MSS-1
Acute dermatosis

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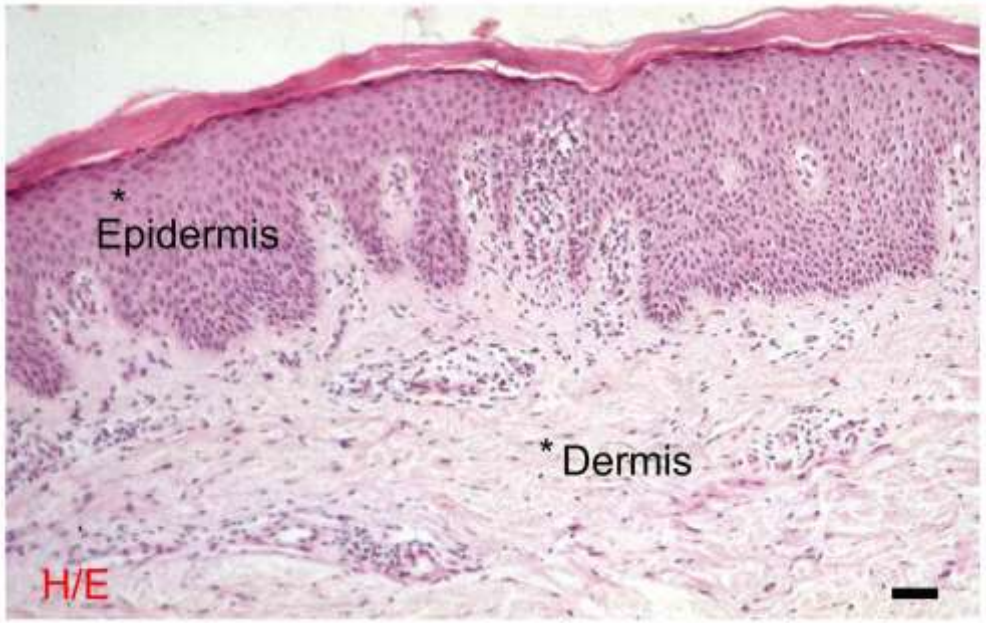
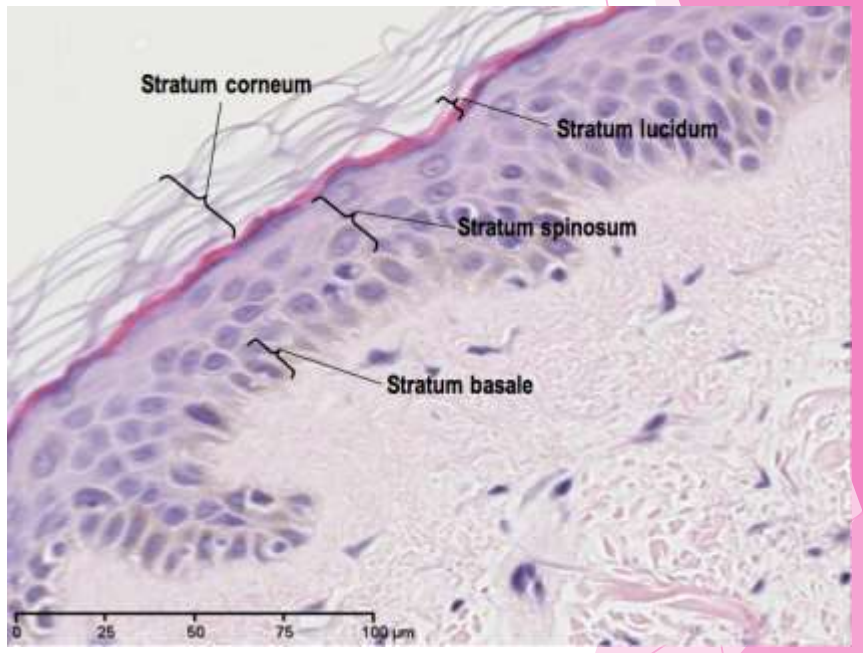
Histology


*
Epidermis
* Dermis
H/E


Stratum corneum
Stratum lucidum
Stratum spinosum
Stratum basale
0
25
50
75
100 µm

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.

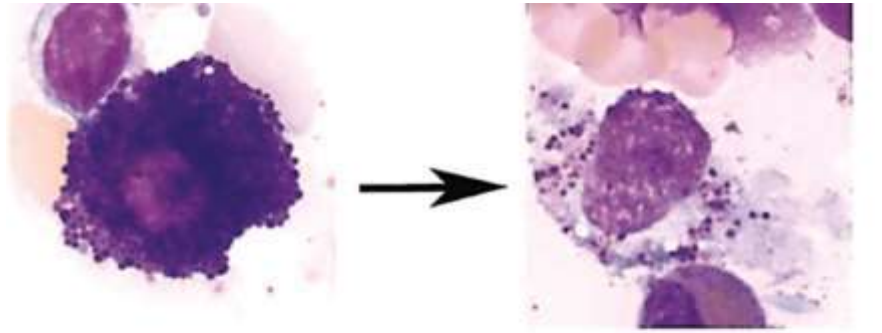
- Acute inflammatory dermatoses.
- Chronic Inflammatory Dermatoses.
- Blistering (Bullous) Disorders.

