#### Seborrhoeic dermatitis

#### **Definition:**

 Seborrheic dermatitis is a common mild chronic eczema affecting skin regions with high sebum production.

### **Epidemiology**

- Age of onset: 2 forms of seborrheic dermatitis:
- 1. Infantile form: confined to the first 3 months of life, self-limited.
- 2. Adult form: peaks in the 4th 6th decades, chronic.
- Incidence / prevalence: adult seborrheic dermatitis is probably the most common type of eczema with prevalence of about 5%.
- .Sex: men are affected more common than women •

### **Aetiopathogenesis**

- (1) Role of sebum overproduction (seborrhea).
- (2) Role of altered sebum composition:
- In patients with seborrheic dermatitis, triglycerides and cholesterol are elevated but squalene and free fatty acids are significantly decreased.
- Free fatty acids (which have antimicrobial effect) are formed from triglycerides by bacterial lipases, produced by the lipolytic P. acnes.
- (3) Role of *Malassezia*:
- The genus *Malassezia* is part of the normal resident skin flora.

### **Clinical features:**

- Skin lesions: well-defined red patches with yellowish "greasy" scales sometimes itchy.
- Sites: areas rich in sebaceous glands scalp, face, ears, pre sternal region (central chest) and the intertriginous areas
- Clinical forms: Infantile / Adult
- (A) Infantile seborrheic dermatitis
- Scalp affection (cradle cap): mild greasy scales
- —» a coherent scaly and crusty mass covering most of the scalp (cradle cap).
- Trunk affection (psoriasiform, flexures and napkin area).

- (B) Adult seborrheic dermatitis
- Scalp affection:
- *Dandruff:* the common mildest form non-inflammatory.
- **■** Inflammatory
- Face: symmetric affection of the forehead, medial eyebrows, upper eyelids, naso-labial folds
- *Trunk:* morphological variants include petaloid (petal-shaped), flexural (intertrigo-like), eczematous plaques, pityriasiform and follicular.
- Generalized / Erythrodermic.

# <u>Prognosis</u>

Adult form has a chronic relapsing course (infantile form is self-limited).

# **Treatment**

- 1. General skin care (important for infants): emollients, mild shampoos to remove scalp scales and crusts and avoidance of irritation (e.g. strong keratolytic shampoos and mechanical measures to remove the scales from the scalp)
- 2. Topical antifungals e.g. Ketoconazole cream (2%) or shampoos and zinc pyrithione and tar shampoos.
- 3. Topical anti-inflammatory drugs: corticosteroids / calcineurin inhibitors.



#### Venous eczema

Synonyms(Stasis dermatitis, Gravitational dermatitis, Varicose eczema, Congestion eczema)

## **Definition**

Stasis dermatitis is a common component of the clinical spectrum of chronic venous insufficiency of the lower extremities (i.e. it may be associated with other signs of venous hypertension).

#### **Development of venous hypertension of the lower limbs:**

- This is linked to the upright position and valvular incompetence of the deep leg veins.
- Impact on the microvasculature:

Venous hypertension slows the blood flow in the micro vasculature —> distends the capillaries —> damages the capillary permeability barrier —▶

- passage of fluid into the tissue (edema).
- passage of proteins (particularly fibrin) into the tissue —>
  deposited around the vessels as hyaline cuffs —> inhibit oxygen
  diffusion and metabolic exchange.
- extravasation of erythrocytes (purpura) and hemosiderin deposition
   —» free iron ions —> proinflammatory effect.
- release of inflammatory mediators —> peri-capillary inflammation.
- peri-capillary fibrosis and venous ulcers.

### Clinical features of chronic venous insufficiency:

- Pitting edema (the first sign of chronic venous insufficiency).
- Purpura and spotty hemosiderin deposits.
- Dry and itchy skin.
- Over a period of years, the skin, subcutaneous adipose tissue and deep fascia become progressively indurated and adherent.
- A firm circular cuff is formed which appears to strangle the distal calf, creating an inverted wine bottle appearance.
- Intense hemosiderin pigmentation.
- Venous ulcers (develop spontaneously or are triggered by scratching or other trauma).

#### Clinical features of stasis dermatitis:

- **Symptoms**: severe pruritus (multiple scratch marks).
- <u>Site</u>: mostly around the medial malleoli (location of the major communicating veins) may extend to involve the whole distal lower limb.
- <u>Signs</u>: erythema and scaling, oozing and crusting with episodes of vesiculation (superimposed contact sensitization).
- Chronic lesions of stasis dermatitis show lichenification.
- Contact sensitization leads to secondary dissemination (patches of eczema arise in a symmetric distribution on the anterior aspect of the contralateral leg, the anterior thighs, the extensor surface of the upper extremities and may generalize to involve the trunk and face).

