Oral Medicine

Lecture 8

Red and White Lesions of the Oral Mucosa

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Oral Potentially Malignant Disorders (PMD)

The development of oral **leukoplakia** and **erythroplakia** as potentially malignant lesions, involves different **genetic events**.
This concept is supported by the fact that **markers** of genetic defects are differently expressed in different leukoplakias and erythroplakias.

Activation of oncogenes and deletion and injuries to suppressor genes and genes responsible for DNA repair will all contribute to a defective functioning of the **genome** that governs cell division.

- Following a series of **mutations**, a malignant transformation may occur.

Carcinogens such as **tobacco** may induce hyperkeratinization, tobacco-associated leukoplakia; which **is reversible** following cessation, but at some stage, mutations will lead to an unrestrained proliferation and cell division.

Oral Leukoplakia

Leukoplakia is defined as a **white plaque** of questionable risk for malignant transformation having excluded other known white lesions or disorders that carry **no increased** risk for cancer. Leukoplakia is thus a diagnosis of exclusion.

- By definition, leukoplakia is idiopathic, having excluded white patches of known etiology.
- The exception to this rule is that smoking is recognized as an etiological or exacerbating factor and smoker's keratosis may regress when the irritant is removed.

- **The prevalence** of oral leukoplakia according a comprehensive global review points 1.5 - 2.6%.
- **Most** oral leukoplakias are seen in patients **beyond** the age of 50 and infrequently encountered below the age of 30.
- Leukoplakias are more common in men but a slight majority for women has been found in some studies.

It can be further divided into:

- **Homogeneous type** characterized as a white, often well-demarcated plaque with an identical reaction pattern throughout the entire lesion.
- **Non-homogeneous type** combined appearance of white and red areas, the nonhomogeneous oral leukoplakia has also been called **erythroleukoplakia** and **speckled leukoplakia.**

- Oral leukoplakia may be found at **all sites** of the oral cavity
- Non-smokers have a higher percentage of leukoplakias at the **border of the tongue compared with smokers.**

Oral erythroplakia

Erythroplakia is a fiery red patch that cannot be characterized clinically or pathologically as any other definable disease.

Oral erythroplakia is **not** as common as oral leukoplakia, and the prevalence has been estimated to be in the range of 0.02 – 0.1%.

- The gender distribution is reported to be **equal**

Erythroplakia is defined as a red lesion of the oral mucosa that excludes other known pathologies.

Erythroplakia is usually asymptomatic, although some patients may experience a burning sensation with food intake.

A special form of erythroplakia has been reported that is related to reverse smoking of *chutta*, predominantly practiced in India.

Both those diagnoses thus require exclusion of other similar-looking lesions of known causes or mechanisms before being applied.

The relative importance of one versus the other is that leukoplakia is very common and can sometimes transform into cancer, whereas erythroplakia is rather uncommon but frequently represents a precursor to cancer.

- **The floor of the mouth & the lateral borders of the tongue have been considered high-risk sites for malignant transformation**

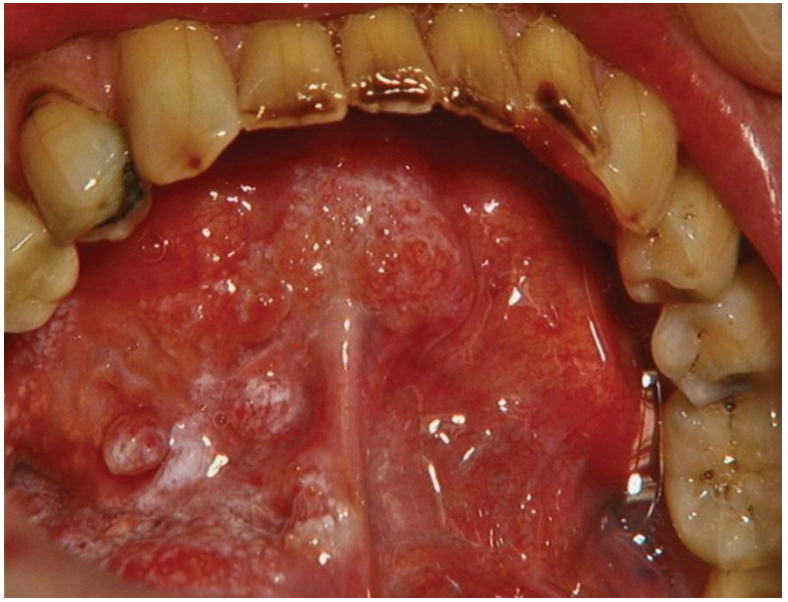
- These sites have also been found to have a higher frequency of loss of heterozygosity compared with low-risk sites.

