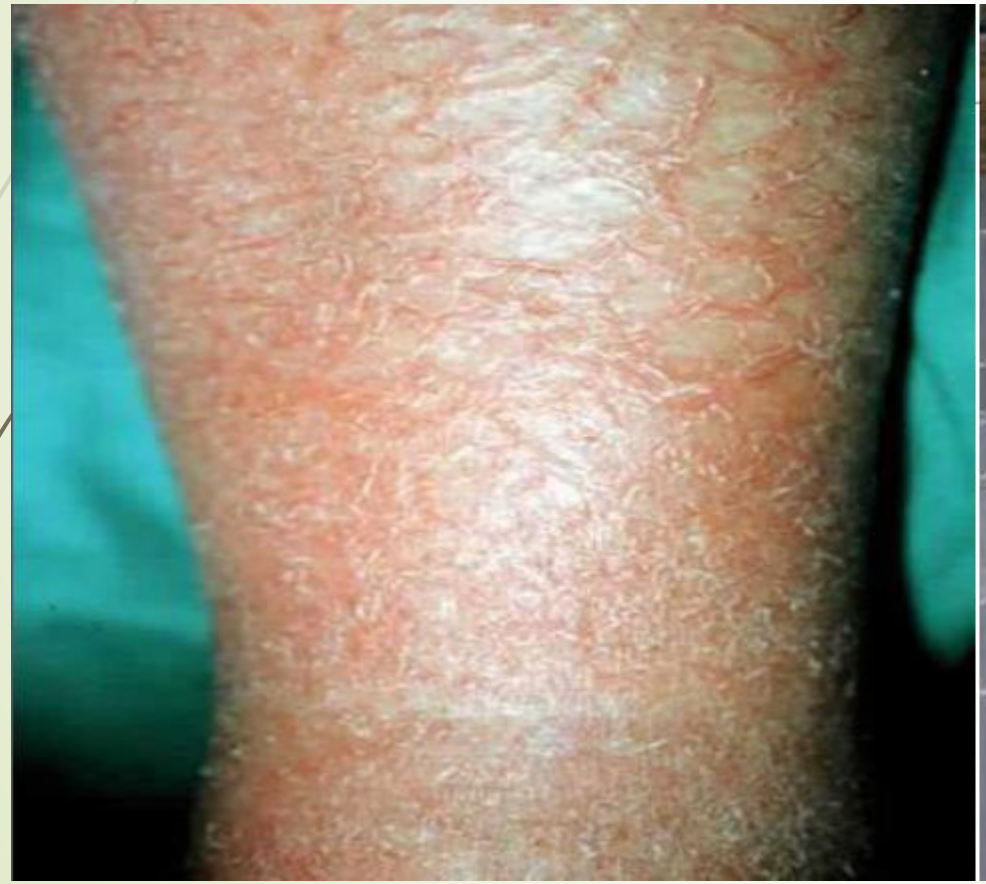
Stasis (varicose) dermatitis



Asteatotic dermatitis

- Pruritic dermatitis that typically occurs on the lower extremities **(shins)** of older individuals with **dry skin**.
- Its incidence peaks during cold winter months.
- Exacerbating factors:
  - Low environmental humidity
  - Exposure to detergents or irritants
- Pathogenesis → Water loss from the stratum corneum due to age-related skin barrier impairment.
- Treatment → Repeated used of emollients + mild potency topical steroids

