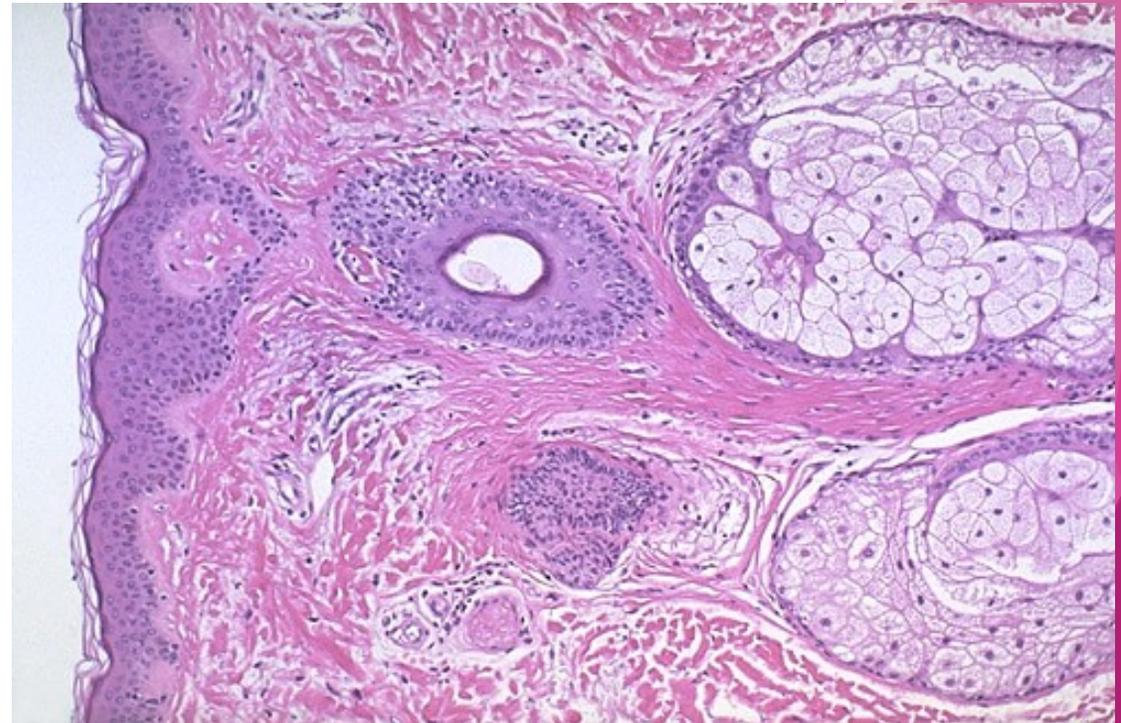
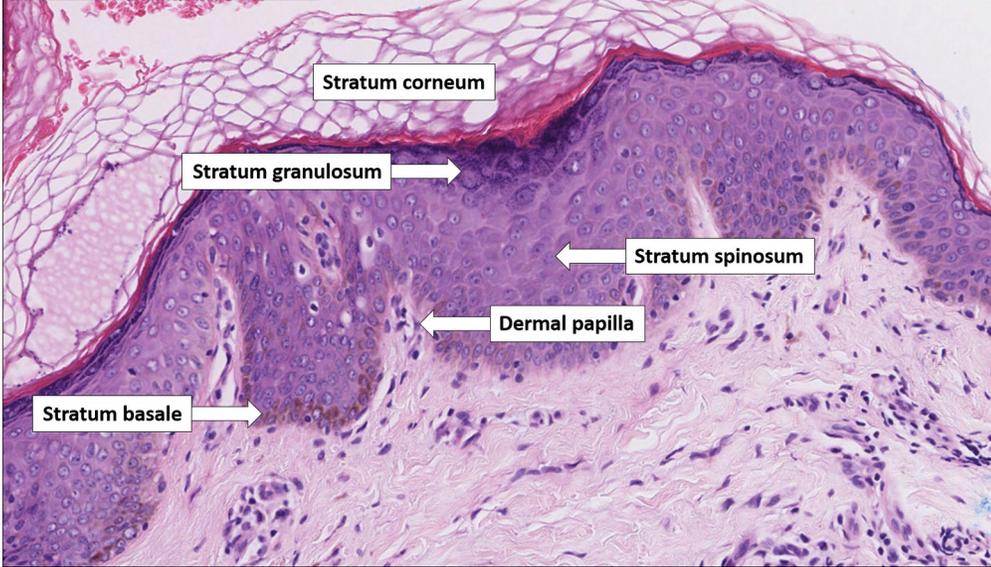


