RED & WHITE LESIONS OF ORAL MUCOSA Part II

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RED & WHITE LESIONS OF ORAL MUCOSA

1. INTRODUCTION
2. CLASSIFICATION
3. DESCRIPTION OF INDIVIDUAL LESIONS
4. DIFFERENTIAL DIAGNOSIS

Classification

1. Variations from normal
2. Non-Keratotic white lesions
3. Oral candidiasis
4. Keratotic white lesions with no increased potential for oral cancer
5. Red and white lesions with unknown or increased malignant potential

Red and white lesions with unknown or increased malignant potential

~ Leukoplakia
~ Erythroplakia
~ Oral submucous fibrosis
~ Lichen planus
~ Lichenoid reactions
~ Actinic keratosis, elastosis, Cheilitis
~ Discoid lupus erythematosus
~ Dyskeratosis congenita
~ Carcinoma in situ
~ Bowen's disease
~ Erythema multiforme, lupus erythematosus

Leukoplakia

Epidemiology

1. More prevalent in Southeast Asia.
2. Prevalence in India=0.2% to 4.9%.
3. Tobacco abuse is the most common etiological factor.
4. Smoking causes more number of leukoplakias than tobacco chewing. But, malignant transformation is more frequent in leukoplakias caused by chewing.
5. 1.3% of lesions in India are idiopathic.
6. Malignant transformation rate=3% to 6%
7. Age: majority of cases in the range of 35-45 years.
8. Sex: males are much more frequently affected than females.
9. Site: buccal/vestibular mucosa most frequently affected (habit related). Floor of mouth least affected.

Lesson objectives

1. You should know the etiology and pathogenesis.
2. You should be able to diagnose different clinical forms of leukoplakia, based upon history and clinical features.
3. You should be able to decide when histological investigations are indicated.
4. You should be able to decide the treatment plan and execute it.
5. You should be able to counsel and educate patients on tobacco abuse.

Leukoplakia

Definition

1. Leukoplakia literally means *white patch*
2. WHO definition (1978):

"a white patch or plaque that cannot be characterized clinically or pathologically *as* any other disease"

Leukoplakia

Definition

1. Leukoplakia literally means *white patch*
2. WHO definition (1978):
3. Modified definition (1984):

"a white patch or plaque that cannot be characterized clinically or pathologically *as* any other disease, *and* which is not associated with any physical or chemical agent *except* the use of tobacco"

Leukoplakia

Etiology and pathogenesis

1. Tobacco abuse- smoking, quid
2. Predisposing factors

Local
~ poor oral hygiene
~ chronic trauma
~ alcohol
~ candidiasis, syphilis, EB virus infection
~ electro-galvanism

Leukoplakia

Etiology and pathogenesis

1. Tobacco abuse- smoking, quid
2. Predisposing factors

Systemic
~ genetic
~ nutritional deficiency

Assignment

Elaborate the role of following in etiology and pathogenesis of leukoplakia:

1. Tobacco
2. Local predisposing factors
3. Systemic predisposing factors

Leukoplakia

Clinical features

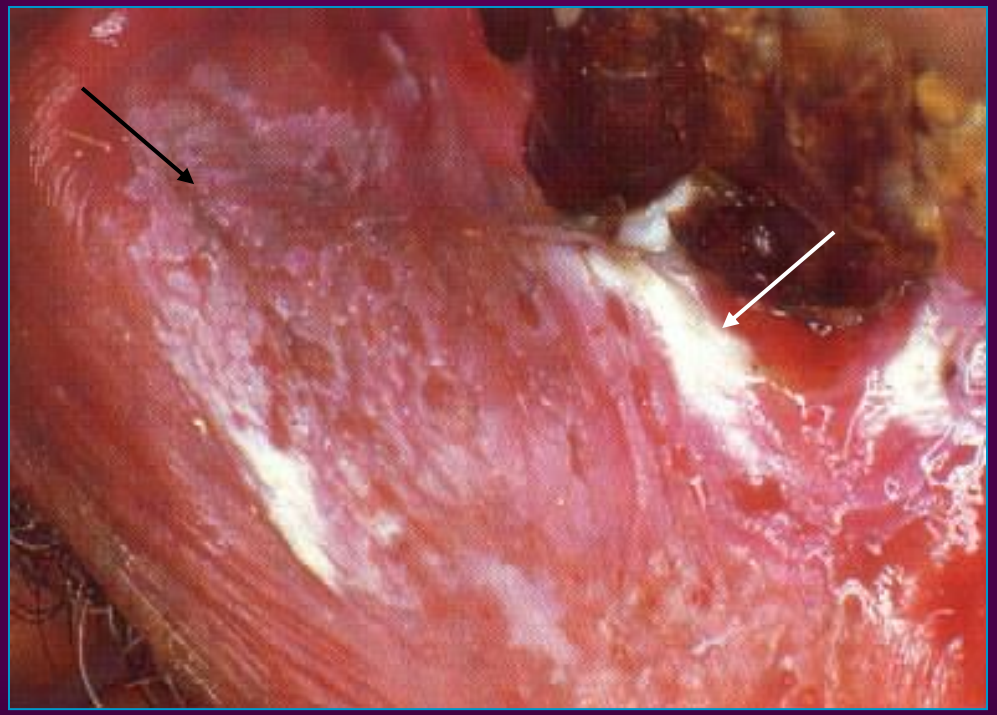
1. Age: majority of cases in the range of 35-45 years.
2. Sex: males are much more frequently affected than females.
3. Site: buccal/vestibular mucosa most frequently affected (habit related). Floor of mouth least affected.
4. Symptoms: mostly asymptomatic, discovered on routine examination, sometimes patients may be aware of a white lesion/roughness/thickening, speckled variety may cause burning sensation

Leukoplakia

Clinical features

5. Signs: 4 clinical forms-
   - Pre-leukoplakia
   - Homogenous: most common form
   - Nodular or speckled
   - Verrucous: least common

Clinical features



Pre-leukoplakia

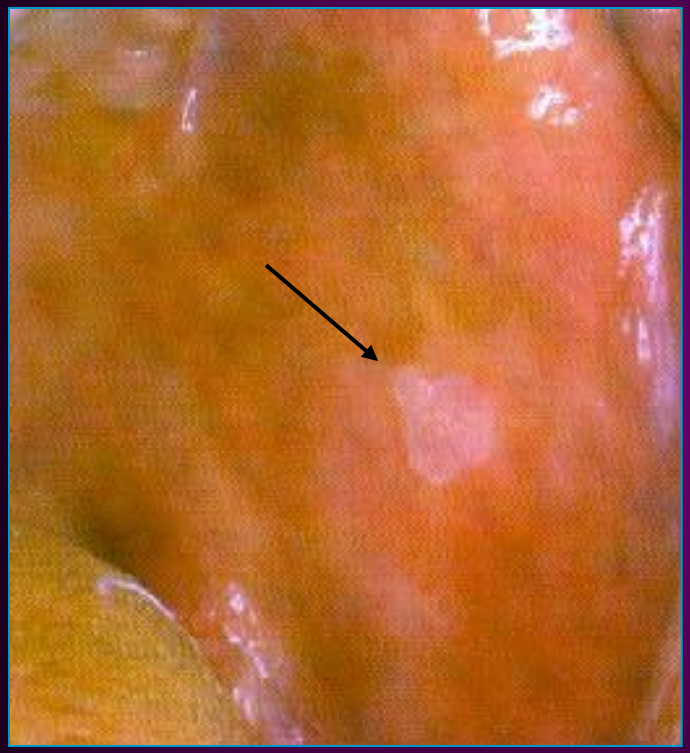
Clinical features

- Precursor of leukoplakia
- Prevalence=0.5% to 4.1%
- Low malignant potential
- Appears as a gray or grayish-white, flat lesion with indistinct borders, lending into the adjacent mucosa
- Partially scrapable

