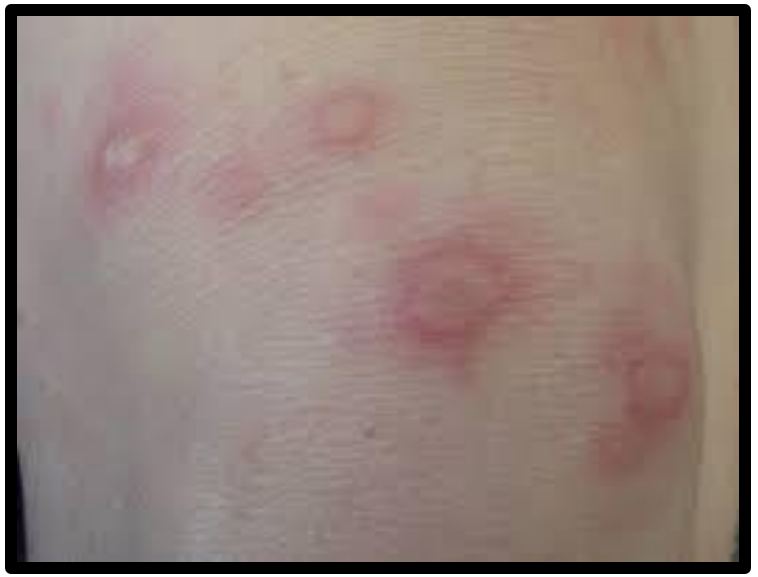
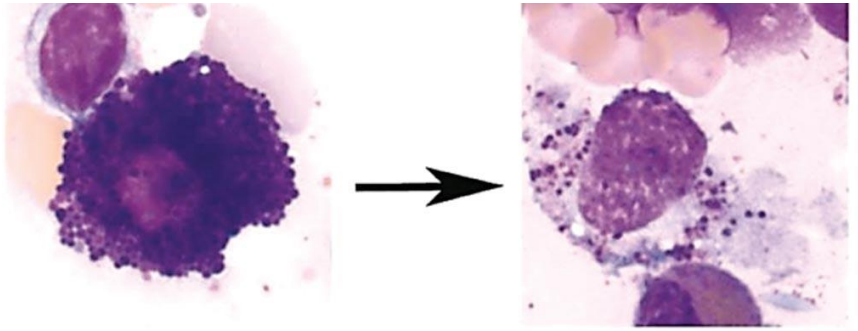
MSS MODULE- PATHOLOGY LAB I

A. URTICARIA.



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

- **The resulting erythematous, edematous, and pruritic plaques are termed wheels.**

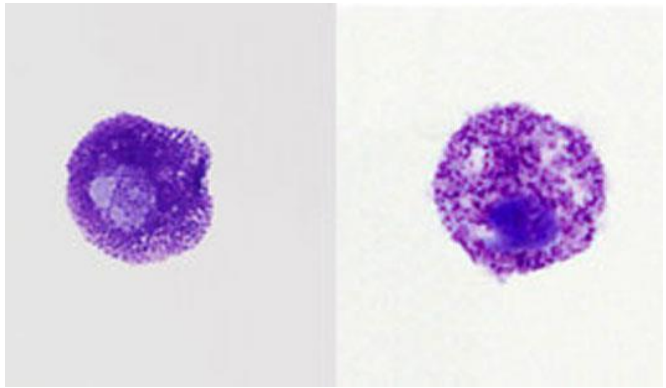


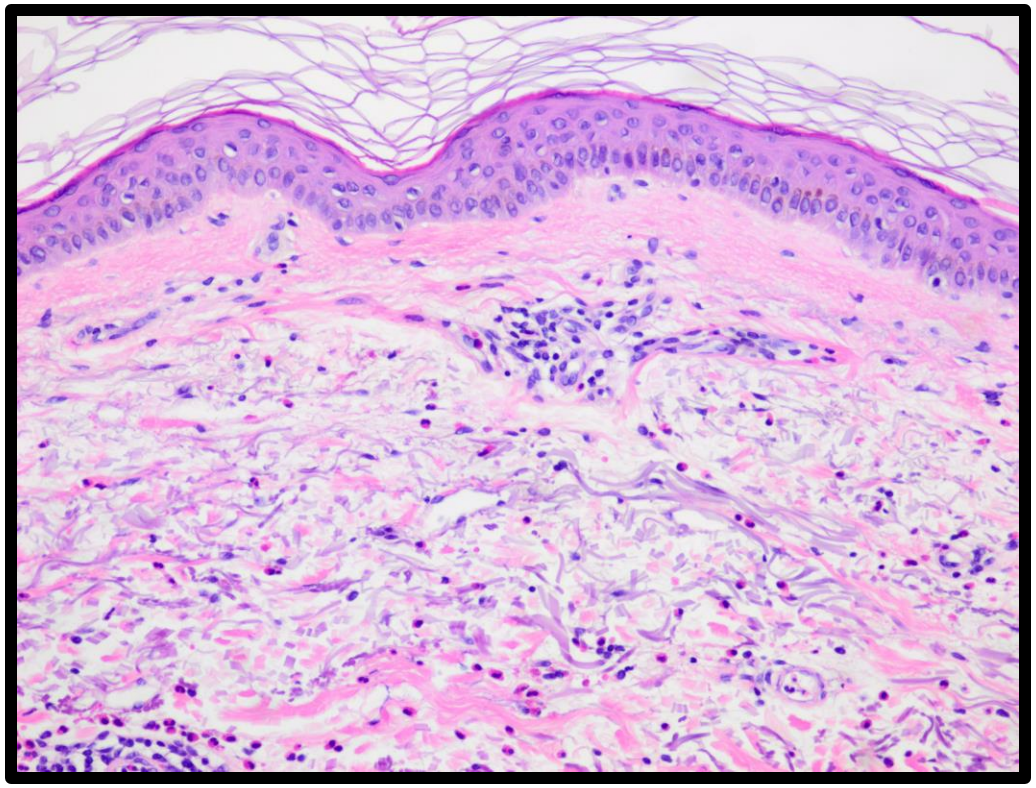
Resting mast cell

Activated mast cell

HISTOLOGIC FEATURES OF URTICARIA

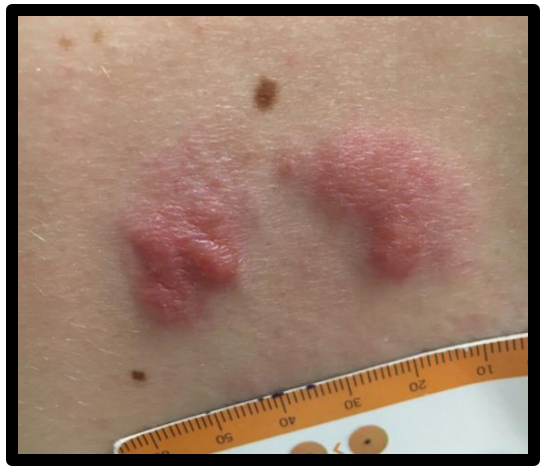
- *sparse superficial perivenular infiltrate of mononuclear cells, rare neutrophils, and sometimes eosinophils.
- *dermal edema causes splaying of collagen bundles.
- *Degranulation of mast cells, can be highlighted using a Giemsa stain.

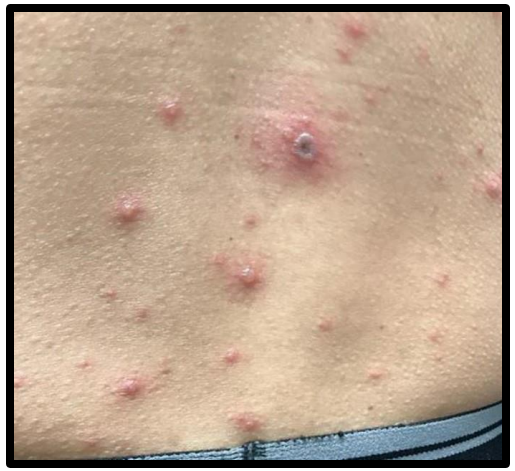


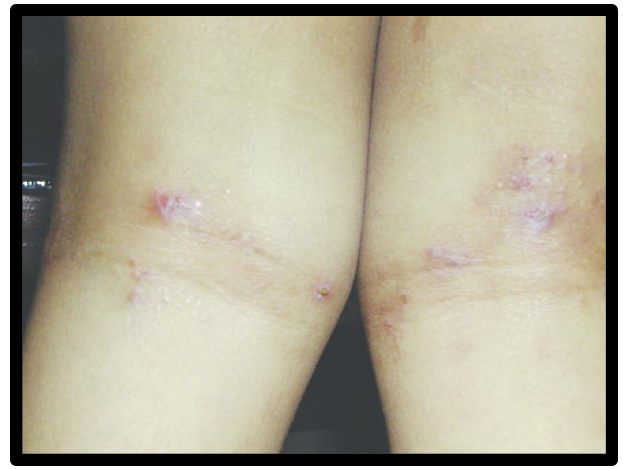


B. ACUTE ECZEMATOUS DERMATITIS.

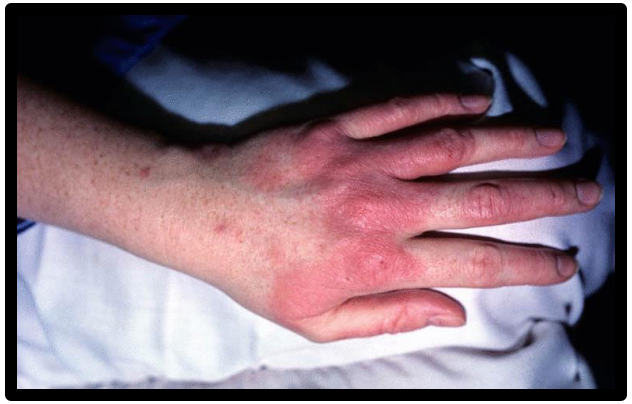
- **Eczema is a clinical term that embraces a number of conditions with varied underlying etiologies.**
- **Clinically the patient may has:**
- **erythematous papules with overlying vesicles, which ooze and become crusted.**
- **Pruritus is characteristic.**
- **With persistence, these lesions coalesce into raised, scaling plaques.**