1. Acute inflammatory dermatoses.

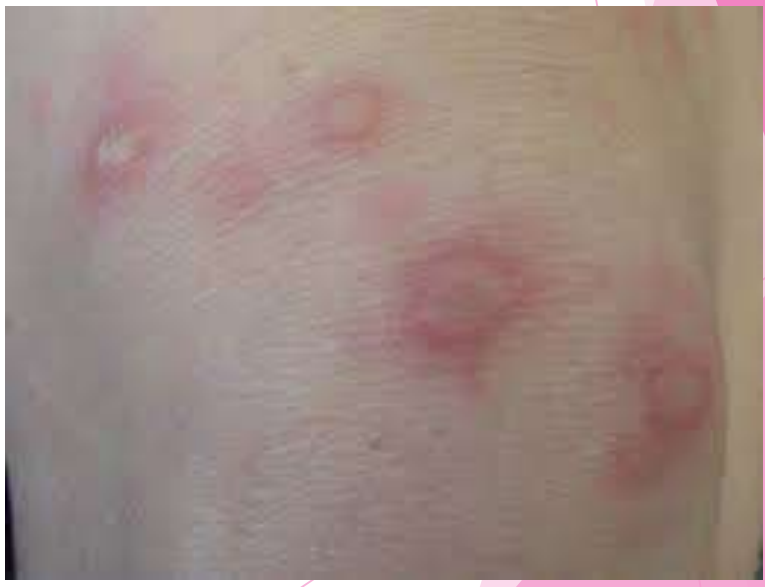
- Acute lesions, defined as:
- days to several weeks in duration.
- characterized by inflammation, edema, and sometimes epidermal, vascular, or subcutaneous injury.
- Marked by infiltrates consisting of mononuclear cells rather than neutrophils, (unlike acute inflammatory disorders at most other sites).
- Some acute lesions may persist, transitioning to a chronic phase, while others are self-limited

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



pathogenesis

**IgE-dependent urticaria

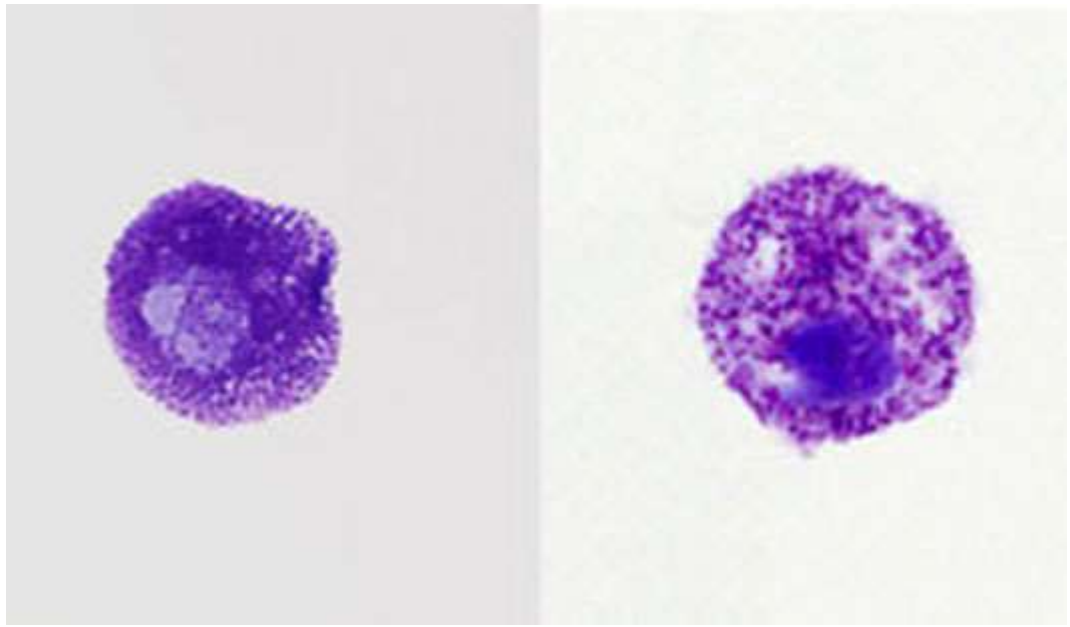
Responsible antigens
include viruses, pollens, foods, drugs, and insect venom.

