Sri Aurobindo College of Dentistry

Indore, Madhya Pradesh

INDIA

MODULE PLAN

- TOPIC :RED AND WHITE LESIONS OF ORAL MUCOSA
- SUBJECT: OMDR
- TARGET GROUP: UNDERGRADUATE DENTISTRY
- MODE: POWERPOINT – WEBINAR
- PLATFORM: INSTITUTIONAL LMS
- PRESENTER: Dr. Tushar Phulambrikar

RED
&
WHITE LESIONS
OF ORAL MUCOSA

Presented By- Dr. Tushar Phulambrikar
(M.D.S)

Lesions most often appear white because of:

1. Thickening of the keratin layer or hyperkeratosis.
2. Acanthosis (a thickening of the spinous cell layer)
3. Intra or Extracellular accumulation of fluid in the epithelium (i.e, leukoedema)
4. Reduced vascularity in the underlying lamina propria.
5. Fungi can also produces white appearance.
6. Immunopathogenesiscan also give the similar kind of appearance.

What was known?

- White lesions in the oral cavity may be benign, pre-malignant or malignant.
- Early diagnosis can minimize progression of oral cancer.

- It can be grouped into five categories according to the pathological process, known as the 5 Is:

1. Inherent (congenital or hereditary, e.g. white sponge nevus)
2. Inflammation (e.g., oral lichen planus, some variants of geographic tongue)
3. Infection (e.g., oral candidiasis)
4. Iatrogenic (e.g., drug-induced lichenoid reaction, frictional hyperkeratosis) and
5. Idiopathic (e.g., oral premalignant lesion or neoplasm).

CLASSIFICATION OF WHITE LESIONS

CLASSIFICATION OF WHITE LESIONS:

- 1. Hereditary/Developmental:
- (a)Leukoedema (b) White spongy nevus
- (c) Hereditary benign intraepithelial dyskeratosis
- (d)Pachyonychia congenita (e)Dyskeratosis congenita

- 2. Reactive:
- (a) Frictional keratosis (b) Morsicatio buccarum
- (c) Nicotine stomatitis (d) Tobacco pouch keratosis
- (e) Chemical burn

- 3. Immunologic:
- (a) Lichen planus (b) Lichenoid mucositis
- (c) Discoid lupus erythematosus
- (d) Graft-versus- host disease

- 4. Bacterial/Viral/Fungal:
- (a) Candidiasis (b) Mucous patches in secondary
- syphilis (c) Oral hairy leukoplakia

- 5. Systemic disease:
- (a) Uremic stomatitis

- 6.Potentially malignant disorders:
- (a) Leukoplakia (b) Actinic cheilitis

- 7. Neoplastic:
- (a) Squamous cell carcinoma

Wood N.K, Goaz P W, Differential Diagnosis of Oral and Maxillofacial Lesions, 5th edition, Elsevier.
Burket L W, Oral Medicine Diagnosis and Treatment, 9th edition, J.B Lippincott Company

SCRAPPABLE LESIONS:

1. Acute pseudomembranous candidiasis
2. Burns

- ✓ Chemical
- ✓ Physical
- ✓ Thermal

3.ANUG

4.Secondary syphilis

5.Diphtheritic lesions.

NON-SCRAPPABLE LESIONS:

1. Leukoplakia
2. Tobacco pouch keratosis
3. Lichen planus
4. Oral submucous fibrosis
5. Traumatic keratosis
6. White sponge nevus
7. Psoriasis

A) HEREDITARY WHITE LESIONS:

1. Leukoedema
2. White sponge nevus
3. Hereditary benign intraepithelial dyskeratosis or whitkop's disease
4. Dyskeratosis congenita

B) DEVELOPMENTAL LESIONS:

1. Ectopic lymphoid tissue or oral lymphoepithelial cyst.

A) KERATOTIC WHITE LESIONS:

1. Stomatitis nicotina
2. Traumatic keratosis
3. Keratosis associated with dental restoration
4. Focal epithelial hyperplasia
5. Psoriasis

B) NON-KERATOTIC WHITE LESIONS:

1. Burns- thermal, chemical and electrical
2. Desquamative gingivitis
3. Pemphigus
4. Diphtheritic lesions
5. Syphilitic mucous patches
6. ANUG
7. Candidiasis
8. Cheek biting

A) WHITE LESIONS WITH DEFINITE PRECANCEROUS POTENTIAL:

1. Leukoplakia
2. Lichen planus
3. Verrucous leukoplakia
4. Submucous fibrosis

B) LESIONS WITHOUT PRECANCEROUS POTENTIAL:

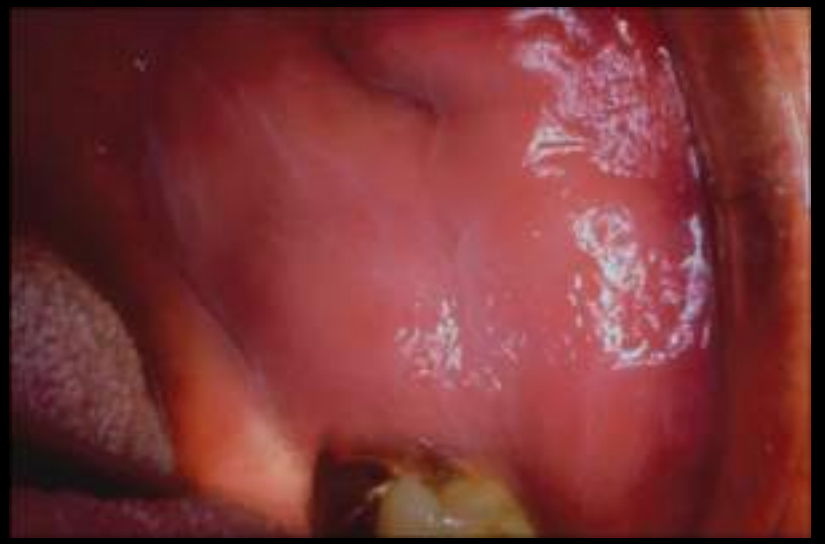
1. Traumatic keratosis
2. Geographic tongue
3. Tobacco pouch keratosis
4. Hereditary white lesions.

HEREDITARY WHITE LESIONS

LEUKOEDEMA

- Common mucosal alteration that represents a variation of the normal condition rather than a true pathologic change.
- 90% of black adults and up to 50% of black teenagers.
- White persons- highly variable (10 to 90%).
- This difference can be attributed to the darker coloration of the mucosa in black persons, rendering the alteration more visible.

- The most frequent site- Buccal mucosa bilaterally.
- Rarely seen on the labial mucosa, soft palate, and floor of the mouth.
- It usually has a faint, white, diffuse, and filmy appearance, with numerous surface folds resulting in wrinkling of the mucosa.
- It cannot be scraped off, and it disappears or fades upon stretching the mucosa.
- Microscopic examination reveals thickening of the epithelium, with significant intracellular edema of the stratum spinosum



TREATMENT

- No treatment is indicated for leukoedema since it is a variation of the normal condition.

REACTIVE
AND
INFLAMMATORY
WHITE LESIONS

Linea Alba (White Line)

- Horizontal streak on the buccal mucosa at the level of the occlusal plane extending from the commissure to the posterior teeth.
- It is a very common finding and is most likely associated with **pressure, frictional irritation, or sucking trauma from the facial surfaces of the teeth.**

CLINICAL FEATURES:

- Linea alba is usually present bilaterally and may be pronounced in some individuals.
- It is more prominent in individuals with **reduced overjet of the posterior teeth.**
- It is often scalloped and restricted to dentulous areas.

TREATMENT:

- No treatment is indicated for patients with linea alba.

Frictional (Traumatic) Keratosis

CLINICAL FEATURES:

- White plaque with a **rough and frayed surface** that is clearly related to an identifiable source of mechanical irritation and that will usually resolve on elimination of the irritant.
- Frictional keratosis is frequently associated with rough or maladjusted dentures and with sharp cusps and edges of broken teeth.

TREATMENT:

- Upon removal of the offending agent, the lesion should resolve within 2 weeks.
- Biopsies should be performed on lesions that do not heal to rule out a dysplastic lesion.

Morsicatio buccarum

- Morsicatio is a condition caused by chronic physical irritation such as continuous biting of the tongue (morsicatio linguarum), buccal mucosa (morsicatio buccarum) or labial mucosa (morsicatio labiorum)

•Morsicatio' represents changes of surface features of the tongue, buccal mucosa, or lips caused by chronic tissue irritation, such as biting or sucking.

•It is a self-induced injury caused by habitual behaviors, which may be associated with stress or mental illness in some patients.

- It can be misdiagnosed when missing a prudent history taking.
- We herein report three cases of this condition, with emphasis on habit-related histories such as self-mutilation.

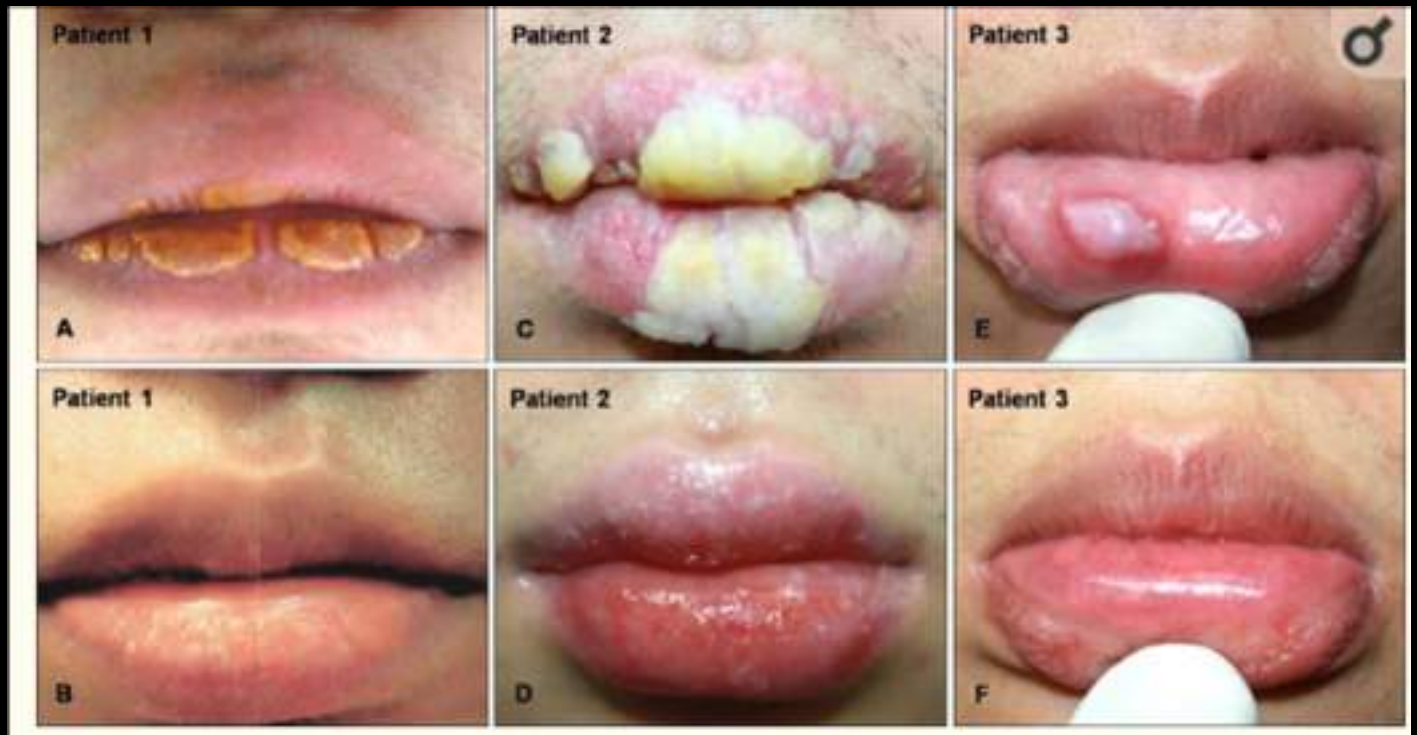


Fig 1

(A, C) Localized yellow hyperkeratotic plaques on the upper and lower lips in patients 1 & 2. (E) A well-demarcated, white, smooth plaque on the lower lip in patient 3. (B, D, F) The lesions had completely disappeared after the patients retained from their lesion causing habits.