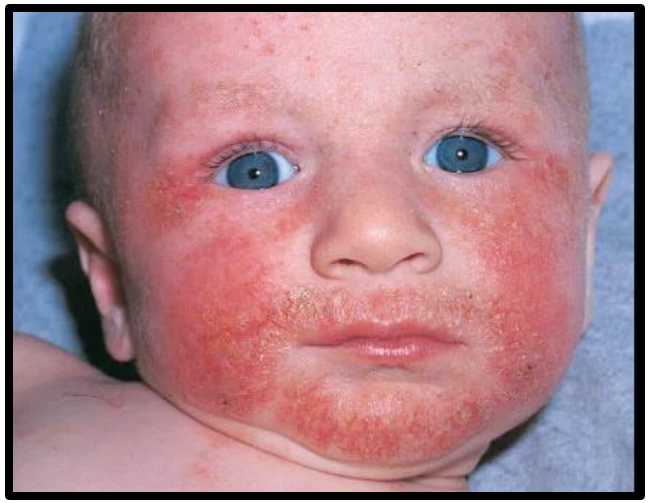


THE CLINICAL SUBTYPES INCLUDE :



- **1. Allergic contact dermatitis:**

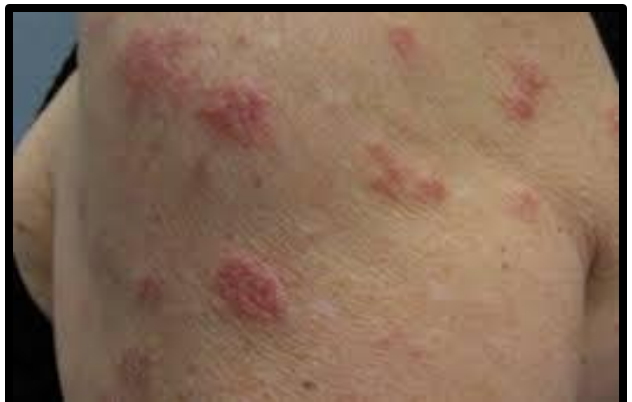
stems from topical exposure to an allergen and is caused by delayed hypersensitivy reactions.



- **2. Atopic dermatitis:**

stem from defects in keratinocyte barrier function, defined as skin with increased

permeability to substances to which it is exposed, such as potential antigens



- **3. Drug-related eczematous dermatitis:**

Hypersensitivity reaction to a drug.

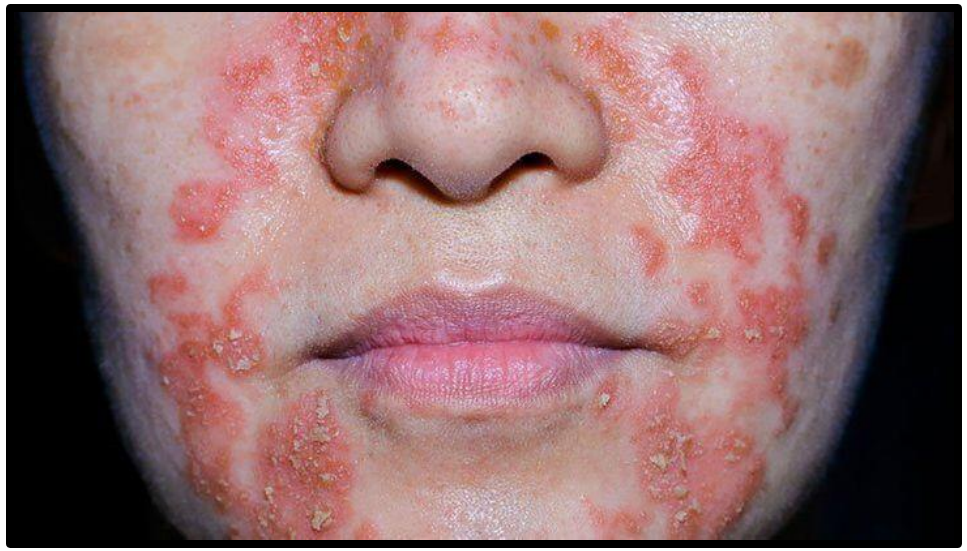
- **4. Photoeczematous dermatitis:**

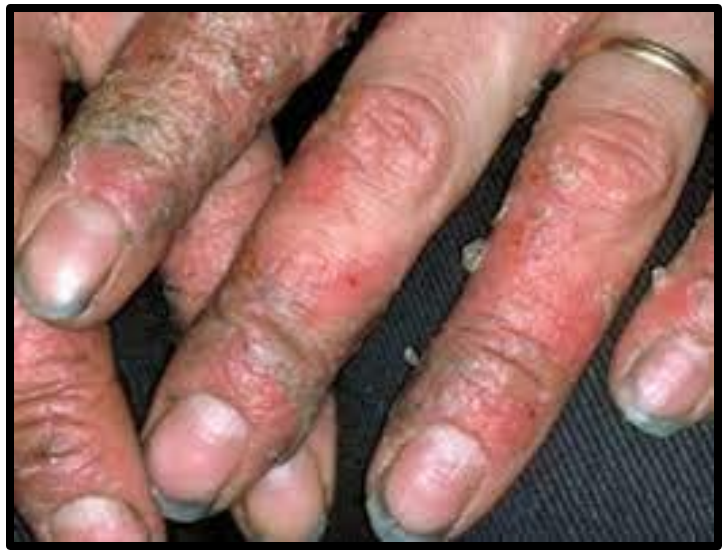
appears as an abnormal reaction to UV or visible light

- **5. Primary irritant dermatitis:**

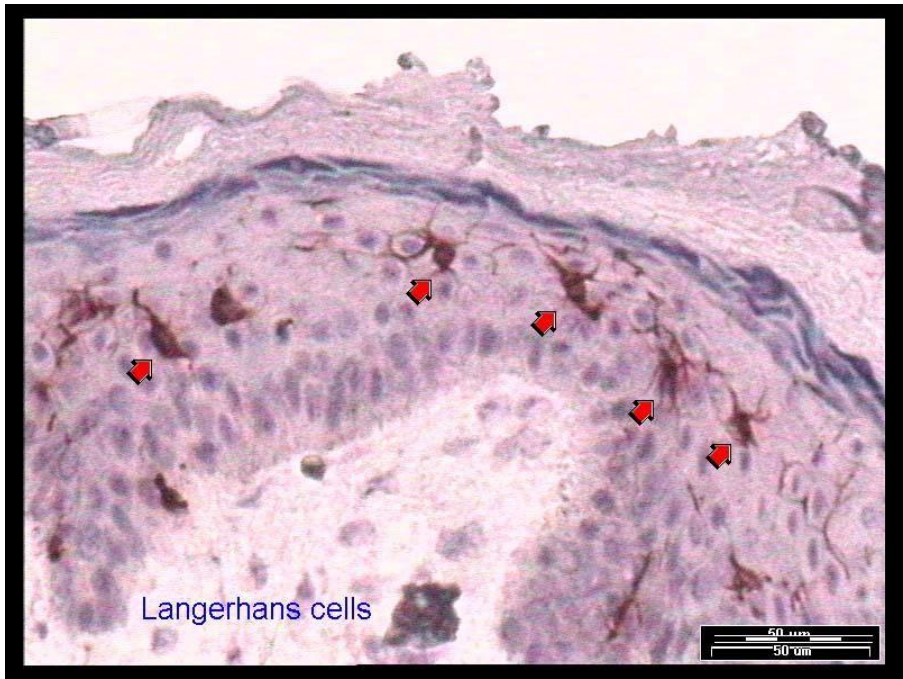
results from exposure to substances that chemically, physically, or mechanically

damage the skin.



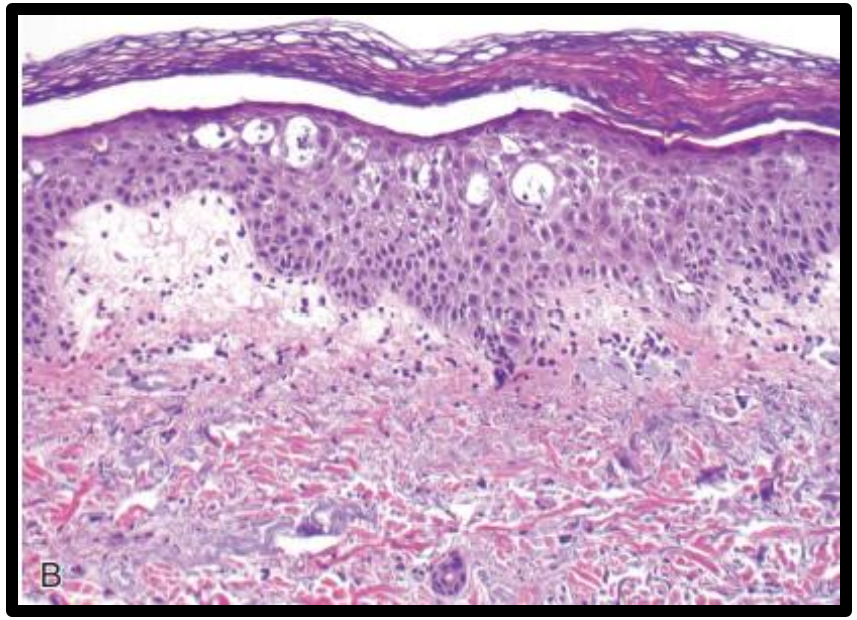


- The self-proteins modified by the agent are processed by epidermal Langerhans cells
- which migrate to draining lymph nodes and present the antigen to naïve T cells.
- This sensitization event leads to acquisition of immunologic memory
- on reexposure to the antigen, the activated memory CD4+ T lymphocytes migrate to the affected skin sites during the course of normal circulation

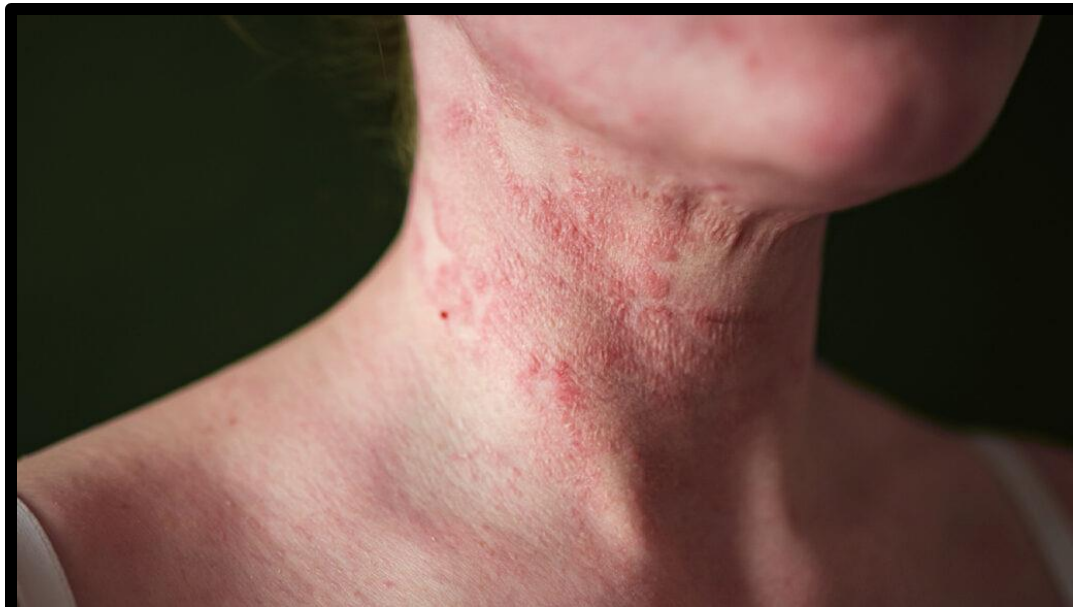


HISTOLOGY

- Spongiosis or epidermal edema, Edema fluid seeps into the
- epidermis, where it splays apart keratinocytes .
- Intercellular bridges are stretched and become more prominent and are easier to visualize.
- superficial perivascular lymphocytic infiltrate
- edema of dermal papillae.
- mast cell degranulation.



