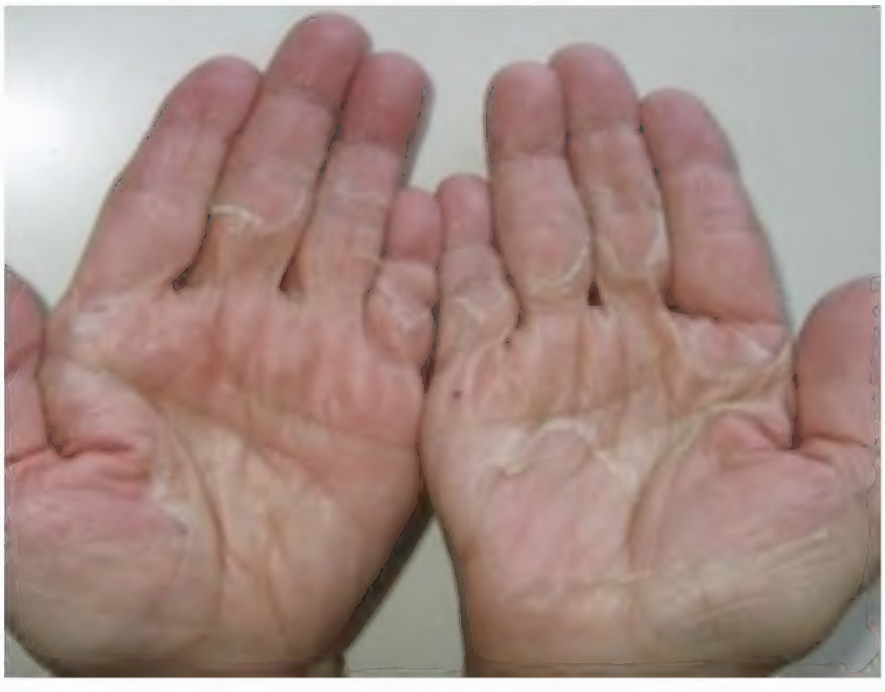
1. What are chronic inflammatory dermatoses?

- Chronic inflammatory dermatoses are persistent skin conditions characterized by exhibiting their most characteristic features over many months to years.

2. What happens to the skin surface in some chronic inflammatory dermatoses?

- The skin surface in some chronic inflammatory dermatoses becomes roughened due to excessive or abnormal scale formation and shedding (desquamation).



1. What is Psoriasis?
- Psoriasis is a common chronic inflammatory dermatosis.

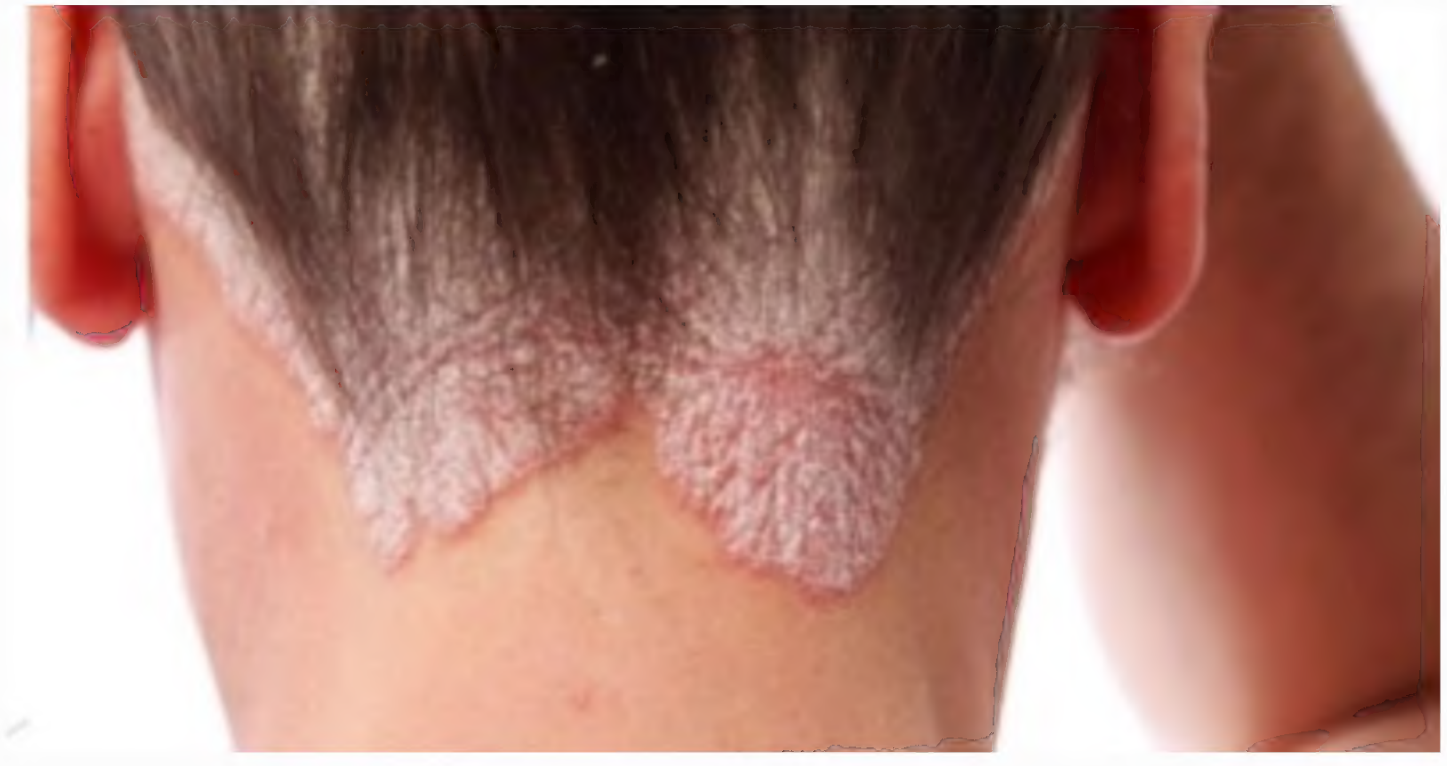
2. What percentage of individuals does psoriasis typically affect?
- Psoriasis affects 1% to 2% of individuals.

3. What health risks are associated with psoriasis?
- Psoriasis is associated with an increased risk for heart attack and stroke.

4. What is the relationship between psoriasis and heart attack/stroke?
- The relationship between psoriasis and heart attack/stroke may be related to a

chronic inflammatory state.

5. What percentage of patients with psoriasis develop arthritis?
- Up to 10% of patients with psoriasis develop arthritis.



6. What is the presumed origin of psoriasis?
- Psoriasis is presumed to be autoimmune

in origin.

7. What contributes to the risk of psoriasis?
- Both genetic and environmental factors contribute to the risk of psoriasis.