Asteatotic dermatitis

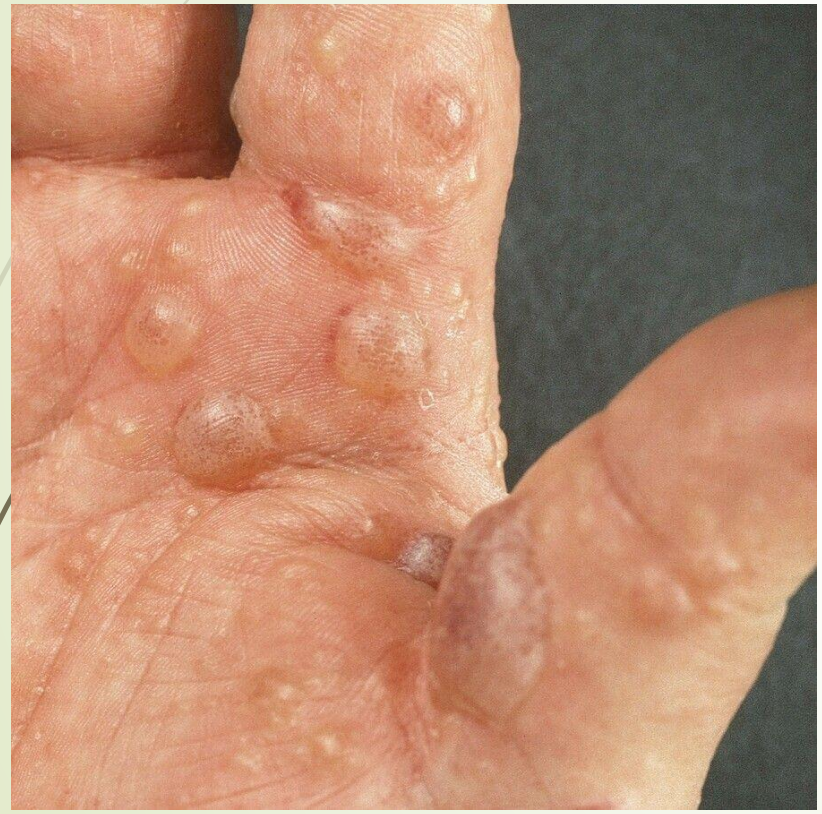


Crazy-paving appearance

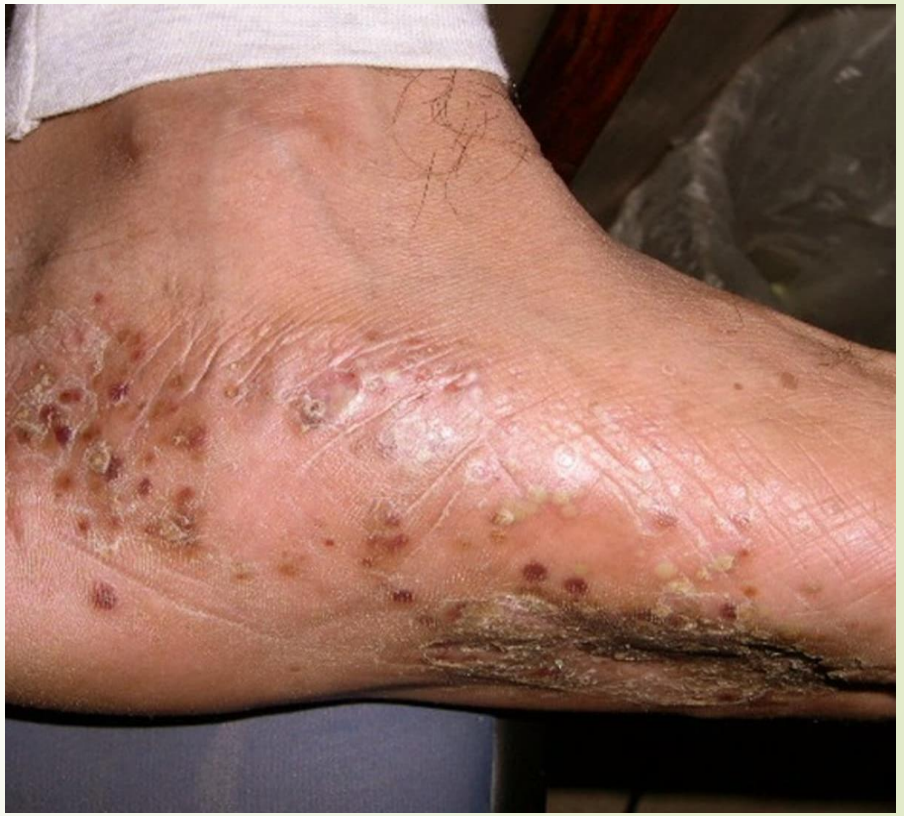
Dyshidrotic eczema (pompholyx)