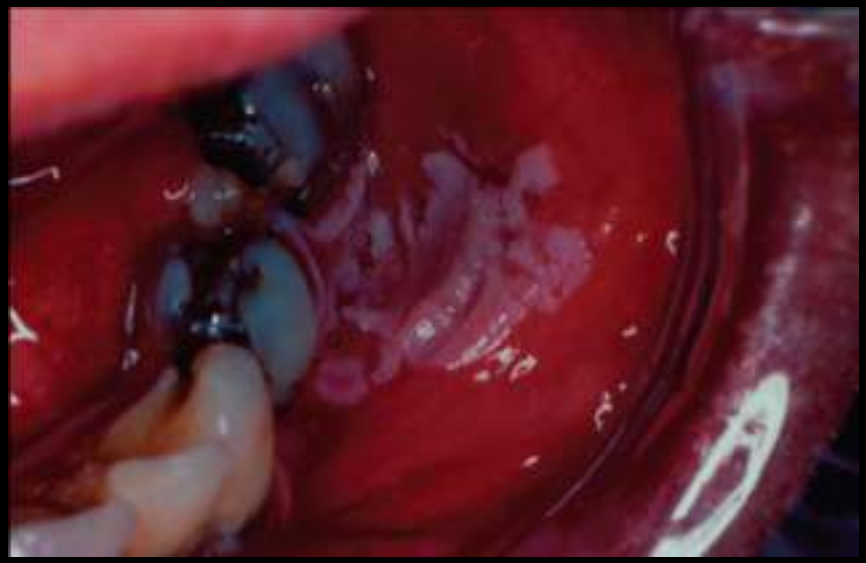
Journal of annals of dermatolgy, 2012

Chemical Injuries of the Oral Mucosa

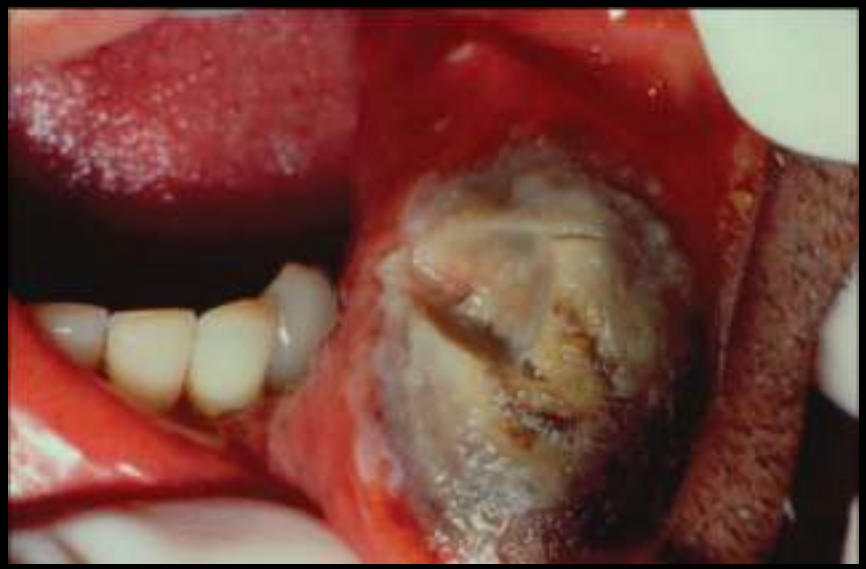
- Transient nonkeratotic white lesions of the oral mucosa are often a result of chemical injuries caused by a variety of agents that are caustic when retained in the mouth for long periods of time, such as **aspirin, silver nitrate, formocresol, sodium hypochlorite, paraformaldehyde, dental cavity varnishes, acidetching materials, and hydrogen peroxide.**

- The white lesions are attributable to the formation of a superficial pseudomembrane composed of a necrotic surface tissue and an inflammatory exudate.

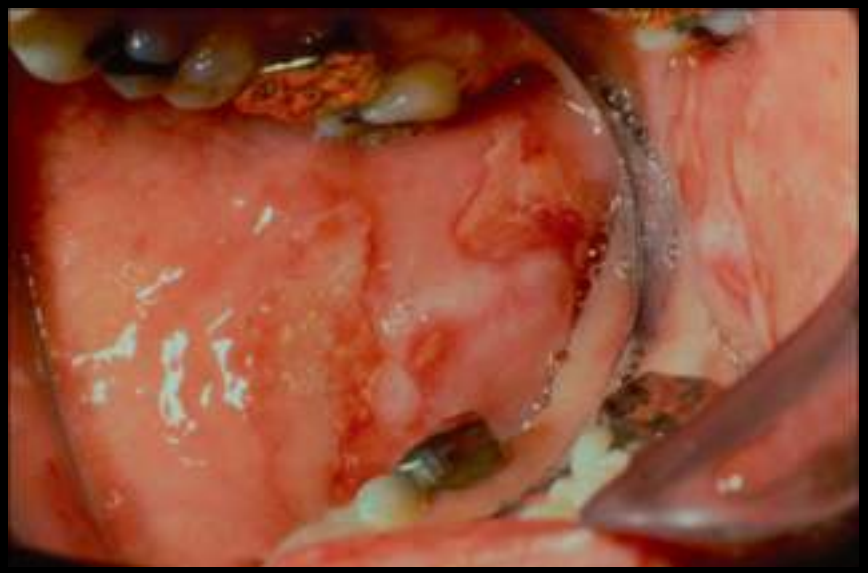
Aspirin burn, creating a pseudomembranous necrotic white area.



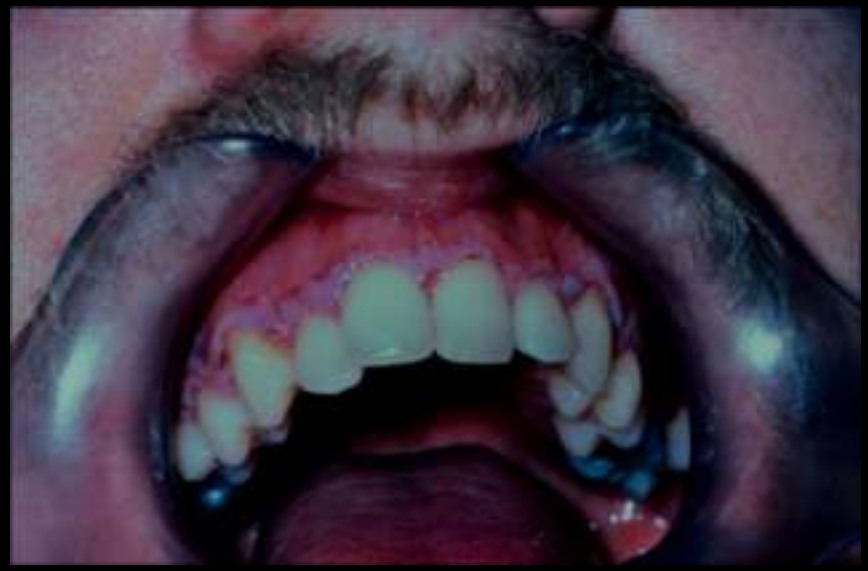
Extensive tissue necrosis caused by injudicious use of silver nitrate.



Unusual sensitivity reaction with severe ulcerations and sloughing of the mucosa has been reported to have been caused by a cinnamon-flavored dentifrice

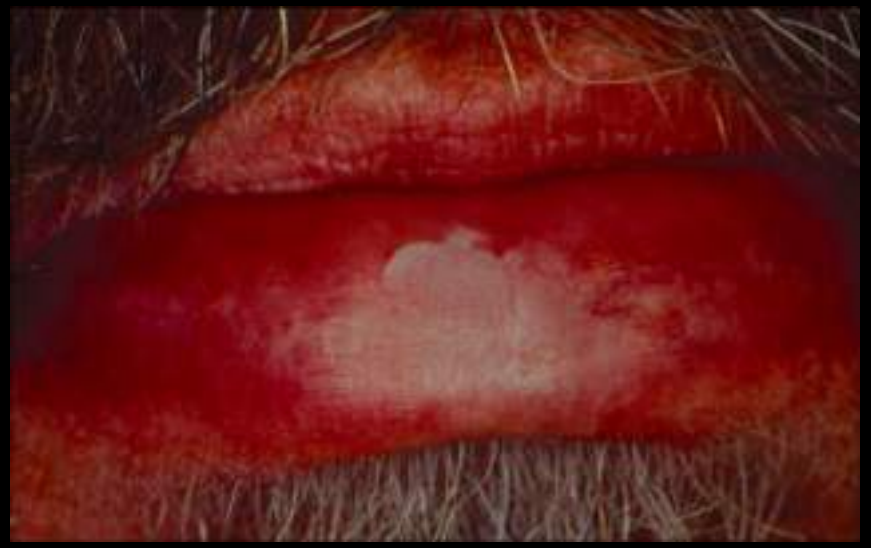


Diffuse slough of marginal gingivae due to misuse of commercial mouthwash.



Actinic Keratosis (Cheilitis)

- Actinic (or solar) keratosis is a **premalignant epithelial lesion** that is directly related to long-term sun exposure.
- These lesions are classically found on the **vermilion border of the lower lip** as well as on other sun-exposed areas of the skin.
- A small percentage of these lesions will transform into squamous cell carcinoma.



WHY SHOULD BE CONCERNED ??

- Though the vast majority of actinic keratoses remain benign, they reveal that you have sustained sun damage and could develop any kind of skin cancer, especially the second most common form of the disease, squamous cell carcinoma (SCC).

Smokeless Tobacco–Induced Keratosis

- **Other names:** Smokeless tobacco keratosis, snuff dipper's keratosis, or tobacco pouch keratosis.

- Dipping snuff results in the development of a well-recognized white mucosal lesion in the area of tobacco contact.

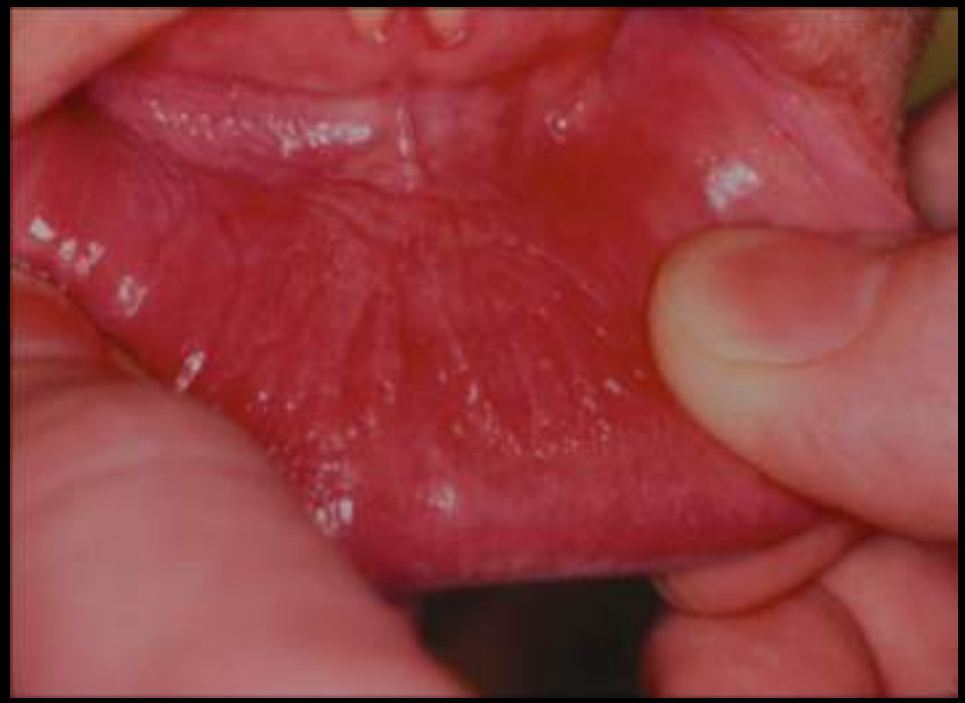
- Much lower risk of malignant transformation.

- Most changes associated with the use of smokeless tobacco are seen in the area contacting the tobacco.

- The most common area of involvement is the anterior mandibular vestibule, followed by the posterior vestibule.

- The surface of the mucosa appears white and is granular or wrinkled

- A folded character may be seen (tobacco pouch keratosis).



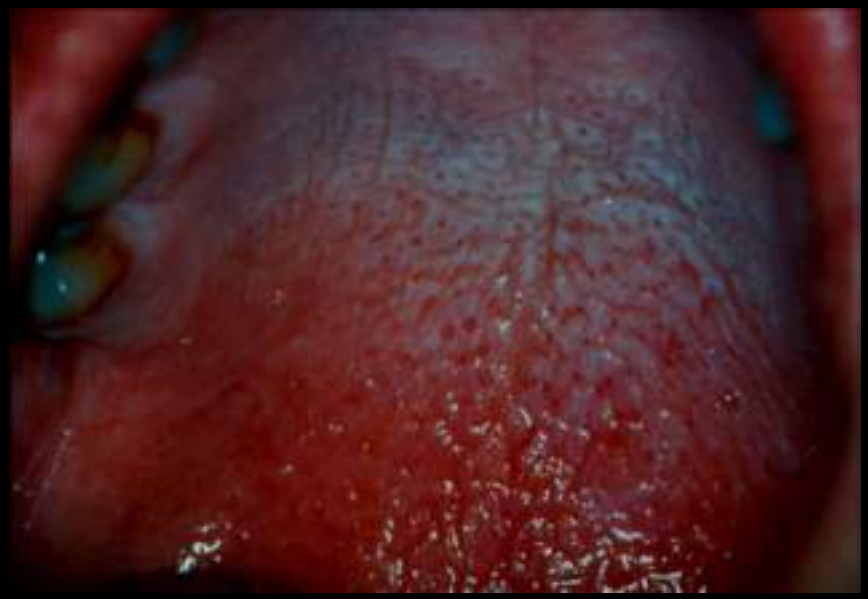
- Commonly noted is a characteristic area of gingival recession with periodontal-tissue destruction in the immediate area of contact.

