



الكلية : الطب

القسم او الفرع : الامراض والطب العدلي

المرحلة: الثالثة

أستاذ المادة : امراض

اسم المادة باللغة العربية : علم الامراض النسيجي

سم المادة باللغة الإنكليزية : pathology

اسم المحاضرة الرابعة عشر باللغة العربية: امراض الجلد/ المحاضرة الرابعة عشر

اسم المحاضرة الرابعة عشر باللغة الإنكليزية : skin pathology

## Terms

- ❑ **Acanthosis:** marked epidermal thickening.
- ❑ **Spongiosis:** intercellular epidermal edema.
- ❑ **Parakeratotic:** pyknotic keratinocyte nuclei with in stratum corneum
- ❑ **Hyperkeratosis:** increase thickness of stratum corneum
- ❑ **Acantholysis:** loss of coherence between epidermal cells due to breakdown of intercellular bridge

## ***ACUTE INFLAMMATORY DERMATOSES***

### **Urticaria:**

Urticaria (“hives”) is a common disorder mediated by localized mast cell degranulation, which leads to dermal microvascular hyperpermeability. The resulting erythematous, edematous, and pruritic plaques are termed wheals

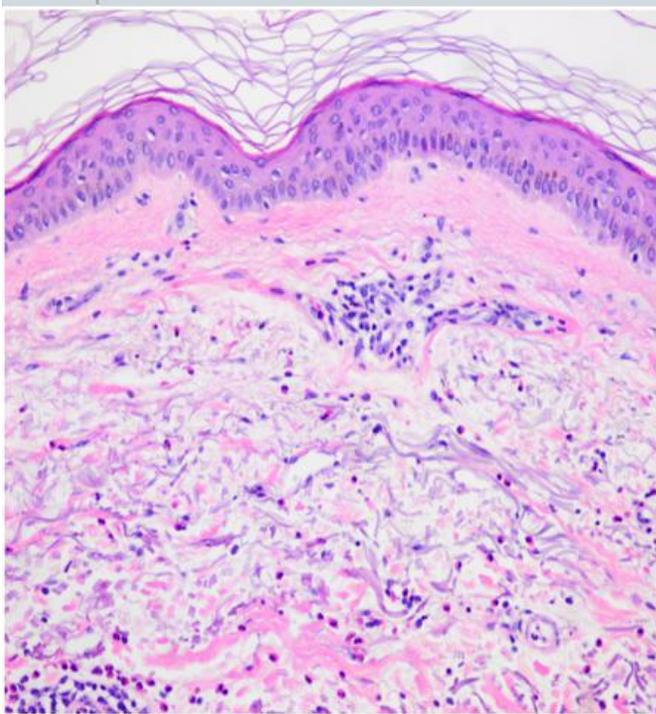
### ***MORPHOLOGY***

There is usually

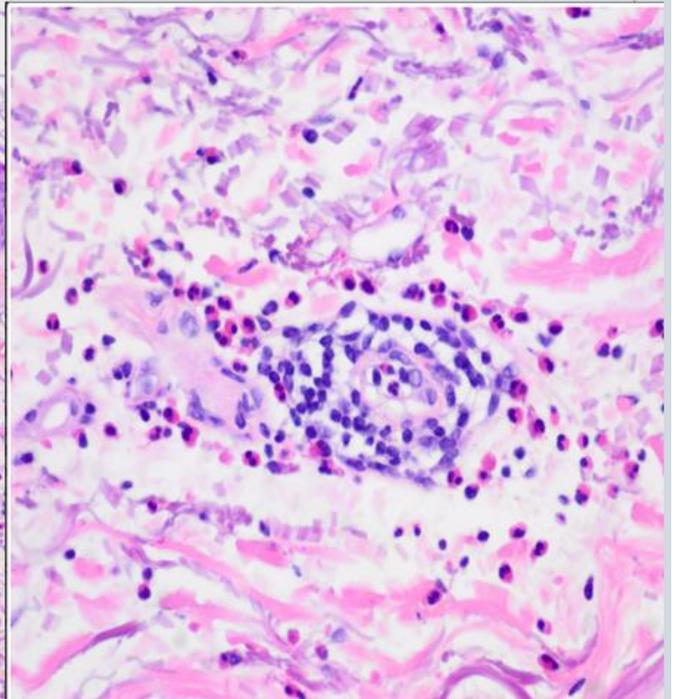
- ❑ a sparse superficial perivenular infiltrate of mononuclear cells.
- ❑ Superficial dermal edema causes splaying of collagen bundles, making them appear to be more widely spaced than normal.
- ❑ Degranulation of mast cells, which reside around superficial dermal venules, can be identified a Giemsa stain



Widespread lesions. They can be polycyclic or annular.



