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Pathology #5

مجموعة التفريغ السريع

MSS

Subject: *Skin Pathology*

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Date: *Tuesday, October 08, 2013*

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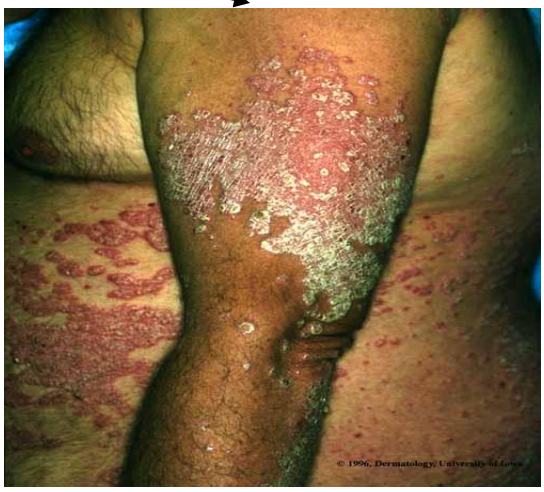
بسم الله الرحمن الرحيم

MACROSCOPIC TERMS

- **MACULE**
- **A circumscribed flat lesion of up to 5 mm in diameter, usually distinguished from surrounding skin by its coloration.**

- **PATCH**
- **A circumscribed flat lesion >5 mm in diameter, usually distinguished from surrounding skin by its coloration, sometimes (not always) it's seen with the macule .**The figure next

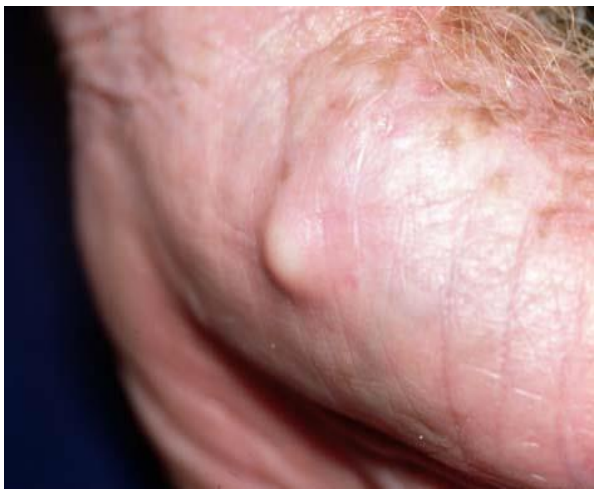


- **PAPULE**
- **Elevated dome-shaped or flat-topped (**its top is flat**) lesion 5 mm or less across.**

- **PLAQUE**
- **Elevated flat-topped lesion usually greater than 5 mm across (coalescent papules)**

- **NODULE**
- **Elevated lesion with spherical contour greater than 5 mm across.**

- **VESICLE**
- **Fluid-filled raised lesion 5 mm or less across.**



- **BULLA**
- **Fluid-filled lesion greater than 5 mm across.**

- **BLISTER**
- **Common term used for vesicles and bullae. (see slides)**

- **PUSTULE**
- **Named If any of the previous is infected → Discrete, pus-filled, raised lesion.**

MICROSCOPICAL TERMS

HYPERKERATOSIS:

Thickening (hyperplasia) of the stratum corneum with keratin.

PARAKERATOSIS:

Presence of the nuclei in the stratum corneum (Normally there is no nuclei).

Note: Parakeratosis on squamous mucosal membranes (such as buccal mucosa) is normal.

ACANTHOSIS:

Diffuse epidermal hyperplasia.