- Also known as: **Acute palmoplantar eczema**
- An intensely pruritic, chronic and recurrent, vesicular dermatitis of *unknown etiology* that typically involves the **palms** and **soles** and lateral aspects of the fingers → Paronychia with nail dystrophy in severe cases
- Occurs most commonly in young adults
- Multiple small, deep-seated vesicles on the palmar or plantar skin that may coalesce to form large bullae
- **Superinfection is common**
- Treatment → $KMnO_4$ soaks + potent topical steroid. Add systemic antibiotic if needed.

Dyshidrotic eczema (pompholyx)



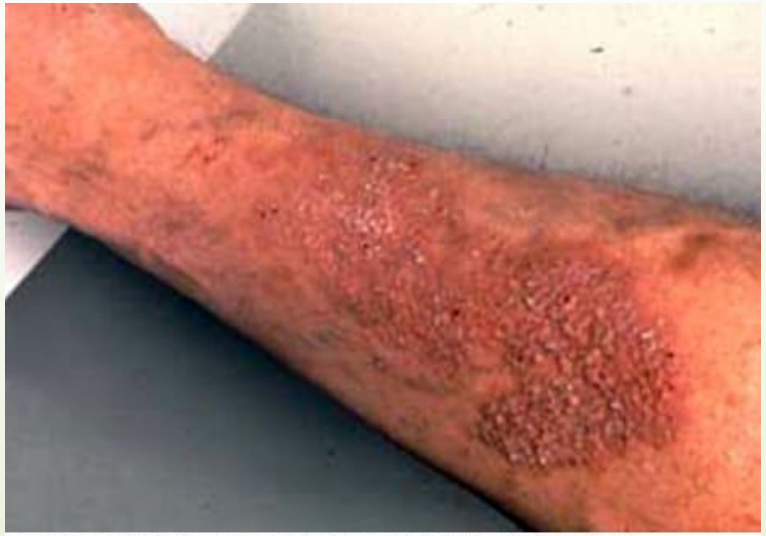
Cheiropompholyx



Podopompholyx

Neurodermatitis (Lichen simplex chronicus)

- A skin condition that starts with an itchy patch of skin, where scratching it makes it **more itchier**
- The itch-scratch cycle causes the affected skin to become thick and leathery **(lichenification**)
- Treatment → Breaking the itch-scratch cycle by resisting the urge to scratch + OTC anti-pruritic medications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Exogenous eczemas

Irritant contact dermatitis

- A **localized**, inflammatory skin response to a wide range of chemical or physical agents.
- **Most common** type of contact dermatitis.
- Direct cytotoxic effect of irritant agents → **NOT** immune-mediated.
- If applied in **high enough** concentrations, may cause an eczematous response in previously **normal skin**
- Clinical manifestations as well as histopathological features of ICD are similar to those of other acute or chronic eczematous dermatites, including atopic dermatitis → Occupational history & physical examination give clues