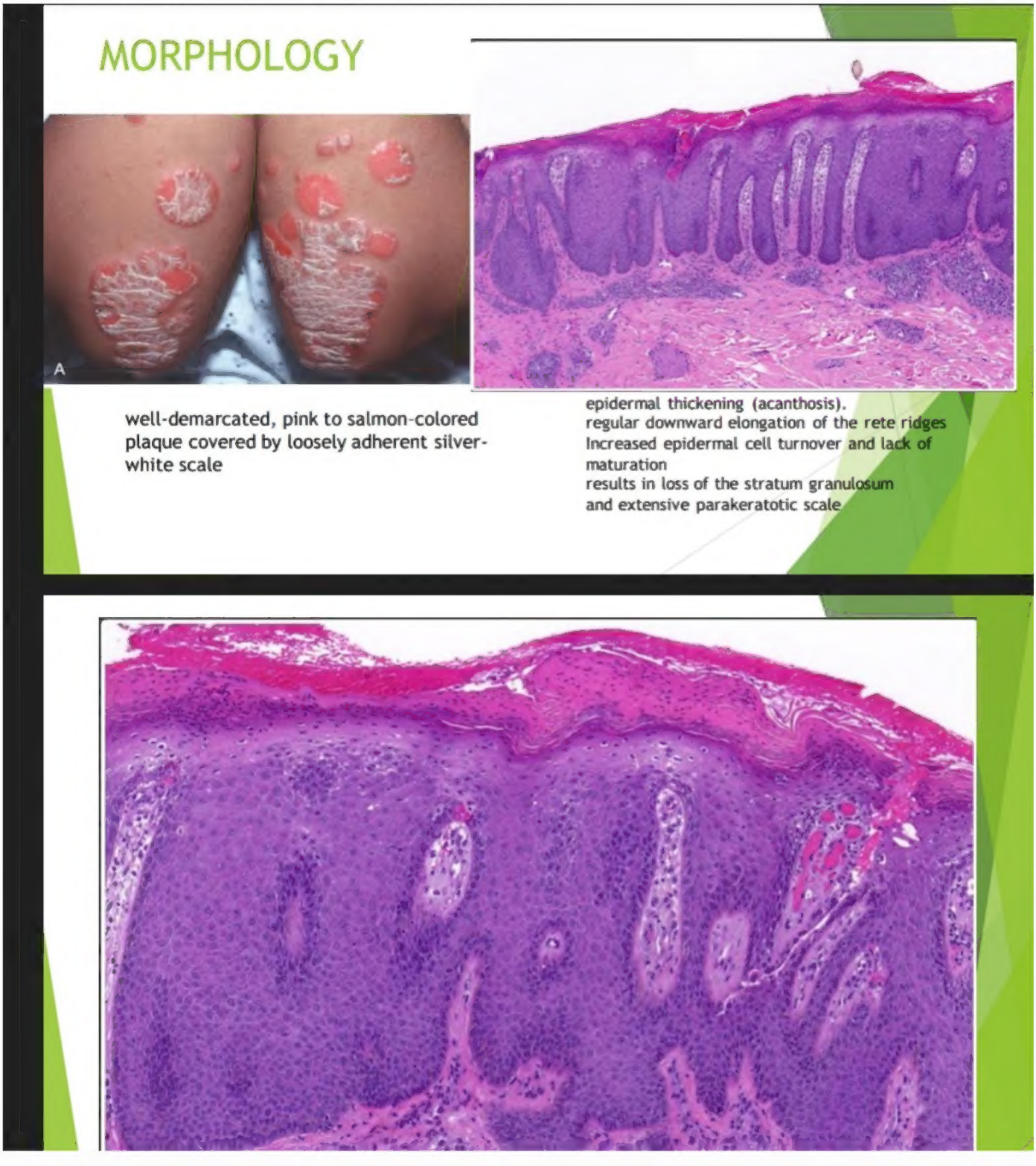
8. Which type of cells are involved in the pathogenesis of psoriasis?
- Sensitized populations of T cells, including CD4+TH17, TH1 cells, and CD8+ T cells, are involved in the pathogenesis of psoriasis.

9. Where do sensitized populations of T cells accumulate?
- Sensitized populations of T cells home to the dermis and accumulate in the epidermis.

**10. What do these cells secrete, leading

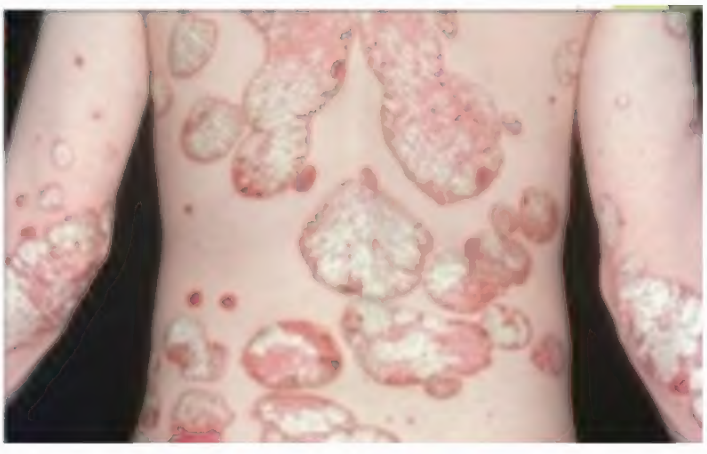
to characteristic lesions?**

- These cells secrete cytokines and growth factors that induce keratinocyte hyperproliferation, resulting in characteristic lesions.



1. What are the common skin areas affected by psoriasis?

- Psoriasis most frequently affects the skin of the elbows, knees, scalp, lumbosacral areas, intergluteal cleft, glans penis, and vulva.



2. What percentage of psoriasis cases exhibit nail changes on the fingers and toes?

- Nail changes on the fingers and toes occur in 30% of psoriasis cases.

3. How is mild psoriasis treated topically?

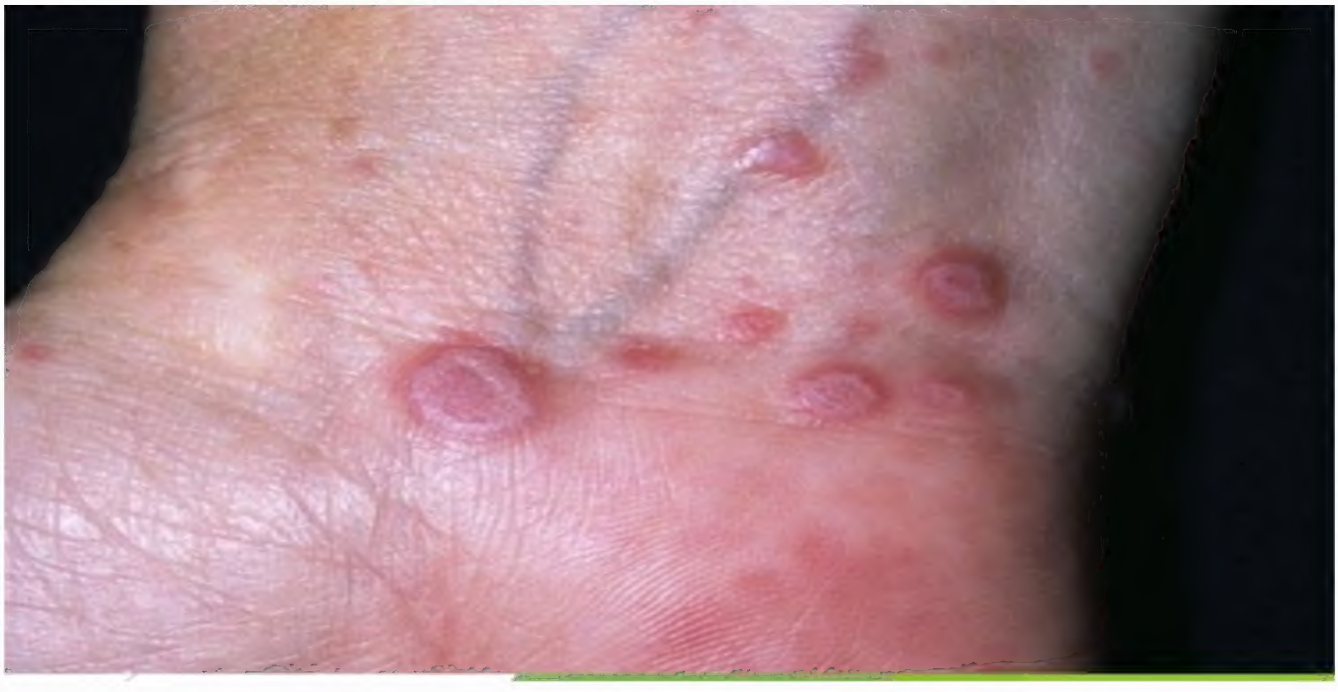
- Mild psoriasis is treated topically with ointments containing corticosteroids or other immunomodulatory agents.