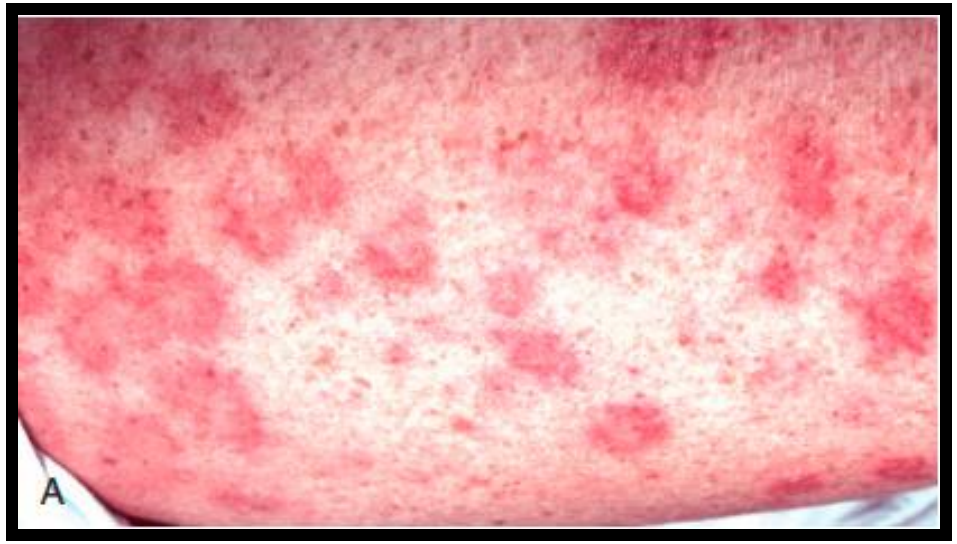
- Lesions of acute eczematous dermatitis are
- pruritic, edematous, oozing plaques, often containing vesicles and bullae.
- With persistent antigen exposure, lesions may become scaly (hyperkeratotic) as the epidermis thickens (acanthosis).
- It usually appears in early childhood and remits spontaneously as patients mature into adults. Children with atopic dermatitis often have asthma and allergic rhinitis, termed the atopic triad.



Erythema Multiforme

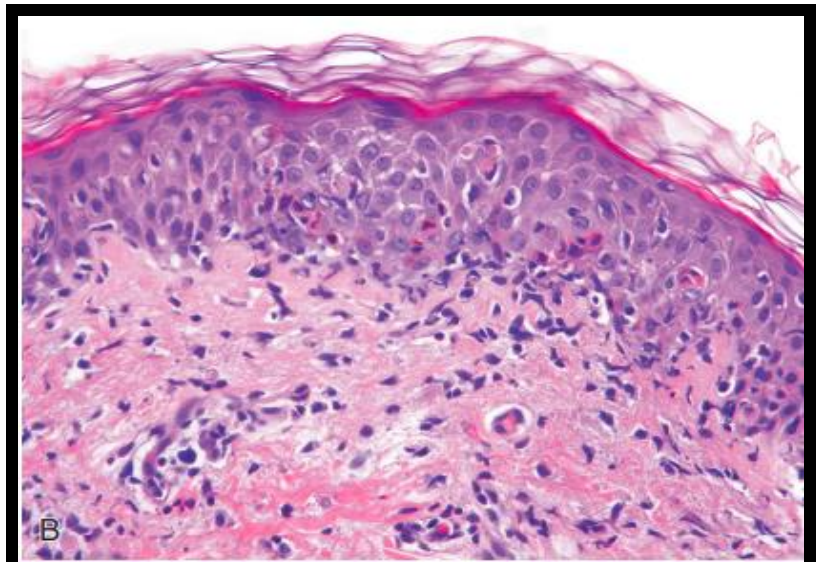
MORPHOLOGY

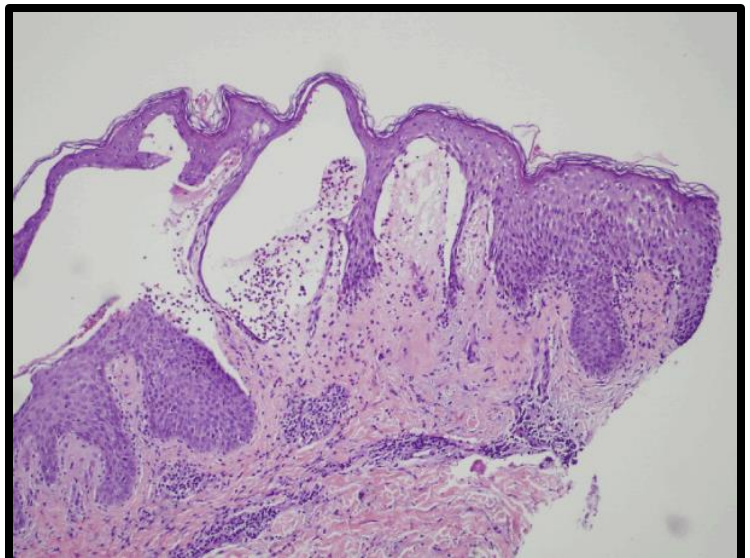
- **Affected individuals present with a wide array of lesions, which may include macules, papules, vesicles, and bullae (hence the term multiforme)**
- **Well-developed lesions have a characteristic “targetoid” appearance**



- Early lesions show
- superficial perivascular lymphocytic infiltrate
- dermal edema
- margination of lymphocytes along the dermoepidermal junction with apoptotic keratinocytes

- With time
- discrete, confluent zones of basal epidermal necrosis appear, with concomitant blister formation.