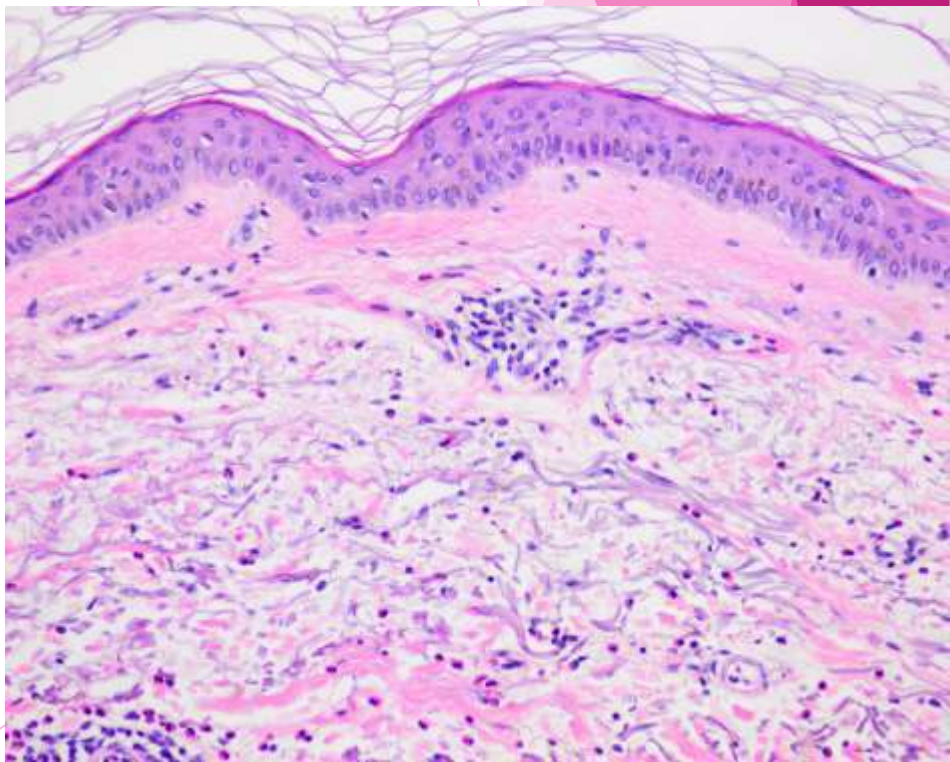
**IgE-independent urticaria

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.



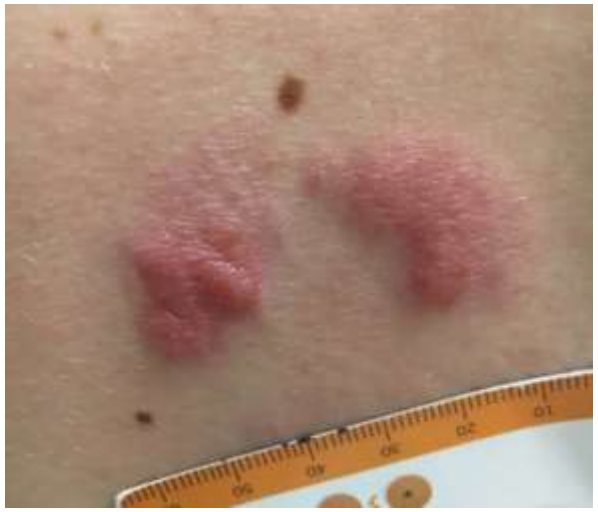


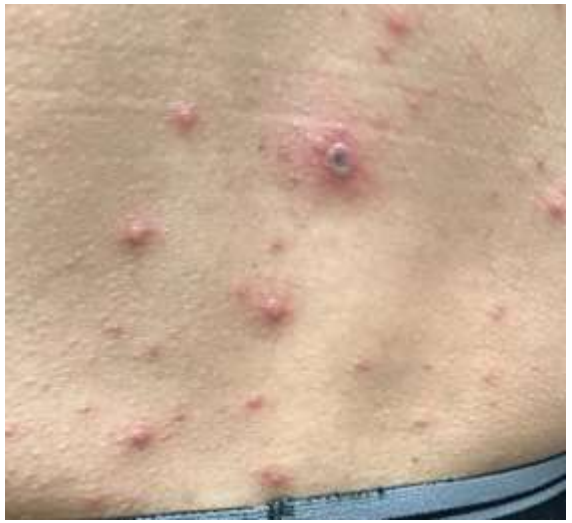
Clinical Features

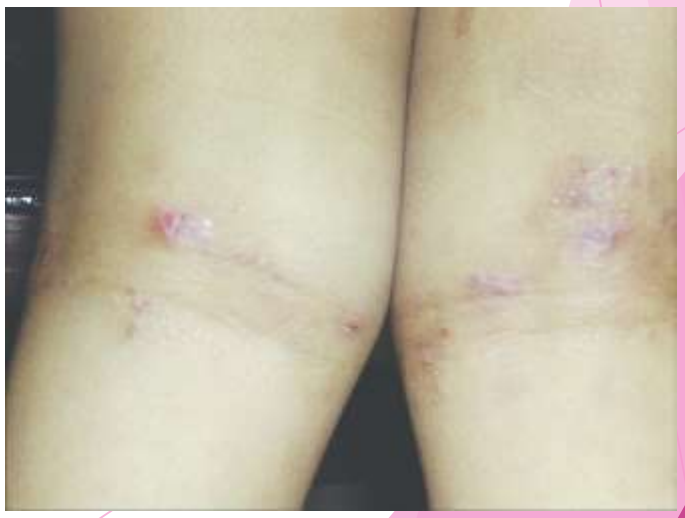
- Typically affects individuals between 20 and 40 years of age.
- Individual lesions usually develop and fade within hours, but episodes can persist for days or even months.
- Lesions range in size and nature from small, pruritic papules to large, edematous, erythematous plaques.
- Treatment:
- Antihistamines.
- 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 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.

