

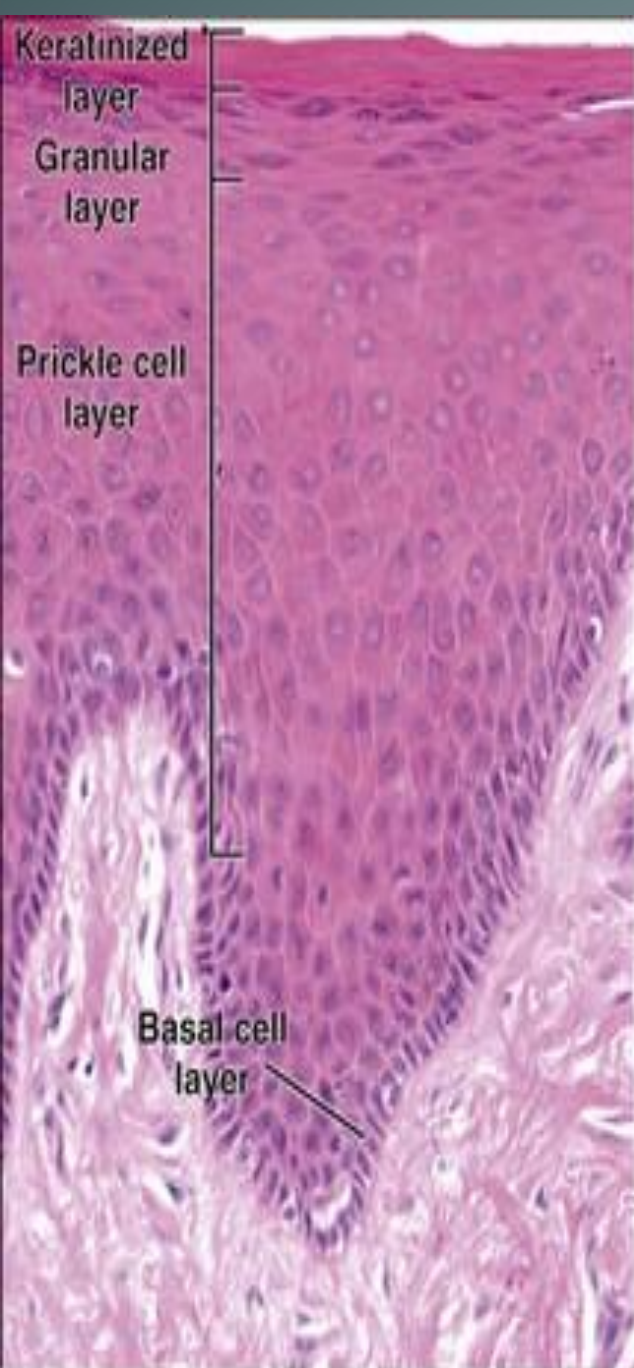


White Lesions of Oral Mucosa

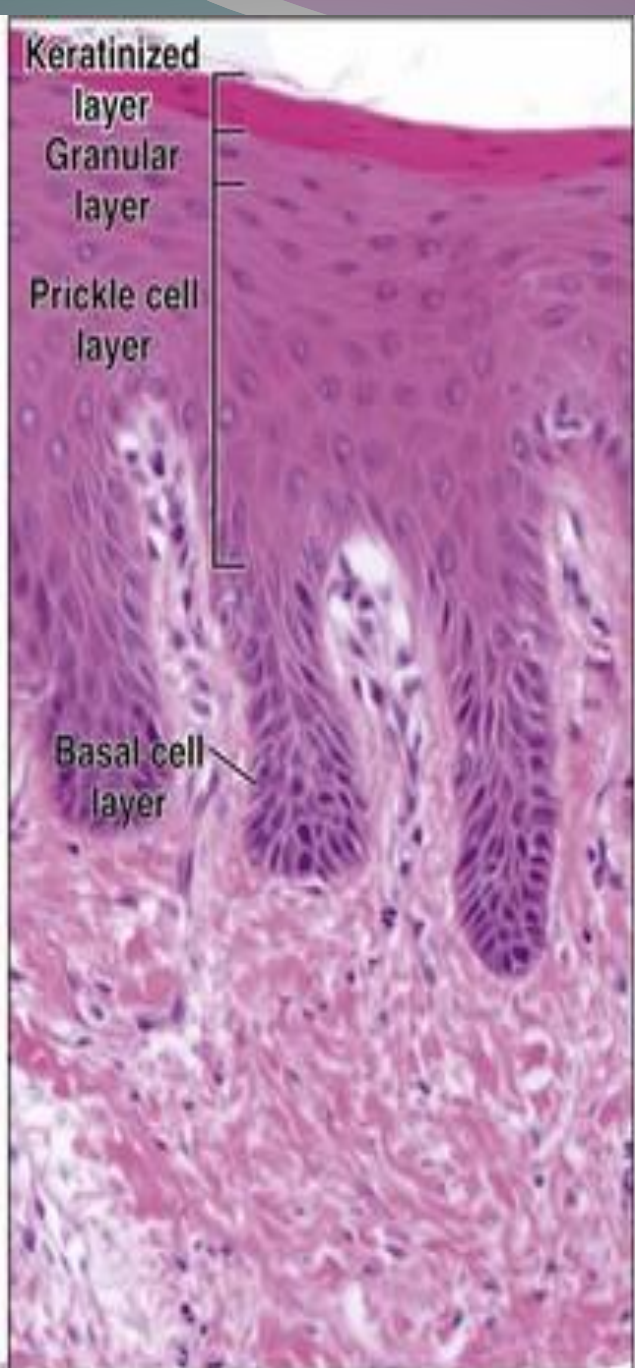
Color of oral mucosa depend on:-

- 1-Surface keratin (keratinization).
- 2-Thickness of covering epi.
- 3-Melanin pigmentation.
- 4-Blood vessel in the underlying C.T(pink or red color).

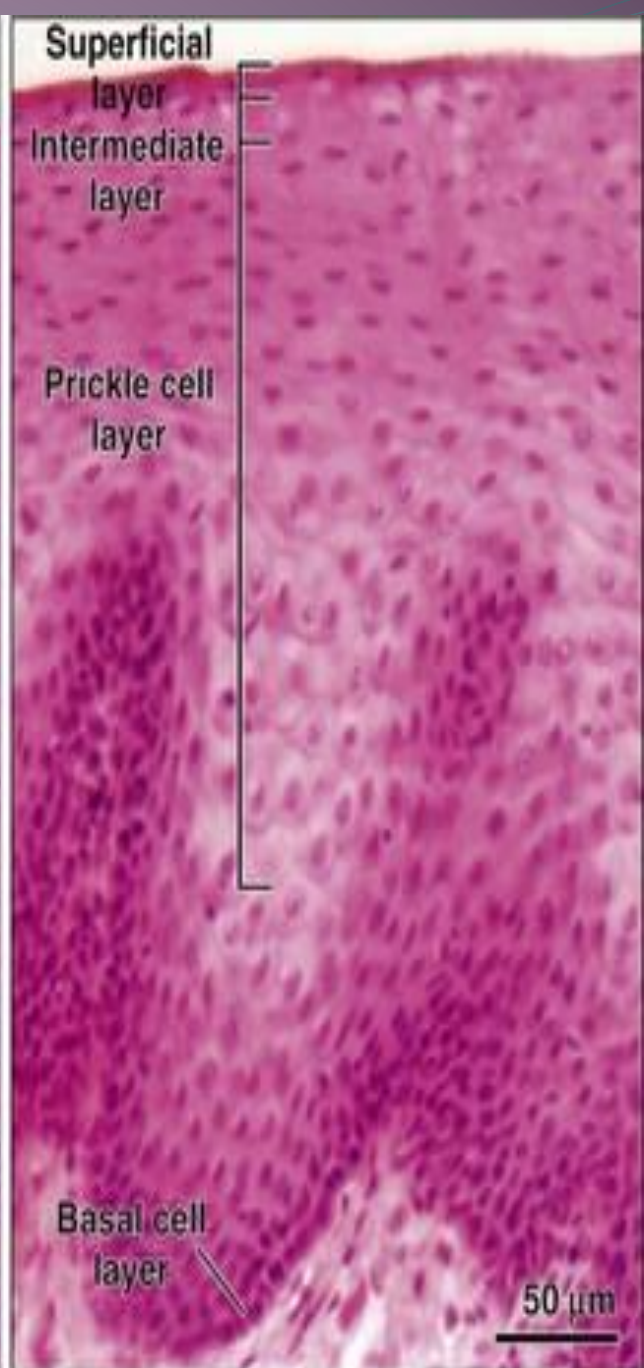
An increase in keratin layer and covering epi. lead to whitish oral mucosa.



A



B



C

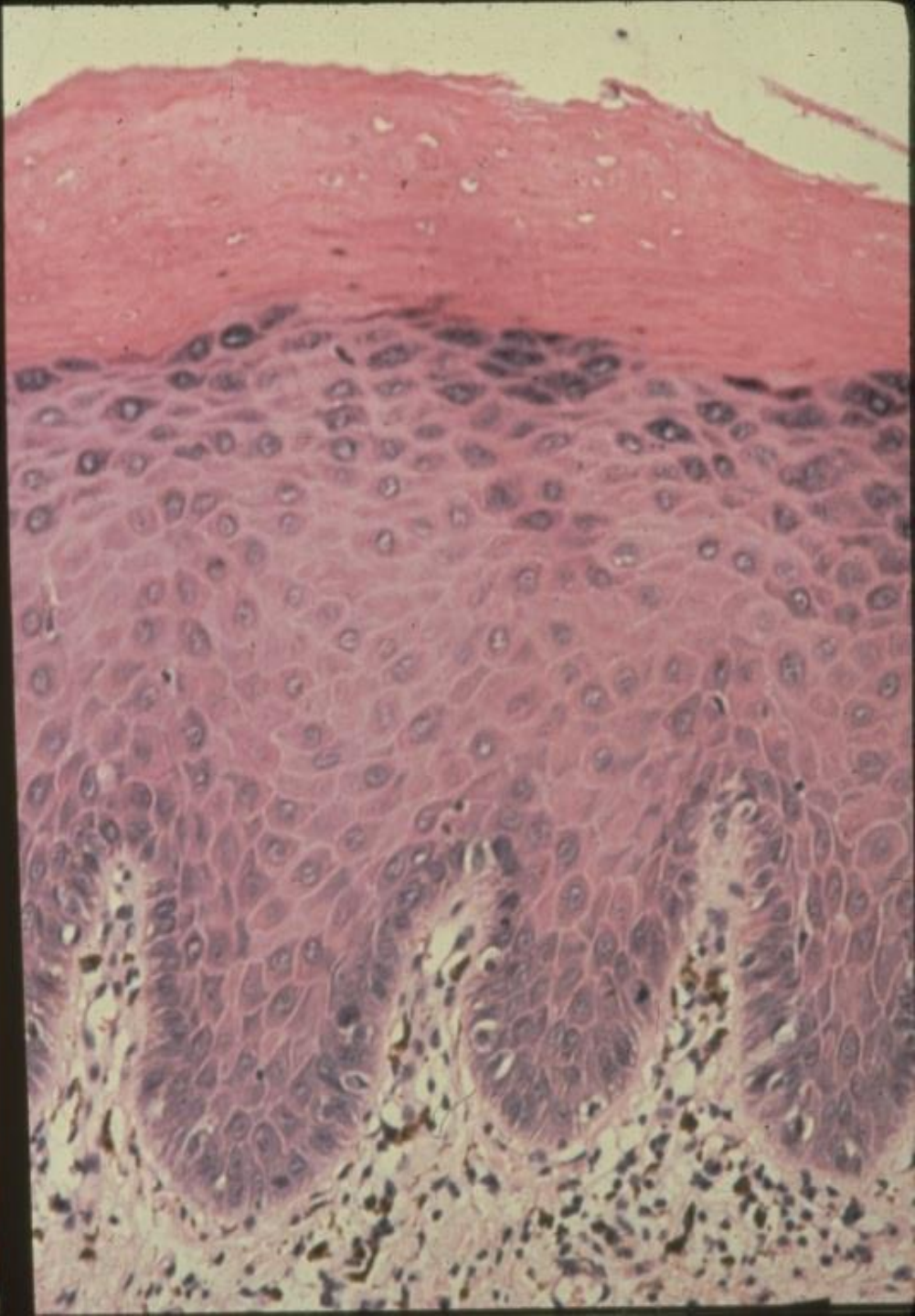
Clinically , white color lesions result from:-

- 1-Thickened surface keratin layer = **hyperkeratosis** .i.e increase or widening of stratum Corneum (hyperparakeratosis or hyperorthokeratosis).
- 2-Thickened spinous cell layer, **epith. hyperplasia** , which acts as a barrier masking the normal vascularity of the underlying C.T
- 3-Change in the underlying C.T: decrease the number of blood vessel and increase in collagen fibers.