4. What treatment options are available for more severe cases of psoriasis?
- More severe cases of psoriasis are treated with phototherapy (which has immunosuppressive effects) or systemic therapy with immunosuppressive agents such as methotrexate or TNF antagonists.

5. How is lichen planus described clinically?
- Lichen planus is described as "pruritic, purple, polygonal, papules, and plaques."

6. What type of immune response may cause the lesions in lichen planus?
- The lesions in lichen planus may result from a CD8+ T cell-mediated cytotoxic

response against antigens in the basal cell layer and the dermoepidermal junction.



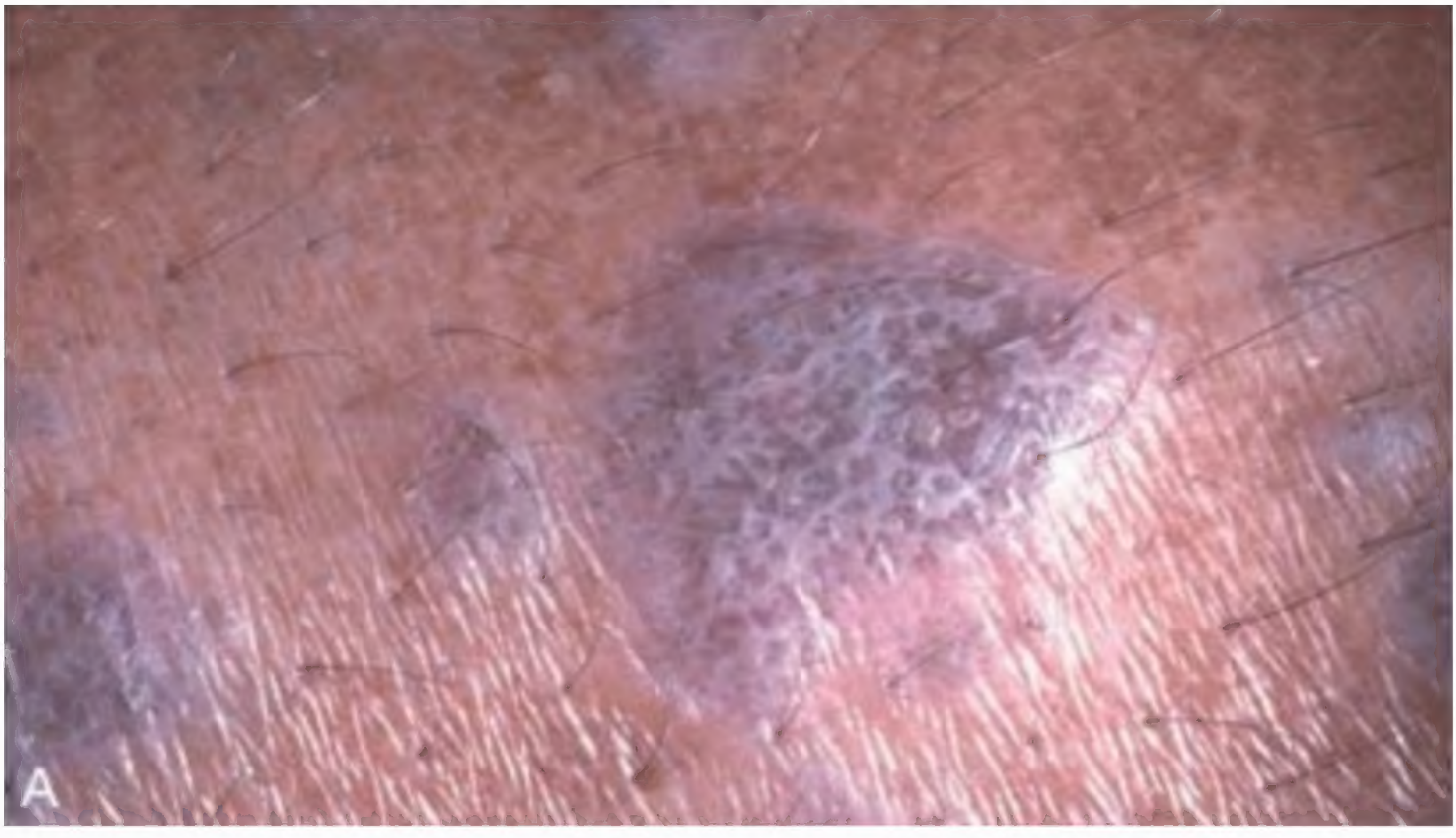
**7. What is highlighted by white dots or lines in cutaneous lesions of lichen planus? **

- Cutaneous lesions of lichen planus are highlighted by white dots or lines termed Wickham striae.

8. What may result in hyperpigmentation in lichen planus?

- Hyperpigmentation in lichen planus may

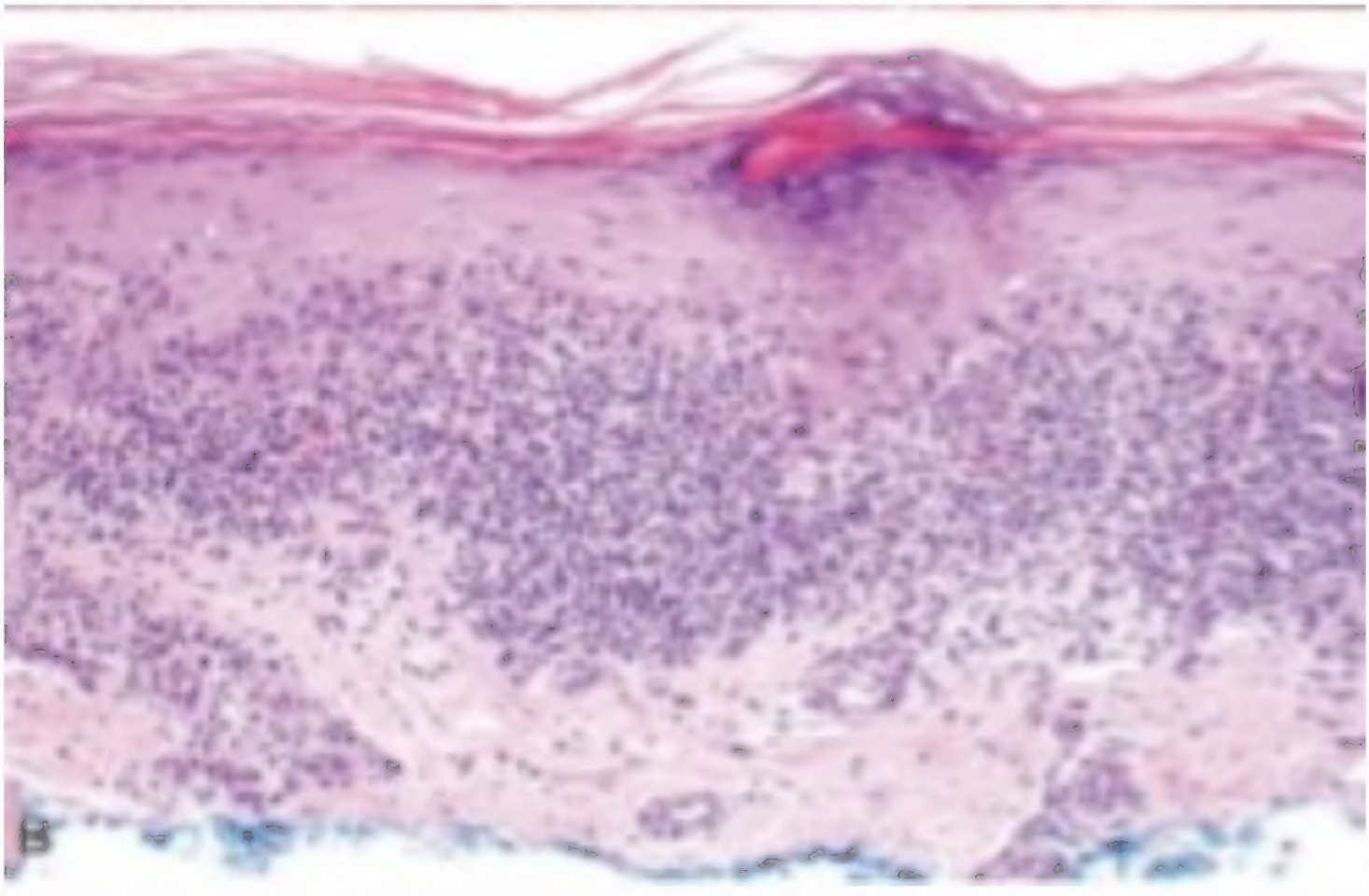
result from melanin loss into the dermis from damaged keratinocytes.