# Acute dermatosis

Dr.Eman Kreishan, M.D

Hair-bearing skin (high power):  
Layers of the epidermis.



# 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:  $\gamma/\delta$  T cells.

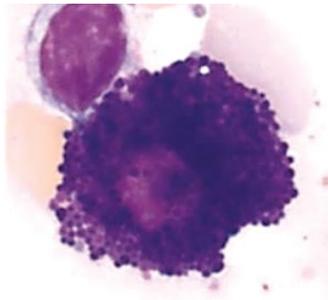
- ▶ Acute inflammatory dermatoses.
- ▶ Chronic Inflammatory Dermatoses.
- ▶ Blistering (Bullous) Disorders.

# 1. Acute inflammatory dermatoses.

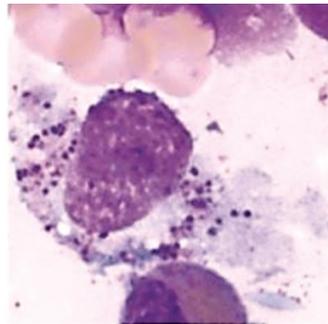
- ▶ Acute lesions, defined as:
  - ❑ days to several weeks in duration.
  - ❑ characterized by inflammation, edema, and sometimes epidermal, vascular, or subcutaneous injury.
  - ❑ Marked by infiltrates consisting of mononuclear cells rather than neutrophils, (unlike acute inflammatory disorders at most 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 wheals.