However, **the size of the lesion and the homogeneous/ nonhomogeneous pattern** are also decisive characteristics for the prognosis, whatever the site.

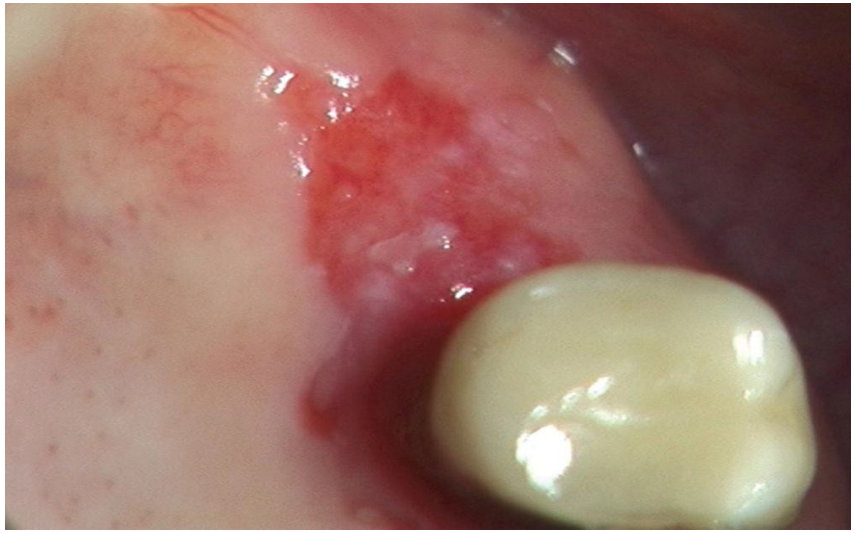
- Hairy leukoplakia is **not considered a true leukoplakia**, since the etiology and infective agent (EBV) are known and the risk of malignant transformation appears to be almost nonexistent.



A homogeneous leukoplakia



A nonhomogeneous leukoplakia.



Erythroplakia at the alveolar ridge. The patient later developed a squamous cell carcinoma



upper buccal sulcus, which transformed into a squamous cell carcinoma two years later.
A

Diagnosis

- The diagnostic procedure of oral leukoplakia and erythroplakia is identical.
- The provisional diagnosis is based on the clinical observation of a white or red patch that is **not** explained by a definable cause, such as **trauma**.
- If trauma is suspected, the cause, such as a sharp tooth or restoration, should be eliminated.
- If healing does not occur in **two weeks, a tissue biopsy** is essential to rule out malignancy.

- It has been reported that **91%** of histologically assessed erythroplakias showed invasive carcinoma or carcinoma in situ, and in **9%** there was moderate to severe dysplasia.
- Another study showed that severe dysplasia and frank carcinoma in 75% and mild to moderate dysplasia in 25%.
- Thus, **all** erythroplakias should be considered as **sinister**.
- Any **red mucosal lesion** without an apparent local cause or not fitting into other known red lesions, and not regressing following removal of possible cause or two weeks of treatment, should be considered a cancer unless histologically proven otherwise.

surgery has been strongly questioned, as squamous cell carcinomas are almost equally prevalent in patients subjected and not subjected to surgery ?

This may be explained by **genetic defects** even in clinically normal mucosa surrounding the removed lesion and is supported by a concept referred to as **Field Cancerization**.

