

Musculoskeletal system (MSS)-Pathology 1

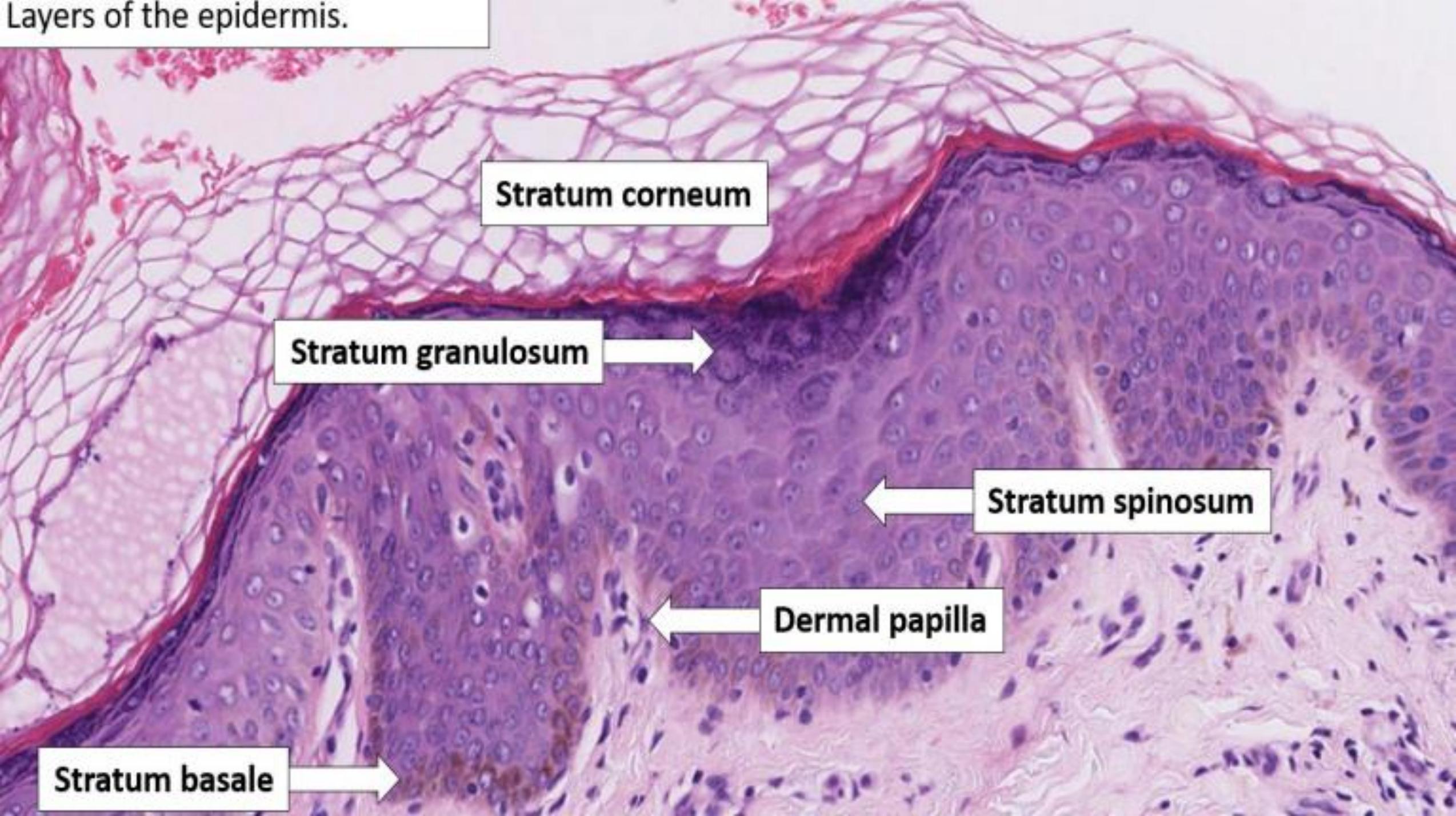
Acute Dermatitis

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Layers of the epidermis.



Stratum corneum

Stratum granulosum

Stratum spinosum

Dermal papilla

Stratum basale

Functions of skin



1. Squamous cells (keratinocytes) help maintain skin homeostasis by providing a physical barrier to environmental insults.



2. Has a major role in immunity.

- dermis contain: CD4+ and CD8+ cells.
- epidermis contain: γ/δ T cells.



Acute inflammatory dermatoses.



Chronic Inflammatory Dermatoses.