Pre-leukoplakia

Leukoplakia

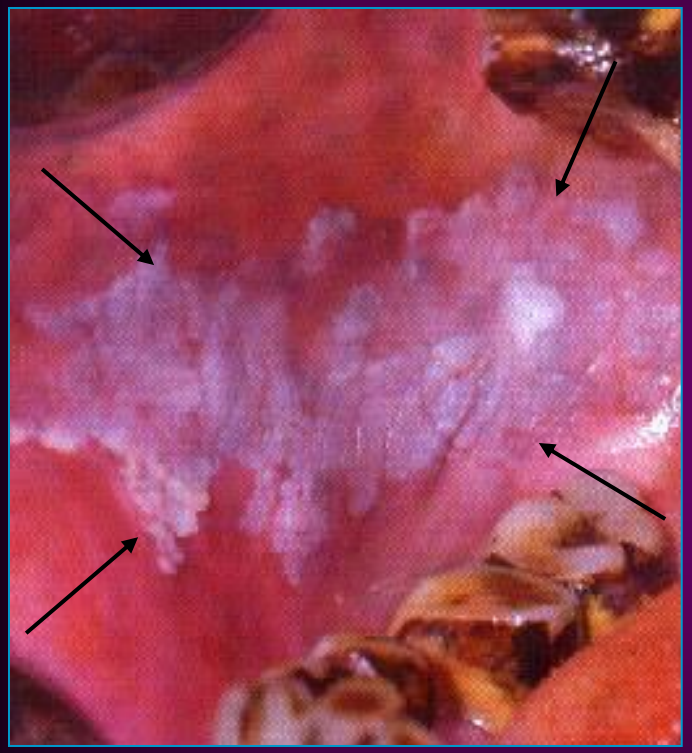
Clinical features



Homogenous

Leukoplakia

Clinical features

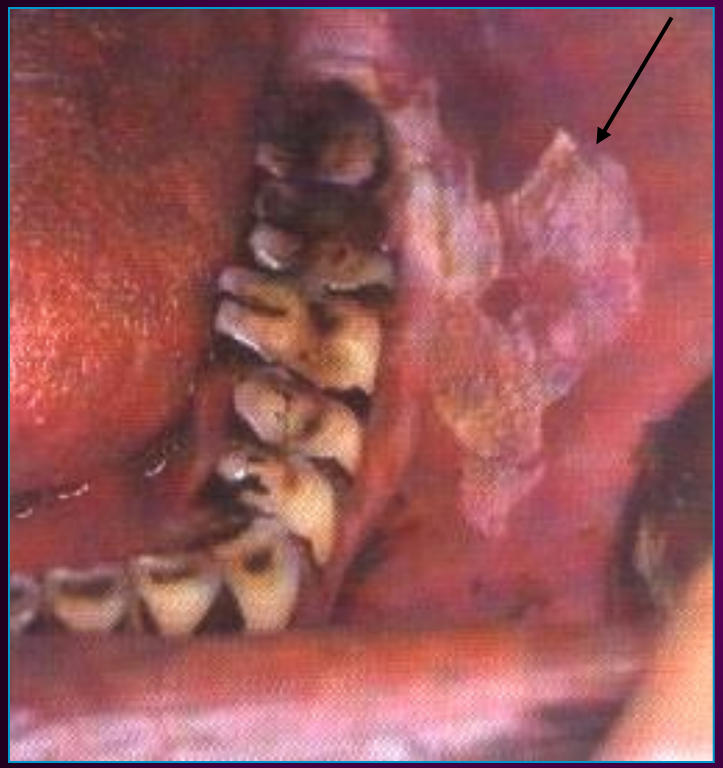


‘cracked-mud’ appearance

Homogenous

Leukoplakia

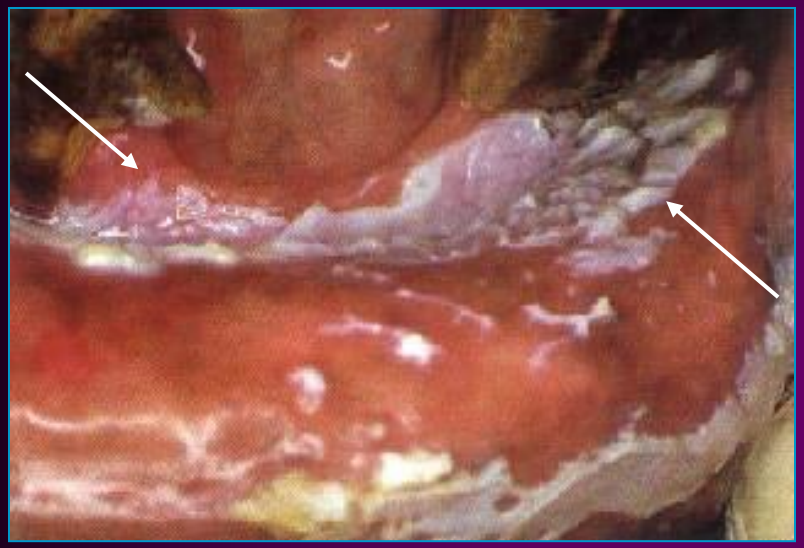
Clinical features

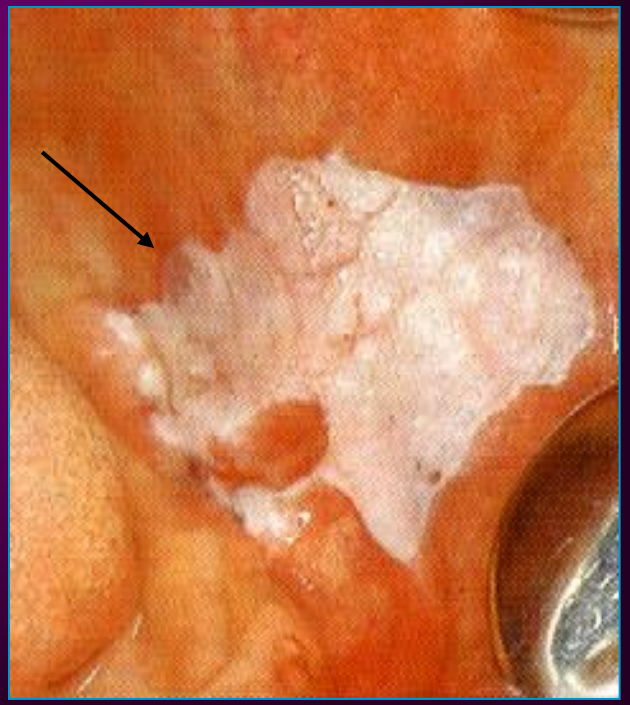


Homogenous

Leukoplakia

Clinical features

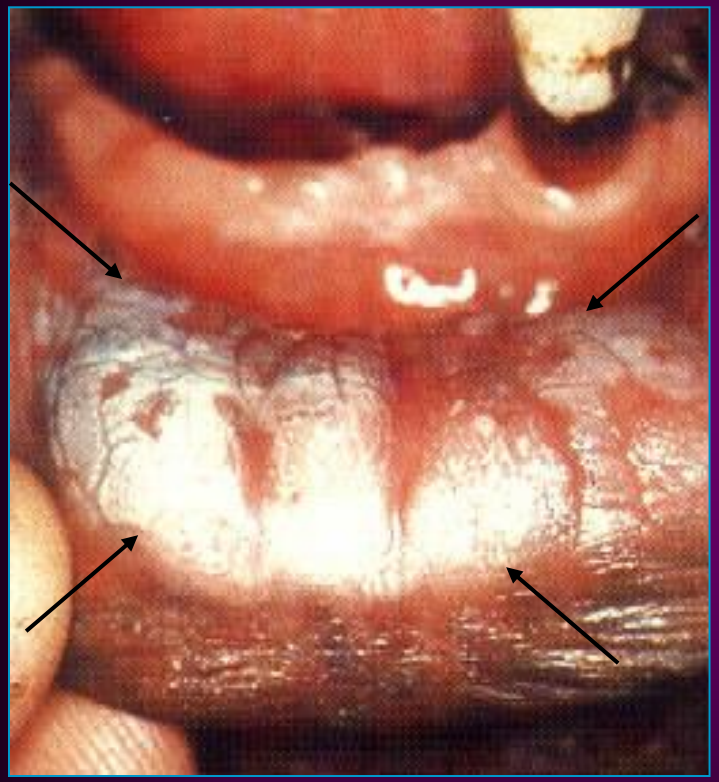




Homogenous

Leukoplakia

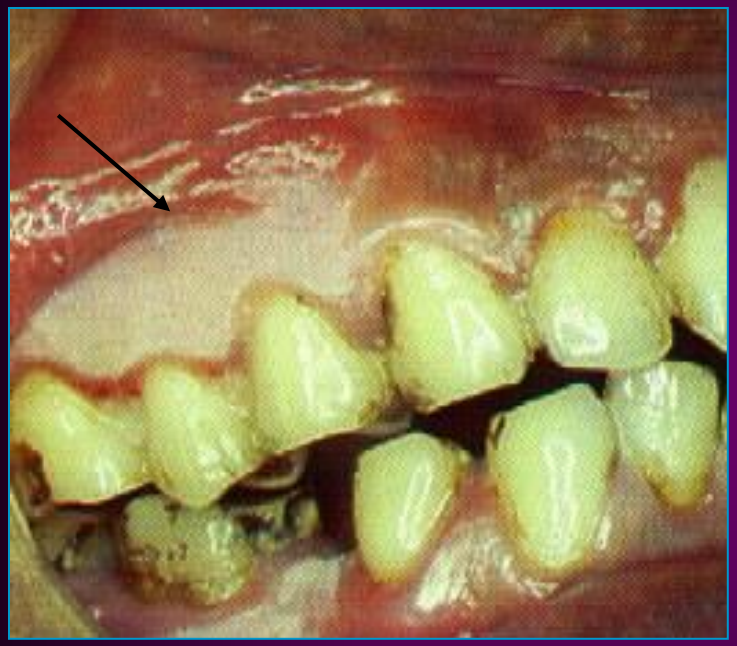
Clinical features

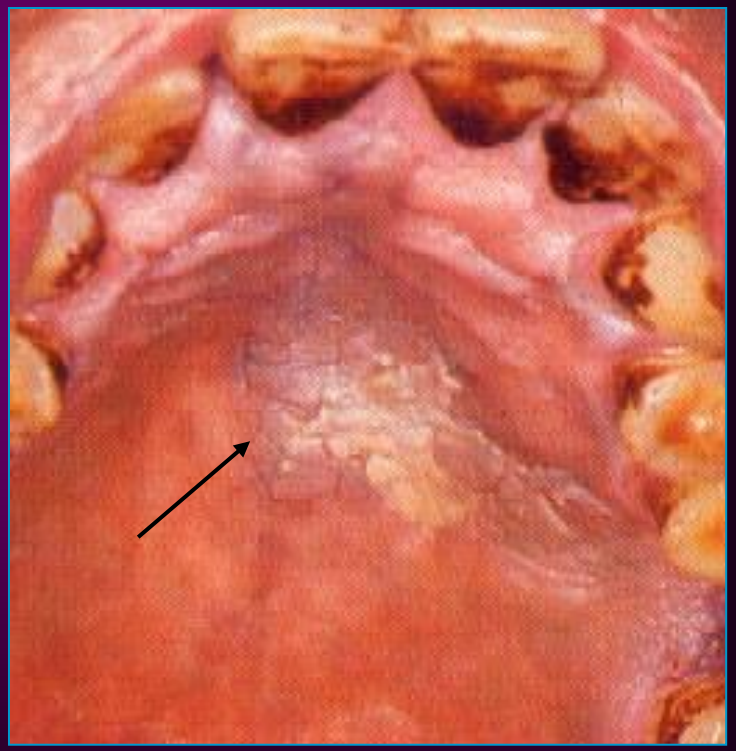


Homogenous

Leukoplakia

Clinical features

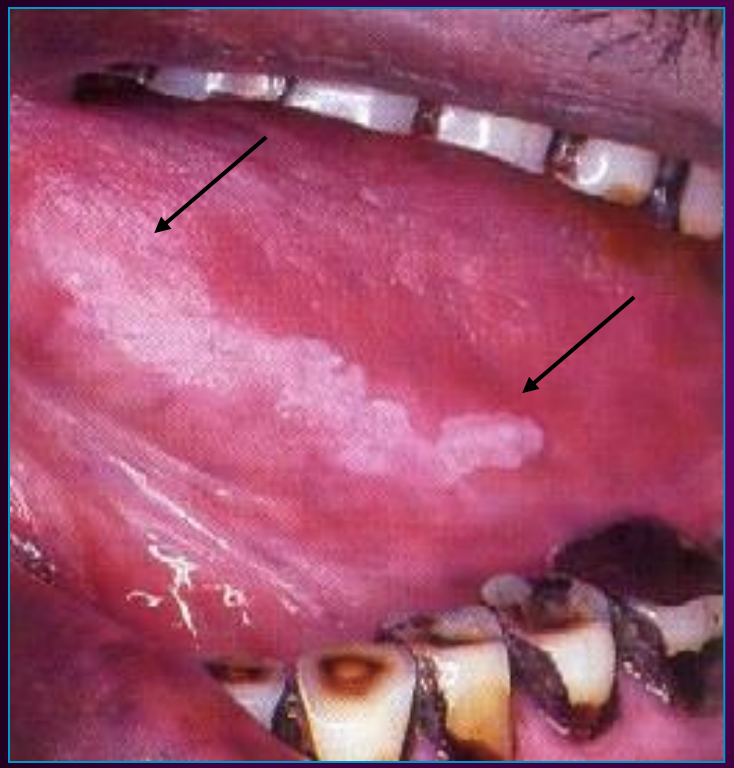




Homogenous

Leukoplakia

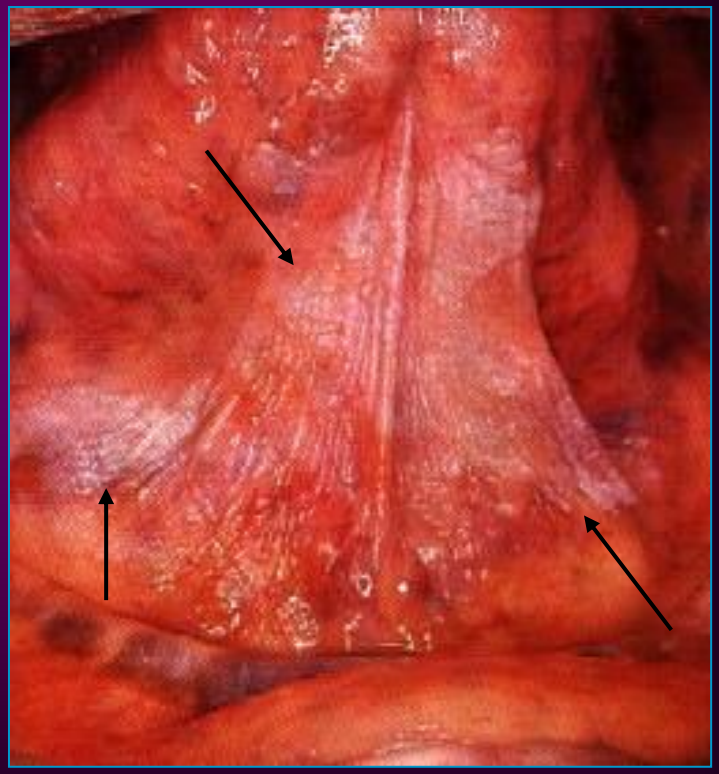
Clinical features



Homogenous

Leukoplakia

Clinical features



Homogenous

Leukoplakia

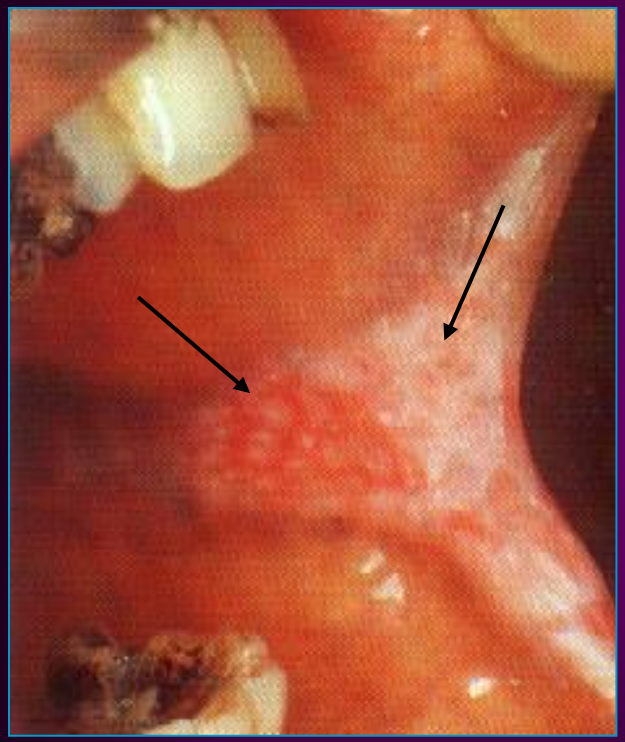
Clinical features

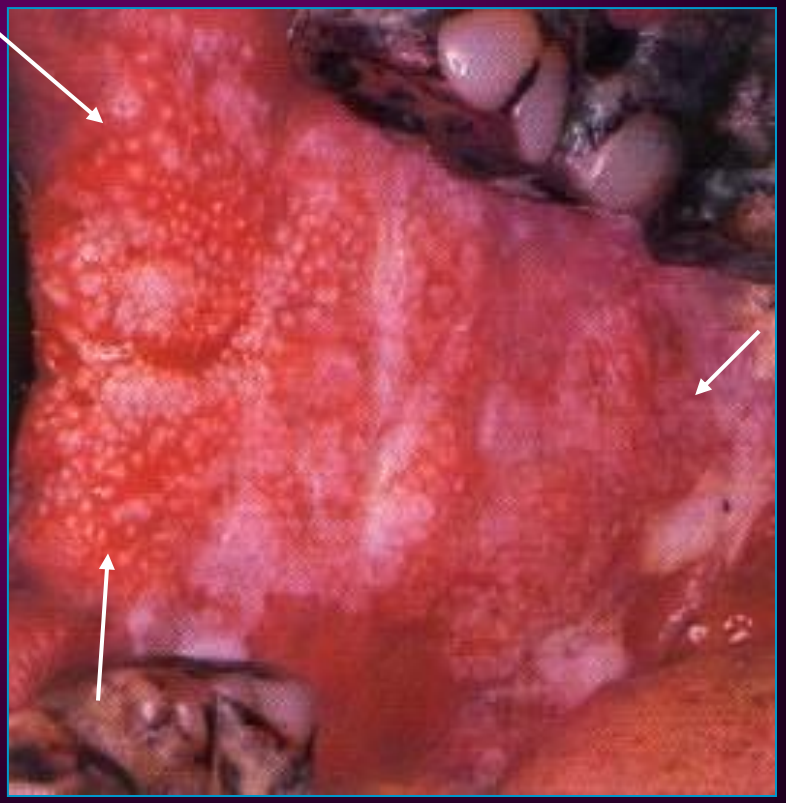
- At a site that comes in contact with tobacco
- White, brownish-white plaque with more or less uniform appearance
- 'cracked-mud', 'corrugated' appearance
- Size: from about 10 mm to extensive lesions
- Distinct borders
- Non-scrapable
- Loss of elasticity/pliability of affected mucosa
- Partially scrapable
- Loss of papillae, if on tongue dorsum