Field cancerization is caused by simultaneous genetic instabilities in the epithelium of several extra lesional sites that may lead to SCC.

However, in the absence of evidenced-based treatment strategies, **Surgery** will remain the treatment of choice for oral leukoplakias and erythroplakias.

Malignant transformation of oral leukoplakias; up 20% over 30 years depending on site &habits

- Homogeneous leukoplakias are associated with a decreased risk for malignant transformation than nonhomogeneous and erythroplakias, and lesions not exceeding **200 mm2** appear to have a better prognosis than larger lesions .

Proliferative Verrucous Leukoplakia (PVL)

Oral leukoplakias, where the white component is dominated by papillary projections, similar to oral papillomas, are referred to as **verrucous or verruciform leukoplakias**

Oral leukoplakias with a more aggressive proliferation pattern and high recurrence rate are designated as **proliferative verrucous leukoplakia** (PVL)

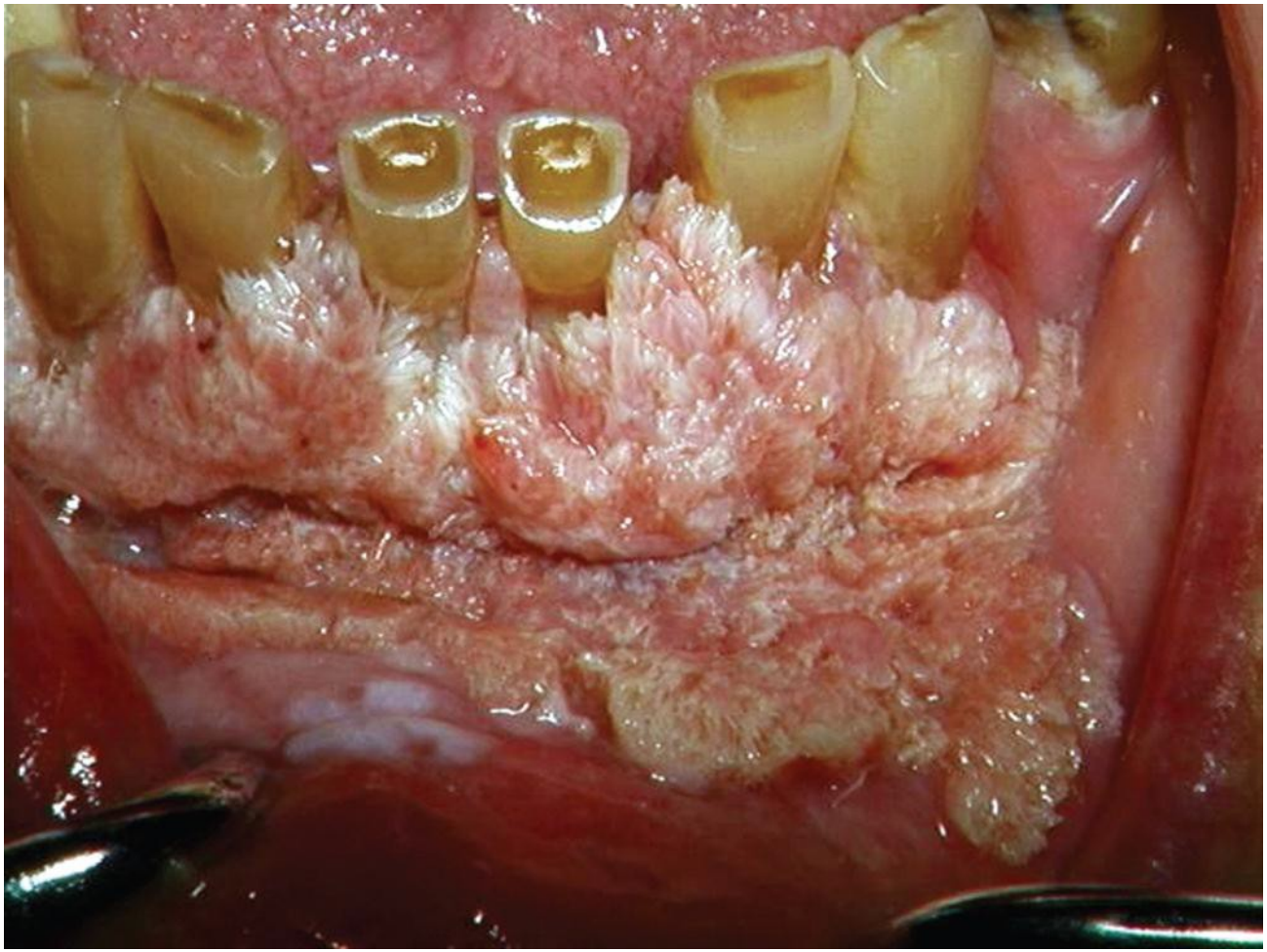
Diagnosis cannot usually be established at a single consultation. **Histologically** the lesion may appear benign, but **clinically** it behaves as a malignancy with gradually spreading leukoplakia, often gingivally.