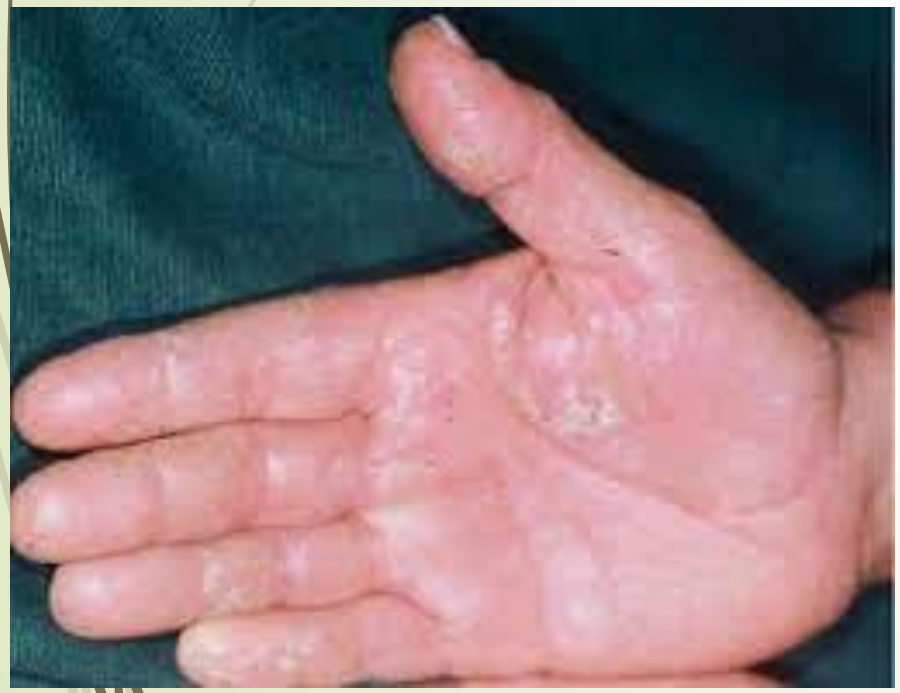
Irritant contact dermatitis

- Types of irritants:
  - 1. Chemical irritants:
    - Acids → Coagulative necrosis
    - Alkali → Liquefactive necrosis + lipid saponification → cellular swelling
    - Solvents → Remove lipids & damage cell membranes
    - Water → Prolonged contact with water causes swelling of the stratum corneum → Disruption of the intercellular lipids → Enhancement of skin permeability and susceptibility to irritants
    - Oxidizing agents (e.g. bleach, benzoyl peroxide) → cytotoxic agents