Homogenous

Leukoplakia

Clinical features

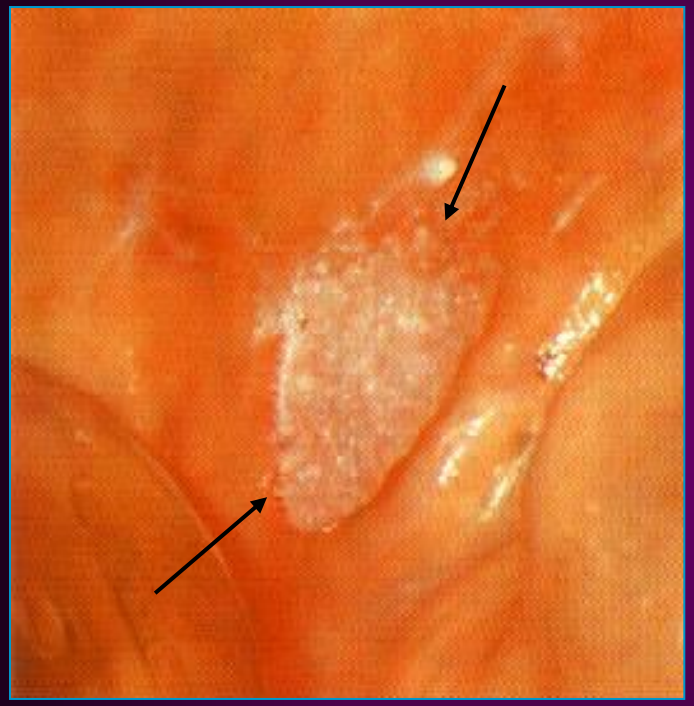


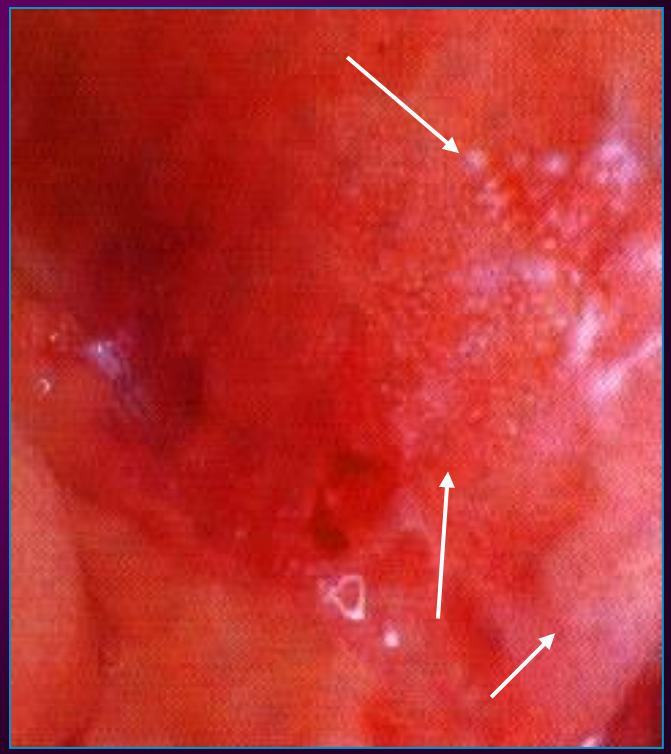


Nodular ('speckled')

Leukoplakia

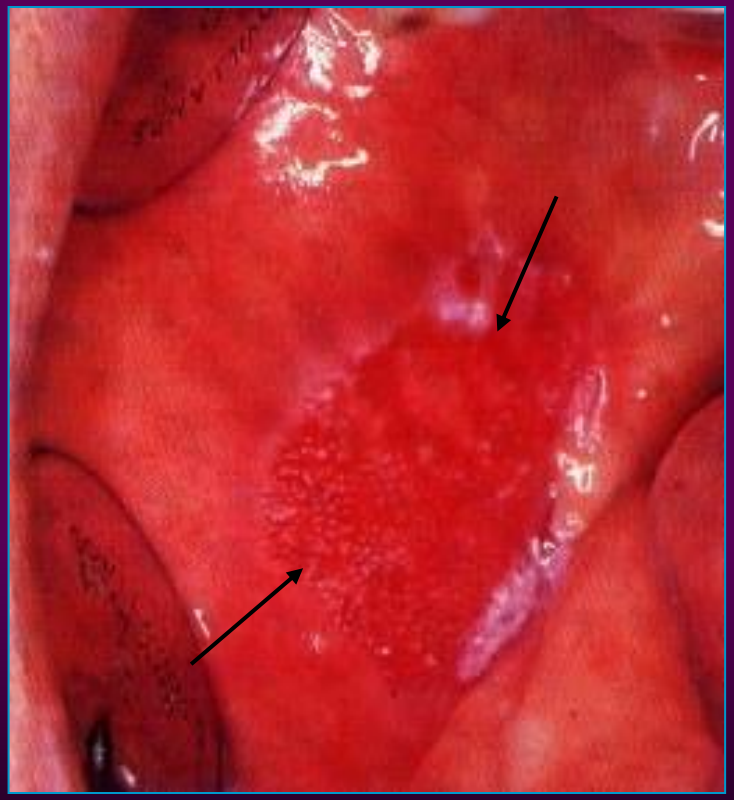
Clinical features





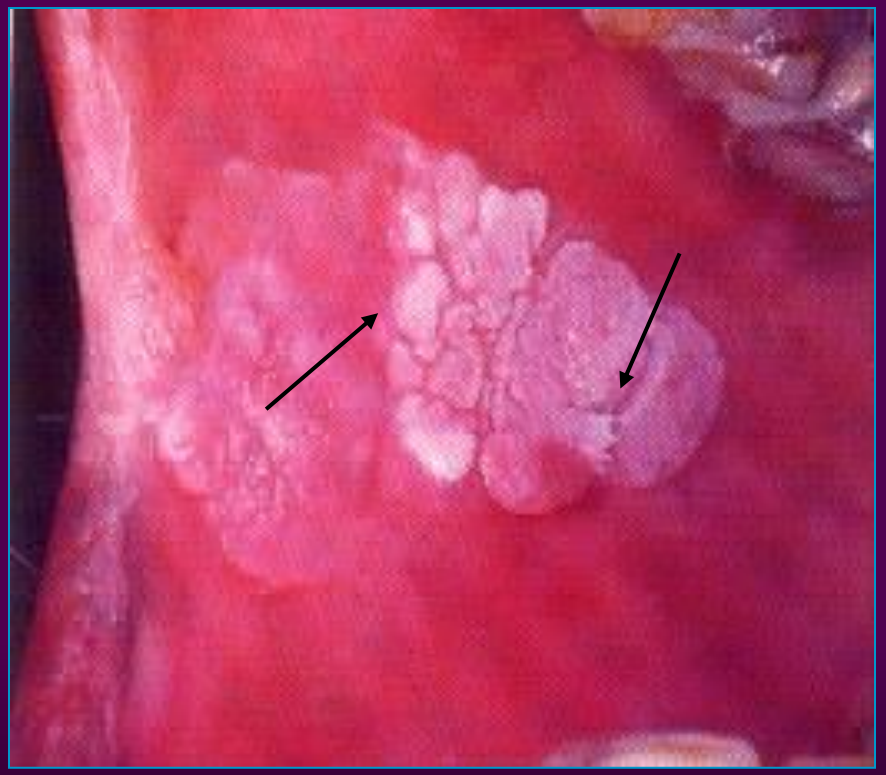
Nodular ('speckled')

Clinical features



Nodular ('speckled erythroplakia')

Clinical features



Verrucous

Leukoplakia

Clinical features

- At a site that comes in contact with tobacco
- Appears as a mixed red and white lesion, i.e. small multiple keratotic (white) nodules scattered over an atrophic (red) patch of mucosa
- Size: from about 10 mm to extensive lesions
- Relatively less distinct borders
- Non-scrapable
- Higher rate of malignant transformation

Nodular ('speckled')

Leukoplakia

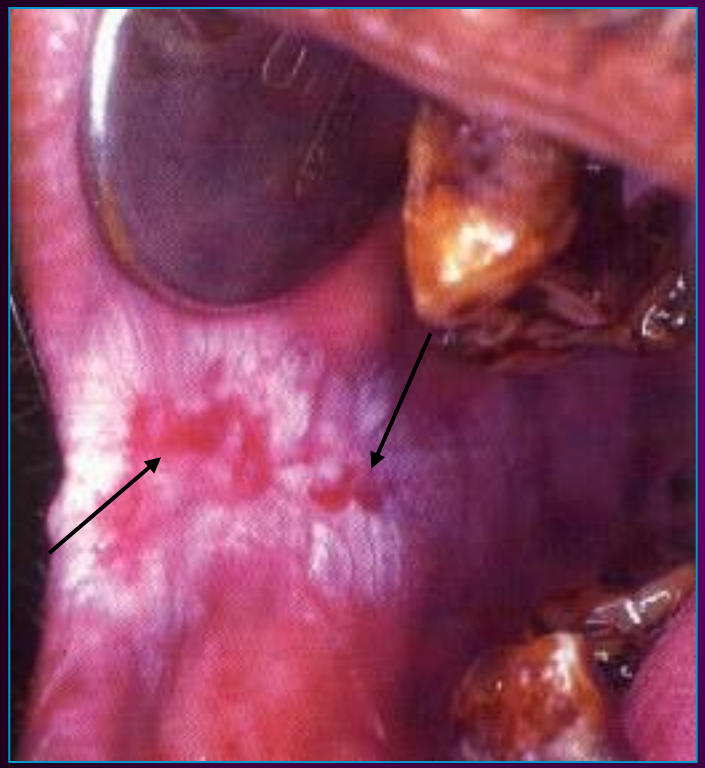
Clinical features

- At a site that comes in contact with tobacco
- Appears as a white plaque with multiple finger-like projections from surface
- Size: usually only some areas of a homogenous leukoplakia show verrucous pattern
- Distinct borders
- Non-scrapable

Verrucous

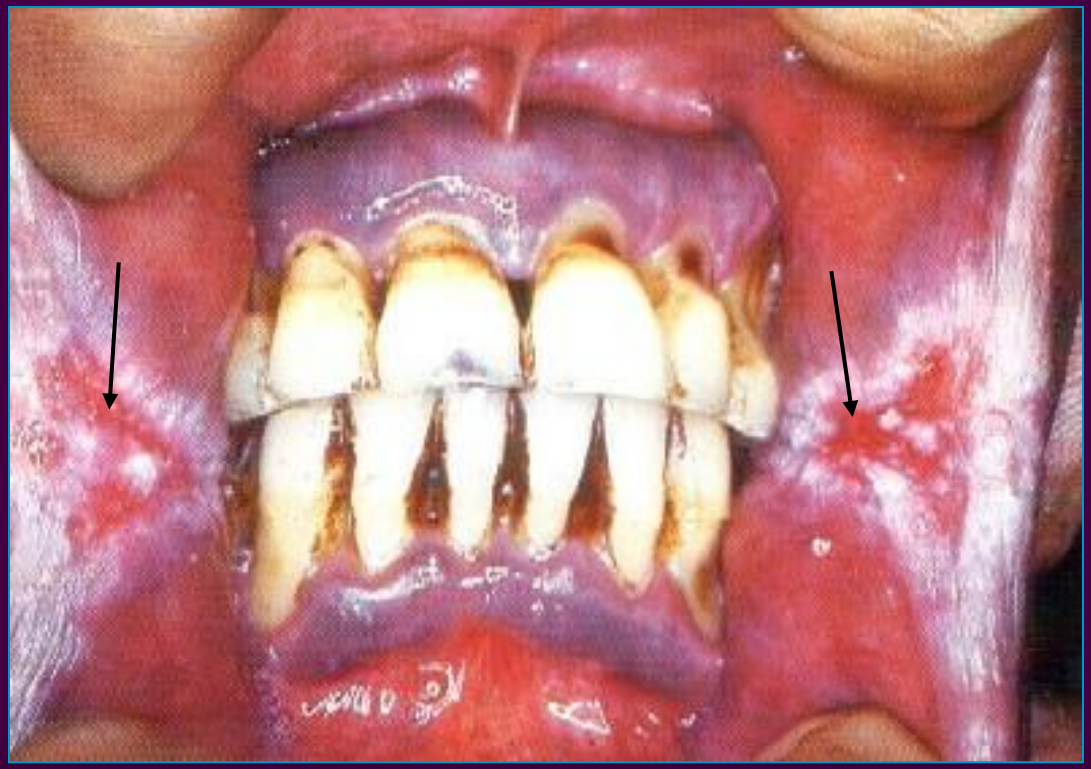
Leukoplakia

Clinical features



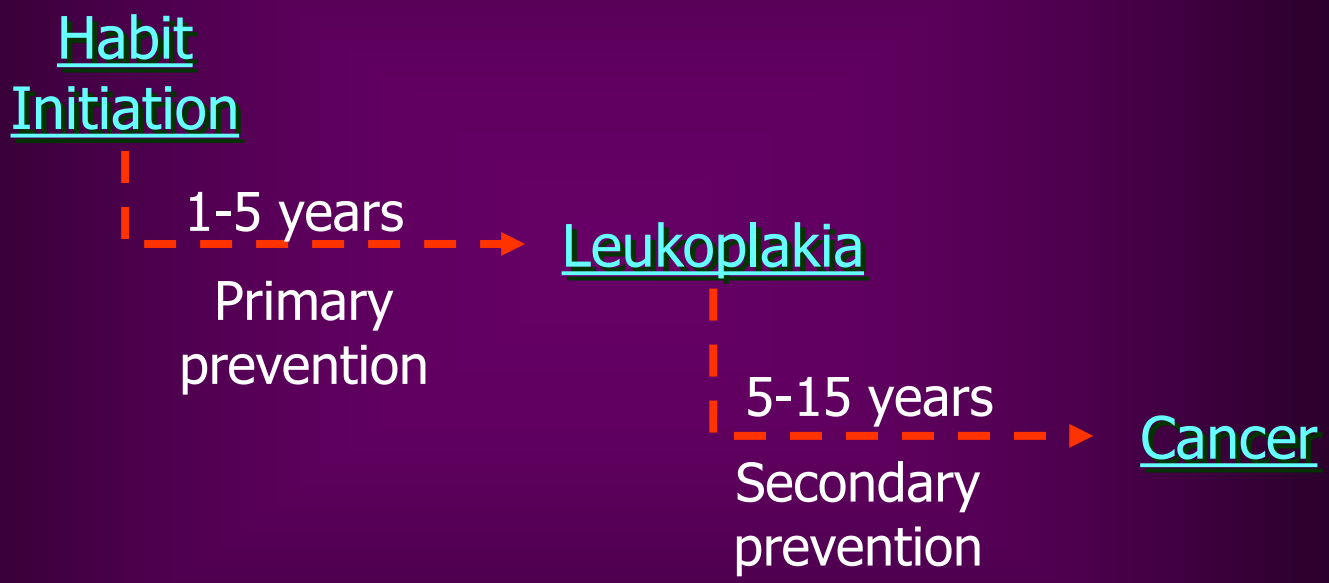
Ulcerated lesion

Clinical features



Ulcerated lesion

Natural history: evolution



Leukoplakia

Natural history: long term behavior

- May persist without any change.
- May spontaneously regress, especially when habit is discontinued.
- May recur.
- May progress to cancer: 'malignant transformation' denotes development of oral cancer in pre-existing leukoplakia. Overall rate of malignant transformation is 3% to 6%. The rate is 20% in nodular/speckled lesions.
- Nodular area, ulceration, rolled margins, induration, growth are the signs of malignant transformation.
- Therefore, long term follow-up is essential.