PAPILLOMATOSIS:

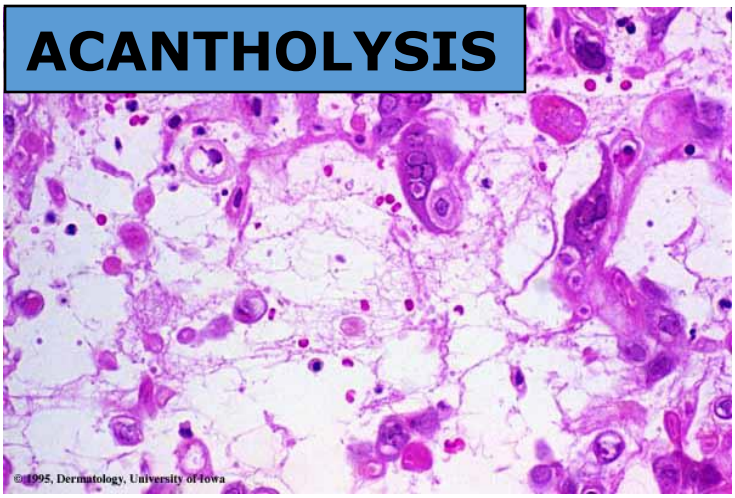
Surface elevation caused by hyperplasia and enlargement of contiguous dermal papillae.

DYSKERATOSIS: Abnormal keratinization occurring prematurely within individual cells or groups of cells below the stratum granulosum.

Note: Keratin is normally produced within keratinocytes and accumulate on the surface but in dyskeratosis keratin is accumulated within keratinocytes themselves ☺

ACANTHOLYSIS: Loss of intercellular connections (such as desmosomes**) resulting in loss of cohesion between keratinocytes.**

Spongiosis: Edema of epidermis.



Infectious Dermatoses

Has many types:

A) Bacterial Infections

-It's acute. And the commonest type is called Impetigo.

-Impetigo is a Common superficial bacterial infection of the skin.

-It's commonly caused by Staphylococcus aureus. Sometimes it can be caused by Beta-hemolytic streptococci

-It will result in a Spongiotic epidermis with heavy neutrophil infiltrate that means it's acute infection.

B) Viral Infections

-It's called Verrucae (warts / ثواليل)

-It's a common lesion which can happen at any age but it's most common in children and adolescents.

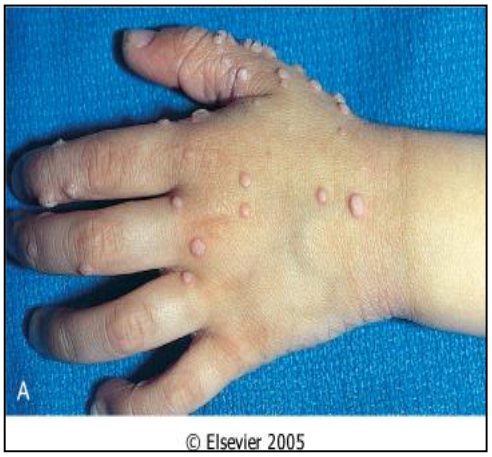
-It's caused by human papillomaviruses (the low risk types 6 &11)

(there are other severe types, which can cause squamous cell carcinoma.)

-Generally it's self-limited and have spontaneous regression but sometimes it needs a treatment by cauterization (الكوي) or excision.

-And there are many types of viral infection; for e.g. we have:

- **Verruca vulgaris**
- **Verruca plana**
- **Verruca plantaris**
- **Verruca palmaris**



- biggest type is called Condylomaacuminatum (venereal wart) which could happen with venereal diseases. And the big difference from other types is that it could recur and rarely it could be malignant.