1. What is the microscopic characteristic of lichen planus?

- Lichen planus is characterized microscopically as a prototypical interface dermatitis, with inflammation and damage concentrated at the interface of the squamous epithelium and papillary dermis. There is a dense, continuous infiltrate of lymphocytes along the

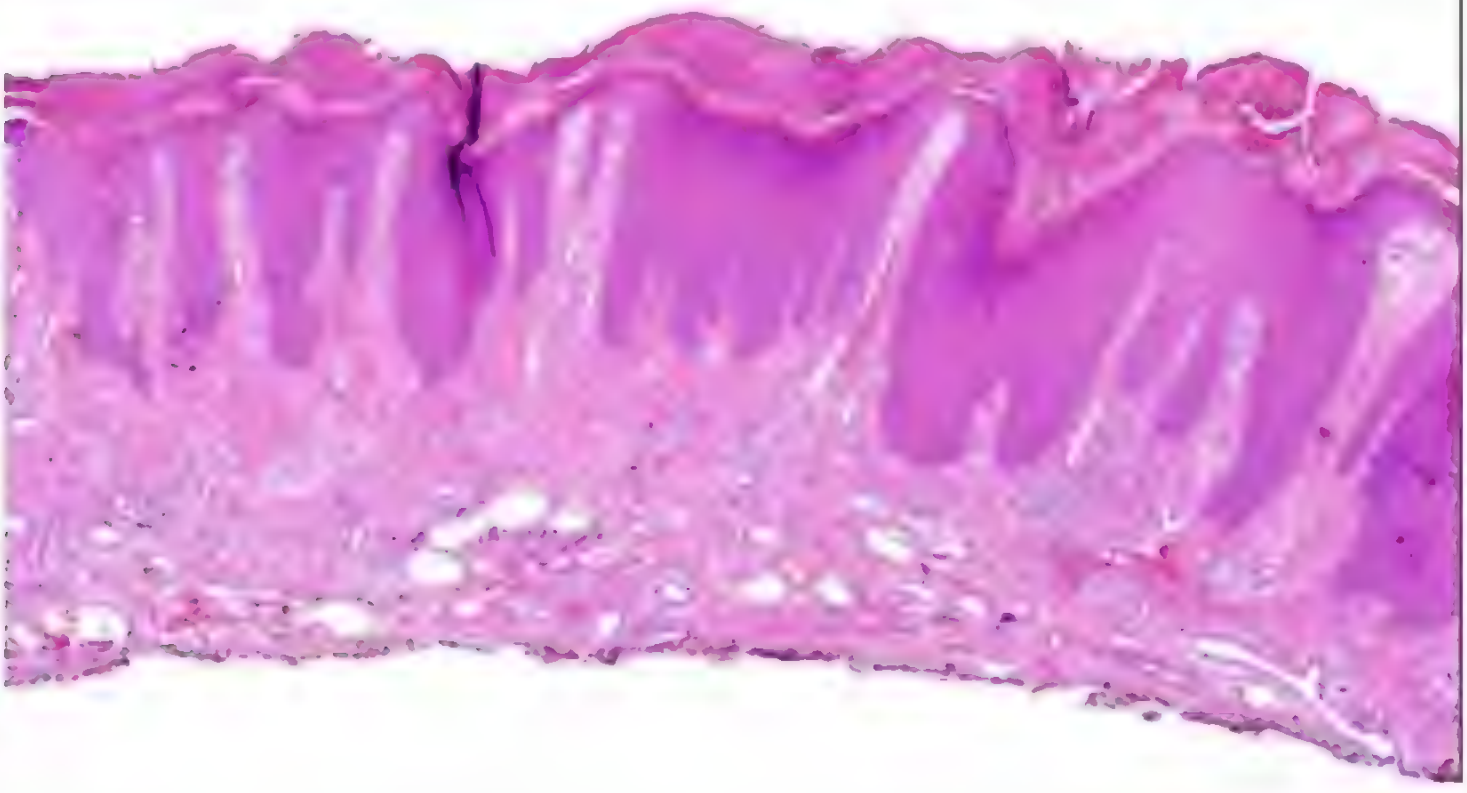
dermoepidermal junction.



2. What are Civatte bodies?
- Civatte bodies are observed microscopically in lichen planus.

3. How does lichen simplex chronicus manifest clinically?
- Lichen simplex chronicus manifests as roughening of the skin, typically in response to local repetitive trauma, such

as rubbing or scratching.



4. What is the pathogenesis of lichen simplex chronicus?
- The pathogenesis of lichen simplex chronicus is not well understood, but it is believed that trauma induces epithelial hyperplasia and eventual dermal scarring.

5. What are the microscopic characteristics of lichen simplex chronicus?
- Microscopically, lichen simplex chronicus is characterized by acanthosis,

hyperkeratosis, and hypergranulosis.

6. What distinguishes blistering disorders from other conditions where vesicles and bullae occur as secondary phenomena?

- Blistering disorders, such as pemphigus (vulgaris and foliaceus) and bullous pemphigoid, are characterized by blisters as the primary and most distinctive feature. The blisters tend to occur at specific levels within the skin, which is a morphologic distinction critical for diagnosis.

1. What forms the basis of the differential diagnosis for blistering disorders?

- The level of epidermal separation forms the basis of the differential diagnosis for

blistering disorders.