- Cessation of use almost always leads to a normal mucosal appearance within 1 to 2 weeks.

NICOTINE STOMATITIS

- *Synonyms:* Stomatitis nicotina palati, smoker's palate.

- White lesion that develops on the hard and soft palate in heavy cigarette, pipe, and cigar smokers.



- The lesions are restricted to areas that are exposed to a relatively concentrated amount of hot smoke during inhalation.

INFECTIOUS WHITE LESIONS AND WHITE & RED LESIONS

Oral Hairy Leukoplakia

- Oral hairy leukoplakia is a corrugated white lesion that usually occurs on the **lateral or dorsal surfaces of the tongue and also on the buccal mucosa** in patients with severe immunodeficiency.
- The most common disease associated with oral hairy leukoplakia is **HIV infection(prevalance is ass high as 80% in AIDS pt.)**.
- **Epstein-Barr virus (EBV)** is implicated as the causative agent kia

D/D

- Hyperplastic candidiasis
- Idiopathic leukoplakia
- Frictional keratosis induced by tongue chewing

CANDIASIS

Candidiasis

- Multiplicity of diseases caused by a yeast like fungus, ***Candida.***
- Most common oral fungal infection in humans.
- It is the most prevalent opportunistic infection affecting the oral mucosa.

Predisposing factors

1. Marked changes in oral microbial flora (due to the use of antibiotics [especially broad-spectrum antibiotics] excessive use of antibacterial mouth rinses).
2. Chronic local irritants (dentures and orthodontic appliances)
3. Administration of corticosteroids (aerosolized inhalant and topical agents are more likely to cause candidiasis than systemic administration)
4. Poor oral hygiene
5. Pregnancy

6. Immunologic deficiency:

— Congenital or childhood (chronic familial mucocutaneous candidiasis ± endocrine candidiasis syndrome [hypoparathyroidism, hypoadrenocorticism], and immunologic immaturity of infancy)

— Acquired or adult (diabetes, leukemia, lymphomas, and AIDS)

— Iatrogenic (from cancer chemotherapy, bone marrow transplantation, and head and neck radiation)

7. Malabsorption and malnutrition

Candida

- *Candida* species are normal inhabitants of the oral flora of many individuals, but are present in the mouth of the healthy carrier in a low concentration of 200 to 500 cells per milliliter of saliva.

Species

- *Candida albicans*
- *Candida tropicalis*
- *Candida glabrata*
- *Candida parapsilopsis*
- *Candida guilliermondii*
- *Candida krusei*
- *Candida pseudotropicalis*

CLASSIFICATION

a) Acute

- Pseudomembranous
- Atrophic (erythematous) - Antibiotic stomatitis

(b) Chronic

- Atrophic
  - Denture sore mouth
  - Angular cheilitis
  - Median rhomboid glossitis
- Hypertrophic/hyperplastic
  - Candidal leukoplakia
  - Papillary hyperplasia of the palate
  - Median rhomboid glossitis (nodular)
- Multifocal

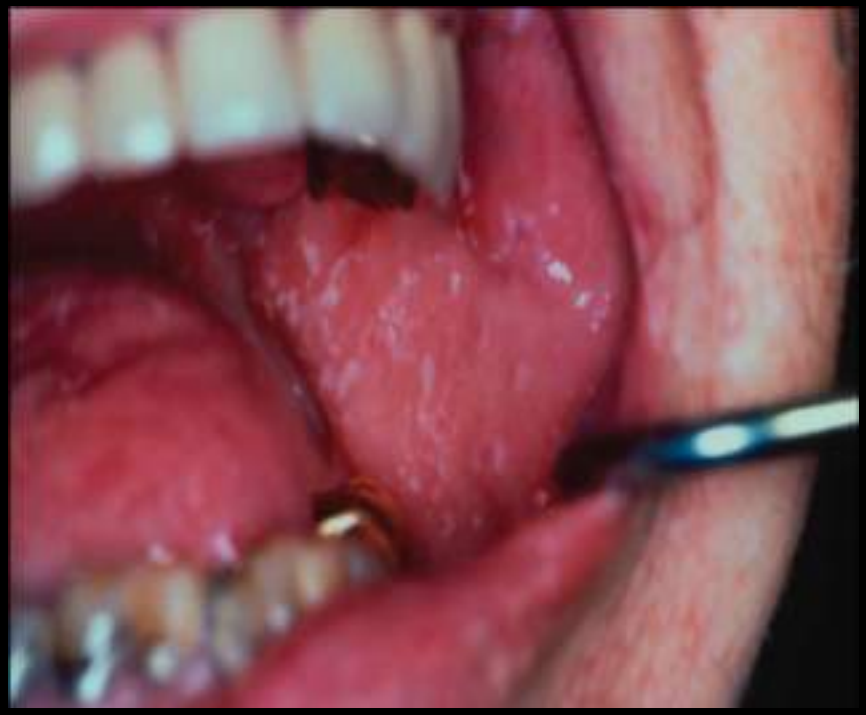
(c) Mucocutaneous

- Syndrome associated
  - Familial +/– endocrine candidiasis syndrome
  - Myositis (thymoma associated)
- Localized
- Generalized (diffuse)

(d) Immunocompromise (HIV) associated

Acute pseudomembranous candidiasis

- Is the prototype of the oral infections caused by *Candida*.
- It is a superficial infection of the outer layers of the epithelium, and it results in the formation of patchy white plaques or flecks on the mucosal surface
- Removal of the plaques by gentle rubbing or scraping usually reveals an area of erythema or even shallow ulceration.

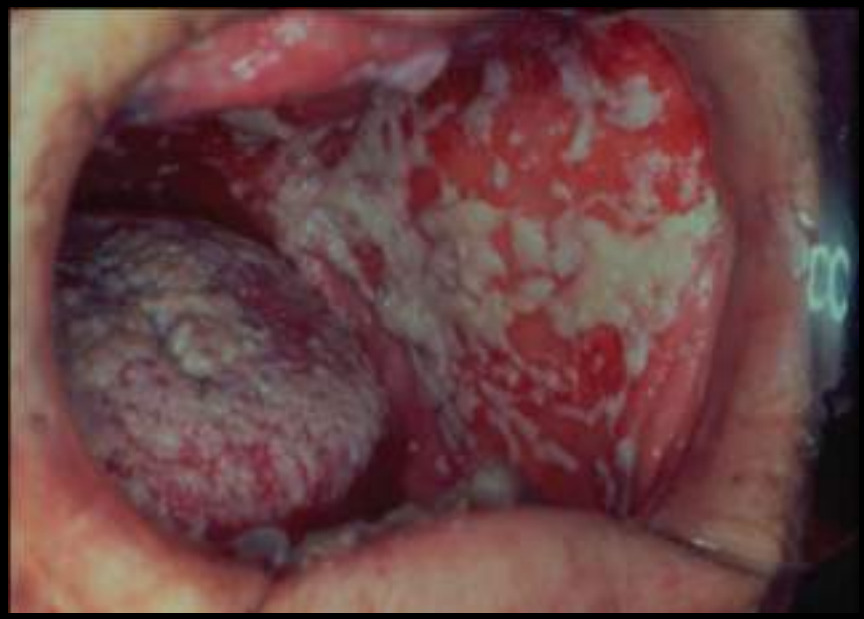


Acute pseudomembranous candidiasis (thrush)

- Thrush is seen in children and in adults of all ages whenever the number of *Candida* organisms in the oral cavity increases significantly.
- The typical lesions in infants are described as soft white adherent patches on the oral mucosa, which are generally painless and can be removed with little difficulty.
- In the adult, inflammation, erythema, and painful eroded areas are present, and the typical pearly white plaque like lesions are relatively inconspicuous at times

Acute pseudomembranous candidiasis (thrush)

- Any mucosal surface may be involved.
- The lesions may involve the entire oral mucosa or may involve relatively localized areas where normal cleansing mechanisms are poor.



Symptoms

- A prodromal symptom of a rapid onset of a bad taste and the loss of taste discrimination is present.
- A burning sensation of the mouth and throat may also precede the appearance of the white pseudo membranous lesions.
- Symptoms of this type in a patient receiving broad-spectrum antibiotics are strongly suggestive of thrush or other forms of oral candidiasis.

Differential diagnosis

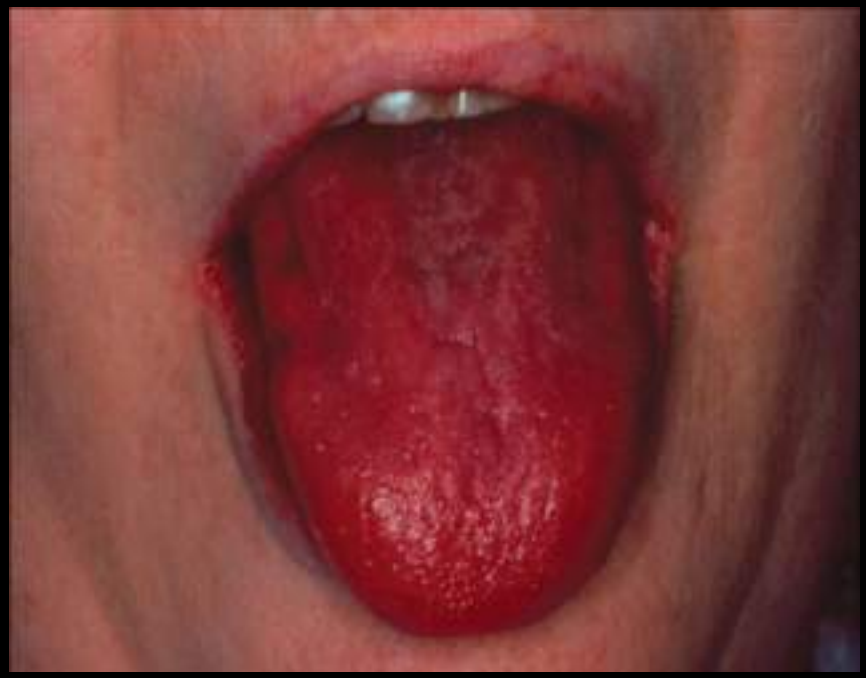
- Food debris
- Habitual cheek biting
- Genetically determined epithelial abnormality such as white sponge nevus.

Diagnosis

- Exfoliative cytology
- Histopathology

Acute atrophic candidiasis

- Red patch of atrophic or erythematous raw and painful mucosa, with minimal evidence of the white pseudomembranous lesions observed in thrush.
- Predominantely seen in the palate and dorsum of the tongue



Antibiotic sore mouth

- Antibiotic sore mouth, a common form of atrophic candidiasis, should be suspected in a patient who develops symptoms of oral burning, bad taste, or sore throat during or after therapy with broad-spectrum antibiotics.
- Patients with chronic iron deficiency anemia may also develop atrophic candidiasis.

Chronic atrophic candidiasis (CAC)

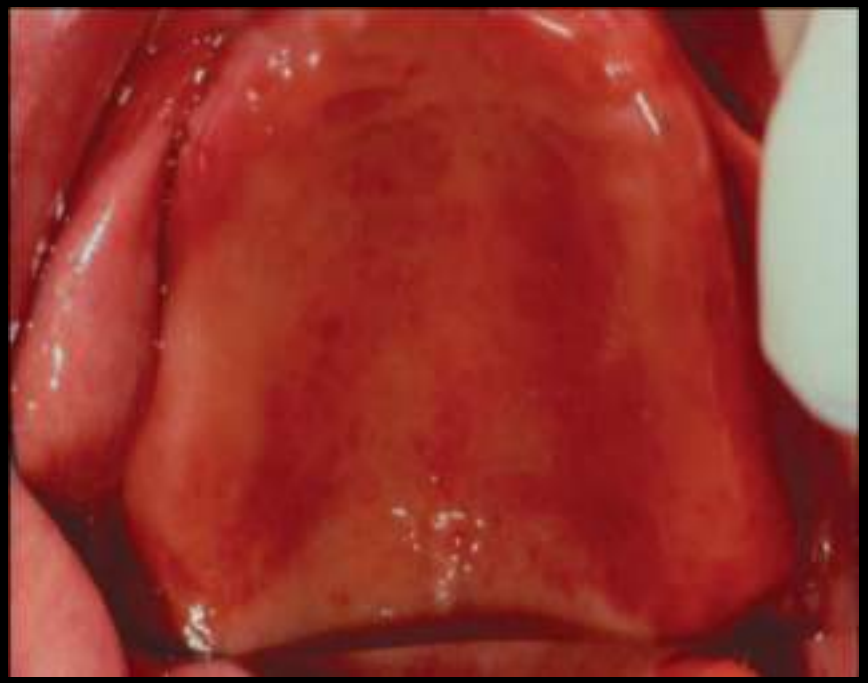
- Include :-
  - Denture stomatitis (denture sore mouth)
  - Angular cheilitis
  - Median rhomboid glossitis.