These white lesion will described as **keratosis** or **keratotic lesion**.

-Keratosis appear white because the thickened or abnormal keratin become hydrated as a result of being bathed by saliva and evenly reflect light.



- Most of white lesions are symptomatic, rough to palpation and can't be rubbed off with a gauze.
- White lesion that rubs off, they are due to accumulation of epithelial debris, or inflammatory exudates on the surface due to infection.
- White lesions classified **Microscopically** into:-
 - 1-Focal keratosis :-simple hyperkeratosis and epithelial hyperplasia without epithelial dysplasia.
 - 2-Hyperkeratosis and epithelial hyperplasia with epithelial dysplasia (mild, moderate, Severe).

3-Carcinoma in situ.

4-Squamous cell carcinoma.

-Cellular atypia :- cellular changes which cytologically characterized malignant and premalignant lesions.

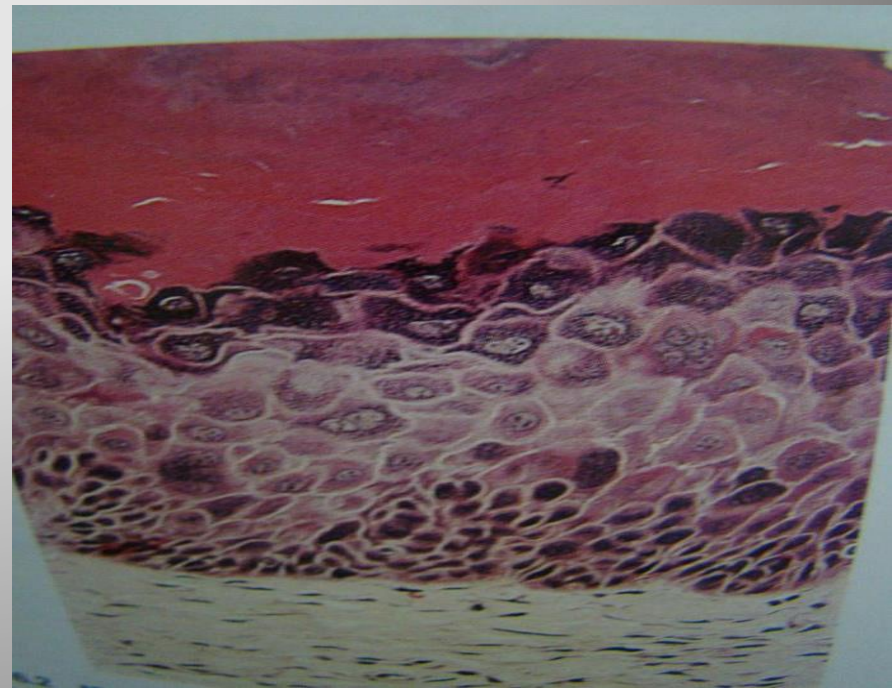
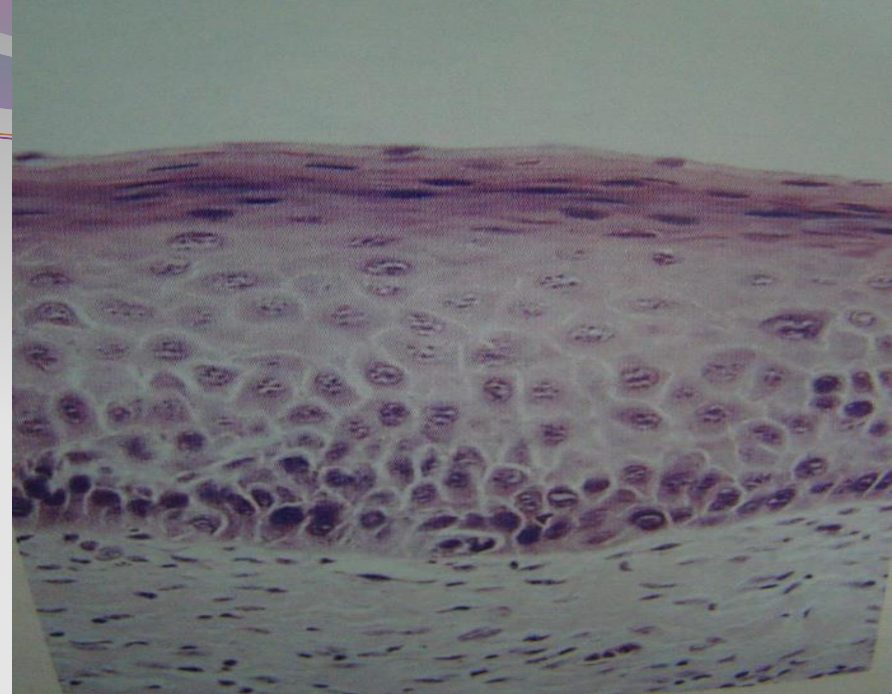
-Epithelial dysplasia:- term describing epithelium when features of cellular atypia is present.

Atypia :- changes in individual cell.

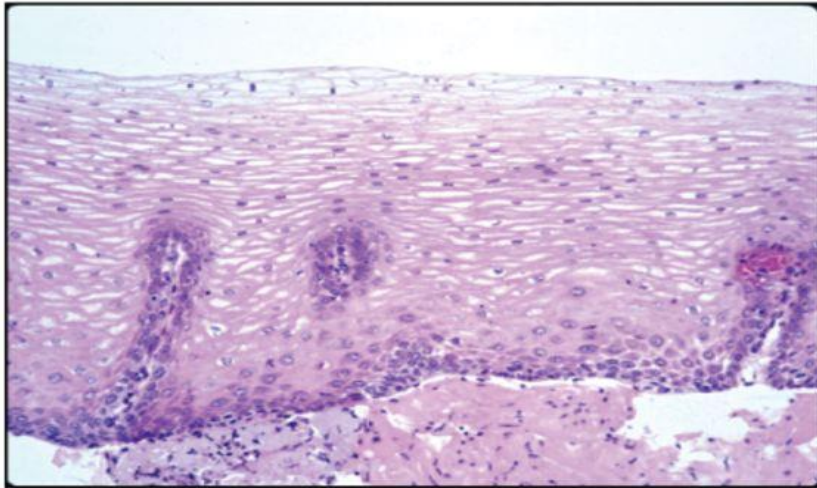
Dysplasia :- changes in epith. (as tissue).

Epithelial dysplasia

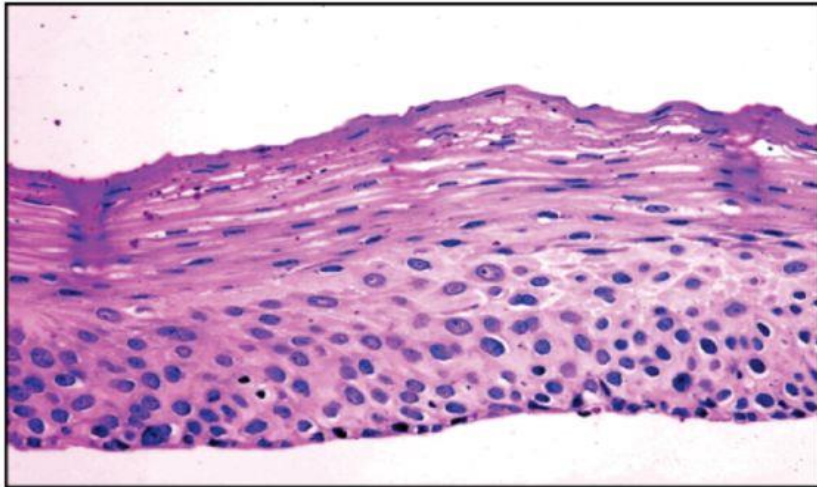
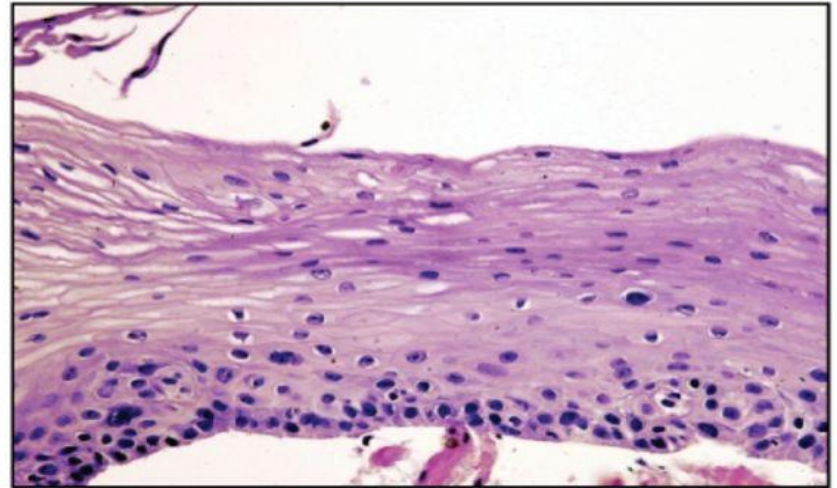
- Serious condition refers to abnormal growth pattern, or disorientation of normal layers of epith.
- It indicates premalignant changes due to this cytological abnormality.
- Changes can be so severe resemble cancer called **Carcinoma in situ**, which is a severe stage of epithelial dysplasia involving the entire thickness of epith. with no invasion to underlying C.T (intact B.M).