Blistering (Bullous) Disorders.

1. Acute inflammatory dermatoses.

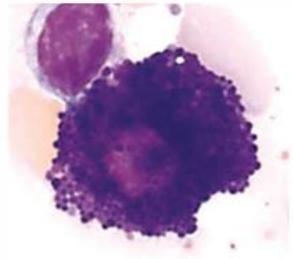
Acute lesions, defined as:

- days to several weeks in duration.

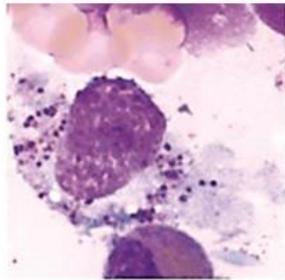
- characterized by inflammation, edema, and sometimes epidermal, vascular, or subcutaneous injury.

- Marked by infiltrates consisting of mononuclear cells rather than neutrophils, (unlike acute inflammatory disorders at most other sites).

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



Resting mast cell



Activated mast cell

A. Urticaria.

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

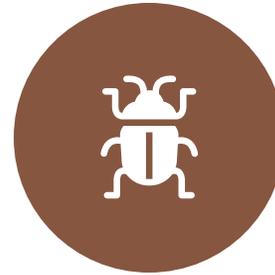
The resulting erythematous, edematous, and pruritic plaques are termed wheels.



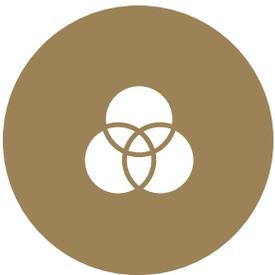
Pathogenesis



**IgE-independent urticaria



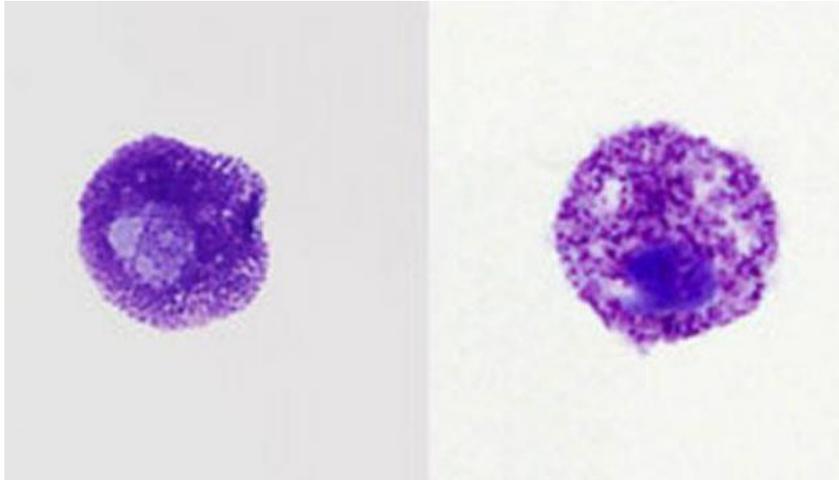
Responsible antigens include viruses, pollens, foods, drugs, and insect venom.



**IgE-dependent urticaria



pathogenesis Result from exposure to substances that directly incite mast cell degranulation, such as opiates and certain antibiotics.

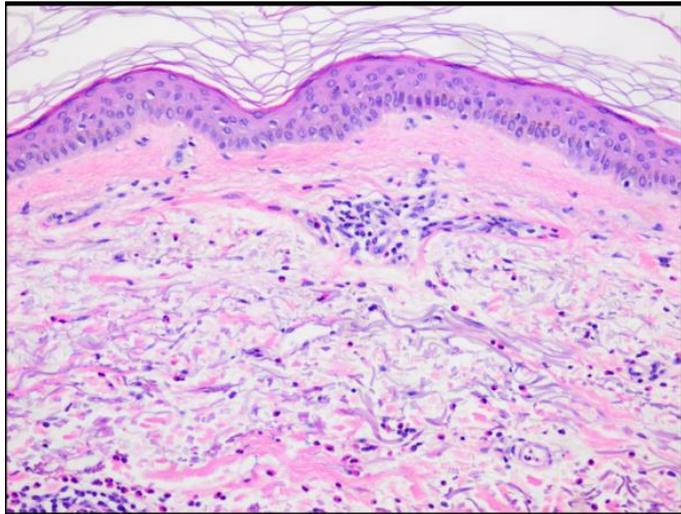


Histologic features of urticaria

*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.