PVL is usually

- Seen in older women, and the lower gingiva is a predilection site.
- The malignant potential is very high and verrucous carcinoma or squamous cell carcinoma may be present at the primary examination.
- Similar to what is seen in oral papillomas, the PVL has been suspected to have a **viral etiology.**



A proliferative verrucous leukoplakia progressively extending from the buccal mucosa, across the gingiva to the ventral surface of the tongue in a 48-year-old female.



A proliferative verrucous leukoplakia in an80-year-old woman

In general, **clinical management of leukoplakia** relates to a combination of clinical (site, homogeneity, size, behavior) and histologic features (presence and degree of dysplasia and inflammation). While those leukoplakias that are homogeneous, stable, on lower-risk sites such as the buccal mucosa and show no dysplasia, can be reviewed annually (assuming no change in clinical characteristics).

Those showing clinical characteristics of mixed appearance, on high-risk sites, changing in size, in smokers, and histologically showing a degree of dysplasia will need to be reviewed more regularly. It can be deduced that a reproducible risk score from this combination of features would be very helpful for clinicians. An algorithm for management is shown in Figure 4-25.

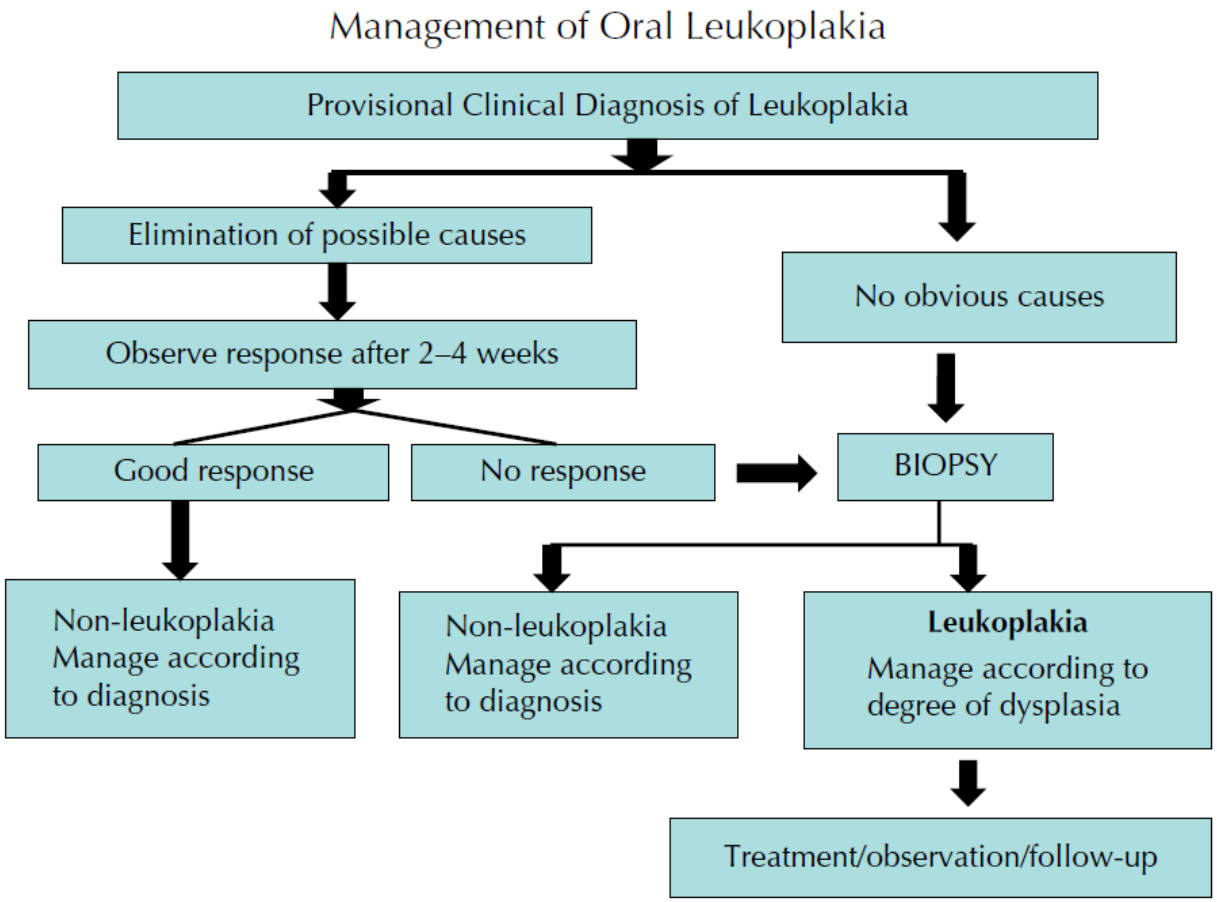


Figure 4-25 An algorithm for the management of oral leukoplakias.

No consensus has been reached regarding management and follow-up of oral leukoplakias & erythroplakias.

- A general recommendation is to re-examine the premalignant site irrespective of surgical excision
- **every three months** for **the first year**.
- If the lesion **does not** relapse or change in reaction pattern, the follow-up intervals may be extended to **once every six months**.

- New biopsies should be taken if **new clinical** features emerge.