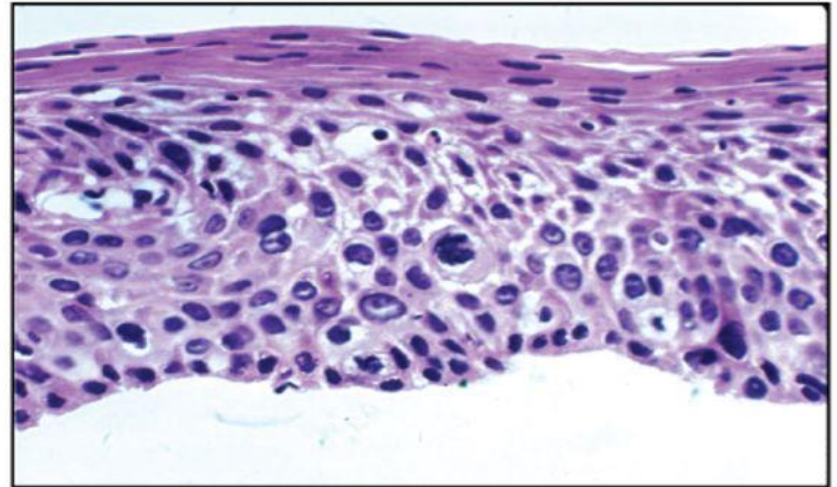
Normal



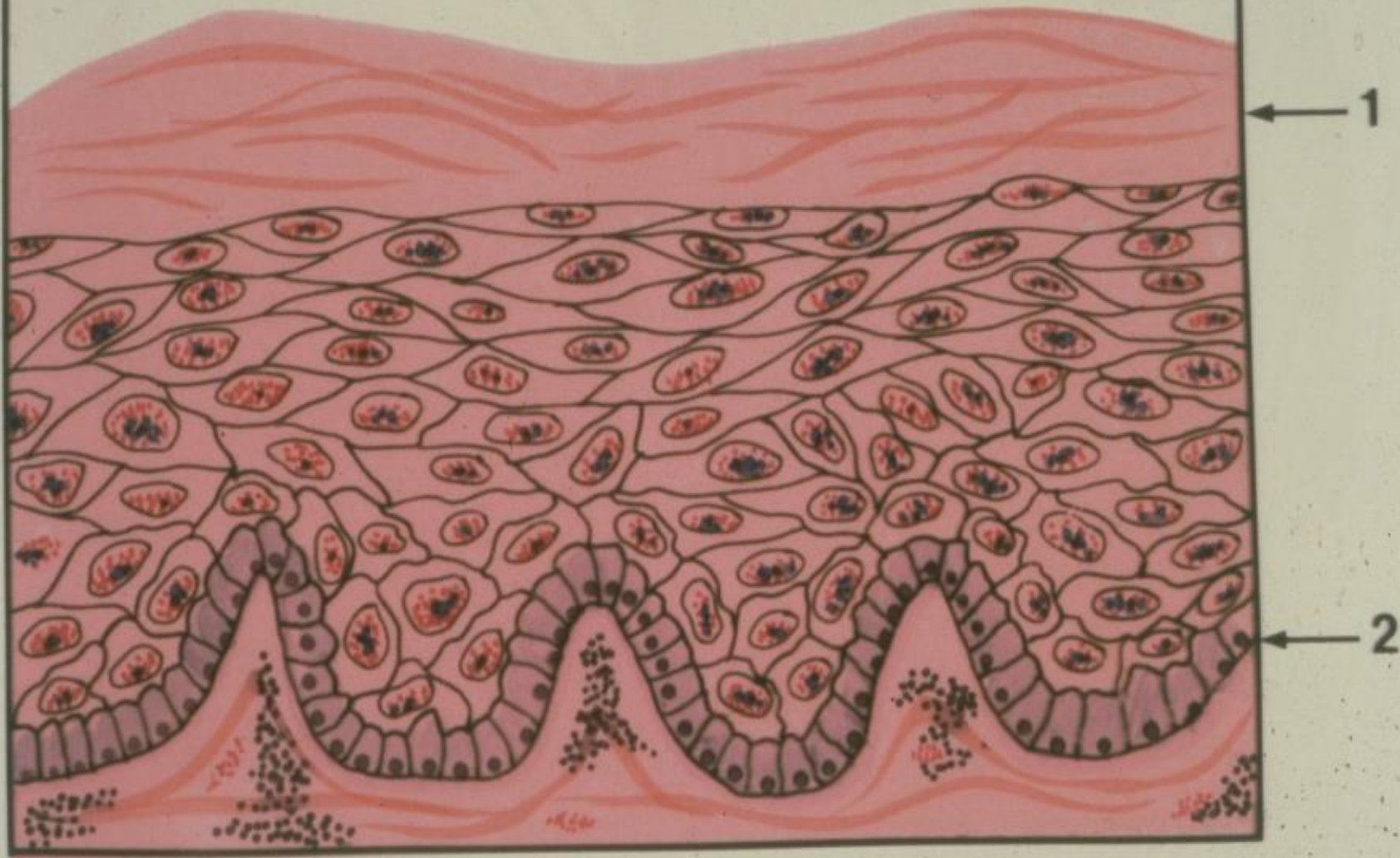
Mild dysplasia



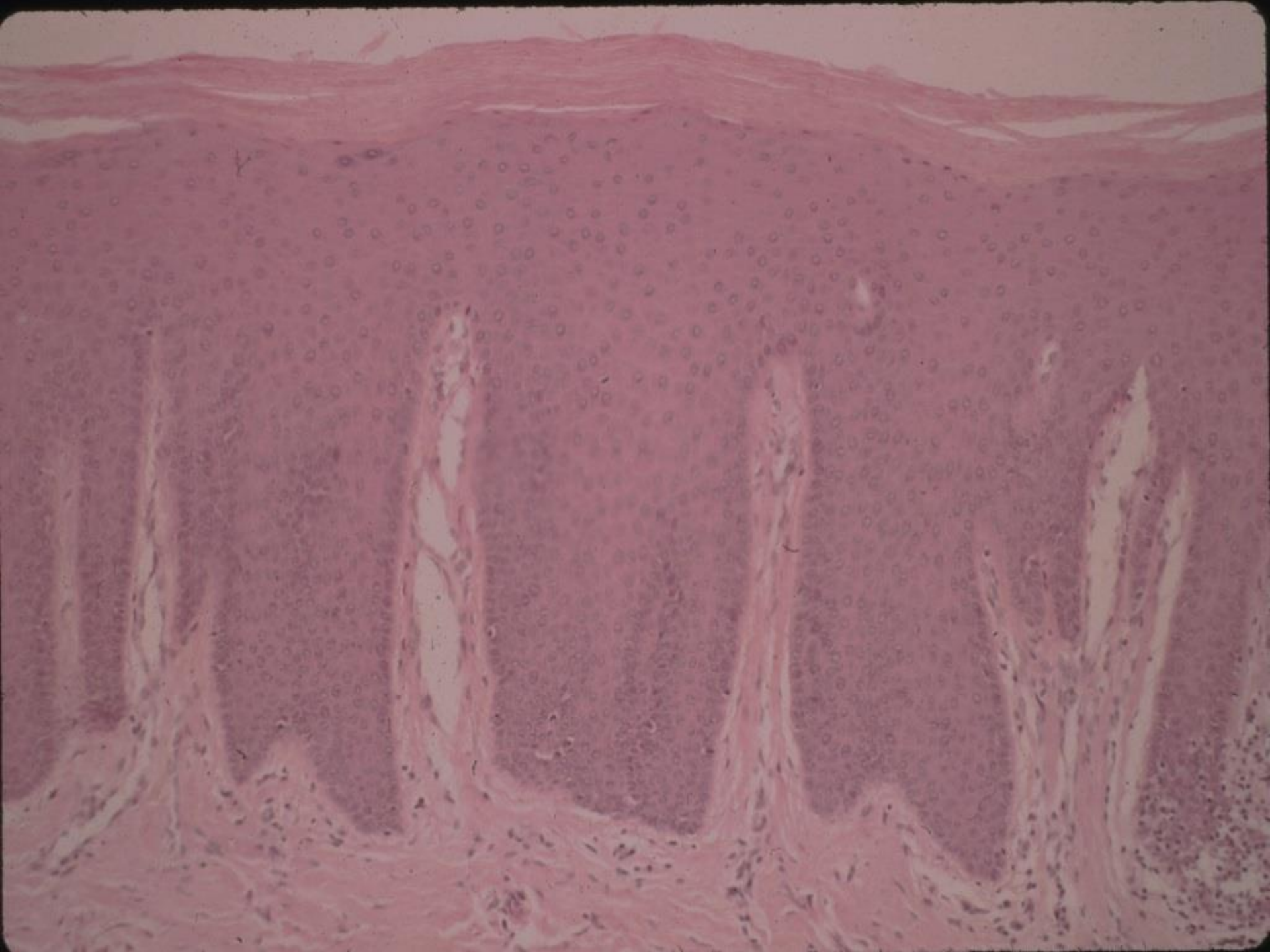
Moderate dysplasia

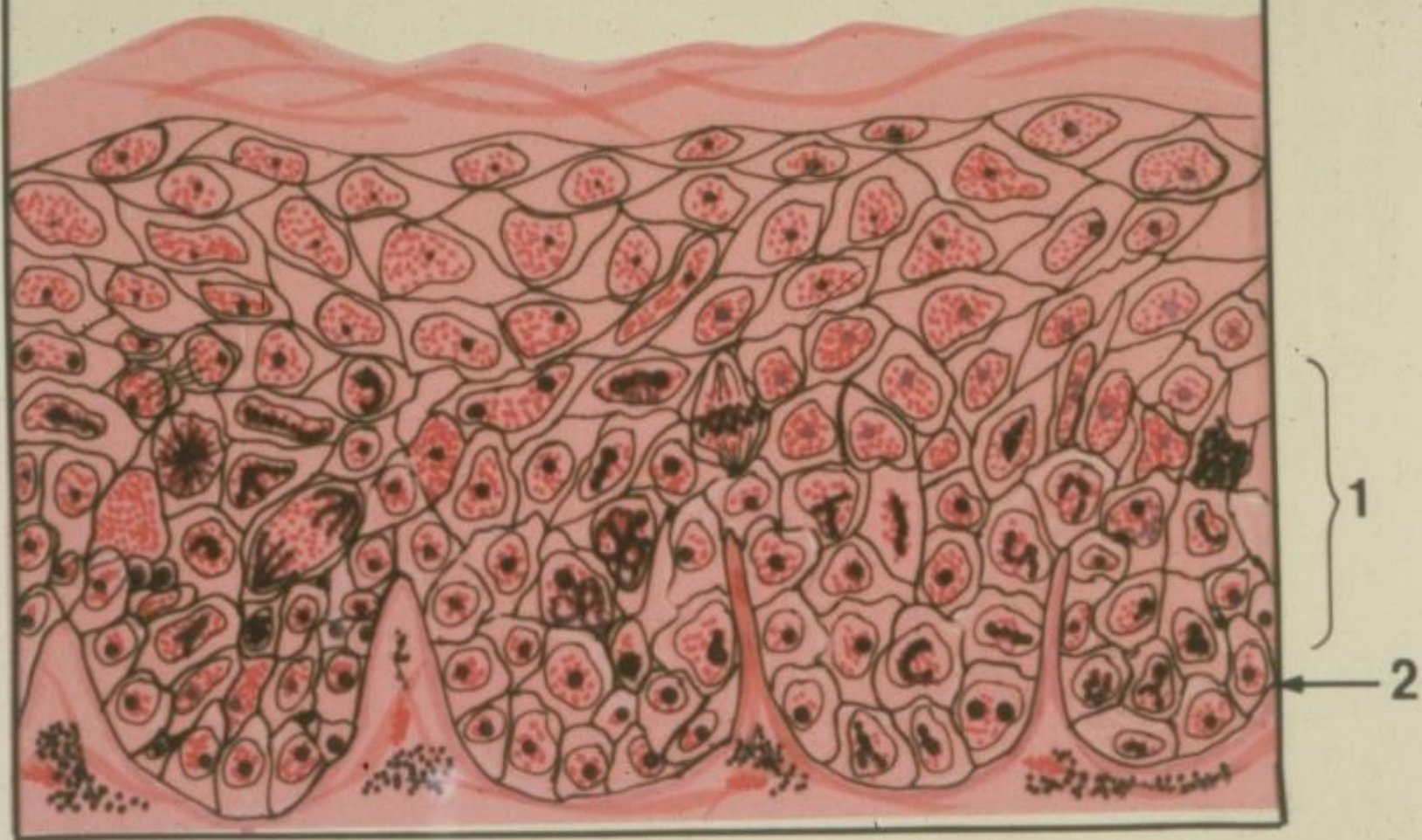


Severe dysplasia



Hyperorthokeratosis



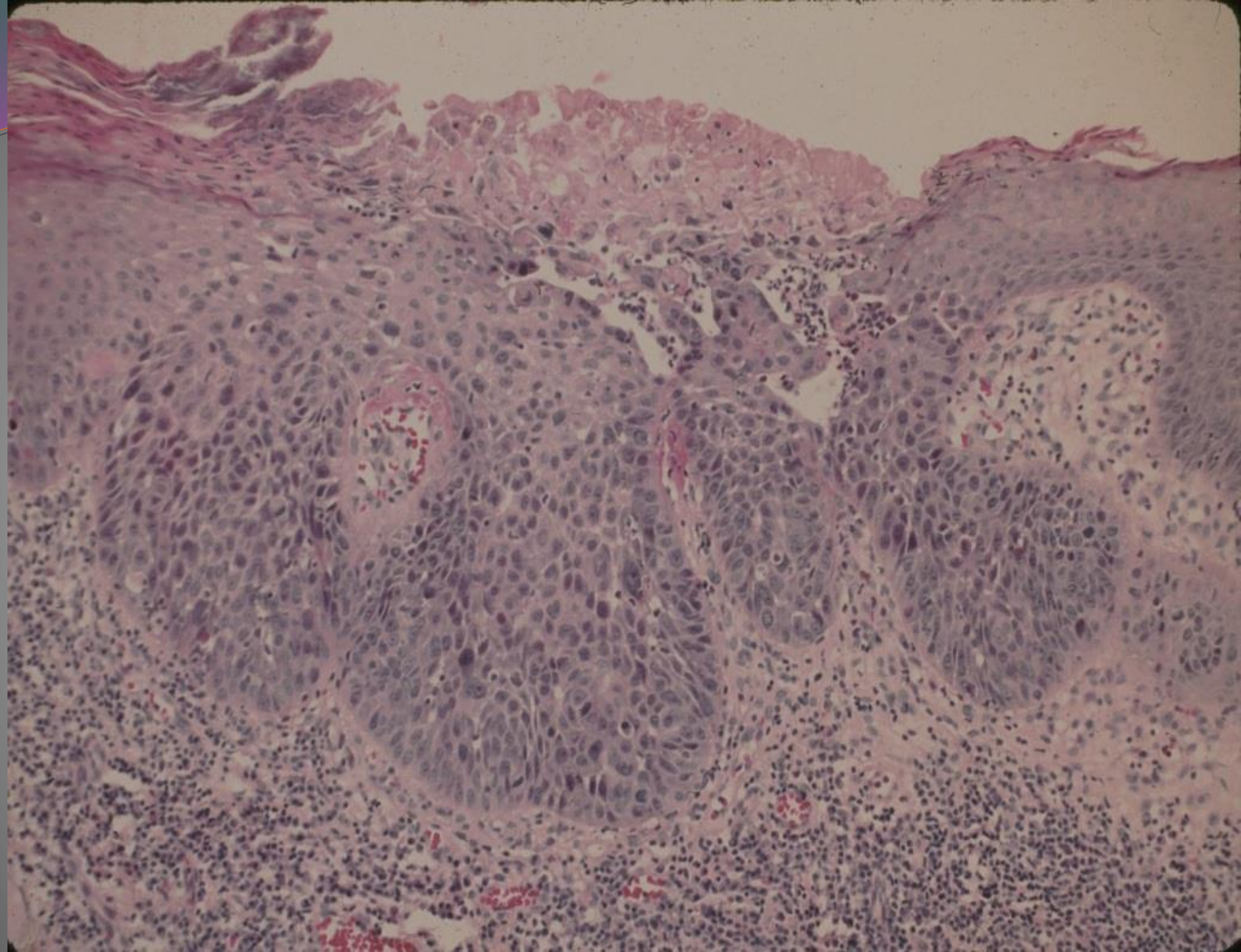


**Moderate Atypicalities
(dysplasia)**





Carcinoma in situ



Classification of white lesions according to etiology:-

1-Hereditary (genokeratosis)

2-Traumatic

3-Infective

4-Idiopathic

5-Immunologically mediated white lesion or dermatological

6-Neoplastic

1-Hereditary white lesion :-

a-White sponge nevus (oral epi. Nevus).

b-leukoedema.