Resting mast cell



Activated mast cell



# pathogenesis

## **\*\*IgE-dependent urticaria**

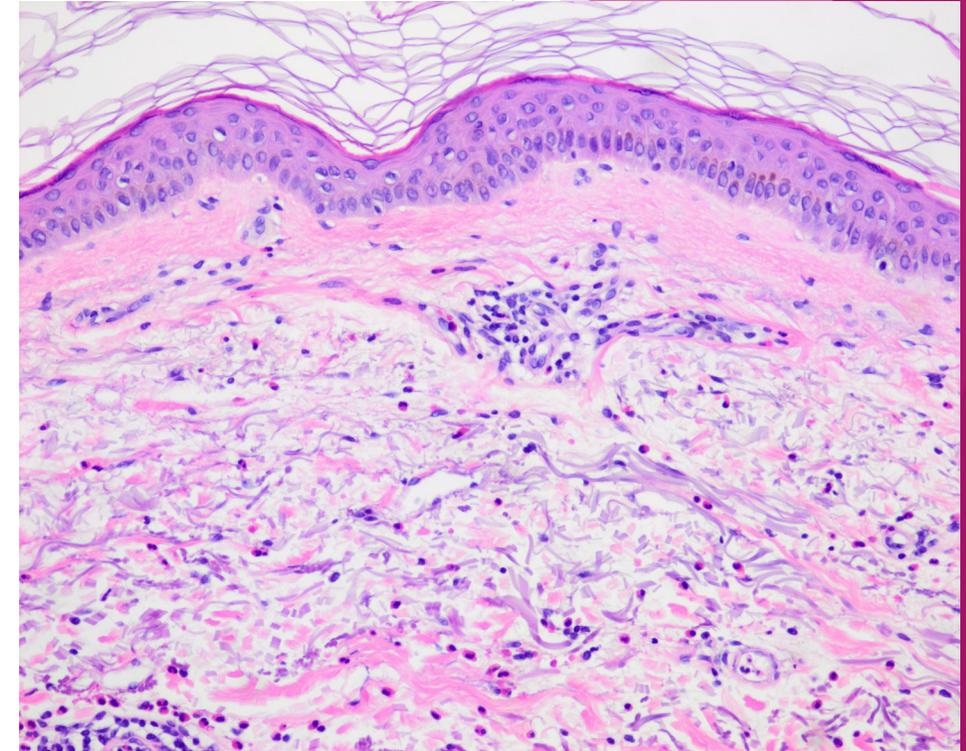
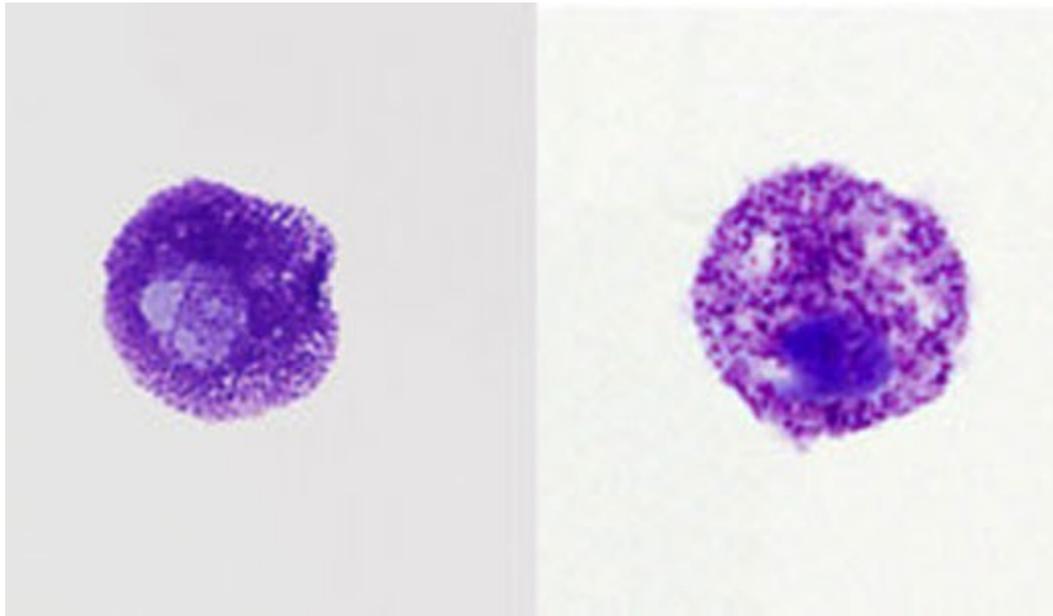
Responsible antigens include viruses, pollens, foods, drugs, and insect venom.