Denture stomatitis (denture sore mouth)

- Common form of oral candidiasis.
- Manifests as a diffuse inflammation of the maxillary denture-bearing areas and that is often (15 to 65% of cases) associated with angular cheilitis.

Stages of denture stomatitis

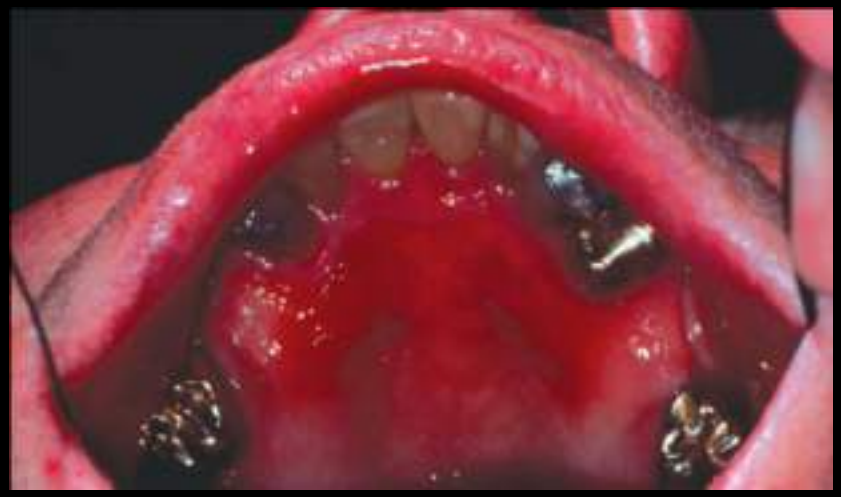
- 3 stages:
- 1st stage- consists of numerous palatal petechiae.

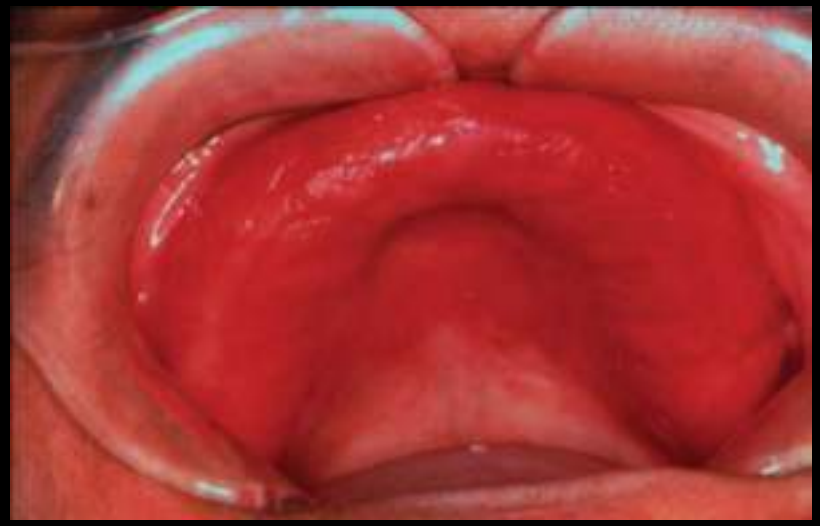


Stages of denture stomatitis

- 2nd stage-

  More diffuse erythema involving most (if not all) of the denture- covered mucosa

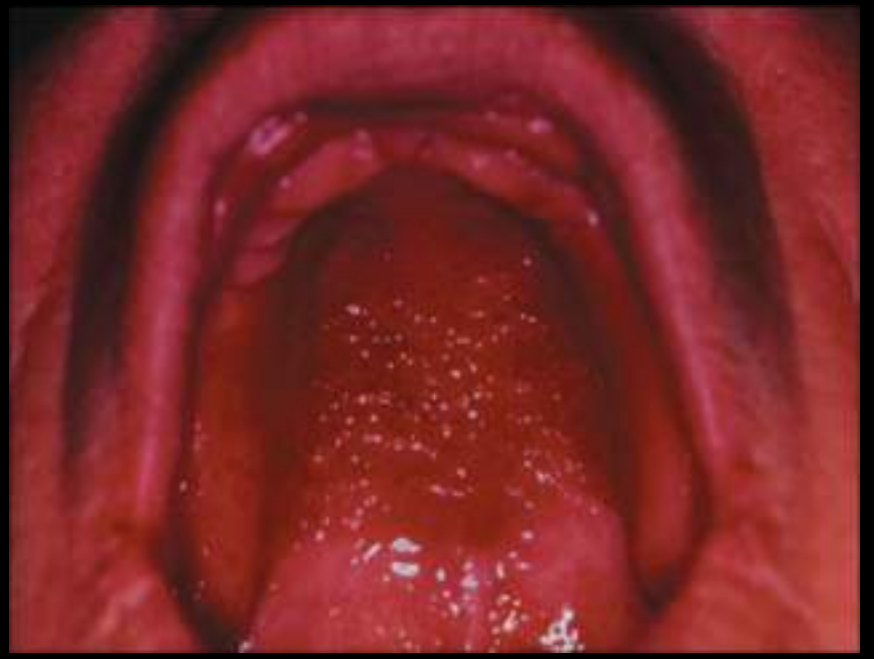




Stages of denture stomatitis

- **3rd stage-**

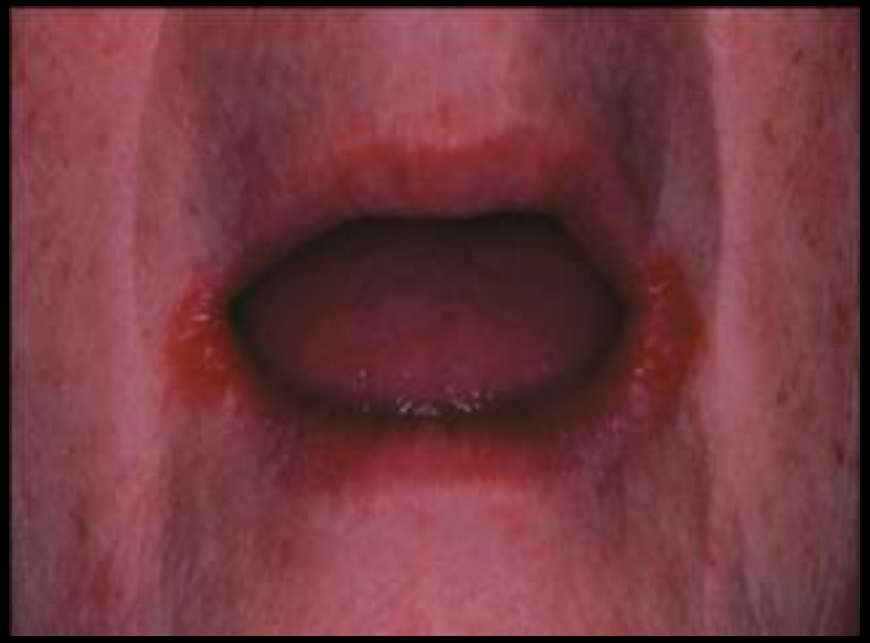
  Includes the development of tissue granulation or nodularity (papillary hyperplasia) commonly involving the central areas of the hard palate and alveolar ridges.



- Yeast attached to the denture plays an important etiologic role in chronic atrophic candidiasis.
- The attachment of yeast to the patient's appliances is increased by mucus and serum and decreased by the presence of salivary pellicle.
- Rinsing the appliance with a dilute (10%) solution of household bleach, soaking it in boric acid, or applying nystatin cream before inserting the denture will eliminate the yeast.
- Disinfection of the appliance is an important part of the treatment of denture sore mouth.
- Soft liners in dentures provide a porous surface and an opportunity for additional mechanical locking of plaque and yeast to the appliance

Angular cheilitis

- Infection involving the **lip commissures.**
- Frequently seen in association with denture stomatitis & rarely seen in patients with natural dentition.

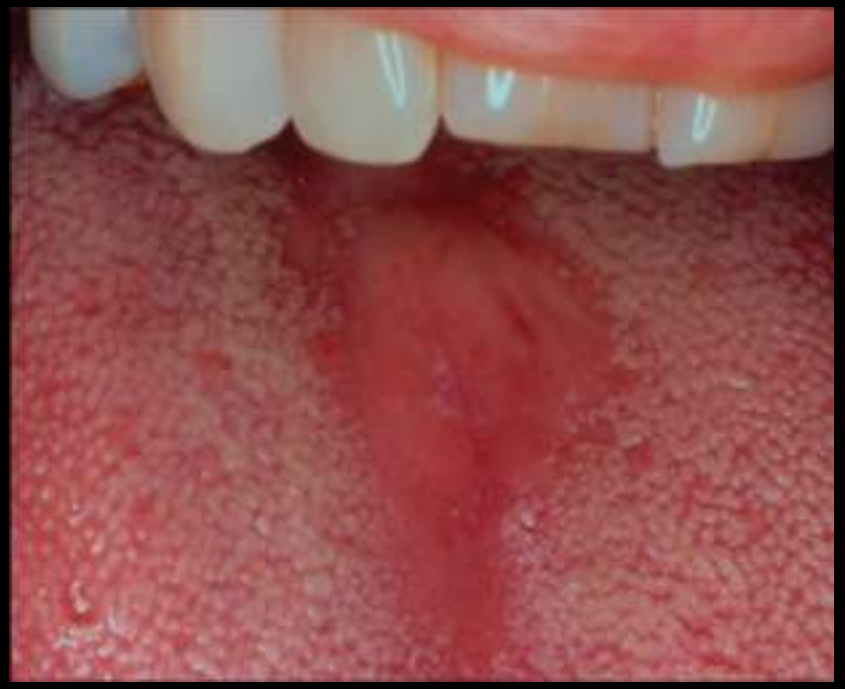


Angular cheilitis

- Other possible etiologic cofactors include:
  - Reduced vertical dimension
  - Nutritional deficiency (iron deficiency anemia and vitamin B or folic acid deficiency) sometimes referred to as perlèche
  - Diabetes, neutropenia, and AIDS, as well as co-infection with *Staphylococcus* and beta-hemolytic *Streptococcus*.
- More-extensive desquamative lesions affecting the full width of the lip and sometimes extending to the adjacent skin are associated with habitual lip sucking and chronic *Candida* infection.

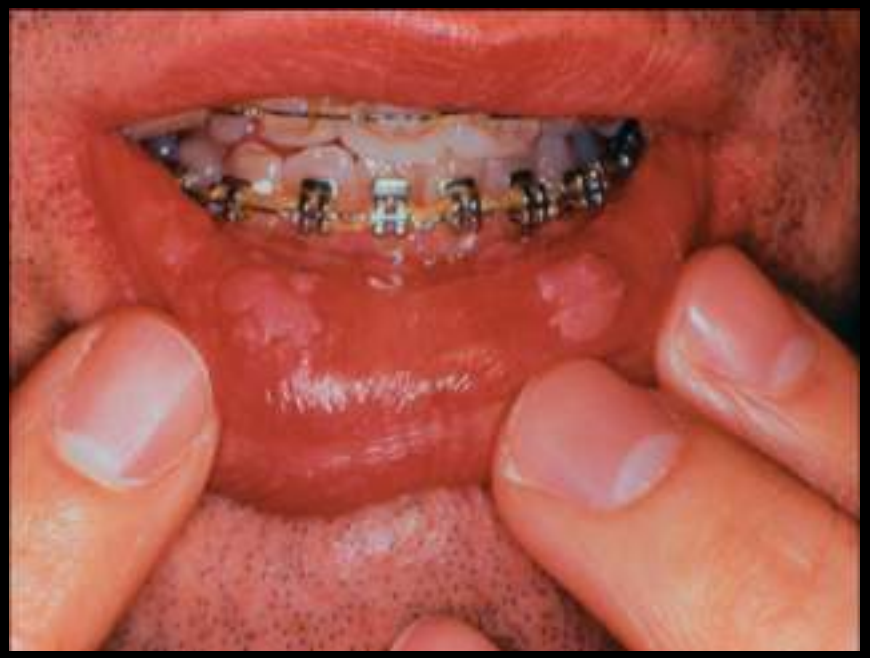
Median rhomboid glossitis

- Erythematous patches of atrophic papillae located in the **central area of the dorsum of the tongue.**
- When these lesions become more nodular, the condition is referred to as **hyperplastic** median rhomboid glossitis.
- These lesions were originally thought to be **developmental in nature** but are now considered to be a manifestation of chronic candidiasis.



