# Dermatopathology
# (Skin pathology )

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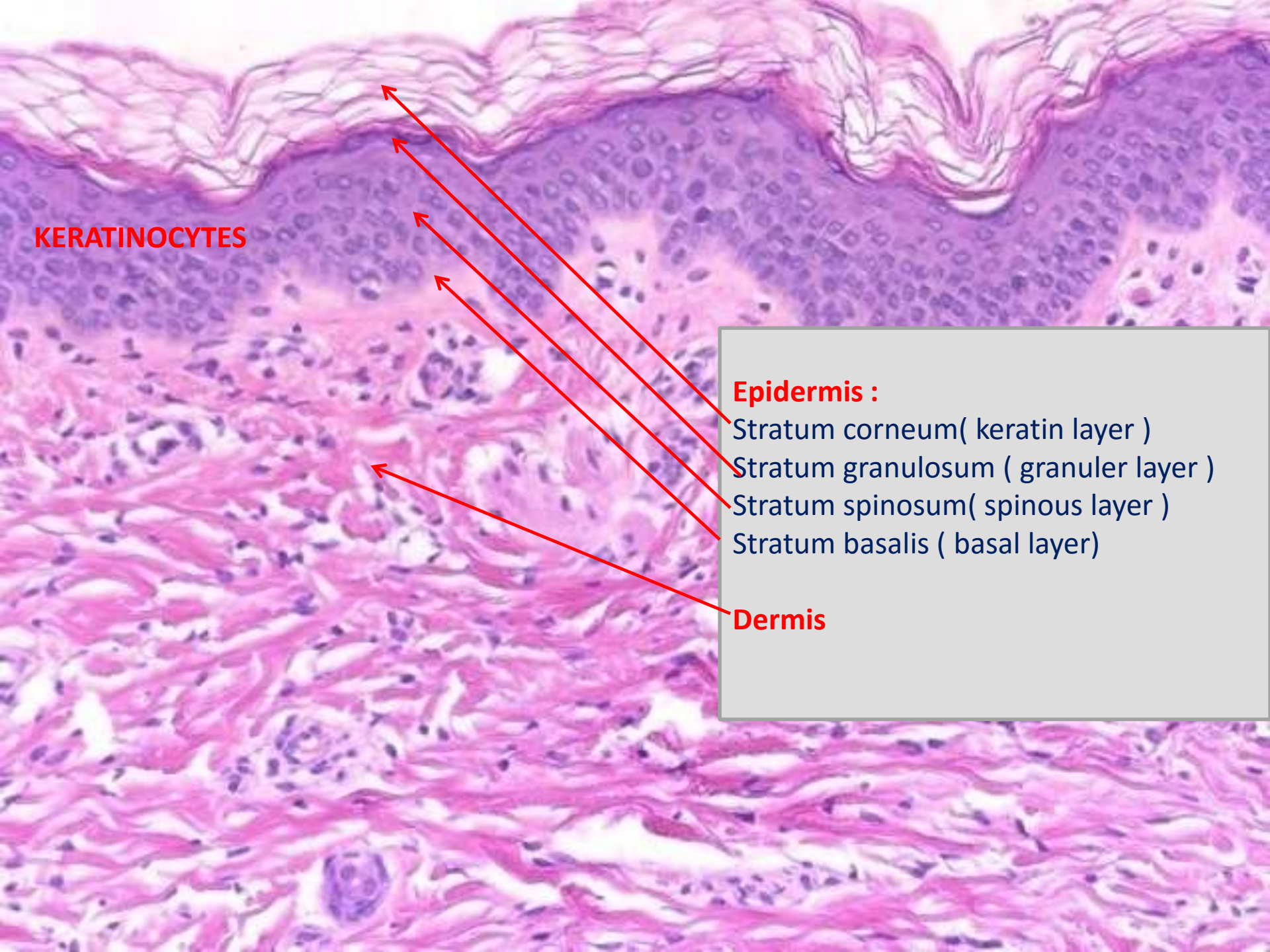
# Dermatopathology:

1. Acute Inflammations:
   - Urticaria,
   - Acute Eczema,

2. Chronic Inflammations:
   - Psoriasis,
   - Lichen planus.
3. Infections
   - Bacterial (Impetigo),
   - Fungal(tinea) &
   - Viral(warts).

1. Blistering Diseases
   - Pemphigus,
   - Pemphigoid,
   - Dermatitis herpetiformis.
5. Neoplastic:
- Benign:
  - Nevi,
  - Malignant:
  - BCC,
- - SCC,
  - Melanoma.


KERATINOCYTES
Epidermis :
Stratum corneum( keratin layer )
Stratum granulosum ( granuler layer )
Stratum spinosum( spinous layer )
Stratum basalis ( basal layer)
Dermis

- **The skin** composed of epidermis and dermis and subcutaneous fatty tissue (hypodermis, subcutis or pannus)

The epidermis is a stratified squamous keratinizing epithelium composed of several layers of keratinocytes

1-basal layer (stratum basale)of proliferative cells.

2-spiny layer (stratum spinosum)Prickle cell layer of polygonal cells

3-granular cell layer(Stratum granulosum) of flattened cells rich in dark granules(keratohyaline granules).

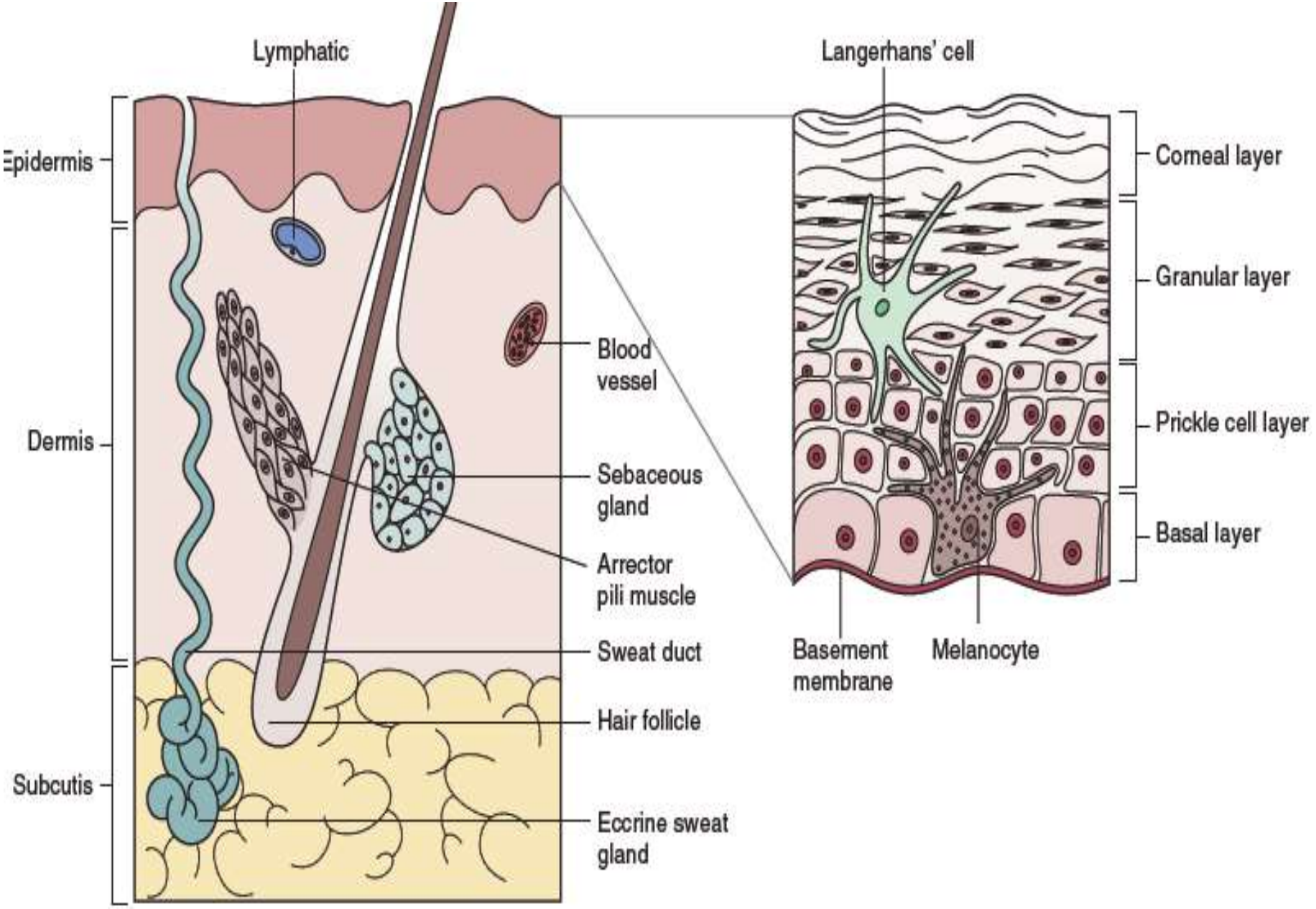
4-Corneal layer: Stratum corneum (horny layer)

of differentiated keratinocytes(dead cell without nuclei, The top layer of cells loosens and falls off).

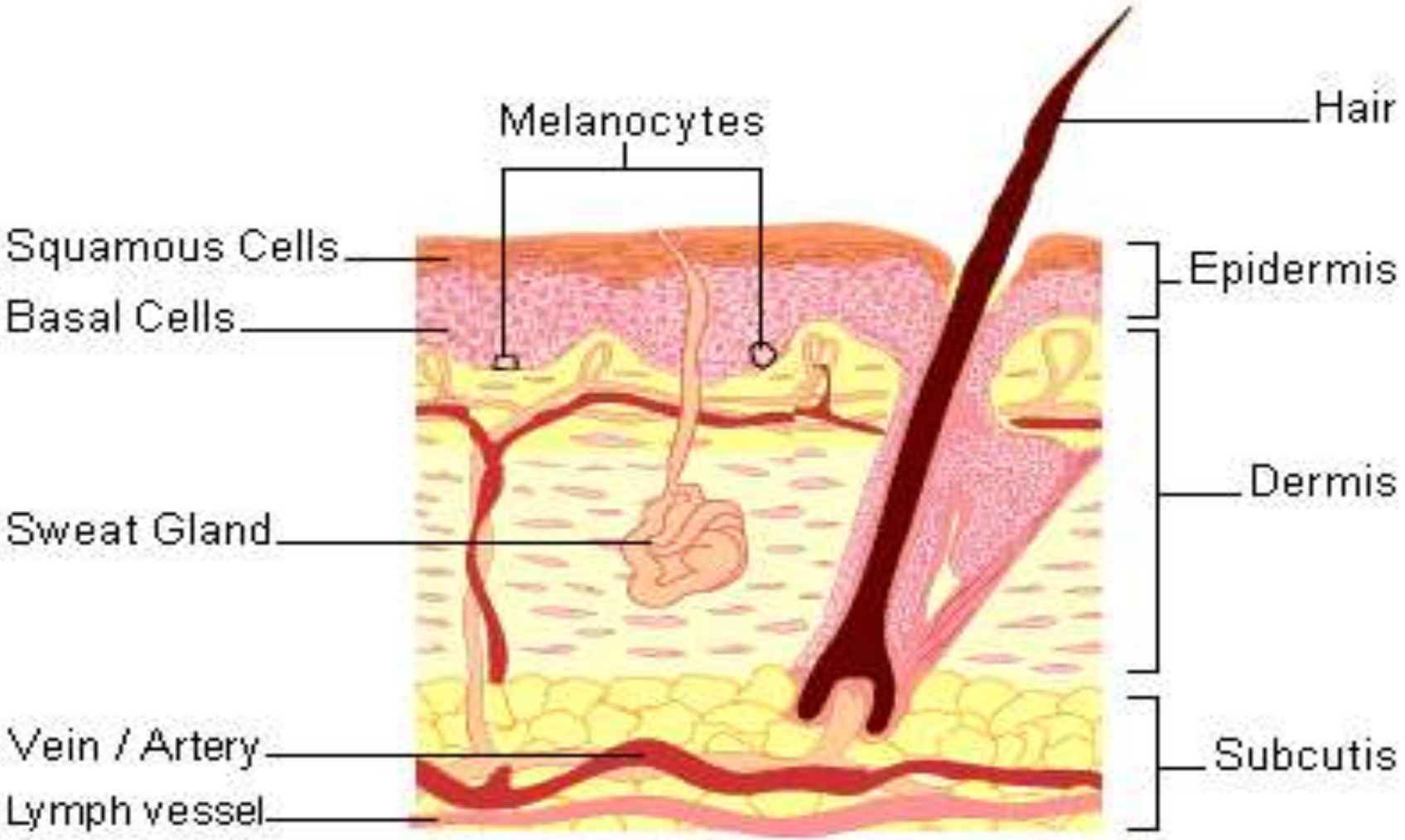
The dermis is composed of dermal connective tissue composed of collagen and elastic fibers. It has two distinctive areas

1- papillary dermis rich in small nerves and capillaries.

2-reticular dermis rich in skin appendages ( sweat glands, pilosebaceous unit).


Lymphatic
Epidermis
Dermis
Subcutis
Blood vessel
Sebaceous gland
Arrector pili muscle
Sweat duct
Hair follicle
Eccrine sweat gland
Langerhans' cell
Corneal layer
Granular layer
Prickle cell layer
Basal layer
Basement membrane
Melanocyte

# ANATOMY OF THE SKIN



# Terms user in Dermatopathology

**MACROSCOPIC TERMS**

**MICROSCOPIC TERMS**

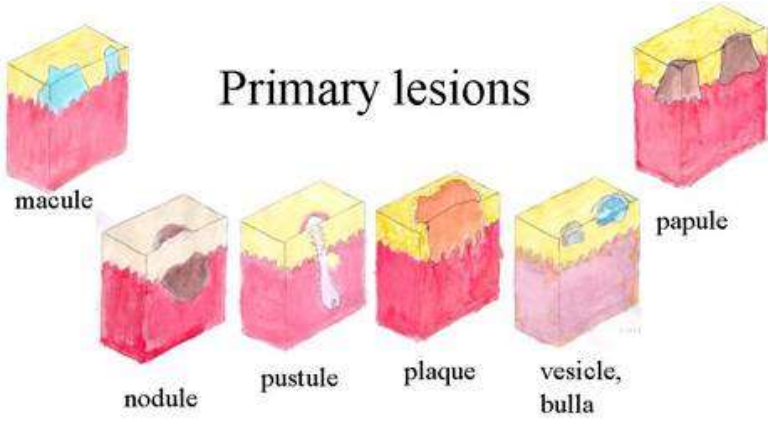
# MACROSCOPIC TERMS

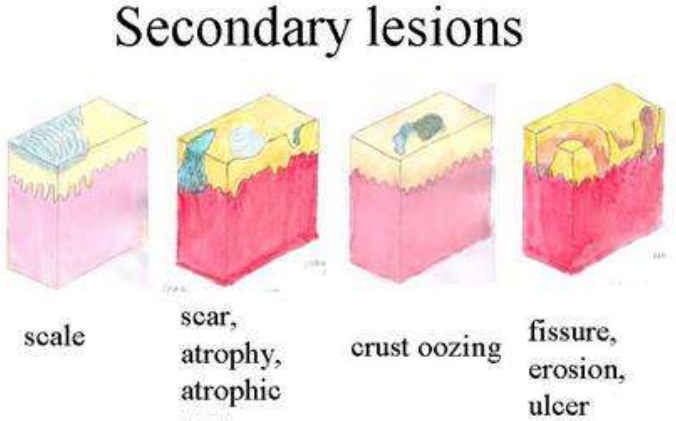
**Primary lesions- The original lesions**

- Flat :Macule, patch
- Elevated: Papule, plaque, nodule,
- Fluid filled(blister): Vesicle , Bullae
- Wheals: pruritic edematous plaque
- pus filled: Pustule

**Secondary Lesions: the primary lesions continue to full development or may be modified by regression, trauma or other factors like scratching or rubbing**

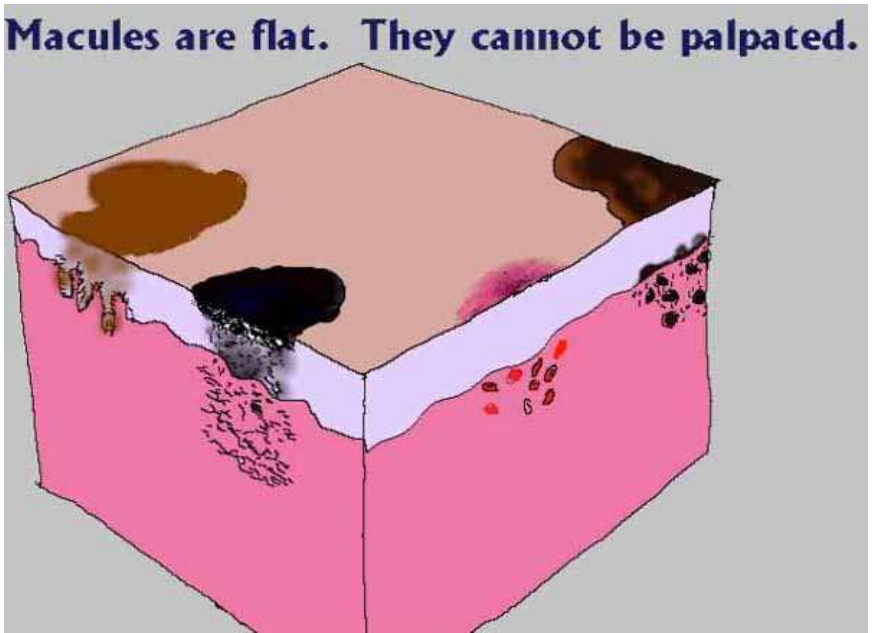
- Scales
- Crusts
- Erosions , Ulcers
- Scar-hypertrophic scars
- Keloid
- Atrophy



# Macroscopic Terms(clinical )

- Macule: A  flat ,circumscribed change in skin color without elevation or depression.
- Macule := < 5 mm
- *Latin: macula, “spot”* بقعة




Patch: A flat ,circumscribed change in skin color without elevation or depression> 5 mm

- Clinical terms

# Flat lesion:no elevation no depression

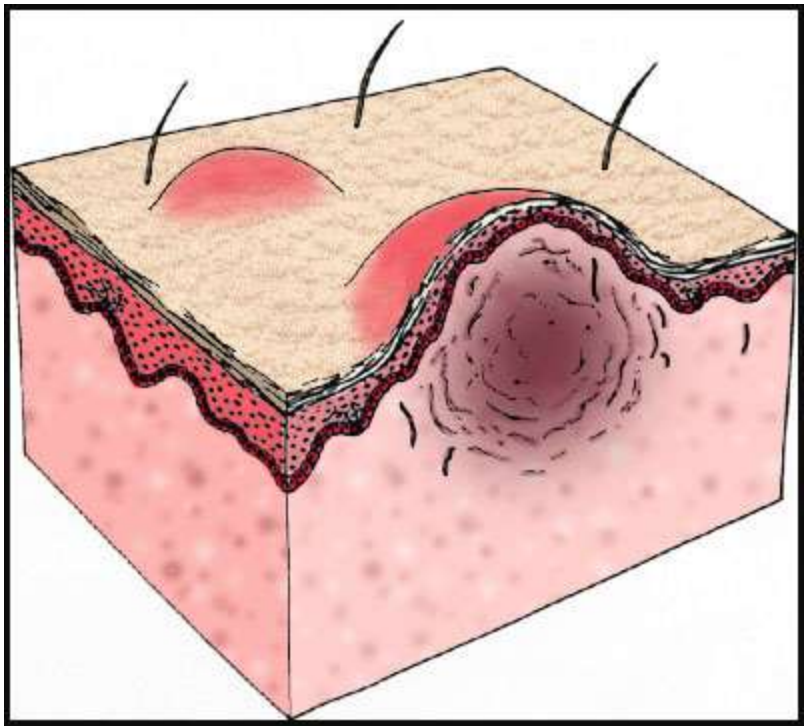
**Macule := < 5 mm**

**Patch > 5 mm**

- Flat, circumscribed area distinguished from surrounding skin by coloration


# Papule : A solid elevated lesion usually 5 mm or less in diameter.

- *Papule- (Latin Paula, “Pimple”)* بثرة



**Nodule:** elevated , solid lesion greater than 5 mm in diameter

*(Latins: nodulus- "small knot")-عقدة*

❑Clinical terms

Elevated solid area

= < 5 mm

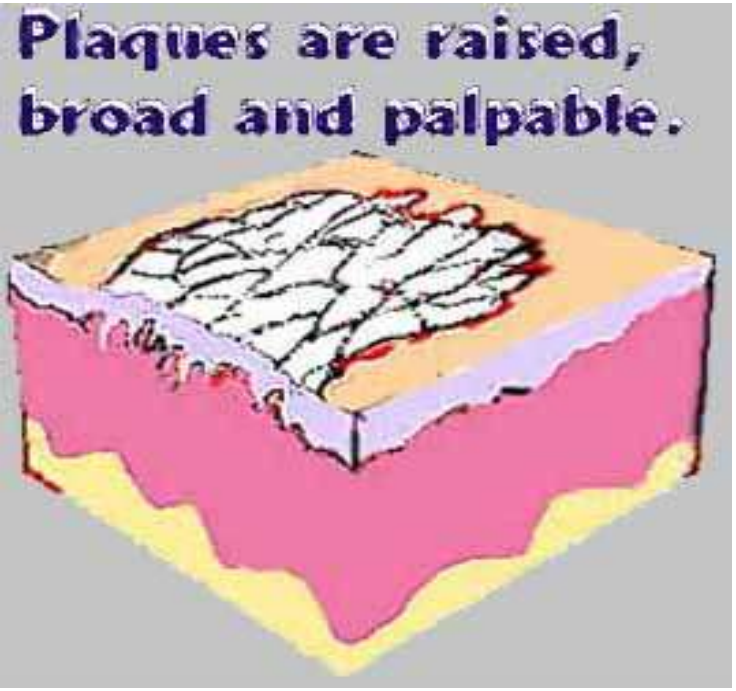
papule

> 5 mm

nodule

# Plaque : elevated flat topped lesion that has a greater than 5 mm across.

- *Plaques: (French- Plaque- "Plate")*




❑Clinical terms

# Blister

## Fluid-filled raised area

### Vesicle

- = < 5 mm

e.g. Herpes

### Bulla

- > 5 mm

**Vesicle**: elevated fluid-filled lesion 5 mm or less in diameter.

*(Latin "Little bladder")*

Bulla ( blister ) elevated fluid-filled lesion more than 5mm across.