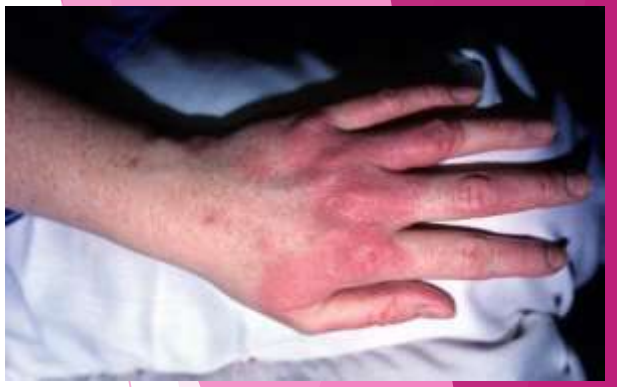
- 2. Atopic dermatitis:

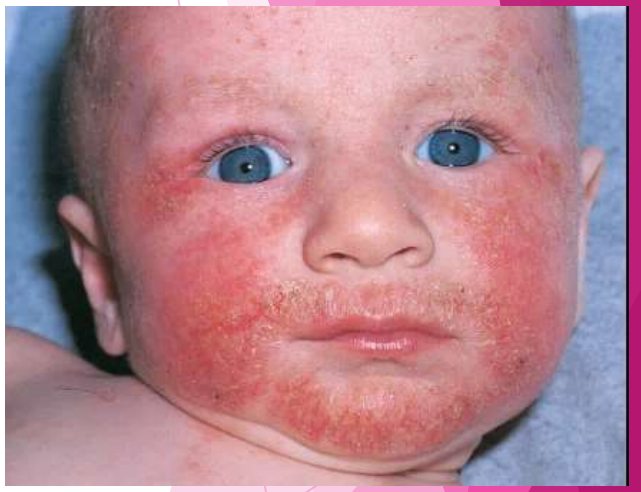
stem from defects in keratinocyte barrier function, defined as skin with increased

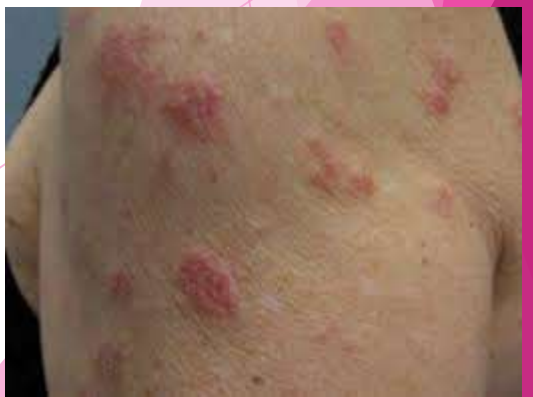
permeability to substances to which it is exposed, such as potential antigens

- 3. Drug-related eczematous dermatitis:

Hypersensitivity reaction to a drug.





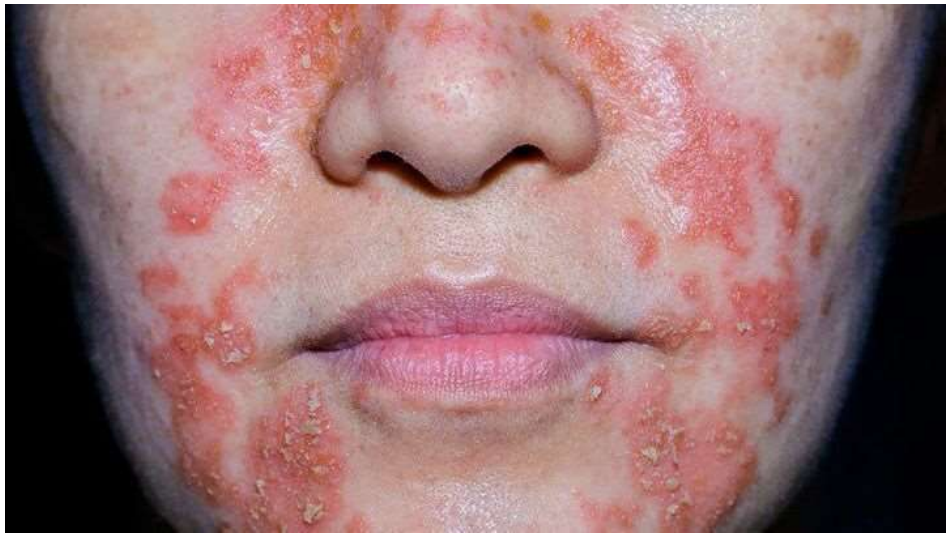


- 4. Photoeczematous dermatitis:

appears as an abnormal reaction to UV or visible light

- 5. Primary irritant dermatitis:

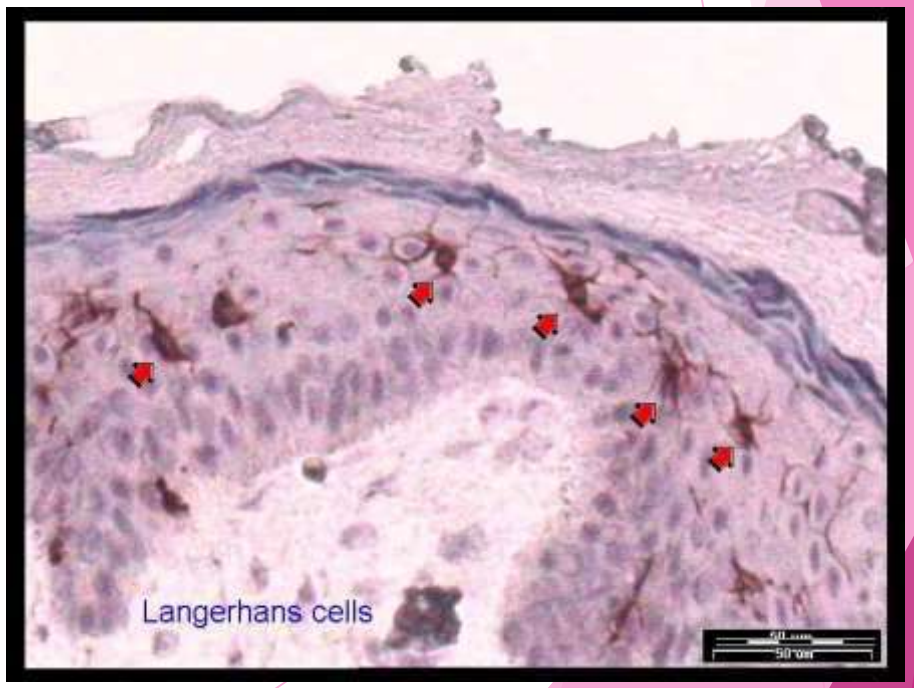
results from exposure to substances that chemically, physically, or mechanically damage the skin.



Allergic contact dermatitis

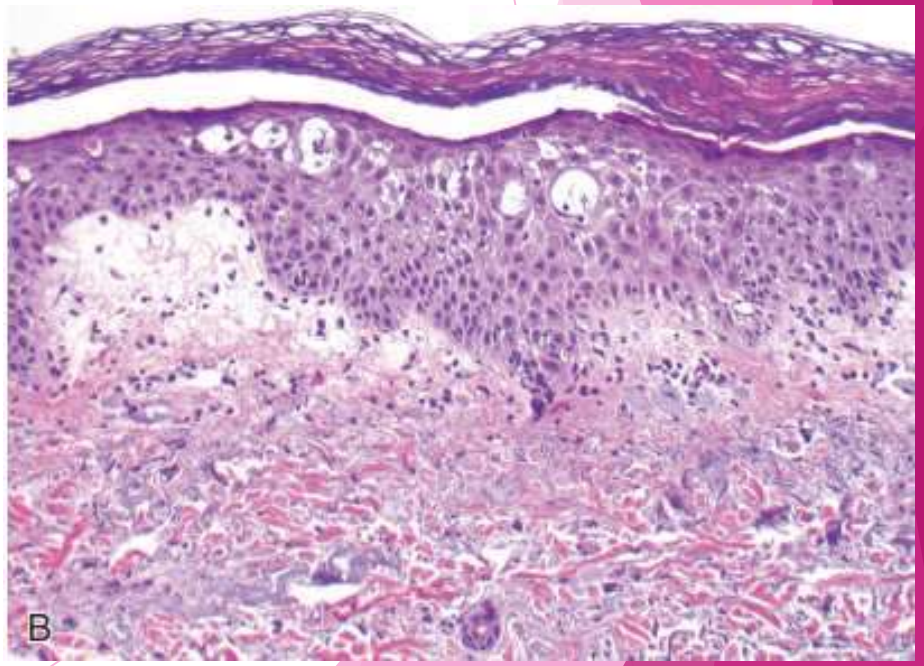
- Allergic contact dermatitis is triggered by exposure to an environmental contact-sensitizing agent, such as poison ivy, that chemically reacts with self-proteins, creating neoantigens that can be recognized by the T cell arm of the adaptive immune system.