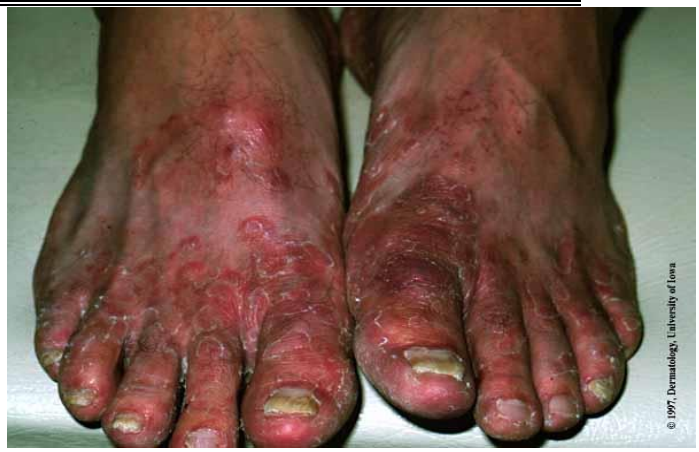
We can test the presence of virus by immunohistochemistry.

C- Fungal Infections

Is 2 types:

1) Superficial Skin Infection :

It's Caused primarily by dermatophytes mainly the tinea group;



⇨Tinea capitis(hair shaft)

⇨ Tinea corporis (arms & legs)

⇨Tinea pedis (athlete's foot)

-We usually test for the presence of fungi by **PAS** (periodic acid-schiff) satin.

The spread to or primary infection of the nails leading to discoloration of it is referred to as onychomycosis.

2) **Mucosal & systemic (deep) : caused by Candida especially in immunocompromised patients**.

D- Arthropod bites & stings

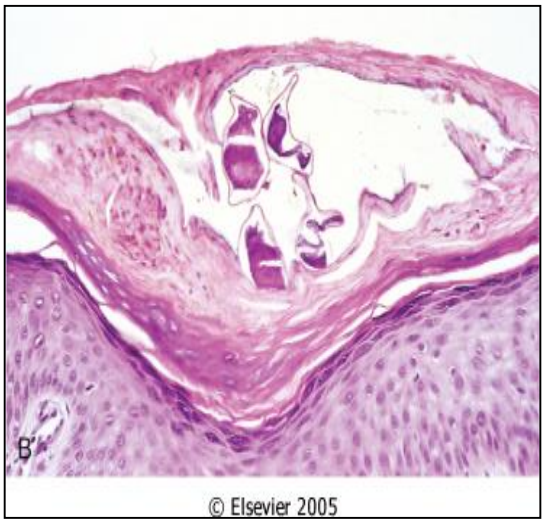
Arthropods can produce lesions by several means:

⇨ **By direct irritant effects of insect parts impeded in the tissue or secretions.**

⇨ **By immediate or delayed hypersensitivity reactions (including an anaphylactic shock) like what bees do.**

⇨ **By specific venoms.**

⇨ **By serving as vectors for secondary invaders for e.g. it can carry a virus, bacteria or even a parasite.**



- **In the picture, inside the corneal layer there is a group of parts of insects. It can be along the hair such as the flea (البرغوث) causing irritation in that area.**

Acute Inflammatory Dermatoses

▶Urticaria

-Common Localized mast cell degranulation resulting in dermal microvascular hyperpermeability Causing tranzient pruritic ' Wheels' (few hours).

-Results from antigen induced release of mediators from mast cells after specific sensitization to food, pollen, drugs ….etc