Leukoplakia

Investigations:

- Exfoliative cytology
- Brush / incisional / excisional biopsy
- Toluidine blue vital staining to select biopsy site

Procedure:

- 20-seconds rinse with 1% acetic acid
- 20-seconds rinse with water twice
- 10-seconds rinse with 5-10 ml of 1% toluidine blue solution
- 1-minute rinse with 1% acetic acid
- Water rinse

Leukoplakia

Histological features

- Clinical appearance is not always an indicator of histological features.
- Homogenous lesions usually show only keratosis. Mild dysplasia may be seen.
- Nodular/speckled lesions usually show varying degrees of dysplasia. Frank malignancy may also be evident.
- Histological features influence treatment decisions.

Differential diagnosis

Leukoplakia

Treatment: *Homogenous leukoplakia*

1. Patient education and motivation to discontinue tobacco habit.
2. Identification and removal of any predisposing factors.
3. Anti-fungal agents for 2 weeks may reduce lesion size.
4. Periodic observation.
5. Increase in lesion size or change in appearance indicates need for histological examination.
6. Dysplastic changes indicate surgical removal and long term follow-up.

Leukoplakia

Treatment: *Nodular and verrucous leukoplakias*

1. Patient education and motivation to discontinue tobacco habit.
2. Identification and removal of any predisposing factors.
3. Histological examination.
4. Dysplastic changes indicate surgical removal and long term follow-up.
5. Non-dysplastic lesions may be treated similar to homogenous variety.

Leukoplakia

Treatment:

Vitamin A and its analogues have shown variable results in obtaining clinical remission of leukoplakia. However, they do not prevent dysplasia or malignant transformation.

Erythroplakia

Definition

1. Erythroplakia literally means *red patch*
2. Definition:

"a velvety, red patch that cannot be characterized clinically or pathologically *as* any other disease"

Erythroplakia

Etiology and pathogenesis

1. Tobacco abuse

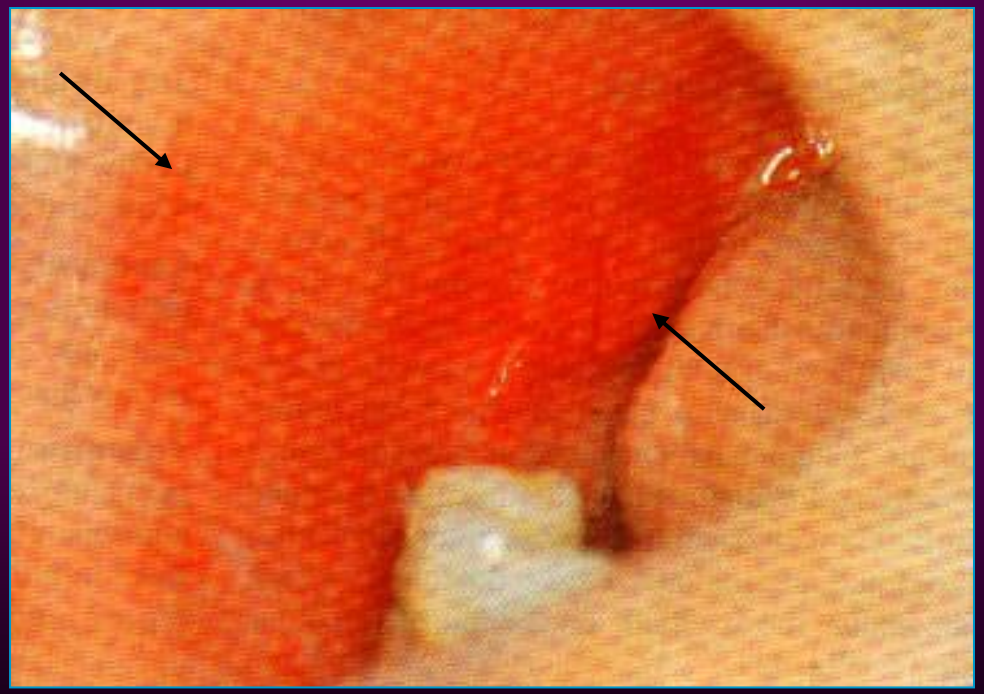
Clinical features

1. Very rare lesion- 0.02%
2. Age: majority of cases in the 5^{th}-6^{th} decades.
3. Site: buccal or palatal mucosa most frequently affected.
4. Symptoms: mostly asymptomatic, discovered on routine examination, sometimes patients may experience burning sensation.

Erythroplakia

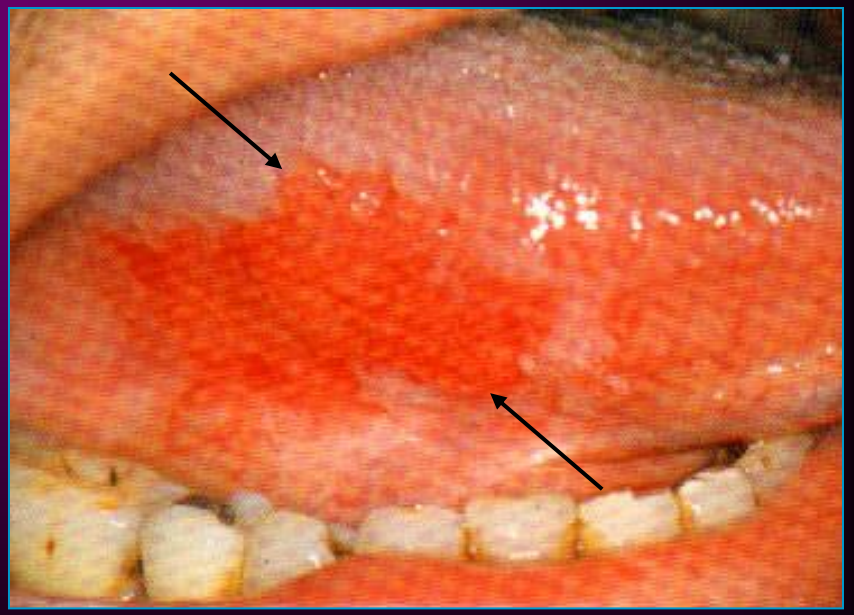
Clinical features

5. Signs: 2 clinical forms-
   - Homogenous with velvety surface
   - Speckled with white keratotic nodules



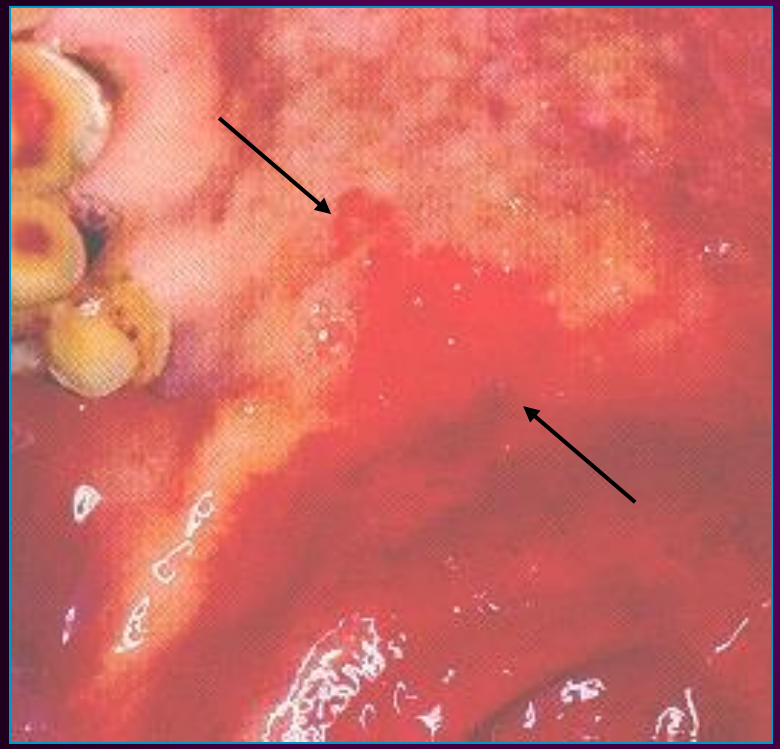
Erythroplakia

Clinical features

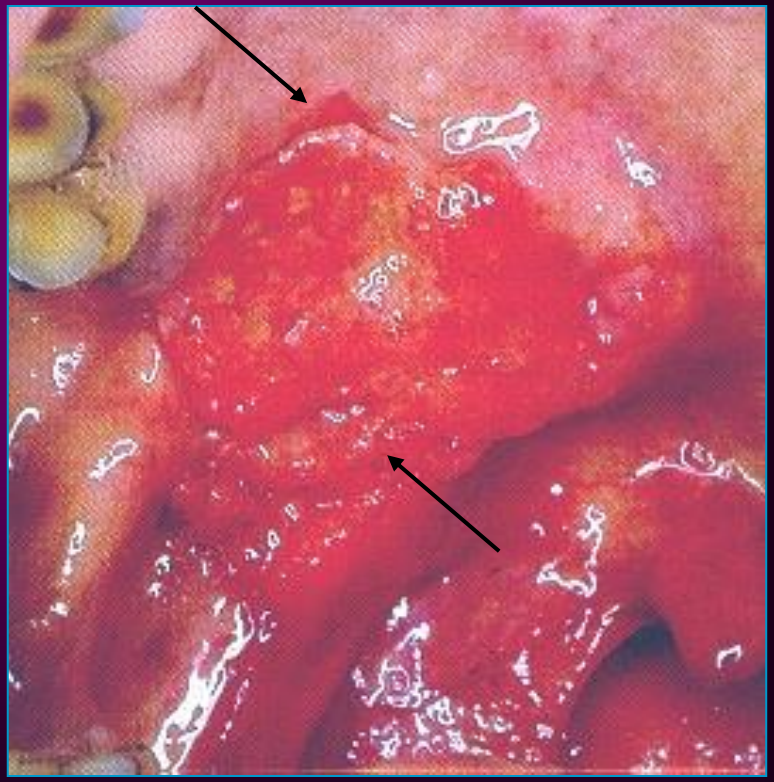


Erythroplakia

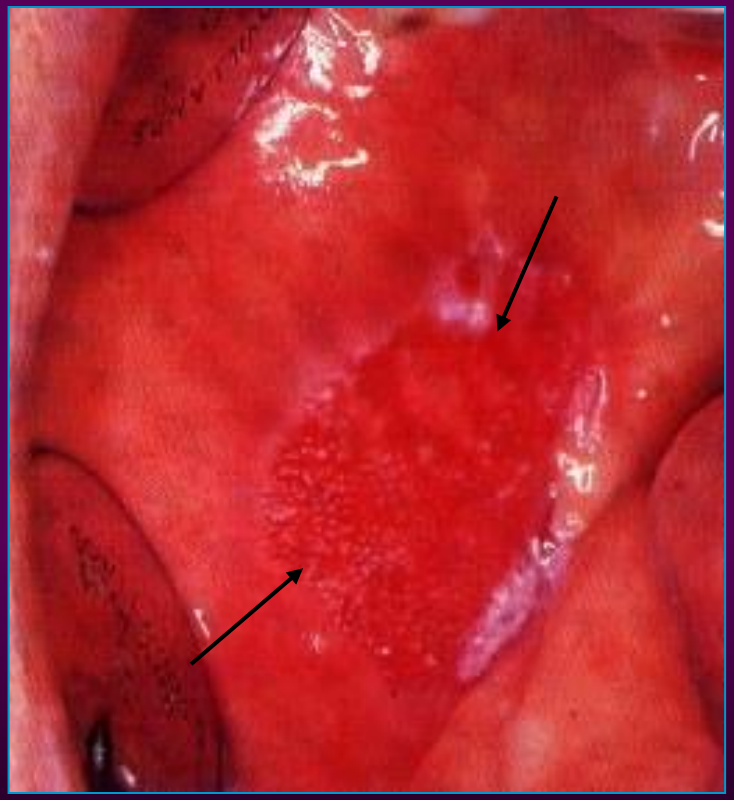
Clinical features



Malignant transformation



Clinical features



speckled erythroplakia

Erythroplakia

Investigations:

- Exfoliative cytology
- Brush / incisional / excisional biopsy
- Toluidine blue vital staining to select biopsy site

Histological features

- Severe degree of dysplasia to ca-in-situ.
- Frank malignancy may also be evident.