*Bulloe (Latin-"Bubble")-*

Wheals are oedematous, flat elevations of various sizes. Associated with itching or burning sensation

Pustule: small elevations of the skin containing pus

*Latin- Pustula-Pus*

- Secondary lesions

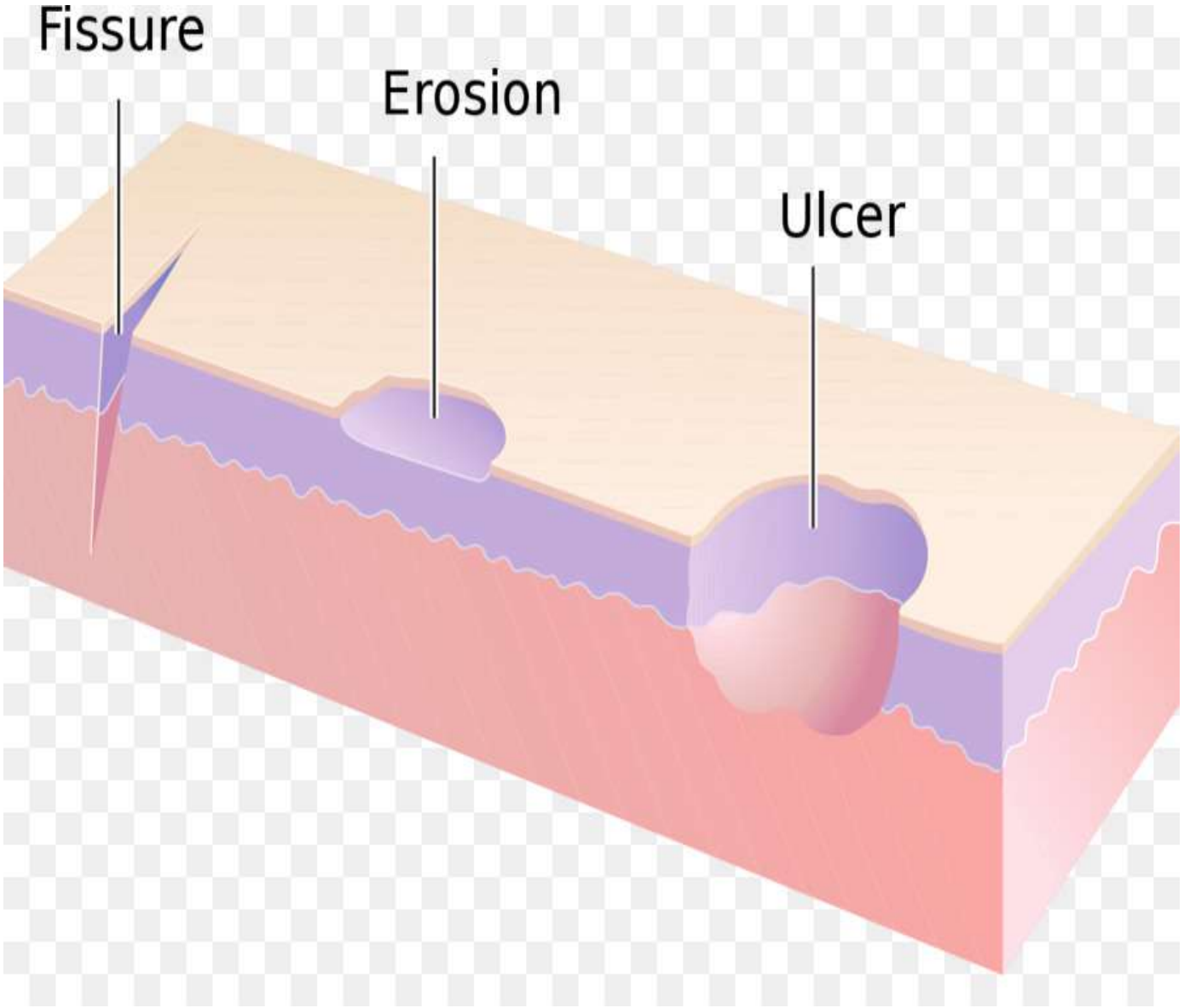
❑Clinical terms

# Scale

- Dry, horny, plate-like excrescence( due to excess dead epidermal cells produced by abnormal keratinization and shedding ).
- E.g psoriasis

# Crust

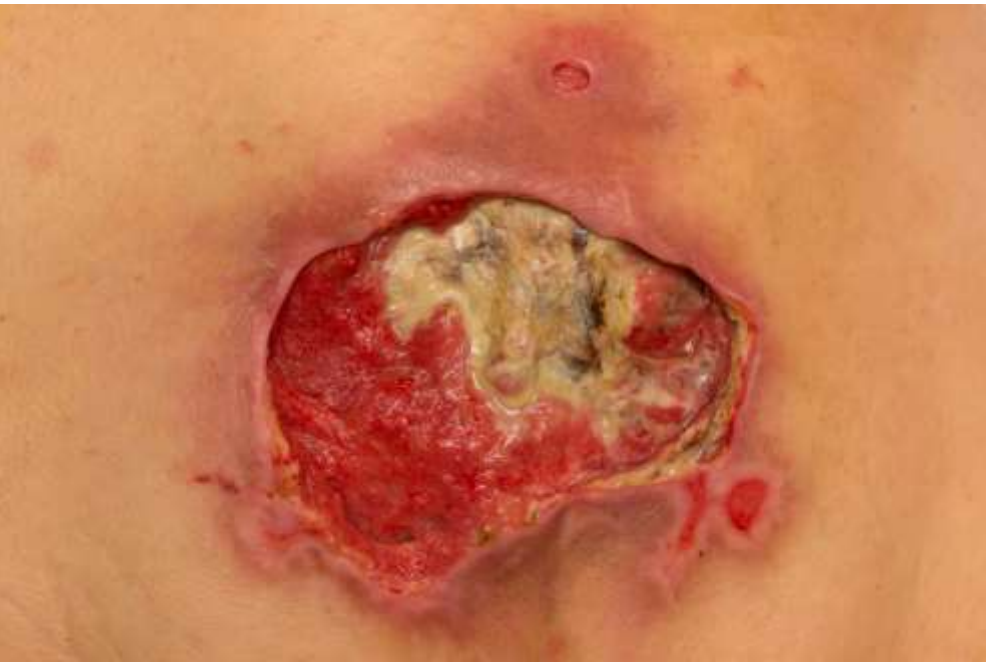
- A “scab” formed from dried serum , blood or exudate on skin usually mixed with epithalial
- and bacterial debris

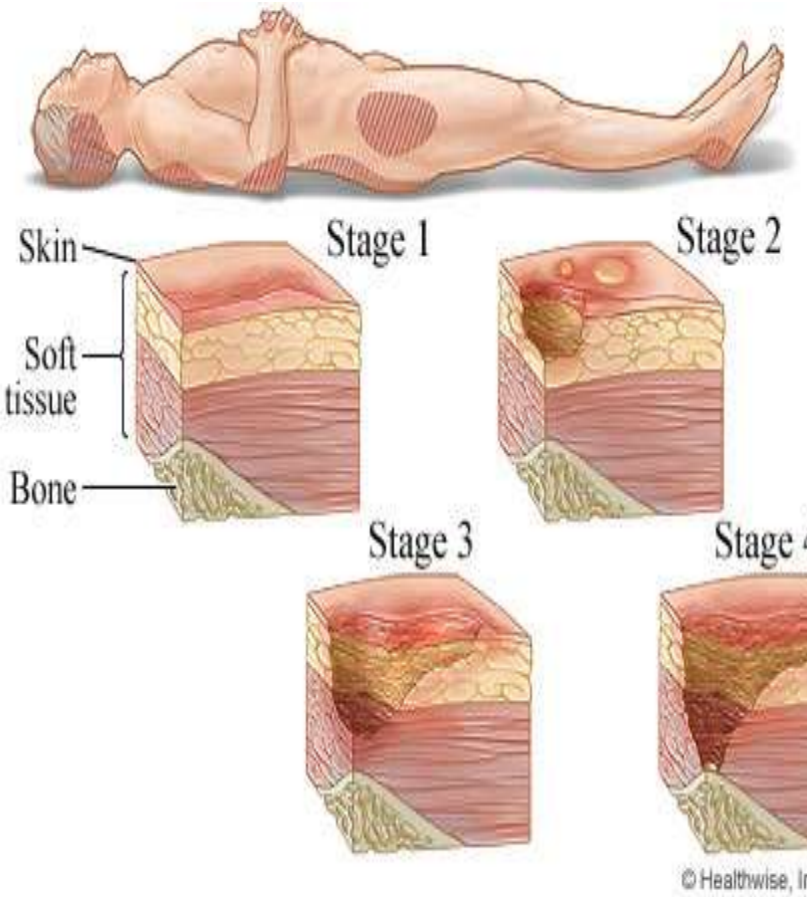

Fissure
Erosion
Ulcer

# Erosion

- Focal loss of epidermis not exceeding below dermo-epidermal junction heal without scar tissue formation (e.g following blister rupture

# **Ulcers** (Latin- Ulcus- “Sore”)- focal loss of epidermis and dermis extending into hypodermis(e.g bedsore ).





# pathologic terms
# (microscopical terms)

# MICROSCOPIC TERMS

Hyperkeratosis

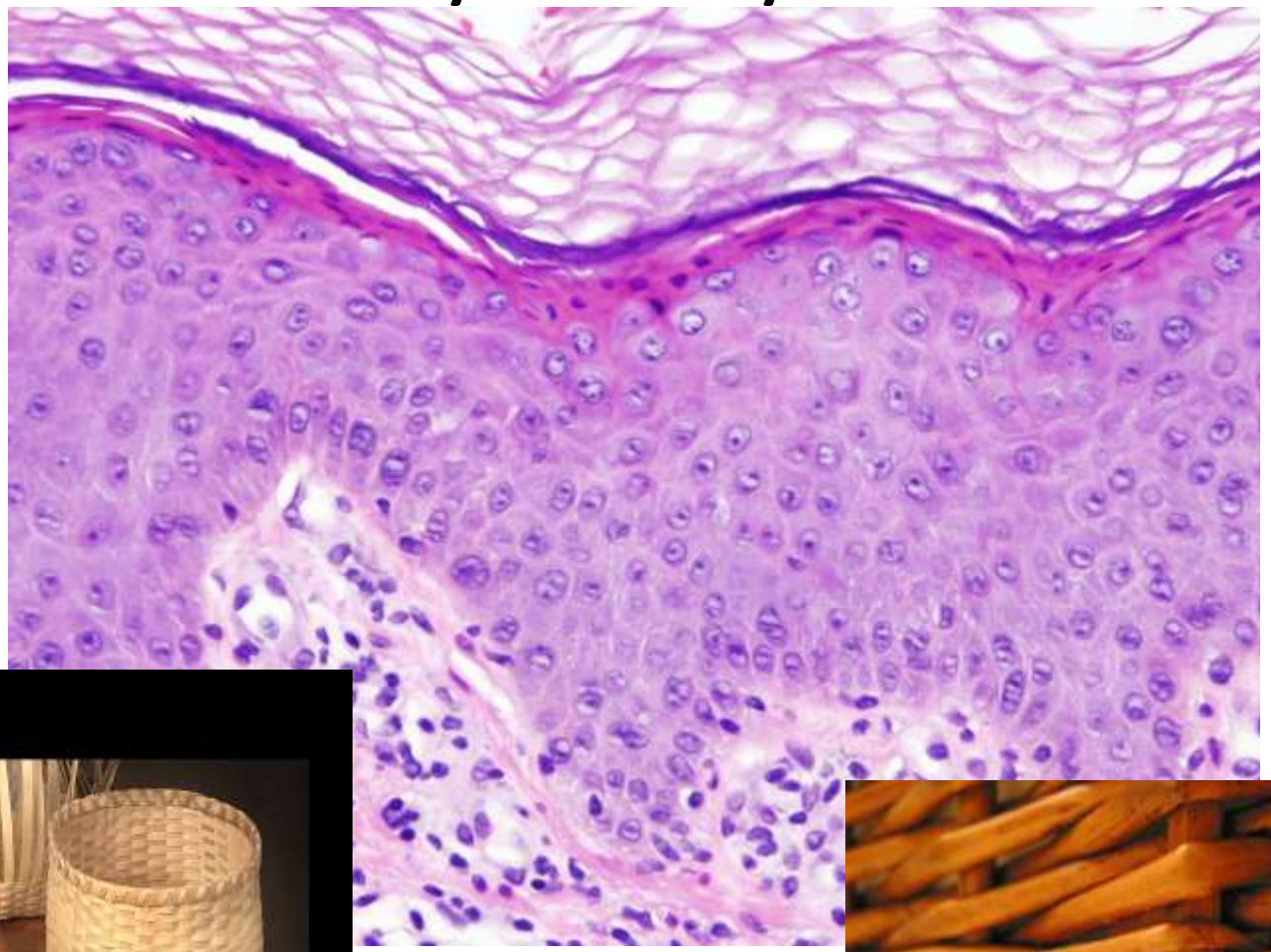
Parakeratosis

Acanthosis

Spongiosis

acantholysis

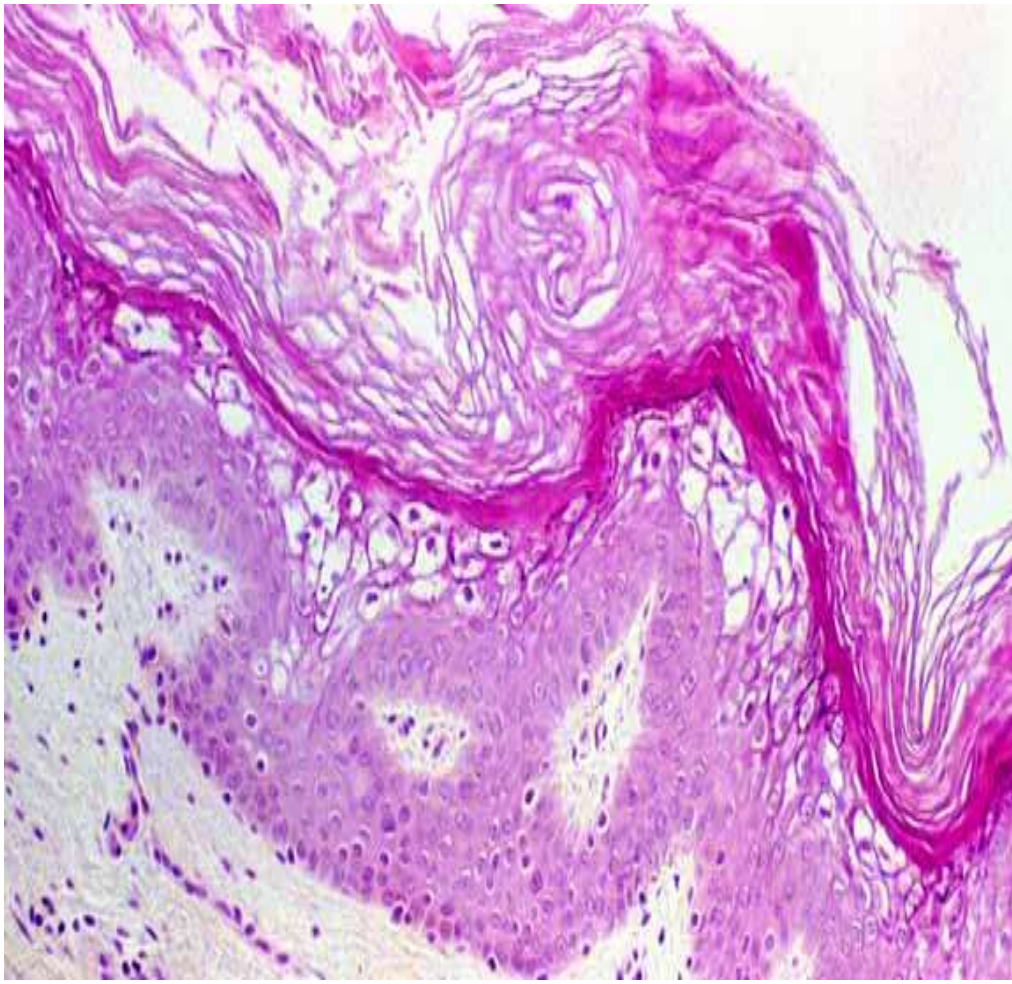
# Normal: Orthokeratosis: basket-weaved horny cell layer




# Hyperkeratosis

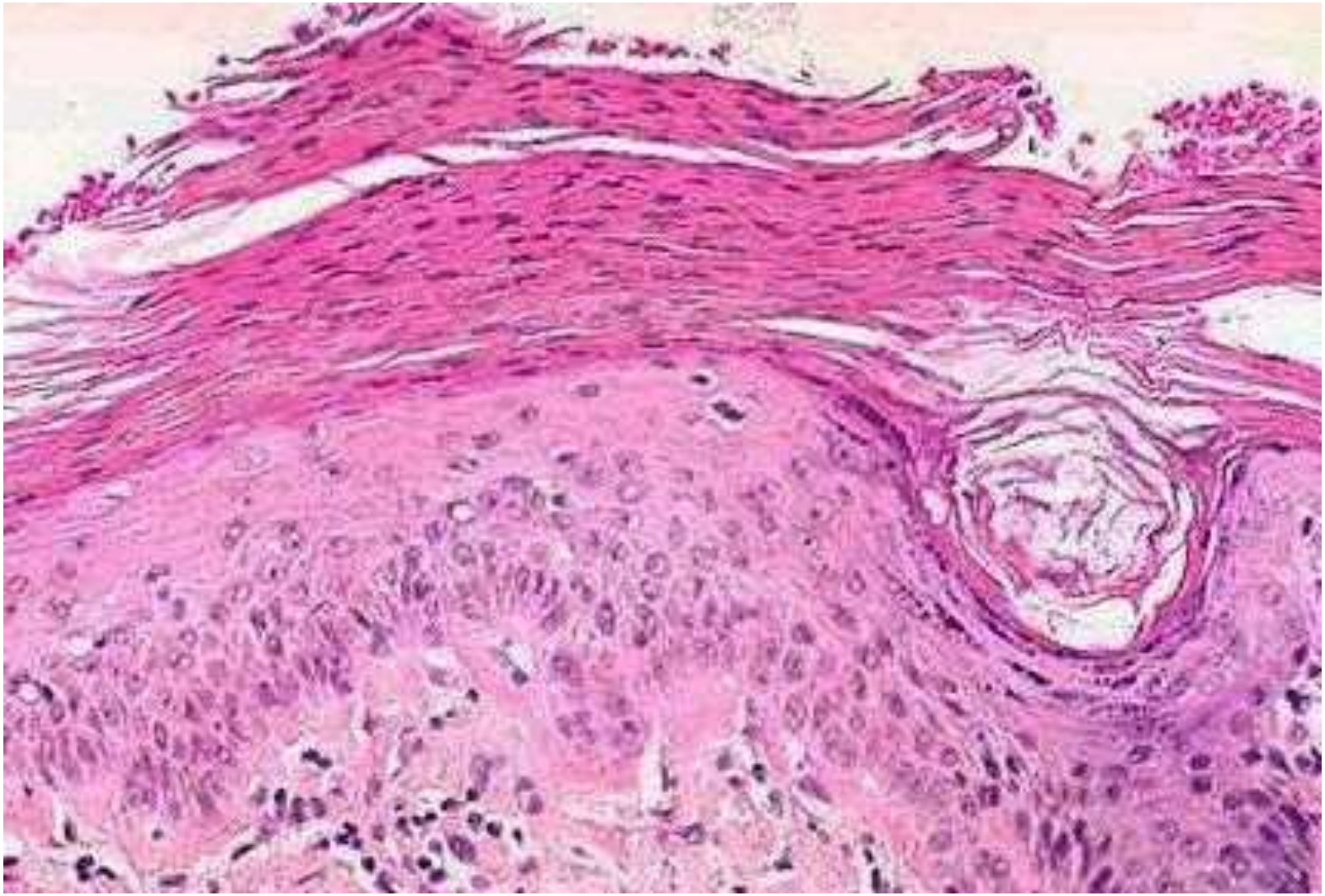
Hyperplasia of the stratum Corneum with abnormal keratin.

The horny cell layer becomes abnormally thick

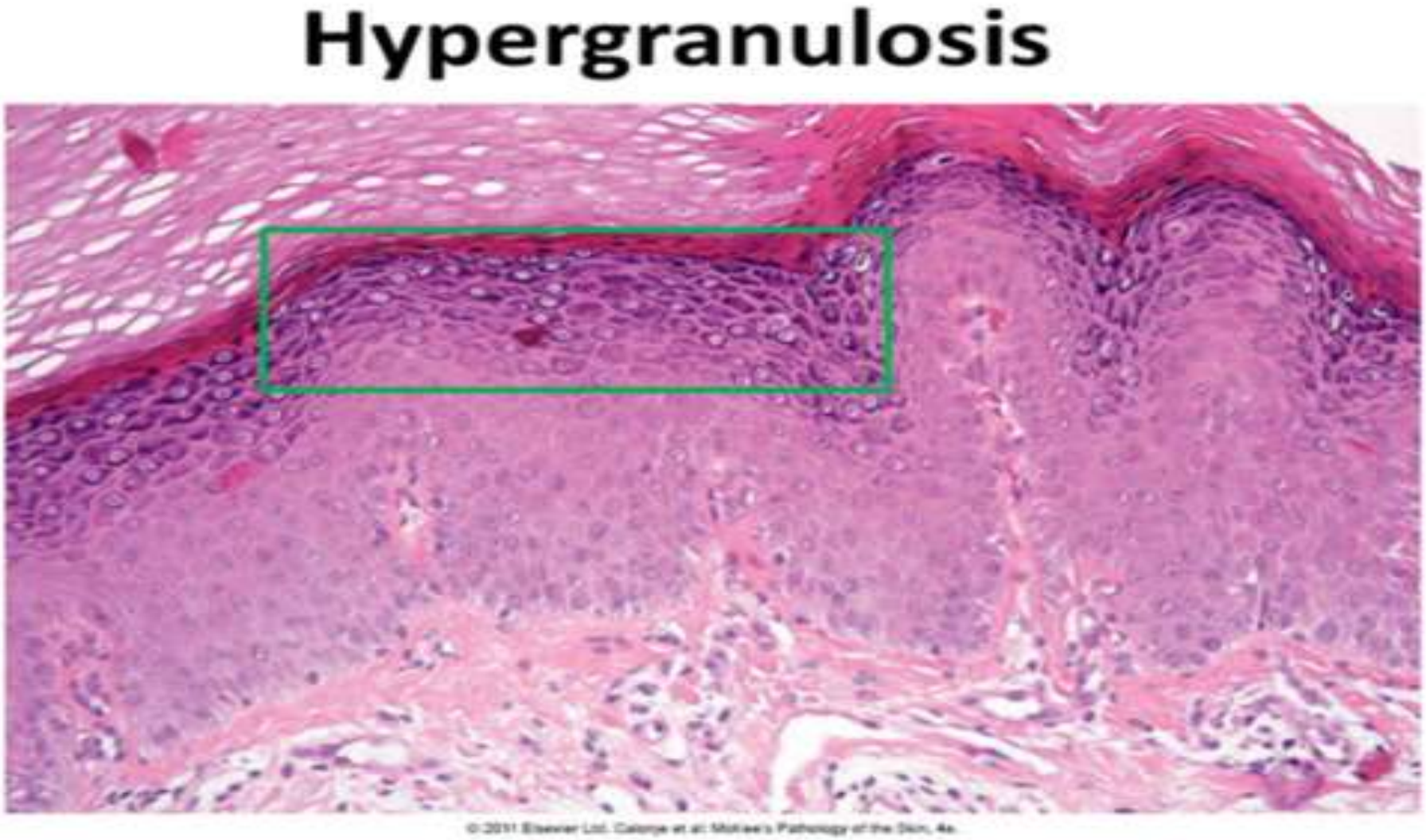


# Parakeratosis

Keratinization characterized by retention of the nuclei.



