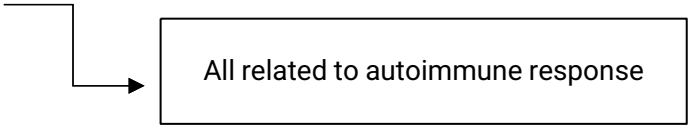
Pathology lecture 5

3 CHRONIC INFLAMMATORY DERMATOSES

- The skin surface in some chronic inflammatory dermatoses is roughened as a result of excessive or abnormal scale formation and shedding (desquamation).

1. Psoriasis
2. Lichen Planus
3. Lichen Simplex Chronicus



psoriasis

-a T cell-mediated inflammatory disease

- autoimmune in origin

-most common location is the skin of elbow and knees along with gluteal

-symmetrical

-prominent parakeratotic scale with infiltrating neutrophils

- Erythematous plaque salmon like covered by loosely adherent silver-white scale

-no granular layer

Lichen Planus

- Autoimmune disease

- symmetrically distributed

- particularly on the extremities.

- Approximately 70% of cases also involve the oral mucosa

- result from a CD8+ T cell–mediated cytotoxic response against antigens in the basal cell layer and the dermoepidermal junction that are produced by unknown mechanisms.

- lichen means : bandlike infiltrate of lymphocytes along the dermoepidermal junction

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

Lichen Simplex Chronicus

-response to local repetitive trauma

-induces epithelial hyperplasia and eventual dermal scarring

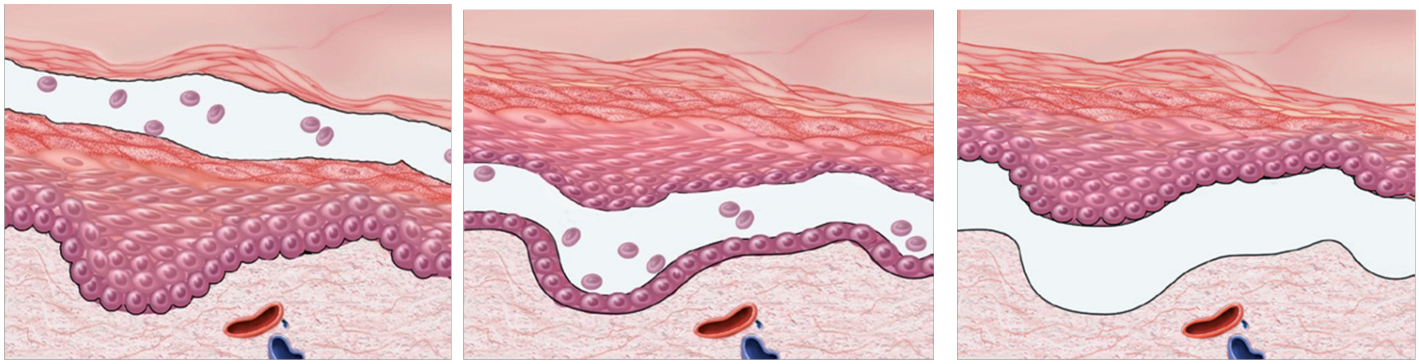
BLISTERING (BULLOUS) DISORDERS

- Blistering in these diseases tends to occur at specific levels within the skin, a morphologic distinction

1-Pemphigus (Vulgaris and Foliaceus)

-an uncommon autoimmune blistering disorder resulting from loss of normal intercellular attachments within the epidermis and the squamous mucosal epithelium

Level of epidermal separation forms the basis of differential diagnosis for blistering disorders.



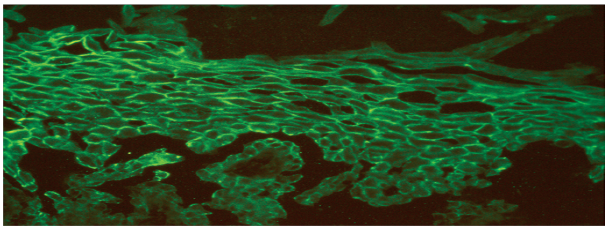
A-Subcorneal **B-Suprabasal** **C-Subepidermal**

-Autoimmune diseases caused by: Antibody mediated hypersensitivity reactions. (Type II)

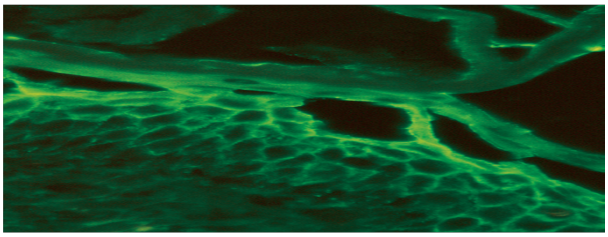
-IgG autoantibodies: Bind to intercellular desmosomal proteins of skin

- **Diagnosis: Direct immunofluorescence study: fishnet-like pattern of intercellular IgG deposits.**

Pemphigus vulgaris:
Uniform deposition of Ig (green) along cell membrane of keratinocytes (fishnet pattern).