Differential diagnosis

Erythroplakia

Treatment:

1. Patient education and motivation to discontinue tobacco habit.
2. Identification and removal of any local irritants.
3. If lesion persists for more than 2 weeks, histological examination is carried out..
4. Dysplastic changes indicate surgical removal and long term follow-up.

Oral submucous fibrosis

Definition

"a chronic mucosal condition affecting any part of oral mucosa, characterized by mucosal rigidity of varying intensity due to fibroelastic transformation of juxtaepithelial connective tissue layer"

Oral submucous fibrosis

Etiology

1. Areca nut chewing- most probable causative factor e.g. *mawa, paan masala*
2. Tobacco abuse, chilly consumption
3. Autoimmune disease
4. Genetic predisposition, nutritional deficiency
5. Oral component of scleroderma
6. Idiopathic

Epidemiology

1. Affects up to 0.4% of Indian population.
2. Incidence is rising, especially amongst younger population.
3. Regional and racial variations- e.g. *Kerala*, *Gujarat.*

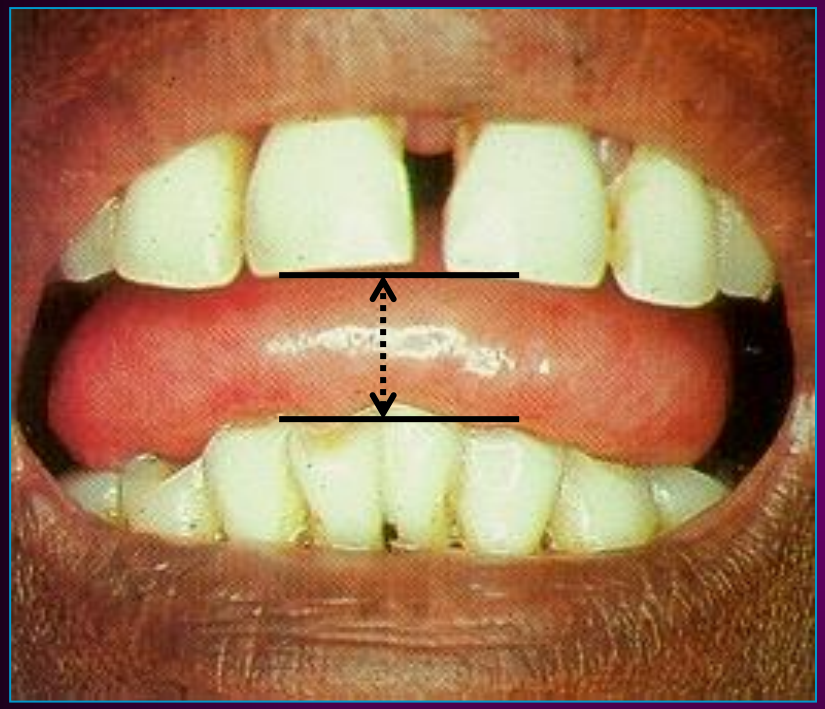
Oral submucous fibrosis

Clinical features

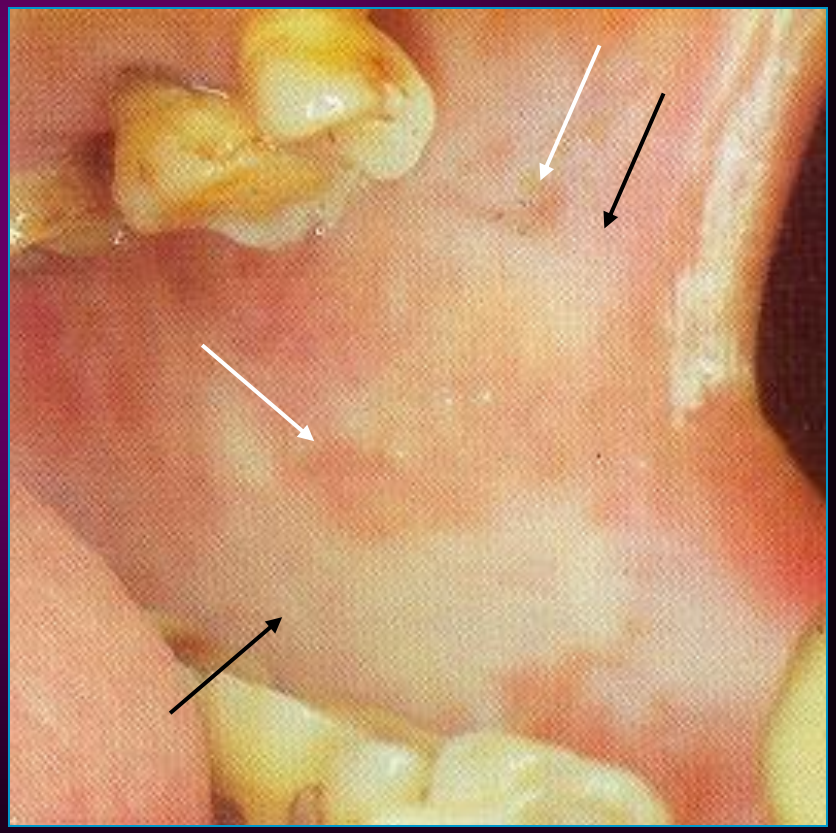
1. Age: majority of cases in the 3rd and 4th decades.
2. Sex: occurs in both sexes
3. Site: bilateral buccal involvement followed by labial, lingual, palatal mucosae. Gingiva is less commonly involved.
4. Symptoms:
   - Progressive reduction in mouth opening
   - Burning sensation, intolerance to spicy food
   - Hypersalivation or dryness of mouth
   - Difficulty in speech, swallowing or mastication

Oral submucous fibrosis

Clinical features

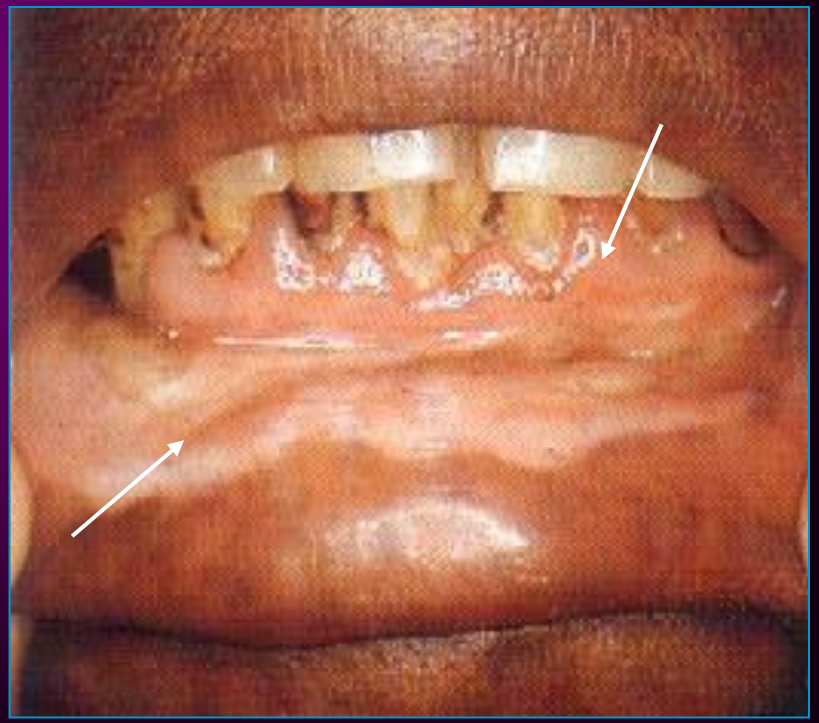


Elliptical rima oris



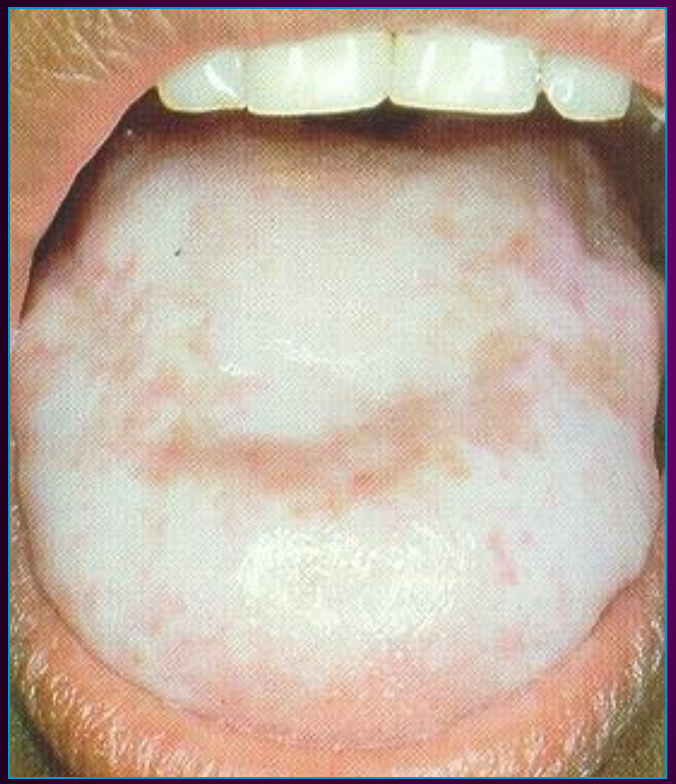
Oral submucous fibrosis

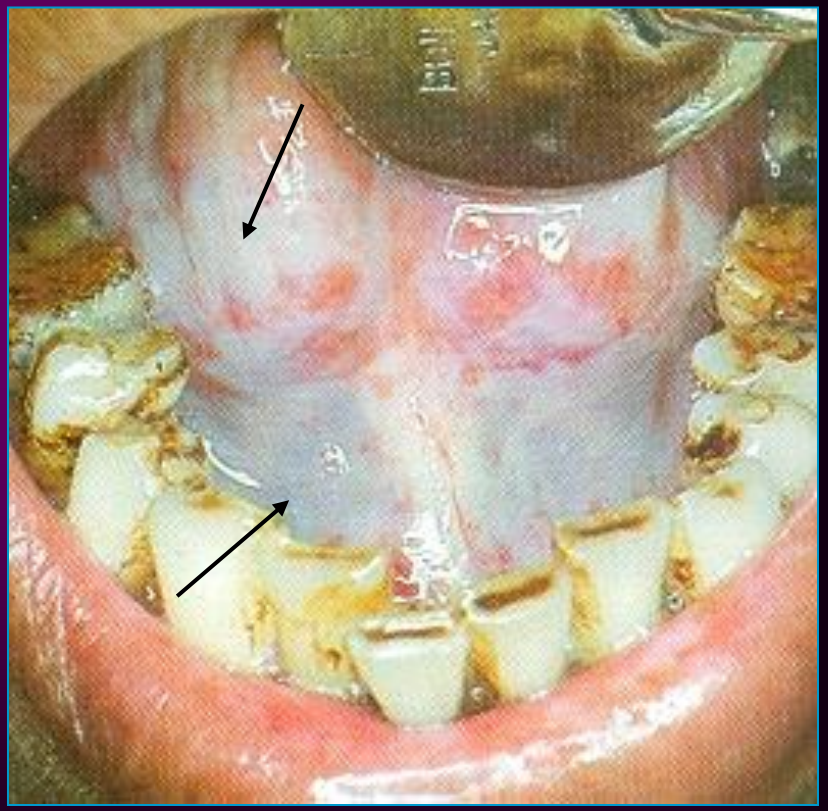
Clinical features



Oral submucous fibrosis

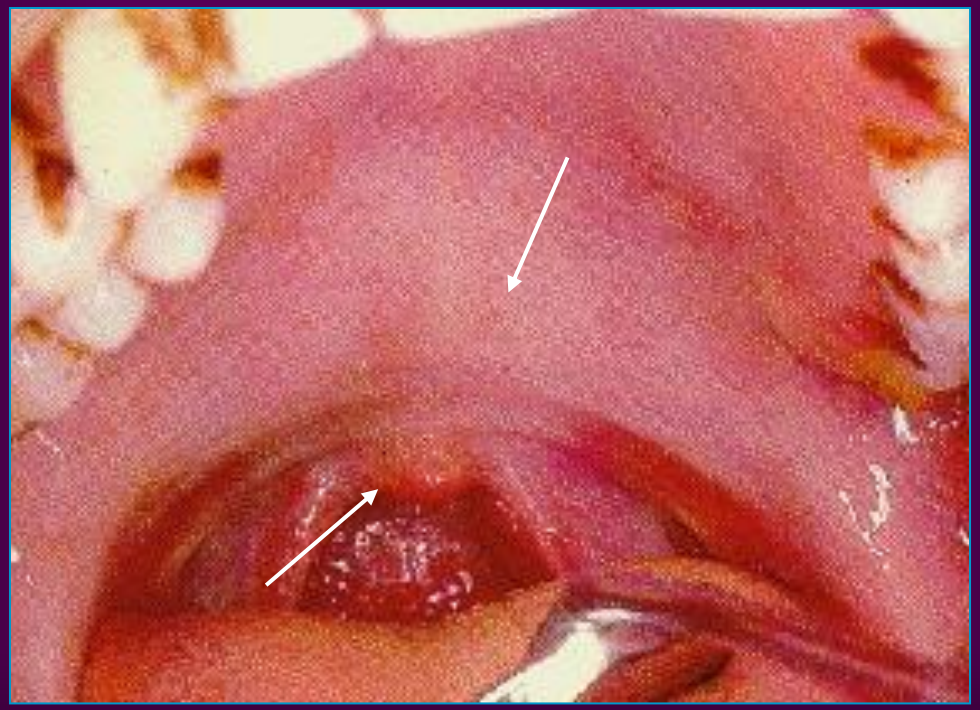
Clinical features





Oral submucous fibrosis

Clinical features



Oral submucous fibrosis

Clinical features

5. Signs:

- Reduced inter-incisal distance.
- Blanching of mucosa- '*marble-like appearance'.*
- On palpation, leathery consistency of mucosa and fibrous bands particularly in posterior parts of buccal mucosa and labial mucosa (*elliptical rima oris*) are the characteristic features.
- Shrunken uvula when soft palate mucosa is involved. Soft palate loses its mobility.
- Tongue involvement causes loss of papillae.
- Involvement of floor of mouth causes loss of tongue movements.
- Hypersalivation or dryness of mouth
- Sometimes- vesicle formation, erosions, ulcerations and hyper/de-pigmentation is seen.