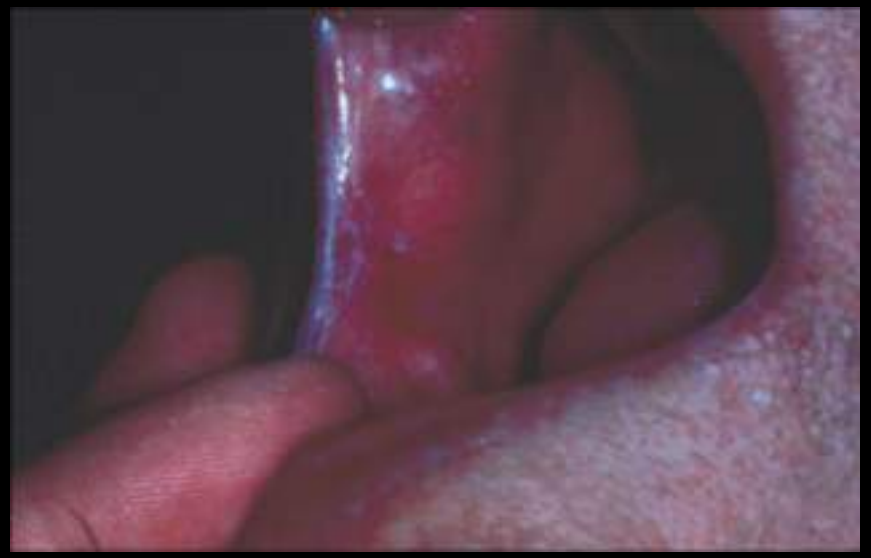
Chronic hyperplastic candidiasis (CHC)

- Mycelial invasion of the deeper layers of the mucosa and skin occurs, causing a proliferative response of host tissue.



Candidal leukoplakia

- *Candidal leukoplakia* is considered a chronic form of oral candidiasis in which firm white leathery plaques are detected on the cheeks, lips, palate, and tongue.



- Differentiated from other varieties of leukoplakia by PAS staining.

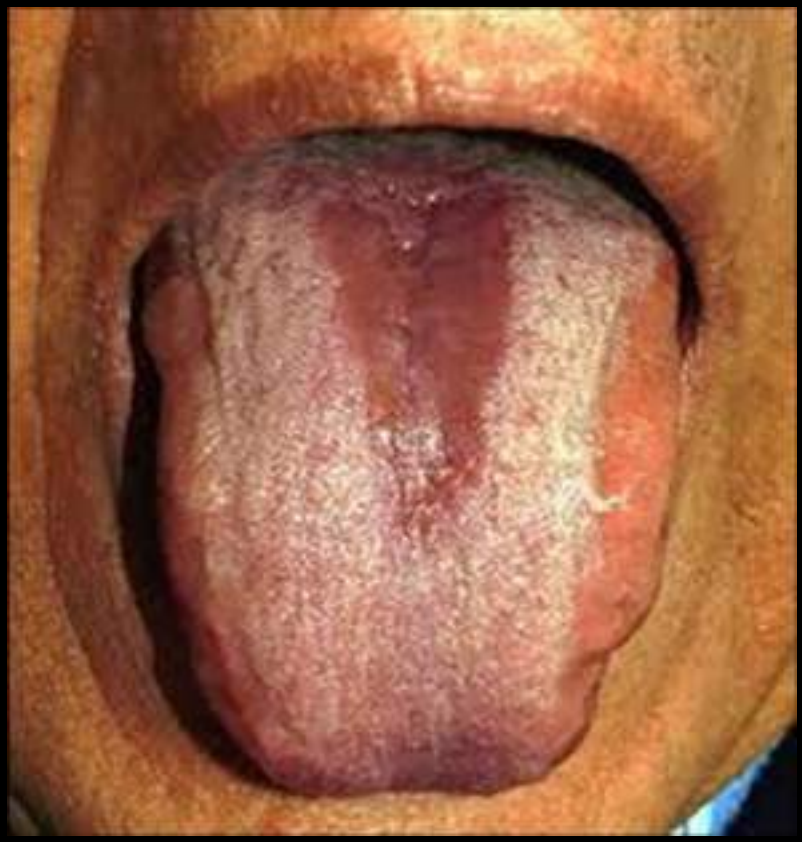
Candidal leukoplakia/ Chronic plaque type

- Approximately 10% of oral leukoplakias satisfy the clinical and histologic criteria for CHC.

- Epithelial dysplasia occurs four to five times more frequently in candidal (speckled) leukoplakia than in leukoplakia in general.

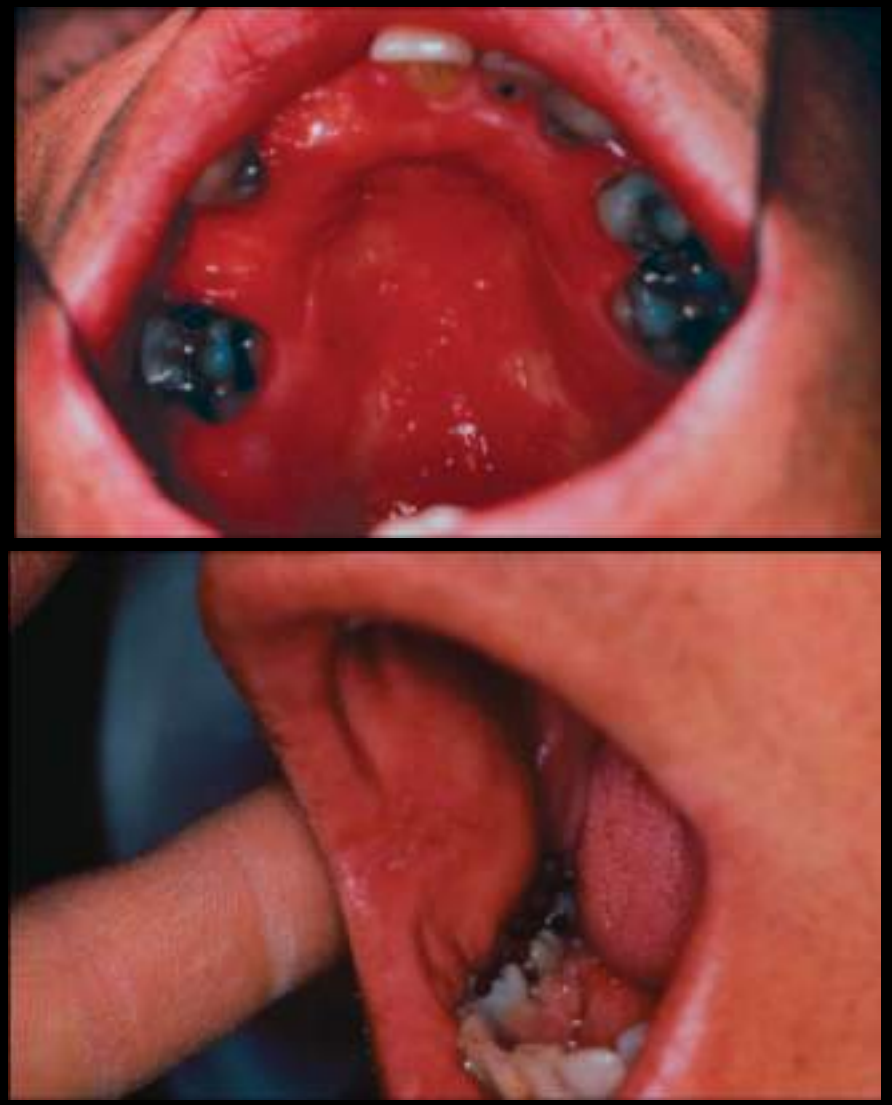
Median rhomboid glossitis (nodular)

- CHC also occurs on the dorsum of the tongue and may resemble median rhomboid glossitis.

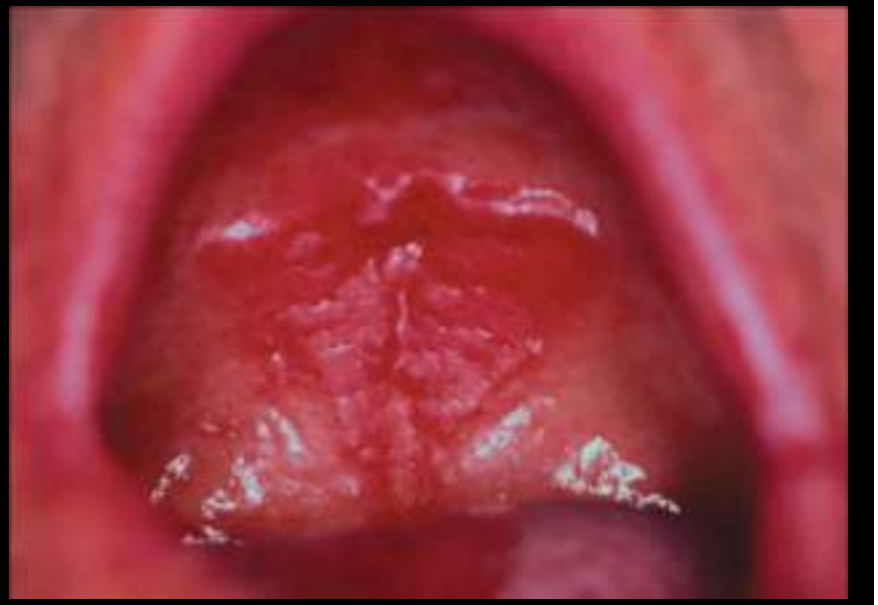


Chronic multifocal candidiasis

- Patients may present with multiple areas of chronic atrophic candidiasis.
- These are most often seen in immunocompromised individuals or in patients with predisposing factors such as ill-fitting dentures.



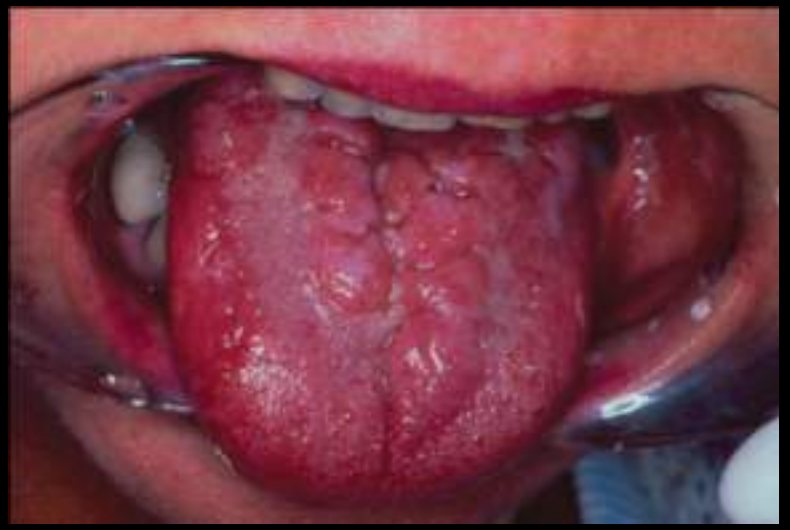
- The changes frequently affect the dorsum of the tongue and midline of the hard palate (kissing lesions), commissure area (angular cheilitis), and denturebearing mucosal surfaces.

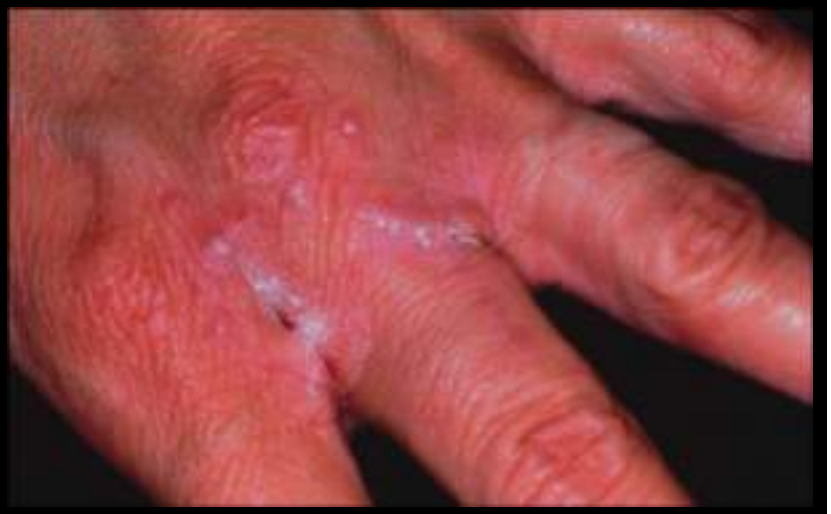


- Smoking may also play an important role in immunocompetent patients.

Chronic mucocutaneous candidiasis

- Persistent infection with *Candida* usually occurs as a result of a defect in cell-mediated immunity or may be associated with iron deficiency.
- Hyperplastic mucocutaneous lesions, localized granulomas, and adherent white plaques on affected mucous membranes are the prominent lesions that identify chronic mucocutaneous candidiasis (CMC)





Oral Candiasis Associated with HIV

- Oral candidiasis is the most frequent opportunistic infection associated with immunocompromised individuals.

- Patients who are on immunosuppressive drug regimens or who have HIV infection,cancer, or hematologic malignancies have an increased susceptibility to oral candidiasis.