Clinical Features

Typically affects individuals between 20 and 40 years of age.

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

Lesions range in size and nature from small, pruritic papules to large, edematous, erythematous plaques.

Treatment:

- Antihistamines.
- leukotriene antagonists.
- monoclonal antibodies that block the action of IgE.
- immunosuppressive drugs

B. Acute Eczematous Dermatitis.

Eczema is a clinical term that embraces a number of conditions with varied underlying etiologies.

Clinically the patient may have:

- 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 hypersensitivity 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.

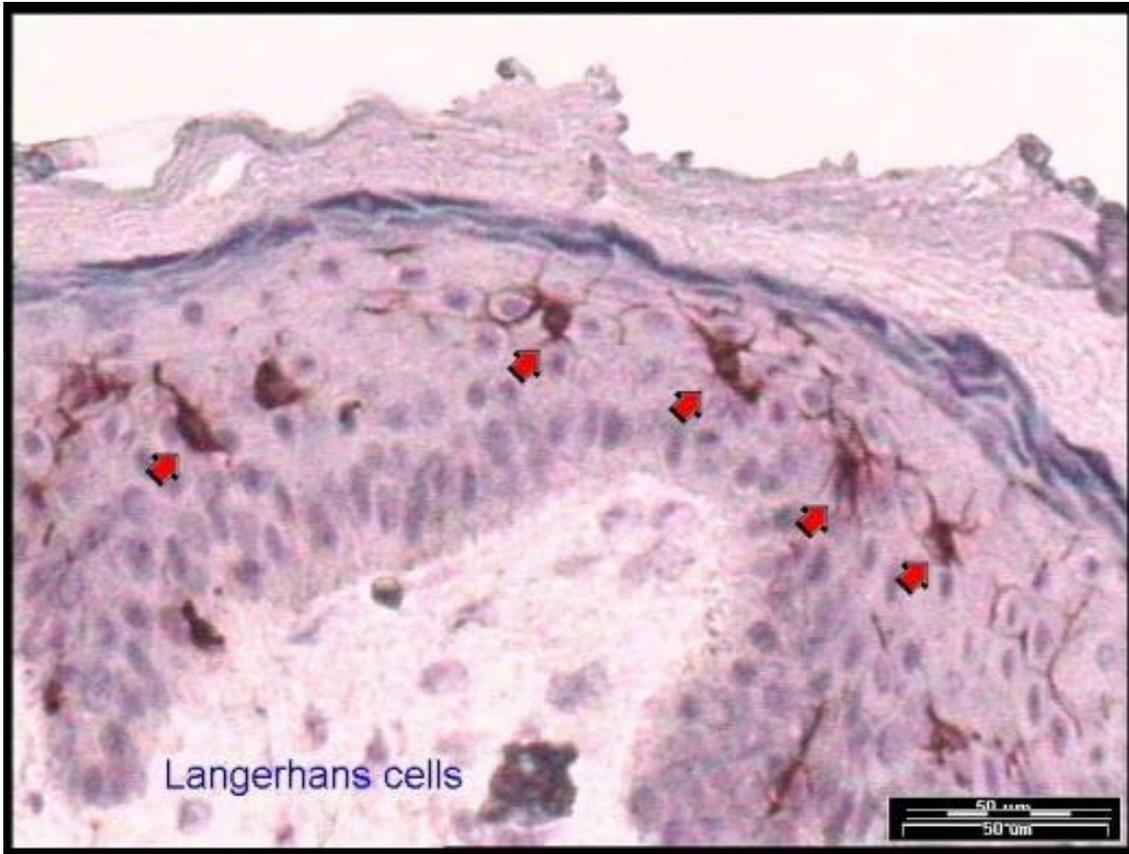
4. Photoeczematous dermatitis: appears as an abnormal reaction to UV or visible light

5. Primary irritant dermatitis: results from exposure to substances that chemically, physically, or mechanically damage the skin.



Allergic contact dermatitis

Allergic contact dermatitis is triggered by exposure to an environmental contact-sensitizing agent, such as poison ivy, that chemically reacts with self-proteins, creating neoantigens that can be recognized by the T cell arm of the adaptive immune system.



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.

Clinical Features

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

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

The cytotoxic T cell attack is focused on the basal cells of cutaneous and mucosal epithelia, presumably due to recognition of still unknown antigens.

Self-limited disorder that appears to be a hypersensitivity response to certain infections herpes simplex, mycoplasma, and some fungi.

- drugs. sulfonamides, penicillin, salicylates, hydantoins, and anti-malarials.