- **Following five years** of **no** relapse, self-examination may be a reasonable approach.

Oral Submucous Fibrosis

Is a chronic disease affecting the oral mucosa, as well as the pharynx and the upper two-thirds of the esophagus

Areca nuts is **the etiology behind oral submucous fibrosis.**

Areca nut–derived products are commonly used by several hundred million individuals in different parts of ASIA.

- There is dose dependence between **areca quid** chewing habit and the development of this oral mucosal disorder.

Areca nuts **contain alkaloids, coline** seems to be a primary etiologic factor. **Arecoline** has the capacity to modulate **matrix metalloproteinases**, **lysyl oxidases**, and **collagenases**, all affecting the metabolism of collagen, which leads to an **increased fibrosis**.

- During the development of fibrosis, a decrease in water-retaining proteoglycans will occur in favor of increased **collagen type I** production.
- Genetic predisposition of importance for the etiology.
- Polymorphism of the gene, which is coding for **tumor necrosis factor α** (TNF-α), has been reported to promote the development of the disorder.
- Fibroblasts are stimulated by TNF-α, thereby participating in the development of fibrosis.
- Abnormalities of other **cytokines** of importance are transforming growth factor ß and interferon-γ, which may lead to increased production and decreased degradation of collagen.

Oral complications are most commonly observed: on the lips, buccal mucosa, retro molar area, and soft palatal mucosa.
It is widespread in India, Pakistan, Bangladesh, and Sri Lanka and in immigrants coming from these regions.

- The global incidence estimated at 2.5 million individuals.
- The prevalence in Indian populations is 5% for women and 2% for men.
- Individuals in less than 20 years old seem to be affected more commonly.
- This is reflected in the **advertisement** of areca nut products, which is directed against younger age groups.