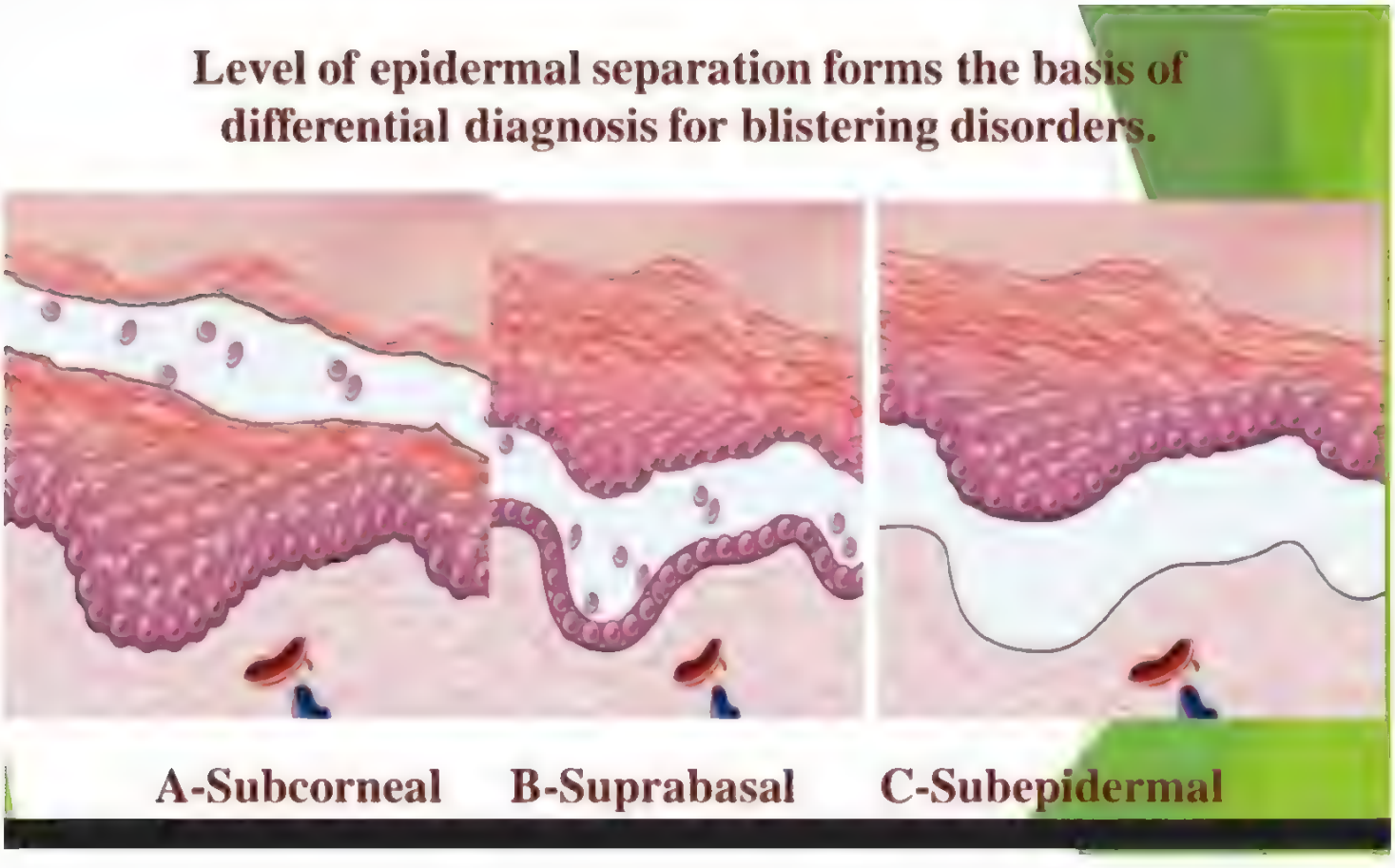
2. What are the three levels of epidermal separation mentioned in the text?

- The three levels of epidermal separation are:

A. Subcorneal

B. Suprabasal

C. Subepidermal



1. What is Pemphigus, and what causes it?

- Pemphigus is an uncommon autoimmune blistering disorder resulting from the loss of normal intercellular attachments within the epidermis and squamous mucosal epithelium. It is caused by antibody-mediated hypersensitivity reactions.

2. What are the three major variants of Pemphigus mentioned in the text?

- The three major variants of Pemphigus are:
- Pemphigus vulgaris (the most common type)
- Pemphigus foliaceus
- Paraneoplastic pemphigus, which is associated with internal malignancy.

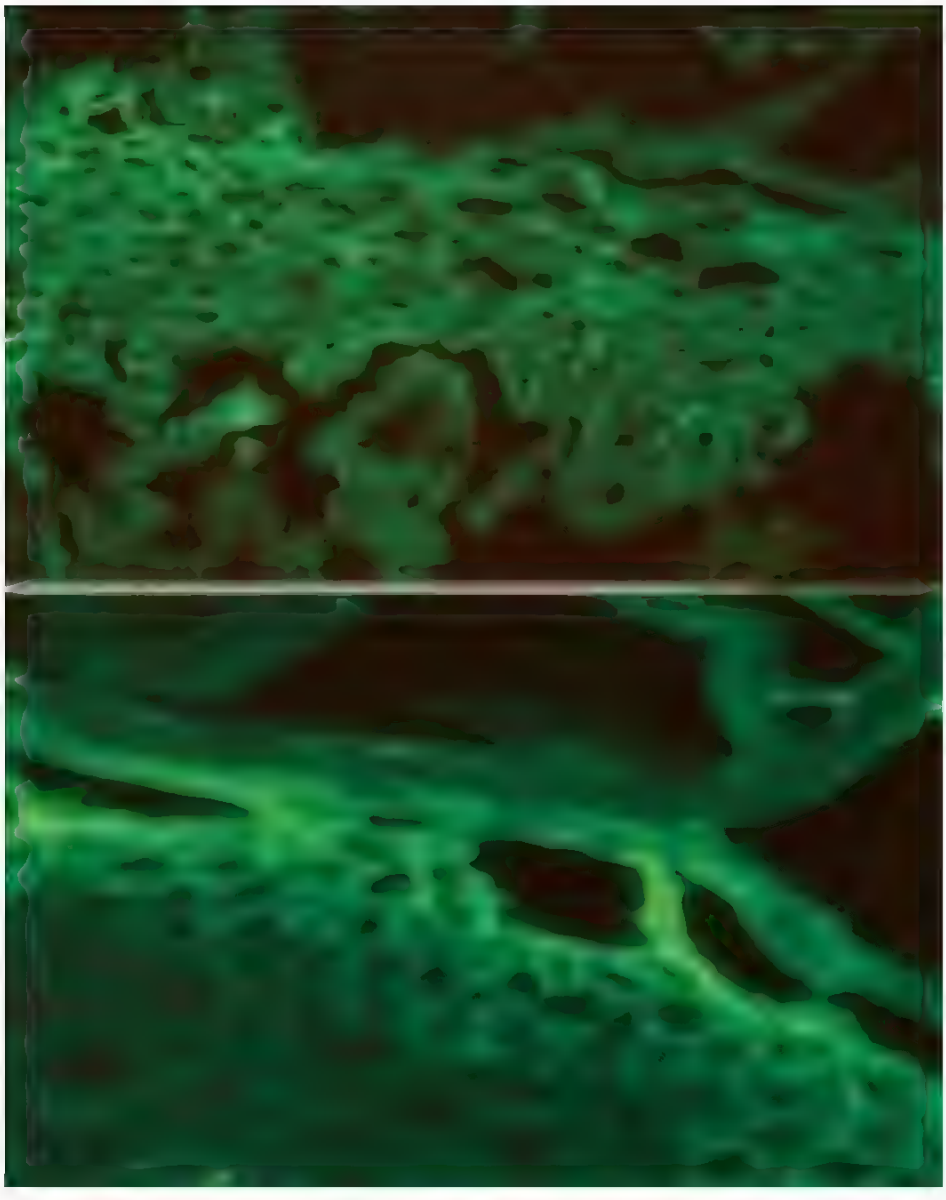
**3. What is the pathogenesis of Pemphigus vulgaris and Pemphigus

foliaceus?**

- Pemphigus vulgaris and Pemphigus foliaceus are autoimmune diseases caused by antibody-mediated hypersensitivity reactions (Type II hypersensitivity). Pathogenic antibodies, specifically IgG autoantibodies, bind to intercellular desmosomal proteins of the skin and mucous membranes, disrupting the intercellular adhesive function of desmosomes and activating intercellular proteases.

4. How is the diagnosis of Pemphigus confirmed?

- The diagnosis of Pemphigus is confirmed through a direct immunofluorescence study, where lesional sites show a characteristic fishnet-like pattern of intercellular IgG deposits.