White sponge nevus (oral epith. nevus):-

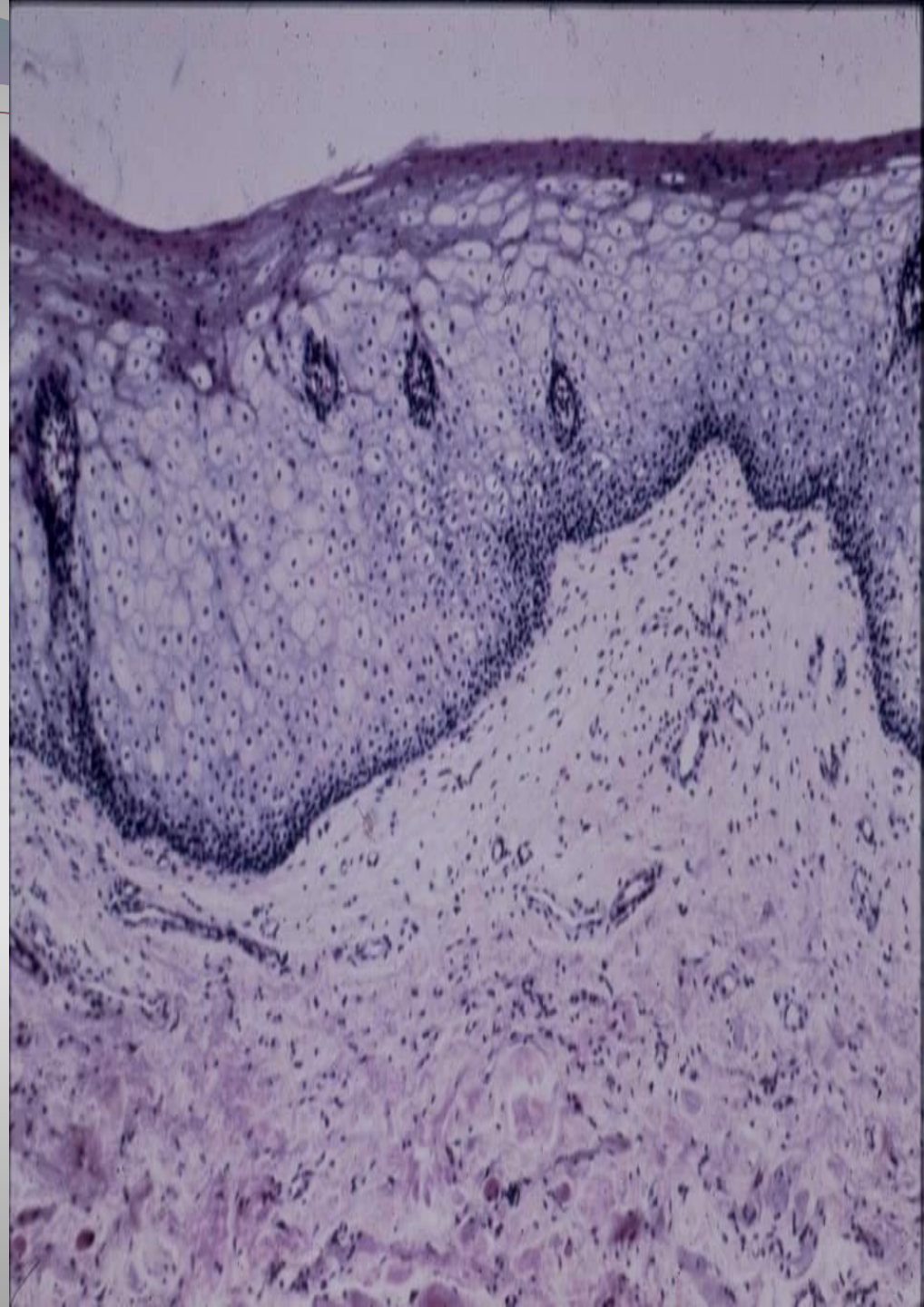
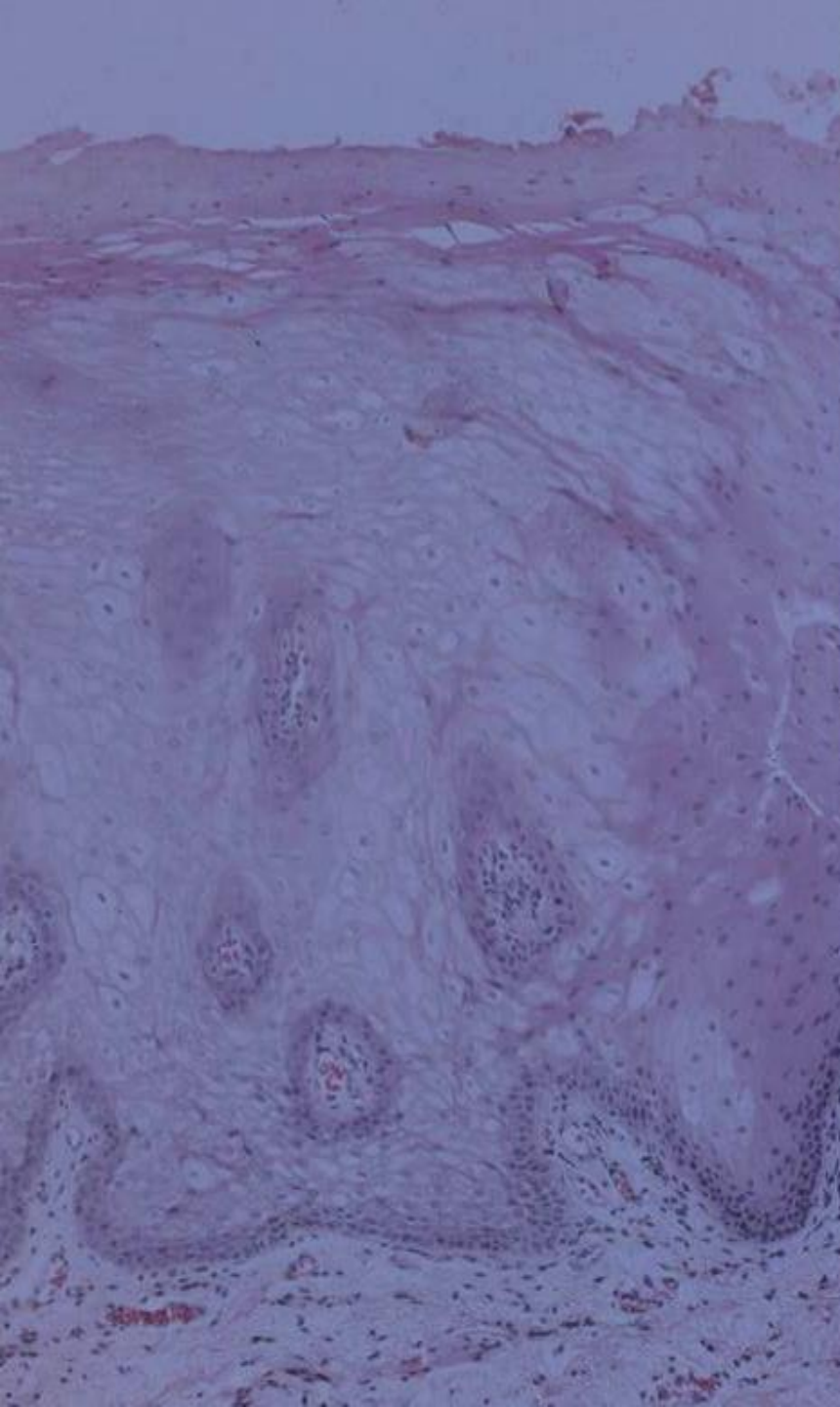
- Appears at birth or early childhood as a thickened & folded lesion, the edge are not well defined, but gradually merge with the normal mucosa.
- May present on buccal mucosa, tongue, gingival & floor of the mouth.
- Benign lesion, painless, require no treatment.



leukoedema:-

- White lesion on the cheek (buccal mucosa), bilaterally, unknown etiology, considered as a deviation from the normal.
- Appear as asymptomatic, translucent, grayish-white filmy appearance. Disappear on stretching.
- No treatment required.