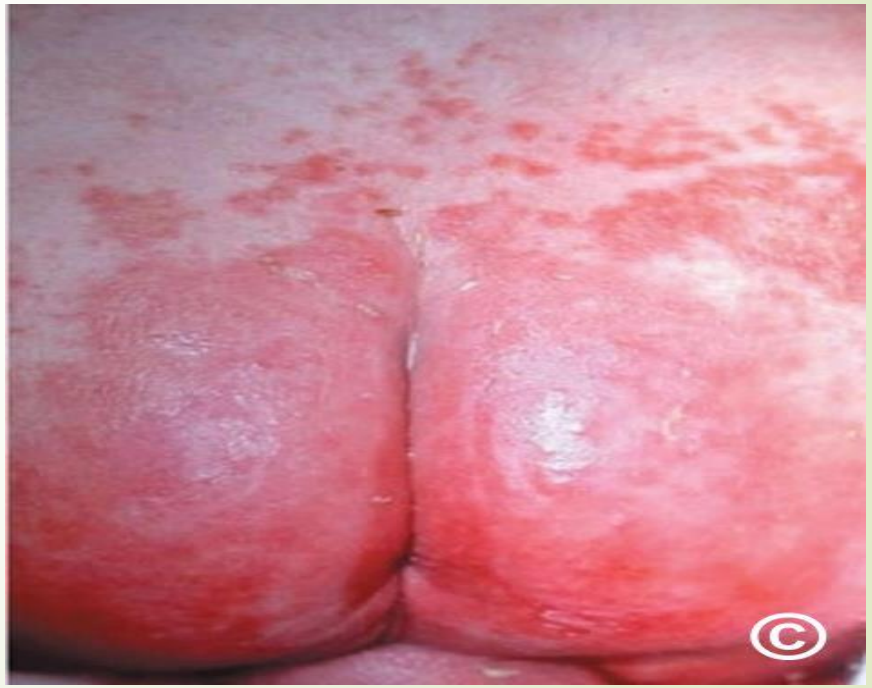
Irritant contact dermatitis

- Types of irritants:
  - 2. Physical irritants: → Chronic microtrauma damages the stratum corneum and releases preformed cytokines from keratinocytes
  - Metal tools
  - Wood
  - Fiberglass
  - Plant parts (e.g. thorns, spines, sharp-edged leaves)
  - Paper
  - Dust/soil