# **Treatment**

 Treatment of the underlying venous hypertension (adequate compression bandages or stockings, lifestyle changes, exercise of the calf muscles and surgical treatment).

Treatment of stasis eczema: topical • .corticosteroids and emollients



### **Discoid eczema**

Synonyms(Nummular dermatitis / eczema)

Discoid eczema is a special type of eczema characterized by its coin-shaped lesions (circular or oval plaques with a well-demarcated edge).

# **Epidemiology:**

- Prevalence; relatively uncommon
- Age and sex; men are affected slightly more and at a later age than women (>50 vs. <30 years, respectively). Discoid eczema is relatively rare in children, except as a manifestation of atopy.

# **Aetiopathogenesis**

- 1. A manifestation of atopic dermatitis, asteatotic eczema or stasis dermatitis.
- 2. Role of infection: secondary to bacterial colonization or hematogenous dissemination of bacterial toxins (from distant "focus" of infection).
- 3. Local physical or chemical trauma (discoid eczema develops at the site of an old injury or scar).
- 4. Dry skin, particularly in the elderly.
- 5 Contact sensitization to aloe, depilating creams, or mercury.
- 6. Discoid eczema of the dorsa of the hands may be a manifestation of chronic irritant or allergic dermatitis.
- 7• Emotional stress may play a role.

# **Clinical features**

- Symptoms; intense pruritus (with prominent excoriations).
- <u>Sites</u>: almost exclusively of the extremities, often the lower legs (in men) and the forearms and dorsal aspects of the hands (in women).
- Lesions; well demarcated round or oval (discoid) eczematous patches 1-3 cm in diameter. Lesions may be acute (vesicles and weeping), but are usually chronic (lichenified and hyperkeratotic).

# **Course**

Discoid eczema usually takes a very chronic course.

# **Treatment**

- Medium- to high-potency topical corticosteroid ointments
- Topical tacrolimus or pimecrolimus
- Emollients
- Other lines of therapy: tar preparations, phototherapy.





# **Pompholyx**

- Synonyms: ( Dyshidrotic eczema,
   Vesicular eczema of palms and soles)
- <u>Definition</u>: A special form of eczema of the palms and soles, in which edema fluid accumulates to form visible vesicles or bullae.

Because of the thick epidermis in these sites, the blisters tend to become larger than in other body areas before they burst.

### Aetiopathogenesis:

The following factors may play a role:

- 1- Hereditary predisposition.
- 2- A manifestation of atopic dermatitis.
- 3- A manifestation of allergic contact dermatitis (nickel, perfumes, fragrances).
- 4- Exposure to irritants (e.g. metal workers exposed to oils).
- 5- Role of drugs e.g. following administration of IVIg.
- 6- Emotional stress, exposure to hot climates and sunlight.
- 7- Remote fungal infection (elsewhere on the body), usually the feet "pompholyx dermatophytide".
- 8- Primary allergic contact dermatitis of the feet (e.g. rubber shoe chemicals) may induce a sympathetic palmar eruption.

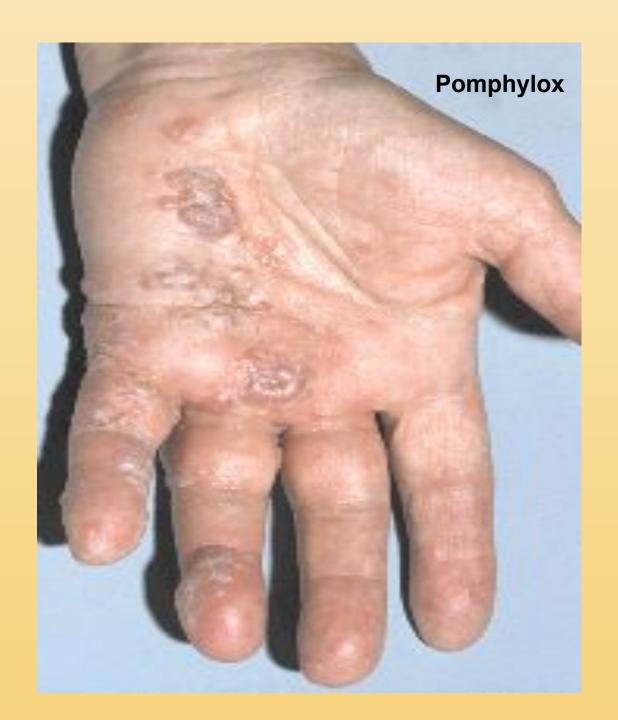
### **Clinical features:**

symmetric, firm, deep-seated vesicles of the palms, the lateral and medial aspects of the fingers, and (less often) the soles and toes. The size of the vesicles varies from pinhead-sized (vesicular type) to several centimeters (bullous type). The lesions are markedly pruritic.