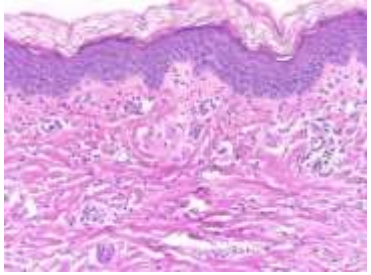
Hypergranulosis is a thickening of the granular cell layers to 4 or more layers from the normal (1-3 layers)



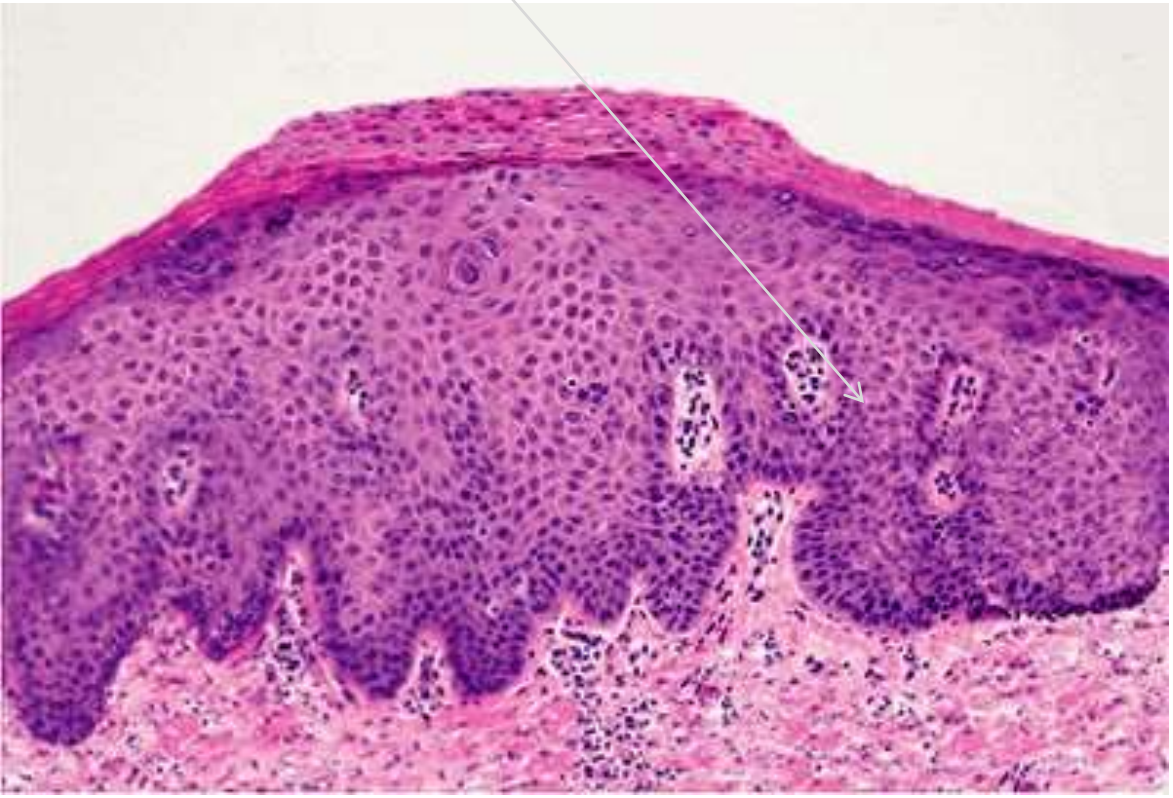

Hyperplasia of stratum granulosum (granular cell layer)

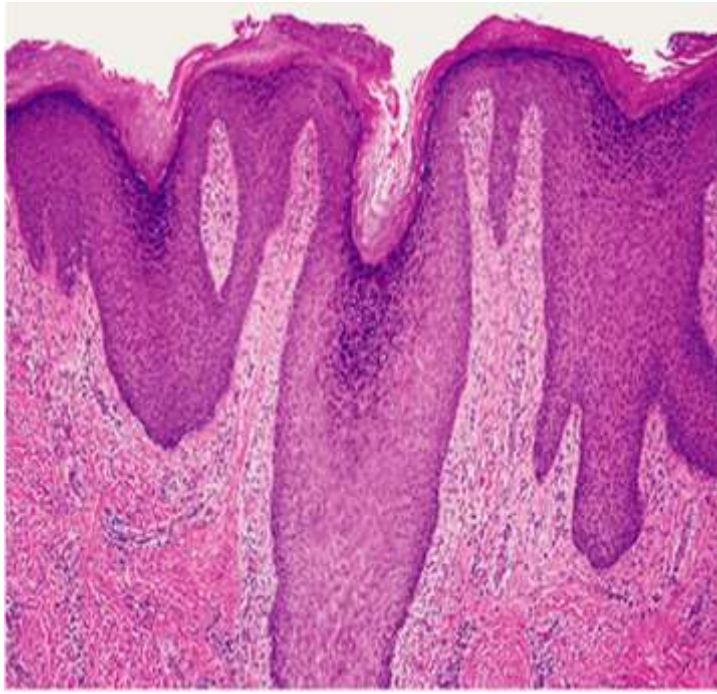
pathologic terms

# Acanthosis



- Epidermal hyperplasia preferentially involving the stratum spinosum



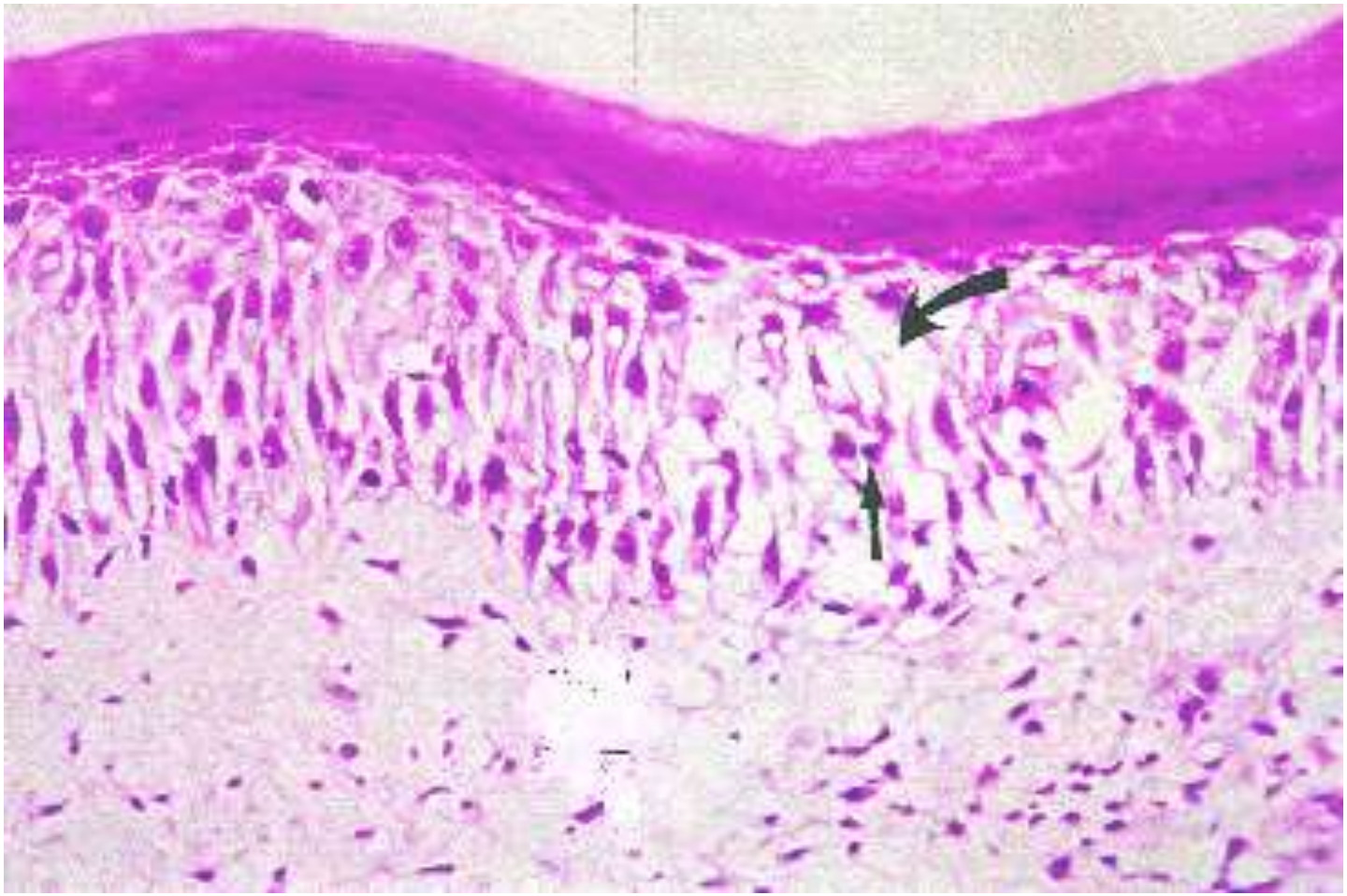


# Acantholysis

**LOSS OF INTERCELLULAR CONNECTION RESULTING IN LOSS OF COHESION.**

# Spongiosis

Intracellular edema of the epidermis.



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# *Inflammatory disorders of skin(dermatosis*

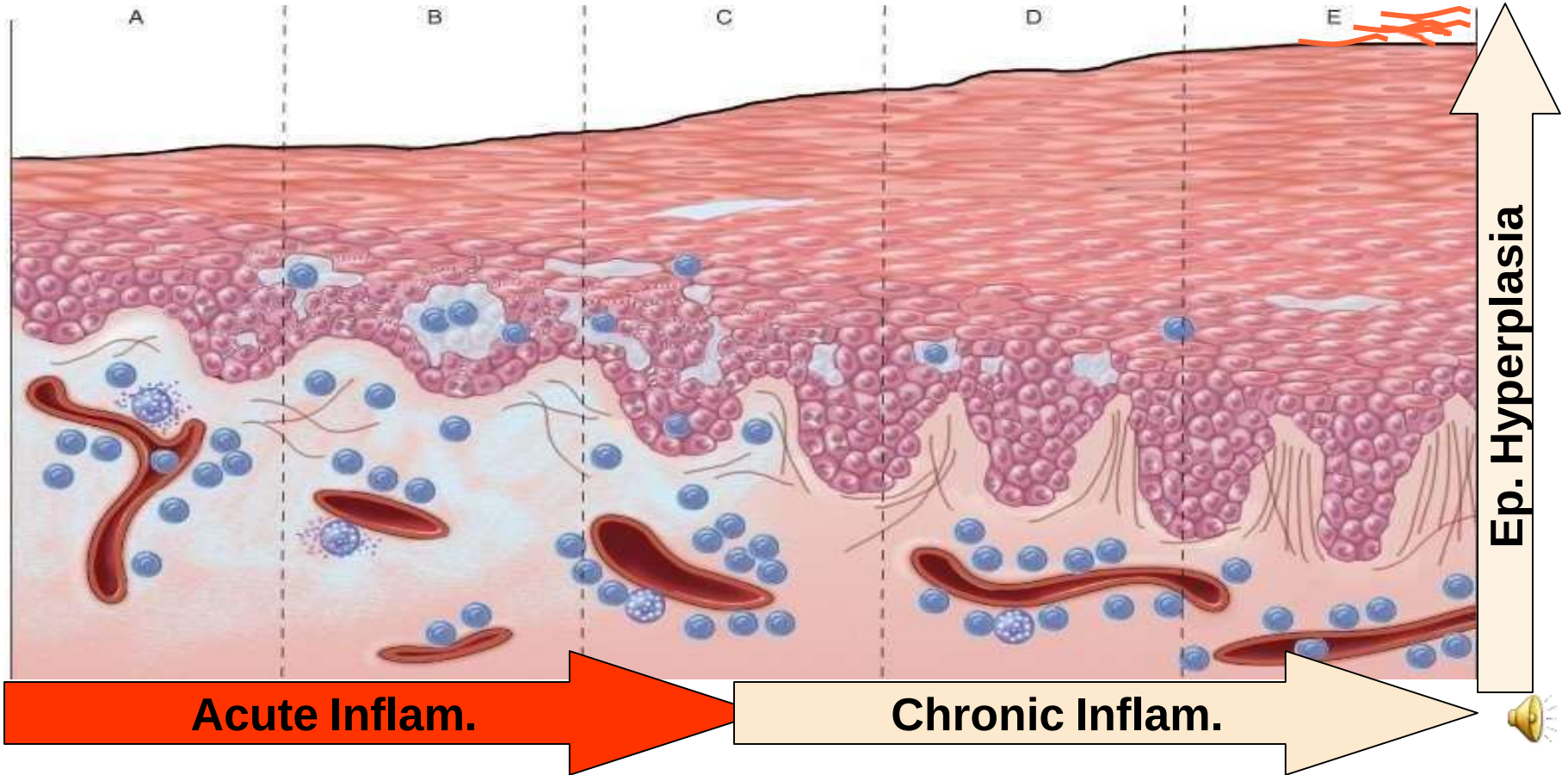
## *1*. Acute inflammatory Dermatosis:

**characterized by**

- **Duration of days to weeks**
- **Acute inflammatory cells infiltration rather than neutrophils.**
- **Edema, vascular, epidermal, & subcutaneous injury.**
- **Examples: like URTICARIA, & ECZEMA**

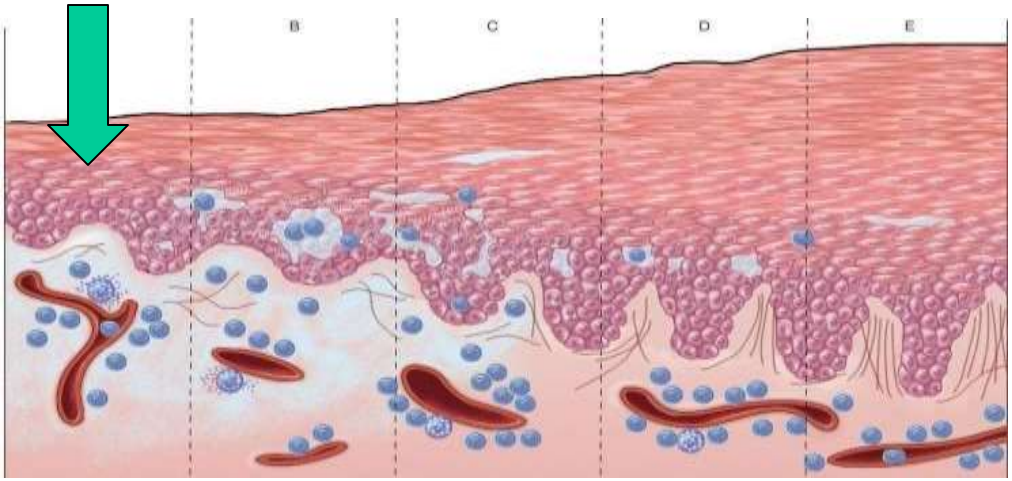
# INFLAMMATORY disorders: Pathogenesis

| Urticaria | Acute Eczema | Chronic Eczema | Lichen Sclerosis |
|---|---|---|---|
| Dermal Infl | Epidermal Infl | Hyperplasia | Hyperkeratosis |



# URTICARIA (Hives)

- Type I hypersensitivity – Allergy
- All ages, more in 20 – 40y.
- Erythematous papules and plaques and wheals
- Individual lesions are transient, usually resolve in 24 hr, but entire episode may last for days.
- Usually on trunk and extremities.

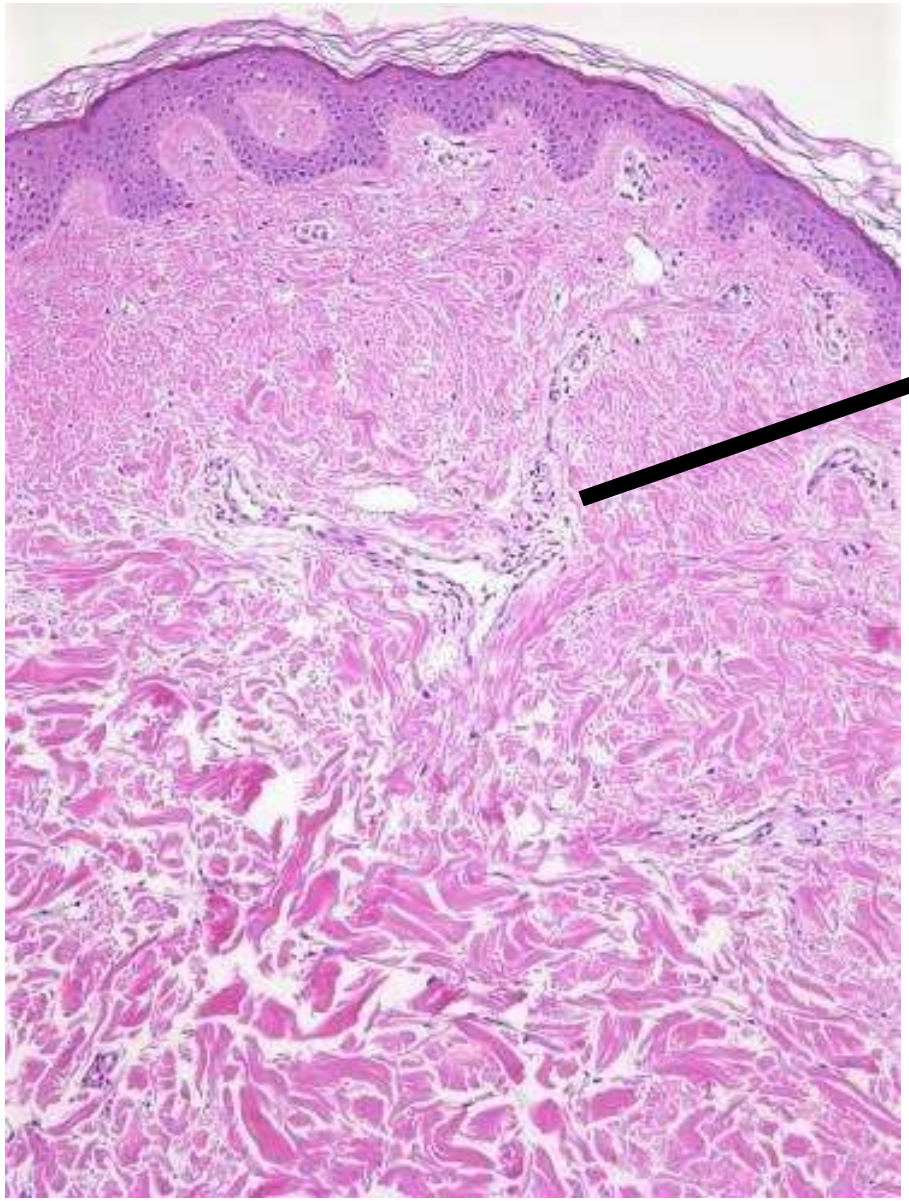
# Urticaria (Hives)

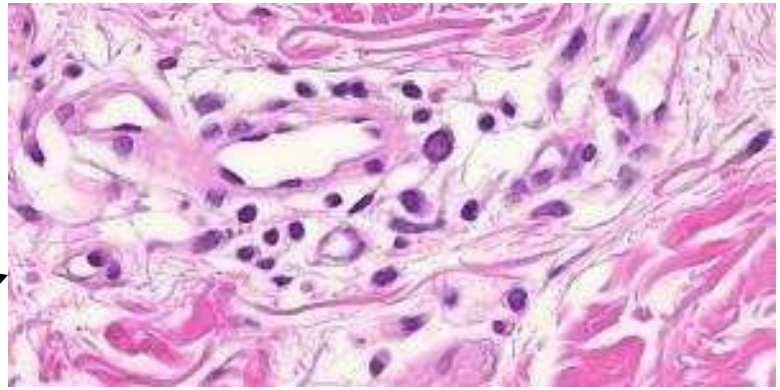
# Urticaria :Mic:

characterized by.

1. Early normal skin biopsy.
2. Superficial perivenular infiltrate consisting of mononuclear cells, rare neutrophils.
3. Widely spaced collagen bundles than in normal skin.

# URTICARIA – Histopathology





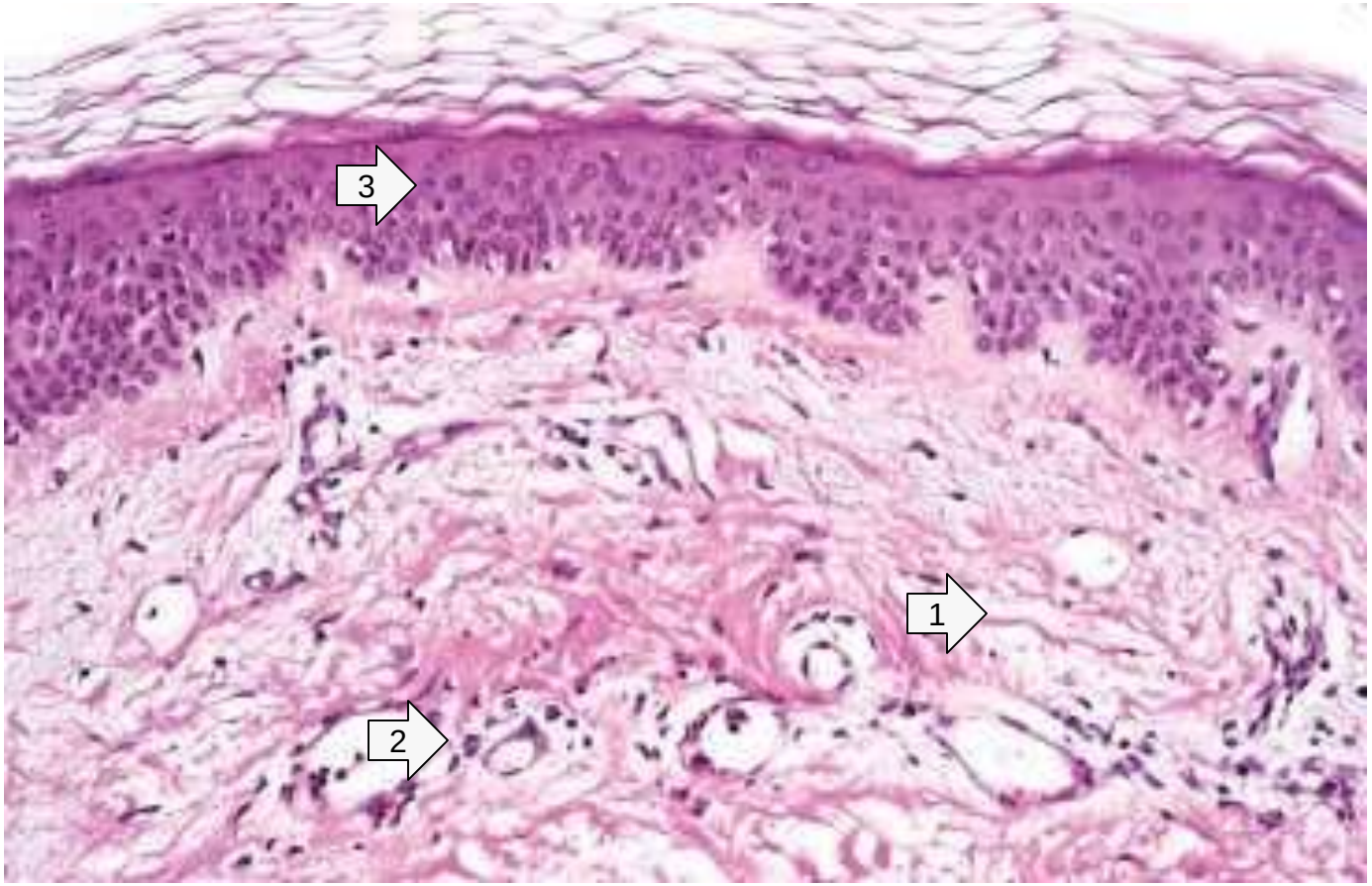
Dermis: Perivascular inflammatory infiltrate: lymphocytes, eosinophils , rare neutrophils, .

* lack of spongiosis or other epidermal changes.

# URTICARIA (Hives)

- Pathogenesis
- Type I hypersensitivity – Allergy
  - Follows exposure to Ag: (pollens, foods, drugs, pressure, temperature, insect…. Etc).
  - Ag → IgE → Mast cell Degranulation → Inflammation.
  - perivascular inflammatory infiltrate: lymphocytes, eosinophils rare neutrophils

# Urticaria – Microscopic features




1. Superficial dermal edema (space between collagen)
2. Dilated blood vessels with perivascular inflammatory cells.
3. Normal Epidermis (no spongiosis or hyperplasia)

# Eczema

- Origin of this word:
- The word ‘eczema’ comes from the Greek ‘boiling’ a reference to the tiny vesicles that are often seen in the early acute stages of the disorder, but less often in its later stage

غليان

# ***Eczema.***

A number of pathogenetically different conditions, all are characterized by red, papulo-vesicular oozing & crusted lesions at early lesions( acute phase),

with time in the presence of persistent antigen stimulation these lesions become less wet (fail to ooze or form vesicles )and progressivey scaly(hyperkeratosis) as the epidermis thickens (acanthosis) .. develop into raised, scaling plaques

( development of chronic form of dermatiotis)

Examples: 1. **C**ontact dermatitis (due to chemicals)

2. **A**topic dermatitis(unknown cause but family history of eczema, allergic rhinitis or **a**sthma)

3. drug- related eczematous dermatitis

***Mic:***

1. epidermis: Spongiosis, which is accumulation of edema fluid within the epidermis.
2. Dermis: Superficial perivascular, lymphocytic infiltrate associated with papillary dermal edema & mast cells degranulation.
3. Prominent eosinophils infiltrate.