Mild dermal edema and predominant perivascular inflammatory infiltrate composed of lymphocytes and eosinophils.



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### *Acute Eczematous Dermatitis*

- Allergic contact dermatitis
- Atopic dermatitis
- Drug-related eczematous dermatitis
- Photoeczematous dermatitis
- Primary irritant dermatitis

## Erythema Multiforme

Erythema multiforme is characterized by epithelial injury mediated by **skin-homing CD8+ cytotoxic T lymphocytes**.

self-limited disorder that appears to be a hypersensitivity response to certain infections and drugs.

- ❑ Infections include those caused by **herpes simplex, mycoplasma, and some fungi, while implicated drugs include sulfonamides, penicillin, salicylates, and anti-malaria's**.

The cytotoxic T cell attack is focused on **the basal cells**, due to recognition of still unknown antigens. Certain human lymphocyte antigen (HLA) haplotypes are associated with the disease.

## 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**
- A superficial perivascular lymphocytic infiltrate associated with dermal edema
- Margination of lymphocytes along the dermo epidermal junction
- Apoptotic keratinocytes.
- **With time**, discrete, confluent zones of basal. epidermal necrosis appear, with concomitant blister formation.
- In a rarer and more severe form of this disease, toxic epidermal necrolysis, the necrosis extends through the full thickness of the epidermis.

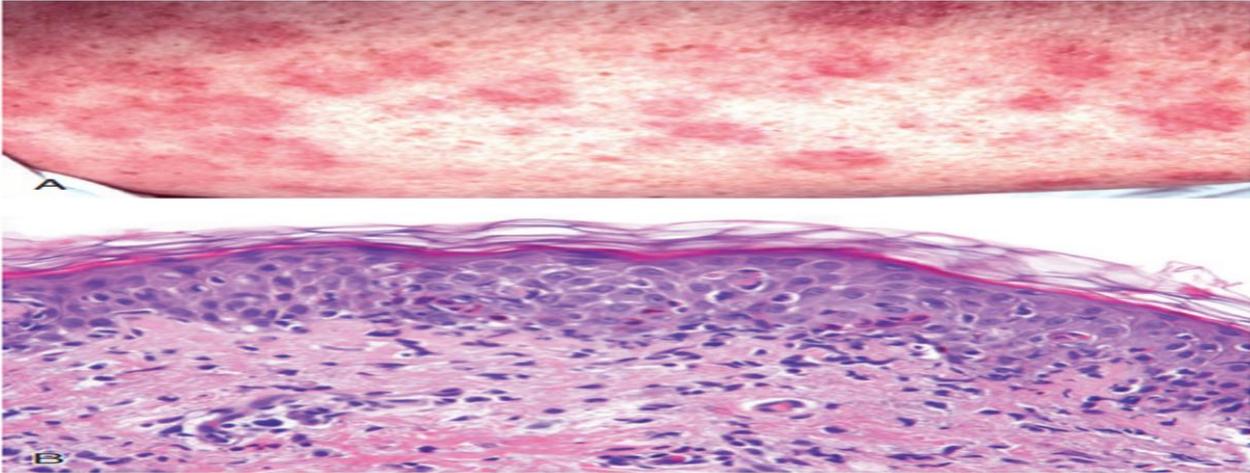


Fig. 24.2 Erythema multiforme. (A) Target like lesions consisting of a pale central blister or zone of epidermal necrosis surrounded by macular erythema. (B) Early lesions show lymphocytes along the dermoepidermal junction (interface dermatitis) associated with scattered apoptotic keratinocytes, marked by dark shrunken nuclei and eosinophilic cytoplasm.