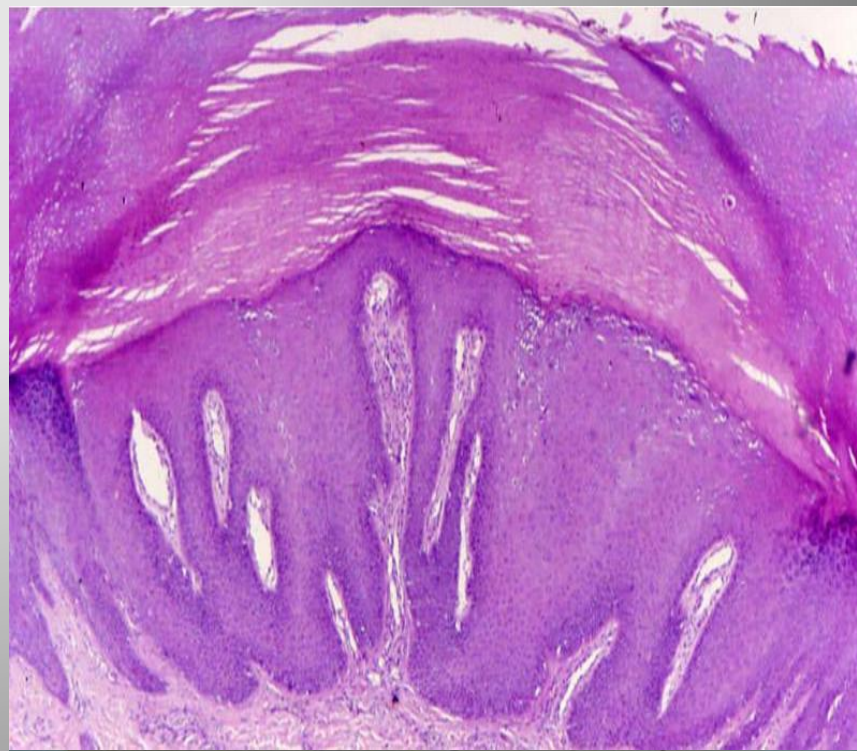
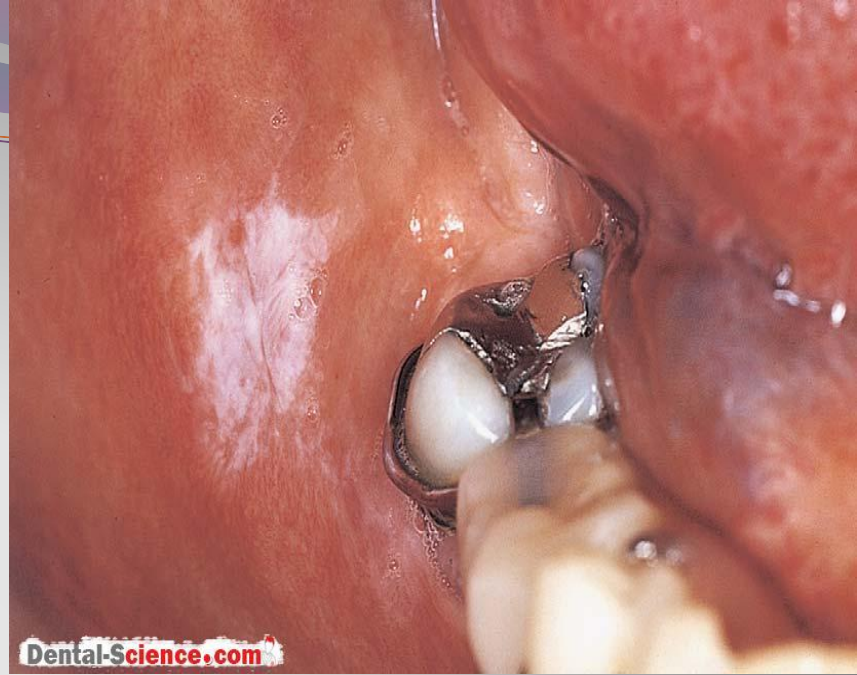
2-Traumatic white lesion :-

Chemical, physical, mechanical factors, ,,white lesion close to the causative factor.

A-Mechanical trauma:-

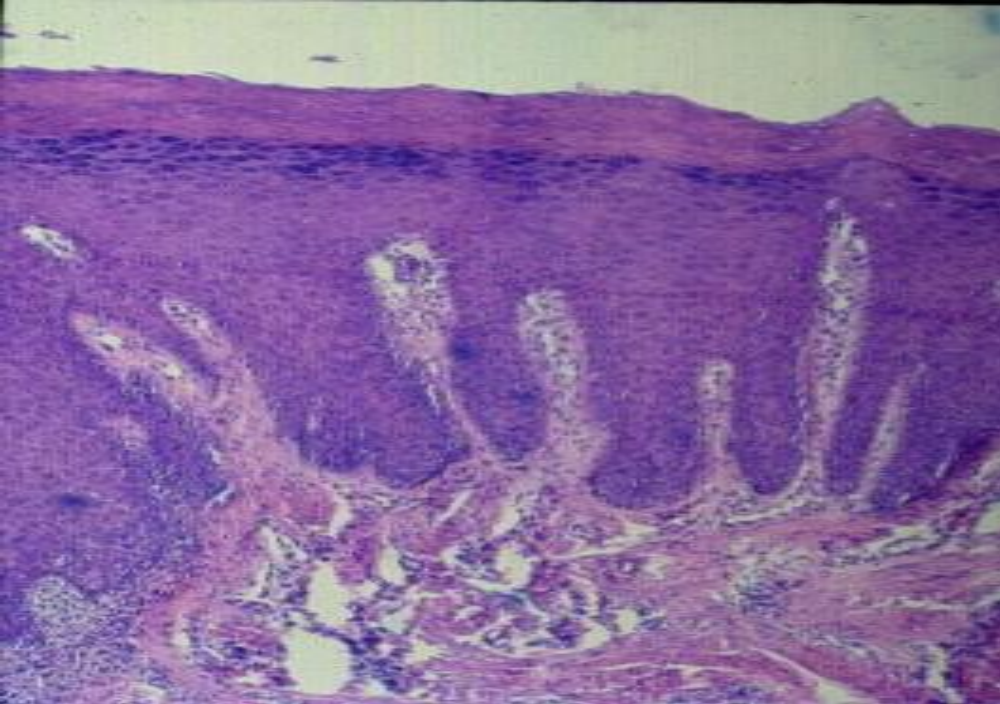
Either

- Acute trauma which lead to ulceration of oral mucosa.
- Chronic trauma lead to epith. hyperplasia and hyperkeratosis, white lesion as a defense mechanism, ex:- sharp tooth, sharp bridge, cheek biting .
- White lesion produced by these trauma called **frictional keratosis**.
- Removal the cause must lead to the disappearance of lesion, if not leukoplakia.
- Microscopically appear as hyperkeratosis with a canthosis , no dysplasia.

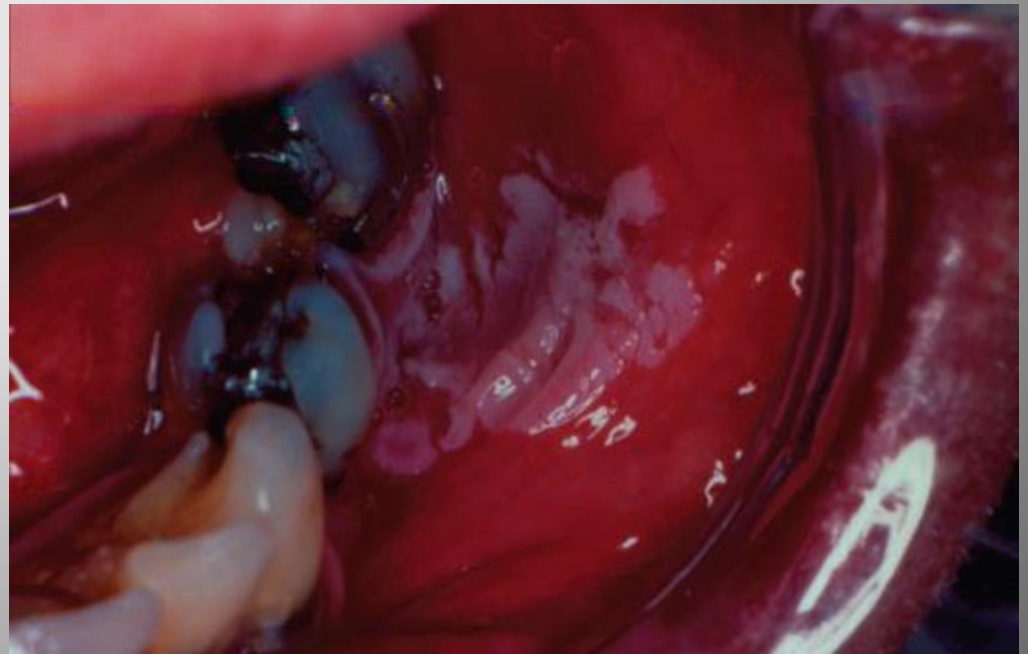


b- Chemical :-ex:-

- 1- **Aspirin Burn** sloughing and ulceration of epi. caused by salicylic acid applied on tooth or oral mucosa in a toothache.
- 2- **Tobacco** whether smoking , chewing, snuffing and betel nut, act as chronic chemical irritation which may lead to hyperplasia & hyperkeratosis. Compound of tobacco lead to leukoplakia and squamous cell carcinoma.
(Tar, nitrous amine compound, with steady continuous mild heat)



Aspirin burn, creating a pseudomembranous necrotic white area.



c-Thermal :- Regular smoking of cigarette, cigar ,pipe → white plaque on oral mucosa , at the anterior part of buccal mucosa, tongue and palate.

-Smoking (both chemically and thermally effect on oral mucosa).

-Constant smoking cigarette from lip= keratosis on lip site.

-Pipe and cigar smoke lead to palatal lesion due to heat==

Nicotinic stomatitis

palatal mucosa become diffusely gray or white with numerous slightly elevated plaque with punctuate red center, representing inflammatory minor salivary gland and their ductal orifice.

-Reversible == stop smoking (1-2) weeks, oral mucosa return to normal.



3-Infectious white lesion:-

a-Candidiasis

it is either chronic hyperplastic candidiasis or acute pseudomembranous candidiasis (thrush ,moniliasis).



b- Syphilis leukoplakia



c-Hairy leukoplakia

Caused by **EBV**, appear as a raised corrugated or hairy surface white lesion, on the lateral border of tongue.



4-leukoplakia:-

- White patch on oral mucosa that are neither scrapped off, nor classified as any other diagnosable disease .
- Only clinical description term (**not diagnostic**).
- Significance of leukoplakia depend on its histological findings.

*Causes :-

- 1-Tobacco smoking it is the major factor
- 2-Alcohol consumption
- 3-Chronic irritation (ill-fitting denture)
- 4-HPV play a role.
- 5-UV radiation.

*Clinically:

Vary from white, small and circumscribed plaque to an extensive lesion involving large area of oral mucosa.

