Acute dermatosis

Histology

epidermis ⇒ composed of 5 layers (keratinocyte squamous cell)
1. stratum corneum
2. stratum granulosum
3. stratum spinosum → squamous
4. stratum basale
5. dermal papilla ⇒ are extensions coming from the dermal layer

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: γ/δ T cells

① Acute inflammatory dermatosis

acute lesions
↳ days to several weeks in duration
characterized by ⇒ inflammation, edema, sometimes ⇒ epidermal / vascular / or subcutaneous ⇒ injury.

* marked by infiltrates consisting of mononuclear cells rather than neutrophils (في acute inflammation)

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

A. Urticaria (الشرية) ⇒ a common disorder mediated by localized mast cell degranulation, which leads to dermal microvascular hyperpermeability

* The resulting ⇒ erythematous (احمرار) / edematous / pruritic (حكة) plaques are termed wheels. (خلايا)

pathogenesis

IgE-dependent urticaria (most common)
- responsible ag: viruses, pollens (حبوب اللقاح), foods, drugs, insect venom (لسعة حشرة)

IgE-independent urticaria.
- result from exposure to substance that directly incite mast cell degranulation such as ⇒ opiates, certain antibiotics

Histologic features ⇒ * sparse superficial perivenular infiltrate of mononuclear cells, rare neutrophils, & some times eosinophils

* dermal edema causes splaying (تشتت وتباعد عن بعضها البعض) of collagen bundles.
* Degranulation of mast cells, can be highlighted using a Giemsa stain.

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

individual lesions usually develop & fade within hours, but episodes can persist for days or even months.

* Lesions range in size & nature.
  - small, pruritic papules ⇒ large, edematous erythematous plaques.

Treatment ⇒
- Antihistamines.
- Leukotriene antagonists
- monoclonal abs ⇒ ↓ IgE action.
- immunosuppressive drug.

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 → fibrous bone

- before birth.

gene mutation.

Osteomyelitis.

bacterial infection.

or

fungi

:

Hypocalcemic

↓ serum Ca level

↳ unbound ionized.

↓ PTH

↓ Vit D.

Hypophosphatasia

disrupts → mineralization.

mutation in ALPL

Clinical features ⇒ Lesions of eczematous dermatitis are ↓

• pruritic • edematous • oozing plaques • often containing vesides & bullae.

with persistent ag 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 → atopic triad.

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 agents

Self-limited disorder that appears to be hypersensitivity response (Type 4) to certain infections (herpes simplex, mycoplasma, some fungi) or drugs.

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 "target" appearance. 2mm-2cm

Early lesion show ⇓
- superficial perivascular lymphocytic infiltrate.
- dermal edema.
- marginalization of lymphocytes along dermoepidermal junction with apoptotic keratinocytes.

→ # 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 ⇒ يصيب أقل من 30% من الجسم
- Toxic epidermal necrolysis ⇒ يصيب أكثر من 30% من الجسم

Minor — Major — SJS — Toxic epidermal necrolysis

Lyell disease

في أي فصل؟ Spring & Fall.

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