5. What is Acantholysis, and what is its histological picture in all forms of Pemphigus?

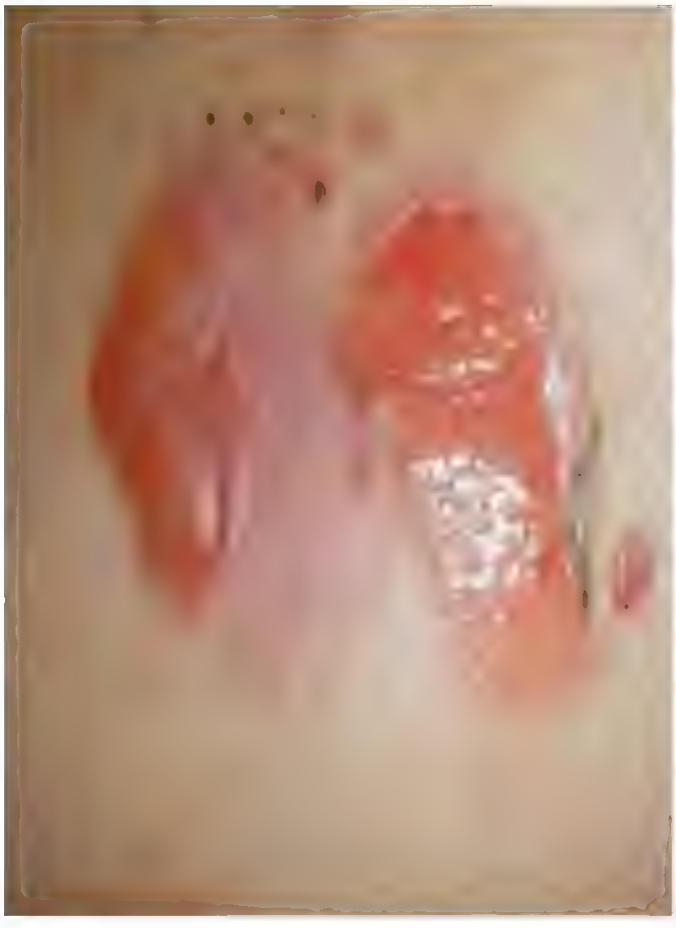
- Acantholysis is the lysis of intercellular adhesive junctions between neighboring squamous epithelial cells, resulting in detached cells. The histological picture in all forms of Pemphigus includes acantholysis and variable superficial

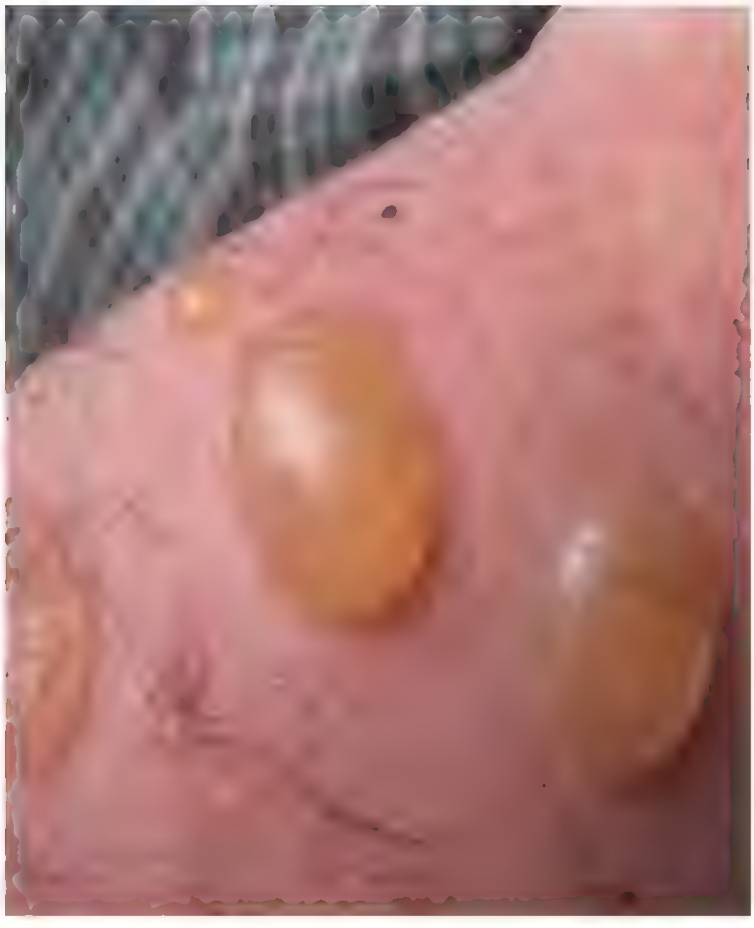
dermal infiltrates comprised of lymphocytes, macrophages, and eosinophils.

6. What are the clinical manifestations of Pemphigus vulgaris?

- Pemphigus vulgaris involves both mucosa and skin, affecting areas such as the scalp, face, axillae, groin, trunk, and points of pressure.

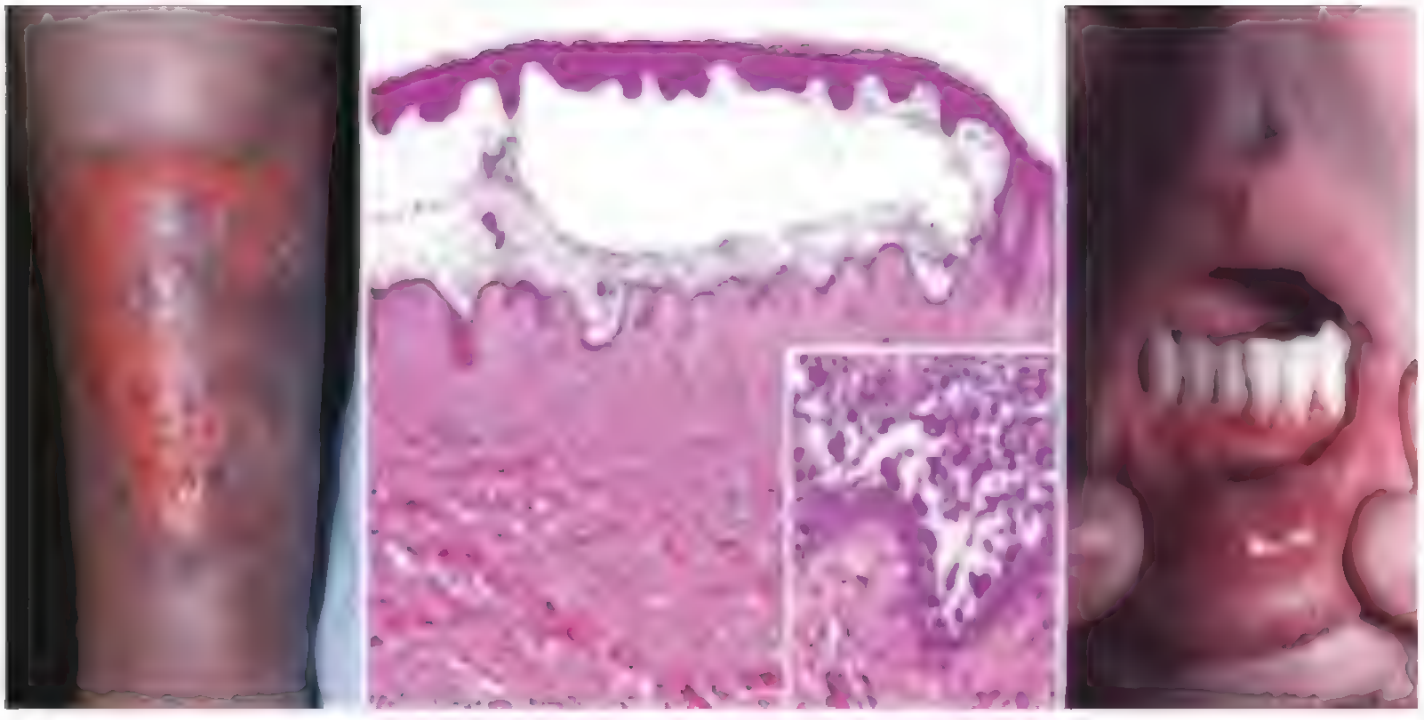
Lesions include superficial vesicles and bullae that rupture easily, leading to extensive erosions covered with serum crust.