Oral submucous fibrosis

Investigations:

- Clinical features are characteristic.
- Histological examination if dysplastic changes are suspected.

Histological features

- Epithelial atrophy. Sometimes dysplasia.
- Juxtaepithelial fibroelastic changes, hyalinization, reduced vascularity, chronic inflammatory response.

Differential diagnosis

Oral submucous fibrosis

Management:

1. Patient education and motivation to discontinue areca nut and tobacco habit.
2. Medical treatment:
   - Short term improvement in mouth opening and relief from burning sensation could be obtained from intra-lesional injections of steroids, placental extract, hyaluronidase. However, none of these provides long lasting relief.
   - Topical applications of anesthetics and steroids may be useful.
   - Long term use of anti-oxidants may improve the condition.

Oral submucous fibrosis

Management:

3. Surgical treatment: stripping with grafting. May add to fibrosis!
4. Oral physiotherapy and maintenance of good oral hygiene.
5. Long term follow-up is essential to detect dysplastic changes and malignant transformation.

Prognosis:

1. Poor, since condition is progressive. Worsens over time, irrespective of therapy and discontinuation of habit.
2. Rate of malignant transformation is approximately 7%-10%.

Oral submucous fibrosis

Management:

3. Surgical treatment: stripping with g… add to fibrosis!
4. Oral physiotherapy and m… hygiene.
5. Long term follo… dysplastic… detect …ant transformation.

Primary prevention is the best policy. Dentists have the opportunity and responsibility. Educate your patients!

Prognosis:

1. Poor, … condition is progressive. Worsens over time, irrespective of therapy and discontinuation of habit.
2. Rate of malignant transformation is approximately 7%-10%.

Assignment

Write 10-12 lines on the following:

~ Actinic keratosis, elastosis, Cheilitis

~ Discoid lupus erythematosus

~ Dyskeratosis congenita

~ Carcinoma in situ

~ Bowen's disease

~ Lupus erythematosus

Oral lichen planus

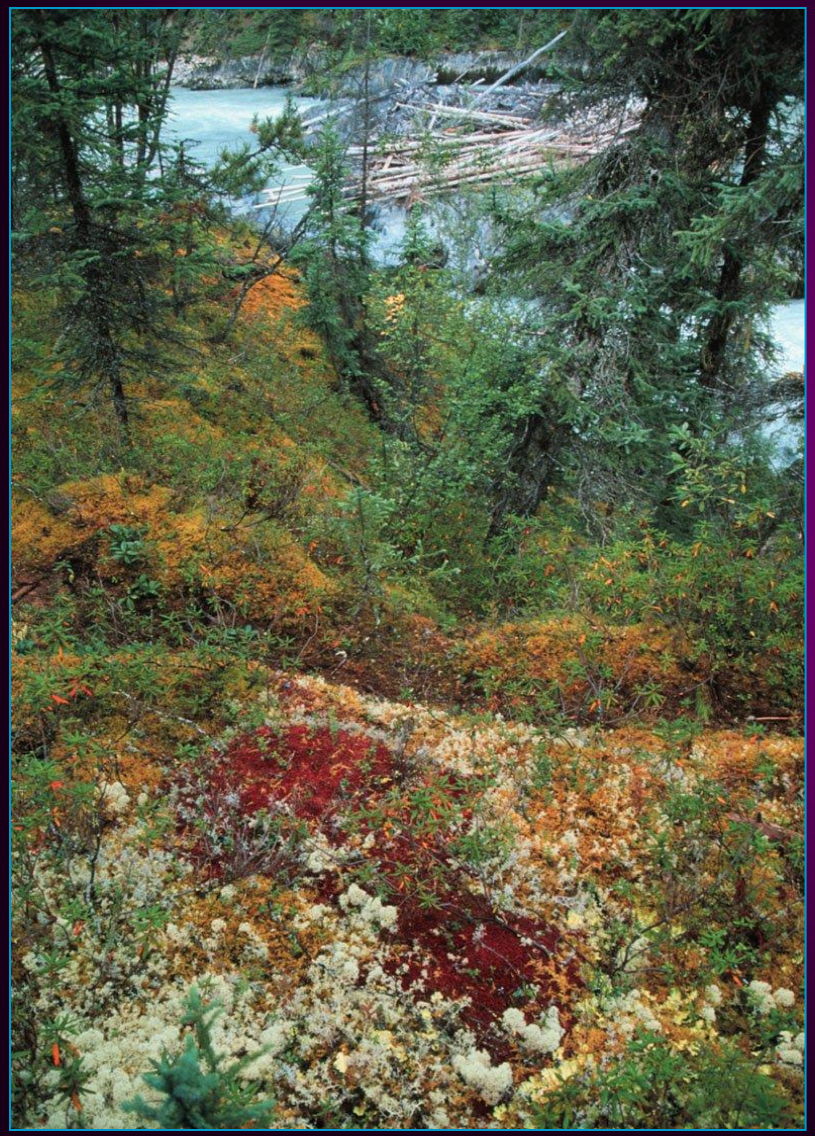
Introduction

- lichen planus is a common, chronic inflammatory disease of the skin and mucous membrane
- Prevalence is approximately 1% of the population
- 40%-50% of patients with oral lesions have skin lesions
- Oral lesions may precede or appear after skin lesions

Etiology: unknown. Probable etiology:

- Autoimmune response to probably basement membrane antigen
- Drug-induced antigen-antibody reaction
- Intense and prolonged emotional stress
- Hereditary tendency

Lichen in nature



Lichen planus is so-called because of similarity to its clinical appearance to lichen.

Lichen is a naturally occurring saprophyte (fungus+algae), that produces white linear patterns on rocks and plant wood.

Lichen in nature



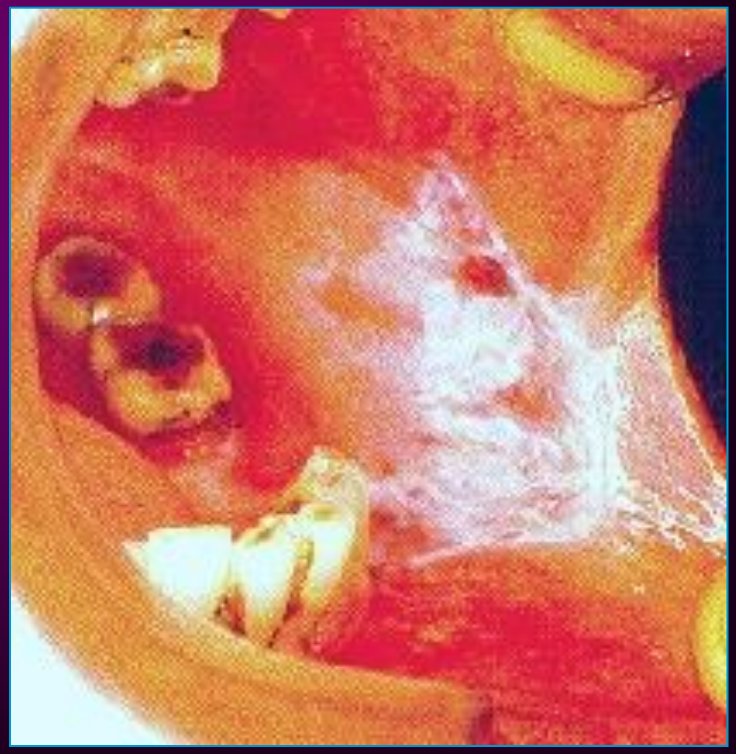






Oral lichen planus

Oral lichen planus



Oral lichen planus

Clinical features

1. Age: majority of cases in the 4rd to 6th decades.
2. Sex: females are more frequently affected
3. Site: bilateral buccal involvement followed by labial, lingual, palatal mucosae. Gingiva is less commonly involved.
4. Symptoms: *some lesions are asymptomatic*
   - Roughness/thickening of mucosa
   - Burning sensation, intolerance to spicy food
   - In some patients, intense pain causing difficulty in speech, swallowing or mastication
   - Anxiety

Oral lichen planus

Clinical features

5. Signs: *skin lesions*

- Site: forearm, anterior leg and genitalia
- Flat, violaceous, polygonal macules and papules
- Covered with fine scaling, white striations (slightly elevated, fine,s whitish lines) arranged in different patterns '*Wickhams' striae*'
- Extremely pruritic, leading to erosions and ulcerations
- Melanin pigmentation may get deposited