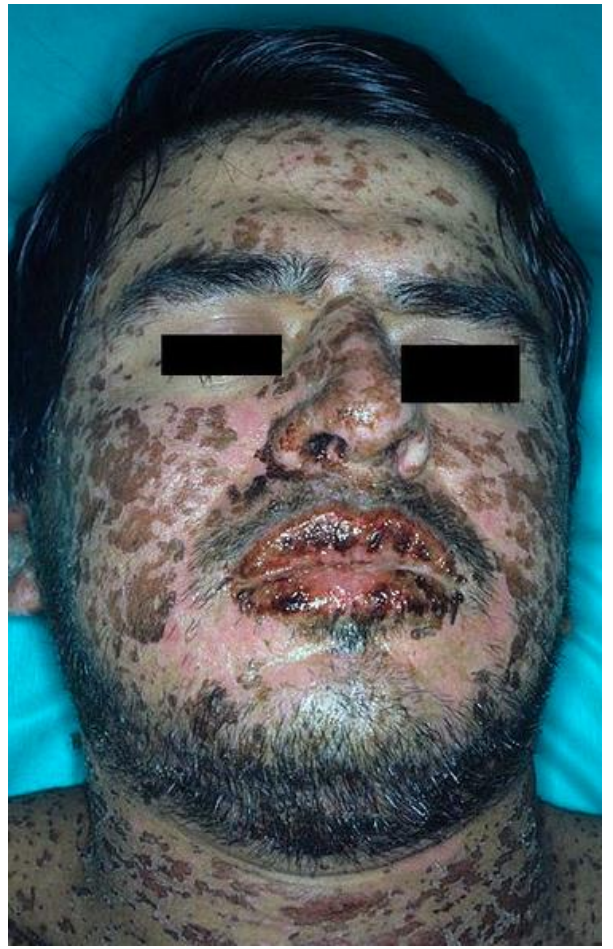


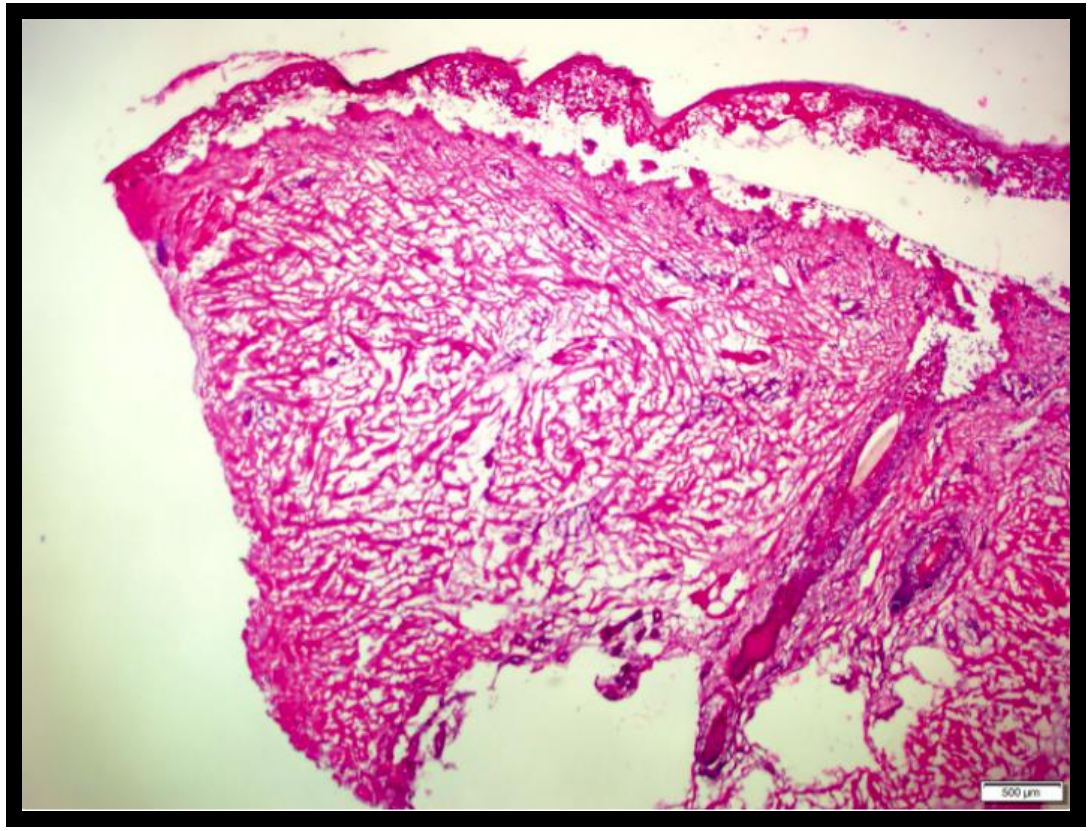


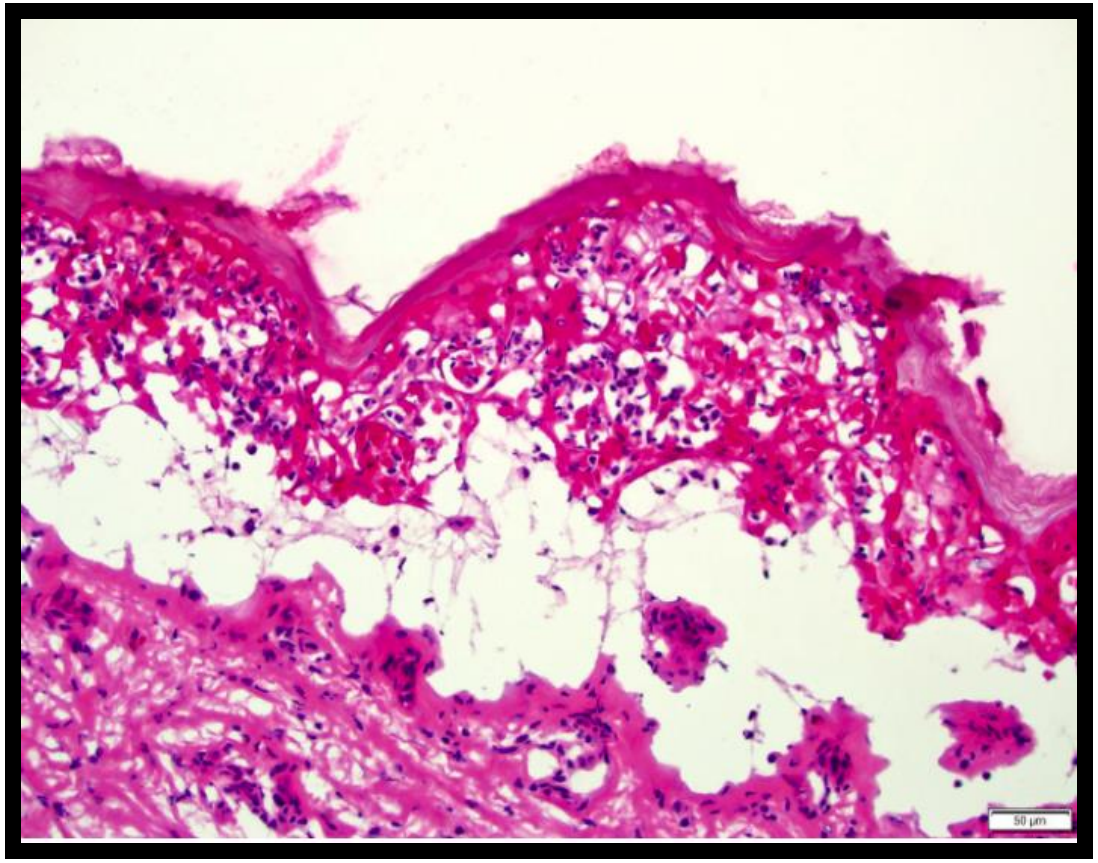
CLINICAL FEATURES

- **Erythema multiforme caused by medications may progress to more serious eruptions, such as**
- **Stevens-Johnson syndrome**
- **toxic epidermal necrolysis.**

- **These forms can be life-threatening, as they may cause sloughing of large portions of the epidermis, resulting in fluid loss and infections complications**



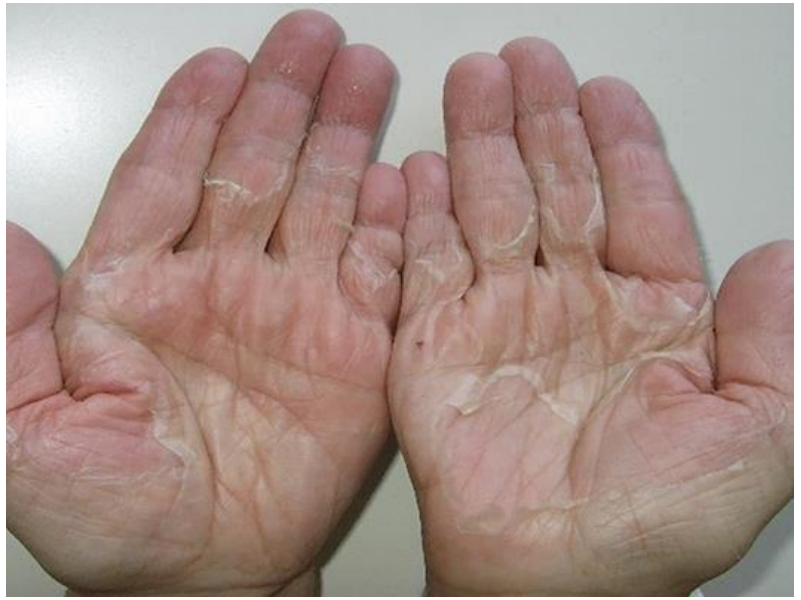

500 µm


50 µm

Chronic inflammatory dermatoses

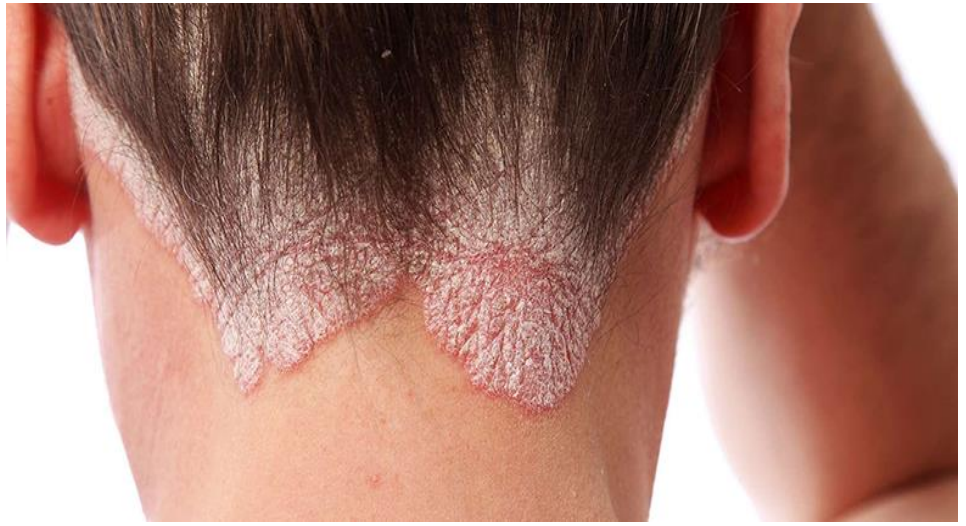
- Chronic inflammatory dermatoses are persistent skin conditions that exhibit their most characteristic features over many months to years.

- The skin surface in some chronic inflammatory dermatoses is roughened as a result of excessive or abnormal scale formation and shedding (desquamation).

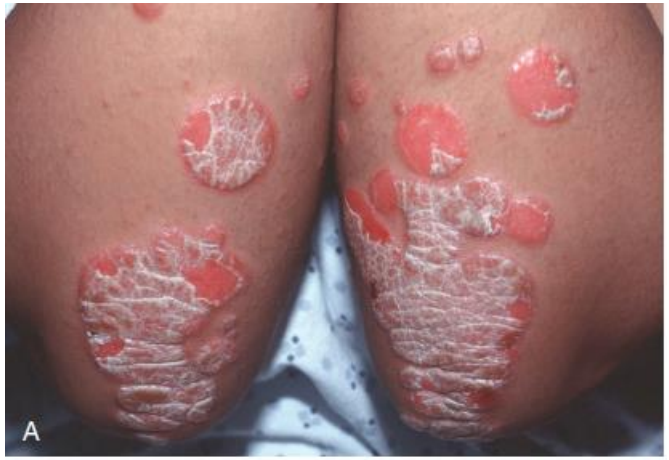


1. PSORIASIS

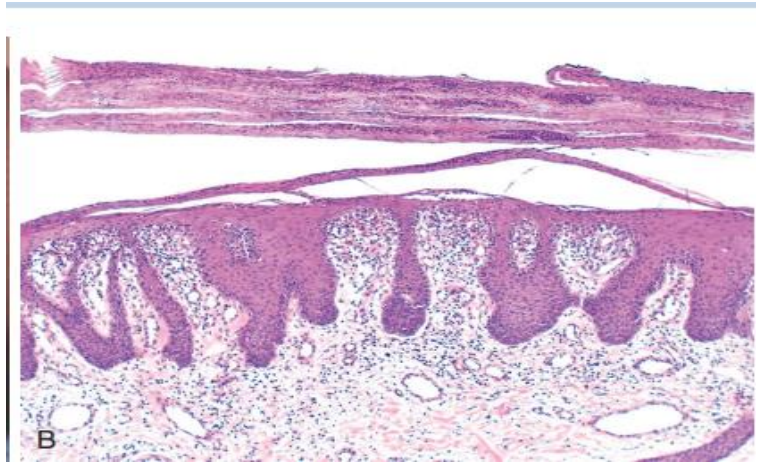
- Psoriasis is a common chronic inflammatory dermatosis, affecting 1% to 2% of individuals.
- psoriasis is associated with an increased risk for heart attack and stroke, a relationship that may be related to a chronic inflammatory state.
- Psoriasis also is associated in up to 10% of patients with arthritis.



