

# Acute dermatoses

**Histology**

**epidermis** → composed of 5 layers

1. stratum corneum
2. stratum granulosum
3. stratum spinosum → squamous
4. stratum basale
5. dermal papilla → are extensions coming from the dermal layer

keratinocytes & squamous cells

make up the skin layer squamous cell (keratinocyte)

**skin**

**dermis**

→ composed of inflammatory cells such as collagen, fibrotic tissue & other components depending on the type of tissue

2 things are always found in dermis

- ↳ hair follicle
- ↳ sebaceous gland small oil producing gland.

\* the amount & intensity varies from one place to another

**Function:**

- squamous cells (keratinocyte) help maintain skin homeostasis by providing a physical barrier to environmental tissue.
- Has major role in immunity.

dermis CD4+ / CD8+

epidermis 8/5 T cells

## 1 Acute inflammatory dermatosis

**acute lesions**

days to several weeks in duration

characterised by

- inflammation
- edema
- sometimes → epidermal or vascular or subcutaneous injury.

marked by infiltrates consisting of **mononuclear cells** rather than **neutrophils**

if cells acute inflammatory

\* some acute lesions may persist, transitioning to a chronic phase while others are self-limited.

**A. Urticaria** → a common disorder mediated by localized mast cell degranulation, which leads to dermal microvascular hyperpermeability

\* The resulting erythematous, edematous, pruritic plaques are termed **wheals**.

**most common**

**IgE-dependent urticaria**

- responsible agents

- viruses
- parasites
- foods
- drugs
- insect venom

**pathogenesis**

**IgE-independent urticaria.**

result from exposure to substance that directly incite mast cell degranulation such as opiates, certain antibiotics

**Histologic features** → sparse superficial perivascular infiltrate of mononuclear cells, neutrophils, & some times eosinophils

\* dermal edema causes splaying of collagen bundles.

\* **degranulation** of mast cells, can be highlighted using a **Giemsa stain**.

individual lesions usually develop & fade within hours

but episodes can persist for days or even months.

**clinical features** → typically affect individuals between 20-40 years of age.

**Treatment** →

- Antihistamines
- leukotriene antagonists
- monoclonal ab → ↓ IgE action.
- immunosuppressive drug.

\* Lesions range in size & nature.

small, pruritic papules → large, edematous erythematous plaques.



## Rickets

↓ metabolism of vit D, Ca or P  
softening of bone

## Osteomalacia

in adult.  
- normal amount of collagen  
↓ Ca absorption from intestine  
↓ dietary  
resistance to vit D action  
↓ P ⇔ ↑ renal loss

## Acromegaly

↑↑ GH

cause → benign tumor of pituitary gland.

## Fibrous dysplasia

abnormal bone growth.

normal bone  $\xrightarrow{\text{disease}}$  fibrous bone

• before birth.  
gene mutation.

## Osteomyelitis

bacterial infection.  
or  
fungi

## Hypocalcemia

↓ serum Ca level

↓ is also unbound ionized.

↓ PTH

↓ Vit D.

## Hypophosphatasia

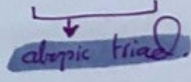
disrupts → mineralization.

mutation in ALPL



**Clinical features** → Lesions of eczematous dermatitis are ↓

- pruritic • eczematous • oozing plaques often containing vesicles & bullae
- # With persistent exposure lesions may become **scaly (hyperkeratotic)** as the epidermis **thickens (acanthosis)**
- # It usually appears in **early childhood** & remits spontaneously as patients mature into adults. Children with atopic dermatitis often have asthma & allergic rhinitis



**Erythema multiforme.**

↓ is characterized by epithelial injury mediated by skin-homing CD8+ cytotoxic T lymphocytes.

- Tc attack is focused on the basal cells of cutaneous & mucosal epithelia, presumably due to recognition of still unknown antigens
- # Self-limited disorder that appears to be hypersensitivity response to certain infections or drugs
  - herpes simplex
  - mycoplasma
  - some fungi.

Type 4

**Morphology**

→ Affected individuals present with a wide array of lesions, which may include:
 

- macules → flat red or pink patches
- papules → solid elevations containing no fluid
- vesicles → small raised fluid fill lesions
- bullae → large raised fluid fill lesions (hence the term multiform)

# Well-developed lesions have a characteristic **targetoid** appearance. 2mm-2cm

**Early lesion shows**

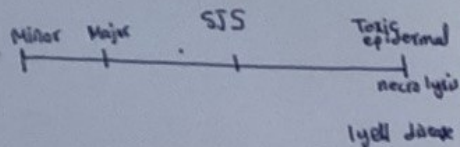
- superficial perivascular lymphocytic infiltrate.
- dermal edema.
- margination of lymphocyte along dermoepidermal junction with apoptotic keratinocyte.

→ # with time ↓  
 → discrete, confluent zones of basal epidermal necrosis appear, with concomitant blister formation.

**Clinical Feature**

Erythema multiforme caused by medications may progress to more serious eruptions, such as:-

- **Stevens-Johnson syndrome** ⇒  $\frac{30\% \text{ من الحالات}}$
- **Toxic epidermal necrolysis** ⇒  $\frac{30\% \text{ من الحالات}}$



Spring & Fall

# These can be life-threatening, as they may cause sloughing of large portions of the epidermis, resulting in fluid loss & infections complications.

12 hours but not sleeping  
 Non-epidermal  
 Heat loss  
 epidermal necrolysis