- 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



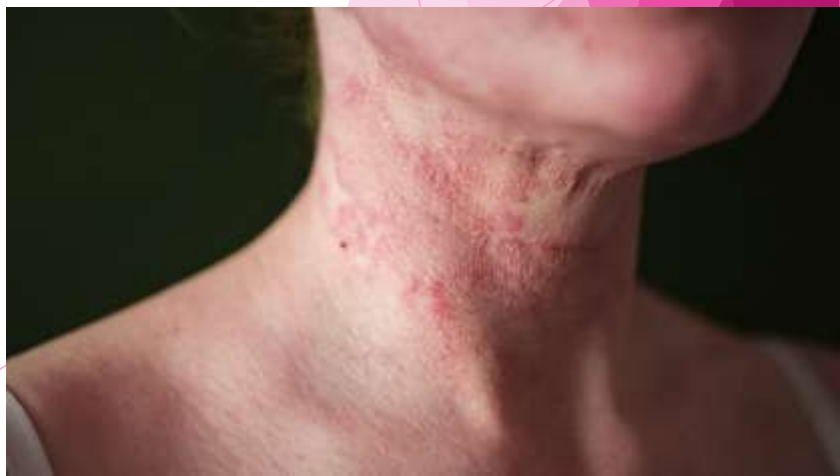
Histology

- 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.



C. Erythema Multiforme

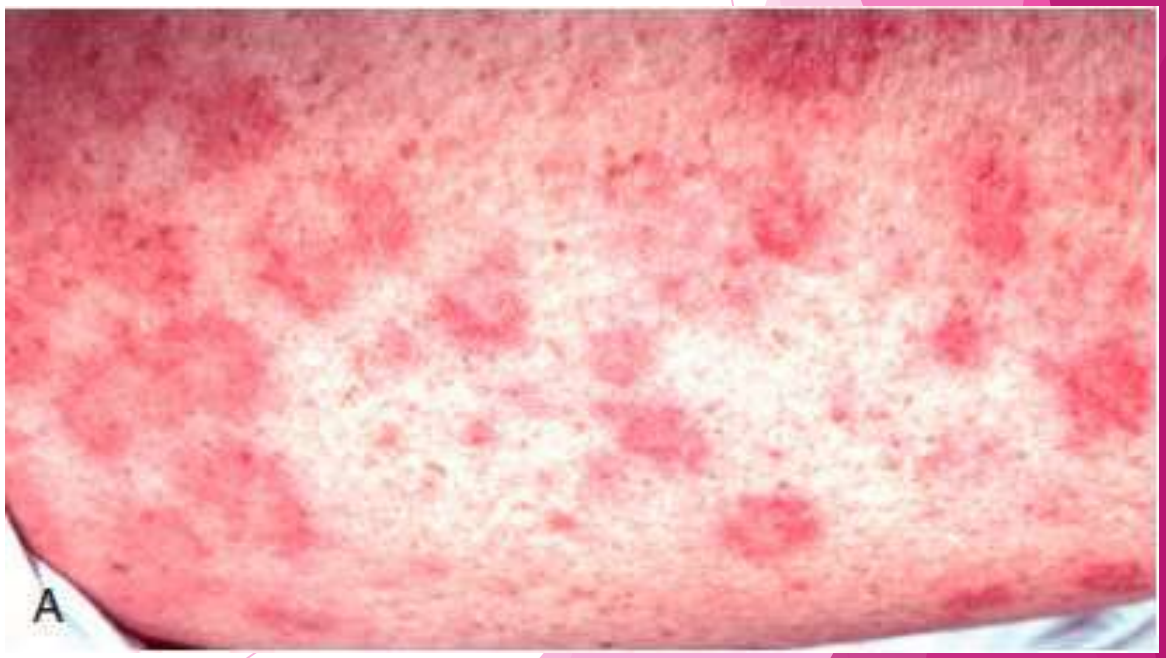
- Erythema multiforme is characterized by epithelial injury mediated by skin-homing CD8+ cytotoxic T lymphocytes.
- The cytotoxic T cell attack is focused on the basal cells of cutaneous and mucosal epithelia, presumably due to recognition of still unknown antigens