MORPHOLOGY



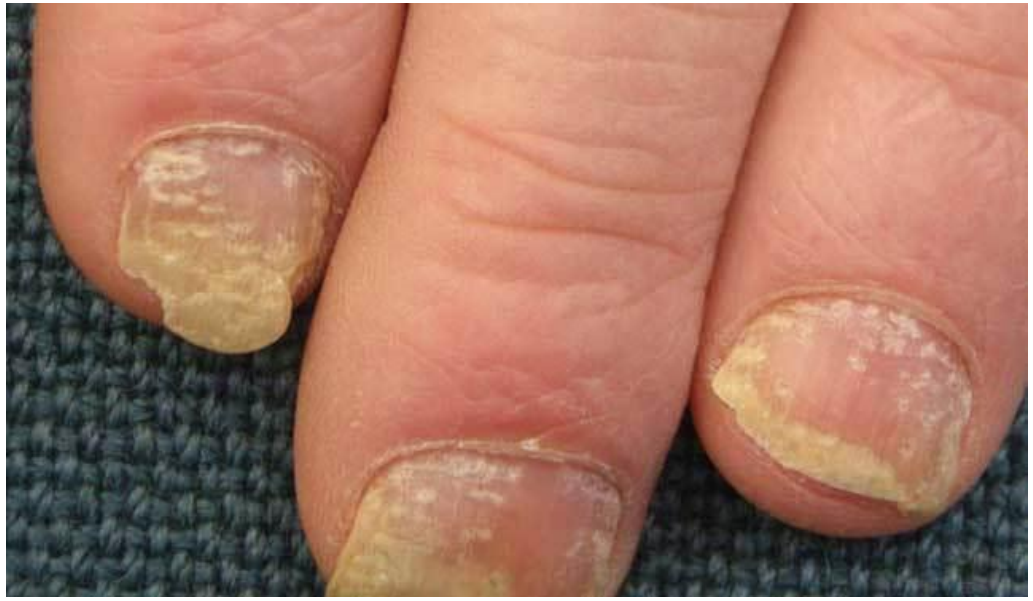
well-demarcated, pink to salmon–colored
plaque covered by loosely adherent silver-white scale

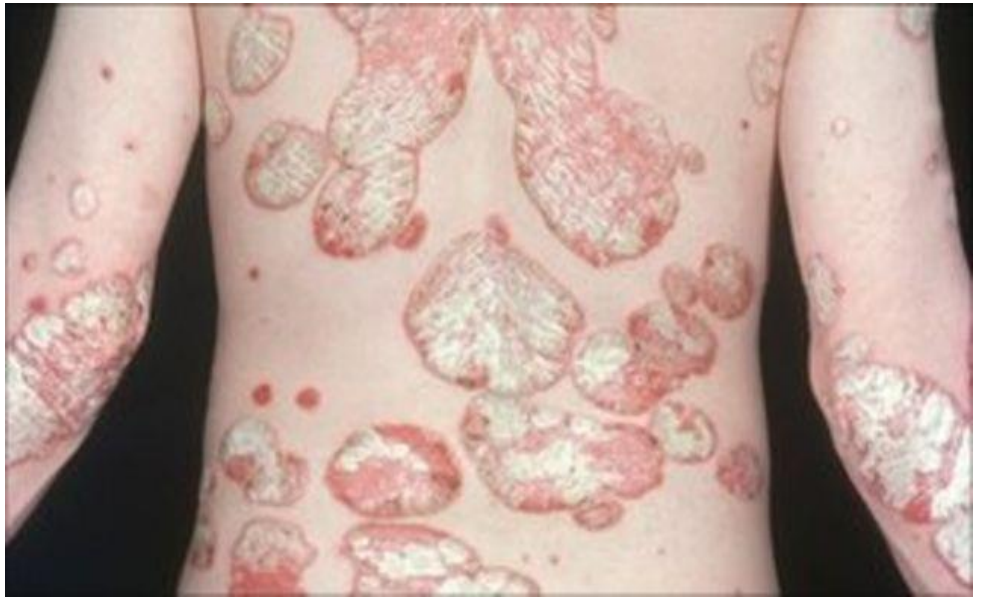


epidermal thickening (acanthosis).
regular downward elongation of the rete ridges
Increased epidermal cell turnover and lack of maturation
results in loss of the stratum granulosum and extensive parakeratotic scale

CLINICAL FEATURES

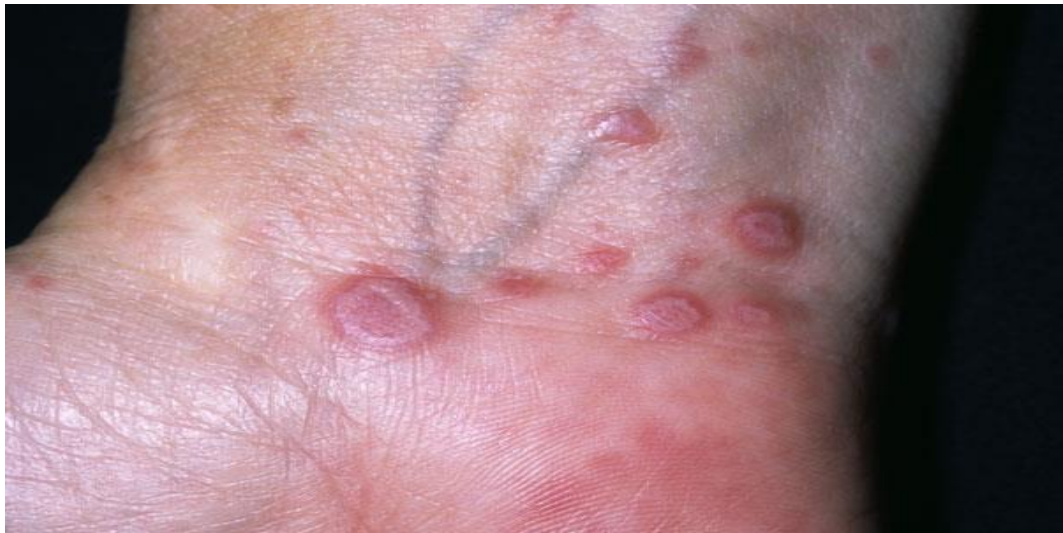
- Psoriasis most frequently affects the skin of the elbows, knees, scalp, lumbosacral areas, intergluteal cleft, glans penis, and vulva.
- Nail changes on the fingers and toes occur in 30% of cases.





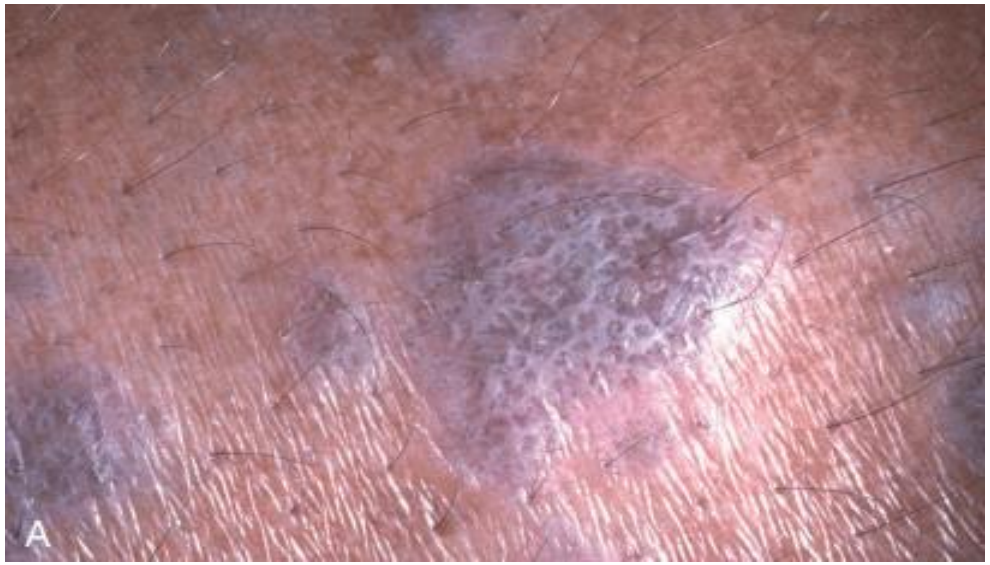
2. LICHEN PLANUS

- "Pruritic, purple, polygonal, papules, and plaques" are the tongue-twisting Ps that describe this disorder of skin and squamous mucosa
- The lesions may result from a CD8+ T cell–mediated cytotoxic response against antigens in the basal cell layer and the dermoepidermal junction that are produced by unknown mechanisms



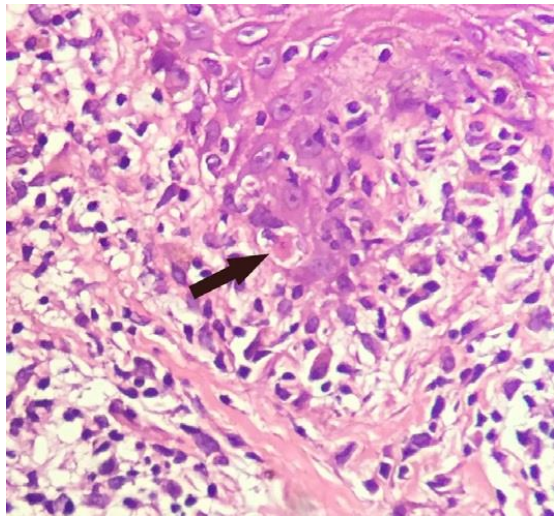
GROSSLY

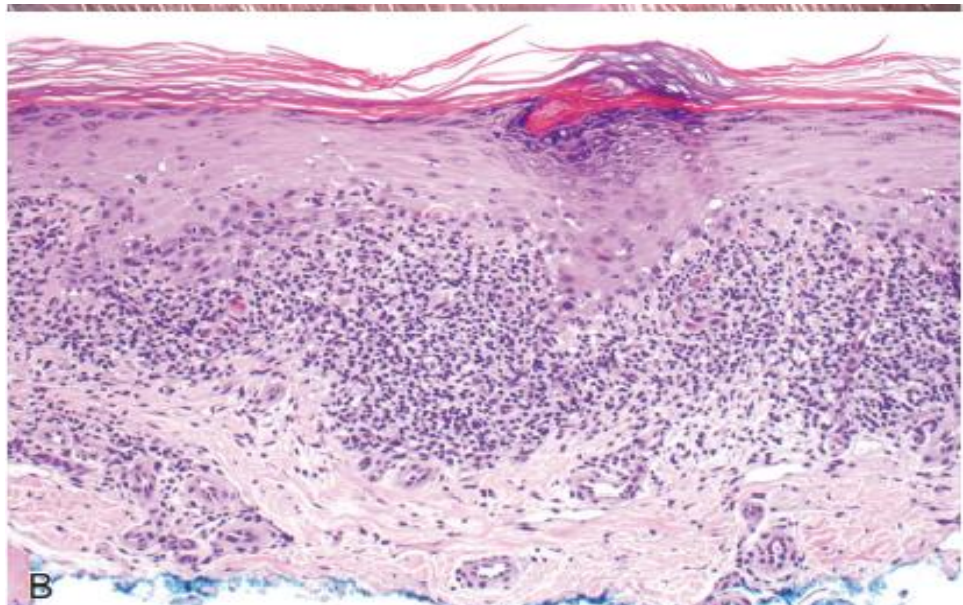
- Cutaneous lesions of lichen planus consist of pruritic, violaceous, flat-topped papules that may coalesce focally to form plaques .
- These papules are highlighted by white dots or lines termed Wickham striae.
- Hyperpigmentation may result from melanin loss into the dermis from damaged keratinocyte