Treatment of oral candidiasis

- **Topical application:** Clotrimazole (imidazole derivative), Nystatin
- **Systemic therapy:** Use of any one of these three: ketoconazole, itraconazole and fluconazole.
- **Intravenous administration:** Fluconazole and amphotericin B for the treatment of the resistant lesions of CMC and systemic candidiasis.

Treatment of oral candidiasis

- The majority of acute oral *Candida* infections respond rapidly to topical nystatin and will not recur, provided that the predisposing factors have also been eliminated.
- Seven to 21 days use of a nystatin rinse three to four times daily is usually adequate although some resistant cases may require a second course of treatment.
- Nystatin in cream form may also be applied directly to the denture or to the corners of the mouth.
- Clotrimazole troches can also be used for treatment of oral lesions.

Treatment of oral candidiasis

- Once-daily dose of 200 mg of ketoconazole, 100 mg of fluconazole, or itraconazole oral suspension (100 to 200 mg/d) for 2 weeks.
- When these medications are used for this short period, side effects such as increased liver enzymes, abdominal pain, and pruritus are rare.
- Fluconazole interacts with a number of other medications and must be prescribed with care for patients who are using anticoagulants, phenytoin, cyclosporine, and oral hypoglycemic agents.

IDIOPATHIC “TRUE” LEUKOPLAKIA

•**The term ‘Leukoplakia’ literally means ‘white plaque’ and it was first used by Schwimmer in 1877 to describe a white lesion of the tongue which probably represented a Syphilitic glossitis.**

The definition of leukoplakia has changed over the time.

- **Leukoplakia is defined as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease" (WHO, 1978).**

- **The WHO working group redefined leukoplakia in 2007 as "the term leukoplakia should be used to recognize white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer."**

Etiology

- A number of locally acting etiologic agents, including tobacco, alcohol, candidiasis, electrogalvanic reactions, and (possibly) herpes simplex and papillomaviruses, have been implicated as causative factors for leukoplakia.

- True leukoplakia is most often related to tobacco usage

- **Alcohol consumption** alone is not associated with an increased risk of developing leukoplakia, but alcohol is thought to serve as a promoter that exhibits a strong synergistic effect with tobacco, relative to the development of leukoplakia and oral cancer.

- **Sunlight (specifically, ultraviolet radiation)** is well known to be an etiologic factor for the formation of leukoplakia of the vermilion border of the lower lip.

- **Candida albicans** *is frequently found in histologic sections* of leukoplakia and is consistently (60% of cases) identified in nodular leukoplakias but rarely (3%) in homogeneous leukoplakias.

- **Human papillomavirus (HPV)**, particularly subtypes HPV-16 and HPV-18, have been identified in some oral leukoplakias.

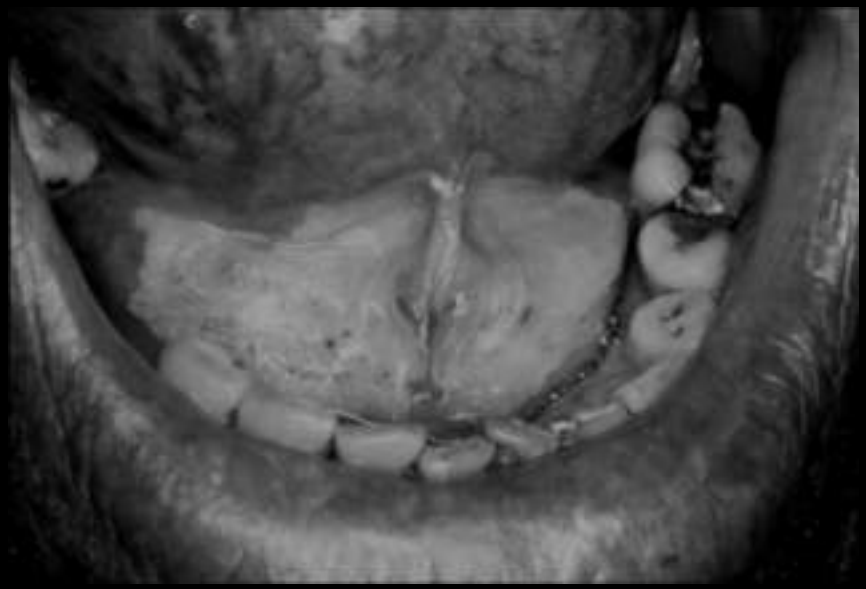
Subtypes

- **“Homogeneous leukoplakia”** (or “thick leukoplakia”)- Well-defined white patch, localized or extensive, that is slightly elevated and that has a fissured, wrinkled, or corrugated surface.

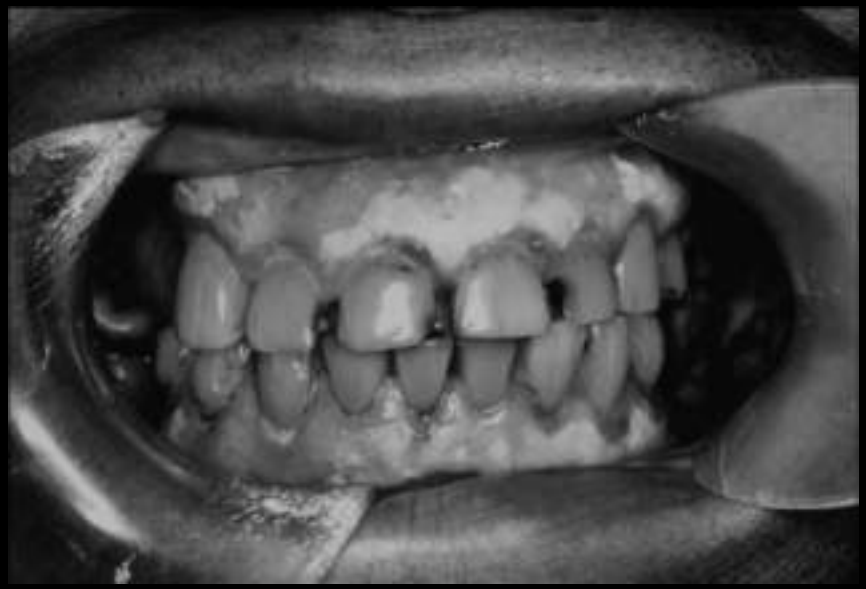
- On palpation, these lesions may feel leathery to “dry, or cracked mud-like.”

“Homogeneous leukoplakia”

Floor of mouth



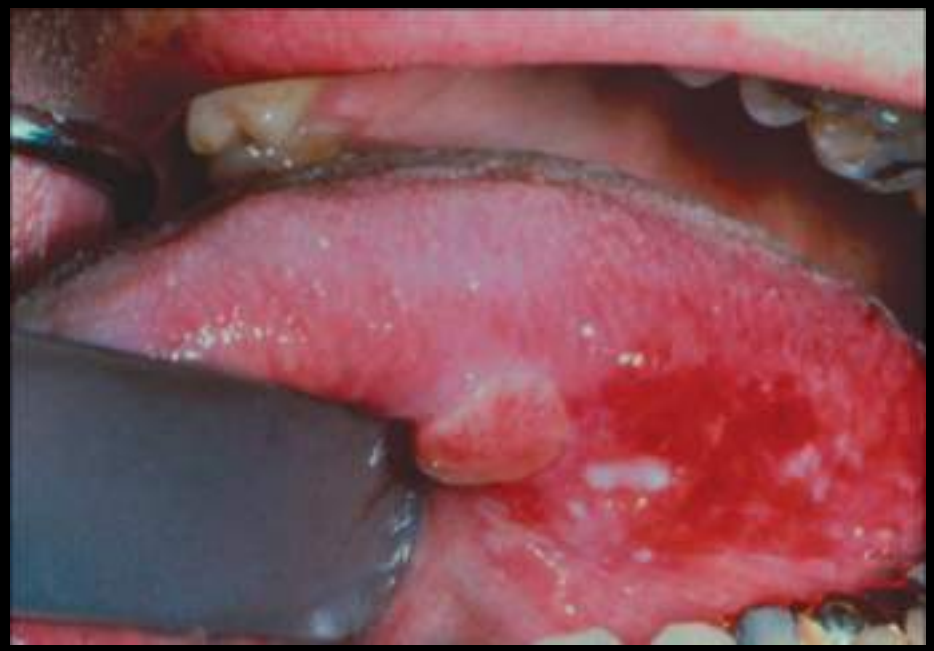
Gingiva



- **Nodular (speckled) leukoplakia** is granular or nonhomogeneous. The name refers to a mixed red-and-white lesion in which keratotic white nodules or patches are distributed over an atrophic erythematous background.

- This type of leukoplakia is associated with a higher malignant transformation rate.

- Nodular or speckled leukoplakia appears as a red velvety plaque with associated white spots or papules on the lateral border of the tongue.



Verrucous leukoplakia” or “verruciform leukoplakia”

- It is a term used to describe the presence of thick white lesions with papillary surfaces in the oral cavity.
- These lesions are usually heavily keratinized and are most often seen in older adults in the sixth to eighth decades of life. Some of these lesions may exhibit an exophytic growth pattern.
- Verrucous carcinoma is almost always a slow growing and well-differentiated lesion that seldom metastasizes.

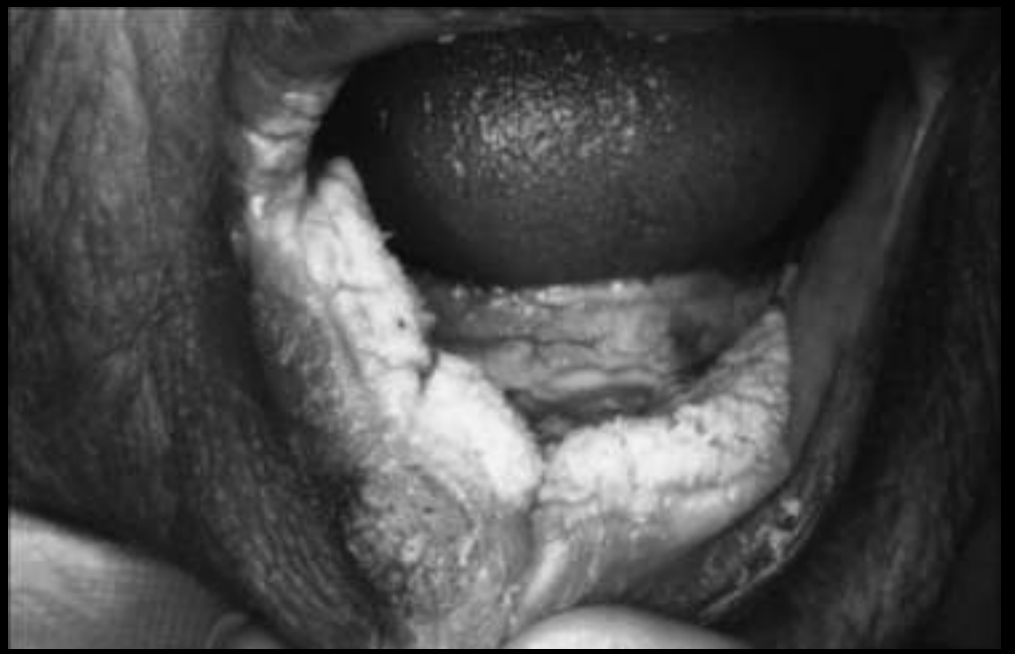
Proliferative verrucous leukoplakia (PVL)

- Extensive papillary or verrucoid white plaques that tend to slowly involve multiple mucosal sites in the oral cavity and to inexorably transform into squamous cell carcinomas over a period of many years.

- PVL has a very high risk for transformation to dysplasia, squamous cell carcinoma or verrucous carcinoma.

- Proliferative verrucous leukoplakia of the floor of the mouth and of the lip.

- In this form of leukoplakia, the risk for malignant transformation is very high.



Diagnosis and Management

- A diagnosis of leukoplakia is made when adequate clinical and histologic examination fails to reveal an alternative diagnosis and when characteristic histopathologic findings for leukoplakia are present.

- Important clinical criteria include location, appearance, known irritants, and clinical course.