# Eczema
# clinical features

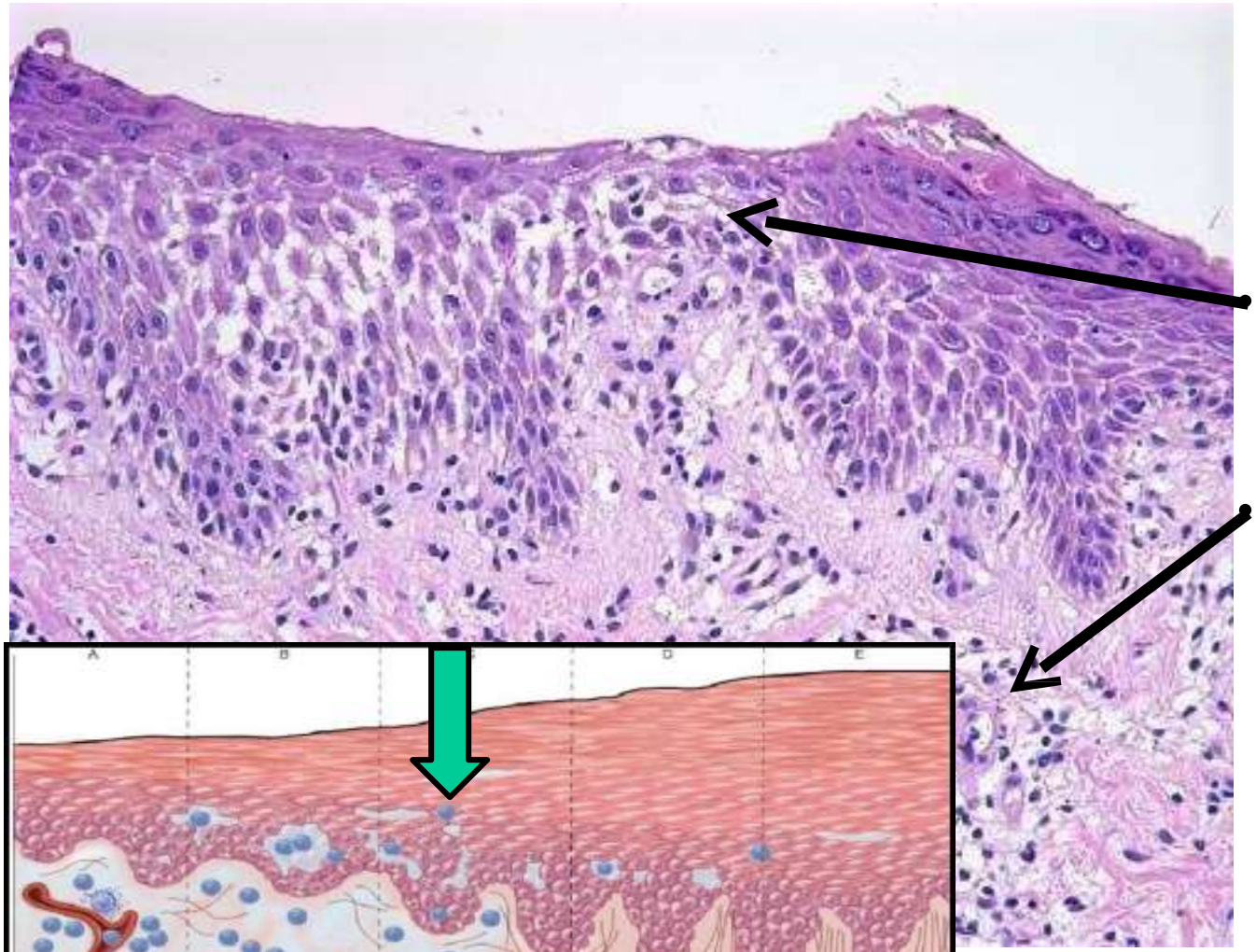
- **Acute** : pruritic (itchy), edematous, plaques, often containing small and large blisters (vesicles and bullae)
- oozing and crusted lesion
- Contact reaction

  to poison ivy,

  laundry detergent.

- **Chronic:** persistent antigen stimulation, lesions may become less "wet" and progressively scaly as the epidermis thickens.

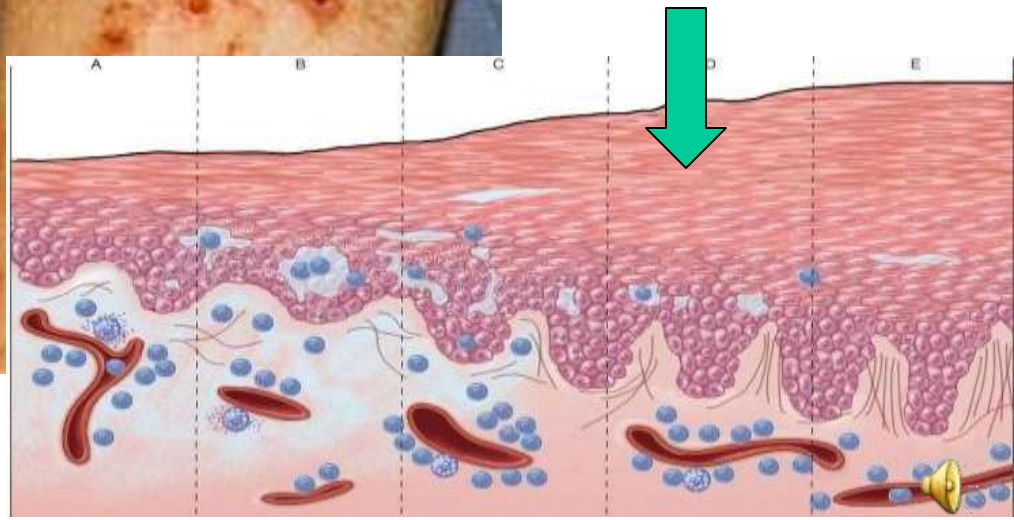
# ECZEMA – histology



Spongiosis (Intraepidermal) edema

Superficial perivascular lymphocytic infiltrate

# ECZEMA dry - (atopic)



# ECZEMA – pathogenesis:

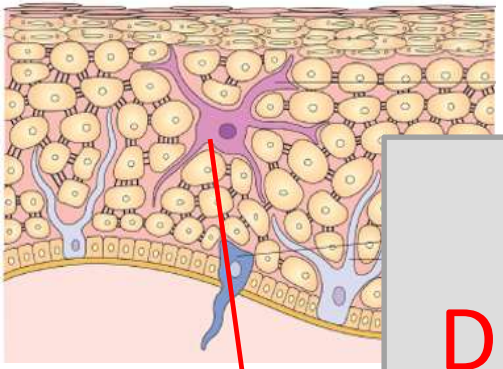
Hypersensitivity Reaction:

- **Initial exposure to antigen:**
  - Antigen processed by Langerhans cells and presented to T cells in the lymph node → T cell activation → memory cells.
- **Re-exposure to antigen:**
  - Quick (memory T cells) response → inflammation → urticaria, erythema, wet eczema
- **Persistence of antigen stimulation:**
  - Chronic inflammation → Acanthosis, hyperkeratosis– dry eczema.

# Allergic Contact Dermatitis

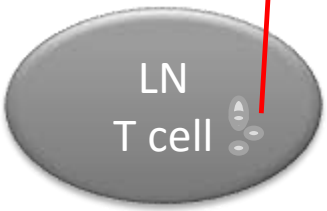
## Pathogenesis

initial exposure to an environmental contact sensitizing agent



DELAYED TYPE HYPERSENSITIVITY

Memory T cell




on re-exposure to the antigen, CD4+ T lymphocytes migrate to the affected skin

they release cytokines that recruit additional inflammatory cells

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  - Seborrheic Keratosis.
- Malignant:
  - BCC, SCC, Melanoma.

# Chronic inflammatory dermatosis:

- Have duration last for many months to years.
- Examples (psoriasis, lichen planus).

# PSORIASIS – الصدفية

- A common chronic inflammatory dermatosis affecting 2% of people in the United States.
- **Etiology :** exact cause :unknown
- Multifactorial: genetic and immune and environmenta
- Sensitized T cells infiltrate the skin and secrete cytokines and growth factors
  - Inflammation, Increased cell turnover
  - **abnormal proliferation and turnover of epidermis (reduced from a month to only 4 days for a cell to transit from basal layer to surface).**
  - Vascular proliferation angiogenesis
  - Trauma precipitates lesions – Koebner phenomena .
- Multi system disorder:
  - Arthritis, myopathy, enteropathy, Immunodefficiency

# *Clinical Features of Psoriasis*

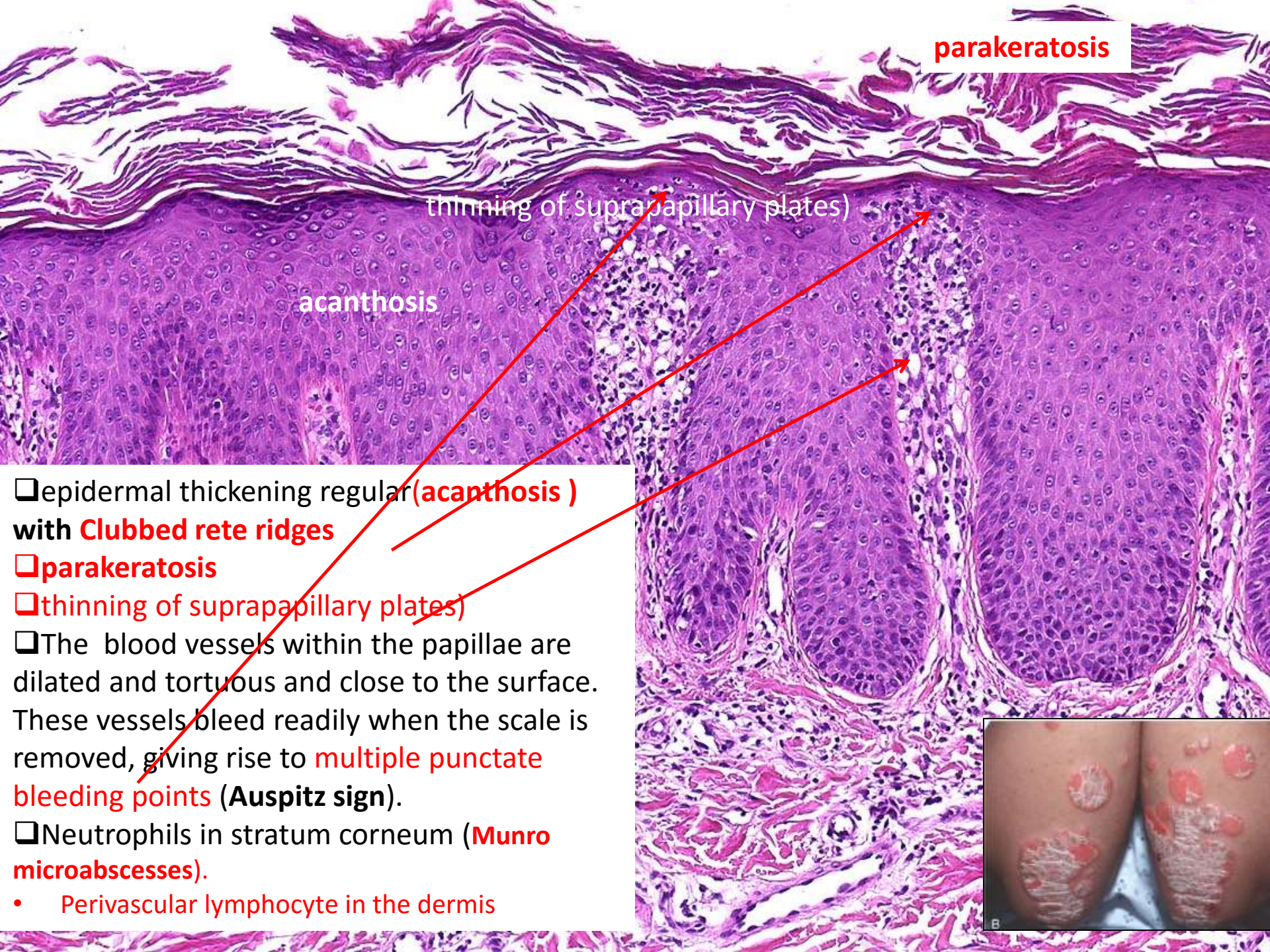
- Site: skin of the elbows, knees, scalp, lumbosacral areas, and nails in 30%of cases
- Appearance: *a well-demarcated pink plaque covered by loosely adherent silver-white scale*
- Removal of scales → point bleeds – Auspitz sign.

**Nail pitting**
**Onycholysis**

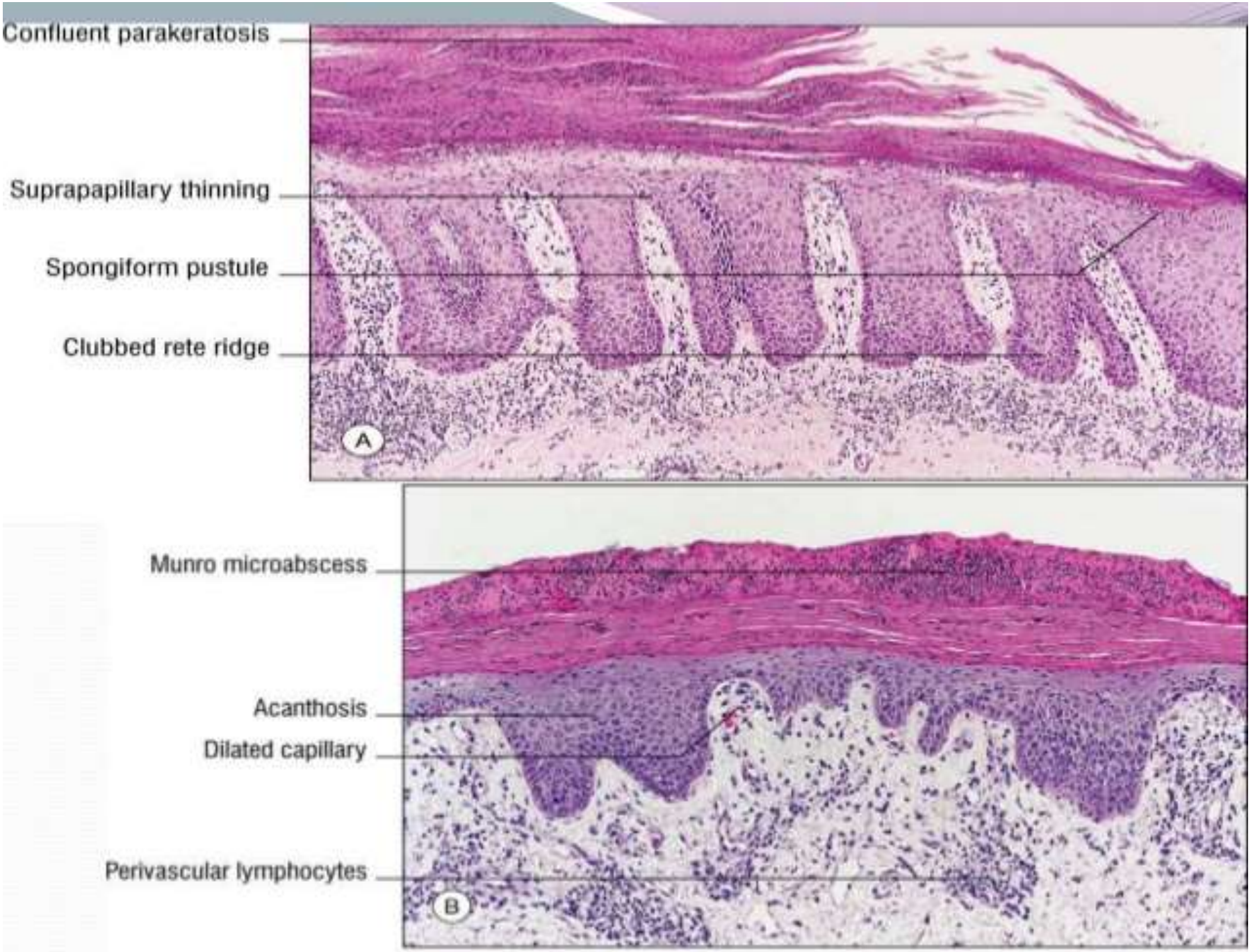
# Psoriasis

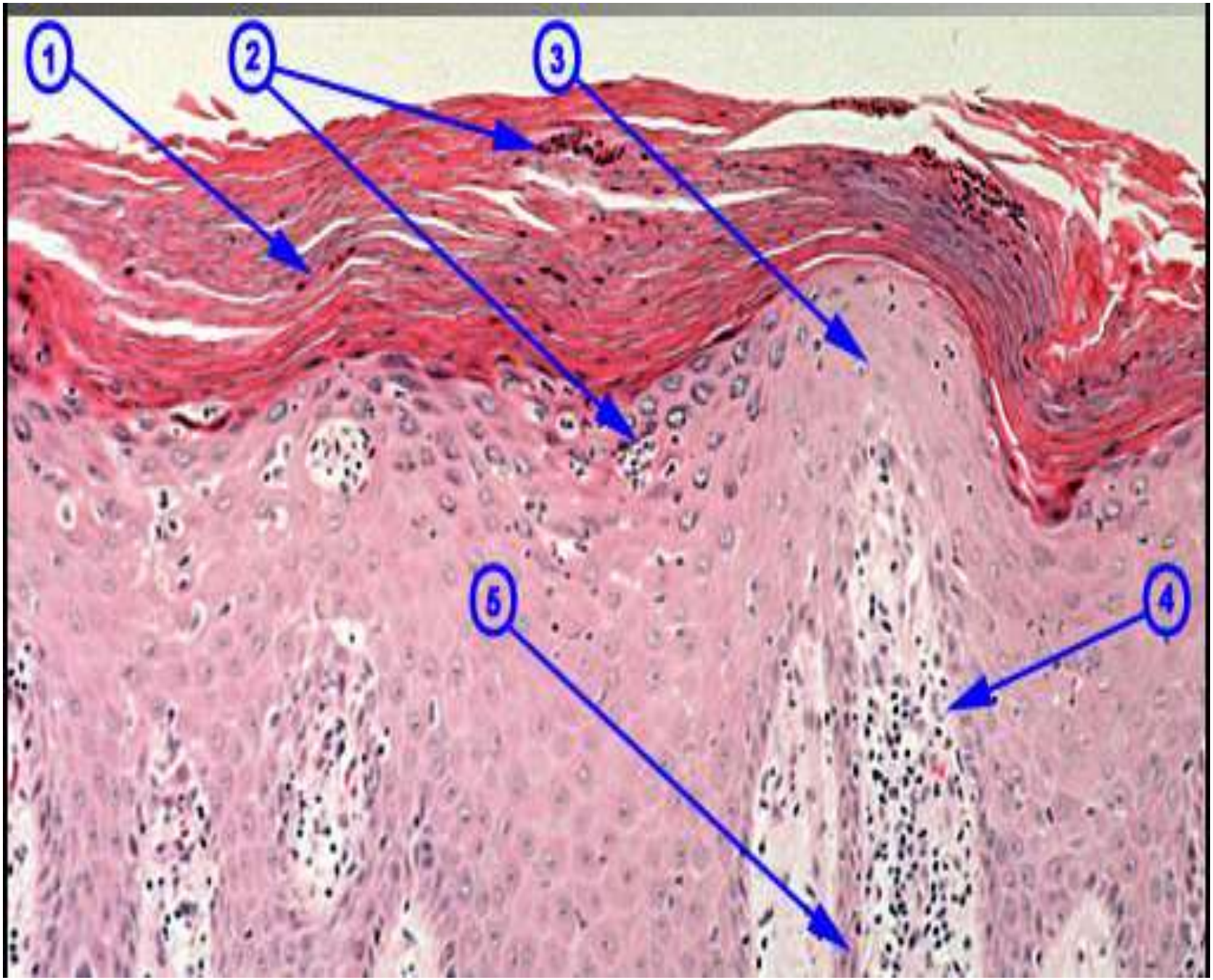
# Psoriasis Microscopically:

- Acanthosis with regular downward elongation with clubbed rete ridges.
- stratum granulosum is thinned or absent with extensive overlying parakeratotic scales,
- Thinning of suprapapillary dermis,
- Blood vessels within the dermal papillae are dilated tortuous and close to the surface. These vessels bleed easily when the scale is removed, giving rise to multiple punctate bleeding points (Auspitz sign).
- Neutrophils aggregate within superficial epidermis & the parakeratotic stratum corneum (Munro microabscess)




- epidermal thickening regular **(acanthosis )** **with Clubbed rete ridges**
- **parakeratosis**
- thinning of suprapapillary plates)
- The blood vessels within the papillae are dilated and tortuous and close to the surface. These vessels bleed readily when the scale is removed, giving rise to multiple punctate bleeding points (**Auspitz sign**).
- Neutrophils in stratum corneum (**Munro microabscesses**).
- Perivascular lymphocyte in the dermis


Confluent parakeratosis
Suprapapillary thinning
Spongiform pustule
Clubbed rete ridge
A
Munro microabscess
Acanthosis
Dilated capillary
Perivascular lymphocytes
B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# Lichen planus الحزاز المسطح .

***Sites:*** characteristically, there are bilateral symmetrical lesions, mainly on the limbs (about the wrists, elbows) , in 70% of cases associated with oral lesions.

**Pathogenesis:** the exact cause is unknown, but suppose to be a release of Ag at the level of the basal cell layer and the dermo-epidermal junction may elicit a cell mediated cytotoxic immune response.

**Prognosis**: is a self limited dis, resolve spontaneously 1-2 years after onset,

Often leaving zones of postinflammatory hyperpigmentation . Oral lesions may persist for years

**Morphology:**

***Gross: 4 Ps : pruritic, purple, papules*** which may coalesce focally to form ***Plaques.***

***Mic:***

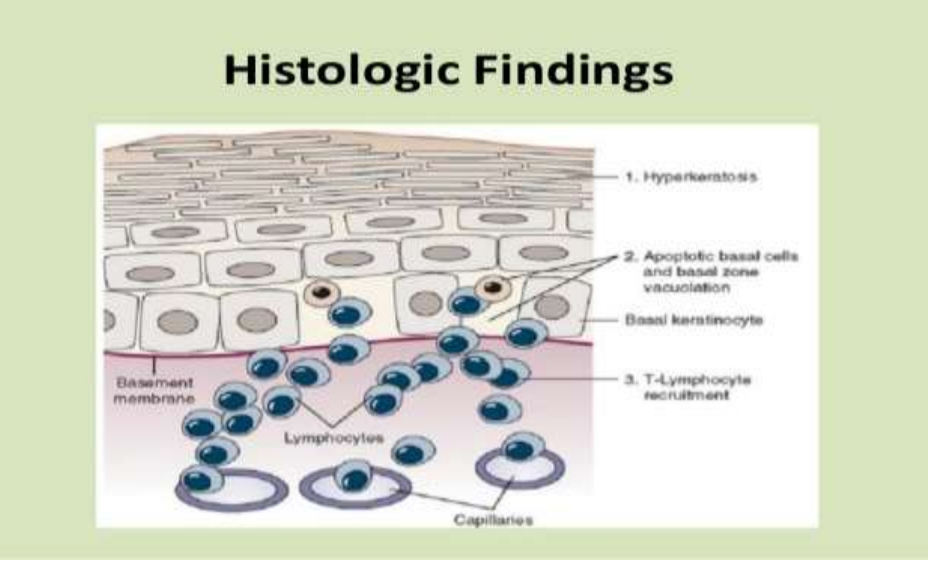
1. Continuous infiltrate of lymphocytes along the dermoepidermal junction.
2. Dermoepidermal junction shows saw toothing appearance.
3. **Civatte bodies** a nucleated, necrotic basal cells incorporated into the inflamed papillary dermis.

# Lichen Planus: **Pruritic, purple, papules**

- Pruritic, Purple, Papules and Plaques.
- Skin , oral,
- Self limited. 1-2 years.
- Basal layer, Interface dermatitis.
- Anucleate dead epidermal cells in basal layer – Civatte bodies.