Oral lichen planus

Clinical features: *skin lesions*

Oral lichen planus

Clinical features: *skin lesions*

Oral lichen planus

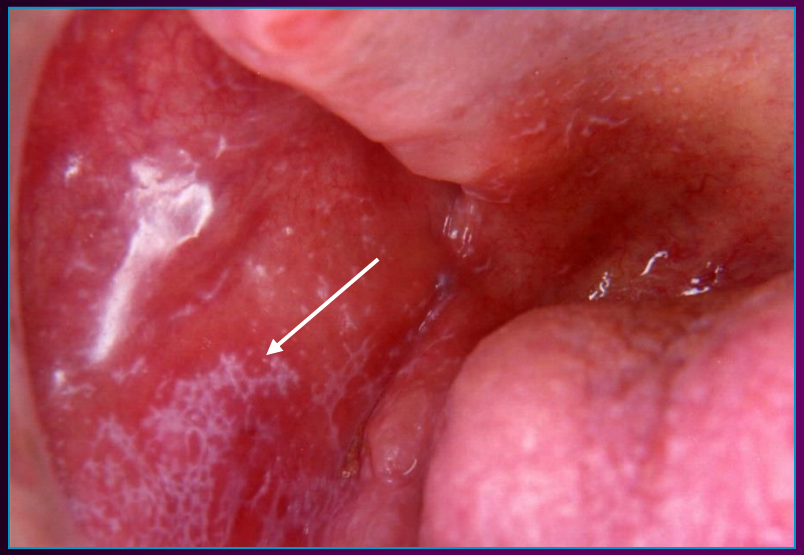
Clinical features

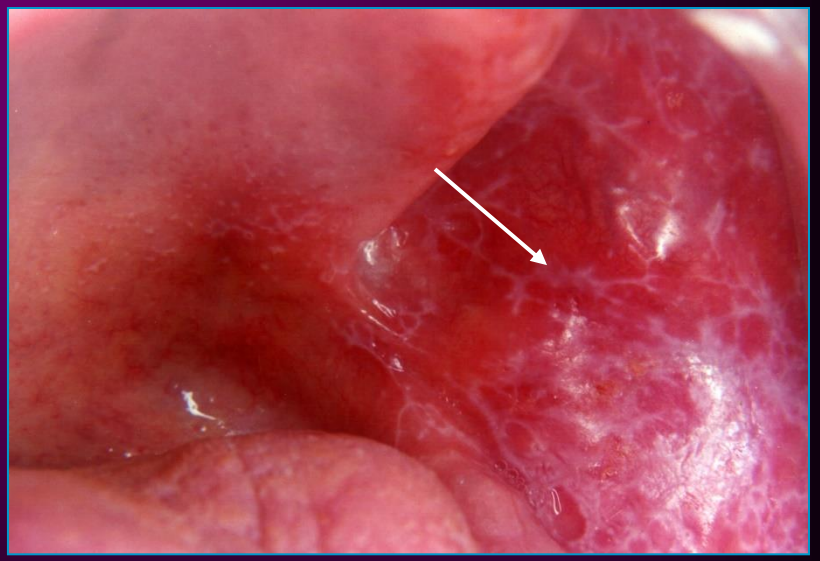
5. Signs: *oral lesions*-

- hypertrophic
- atrophic/ulcerative
- bullous

Oral lichen planus

Clinical features

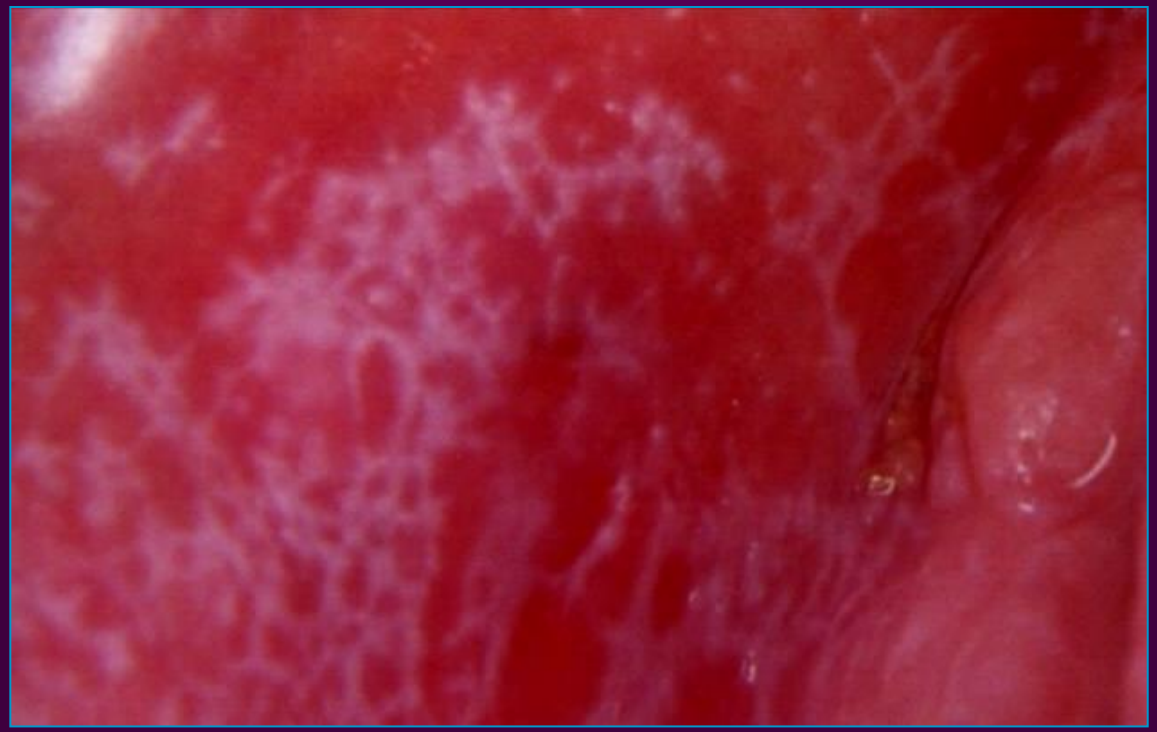




Hypertrophic: reticular

Oral lichen planus

Clinical features



Hypertrophic: reticular

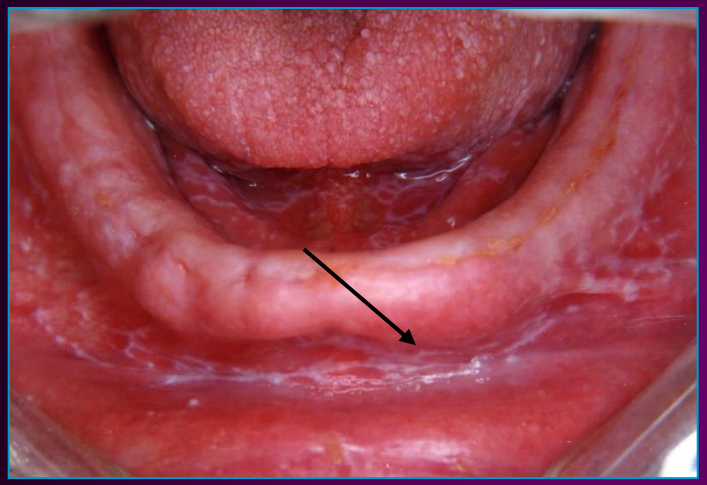
Oral lichen planus

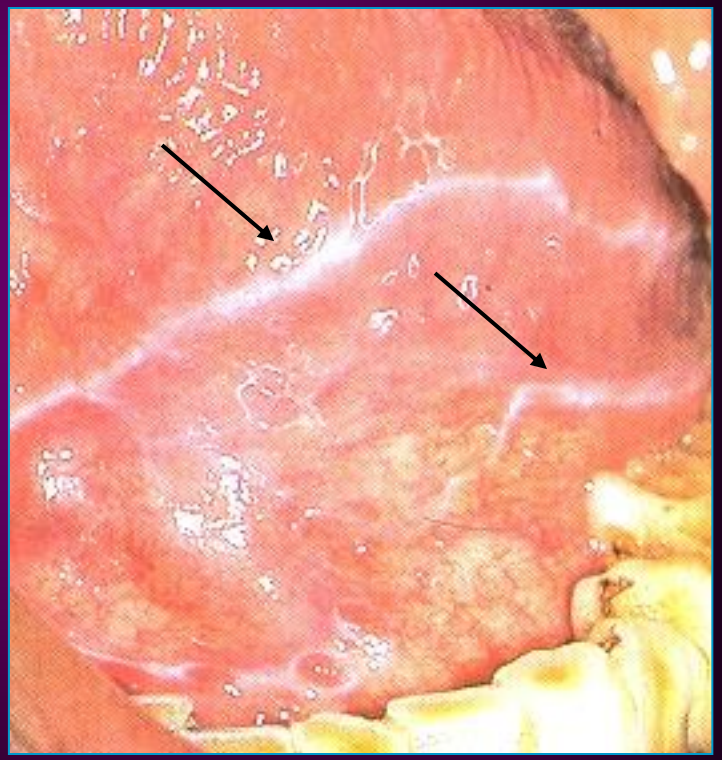
Clinical features

Hypertrophic: reticular

Oral lichen planus

Clinical features

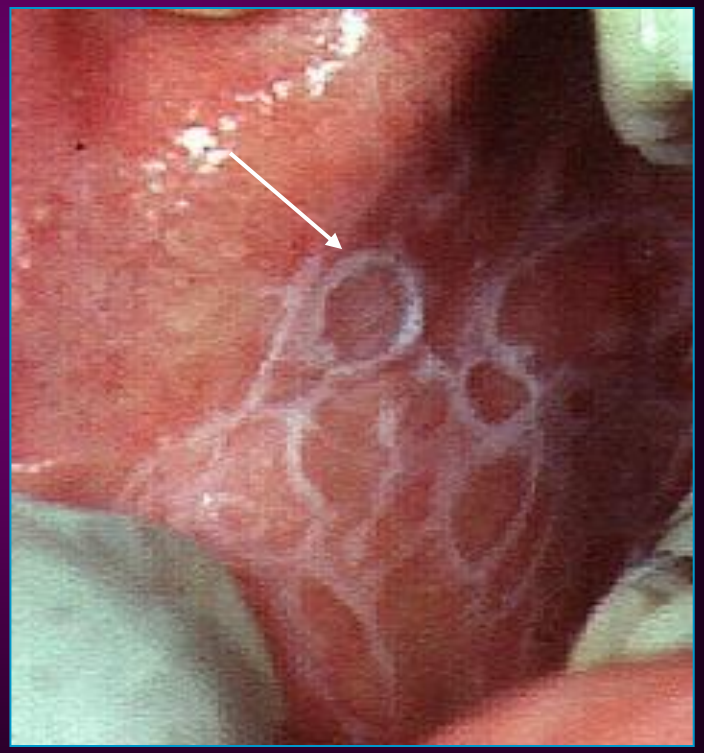




Hypertrophic: linear

Oral lichen planus

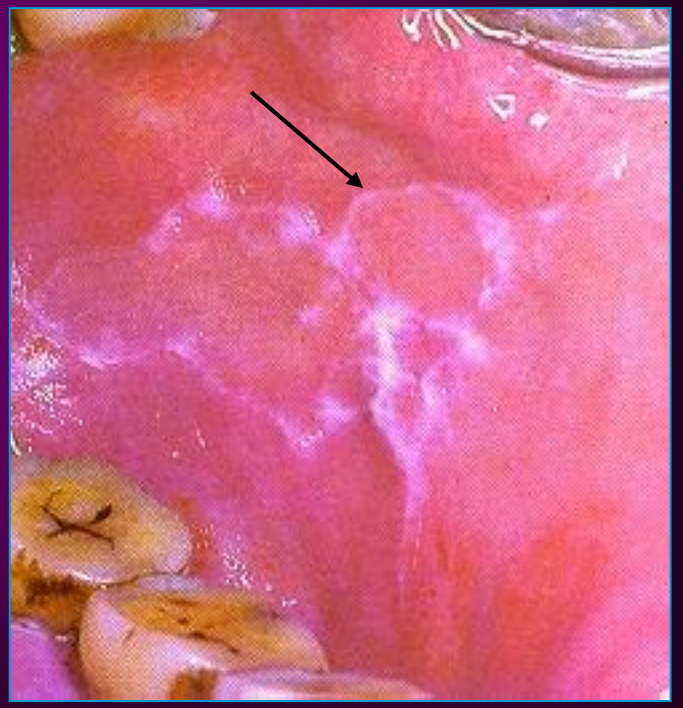
Clinical features



Hypertrophic: annular

Oral lichen planus

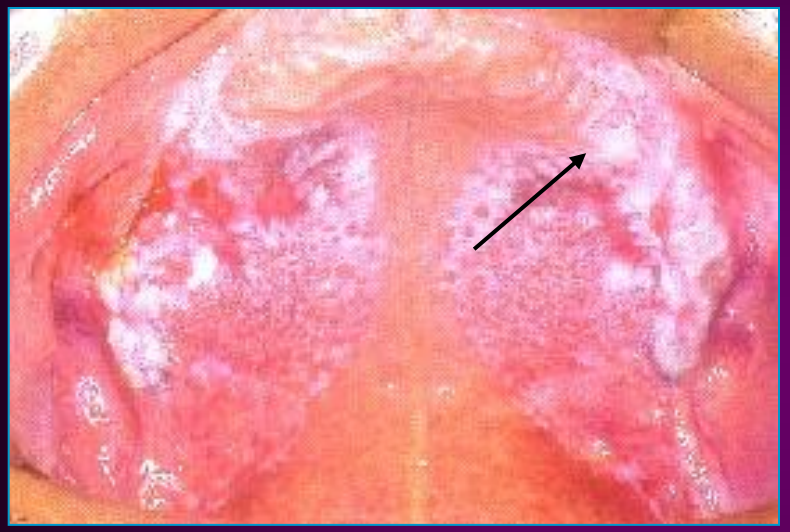
Clinical features

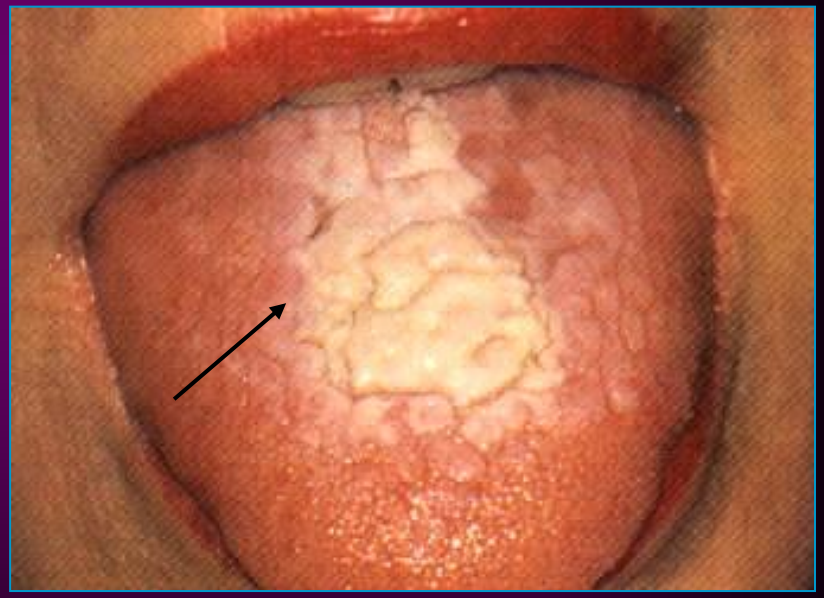


Hypertrophic: annular

Oral lichen planus

Clinical features

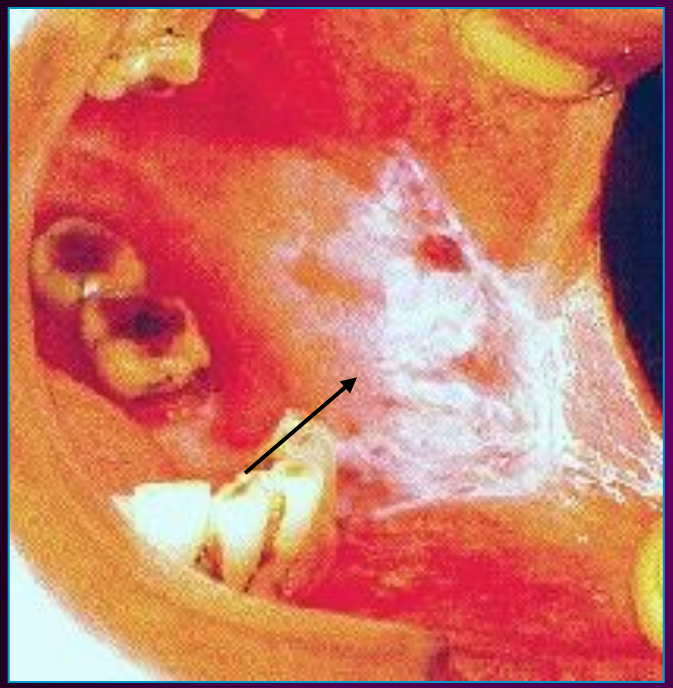


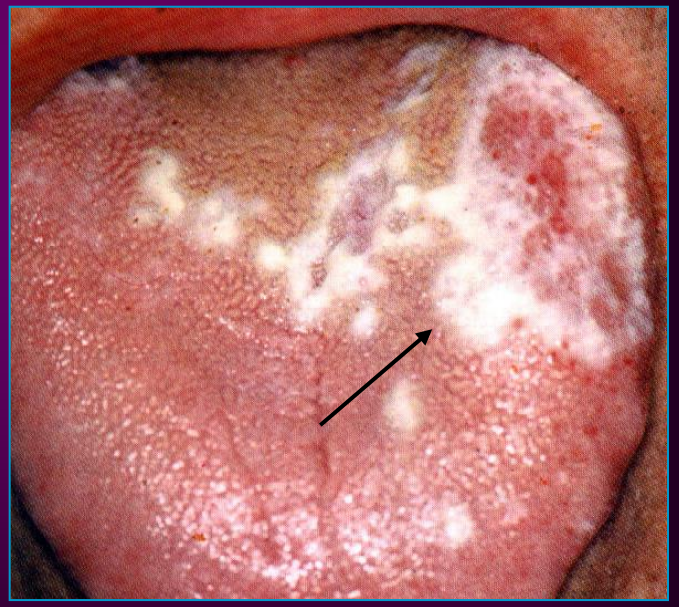


Hypertrophic: plaque-like

Oral lichen planus

Clinical features





Hypertrophic: plaque-like

Oral lichen planus

Clinical features

Hypertrophic: with melanosis

Oral lichen planus

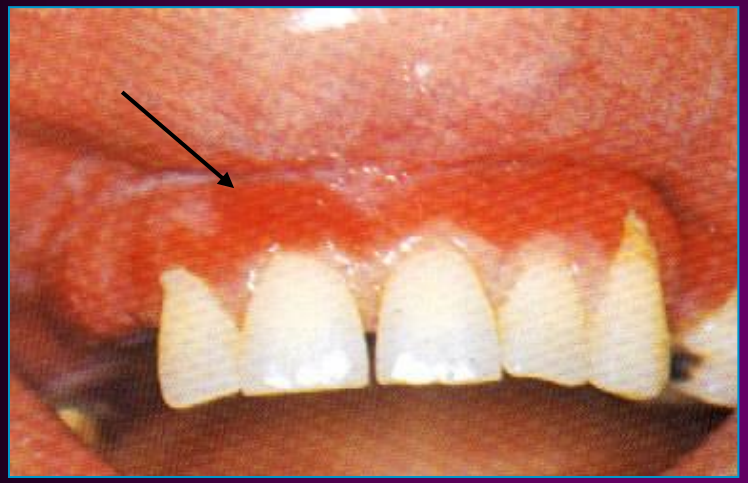
Clinical features

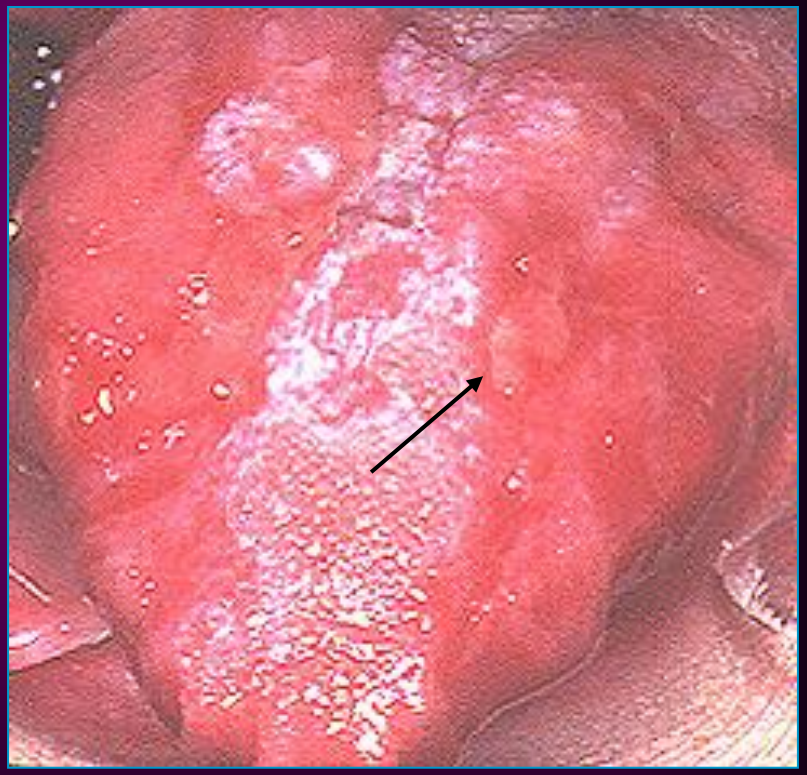
- Hypertrophic form manifests as different arrangement of white striae, viz. *reticular*, *linear*, *annular*, *lace-like*, *papular*
- Sometimes a *plaque-like* white lesions with very few striae is seen
- All forms are non-scrapable
- The pliability of mucosa is not lost
- Papillae on the tongue may not be lost
- Sometimes associated with melanosis

Hypertrophic

Oral lichen planus

Clinical features



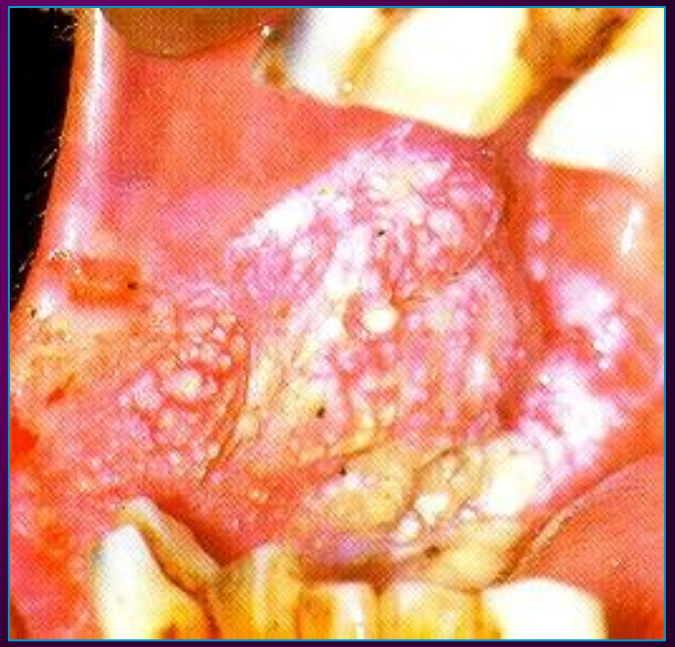


Atrophic/Ulcerative

Oral lichen planus

Clinical features

21 years later:
Malignant transformation



Atrophic/Ulcerative

Oral lichen planus

Clinical features

Atrophic/Ulcerative

Oral lichen planus

Clinical features

Atrophic/Ulcerative

Oral lichen planus

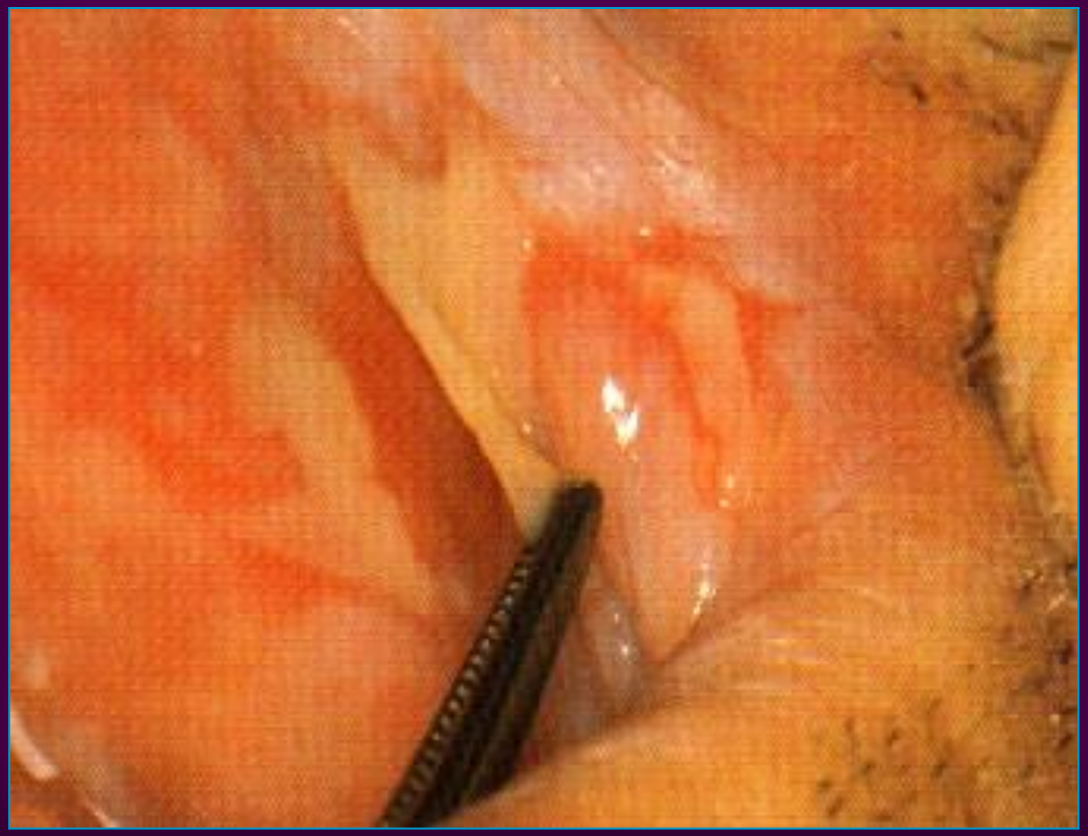
Clinical features

- Atrophic form is less common
- Atrophy may vary from erosions to deep ulcerations
- Usually associated with some hypertrophic (striae) form
- Tender to palpation and may bleed
- More prone to malignant transformation

Atrophic/Ulcerative

Oral lichen planus

Clinical features



Bullous

Oral lichen planus

Clinical features

- Least common
- Appears as a bulla surrounded by some striations
- Ruptures easily to form ulceration
- Tender to palpation and may bleed

Bullous

Oral lichen planus

Investigations:

1. Clinical features are characteristic enough to diagnose hypertrophic forms. Plaque-like lesion can be differentiated from leukoplakia by histological examination.
2. Histological examination may be required to diagnose atrophic/ulcerative and bullous forms.
3. Immunofluorescence studies may be useful to differentiate from other ulcerative or bullous conditions.
4. To detect *Grinspan's syndrome*: OLP, diabetes mellitus, hypertension.

Differential diagnosis

Oral lichen planus

Clinical course: *unpredictable*

1. Hypertrophic forms may remain asymptomatic and unchanged for years. Spontaneous regression may occur in some patients. Rarely, hypertrophic form may change to atrophic/ulcerative form and become symptomatic.
2. Atrophic forms are always symptomatic and rarely regress spontaneously; hence, need treatment.
3. All forms may recur after regression or successful treatment.
4. Malignant transformation is rare (about 0.4%) and usually takes place in atrophic forms.

Oral lichen planus

Management:

- OLP is neither curable nor preventable.
- The clinical course and behaviour is unpredictable.
- Management objectives, therefore, are symptomatic relief, general support and an attempt to cure.
- Asymptomatic lesions should be periodically observed for any changes.