## **\*\*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 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.

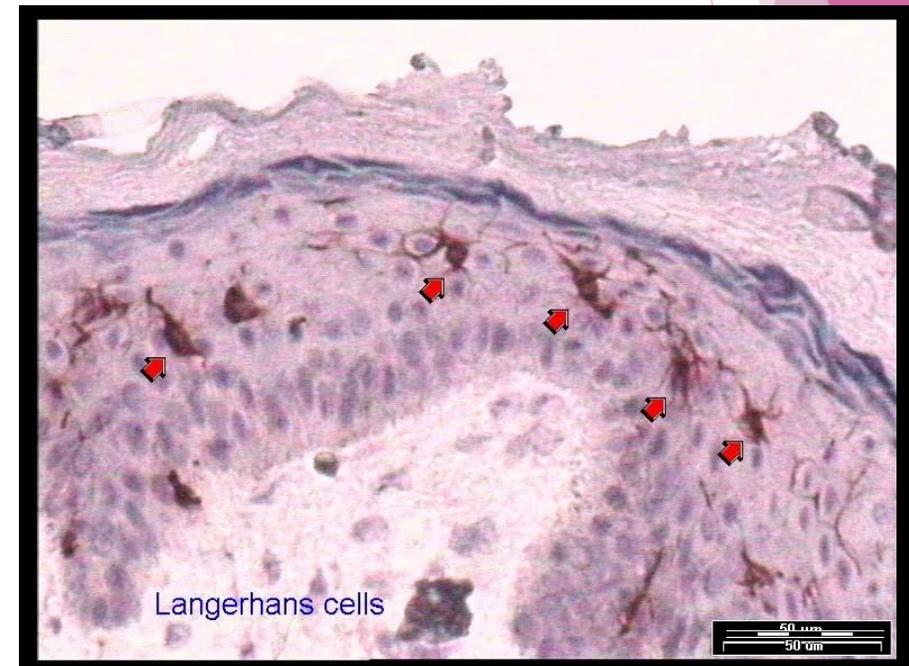


# Allergic contact dermatitis

- ▶ 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.

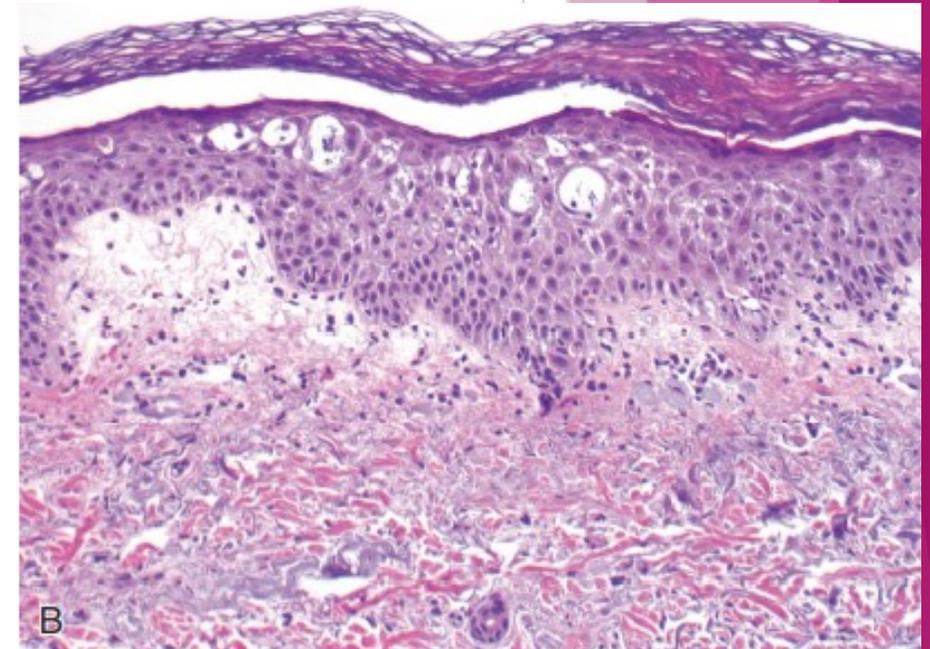


- ▶ The self-proteins modified by the agent are processed by epidermal Langerhans cells
- ▶ which migrate to draining lymph nodes and present the antigen to naïve T cells.
- ▶ This sensitization event leads to acquisition of immunologic memory
- ▶ on reexposure to the antigen, the activated memory CD4+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