MICROSCOPICALLY

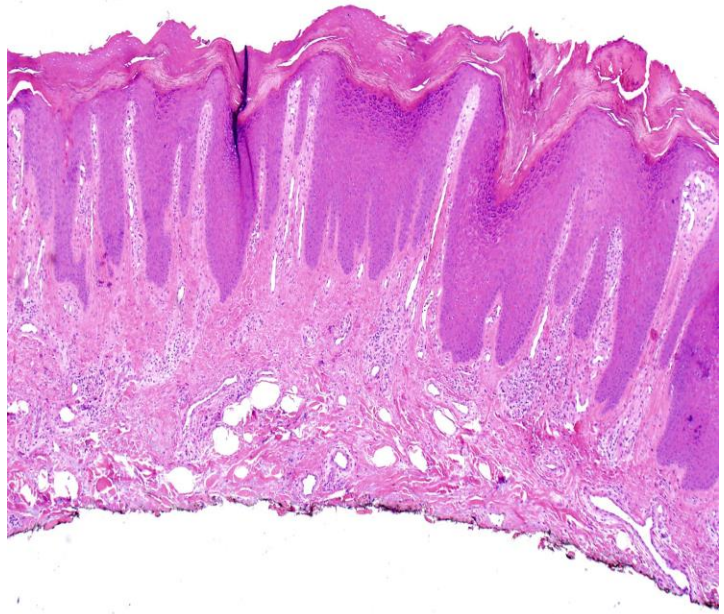
- lichen planus is a prototypical interface dermatitis, so called because the inflammation and damage are concentrated at the interface of the squamous epithelium and papillary dermis.
- There is a dense, continuous infiltrate of lymphocytes along the dermoepidermal junction.
- Civatte bodies*.



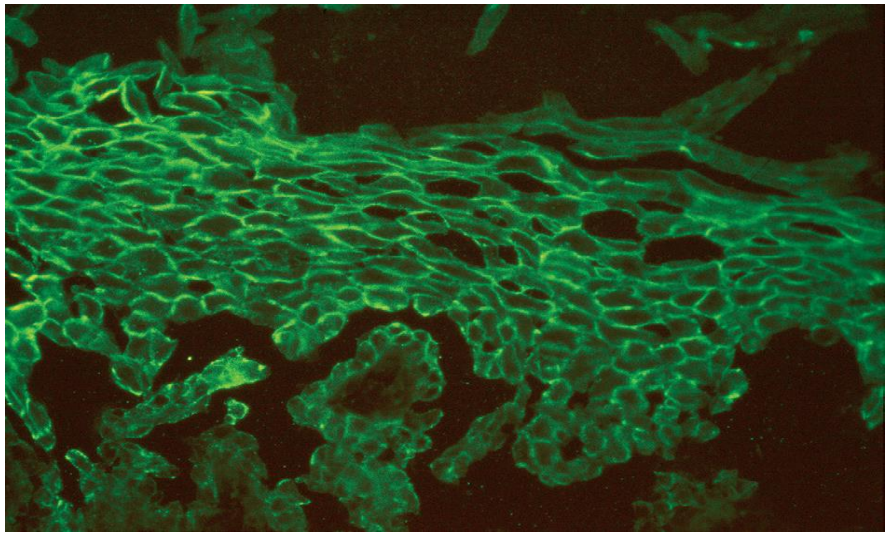


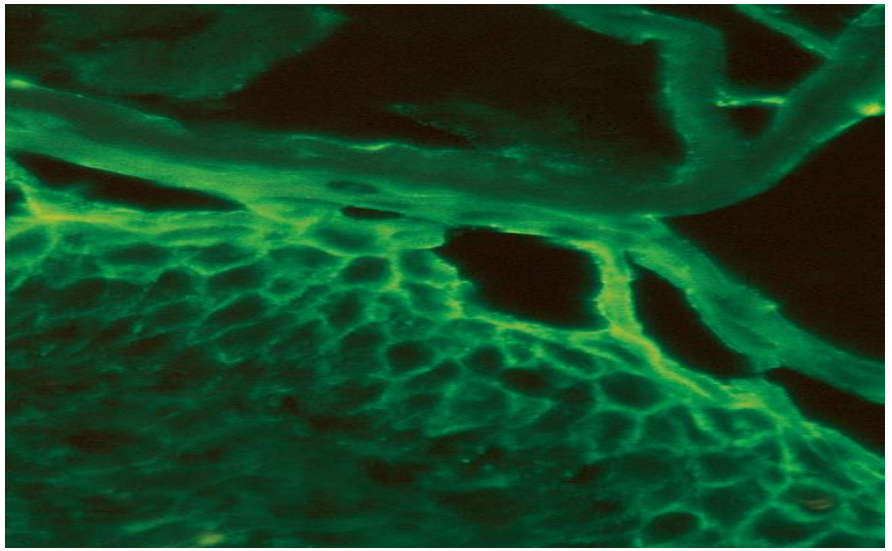
3- LICHEN SIMPLEX CHRONICUS

- Lichen simplex chronicus manifests as roughening of the skin. It is a response to local repetitive trauma, usually from rubbing or scratching.
- The pathogenesis of lichen simplex chronicus is not understood, but the trauma probably induces epithelial hyperplasia and eventual dermal scarring.
- Microscopically :Lichen simplex chronicus is characterized by acanthosis, hyperkeratosis, and hypergranulosis.



- **Diagnosis: Direct immunofluorescence study:**
  Lesional sites show a characteristic fishnet-like pattern of intercellular IgG deposits.





Pemphigus vulgaris:

Uniform deposition of Ig (green) along cell membrane of keratinocytes (fishnet pattern).

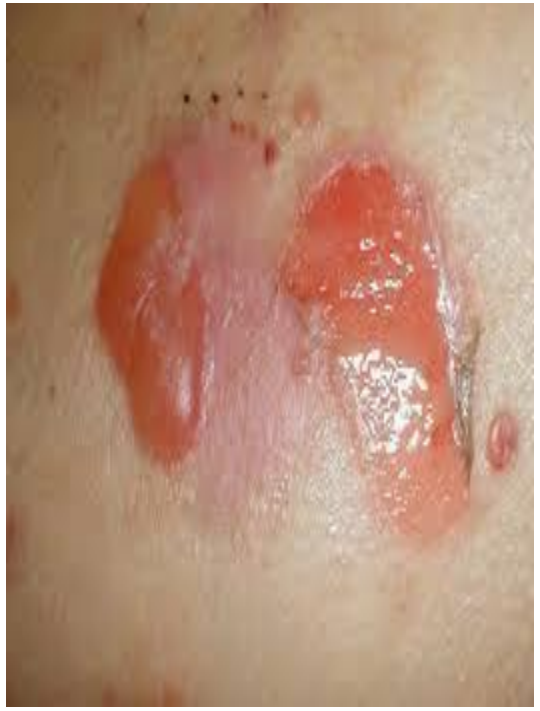
Pemphigus foliaceus:

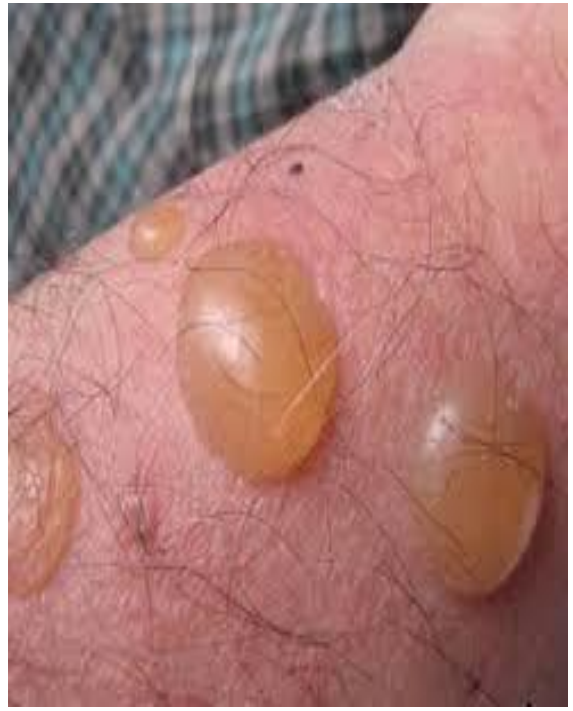
Ig deposits confined to superficial layers of epidermis.

1-Pemphigus vulgaris:

- **Most common type.**
- **Involves both mucosa & skin of scalp, face, axillae, groin, trunk, & points of pressure.**

Lesions:
Superficial vesicles & bullae, rupture easily, leaving deep & extensive erosions covered with serum crust.