- Many white lesions can mimic leukoplakia clinically and should be ruled out before a diagnosis of leukoplakia is made

D/D

1. Lichen planus
2. Lesions caused by cheek biting, frictional keratosis
3. Smokeless tobacco–induced keratosis
4. Nicotinic stomatitis
5. Leukoedema
6. White sponge nevus

BIOPSY

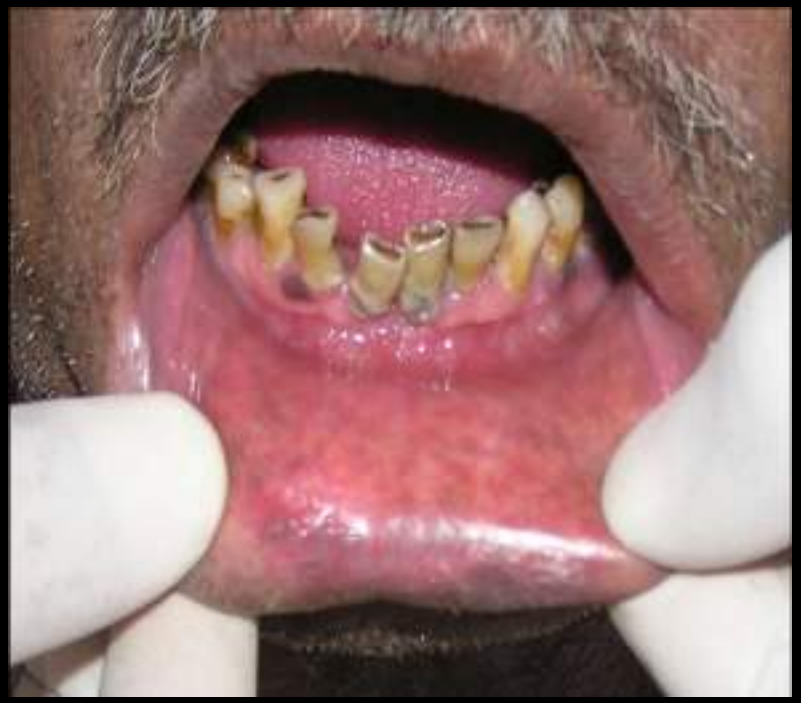
Figure 10-71 • **Moderate epithelial dysplasia.** Dysplastic changes extend to the midpoint of the epithelium and are characterized by nuclear hyperchromatism, pleomorphism, and cellular crowding.

MILD
DYSPLASIA

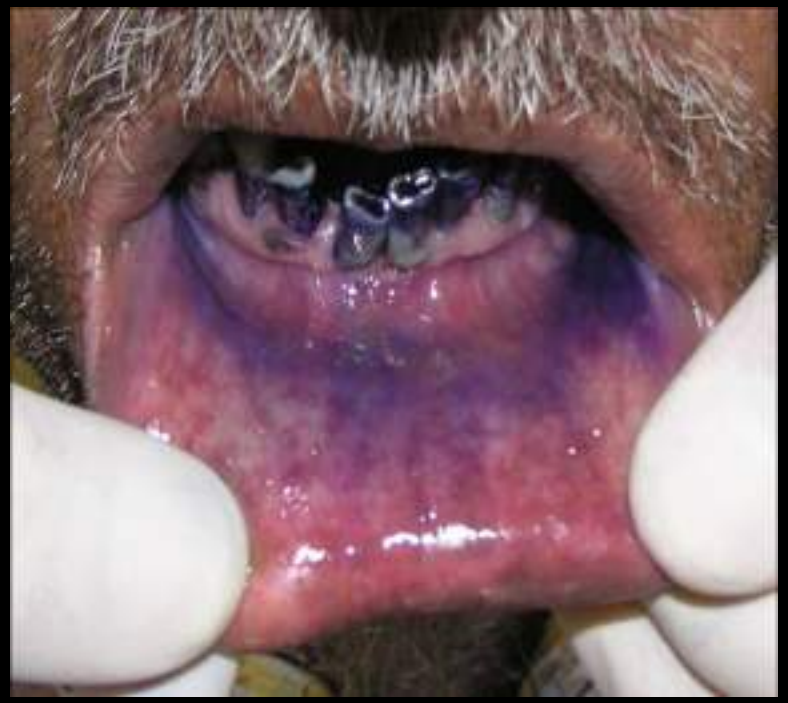
Figure 10-72 • **Severe epithelial dysplasia.** Cellular crowding and disordered arrangement are noted throughout most of the epithelial thickness, although slight maturation and flattening of the cells appears to be present at the epithelial surface.

SEVERE
DYSPLASIA

TOLUIDINE BLUE TEST

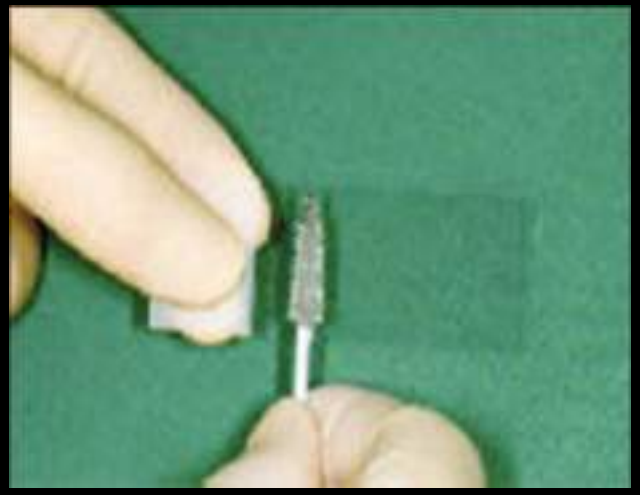


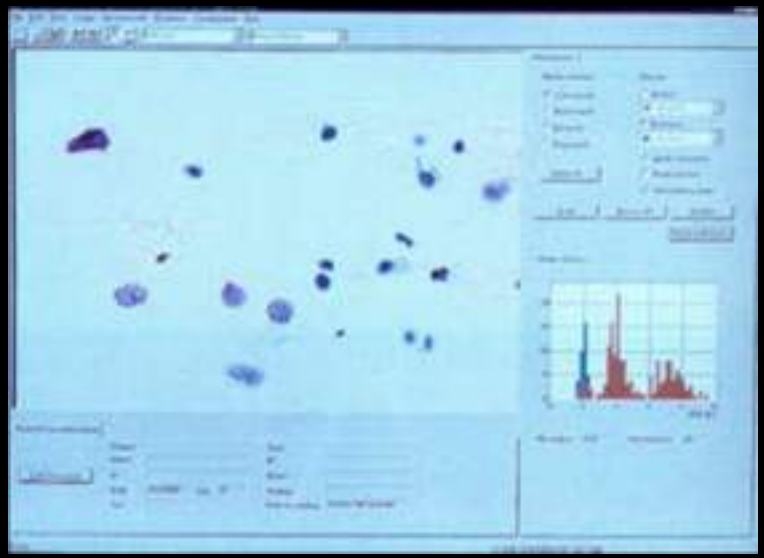
BEFORE
STAINING



AFTER
STAINING

BRUSH BIOPSY





TREATMENT

- Definitive treatment involves surgical excision although **cryosurgery and laser ablation** are often preferred because of their precision and rapid healing.
- **Total excision** is aggressively recommended when microscopic dysplasia is identified, particularly if the dysplasia is classified as **severe or moderate.**
- Most leukoplakias incur a low risk for malignant transformation.
- Following attempts at removal, **recurrences** appear when either the margins of excision are inadequate or the causative factor or habit is continued.
- Programs have included single and combination dosages of vitamins A, C, and E; beta carotene; analogues of vitamin A; and diets that are high in antioxidants and cell growth suppressor proteins (fruits and vegetables).

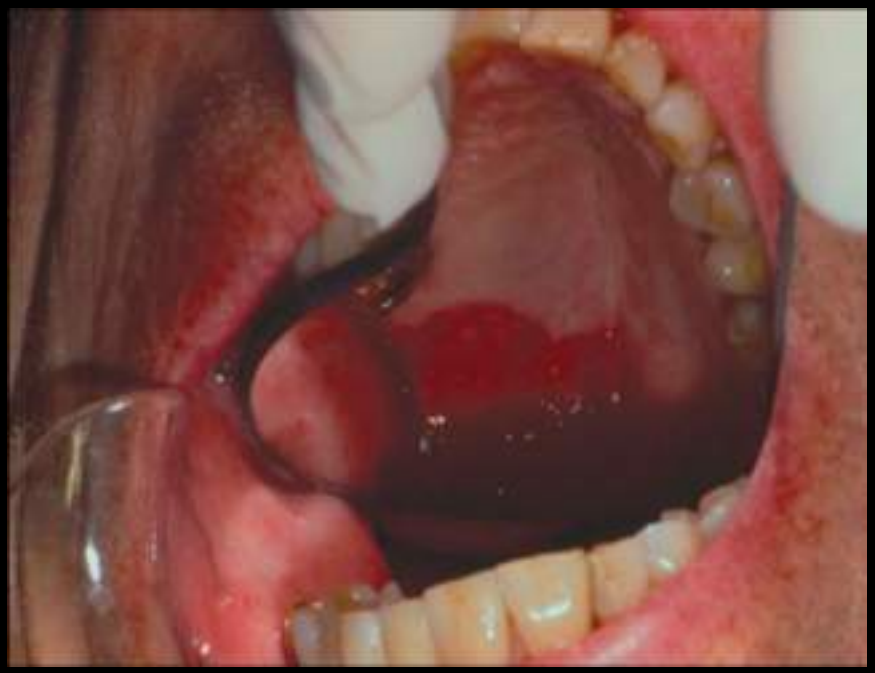
ERYTHROPLAKIA

- “Bright red velvety plaque or patch which cannot be characterized clinically or pathologically as being due to any other condition.

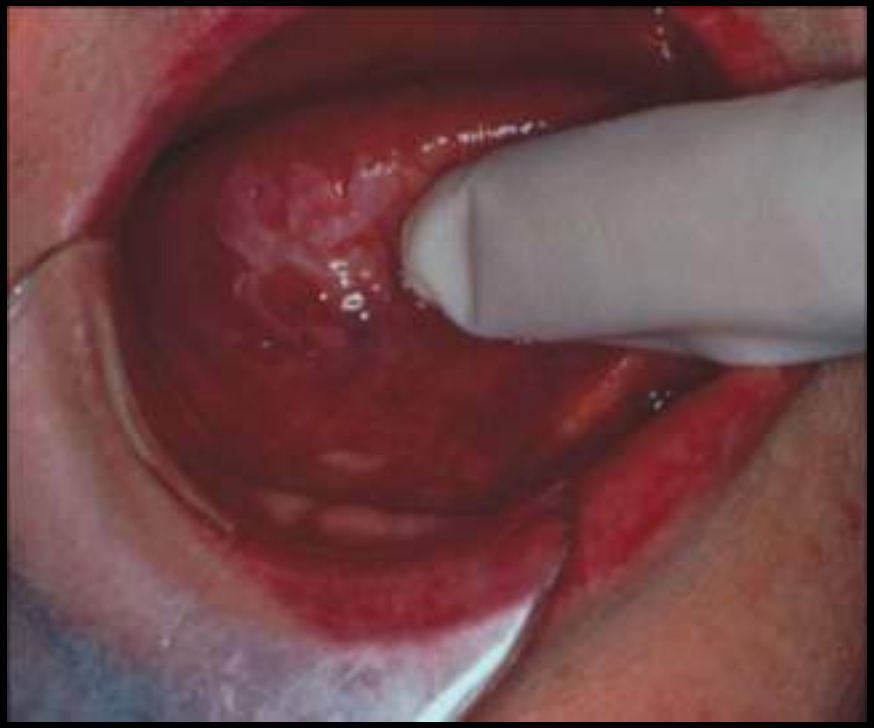
- Although the etiology of erythroplakia is uncertain, most cases of erythroplakia are associated with heavy smoking,with or without concomitant alcohol abuse.

- Erythroplakia occurs predominantly in older men, in the sixth and seventh decades of life.
- Erythroplakias are more commonly seen on the floor of the mouth, the ventral tongue, the soft palate, and the tonsillar fauces, all prime areas for the development of carcinoma.
- Multiple lesions may be present. These lesions are commonly described as erythematous plaques with a soft velvety texture.

Homogeneous erythroplakia consisting of a bright red well-demarcated velvety patch seen here in the posterior hard palate/soft palate area



Homogeneous erythroplakia as a mixed area of leukoplakia and erythroplakia,, seen in the floor of the mouth and on the lateral border of the tongue.



D/D

- Erythematous candidiasis,
- Areas of mechanical irritation,
- Denture stomatitis,
- Vascular lesions
- Nonspecific inflammatory lesions

ORAL SUB MUCOUS FIBROSIS

- Oral submucous fibrosis is an insidious, chronic disease affecting any part of the oral cavity and sometimes pharynx, although occasionally preceded by and/or associated with vesicle formation.

- It is always associated with juxtaepithelial inflammatory reaction followed by fibroelastic changes of lamina propria, with epithelial atrophy leading to stiffness of oral mucosa and causing trismus and inability to eat.

Also known as…

- Atrophica idiopathic mucosae oris... Schwartz (1952)
- Idiopathic scleroderma of mouth- 1954 Su I.P.
- Idiopathic palatal fibrosis - 1962 A.B.N.RAO
- Sclerosing stomatitis-1962 P.N. BEHL
- Juxtra epithelial fibrosis -1966 J.J.PINDBORG

Clinical Features

Age and sex distribution

1. It affects both sexes.
2. The age group varies, although majority of patients are between 20 and 40 years of age.

Site distribution

1. The most frequent location of oral sub mucus fibrosis is the buccal mucosa and the retro molar areas.
2. It also commonly involves soft palate, palatal fauces, uvula, tongue, and labial mucosa. Some times, it involves the floor of mouth and gingiva.