7. What distinguishes Pemphigus vulgaris from Pemphigus foliaceus histologically?

- In Pemphigus vulgaris, suprabasal acantholysis results in intraepidermal blisters,



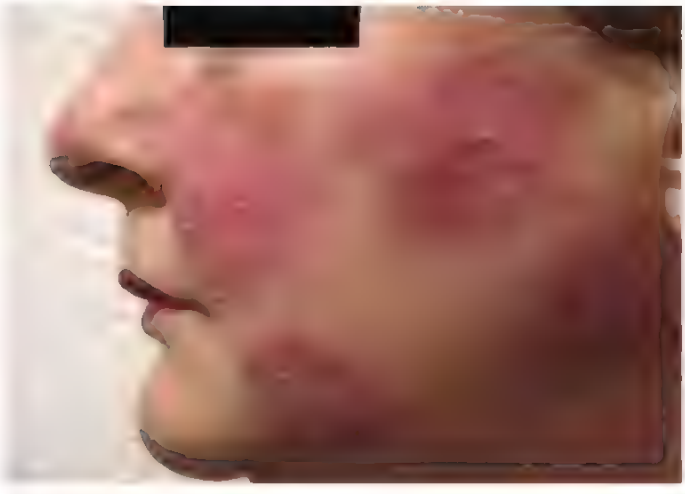
while in Pemphigus foliaceus, acantholysis of the superficial epidermis occurs at the level of the stratum granulosum.

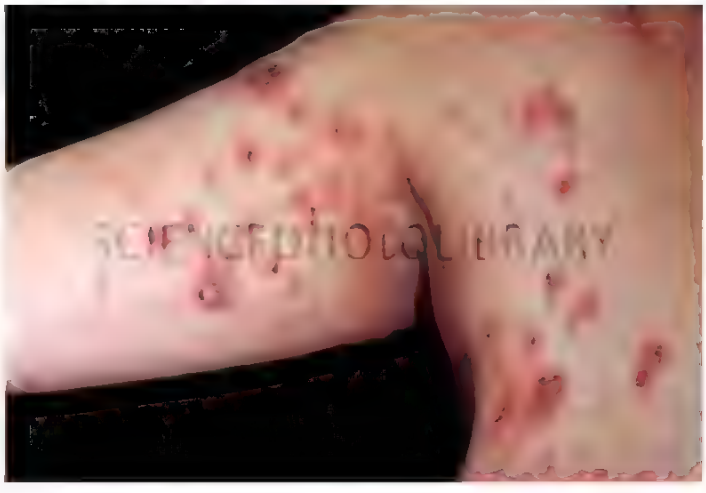
€€What is Pemphigus foliaceus, and how does it differ from Pemphigus vulgaris?

Answer:

Pemphigus foliaceus is a rare and more benign form of pemphigus characterized by bullae confined to the skin. It infrequently involves mucous membranes.

Blisters in Pemphigus foliaceus are superficial and exhibit more limited zones of erythema and crusting of ruptured lesions compared to Pemphigus vulgaris.





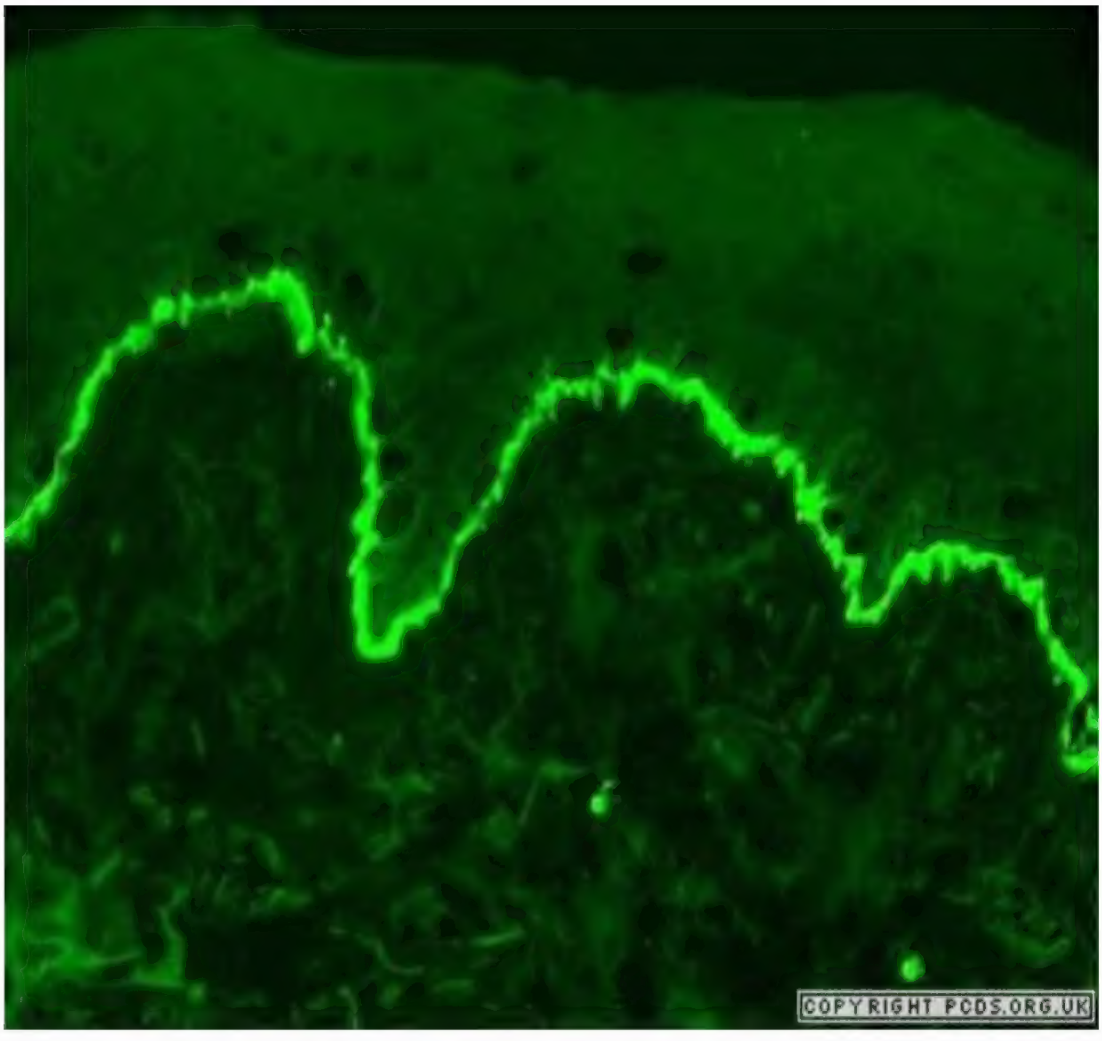
1. What is the gross appearance of a typical blister in Pemphigus foliaceus, and how does it compare to those seen in Pemphigus vulgaris?

- The gross appearance of a typical blister in Pemphigus foliaceus is less severely eroded than those seen in Pemphigus vulgaris.

than those seen in pemphigus vu
Microscopic: Characteristic sub

2. What is the pathogenesis of Bullous pemphigoid, and how is blistering triggered?

- Bullous pemphigoid is an acquired blistering disorder with an autoimmune basis. Blistering is triggered by the linear deposition of IgG antibodies in the epidermal basement membrane.



3. How is the deposition of IgG antibody detected in Bullous pemphigoid?

- The deposition of IgG antibody is detected by direct immunofluorescence as a linear band outlining the subepidermal basement membrane zone.