Irritant contact dermatitis



Wear-and-tear dermatitis

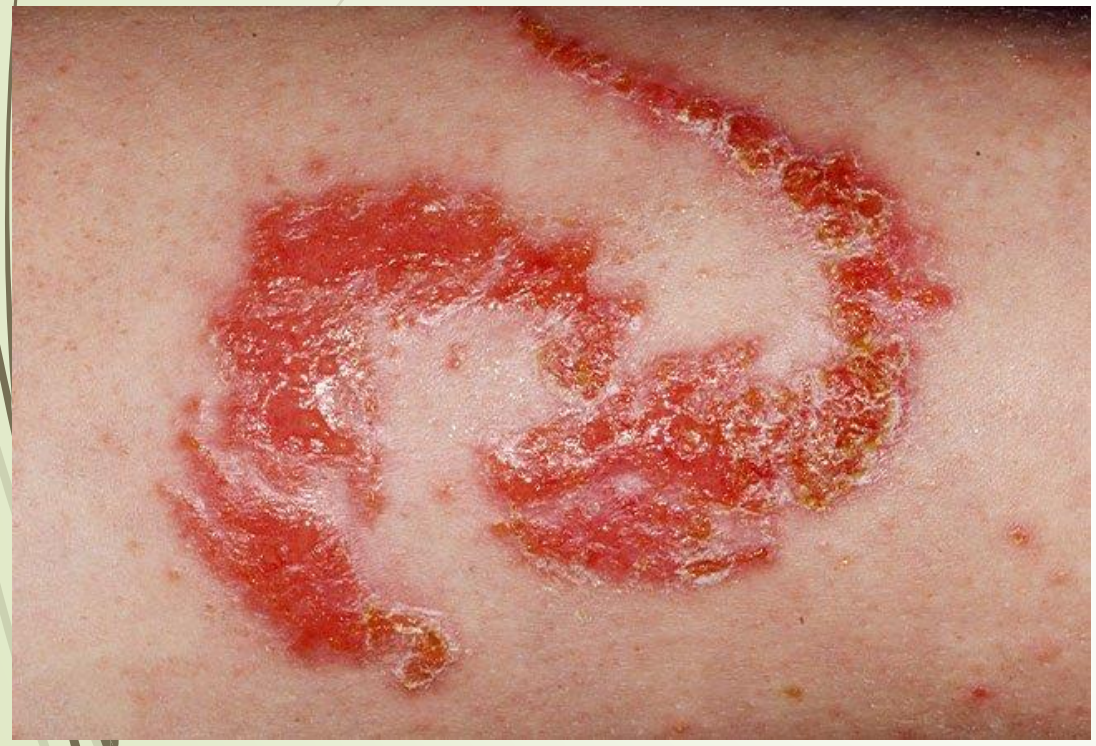


Napkin dermatitis

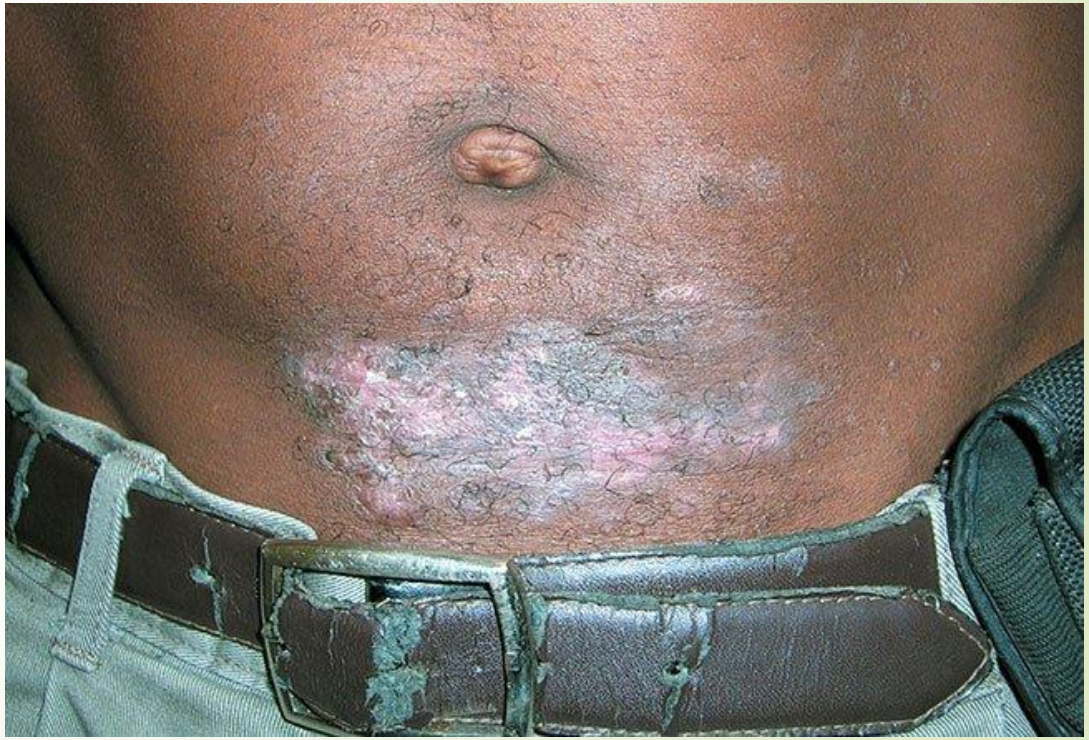
Allergic contact dermatitis

- **Type 4** hypersensitivity reaction ("delayed", "T-cell mediated") in response to a sensitizer or allergen
- Will **NOT** occur in **normal patients** (without sensitization) even if exposed to a large concentration of the allergen.
- Brief exposure in a previously sensitized individual may provoke a severe episode of dermatitis
- Clinically & histopathologically resembles ICD & other forms of eczema → Occupational history, history of predisposition & physical examination give clues

Allergic contact dermatitis



Henna tattoo contact dermatitis



Nickel contact dermatitis

Allergic contact dermatitis

- Top 10 contact allergens:

Nickel sulfate	Metals, metals in clothing, jewelry, catalyzing agents
Neomycin sulfate	Usually contained in creams, ointments
Balsam of Peru	Topical medications
Fragrance mix	Fragrances, cosmetics
Thimerosal	Antiseptics
Sodium gold thiosulfate	Medication
Formaldehyde	Disinfectant, curing agents, plastics
Quaternium-15	Disinfectant
Bacitracin	Ointments, powder
Cobalt chloride	Cement, galvanization, industrial oils, cooling agents, eyeshades