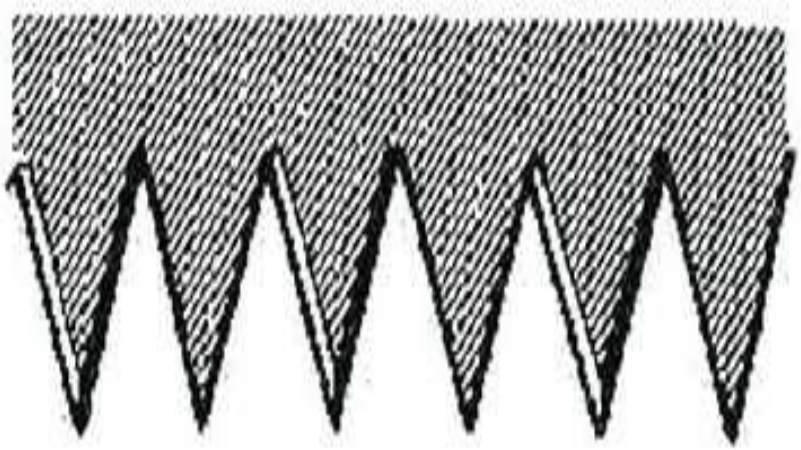
White lines: **Wickham Striae**

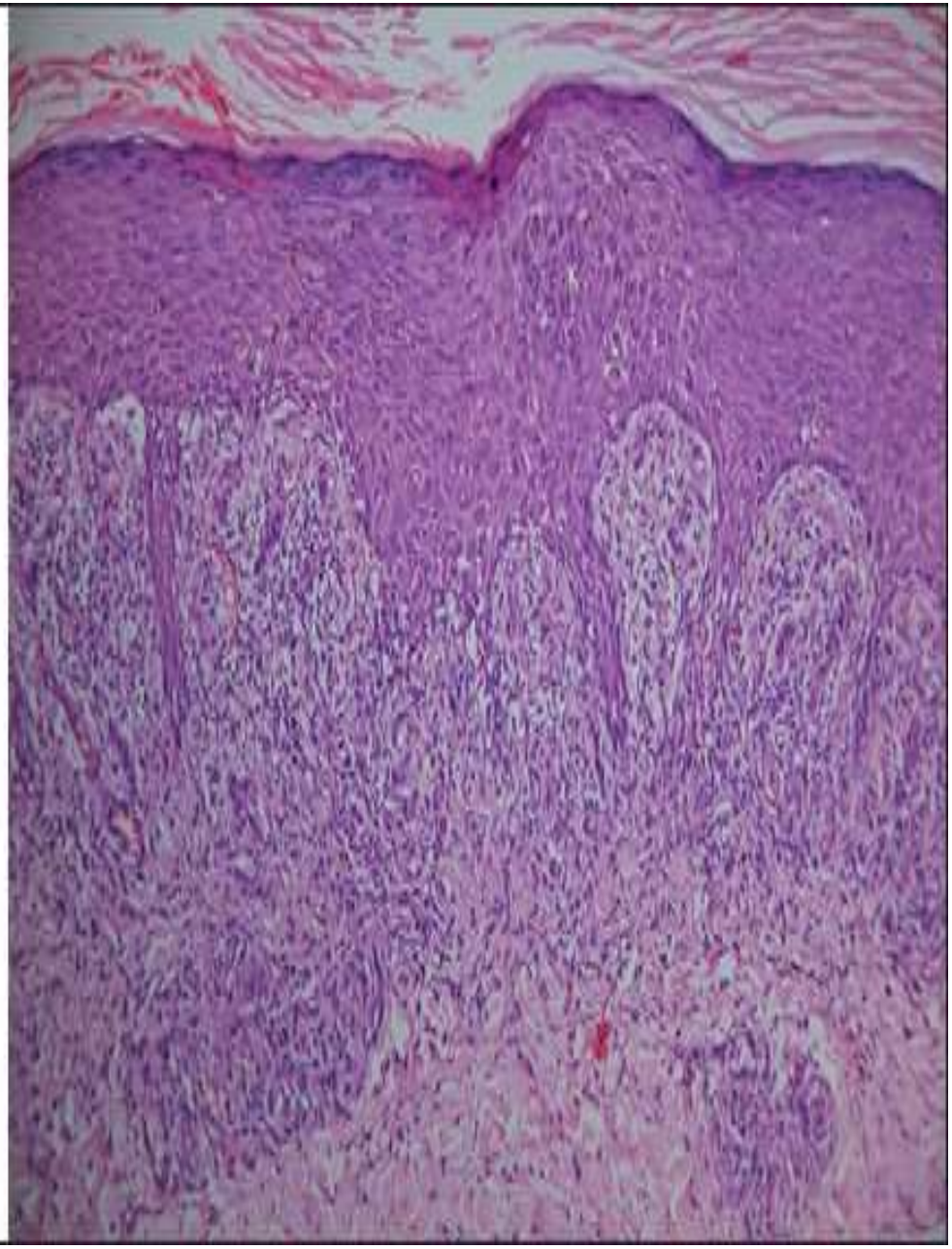
# Lichen Planus
# clinical

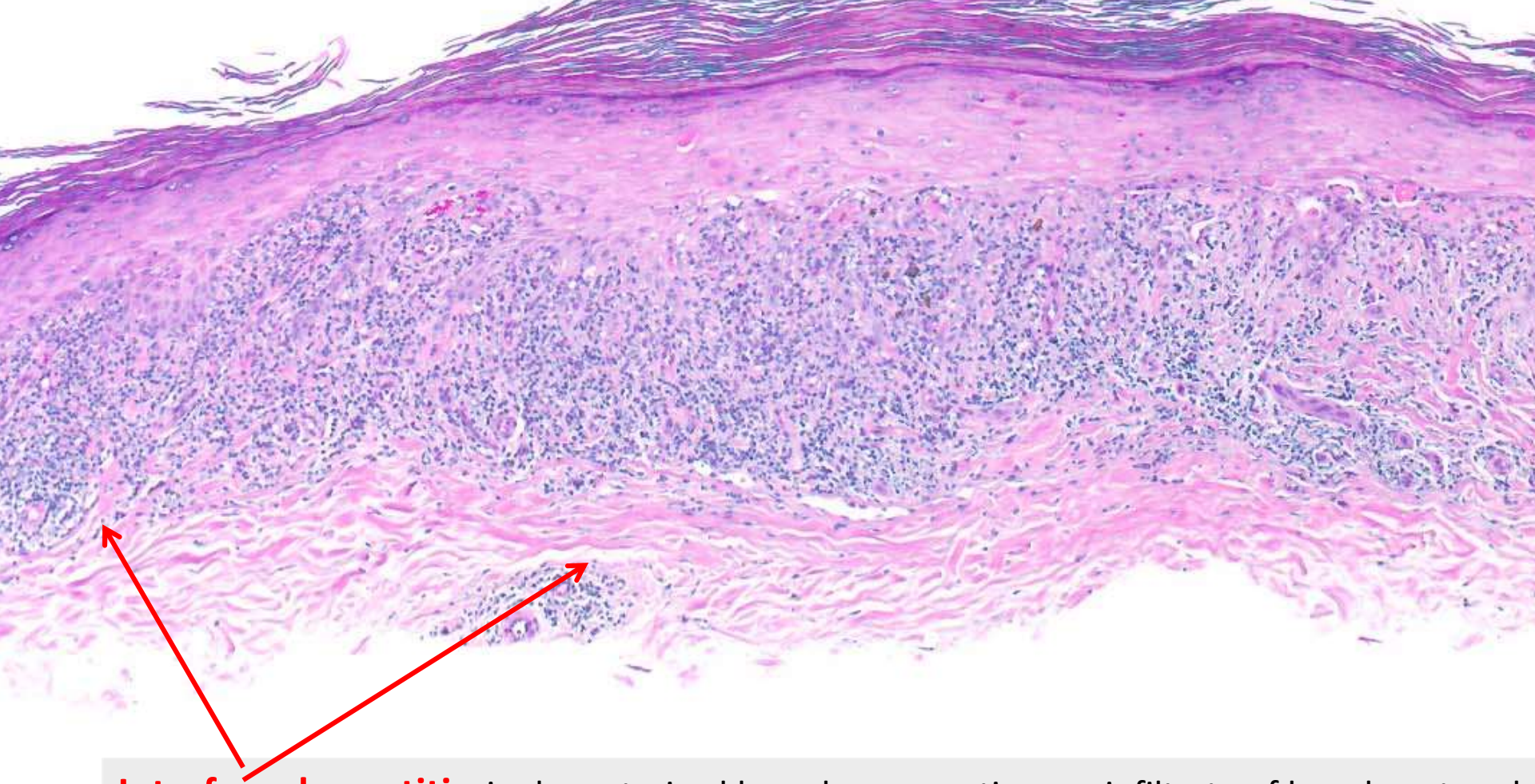



# Lichen planus

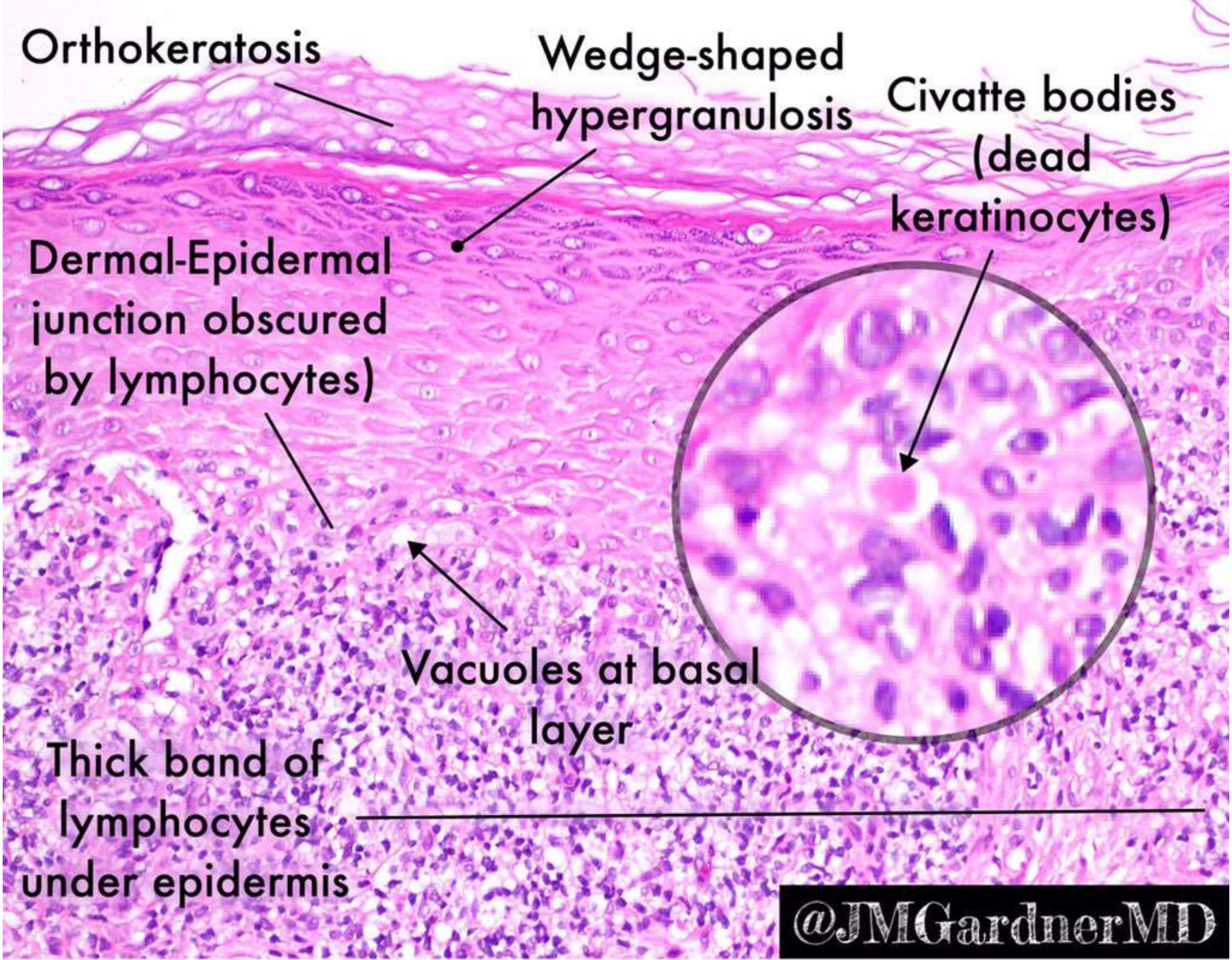
- **microscopic (histologic) description**
- Hyperkeratosis and acanthosis; prominent granular cell layer, saw toothing of rete pegs, bandlike chronic inflammatory infiltrate (T cells and macrophages) that destroys the dermoepidermal junction
- Civatte bodies (apoptotic basal cells)

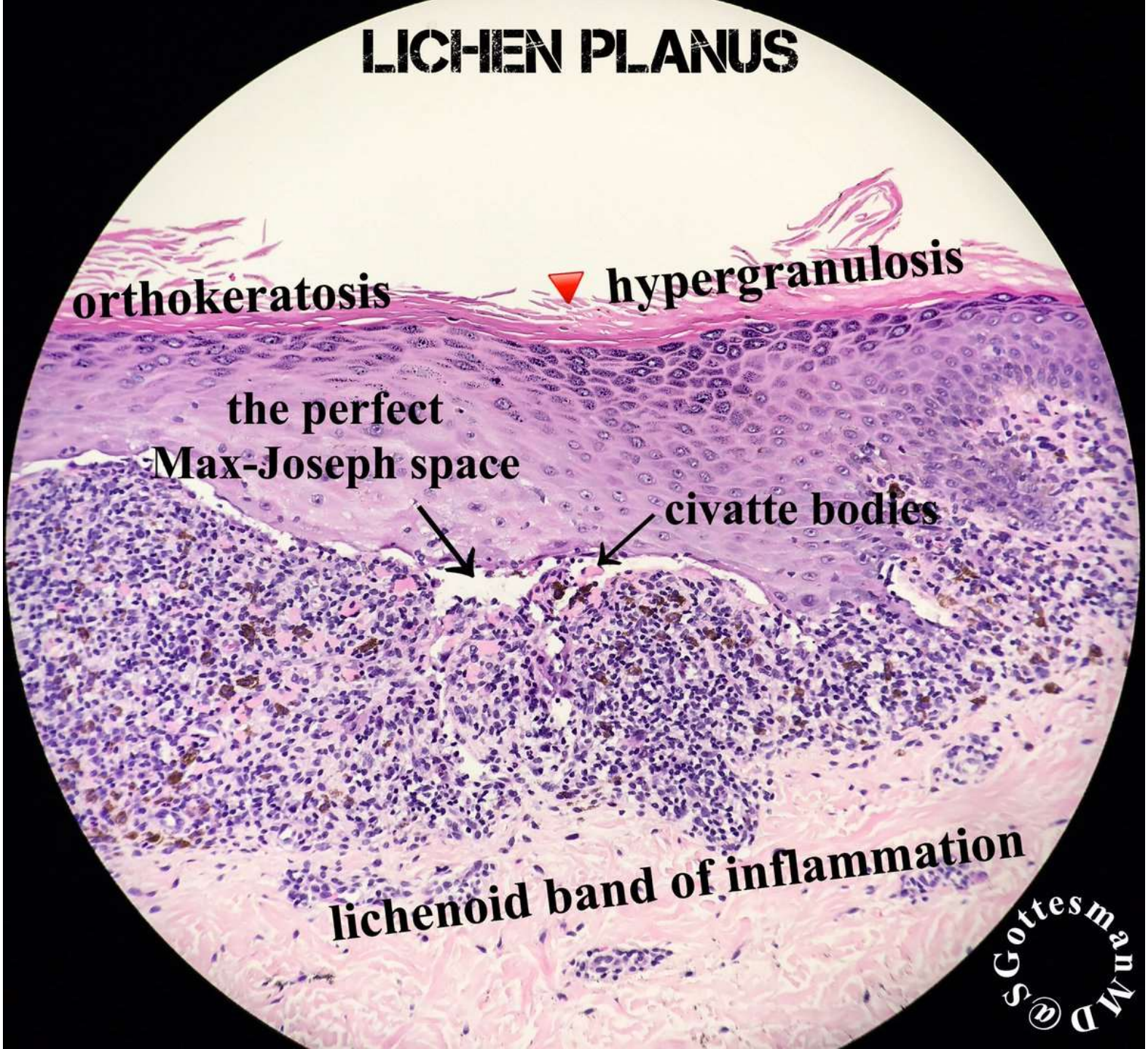






**Interface dermatitis,** is characterized by a dense, continuous infiltrate of lymphocytes alo
dermoepidermal junction
basal keratinocytes show degeneration and apoptosis
This pattern of inflammation causes the dermoepidermal interface to assume an angulated
("sawtoothing").


Orthokeratosis
Wedge-shaped hypergranulosis
Civatte bodies (dead keratinocytes)
Dermal-Epidermal junction obscured by lymphocytes)
Vacuoles at basal layer
Thick band of lymphocytes under epidermis
@JMGardnerMD


LICHEN PLANUS
orthokeratosis
hypergranulosis
the perfect
Max-Joseph space
civatte bodies
lichenoid band of inflammation
©S Gottesman MD

# Dermatopathology

1. Acute Inflammations:
   - Urticaria,
   - Acute Eczema,
   - Erythema Multiforme.
2. Chronic Inflammations:
   - Chronic Eczema,
   - Psoriasis,
   - Lichen planus.
3. Infections
   - Bacterial (Impetigo),
   - Fungal(tinea) &
   - Viral(warts).

1. Blistering Diseases
   - Pemphigus,
   - Pemphigoid,
   - Dermatitis herpetiformis.

5. Neoplastic:

- Benign:
  - Nevi,
  - Actinic Keratosis,
  - Seborrheic Keratosis.
- Malignant:
  - BCC, SCC, Melanoma.

# ***Blistering (bullous)diseases.***

- A group of disorders characterized by formation of bullae.
- These bullae are either subepidermal or intraepidermal

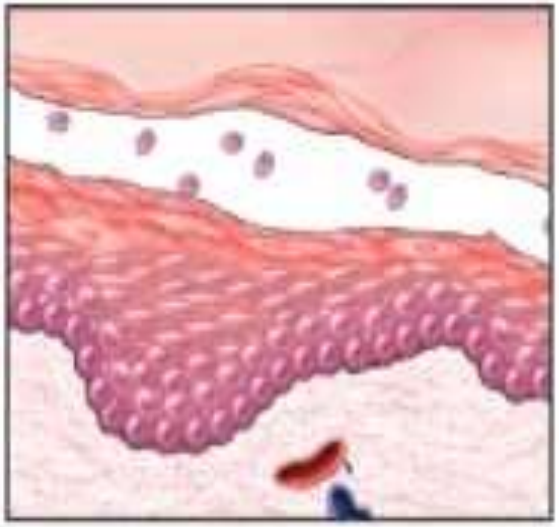
in their location.

- **Cause:** These bullae are due to acantholysis of epidermal cells junctions.
- **Examples** :
- Pemphigus vulgaris, Bullous Pemphigoid, &dermatitis herptiformis.

# BLISTERING DISEASES

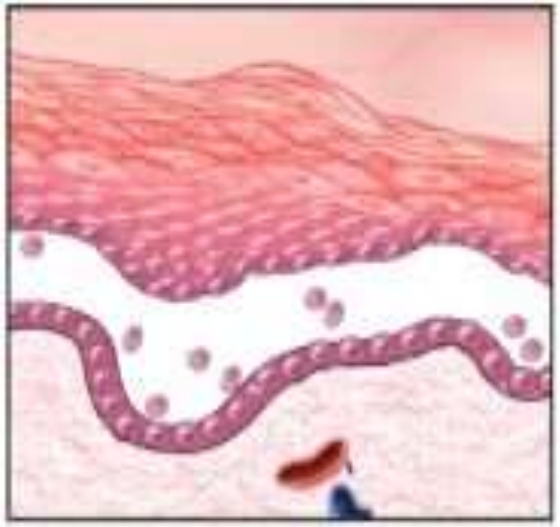
- Subcornial.
- Suprabasal.
- Subepidermal.

PEMPIGUS FOLIACESOUS



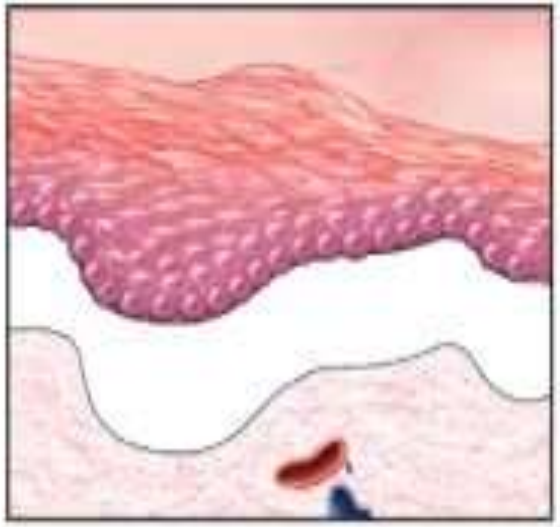
A Subcorneal

PEMPIGUS VULGARIS



B Suprabasal

BULLOUS PEMPHIGOID



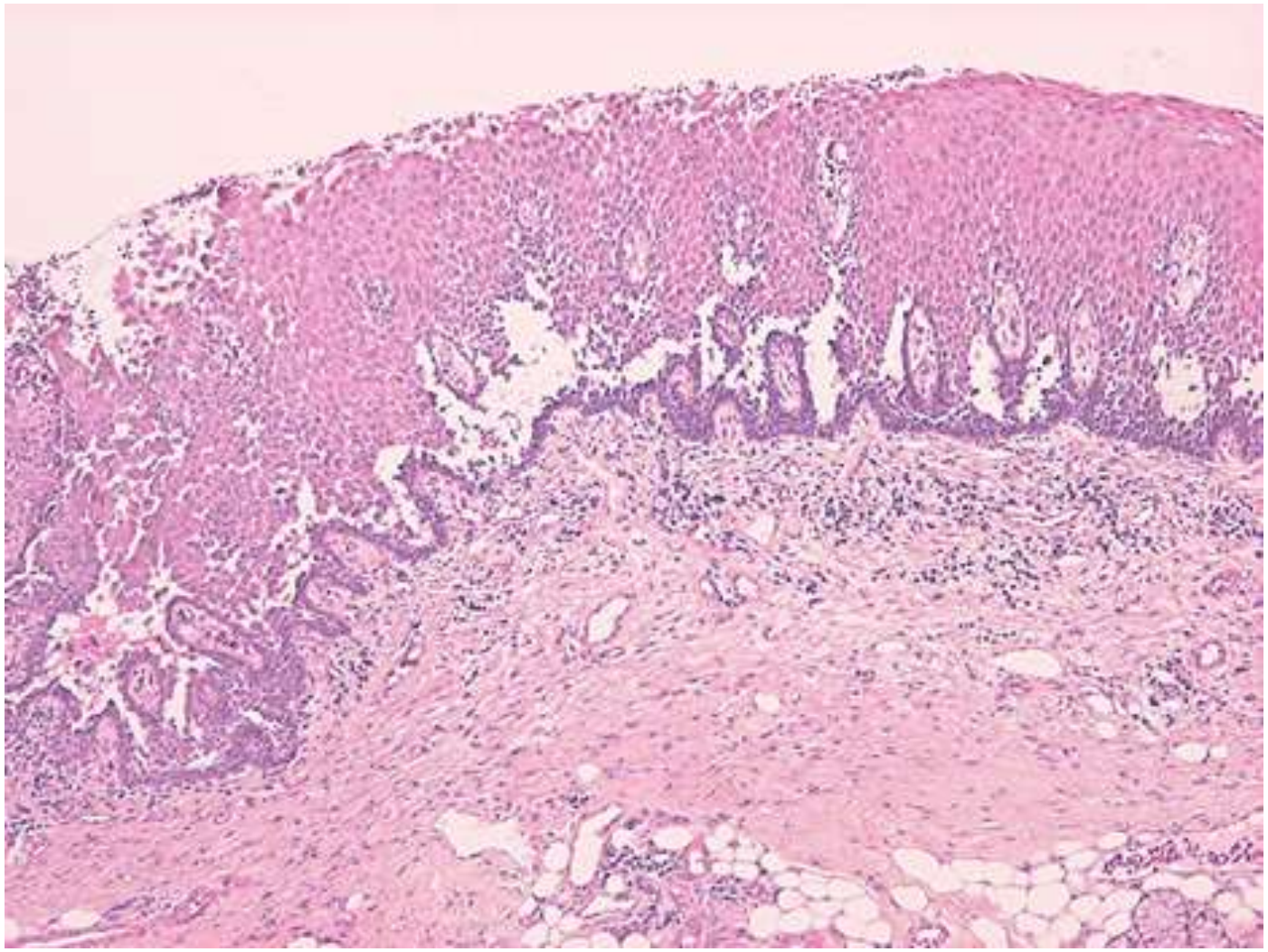
C Subepidermal

# Pemphigus

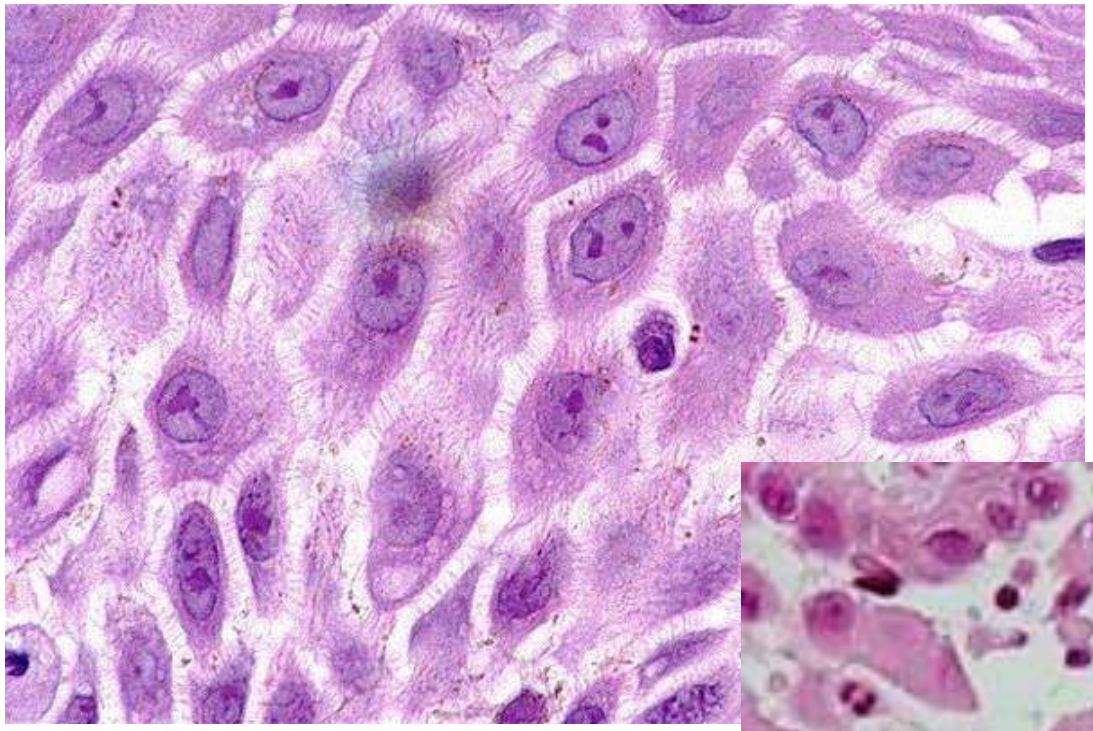
- Characterized by suprabasal acantholytic blisters or bullae.
- **Distribution:** Bullae involve skin & rare the mucous membranes.
- The disease is due to type II hypersensitivity reaction.
- Autoimmune disease Ig G against desmosome
- By immunoflourscent technique, there is netlike pattern of intercellular IgG deposits at the sites of acantholysis