Pemphigus foliaceus:
Ig deposits confined to superficial layers of epidermis.



-Acantholysis (separation)

-Pemphigus vulgaris: Suprabasal blister.

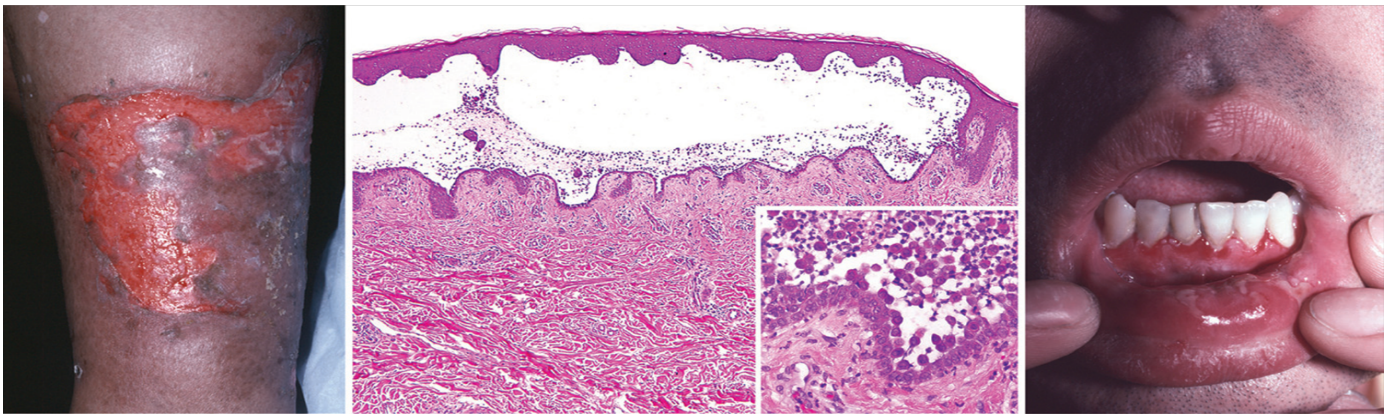
-Pemphigus foliaceus: Subcorneal

1-Pemphigus vulgaris:

- Most common type.
- Involves both mucosa

Lesions:
Superficial vesicles & bullae, rupture easily, leaving deep & extensive erosions covered with serum crust. Easly to rupture

Pemphigus vulgaris:
Erosion on leg:Group of confluent, unroofed blisters.
Suprabasal acantholysis results in intraepidermal blister.

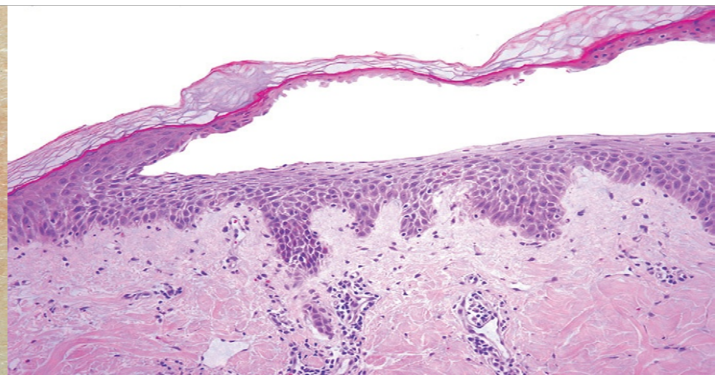


2-Pemphigus foliaceus:

- Mostly in face and extremities
- Rare in oral mucous.
- Infrequent involvement of mucous membranes.
- Blisters are superficial with more limited zones of erythema

Pemphigus foliaceus:

Gross appearance of typical blister, less severely eroded than those seen in pemphigus vulgaris.
Microscopic: Characteristic subcorneal blister.