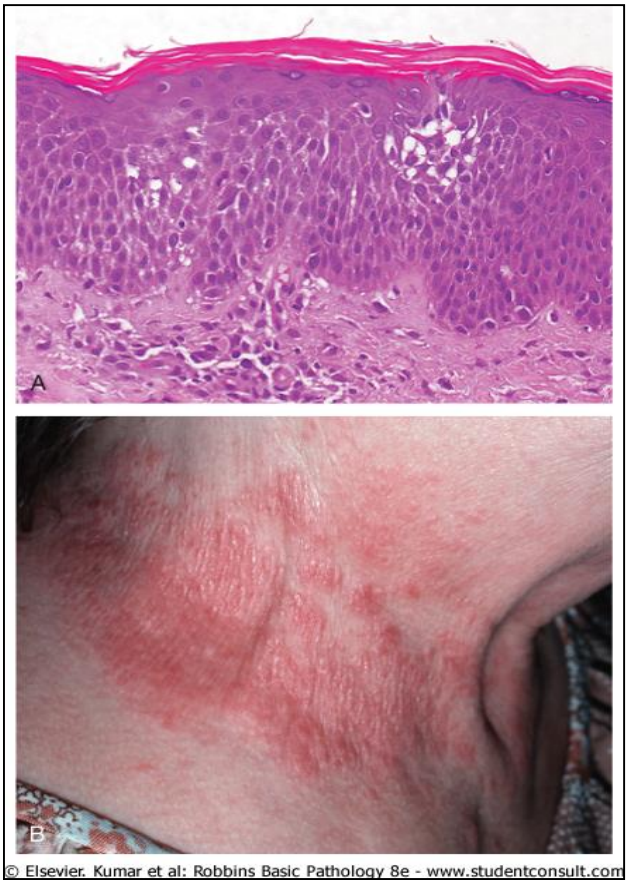
-Morphologically it will appear almost normal but with edema, minimal mononuclear cells with or without eosinophils.

▶ Acute Eczematous dermatitis

Eczema is a clinical term of pathogenetically different conditions. All of these conditions are characterized by papulovesicular, oozing, crusted lesions, may later develop into raised, scaly plaques.

It can be classified into:

1) **Allergic contact dermatitis; like what the sweat under the watch do**
2) **Atopic dermatitis.**
3) **Drug-related dermatitis.**
4) **Photoeczematous dermatitis; (the sun effect)**
5) **Primary irritant dermatitis**



Morphologically :

The main feature in Eczematous dermatitis is Spongiosis which is the accumulation of edema within the epidermis.
We also have superficial perivascular infiltrate and in some cases, eosinophils may be present.

Lesions are pruritic, edematous containing vesicles and bullae.

▶ Erythema Multiforme

- It is usually self-limiting hypersensitivity reaction to some infections , drugs and sometimes the cause is unknown.

- It's very variable with many shapes.

- Erythema multiforme causes variable erythematous lesions (sometimes with vesicles), perivascular inflammation and dermal edema. It may also cause degeneration of keratinocytes with (sometimes) epidermal necrosis in severe cases .

- It can accompany other diseases.

Chronic Inflammatory Dermatoses

- **Many types , unknown cause.**

- **Examples: Psoriasis and lichen planus diseases .**

Psoriasis :

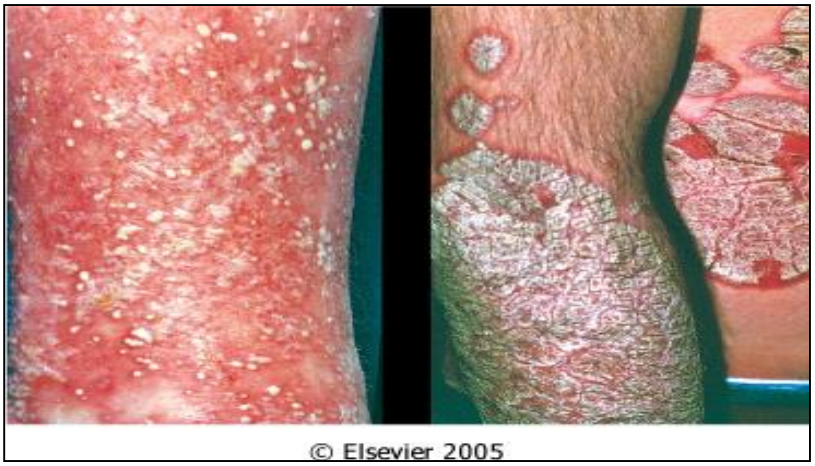
- Common scaly dermatosis, sometimes associated with arthritis, myopathy & enteropathy. It is thought that it can be Immunologically mediated but the mechanism of pathogenesis isn't always very well-defined .

- Affects the extensors surfaces, which means the skin of elbows, knees, scalp and sometimes glans penis.