1. Medical management
2. Surgical management

Oral lichen planus

Management: *medical*

1. Hypertrophic form without symptoms:
   - Patient counseling
   - Periodic observation
2. Hypertrophic form with mild symptoms:
   - Patient counseling
   - Topical anesthetics- 5% lignocain (*Xylocain*), 0.15% benzydamine (*Tantum oral rinse*)
   - Periodic observation

Plaque-like lesions may benefit from a course of anti-fungal therapy.

Oral lichen planus

Management: *medical*

3. Atrophic form with mild to moderate symptoms:
   - Patient counseling
   - Topical anesthetics- 5% lignocain (*Xylocain*), 0.15% benzydamine (*Tantum oral rinse*) before every meal
   - Topical steroids- 0.1% triamcenolone acetonide (*Kenalog*) in Orabase to be applied after every meal and at bed time
   - Anti-anxiety medication- 5 mg diazepam (*Valium*) at bed time for 1-2 weeks
   - Periodic observation

Oral lichen planus

Management: *medical*

4. Atrophic form with severe symptoms and bullous form:
   - Basic management is similar to atrophic form with mild to moderate symptoms
   - Additionally, systemic steroids may be prescribed-
     - Prednisolone (*Wysolone*), 20 mg tablet, twice a day for 5 days.
     - Betamethasone (*Betnesol*), 0.5 mg tablet thrice daily for 5 days.
     - Systemic steroid therapy should not be withdrawn abruptly. Dose must be tapered.
   - Intra-lesional injection of steroid (betamethasone, 4 mg/ml solution, *Betnesol*) may heal stubborn lesions.

Oral lichen planus

Management: *medical*

5. Cyclosporin, chloroquine, retinoids and UV-light have shown resolution of lesions in some patients. However, therapy with these is still under trials and should not be prescribed routinely.

Management: surgical removal (scalpel, cryoprobe, laser) may help in stubborn lesions. However, recurrence may yet occur.

Prognosis: is unpredictable, particularly in atrophic forms. Lesions may resolve completely, only to recur later. Therefore, management is customized to patient's needs.

Differential diagnosis

Red Lesions

1. Acute atrophic candidiasis
2. Chronic atrophic candidiasis
3. Erythroplakia

Red & White Lesions

1. Stomatitis nicotina
2. Speckled leukoplakia/erythroplakia
3. Atrophic form of lichen planus

White Lesions

Differential diagnosis

White Lesions: *scrapability test*	Scrapable
1. Leukoedema	No
2. Linea alba	No
3. Fordyce's spots	No
4. Burns	Yes
5. Habitual cheek biting	No
6. Acute pseudomembranous candidiasis	Yes
7. Chronic hyperplastic candidiasis	No
8. Traumatic keratosis	No
9. Leukoplakia	No
10. Oral submucous fibrosis	No
11. Hypertrophic form of lichen planus	No

Differential diagnosis

Scrapability test

Scrapable lesions

burns, acute pseudomembranous candidiasis

Non-scrapable lesions

leukoedema, linea alba, pots, habitual cheek biting, chron ic candidiasis, traumatic kerat plakia, hypertrophic form of lichen planus, submucous fibrosis

STRETCHABILITY TEST

Differential diagnosis

White Lesions: *stretchability test*	Stretchable
1. Leukoedema	Yes
2. Linea alba	Yes
3. Fordyce's spots	Yes
4. Habitual cheek biting	Yes
5. Chronic hyperplastic candidiasis	No
6. Traumatic keratosis	No
7. Leukoplakia	No
8. Oral submucous fibrosis	No
9. Hypertrophic form of lichen planus	Yes

Differential diagnosis

Scrapability and Stretchability test

Non-scrapable, stretchable lesions

leukoedema, linea alba, Fordyce's spots, habitual cheek biting, hypertrophic form of lichen planus

Non-scrapable, non-stretchable Lesions

chronic hyperplastic candidiasis, traumatic keratosis, leukoplakia, oral submucous fibrosis

Assignment

1. Describe the steps in differential diagnosis of red and white lesions of oral mucosa.
2. List differences in clinical features that aid in differential diagnosis of
   - Red lesions
   - Red and white lesions
   - Scrapable white lesions
   - Non-scrapable, stretchable white lesions
   - Non-scrapable, non-stretchable white lesions.