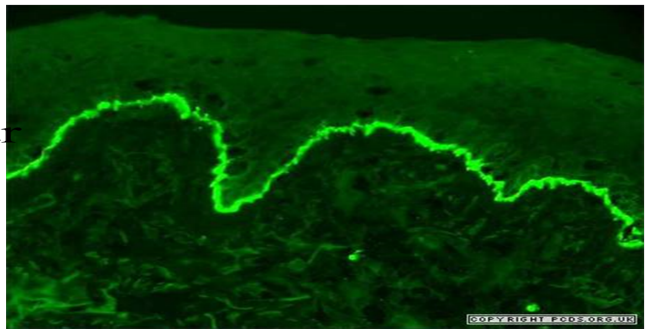


2-Bullous pemphigoid:

- Pathogenesis

Blistering is triggered by linear deposition of IgG antibodies in epidermal basement membrane.

Deposition of IgG antibody detected by direct immunofluorescence as linear band outlining the subepidermal basement membrane zone.

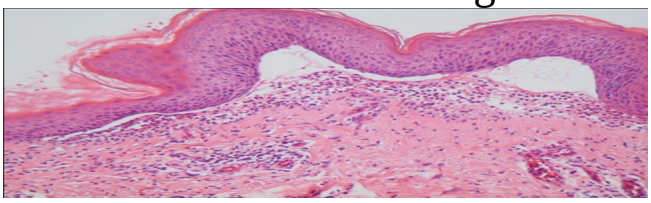


Morphology: Grossly:

- Tense bullae filled with clear fluid.
- Subepidermal nonacantholytic blisters.

- Perivascular infiltrate of lymphocytes & eosinophils.
- Superficial dermal edema.

- Basal cell vacuolization gives rise to



fluid-filled blister.

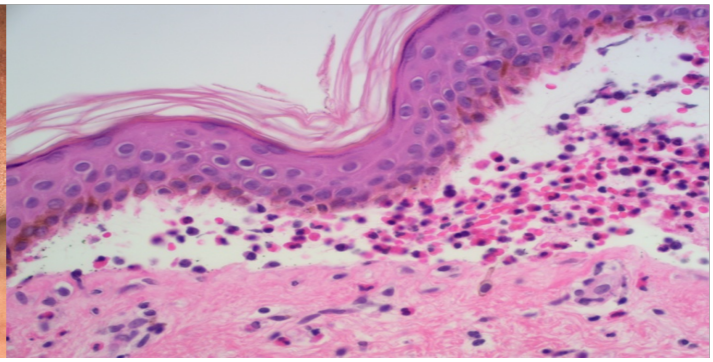
- Blister roof consists of full thickness with intact intercellular junctions so epidermis not rupture easily.

(Key distinction from blisters in pemphigus)

Bullous pemphigoid.

Gross appearance of tense, fluid filled blisters. Like burn

Subepidermal vesicle with inflammatory infiltrate rich in eosinophils.



SKIN TUMORS

غير مطلوب

1- Squamous Cell Carcinoma

-common tumor

-arises on sun-exposed sites in older adult

-mainly caused by UV light exposure, which leads to widespread DNA damage and extremely high mutational loads

-arising at internal sites (oropharynx, lung, esophagus, anus).

2- Basal Cell Carcinoma

-common slow-growing cancer that rarely metastasizes

- loss-of function mutations in PTCH1

- composed of nests of basaloid cells in the periphrey.

-The cleft between the tumor cells and the stroma is a highly characteristic artifact of sectioning

غير مطلوب

Melanocytic nevi

-benign neoplasms

-caused by somatic gain-of-function mutations in BRAF or RAS.

junctional nevi: epidermis

compound nevi: epidermis +dermis

intradermal nevi: deep dermis

-brownish in color

-uniformly pigmented

-uniform border

- Early lesions are composed of round to oval cells that grow in “nests” along the dermoepidermal junction

-Nuclei are uniform and round, and contain inconspicuous nucleoli with little or no mitotic activity

3-Melanoma

-less common

-more aggressive in the skin

- caused by UV light–induced DNA damage that leads to the stepwise acquisition of driver mutations

- initiating event appears to be an activating mutation in BRAF or (less commonly) RAS

-lymphocytic infiltrate

-The main clinical warning signs are as follows:

1. Rapid enlargement of a preexisting nevus
2. Itching or pain in a lesion
3. Development of a new pigmented lesion during adult life
4. Irregularity of the borders of a pigmented lesion
5. Variegation of color within a pigmented lesion

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