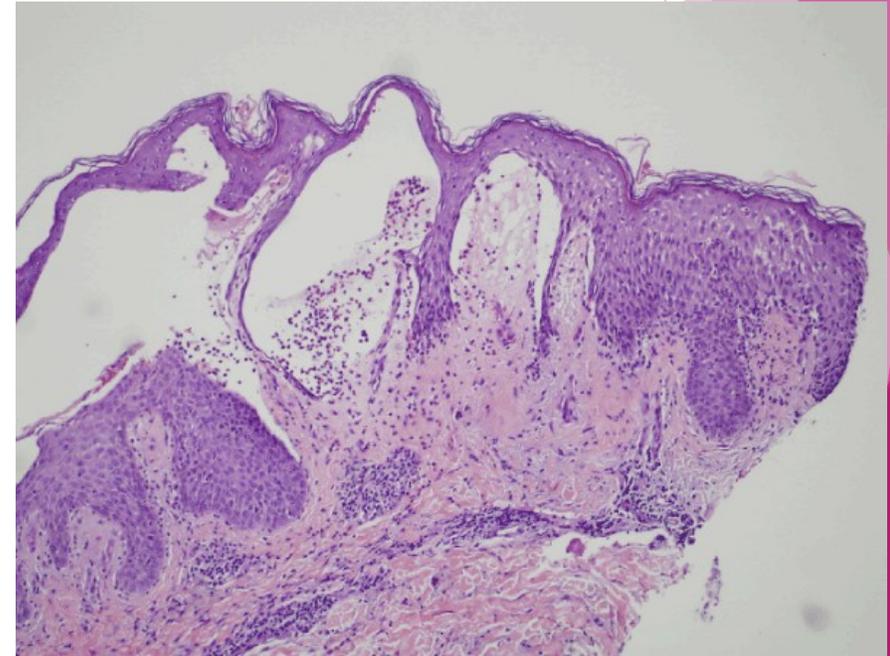
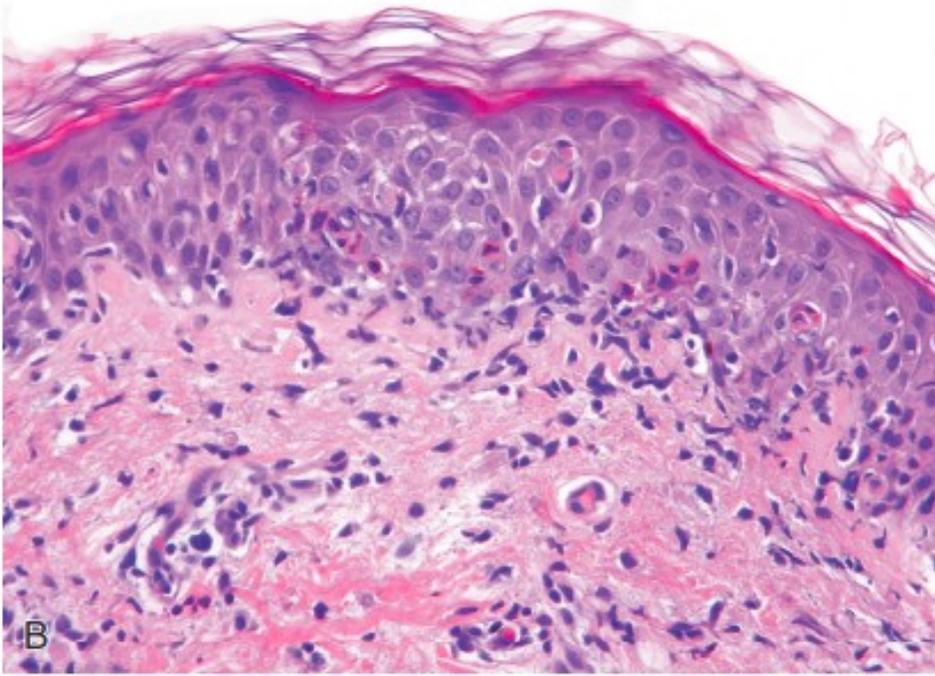
- ▶ Erythema multiforme is characterized by epithelial injury mediated by skin-homing CD8+ cytotoxic T lymphocytes.
- ▶ The cytotoxic T cell attack is focused on the basal cells of cutaneous and mucosal epithelia, presumably due to recognition of still unknown antigens
- ▶ self-limited disorder that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