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

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

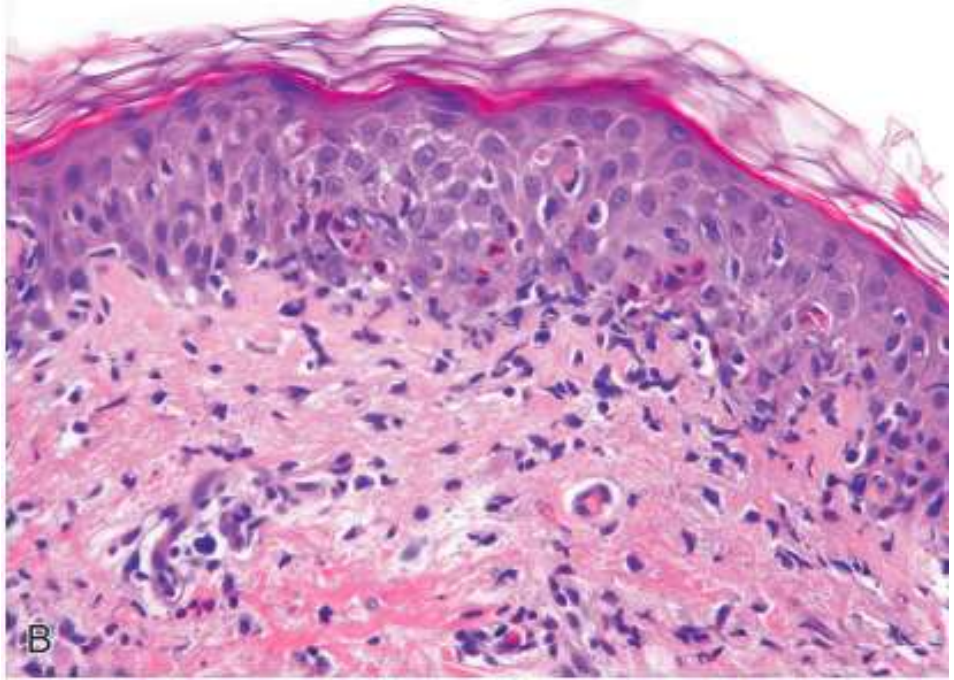
MORPHOLOGY

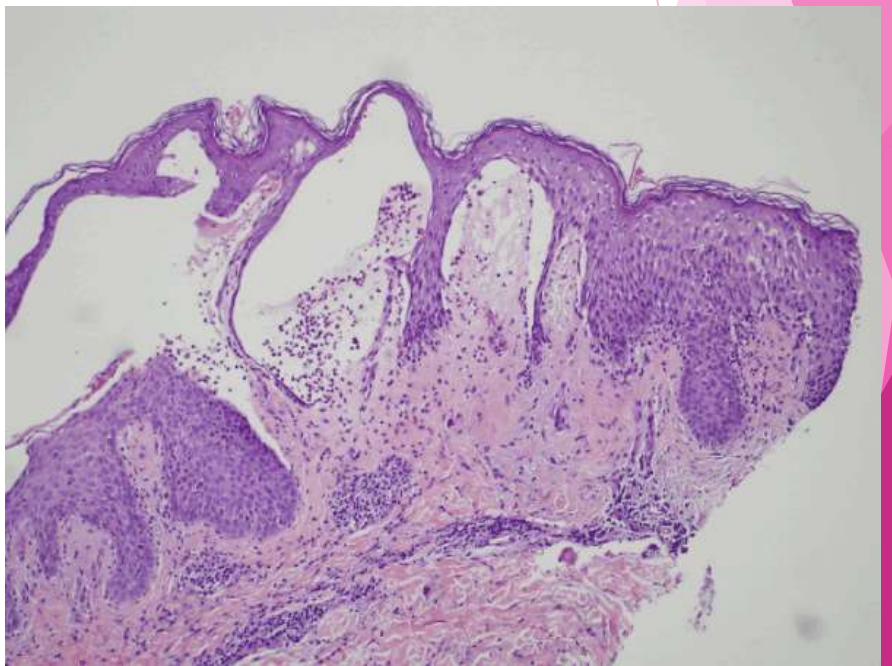
- Affected individuals present with a wide array of lesions, which may include macules, papules, vesicles, and bullae (hence the term multiforme)
- Well-developed lesions have a characteristic “targetoid” appearance



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

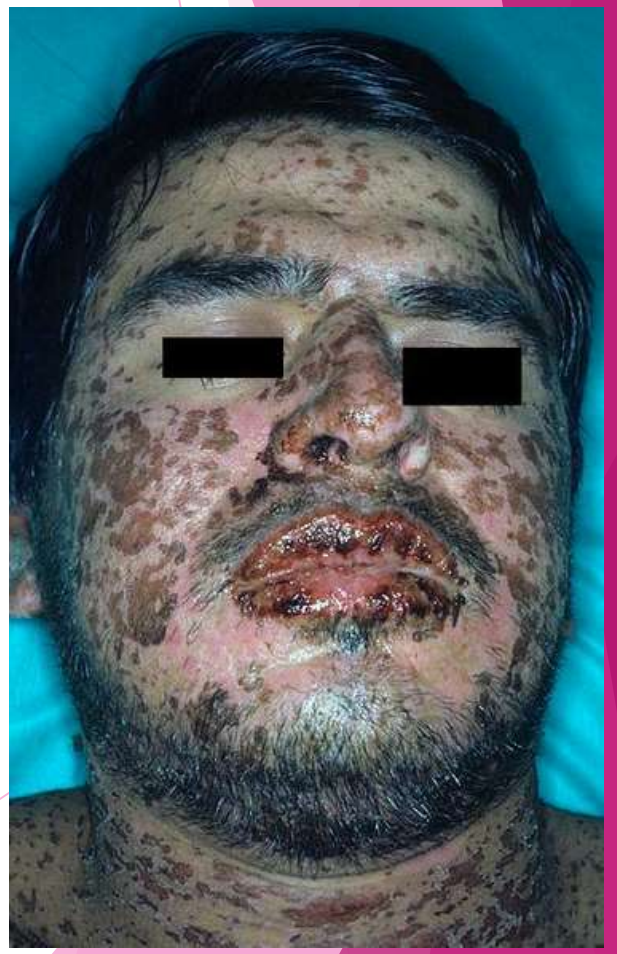
- With time
- discrete, confluent zones of basal epidermal necrosis appear, with concomitant blister formation.