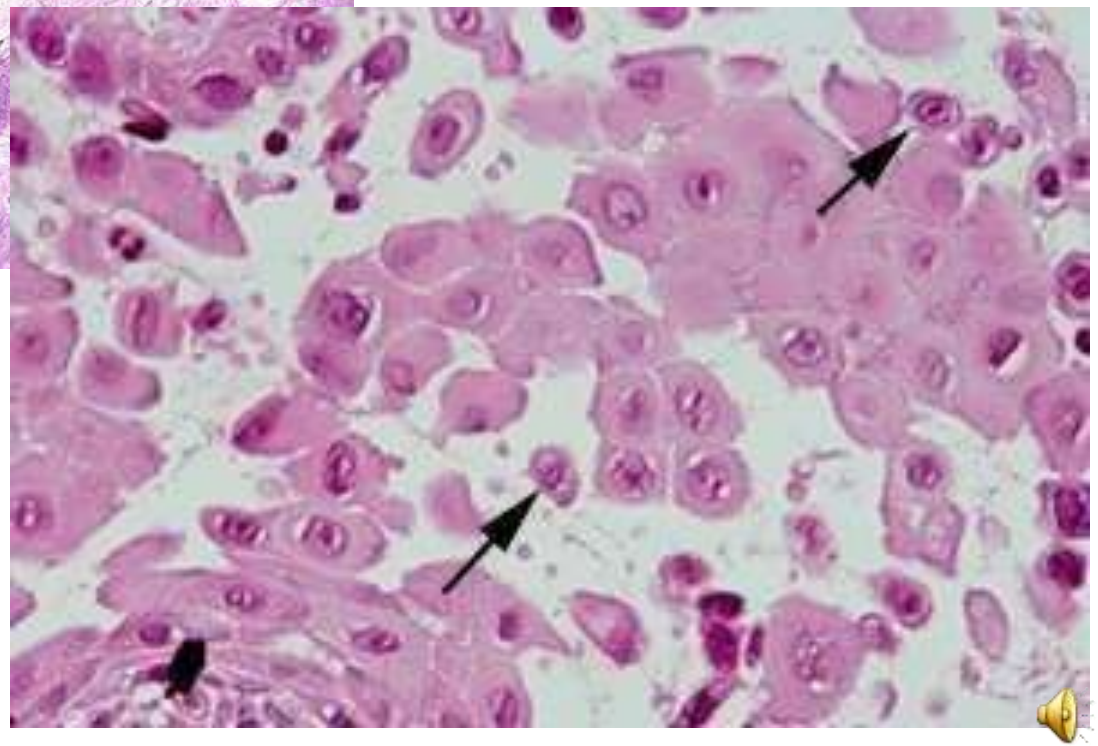
# Pemphigus vulgaris [ **bulla rupture easily and will be covered by dried serum and crust.]**

# Pemphigus vulgaris [ Suparabasal bulla ]

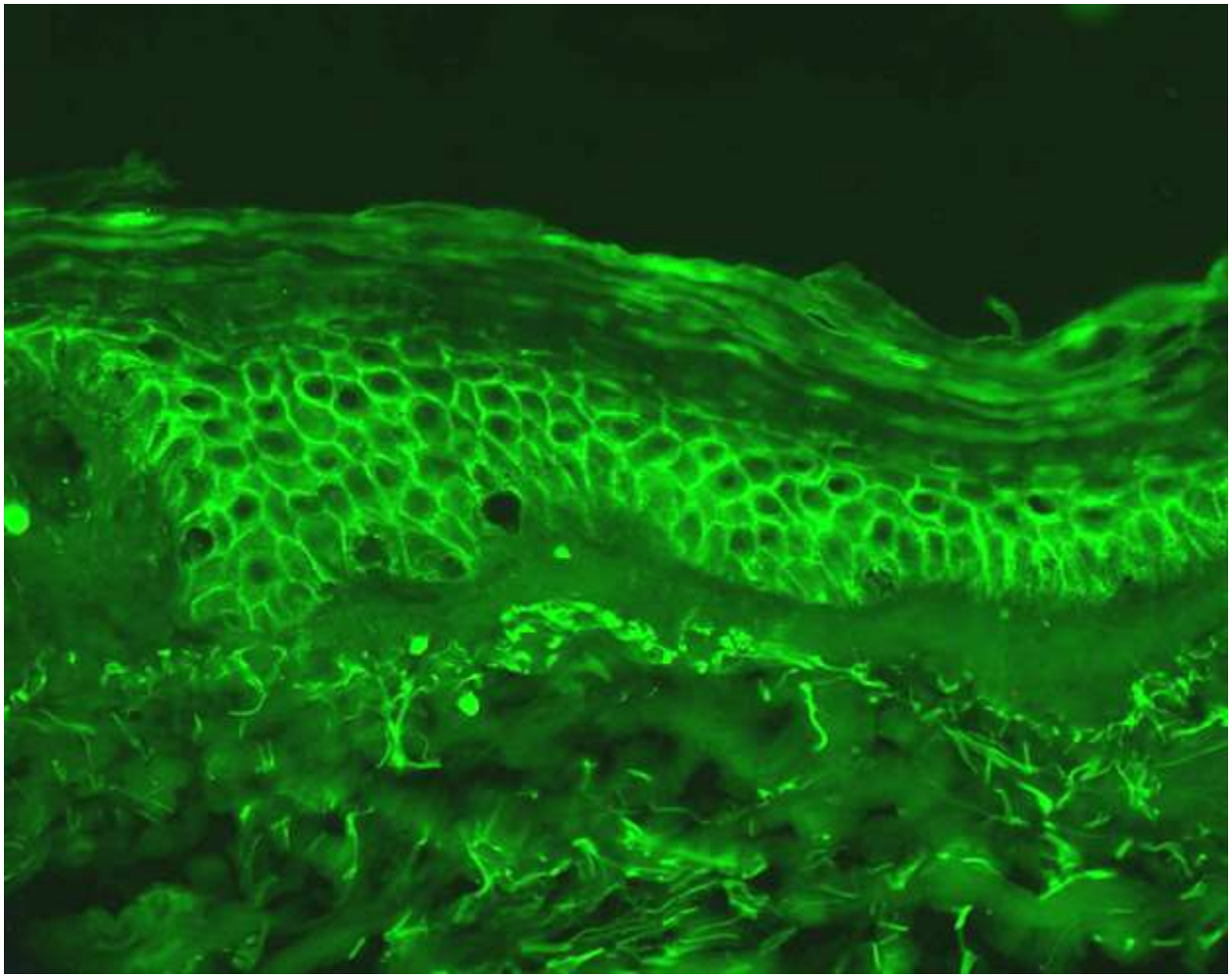


# Acanthocytes - Acantholysis:





**Deposition of IgG and Complement along the cell membrane, giving a Net appearance.**

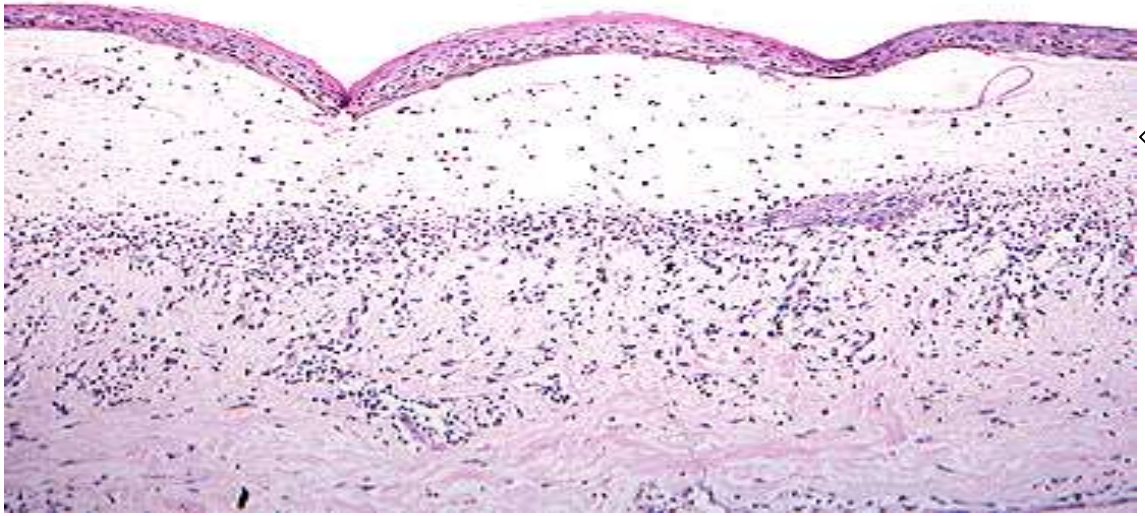


# ***Bullous Pemphigoid.***

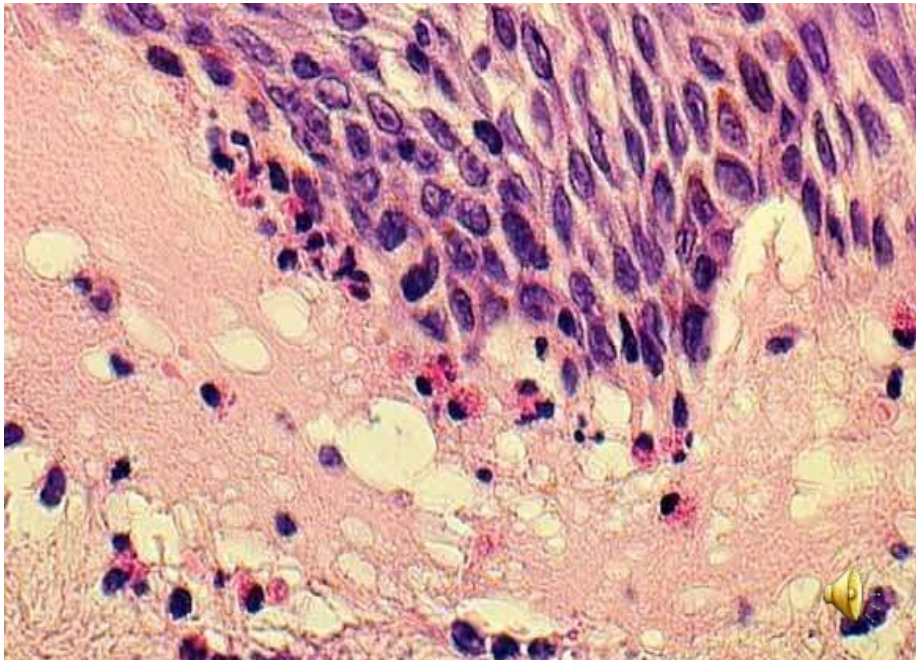
- Affects skin & commonly the mucous membranes (in 30% of cases).
- Characterized by subepidermal, nonacntholytic tense blisters.
- Also caused by type II hypersensitivity reaction(Ab against hemidesmosome)
- By Immunoflourscent shows linear deposits of Immunoglobulins along the basement membrane zone.

# BULLOUS PEMPHIGOID(Tensebulla

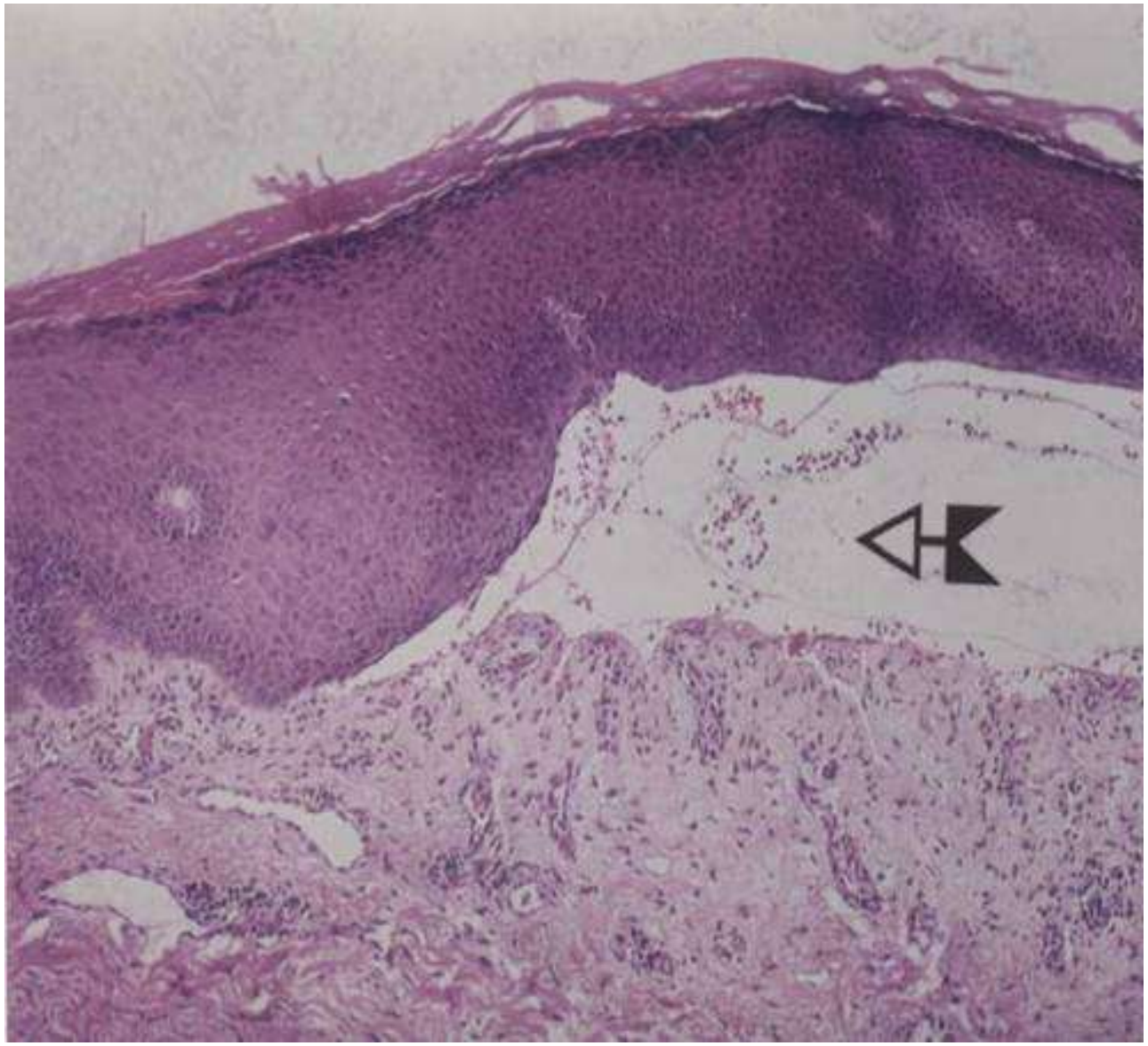

# BULLOUS PEMPHIGOID




Eosinophils at the DE junction.



# BULLOUS PEMPHIGOID - histology



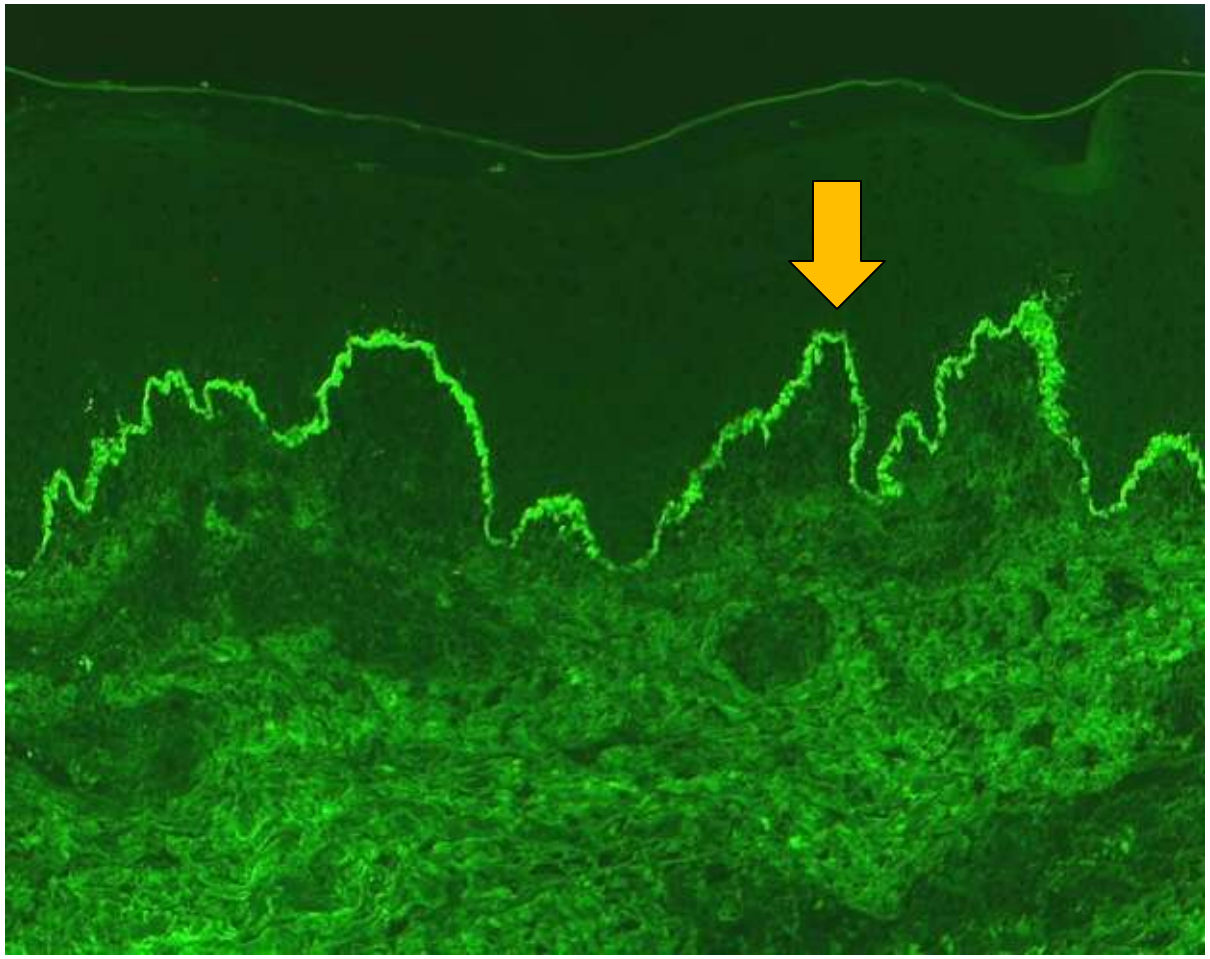
Subepidermal separation

Inflammation characterized by eosinophils

Intact epidermal layer.

# BULLOUS PEMPHIGOID

## IgG and complement staining pattern [ Linear Deposit ]



Antibody against bullous pemphigoid antigen in basement membrane causing subepidermal separation Type II hypersensitivity reaction.

# ***Dermatitis heptiformis***

- Affects male > female, at 3rd- 4th decades of life.
- In 10%- 20% of cases associated with celiac disease and respond to glutean free diet
- Pathogensis: IgA Anti-gluten Ab cross react with basement membrane proteins.
- Clinically: bilateral symmetrical urticarial plaque and vesicles on extensor surfaces , elbows knees upper back
- Vesicles are frequently grouped as are those of true herpes virus therefor called "herptiformis"
- **Mic.**Characterized by subepidermal bullae.
- By immunoflourscent, there are granular deposits of IgA in the tips of dermal papillae.

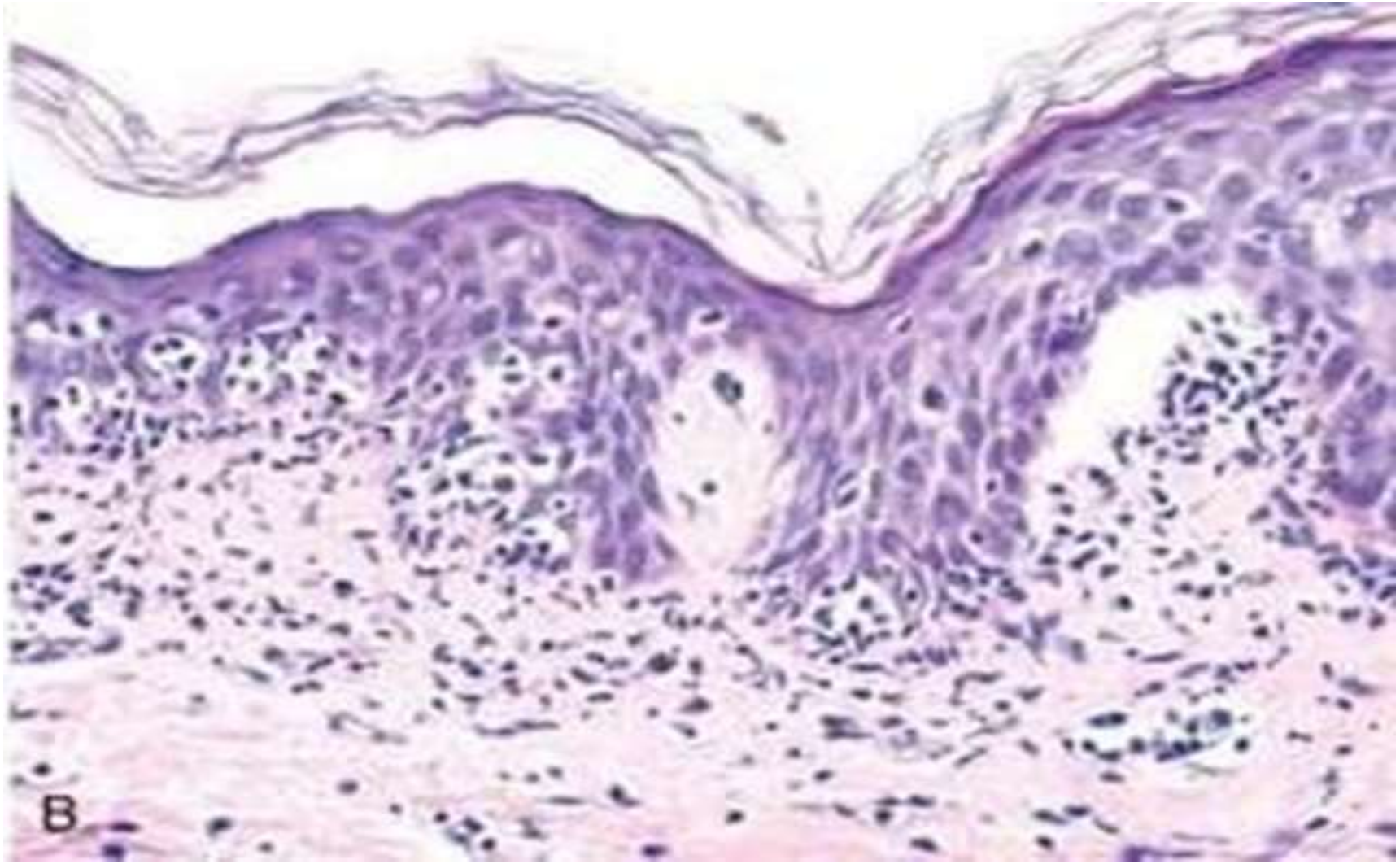
# Dermatitis Herpetiformis

- Extremely pruritic, small vesicles
- Associated with Celiac disease.
- IgA  Anti-gluten Ab cross react with basement membrane proteins.