Allergic contact dermatitis

Irritant vs. allergic contact dermatitis		
	Irritant contact dermatitis	Allergic contact dermatitis
Type of reaction	• Nonimmunologic reaction	• Type IV hypersensitivity reaction
Description	• The agent has a direct cytotoxic effect on skin cells and the inflammatory response is secondary to cutaneous damage, not to the agent itself.	• Presensitized CD4+ T cells recognize antigens on antigen-presenting cells, leading to the release of inflammatory cytokines, while presensitized CD8+ T cells recognize antigens on somatic cells, leading to cell-mediated cytotoxicity.
Subjects at risk	• Health care workers • Individuals working in the cosmetic industry, hairdressers • Metal workers	• Predisposed individuals
Skin involvement	• Borders typically well defined • Limited to contact area	• Ill-defined borders • Extends beyond contact area
Onset	• Subacute to chronic • Repeated exposure to causative agent is necessary (no sensitization)	• Acute to subacute (onset is usually rapid) • Patients need to be sensitized to allergen first
Clinical features	• Eczema with erythema, desquamation, and fissures • Pain, stinging, and burning	• Eczema with erythema, edema, bullae, and vesicles • Itching and pruritus
Diagnostic tests	• No specific test; history and physical examination are usually sufficient for diagnosis	• Positive patch test

Allergic contact dermatitis

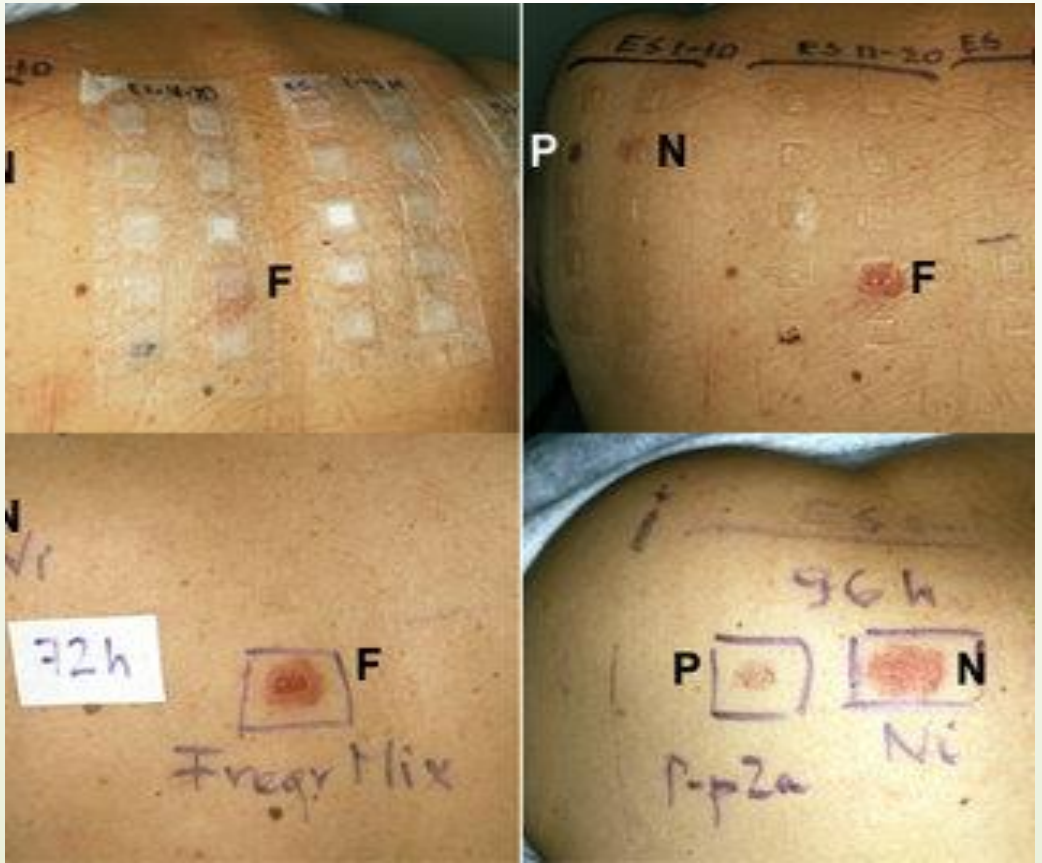
- Patch testing:
  - Identification of specific allergens in allergic contact dermatitis.
  - In **sensitized individuals**, primed antigen-specific T lymphocytes of the Th1 phenotype circulate throughout the body and are able to **recreate a delayed-type hypersensitivity reaction** when non-irritating concentrations of the antigen are applied to normal skin.
  - Patch testing is usually performed on the upper back using a standard series of allergens that are known to cause ACD in a specific geographic region.
    - Other areas of skin result in **false-negatives**
    - Application on areas with existing dermatitis result in **false-positives**