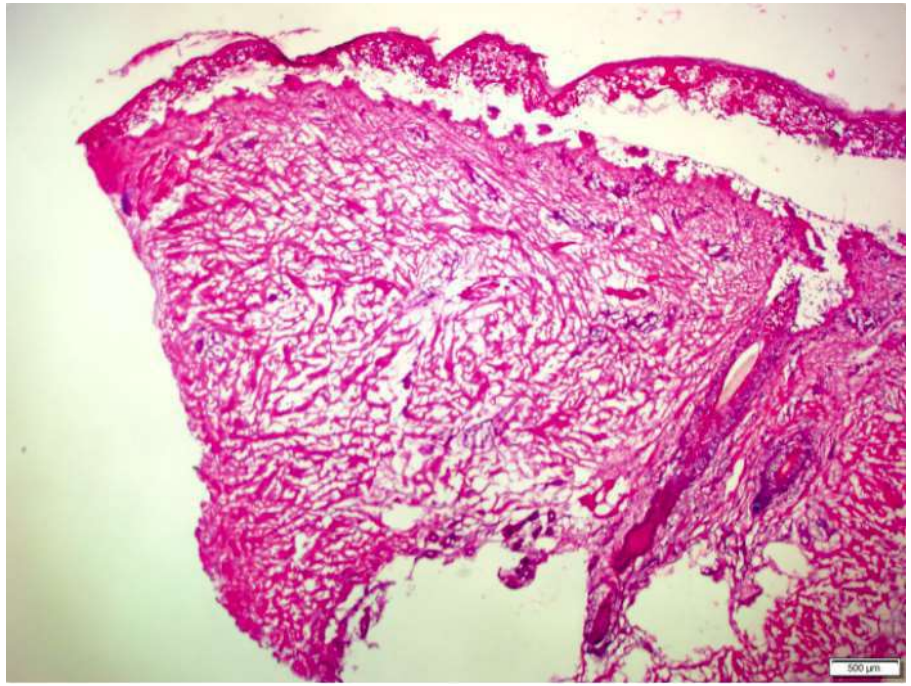


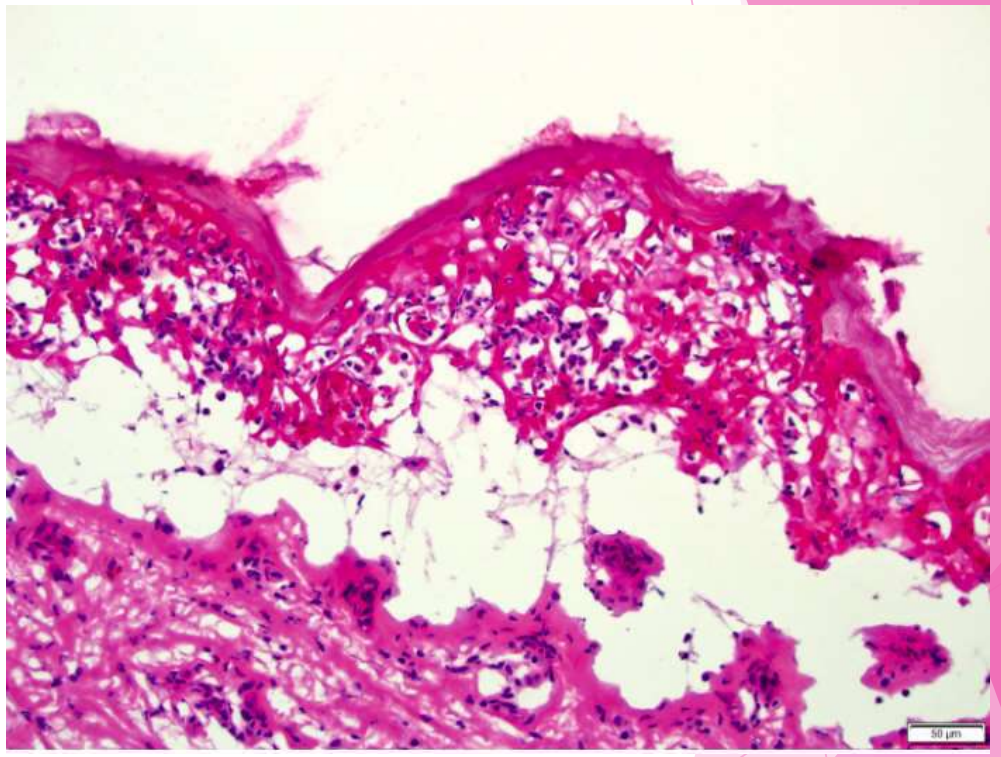


Clinical Features

- Erythema multiforme caused by medications may progress to more serious eruptions, such as
  - Stevens-Johnson syndrome: epidermal detachment < 10% body surface area
  - toxic epidermal necrolysis: epidermal detachment > 30% body surface area
- 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