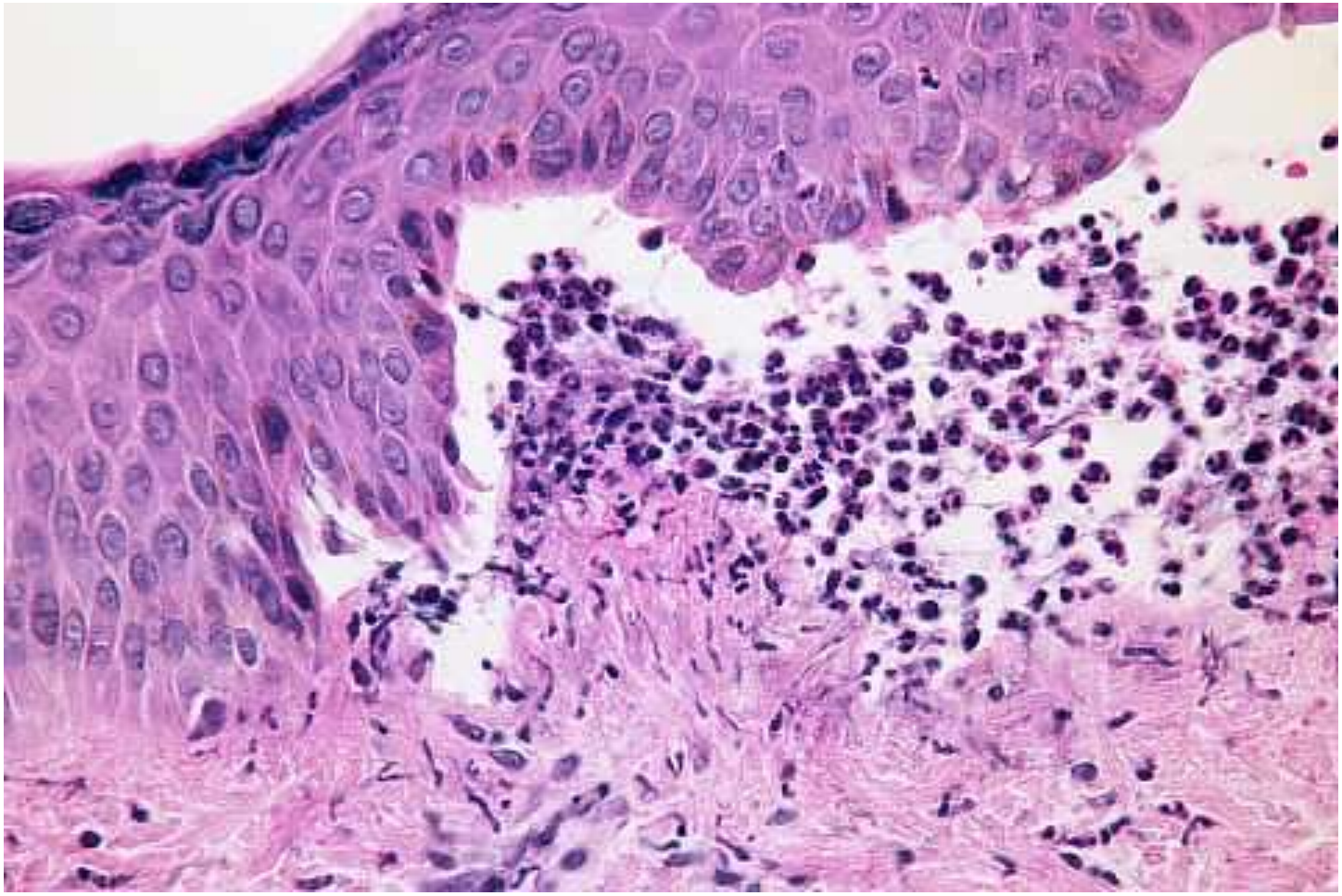
- Gross: Intense Itchy, small, erythematous, papules, small blisters in groups. (sub epithelial)

# Site; extensor surface  elbow, knees upper back.

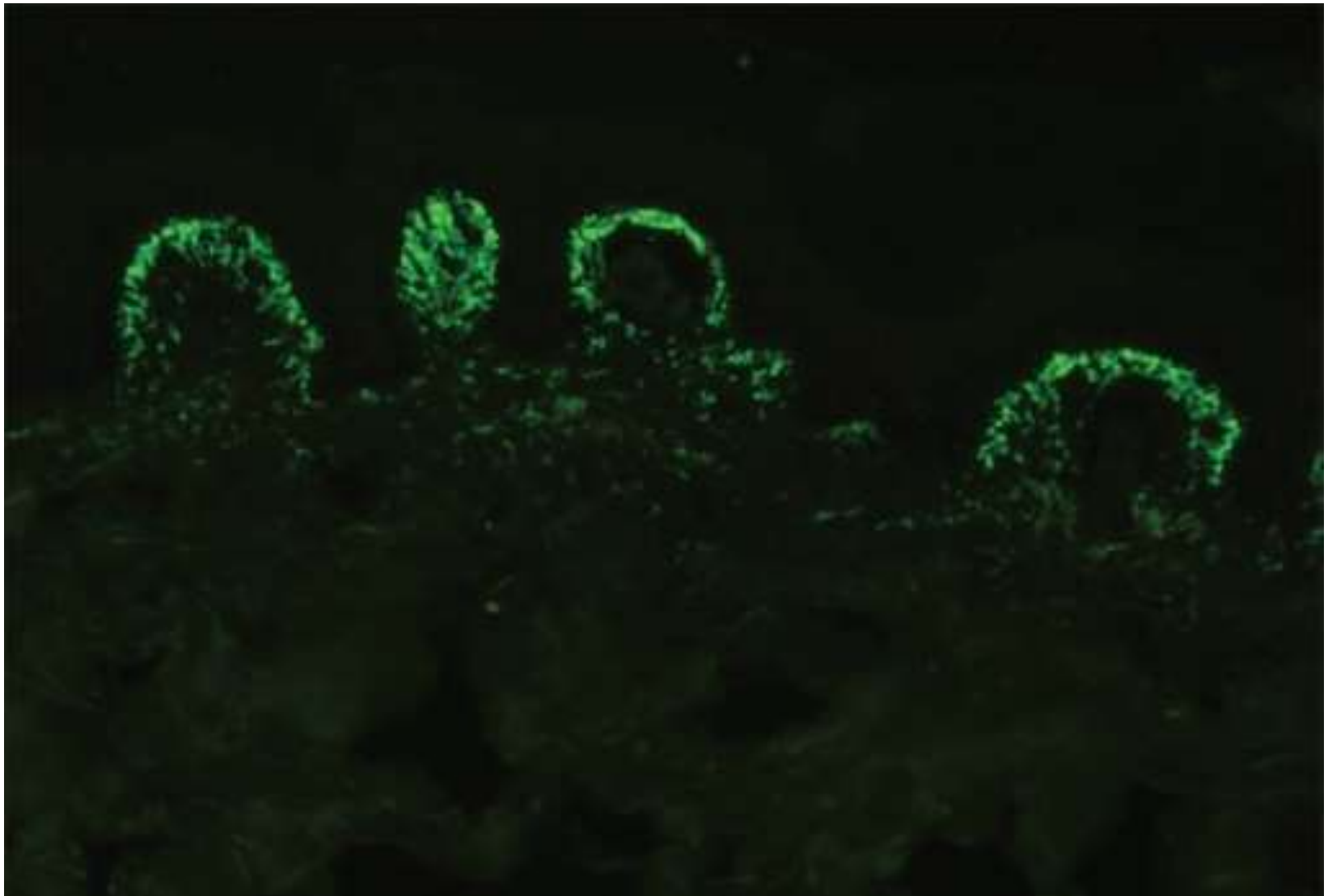

# Dermatitis Herpetiformis Micro: supepidermal, neutrophilic microabscesses in dermal papill



**The inflammatory cells within the vesicle are PMN's. Slight basophilia is seen in the partly necrotic dermal papilla.**



# Granular staining of dermal papillae with IgA in tip of dermal papillae



| Pemphigus vulgaris | Pemphegoid |
| --- | --- |
| Younger patient affected | Elderly are affected |
| Mucosal involvement: uncommon | Is common 1/3 of cases |
| Ab against desmosome | Ab against hemidesmosome |
| Intraepidermal (superficial ) blister | Subspidermal (deep blister |
| Blisters are flaccid and rupture easily | Blisters tense and firm |
| Acantholysis | No acantholysis |
| IF: net like IgG | Linear IgG |

# Dermatopathology

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1. Blistering Diseases
   - Pemphigus,
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   - Dermatitis herpetiformis.

5. Neoplastic:
   - Benign:
     - Nevi,
     - Malignant:
     - SCC,
   - • BCC,
     - Melanoma.

- **The most common benign skin tumor is nevus**
- **Microscopically It composed from:**

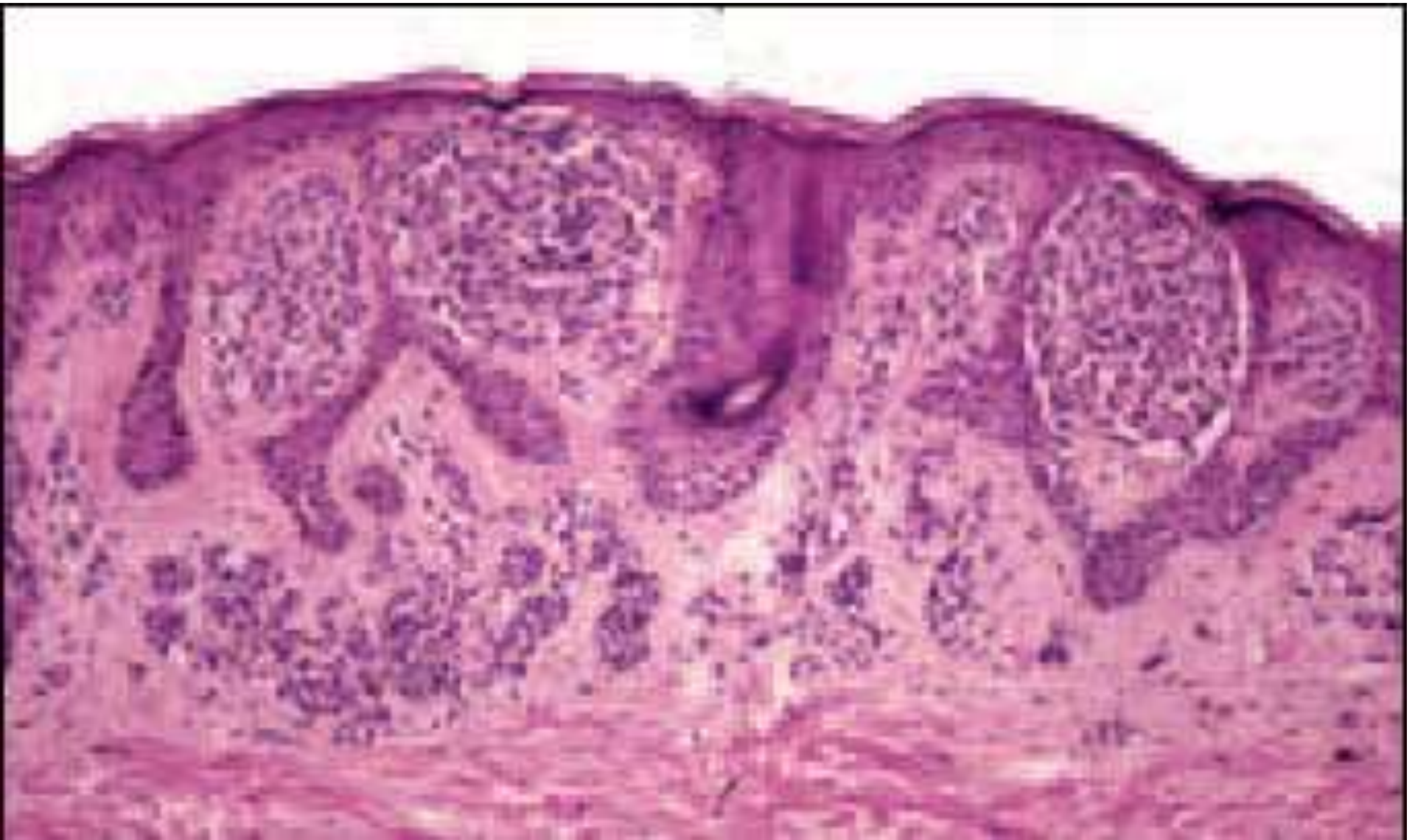
**round to oval cells that grow in nests along dermoepidermal junction (junctional nevi)**

- **that may grow into the underlying derms (compound nevus) &**
- **in older lesions only the dermal nests persist (pure dermal nevus).**

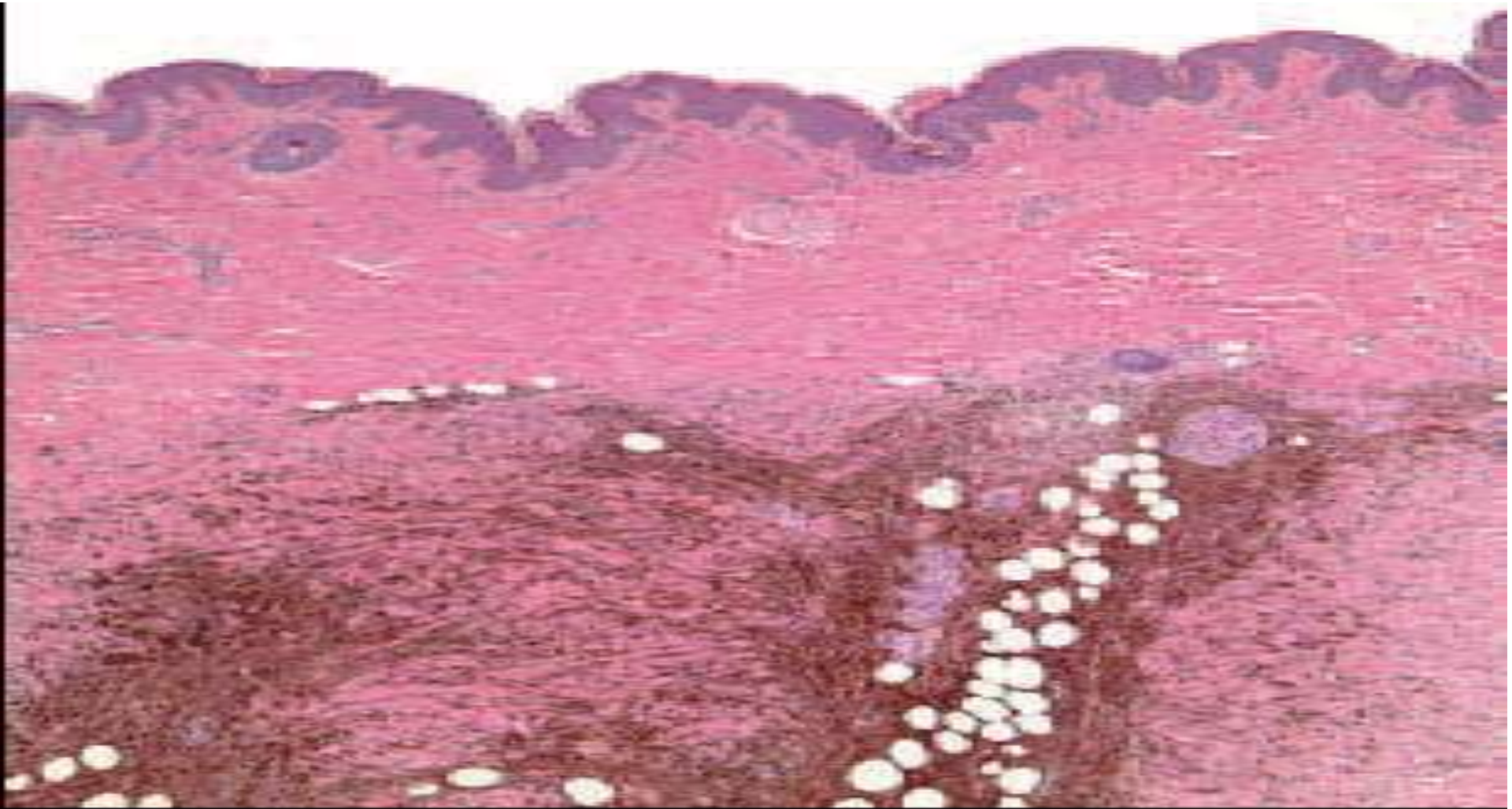
# Junctional Nevus:

- Small, flat, symmetric, uniform lesions.
- Cluster of melanocytes at DE junction. (arrow)

# (compound) melanocytic nevus



# Dermal Nevus



# Malignant Tumors of Skin

***Squamous Cell Carcinoma***

**basal cell carcinoma**

**Melanoma**

# ***Malignant tumors of skin***

## *1. Squamous cell carcinoma:*

### ***Etiology:***

1. Sunlight (ultraviolet).
2. Industrial carcinogens (tar, oils)
3. Chronic ulcers.
4. Sinus of chronic osteomyelitis
5. Old burn scars
6. Arsenic compounds
7. Ionizing radiation
8. Tobacco (squamous cells carcinoma)
9. Immunocompromised patients.
10. Xeroderma pigmentosum (defect in DNA repair gene)

### ***Gross:***

I. In situ carcinoma is usually sharply defined red plaques.
II. Invasive carcinoma is nodular lesion, sometimes ulcerate.

# *Clinical Features of SCC*

- invasive lesions are nodular, show variable scale, and may ulcerate

***Mic***:

I. In situ carcinoma: atypical malignant cells are involved the all levels of epidemis without break through the basement membrane.

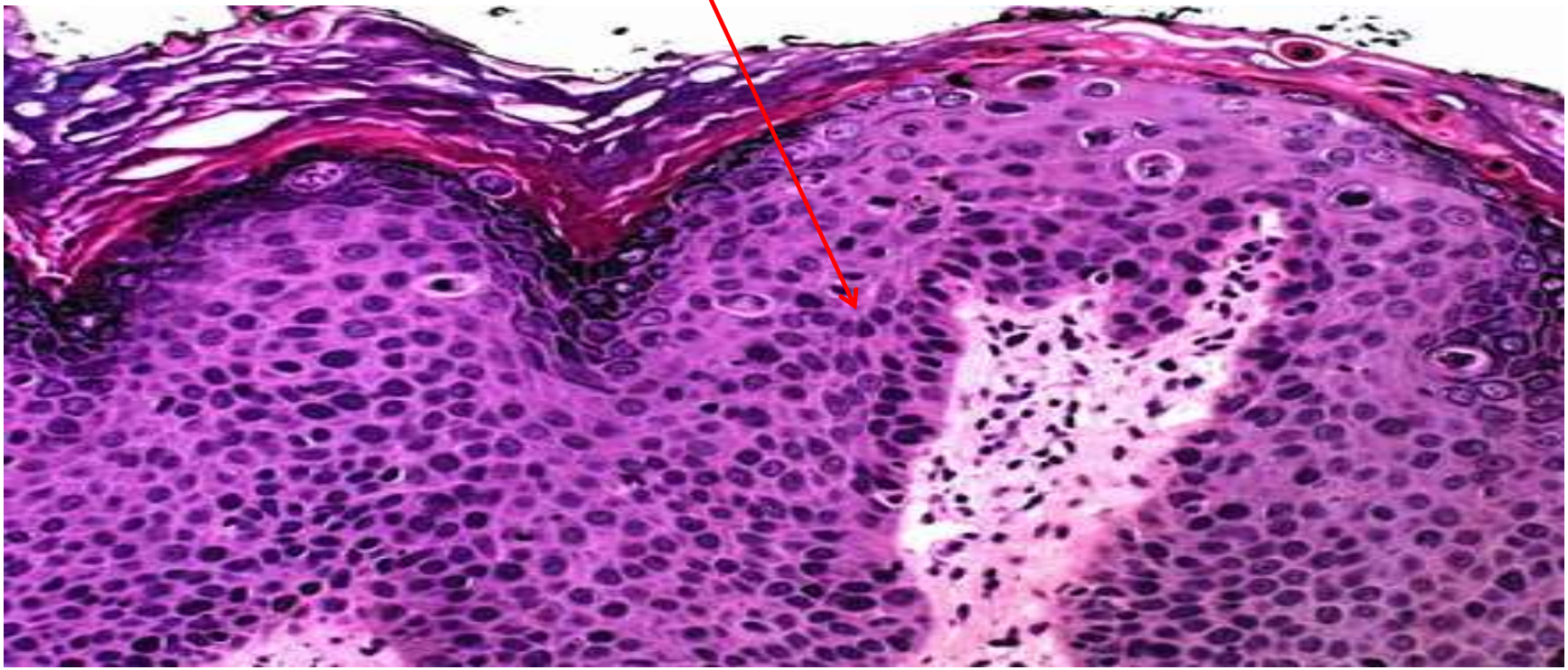
II. Invasive carcinoma: malignant cells are break through the basement membrane.

Invasive carcinomas show variable degrees of differentiation ranging from tumors well differentiated carcinoma which is formed by polygonal squamous cells arranged in orderly lobules that exhibit numerous areas of keratinization to a highly anaplastic carcinoma that is formed by rounded cells with many zones of necrosis & dyskeratosis.

.More aggressive than BCC and can metastasise if untreated. Less than 5% of squamous cells carcinoma shows metastases to regional lymph nodes

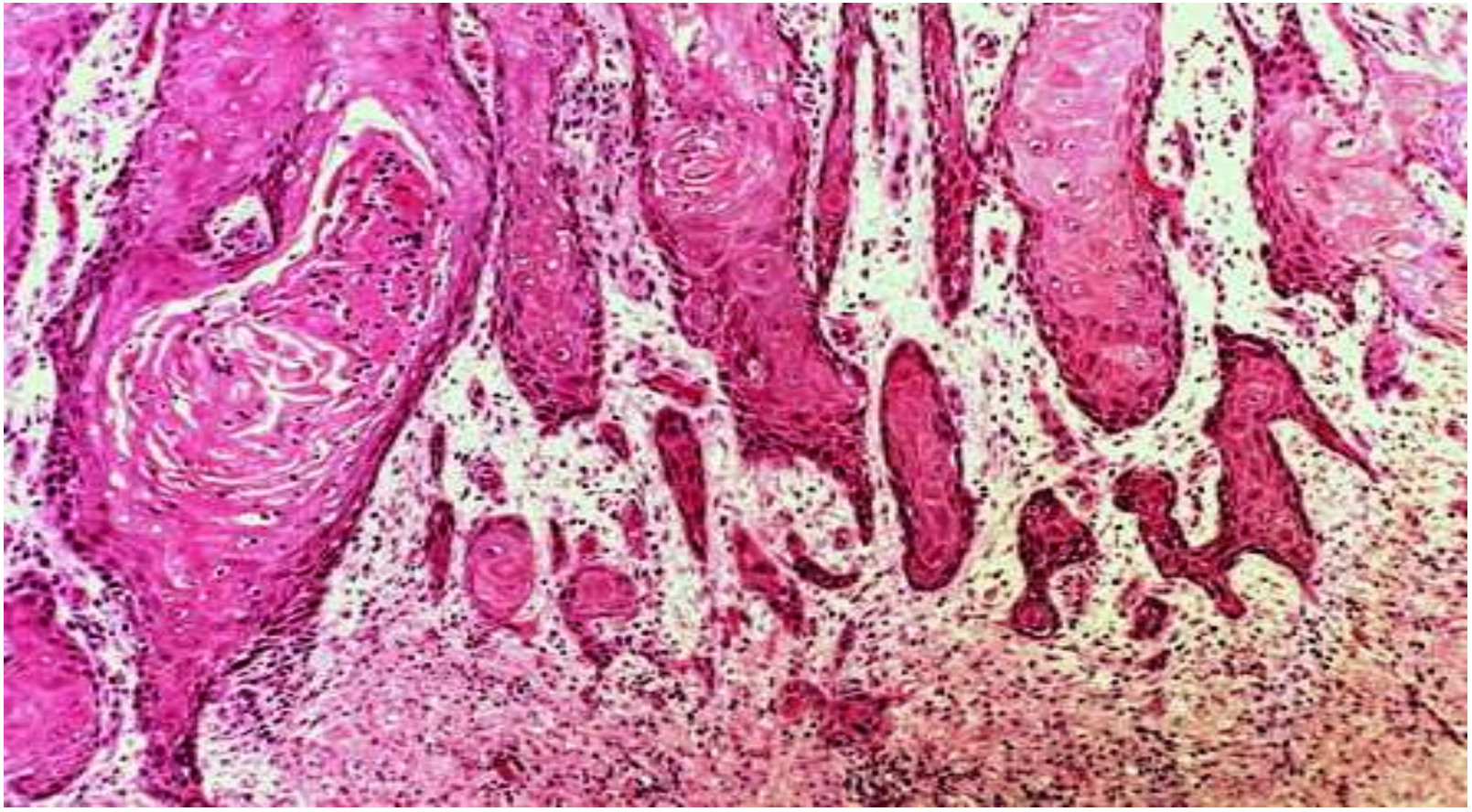
# Morphology of SCC

- Squamous cell carcinoma in situ is characterized by atypical cells at **all levels** of the epidermis, with nuclear crowding and disorganization.