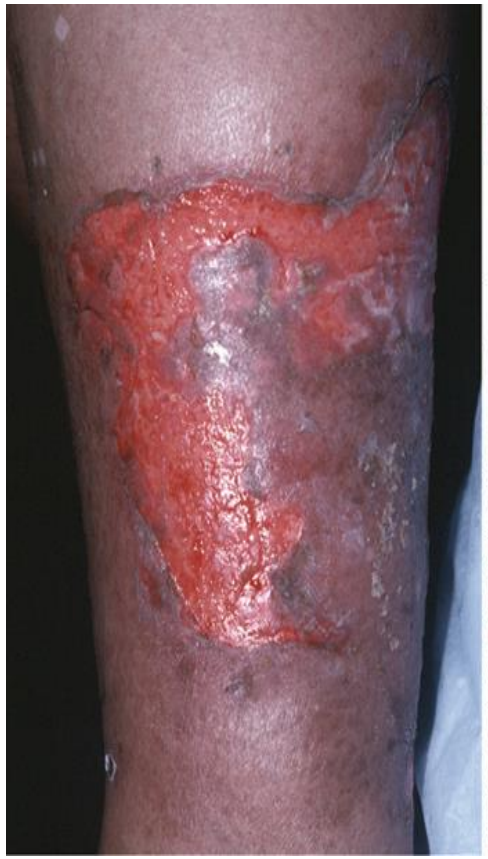


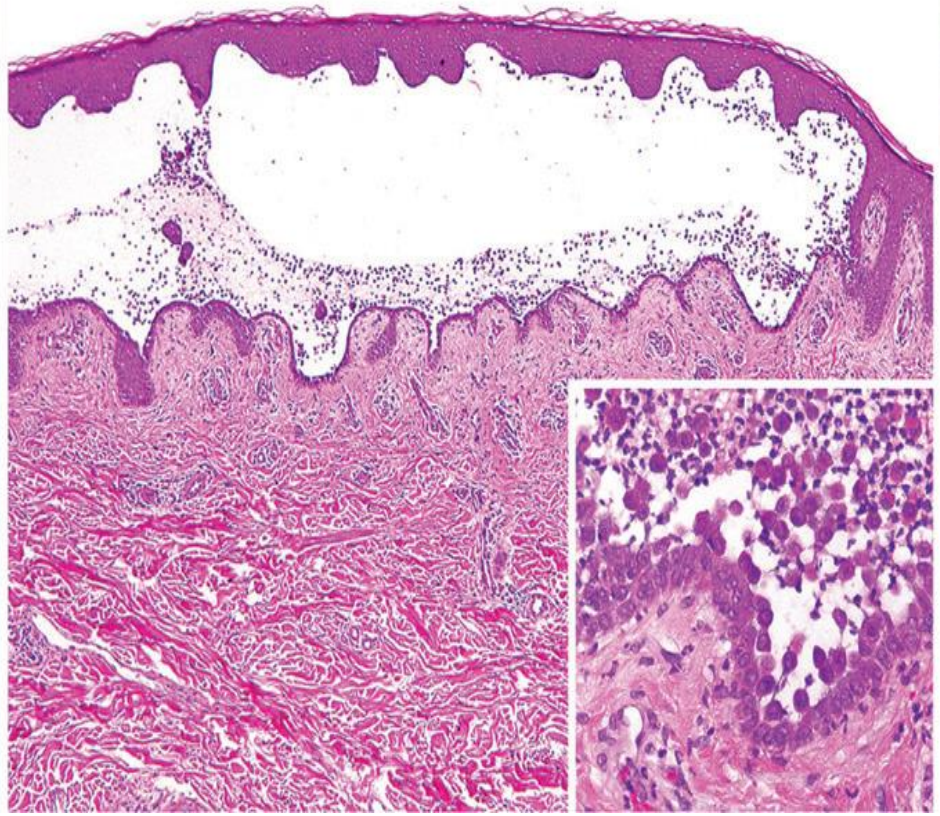


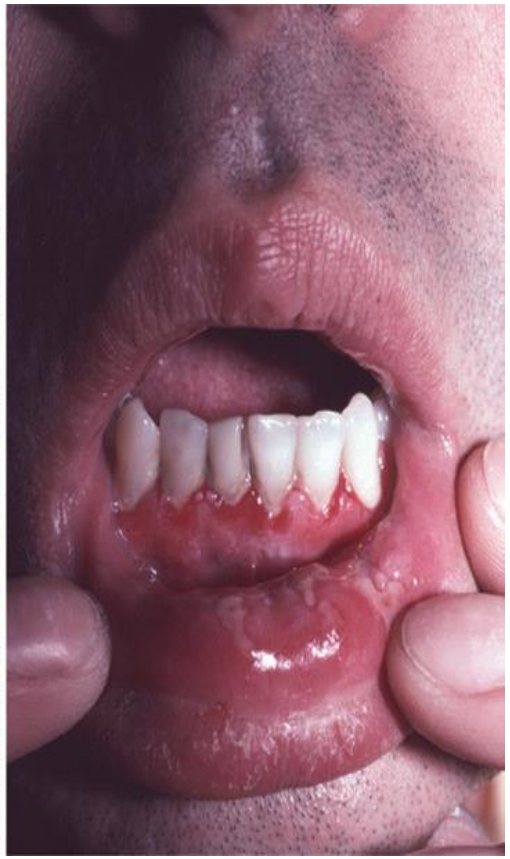
PEMPHIGUS VULGARIS:

EROSION ON LEG:GROUP OF CONFLUENT, UNROOFED BLISTERS.

SUPRABASAL ACANTHOLYSIS RESULTS IN INTRAEPIDERMAL BLISTER.

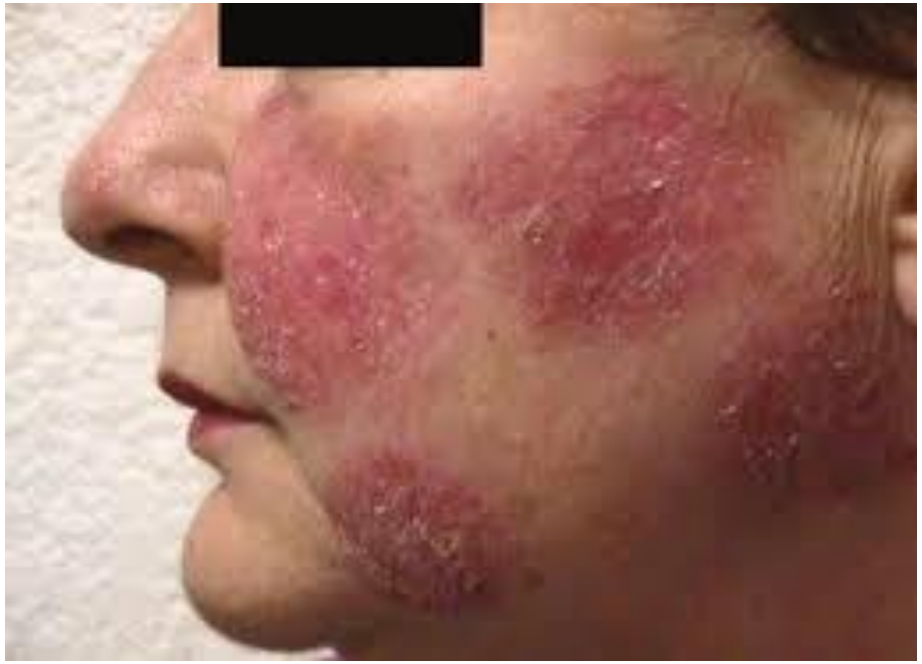


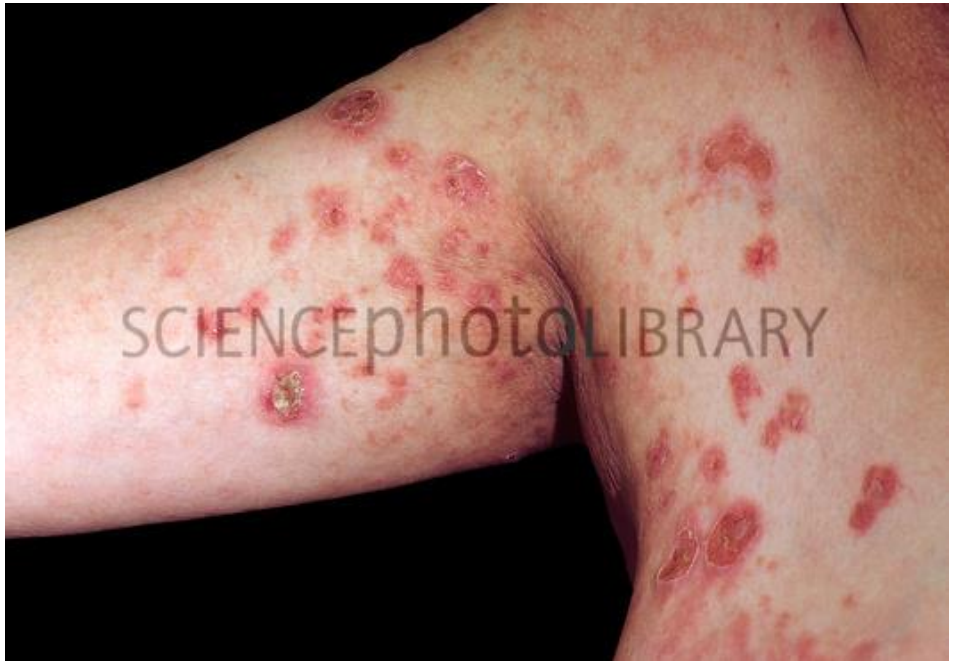




2-Pemphigus foliaceus:

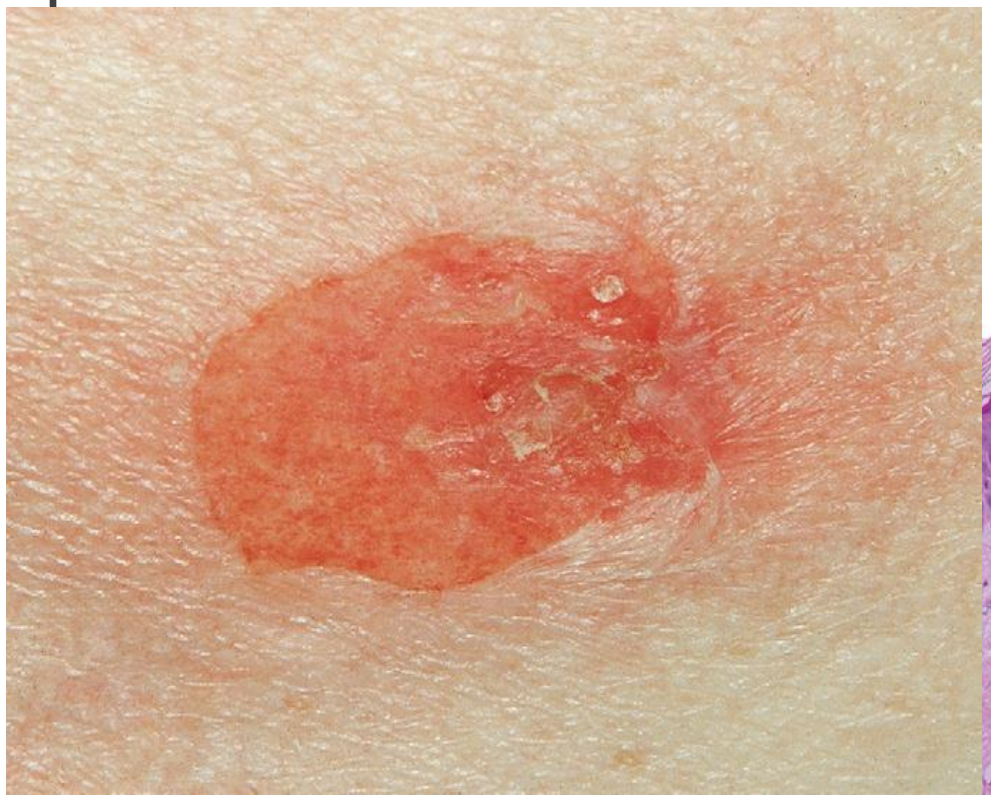
- Rare, more benign form of pemphigus.
- Bullae confined to skin.
- Infrequent involvement of mucous membranes.
- Blisters are superficial with more limited zones of erythema & crusting of ruptured blisters.