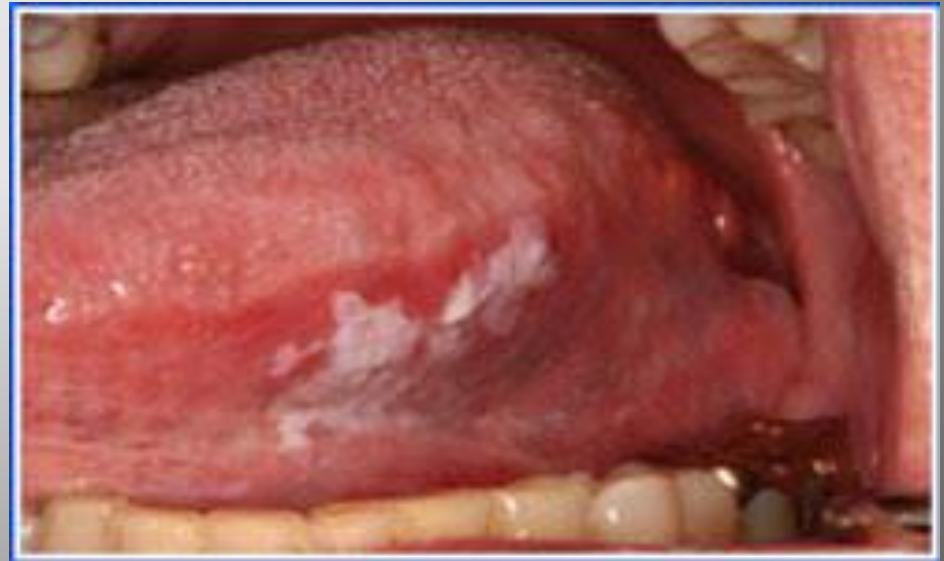
- White, whitish- yellow, grayish-white, with homogenous or non-homogenous surface.
- Surface either smooth or rough and sometimes may be wrinkled or crossed by small cracks or fissures.



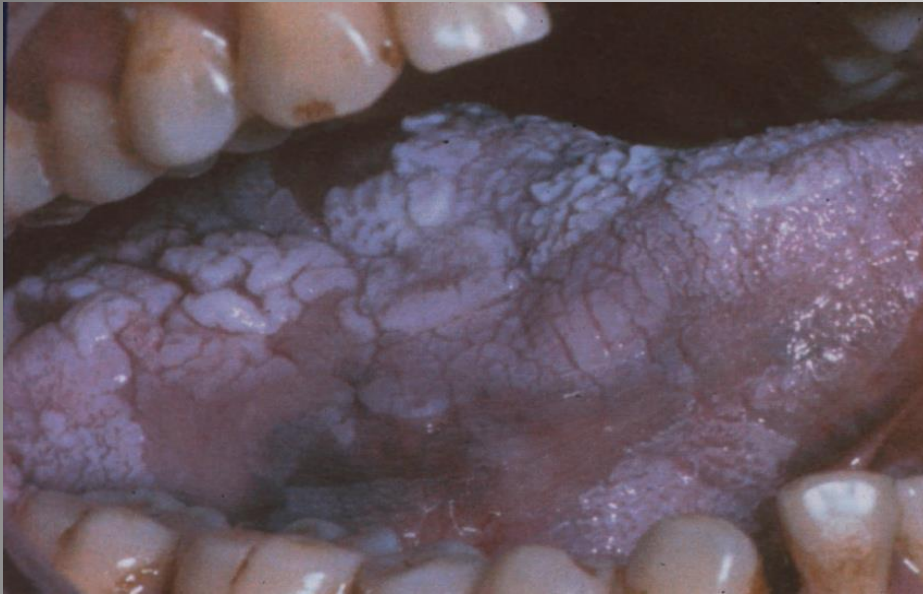
Leukoplakia



-Non-homogenous leukoplakia, shows area of redness, speckled leukoplakia.



- Sometime leukoplakia appear as prominent warty appearance like== **verrucous leukoplakia**.



- Non-homogenous leukoplakia, mostly associated with epith. dysplasia so it consider as a premalignant, some are invasive squamous cell carcinoma at the time of presentation.
- 5% of oral squamous cell carcinoma **clinically** appear as a white lesion.

Pathology:-

show wide range of histological appearance.

1-Hyperkeratosis (ortho or para)

2-Acanthosis (epith. hyperplasia)

3-Epith. dysplasia

4-Carcinoma in situ

5-Invasive squamous cell carcinoma.

- 5-10% of leukoplakia undergo malignant transformation (pre-malignant lesion).

- Take more than one biopsy because the histological changes may differ from area to another in the same lesion.

Prognosis :- some undergo malignant transformation.
5-10%

- Transformation time from one to several years.

- Potential for malignant transformation is greater in a high-risk sites=

ventral surface of tongue, floor of mouth, lingual aspect of lower alveolar mucosa.

- leukoplakia in these sites called **sublingual keratosis**.
- 25% of such lesions are squamous cell carcinoma at time of biopsy, 25% developed carcinoma subsequently .
- Appear as white, soft plaque with finely wrinkled surface, irregular well defined outline, sometime bilaterally.

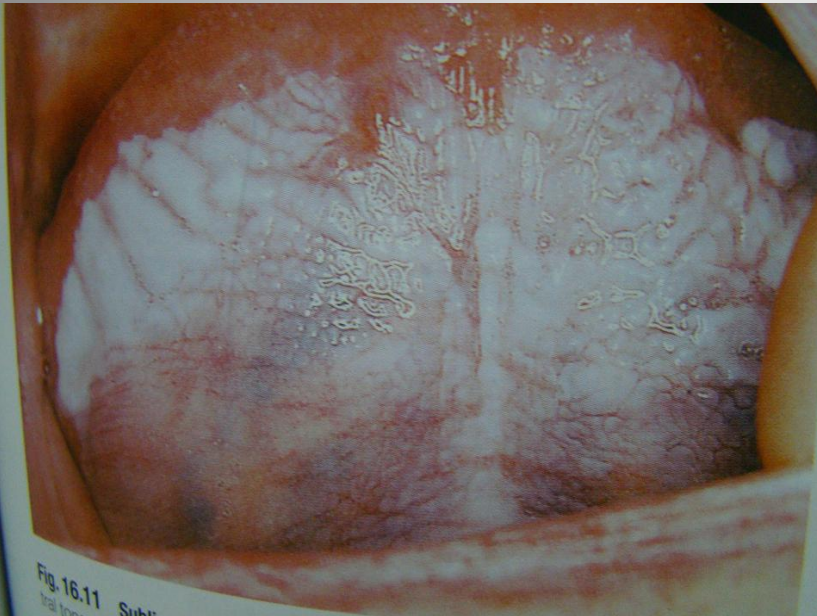


Fig. 16.11 Sublingual keratosis



Erythroplakia :-bright red patches on oral mucosa with well defined border, if it is intermingled with patch of leukoplakia called speckled leukoplakia or leukoerythroplakia

- Mostly in floor of mouth and tongue (lateral and ventral)
- Show epith. dysplasia or invasive carcinoma.



Erythroplakia



Reference: Kumar: Robbins and Cotran: Pathologic Basis of Disease, 7th ed.,
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Speckled leukoplakia :- lesion consisting of white flecks or nodule on atrophic erythematous base.

-Has high degree of suspicion because has high incidence of premalignant and malignant changes.



Lesions with potential for malignant changes, prelignant lesions:-

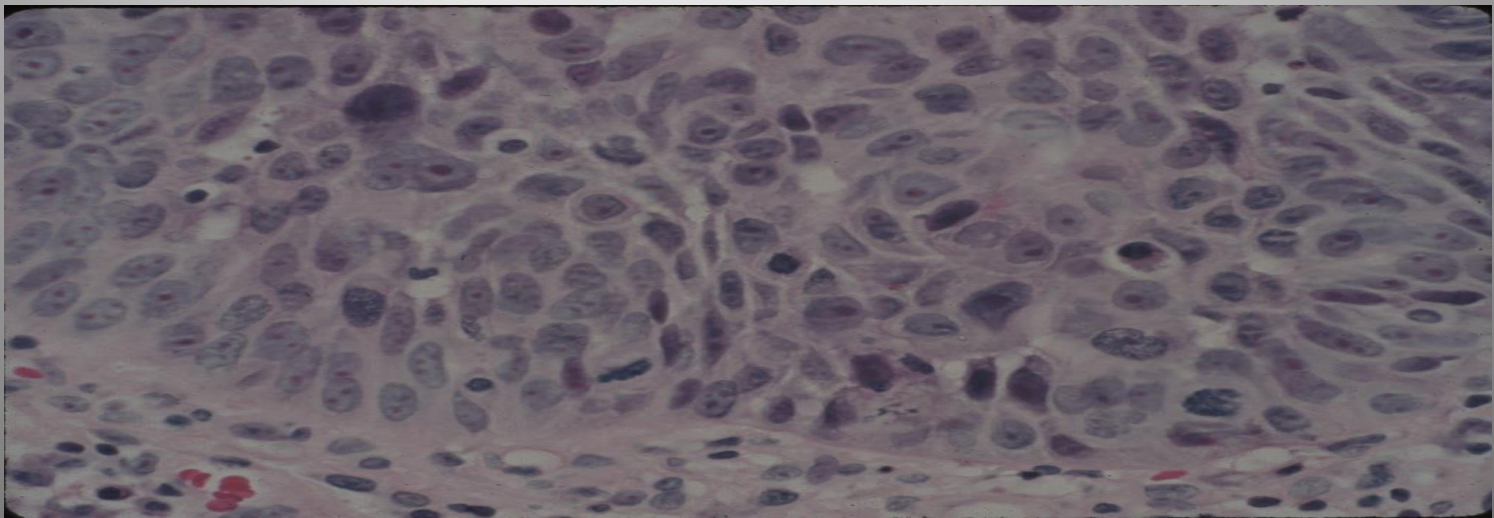
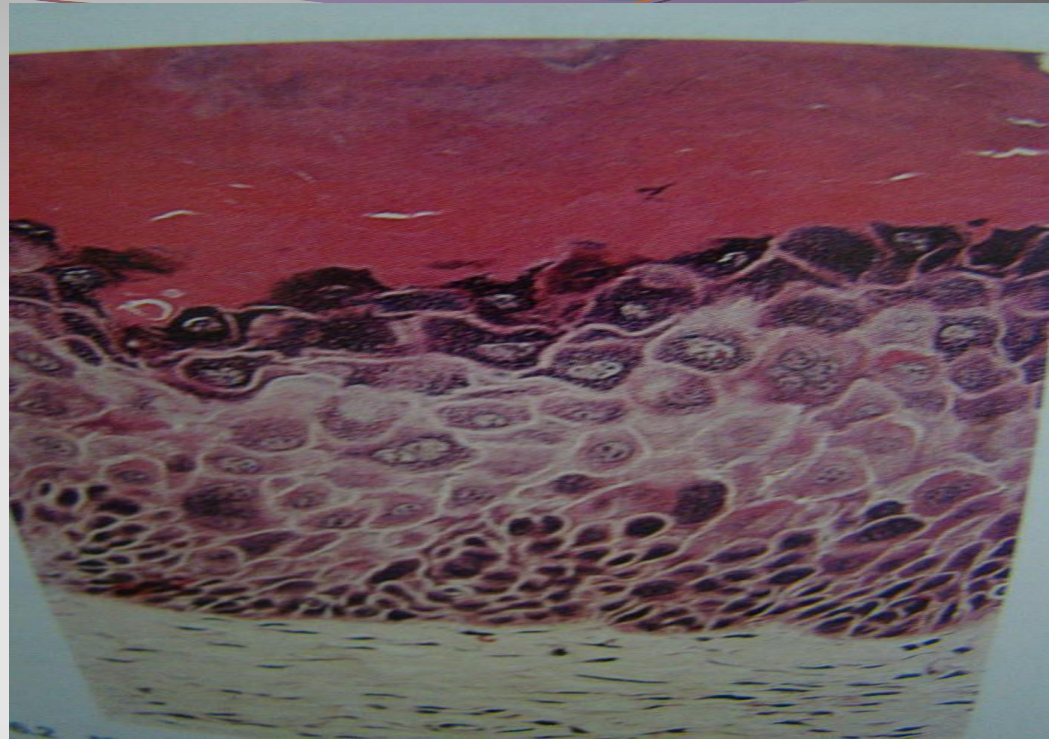
- 1-Idiopathic leukoplakia ----- high risk**
- 2-Erythroplakia (idiopathic) ----- very high risk**
- 3-Speckled leukoplakia (iodiopathic) ----- high risk**
- 4-Chronic candidiasis (candida albicans) ----- low risk**
- 5-Lichen planus (immune mediated) ----- low risk**

-Degree of dysplasia (histologically) is the best predilection for the potential for malignant transformation.