4. What is the gross morphology of blisters in Bullous pemphigoid?

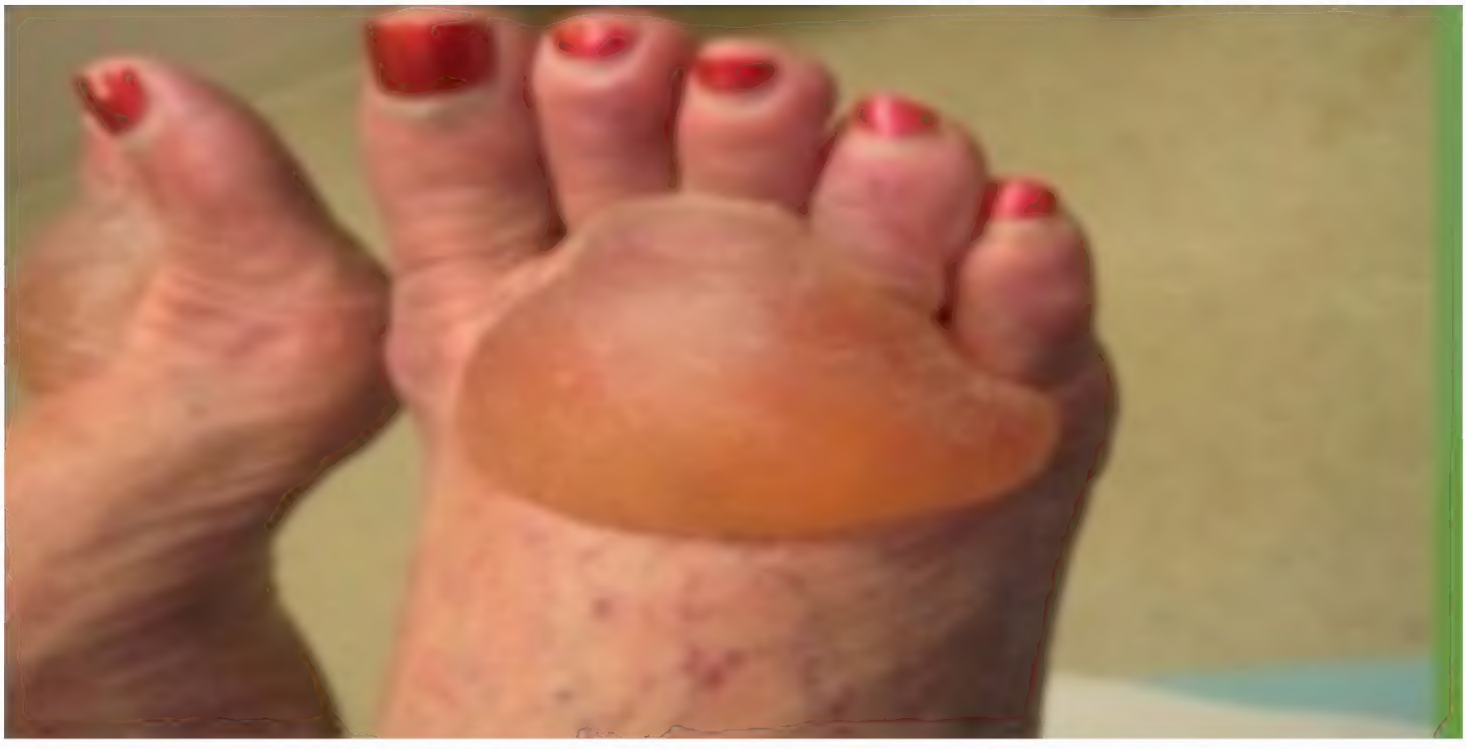
- Grossly, blisters in Bullous pemphigoid

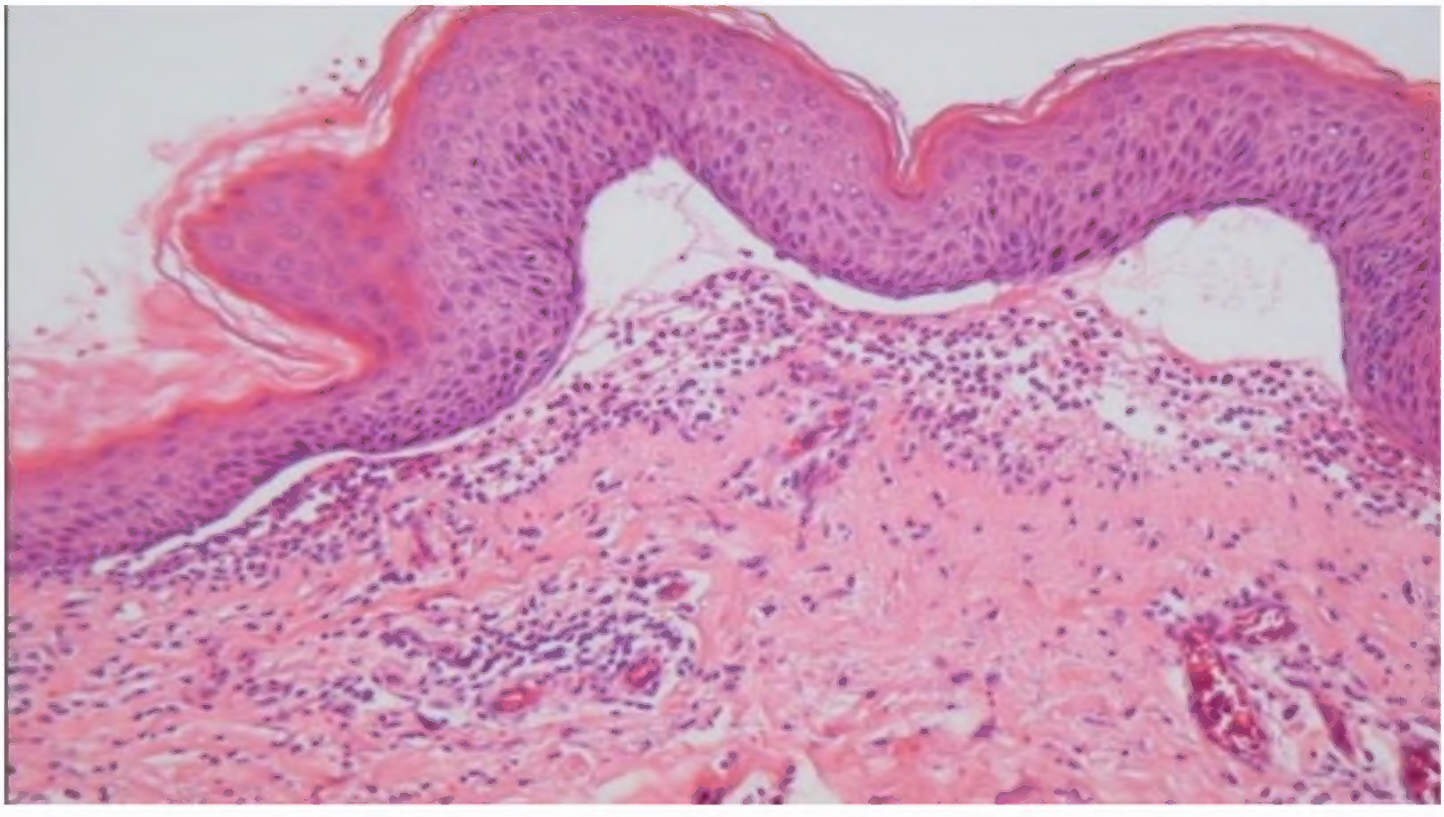
- Grossly, blisters in Bullous pemphigoid appear as tense bullae filled with clear fluid.

5. What is the microscopic characteristic of blisters in Bullous pemphigoid?
- Microscopically, blisters in Bullous pemphigoid are subepidermal non-acantholytic blisters with a perivascular infiltrate of lymphocytes and eosinophils, accompanied by superficial dermal edema.

6. What causes the formation of the blister roof in Bullous pemphigoid, and how does it differ from Pemphigus blisters?
- Basal cell vacuolization gives rise to fluid-filled blisters in Bullous pemphigoid. The blister roof consists of full thickness with intact intercellular junctions, making the epidermis less likely to rupture easily. This

is a key distinction from blisters in Pemphigus.





What is the gross appearance of blisters in Bullous pemphigoid, and what is the microscopic characteristic of the blister?

Answer:

The gross appearance of blisters in Bullous pemphigoid is tense and filled with fluid. Microscopically, Bullous pemphigoid presents with a subepidermal vesicle accompanied by an inflammatory infiltrate rich in eosinophils.