- Typical lesion is a well demarcated pink plaque covered by loosely adherent scales (easily detached , when they detach you will get oozing blood vessels).

- **Nail changes occur in 30% of cases in the form of yellowish brown discoloration , and sometimes may be very bad so that the nail is detached .**

- **Here you can see these patches, they look silvery whitish, all of these produce scales which are chronic and very disturbing.**



- Main Features :

***Parakeratosis, mild hyperkeratosis.**

***Loss of granular layer in the epidermis of the skin (very typical feature).**

***Epidermal hyperplasia (Acanthosis) :it also has rete ridges thickening , broadening and sometimes fusion of more than one.**

***The inflammatory infiltrate which is usually acute may extend to the epidermis causing Munro microabscess.**

***The epidermis between thickened rete rudges is thin and below this thin layer there are dilated capillaries, so when you have prominent capillary and you remove the scale from it you will see this oozing and this is called Auspitz sign.**

Doctor is now talking about the picture below: "These are fused and thickened, here this area is this, here you see the prominent capillary, and here is keratin and it may contain acute inflammatory cells and here even "isn't very evident" the granular layer has disappeared and here the basal layer with no changes in it ".

Lichen planus :

- Results from CD8+ T cell mediated immune response against AG in dermo-epidermal junction just at the basal layer, now this causes:

→ separation between the dermis and epidermis → Interface Dermatitis

- May be caused by viruses or drugs.

- The result is pruritic purple papules & plaques distributed mainly on extremities, often wrists & elbows.

- It is often Self limited and generally resolves within 1-2 years leaving zones of hyperpigmentation.

- Oral lesions may persist for years.

Morphology:

-Dense continuous lymphocytic infiltrate 'hugging' dermoepidermal junction (You see line of infiltrate just under the epidermis).

-Vacuolar degeneration of basal layer and necrotic basal cells in the papillary dermis (Civatte bodies).

-The junction itself appears in zigzag pattern which is called "Saw tooth appearance", unlike psoriasis in which it is thick.

-You can also see Acanthosis, hyperkeratosis & hypergranulosis .

Bullovesicular diseases

-Now bullous diseases can be classified according to the location into:

1- Intra-epidermal : which can be:

Subcorneal : under corneal layer *

Suprabasal : above basal layer *

2- Subepidermal : the bulla will be covered by the whole epidermis.

Note: Each condition (cause) has its known location of bullae.

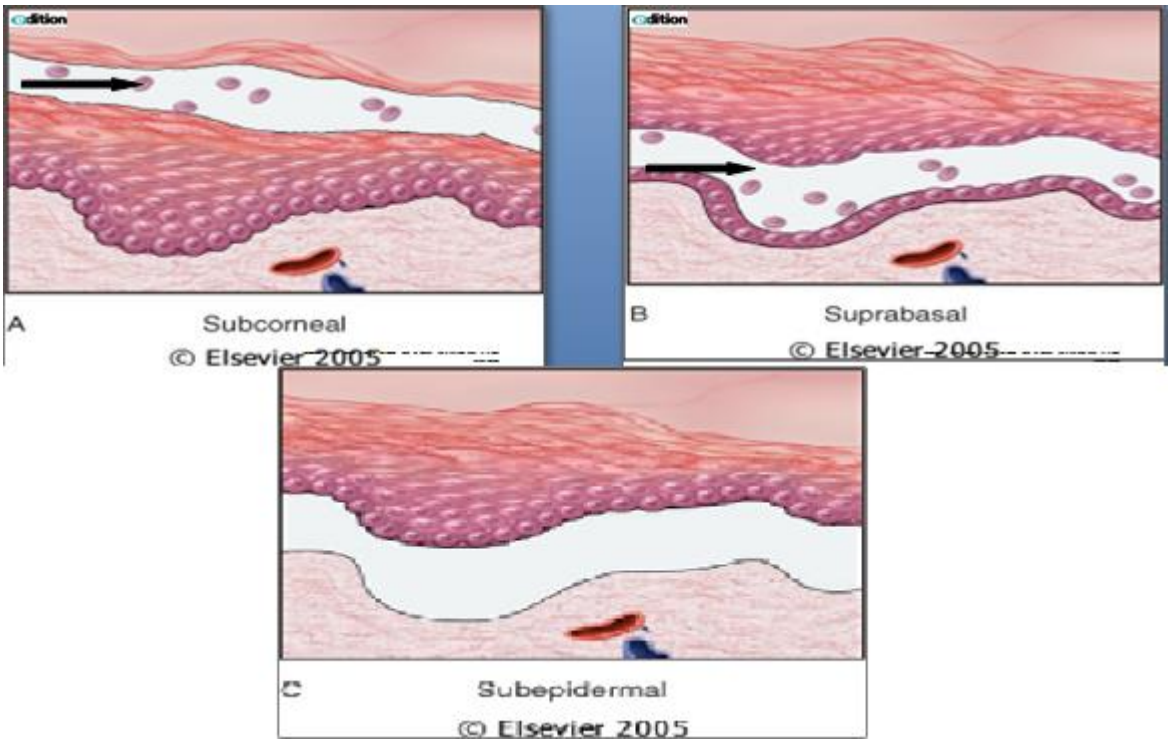
-It is Caused by auto-antibodies to epithelial or Basement Membrane proteins.