-Premalignancy distinguished from malignancy only by the invasiveness and production of metastasis.

-Individual cell alteration in epithelial dysplasia lead to atypia=

- 1-prominent nucleoli
- 2-hyperchromatic nuclei.
- 3-Nuclear pleomorphism
- 4-Altered N/C ratio
- 5-Increase mitotic activity
- 6-Abnormal mitosis
- 7-Multinucleation of cells.



5-Dermatologic cause of white lesion :-

(Immunologically mediated white lesion):-

1- Lichen planus:-

- Common condition involve skin and oral mucosa.
- More in female of (30-50) years.
- Oral lesion detected in 50% of patient, patient present with skin lesion.
- Oral lesion may occur before, at the same time or after the skin lesion development.
- Skin lesion which are itchy papule with a distinct white streaks on the surface called “**wickham’s striae**”

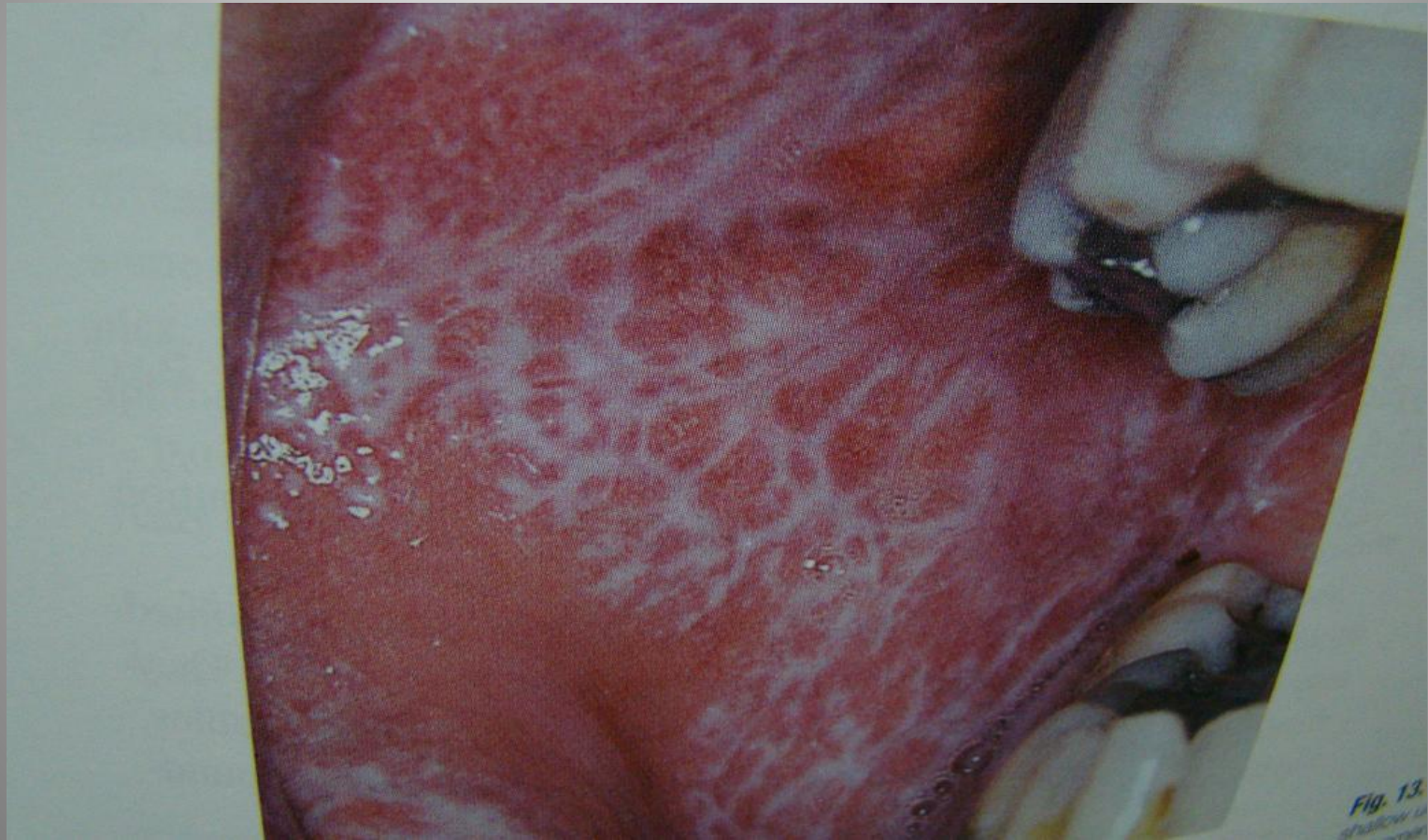


- Any skin area may appear, but commonly on the flexor of wrist .
- Fingernails occasionally involved showing vertical ridge.
- Papule:** it is solid raised lesion less than 5mm in diameter.
- Nodule:** it is solid raised lesion more than 5mm in diameter .
- Skin lesion develop slowly & resolve within **18 months** , recurrence occur.
- In contrast oral lesion much more chronic, it is extends for several years.
- Oral lesion mainly in buccal mucosa, may be in (tongue, gingiva, palate) , floor of mouth uncommon.
- Usually bilateral

-Oral lesion appear with **wide clinical presentation**
occur alone or in combination:-

- 1-Reticuler (lace like striae).
- 2-Atrophic (diffuse red lesion as erythroplakia).
- 3-Plaque like (white patch as leukoplakia).
- 4-Papular (small white papule which may coalesce).
- 5-Errosive (ulcerative).
- 6-Bullous type.

Lichen planus (reticular)



Lichen planus

Atrophic & Errosive



- Reticular , plaque – like & papules are symptoms free.
- Atrophic ,erosive type occurs together, has red glazed mucosal appearance with ulceration (painful and causing discomfort to patient) .
- L.P on gingiva often presenta as a desquamative gingivitis.

***Causes:-**

Unknown etiology, may be T-lymphocytes mediated disorders, when cytotoxic T-lymphocyte stimulated by antigen presenting epith. cell (intraepithelial langerhans cell) which leading to epith. degeneration.

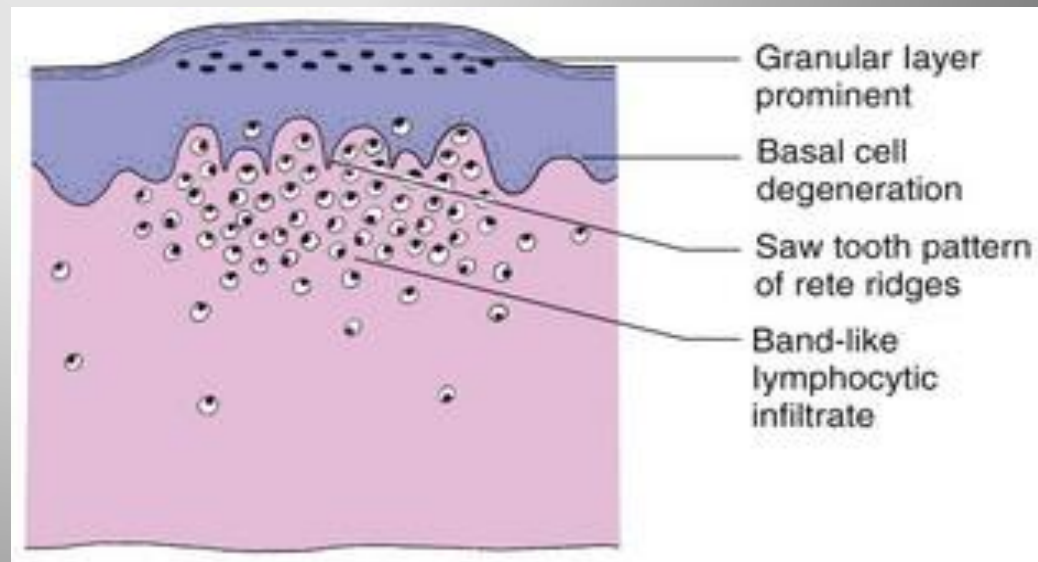
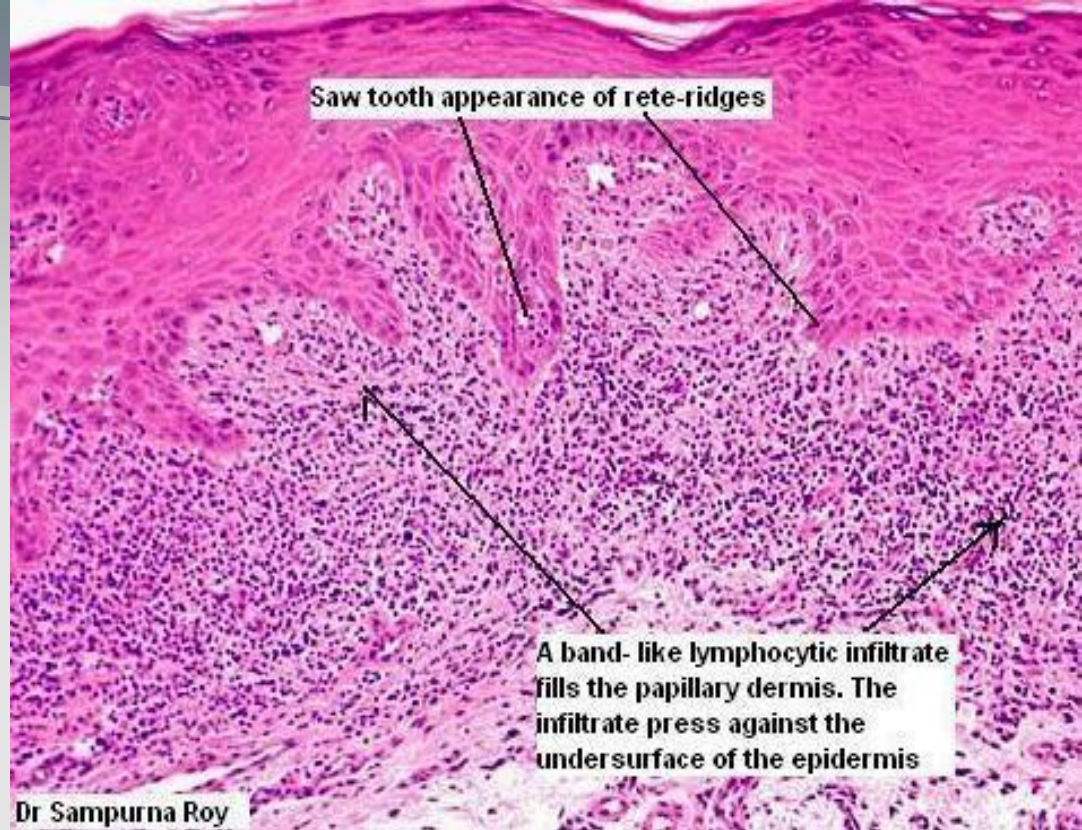
Histopathology:- ortho or parakeratinized epi. of variable thickness (atrophic or acanthotic)