## **CHRONIC INFLAMMATORY DERMATOSES**

### **Psoriasis**

- Psoriasis is a common chronic inflammatory dermatosis
- Recent studies have shown that 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, which in some cases may be severe

### **Pathogenesis:**

- ❑ Psoriasis is a T cell-mediated inflammatory disease, presumed to be autoimmune in origin, although the antigens are not well described. Both genetic (HLA types and other susceptibility loci) and environmental factors contribute to the risk.
- ❑ Sensitized populations of T cells home to the dermis, including CD4<sup>+</sup> TH17 and TH1 cells and CD8<sup>+</sup> T cells accumulate in the epidermis.
- ❑ These cells secrete cytokines and growth factors that induce keratinocyte hyperproliferation, resulting in the characteristic lesions.

❑ Psoriatic lesions can be induced in susceptible individuals by local trauma (Koebner phenomenon), which may induce a local inflammatory response that promotes lesion development.

- **MORPHOLOGY**

- The typical lesion

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

- **Microscopically**

- There is marked epidermal thickening (acanthosis), with regular downward elongation of the rete ridges.

- loss of the stratum granulosum and extensive parakeratotic scale.

- Thinning of the epidermal cell layer overlying the tips of dermal papillae (suprapapillary plates),

- Dilated and tortuous blood vessels within the papillae. These vessels bleed readily when the scale is removed, giving rise to multiple punctate bleeding points (Auspitz sign).

- Neutrophils form small aggregates within both the spongiotic superficial epidermis and the parakeratotic stratum corneum.

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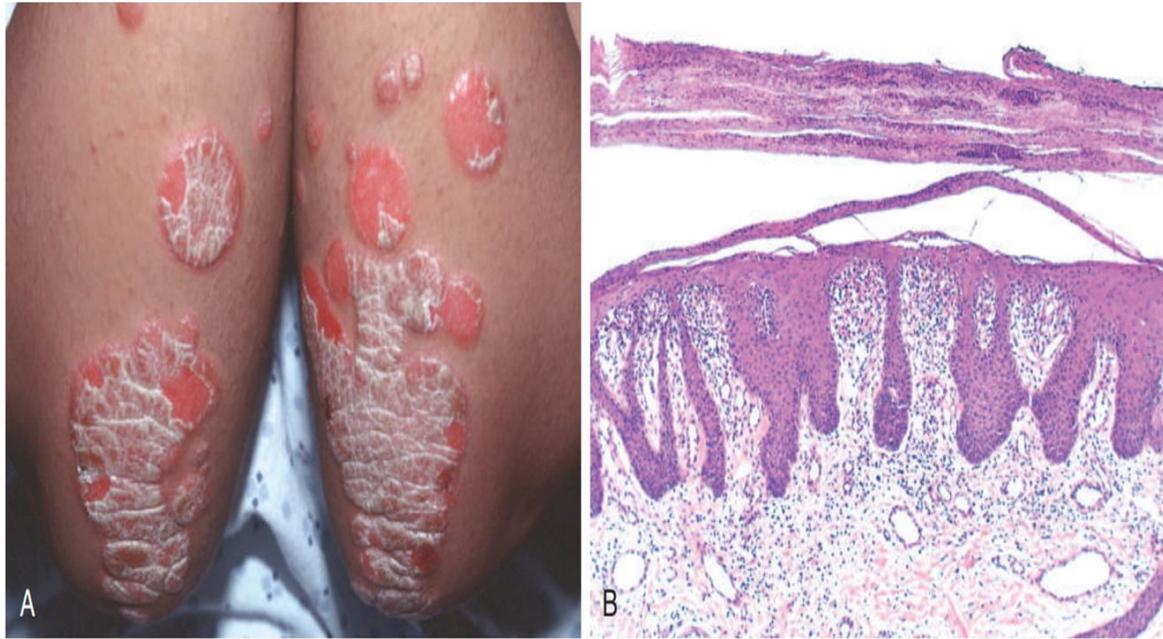


Fig. 24.3 Chronic psoriasis. (A) Erythematous psoriatic plaques covered by silvery-white scale. (B) Microscopic examination shows marked epidermal hyperplasia, downward extension of rete ridges (psoriasiform hyperplasia), and prominent parakeratotic scale with infiltrating neutrophils.