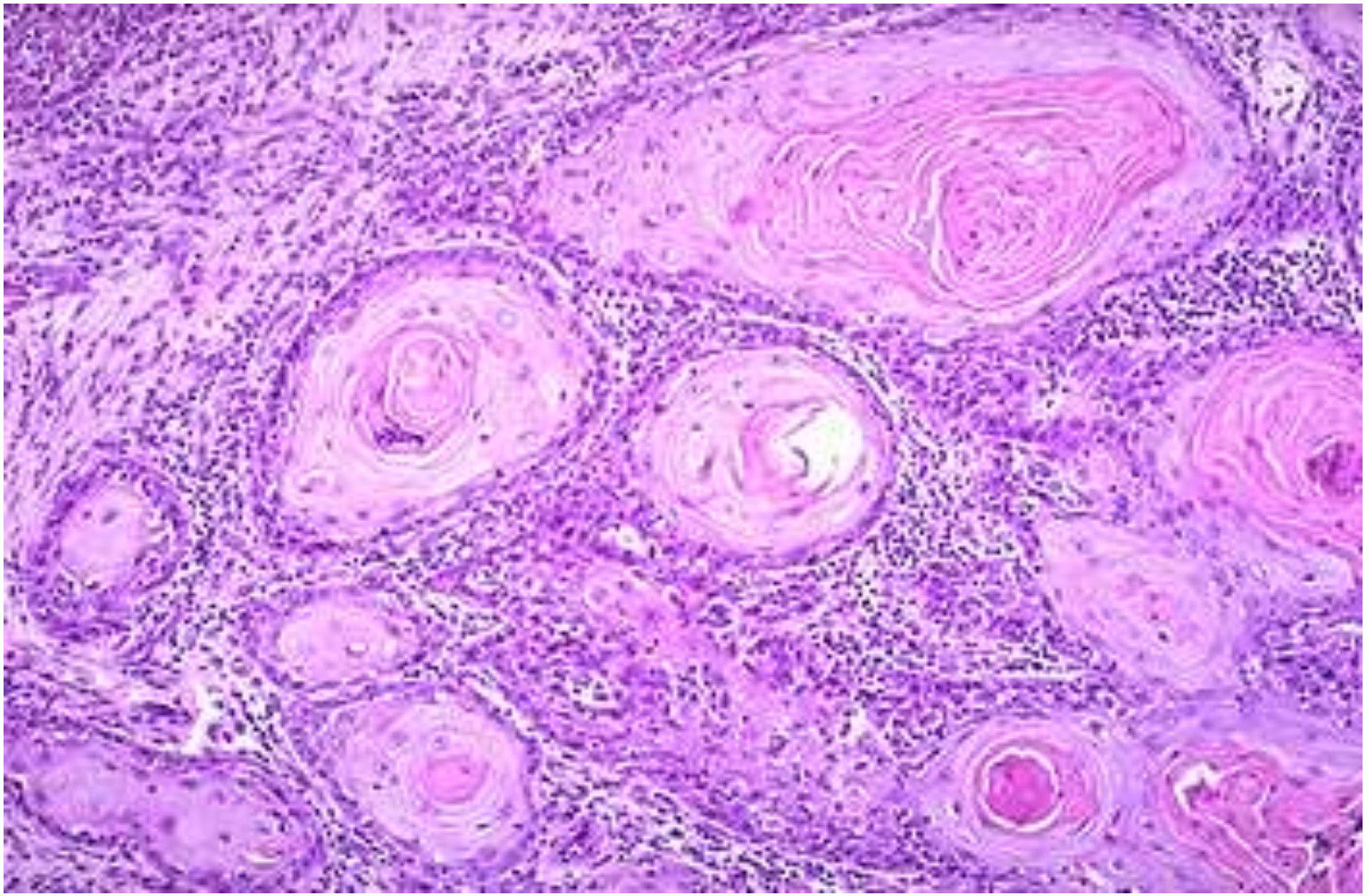


# Invasive Squmous cell carcinoma

- When these cells break through the basement membrane, the process has become invasive



- The neoplastic cells extend downward into the dermis forming keratin pearls.



# Tumors of Skin

*Squamous Cell Carcinoma*

# basal cell carcinoma

**Melanoma**

# 2. Basal cell carcinoma(BCC)

The most common tumor arising on the sun exposed sites in older people

slowly growing tumors that are locally invasive but rarely metastasizing.

Has the same etiology of Squamous cell carcinoma(chronic sun exposure and in lightly pigmented people).

- Increase risk: [same as squamous cell carcinoma]

1. immunosuppressed patients as a result of chemotherapy or organ transplantation,
- 2- xeroderma pigmentosum [inherited defects in DNA repair].

Gross: Pearly papules, often containing prominent, dilated subepidermal blood vessels.

Sometime contain melanin pigment (called pigmented Basal cell carcinoma).

Advanced lesions may ulcerate & extensively invade the local bone or facial sinuses (rodent ulcer).

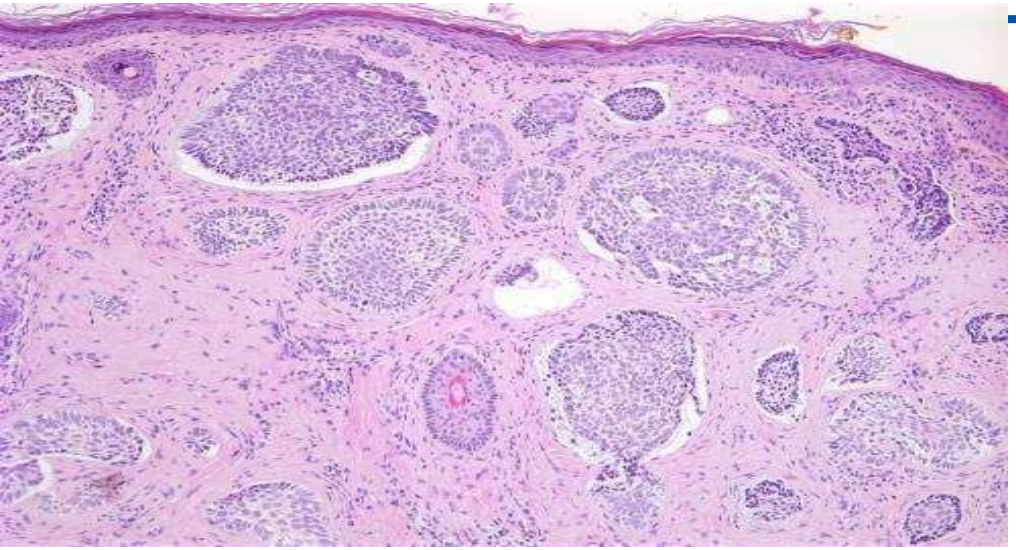
- Mic: tumor cells are resemble the normal basal cells of epidermis,
- with peripheral palisading,
- Clefting between tumor nest and stroma. Mucinous stroma

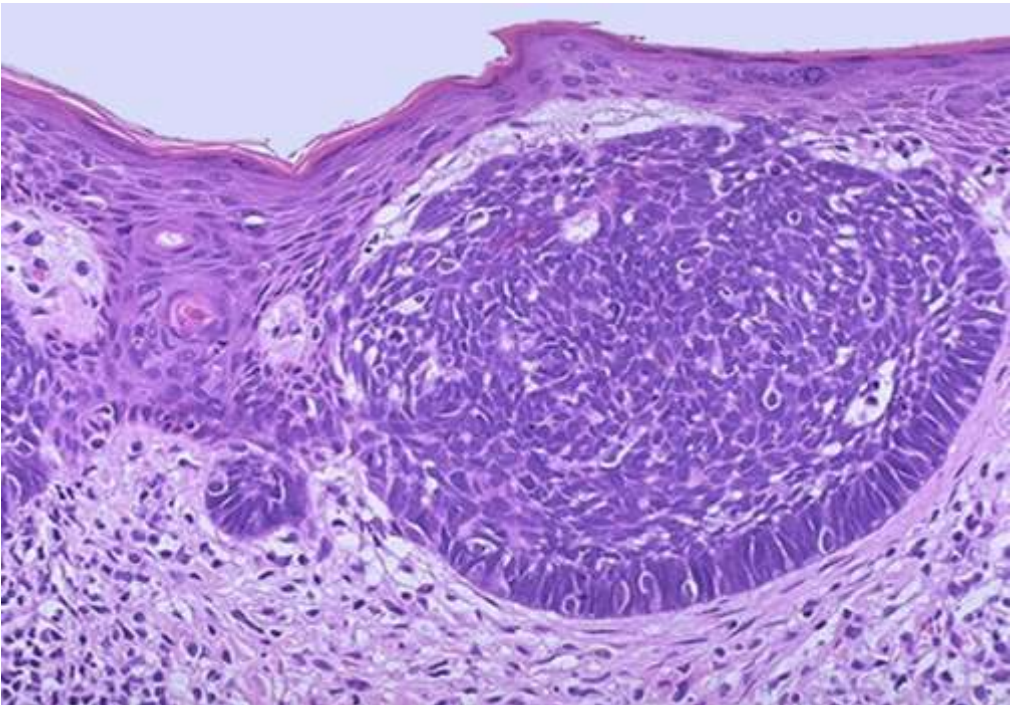
# *Clinical Features of BCC*

***Papule or nodule, often containing prominent, dilated subepidermal blood vessels (telangiectasia)***

**Rodent Ulcer-Ulcerated BCC**

# BASAL CELL CARCINOMA





nests of basaloid cells(small cell, scant basophilic cytoplasm)

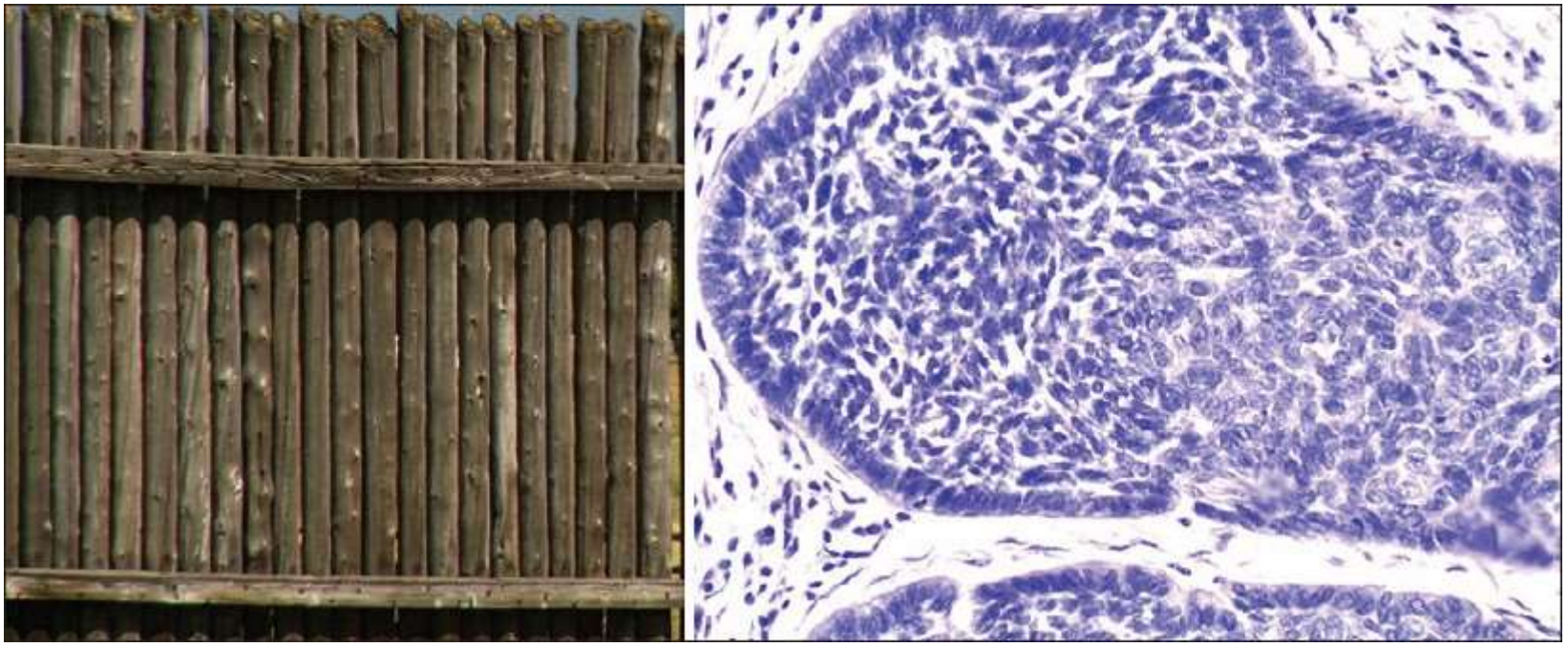
Hyperchromatic nuclei

Peripheral palisading(Peripheral tumor cell nuclei align in the outermost layer (palisading) with separation from the stroma

Clefting between tumor and stroma.

Mucinous stroma

# Peripheral palisading



# ***Malignant Melanoma*.**

Malignant tumor of melanocyte

Melanoma is less common, but much more deadly than basal cell carcinoma or squamous cell carcinoma

*Sites*: skin, oral cavity, anogenital areas, esophagus, meninges, & eyes.

## ***Etiology:***

1. Sunlight, More common in fair skinned persons
2. Preexisting nevus (dysplastic nevus)
3. Industrial carcinogens
4. Hereditary & familial factors

## ***Gross & clinical features:***

### ***Warning clinical signs of malignant melanoma; are***

1. Enlargement of preexisting mole
2. Itching & pain in preexisting mole
3. Development of new pigmented lesion during adult life
4. Irregularity of borders of pigmented lesion
5. Variegation of color within the pigmented lesion

# Melanoma

- sunlight plays an important role in the development of melanoma
- More common in fair skinned persons

***Mic***: there are two patterns of growth in malignant melanoma.

1. ***Radial pattern of growth***: represent the initial tendency of malignant melanoma to grow horizontally within the epidermis & superficial dermal layers, for long period of time, such pattern of growth have no tendency of metastasis & angiogenesis.
2. ***Vertical growth:*** with the time melanoma now grows downward into the deeper dermal layers as an expansile mass, with high tendency of metastasis & angiogenesis.

***Sites of metastasis:*** regional lymph nodes, liver, lung, brain, & heart.

## *Characteristics of melanomas cells:*

1. Melanoma cells are larger than cells of nevus
2. Malignant cells have large nuclei, with irregular contour, & clumped chromatin
3. Have prominent eosinophilic nucleoli
4. Cells grow either in nests or single.

# MELANOMA GROWTH PHASES

## Radial Growth Phase

## Vertical Growth Phase

# Clinical Criteria for Dx

## ABCDE Criteria

**A**symmetry of mole

**B**order irregularity

**C**olour variegation

**D**iameter > 6mm

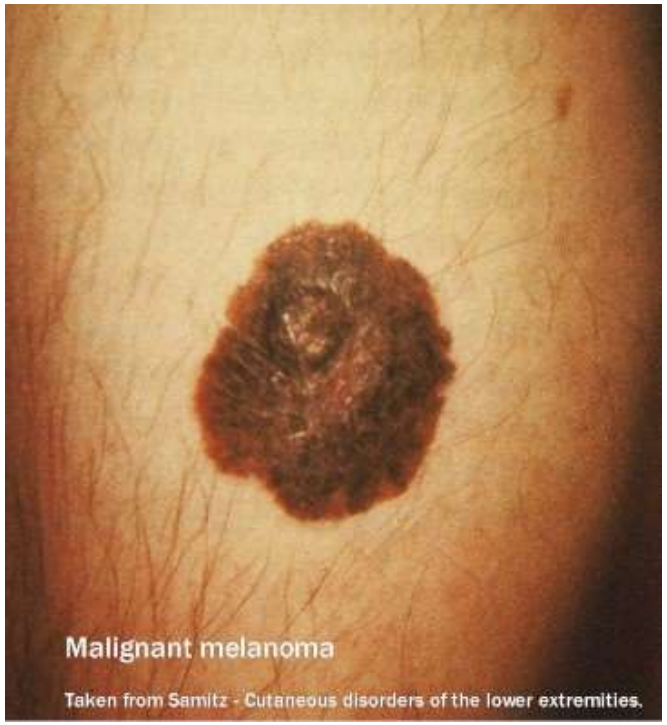
**E**levation

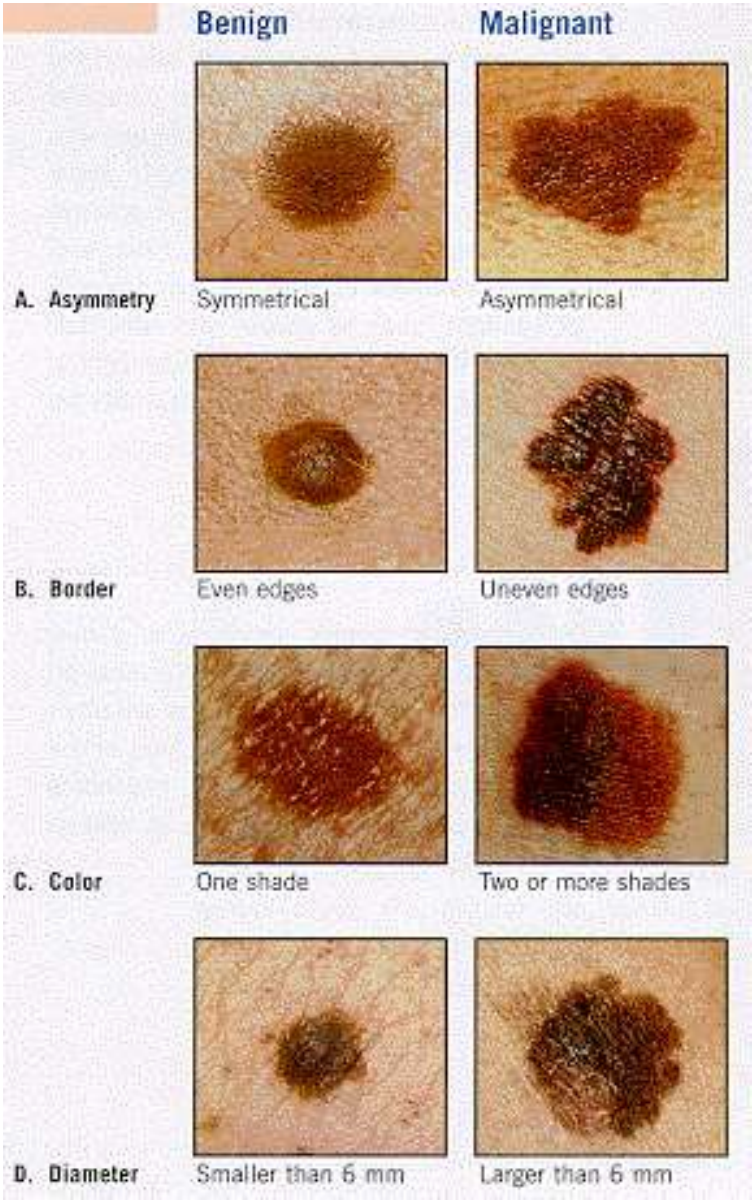


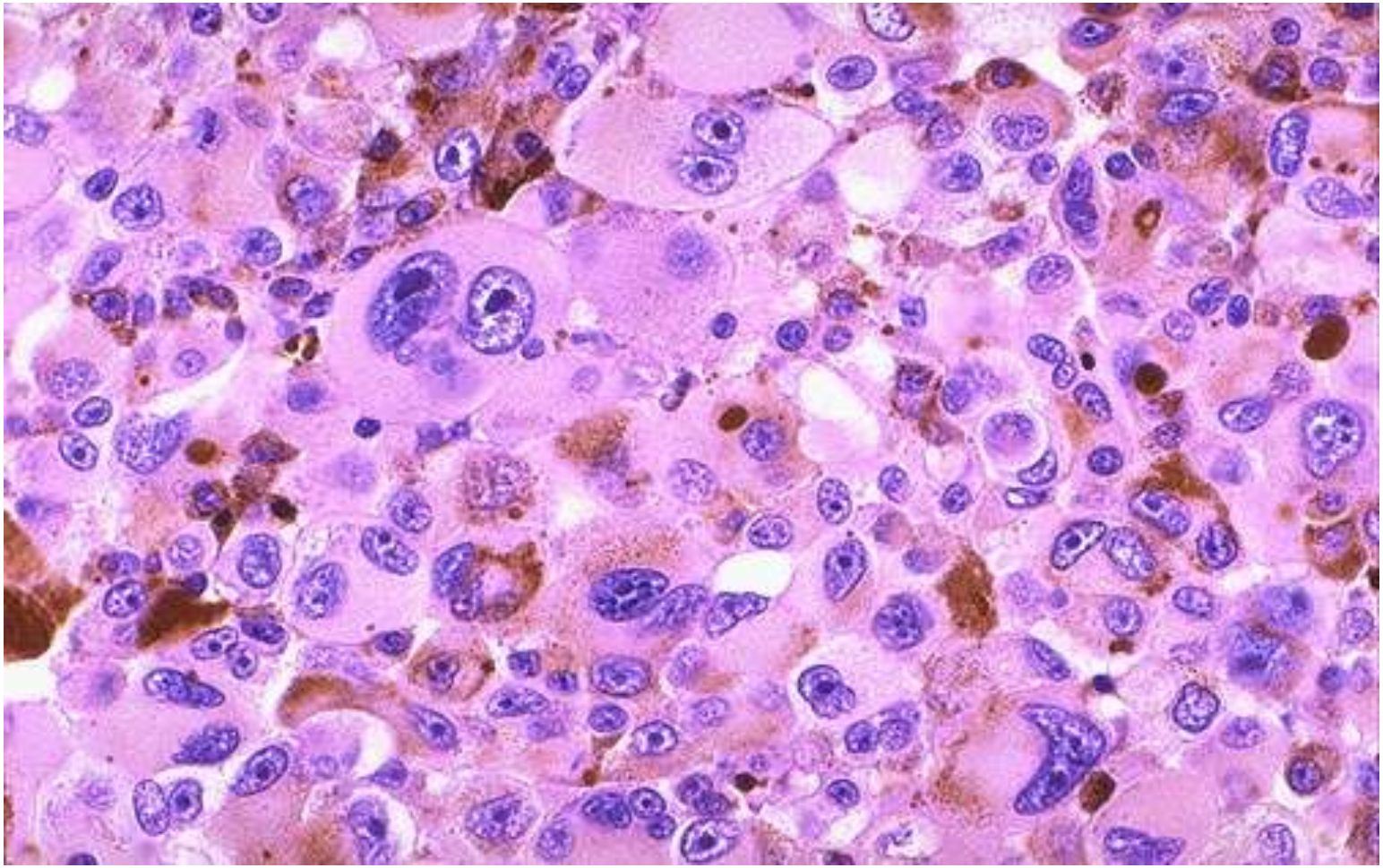
Melanoma Clinical Features: note **ABCD..**




Malignant Melanoma


Malignant melanoma
Taken from Samitz - Cutaneous disorders of the lower extremities.


Benign
Malignant
A. Asymmetry
Symmetrical
Asymmetrical
B. Border
Even edges
Uneven edges
C. Color
One shade
Two or more shades
D. Diameter
Smaller than 6 mm
Larger than 6 mm



- **the microscopic appearance of a malignant melanoma:**
- **Large polygonal cells have very pleomorphic nuclei which contain prominent eosinophilic nucleoli.**
- **The neoplasm is making brown melanin pigment.**