Prodromal symptoms

1. The onset of the condition is insidious and is often of 2 to 5 yrs of duration.

2. The most common initial symptom is burning sensation of oral mucosa, aggravated by spicy food, followed by either hypersalivation or dryness of mouth.

3. Vesiculation, ulceration, pigmentation and recurrent stomatitis have also been indicated as early symptoms.

Late symptoms

1. Gradual stiffening of the oral mucosa occurs in few years after the initial symptoms appear. This leads to inability to open the mouth. Later on patients experience difficulty in protruding the tongue.

2. When the fibrosis extends to pharynx and esophagus, the patient may experience difficulty in swallowing the food.

3. Referred pain in the ears and deafness, due to occlusion of Eustachian tube and a typical nasal voice has been reported.

Signs

1. The most common and earliest sign is blanching of mucosa, caused by impairment of local vascularity. The blanched mucosa becomes slightly opaque and white.

2. The whitening often takes place in spots so that the mucosa acquires a marble like appearance.

3. Blanching may be localized or diffuse, involving greater part of the oral mucosa.

4. As disease progresses the mucosa becomes stiff and vertical fibrous bands appear there.

Staging

Stage 1: Early OSMF

1.Mild blanching

2.No restriction in mouth opening

3.Normal- Distance between Central incisor tips

- Males 35-45mm
- Females 30-42mm

4.No restriction in tongue protrusion

5. Cheek flexibility ...

CF=V1-V2

Two points measured between;

- V 2 = is marked at one third the distance from the angle of the mouth on a line joining the tragus of the ear and the angle of the mouth,
- VI = the subject is then asked to blow his cheeks fully and the distance measured between the two points marked on the cheek
- Mean value for males ... 1.2 cms, Females ... 1.08 cms

6. Burning sensation only on taking spicy food, or hot temperature liquids, etc.

Stage 2: Moderate OSMF

1.Moderate to severe blanching.
2.Mouth opening reduced by 33%.
3.Cheek flexibility also demonstrably decreased.
4.Burning sensation even in absence of stimuli.
5.Palpable bands felt.
6.Lymphadenopathy either unilateral or bilateral.
7.Demonstrable Anemia on hematological examination

Stage 3: Severe OSMF

1. Burning sensation is very severe. Patient unable to do day to day work
2. More than 66% reduction in the mouth opening, cheek flexibility and tongue protrusion
3. The tongue may appear fixed.
4. Ulcerative lesions may appear on the cheek
5. Thick palpable bands.
6. Lymphadenopathy bilaterally evident.

- (According to Durgesh N Bailoor & KS Nagesh)

Differential diagnosis

1. Radiation - related sub epithelial fibrosis and
2. Mucosal scarring secondary to thermal and chemical burns.
3. Scleroderma

TREATMENT

- Cessation of the habit
- Eating bland food without chillies and pepper.
- Nutritional support of high calcium and high protein and iron supplementation.

TREATMENT

1. Intralesional injections of Hyaluronidase and corticosteroids or Placentrax (Contains nucleotides, enzymes, vitamin- A,B,E, Steroids, Fatty acid- Linoleic acid and trace elements- copper, selenium).

2. In severe cases, surgical intervention is the only treatment, but the fibrous bands and other symptoms often recur within a few months to a few years.

3. The use of an oral stent as an adjunct to surgery to prevent relapse has also been studied.

Other modalities

- CO_2 laser surgery
- Intralesional inj. of interferon gamma
- Microwave diathermy- it acts by physio fibrolysis of bands. Useful in early or moderately advanced stages.

ORAL LICHEN PLANUS

- Oral lichen planus (OLP) is a common chronic immunologic inflammatory mucocutaneous disorder that varies in appearance from keratotic (reticular or plaque like) to erythematous and ulcerative.

- About 28% of patients who have OLP also have skin lesions.

- The skin lesions are flat violaceous papules with a fine scaling on the surface.

- Unlike oral lesions, skin lesions are usually self-limiting, lasting only 1 year or less.

- The etiology of lichen planus involves a cell-mediated immunologically induced degeneration of the basal cell layer of the epithelium.

Clinical Features

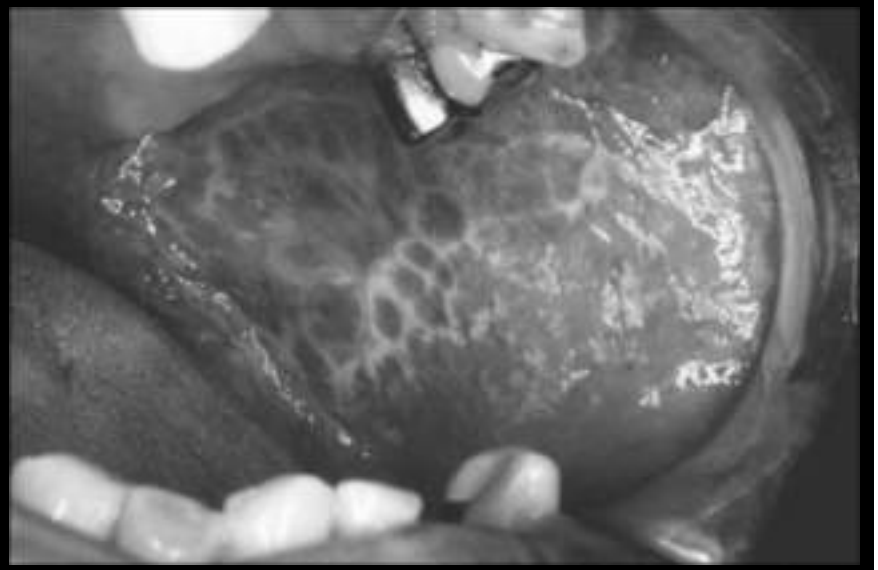
1. The mean age of onset is the fifth decade of life.
2. Female predominance.
3. Buccal mucosa is the most common site.
4. OLP may be associated with pain or discomfort, which interferes with function and with quality of life.
5. Approximately 1% of the population may have cutaneous lichen planus. The prevalence rate of OLP ranges between 0.1 and 2.2%.
6. The skin lesions of lichen planus have been classically described as purple, pruritic, and polygonal papules.

Classification

1. **Reticular** (lacelike keratotic mucosal configurations)
2. **Atrophic** (keratotic changes combined with mucosal erythema)
3. **Erosive** (pseudomembrane-covered ulcerations combined with keratosis and erythema)
4. **Bullous** (vesiculobullous presentation combined with reticular or erosive patterns).

Reticular lichen planus

- The reticular form consists of (a) slightly elevated fine whitish lines (Wickham's striae) that produce either a lacelike pattern or a patern of fine radiating lines or (b) annular lesions.



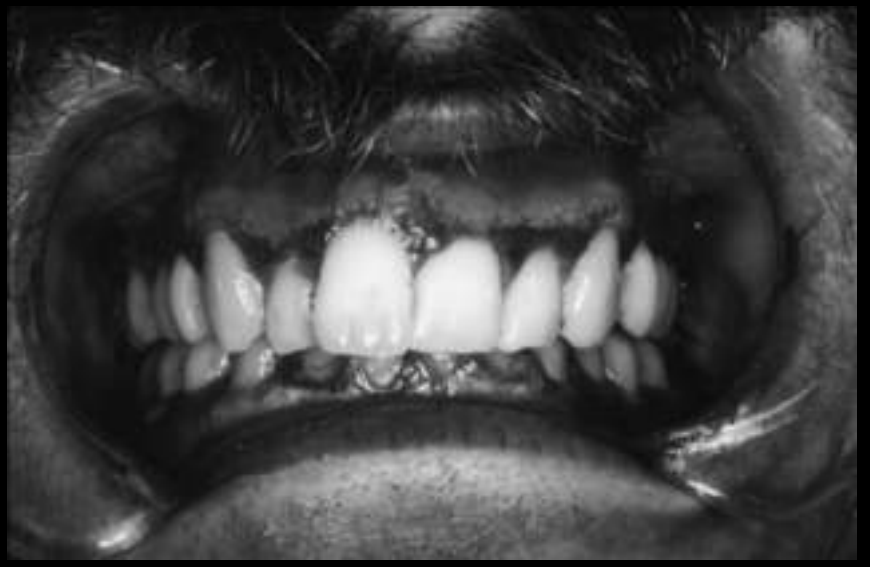
- This is the most common and most readily recognized form of lichen planus.

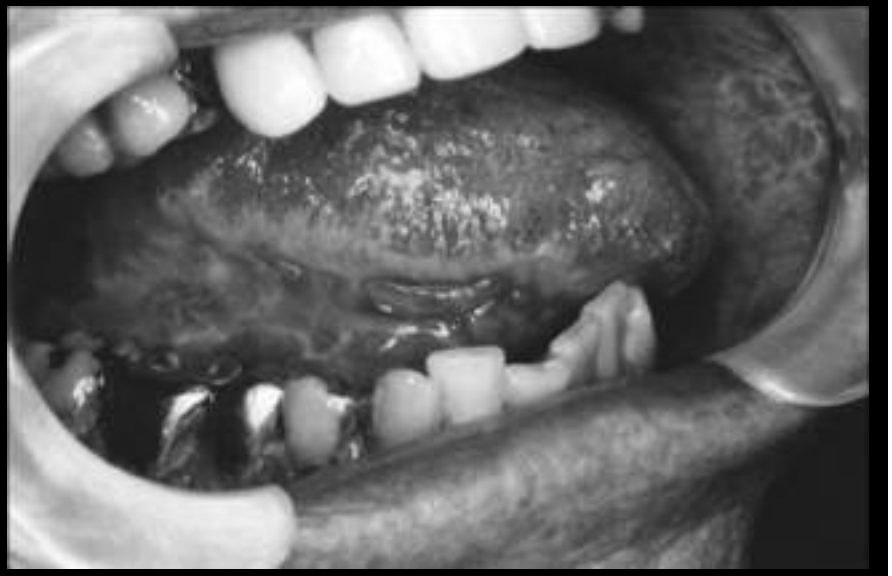
- Most patients with lichen planus at some time exhibit some reticular areas.
- The most common sites include the buccal mucosa (often bilaterally), followed by the tongue; lips, gingiva, the floor of the mouth, and the palate are less frequently involved.
- Whitish elevated lesions, or papules, usually measuring 0.5 to 1.0 mm in diameter, may be seen on the well-keratinized areas of the oral mucosa.
- However, even large plaquelike lesions may occur on the cheek, tongue, and gingivae, and these are difficult to distinguish from leukoplakia.

- **Atrophic lichen planus** presents as inflamed areas of the oral mucosa covered by thinned red-appearing epithelium.

- **Erosive lesions** probably develop as a complication of the atrophic process when the thin epithelium is abraded or ulcerated.

- These lesions are invariably symptomatic, with symptoms that range from mild burning to severe pain.





- Bullous lichen planus is rare and may sometimes resemble a form of linear IgA disease.

Differential Diagnosis

1. Other lichenoid lesions (e.g, drug-induced lesions, contact mercury hypersensitivity, erythema multiforme, lupus erythematosus and graft-versus-host reaction).

2. Leukoplakia

3. Mucous membrane pemphigoid

Treatment

1. There is no known cure for OLP; therefore, the management of symptoms guides therapeutic approaches.

2. Corticosteroids have been the most predictable and successful medications for controlling signs and symptoms.

Topical medications

1. Include high-potency corticosteroids, the most commonly used of which are 0.05% fluocinonide (Lidex), triamcinolone acetonide and 0.05% clobetasol (Temovate).

2. *Candida* overgrowth with clinical thrush may develop, requiring concomitant topical or systemic antifungal therapy.

3. Retinoids are also useful, usually in conjunction with topical corticosteroids as adjunctive therapy for OLP.

4. Systemic and topically administered beta all-*trans retinoic* acid, vitamin A acid, systemic etretinate, and systemic and topical iso-tretinoin are all effective, and topical application of a retinoid cream or gel will eliminate reticular and plaque like lesions in many patients.

5. However, following withdrawal of the medication, the majority of lesions recur.

Systemic administration

1. Systemic administration of prednisone tablets may be done with dosages varying between 40 and 80 mg daily for less than 10 days without tapering.

2. The time and dosage regimens are determined individually, based on the patient's medical status, severity of disease, and previous treatment responses.

- Other topical and systemic therapies reported to be useful, such as dapsone, doxycycline, and antimalarials.

- Lichenoid reactions and lichen planus exhibit similar histopathologic features.

- Lichenoid reactions were differentiated from lichen planus on the basis of

  (1) Their association with the administration of a drug, contact with a metal, the use of a food flavoring, or systemic disease.

  (2) Their resolution when the drug or other factor was eliminated or when the disease was treated.

- Clinically, lichenoid lesions may exhibit the classic appearance of lichen planus, but atypical presentations are seen.

What is new?

- Lacy pattern of white lesion usually seen in oral lichen planus can occur in frictional keratosis also.
- Usage of non-smoked form of tobacco is also a significant risk factor for dysplasia.
- It is advisable to do early biopsy atleast in those oral white lesions with atypical clinical features to decrease the incidence and improve the survival of oral cancer.