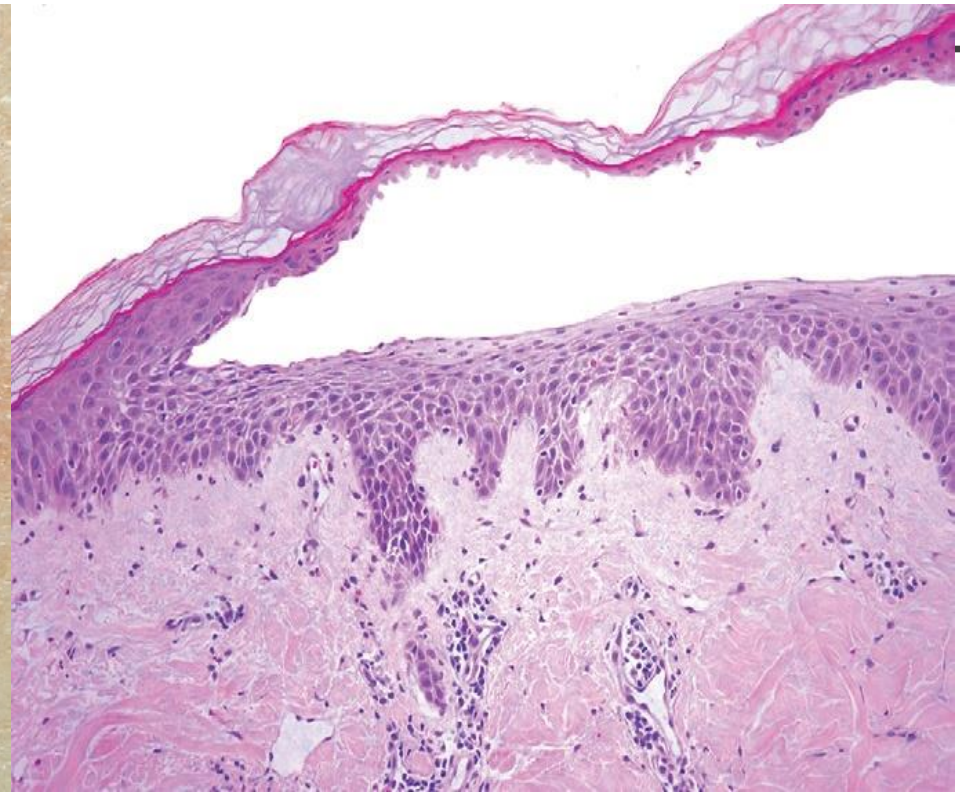




PEMPHIGUS FOLIACEUS:

GROSS APPEARANCE OF TYPICAL BLISTER, LESS SEVERELY ERODED THAN THOSE SEEN IN PEMPHIGUS VULGARIS.
MICROSCOPIC: CHARACTERISTIC SUBCORNEAL BLISTER.





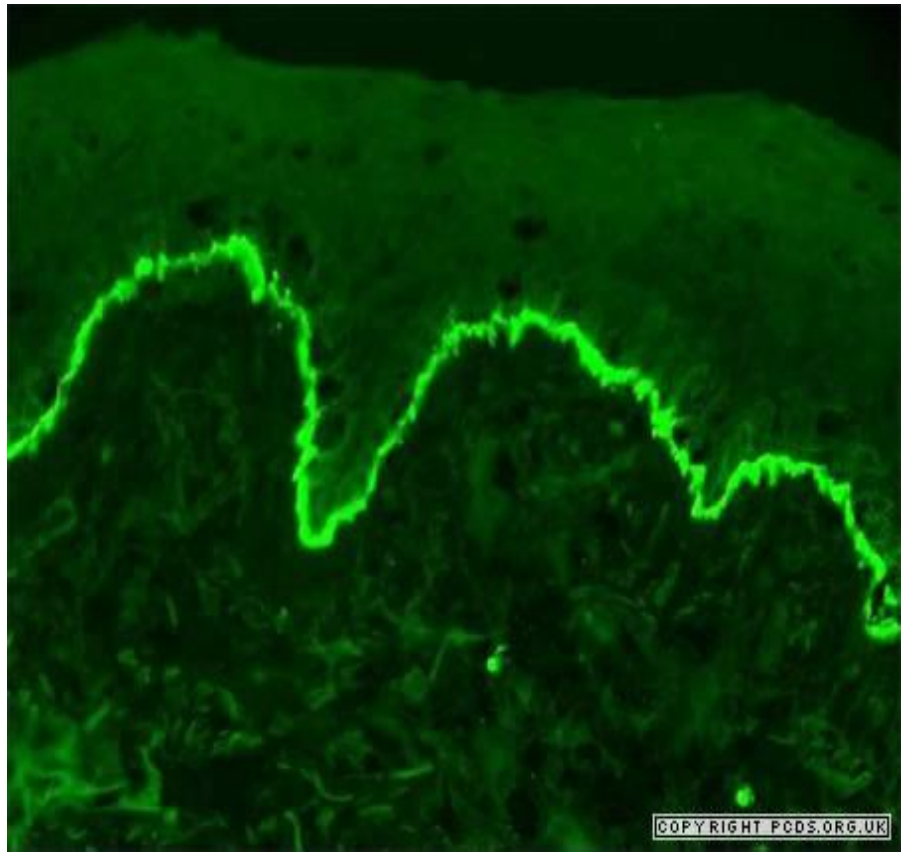
2-Bullous pemphigoid:

Acquired blistering disorder with autoimmune basis.

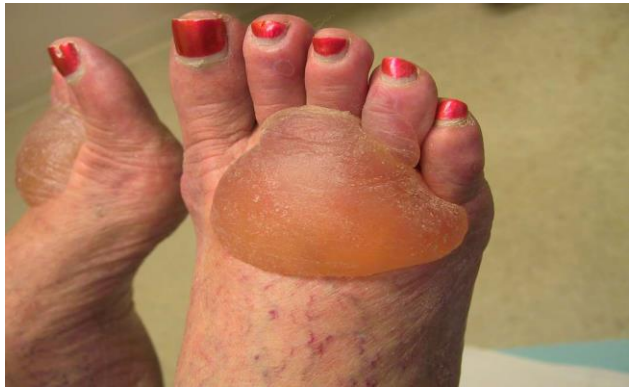
- Pathogenesis

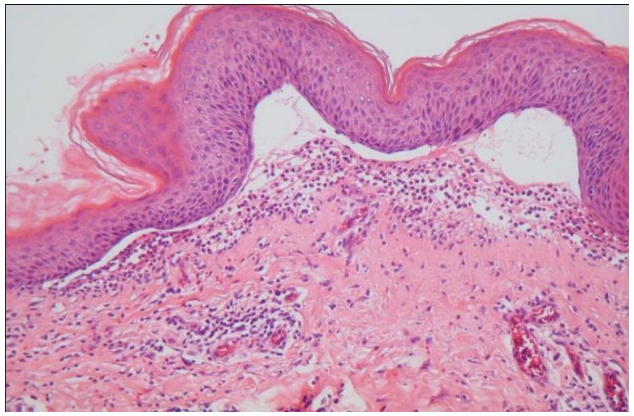
Blistering is triggered by linear deposition of IgG antibodies in epidermal basement membrane.

Deposition of IgG antibody detected by direct immunofluorescence as linear band outlining the subepidermal basement membrane zone.



- Morphology: Grossly:
- Tense bullae filled with clear fluid.
- Subepidermal nonacantholytic blisters.
- Perivascular infiltrate of lymphocytes & eosinophils.
- Superficial dermal edema.
- Basal cell vacuolization gives rise to fluid-filled blister.



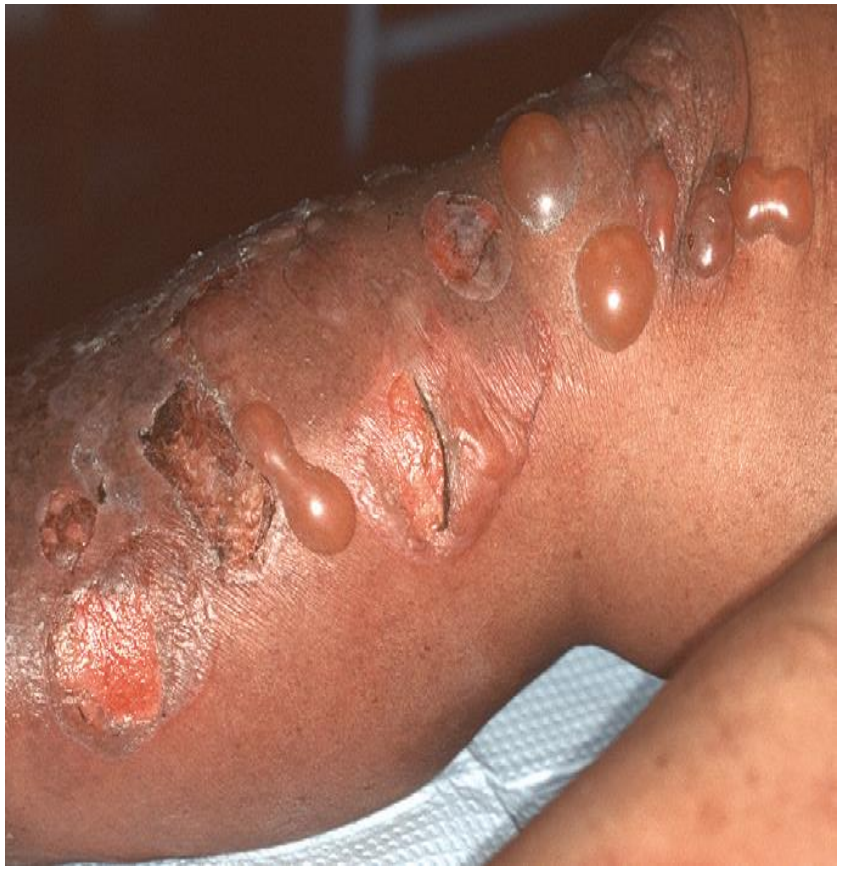


- Blister roof consists of full thickness with intact intercellular junctions so epidermis not rupture easily.

(Key distinction from blisters in pemphigus)

BULLOUS PEMPHIGOID.

Gross appearance of tense, fluid filled blisters.



Subepidermal vesicle with inflammatory infiltrate rich in eosinophils.