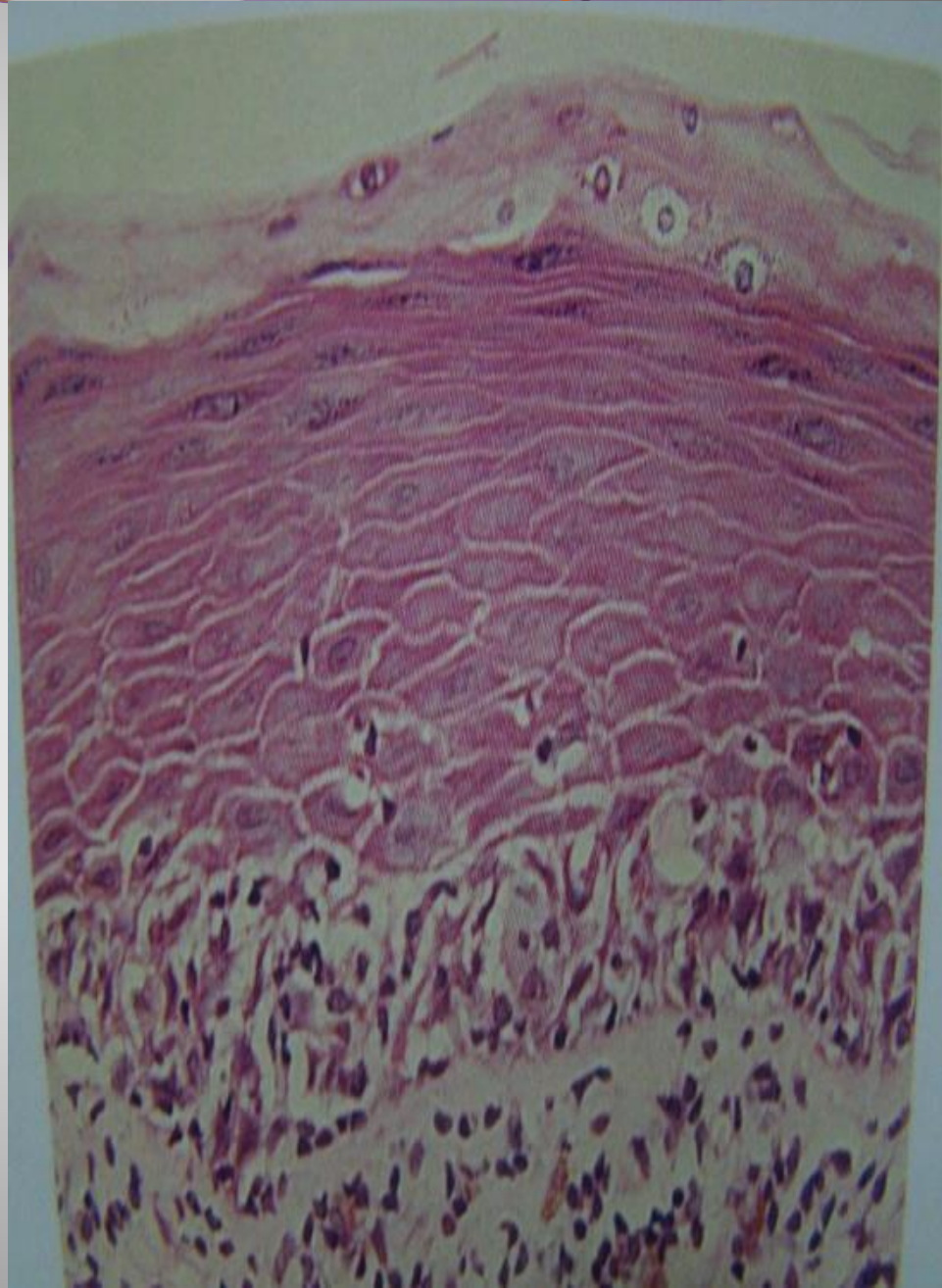
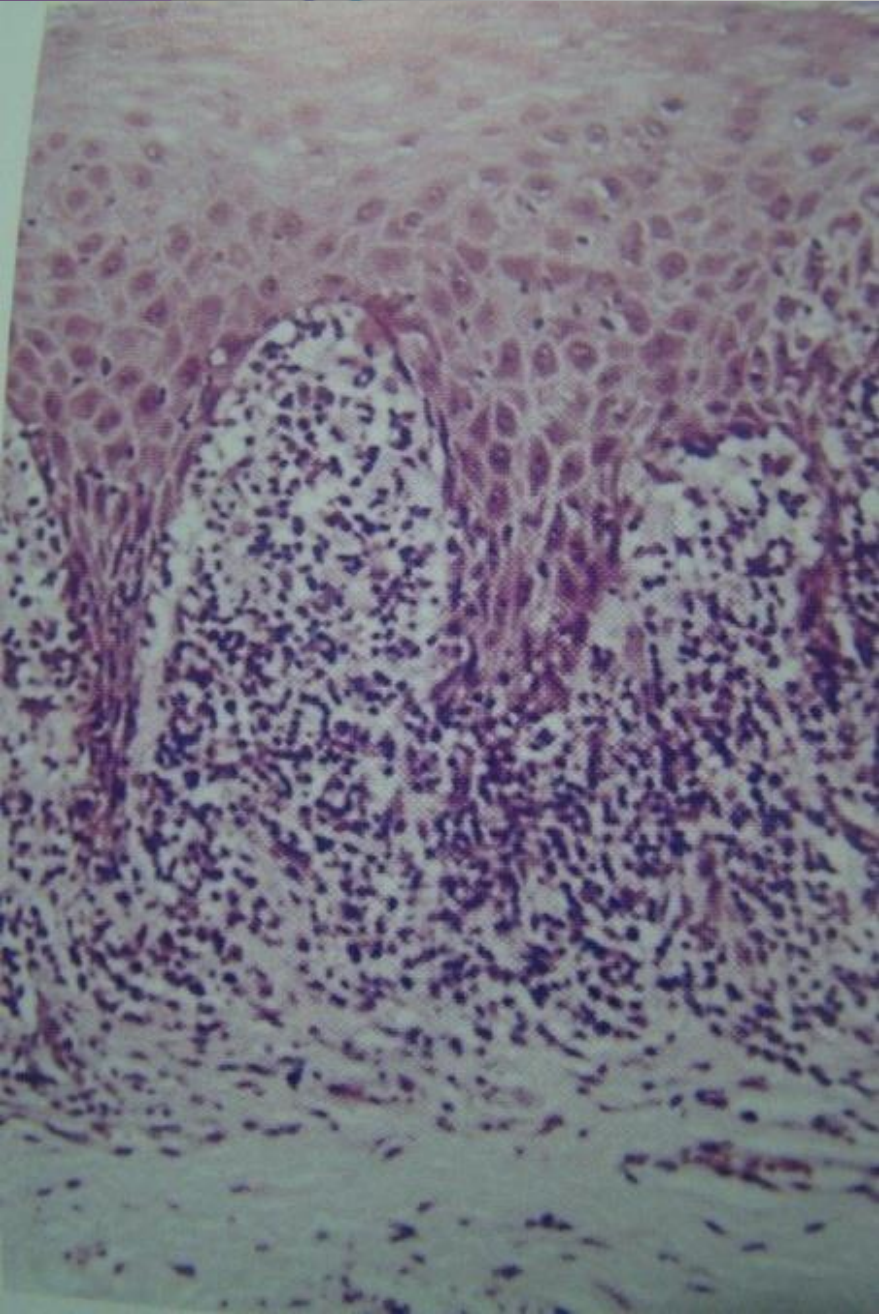
-Acanthotic epi. result in irregular elongation of rete ridges which gives Saw-tooth pattern.

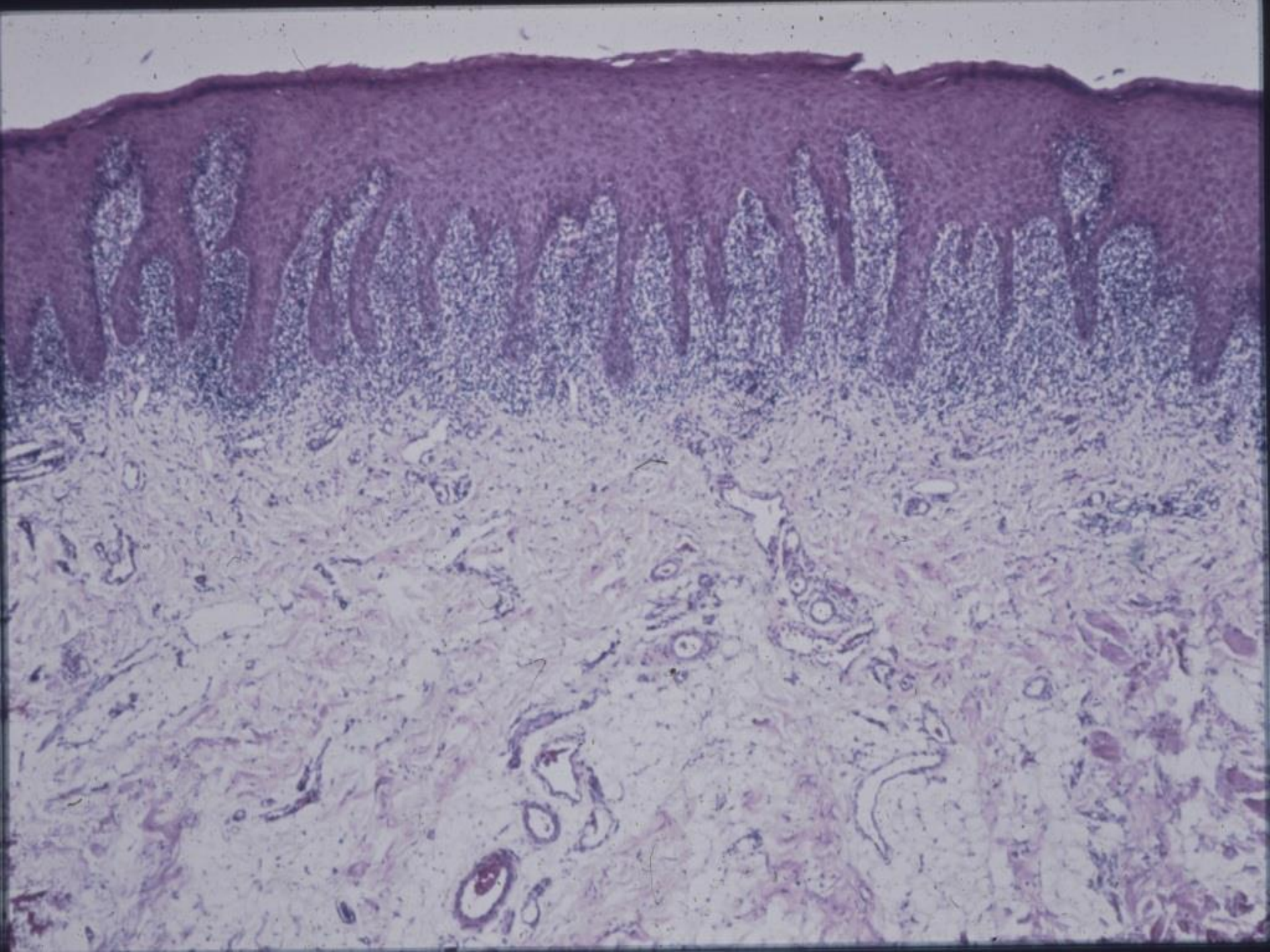
-Dense well defined band of mononuclear

inflammatory cell in sub epi. C.T(mainly T-lymphocyte).

-Characteristic histological finding is degeneration of basal cell layer (liquifactive degeneration), that basal cell layer replaced by hyaline eosinophilic band.







Some patient triggered by hypersensitivity of drugs or dental materials (amalgam) called

Lichenoid reaction

seen mainly on buccal mucosa.

2- **Lupus erythematosus**:- Two forms of this disease:-

1- **Chronic discoid Lupus erythematosus**

(localized form):- **DLE**

- Facial skin may involve,, ,cheek is the commonest oral site,,**discoid area of erythema with keratotic border.**

2- **Systemic (disseminated) :- SLE**

- Involve almost every organ in the body with skin rash, mainly on face as “ **Butter- Fly** ” pattern
- Oral lesion variable.