Vesicles initially contain clear fluid but have a tendency for purulent superinfection. It resolves via desquamation of characteristically thick scales.

# **Treatment:**

- Search for and elimination of a possible trigger (e.g. allergens)
- Topical and systemic corticosteroids, topical calcineurin inhibitors,
- phototherapy (bath PUVA is more effective than oral PUVA or UVB).



# **Contact dermatitis**

### **Definition**

 Contact dermatitis (Contact eczema) is a special type of eczema that occurs when the skin comes in contact with external agents.

### **Types:** Two main types:

- 1. Irritant contact dermatitis (ICD)
- 2. Allergic contact dermatitis (ACD)

The two types share many similarities but also differences exist. These are summarized in table below:

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	Irritant contact dermatitis (ICD)	Allergic contact dermatitis (ACD)
Incidence	80% of all contact dermatitis	20% of all contact dermatitis
Causative agents	Irritant chemicals e.g.soaps. solvents, acids or alkalis, organic solvents, oils.	Allergens e.g. nickel (jewelry, buckles and cell phones), fragrances, formaldehyde, topical antibiotics (neomycin, bacitracin), gold, and others
Pathogenesis	Local toxic effect when the skin comes in contact with irritant chemicals —> damage of keratinocytes (barrier disruption, protein and lipid denaturation, lipid solubilization —> release of inflammatory mediators e.g. TNF- and IL-1 a —> recruitment of leucocytes and initiation of inflammatory cascade.	A delayed-type (type IV) hypersensitivity reaction when the skin comes in contact with an allergen to which an individual has previously been sensitized.  Initial sensitization phase: first exposure to the allergen (uptake and presentation to naive T cells)  The subsequent re-exposure to the same allergen (even at low concentration) —> presentation to already primed T-cells —» release of cytokines —> clinical picture of eczema.

Clinical features	1)Occurs in all subjects exposed 2)Previous contact is not required 3)Occurs soon after exposure 4)Occurs at site of exposure 5)Severity varies with quantity, concentration, and length of exposure to the irritant  Skin lesions: pruritic - Acute (clear fluid-filled vesicles or bullae on red edematous skin), Subacute (less edema, papules), Chronic (minimal edema, scaling, skin Assuring, lichenification)	1)Occurs only in subjects sensitive to the allergen 2)Previous exposure (sensitization is necessary) 3)Occurs within 48-96 hours after exposure 4)Occurs at site of exposure, away from it or disseminated  Skin lesions: pruritic - Acute (clear fluid-filled vesicles or bullae on red edematous skin), Subacute (less edema, papules), Chronic (minimal edema, scaling, skin Assuring,	
	skin Assuring, lichenification)	lichenification)	
Treatment	Avoidance of the irritant / trigger, emollients and topical	Avoidance of the irritant / trigger, emollients and topical	
	steroids	steroids	

### Patch test

#### **Definition:**

a simple office test used for the diagnosis of ACD (the test detects type IV delayed hypersensitivity reaction).

#### **Method:**

standard dilutions of the suspected / common allergens are applied to the back under aluminium discs / patches; the occlusion encourages penetration of the allergen.

The patches are left in place for 48 hours (during that periods, patients are instructed to keep patches dry and in place, avoid excessive sweating or heavy-lifting) and then, after careful marking, are removed.

The sites are inspected 10 minutes later, again at 96 hours and sometimes later (1 week).

# **Interpretation:** the readings are scored according to the reaction seen:

- No reaction
- ± Doubtful reaction (minimal erythema)
- + Weak reaction (erythematous and may be papular)
- ++ Strong reaction (erythematous and oedematous or vesicular)
- +++ Extreme reaction (erythematous and bullous)
- IR Irritant reaction (variable, but often sharply circumscribed, with a glazed appearance and increased skin markings.