-Other causes:

1- Infectious (viral) : like in chickenpox virus or smallpox viruses which can kill the keratinocytes and loosen their attachments and produce spaces filled by fluid .

2- Traumatic: like in burns, anybody who is burnt will produce fluid full bullae or vesicles depending on the degree of the burn.

The figures below show the different types of the bullous diseases ☺



1- Vulgaris Pemphigus:

- Usually a suprabasal condition

- IgG antibodies against the intercellular attachments in the epidermis and mucosa (mucosal surfaces) in congenital area of female.
Now what happens to these intercellular junctions? They are destroyed and loosened, the cells detach and easily rupturing superficial blisters are formed.

- Different types some are associated with internal malignancy.

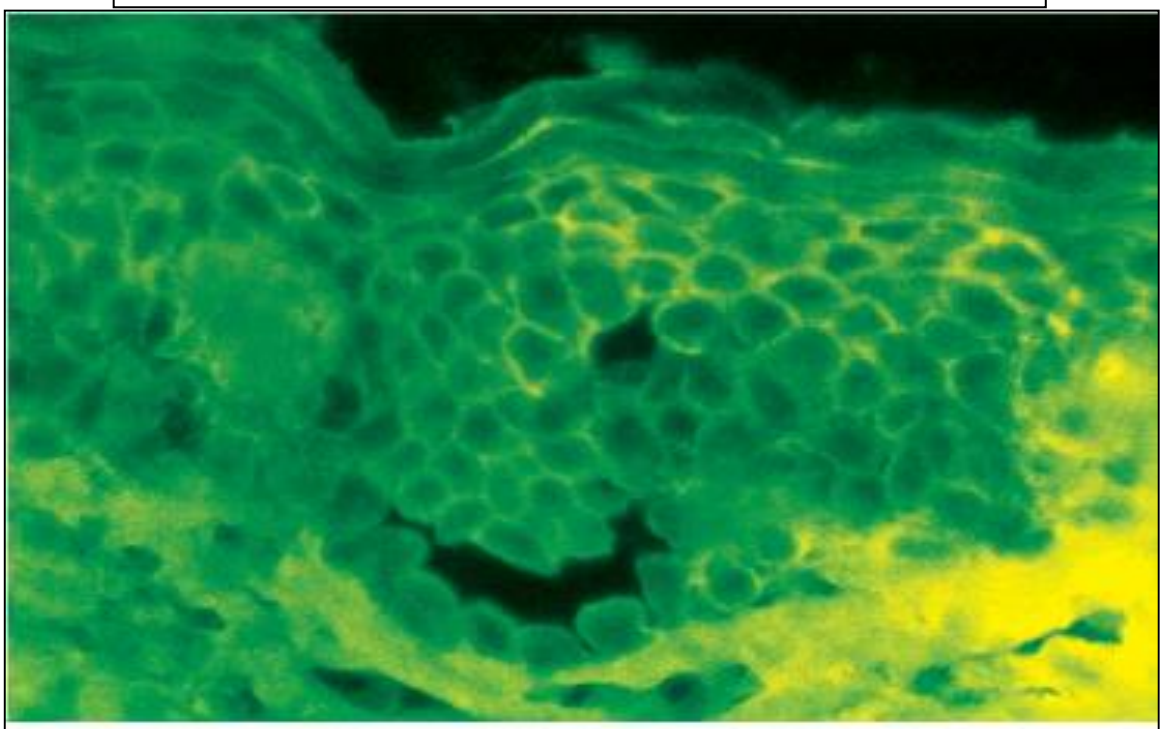
- Morphology:

***The main feature is Acantholysis (Suprabasal acantholytic blister).**

***Infiltration by lymphocytes, histiocytes and eosinophils.**

***Now in testing vesicular diseases we use immunofluorescence why? Because by immunofluorescence you will see where the antigen-antibody reactions are present. And when using this technique in testing pemphigus vulgaris you will get a net-like pattern of intercellular IgG deposits localized to sites of acantholysis.**

Testing pemphigus vulgaris by immune fluorscence



2- Bullous Pemphigoid

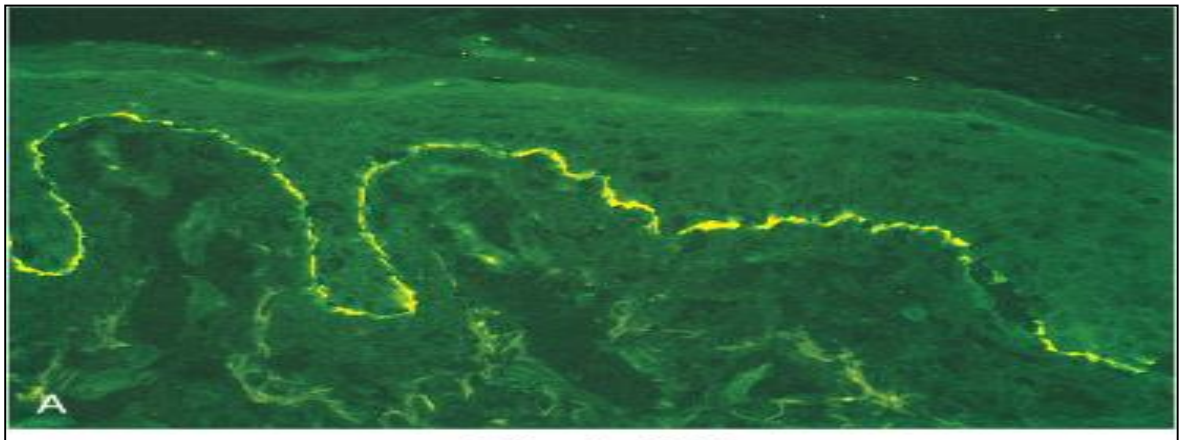
-Subepidermal bullous disease.

-IgG antibodies directed to the basement membrane.

-Tense bullae filled with clear fluid on erythematous base.

-Lesions up to 5 cm and do not rupture easily, why? Because the whole epidermis is covering it, so it's tenser and not easily ruptured.

-Oral involvement is seen in 10-15 % of cases.



3- Dermatitis Herpetiformis

- Subepidemal.

- Grouped vesicles, seen more in males, sometimes associated with celiac disease.

- IgA antibodies (not IgG) directed to dermal epidermal junction.

- Neutrophilic microabcesses at tips of dermal papillae.

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