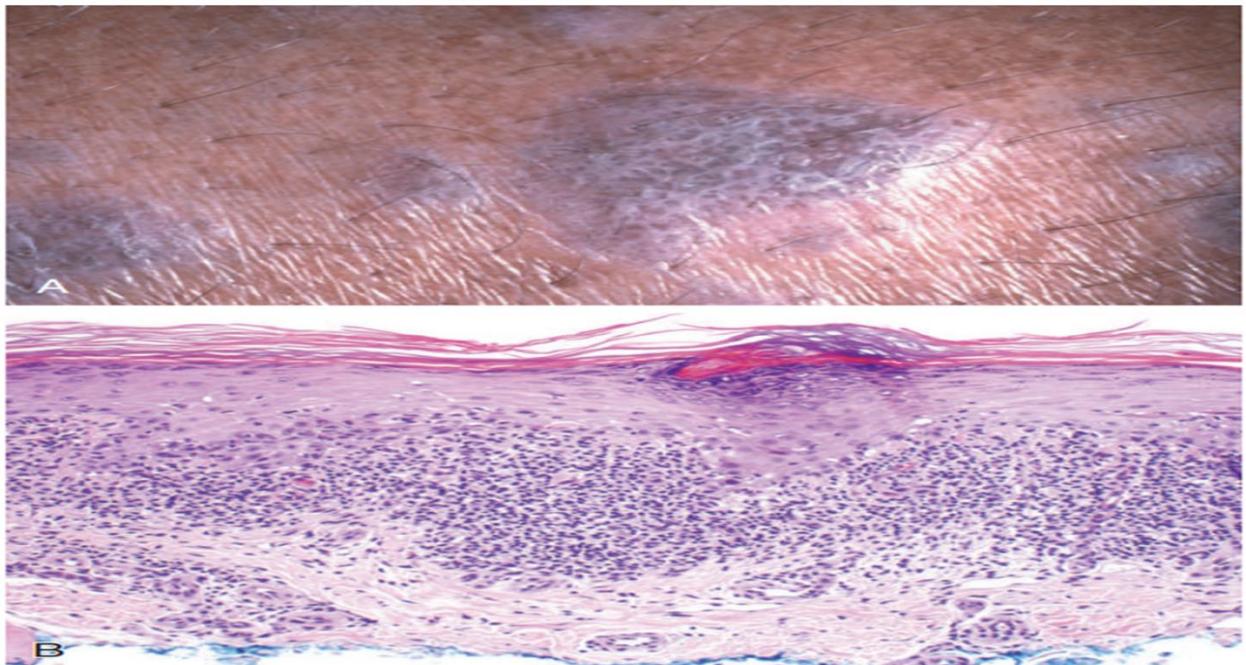
## Lichen Planus

### “Pruritic, purple, polygonal, planar papules, and plaques”

- The lesions may result from a CD8+ T cell–mediated cytotoxic response against antigens in the **basal cell layer and the dermo epidermal junction** that are produced by unknown mechanisms, perhaps as a consequence of a viral infection or drug exposure.
- 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.
- **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.
- Basal keratinocytes, which often atrophy or become necrotic.
- This pattern of inflammation causes the dermoepidermal interface to assume an angulated, zigzag contour (sawtoothing).
- Anucleate, necrotic basal cells are seen in the inflamed papillary dermis and are referred to as colloid bodies or Civatte bodies.
- Although these changes bear some similarities to those in erythema multiforme, lichen planus shows well-developed changes of **chronicity**, including **epidermal hyperplasia, hypergranulosis, and hyperkeratosis**



**Fig. 24.4 Lichen planus. (A) Flat-topped pink-purple polygonal papule with white lacelike markings referred to as Wickham striae. (B) Microscopic examination shows a bandlike infiltrate of lymphocytes along the dermoepidermal junction, hyperkeratosis, hypergranulosis, and pointed rete ridges ("sawtoothing"), which results from chronic injury of the basal cell layer.**