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

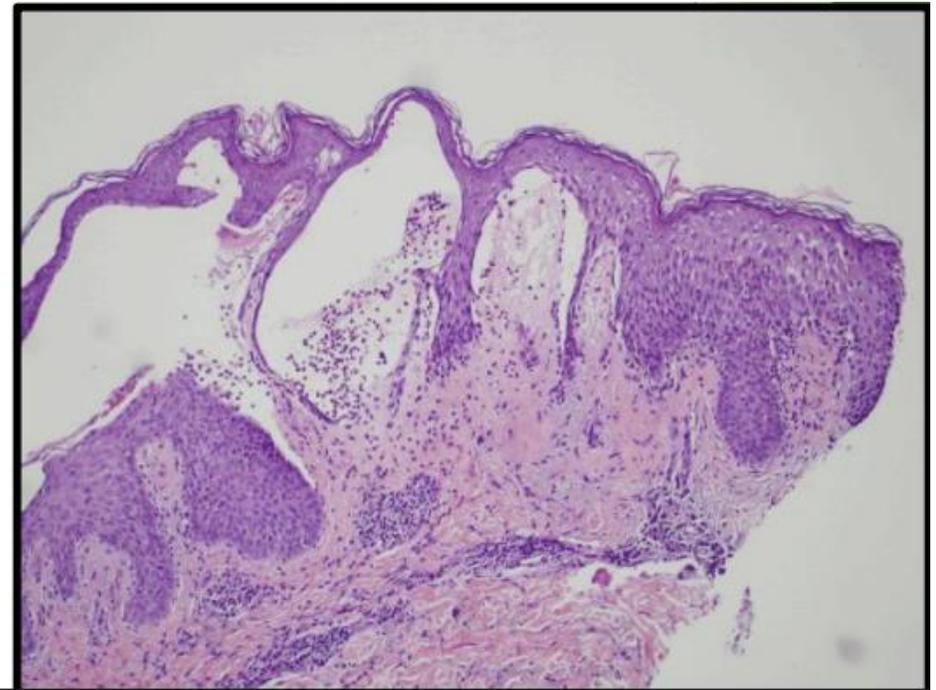
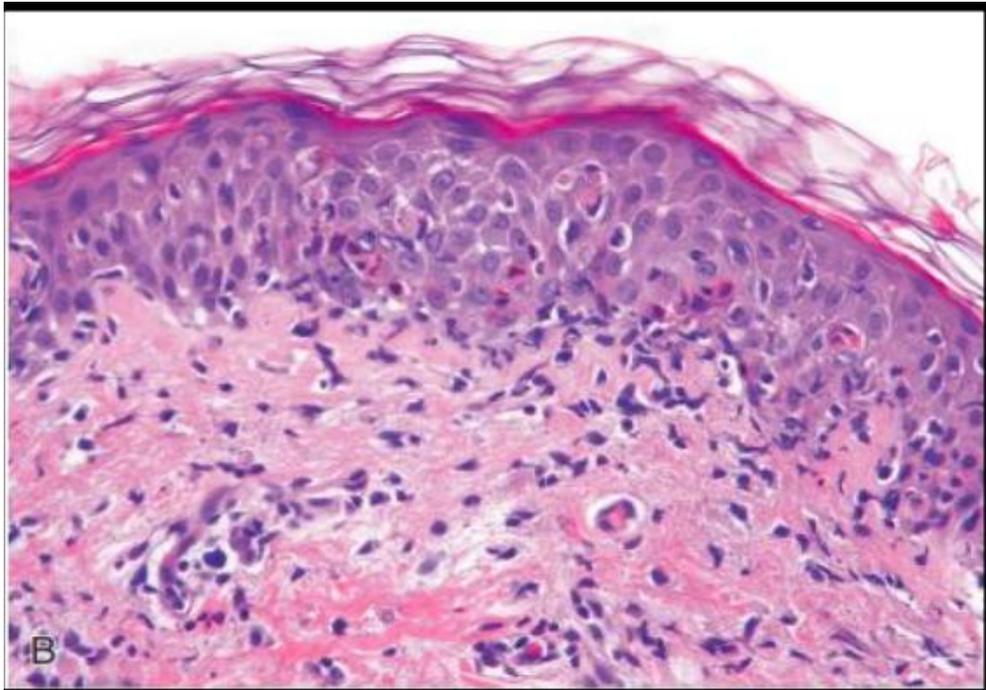


Early lesions show

- 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.