Allergic contact dermatitis

- Patch testing:
  - Several types:
    - Closed test → Most commonly used
    - Open test
    - Semi-open test
  - Interpretation:
    - Initial observation → Patches are removed after **48 hours,** and a response is observed after **15-60** minutes → Erythema, swelling or vesiculations.
    - Second observation → Patches are removed on **Day 4 or 5**
      - To distinguish irritant reactions (**which fade**) from true allergic reactions (**which persist**)
      - To identify allergic reactions that do not appear at the time of initial patch removal

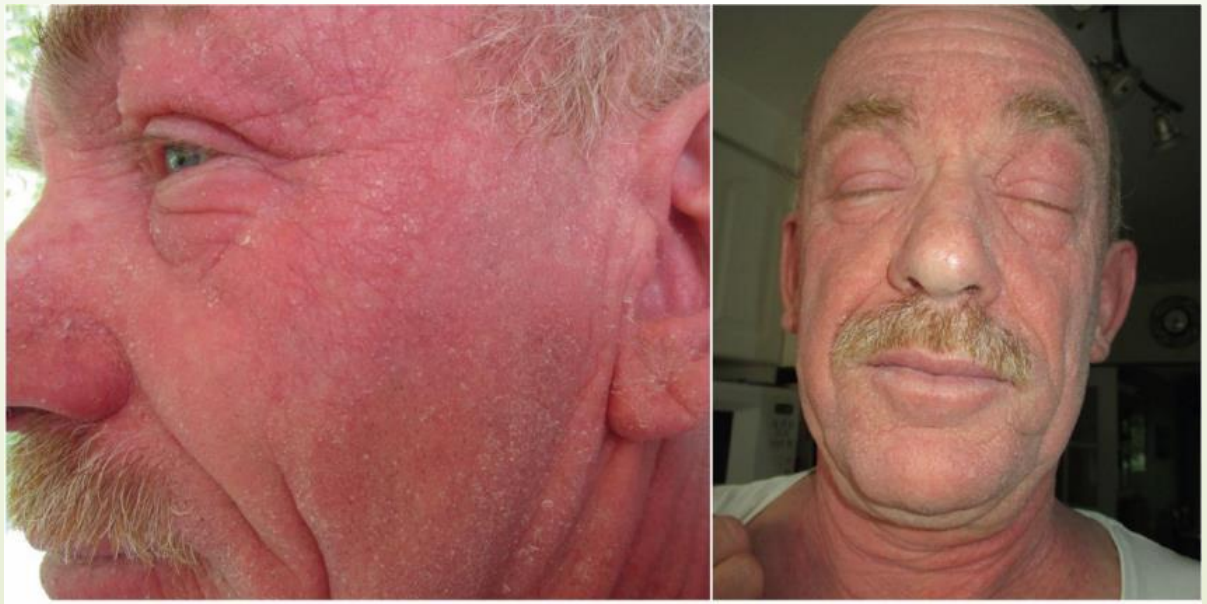
Allergic contact dermatitis

- Patch testing:



Photodermatitis

- Interaction of light and chemicals absorbed by the skin
- Allergic (e.g. chronic actinic dermatitis) vs. Toxic
- Drugs that cause photosensitivity (e.g. doxycycline, isotretinoin)
- Phytophotodermtitis due to contact with plant material and sulight.



Thank You