Clinical Findings

- The **First Signs** are erythematous lesions, sometimes in conjunction with petechiae, pigmentations,and vesicles.
- Followed by a paler mucosa, (white marbling)
- The most prominent clinical characteristics include fibrotic bands located beneath an atrophic epithelium.
- Increased fibrosis eventually interferes with speech, tongue mobility, and a decreased ability to open the mouth.
- The atrophic epithelium may cause inability to eat hot and spicy food.

Diagnosis

The diagnosis of oral submucous fibrosis is based on the clinical characteristics and on the patient's report of a habit of betel chewing.

An international consensus has been reached where at **least one** of the following characteristics should be present :

- Palpable fibrous bands
- Mucosal texture feels tough and leathery
- Blanching of mucosa together with histopathologic features consistent with oral submucous fibrosis.

(atrophic epithelium with loss of rete ridges and juxta-epithelial hyalinization of lamina propria)



Marble-like appearance of the right buccal mucosa in a patient with submucous fibrosis

Management

Products derived from areca nuts are carcinogenic, regardless of concomitant use of tobacco products.

Thus, treatment of oral submucous fibrosis should be focused on cessation of the chewing habits.

If this is successfully implemented, early lesions have a good prognosis as they may regress.

- Several treatment strategies have been tried, such as:
  - Topical and systemic steroids,
  - Hyaluronic acid, interferon-γ,
  - Supplement of vitamins and nutrients,
  - Repeated dilatation with physical devices,
  - and surgery.

None of these treatments have reached general acceptance and the long-term results are uncertain.

- Malignant transformation of oral submucous fibrosis has 0.7–1.3% per annum and the incidence over a 10-year period at approximately 8%.

- Epithelial dysplasia in OSMF tissues appeared to vary from 7- 25% **depending** on the study population.

Immunopathologic Diseases

Lichen Planus

It is a family of lesions with different etiologies

- With a common clinical and histologic appearance.
- Neither clinical nor histopathologic features enable discrimination between different lichenoid reactions but may be used to distinguish **them** from other pathologic conditions of the oral mucosa.
- Skin and mucous membranes are most commonly involved.
- In addition to oral mucosa, other mucous membranes (e.g., genitals in women, esophagus, rectal area) as well as scalp and nails can be affected.

This group include the following disorders:

•• Oral lichen planus
•• Oral lichenoid contact reactions)OLCR)
•• Oral lichenoid drug eruptions
•• Oral lichenoid reactions of graft-versus-host disease (GVHD)

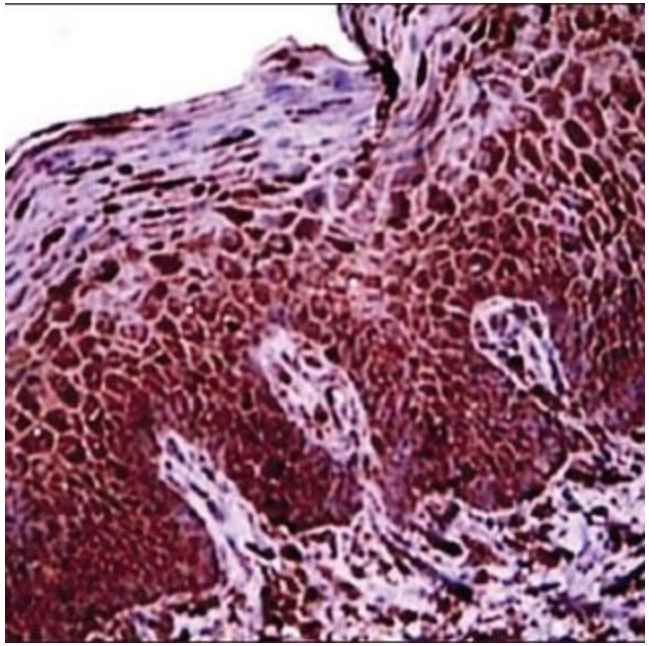
OLCRs

These lesions represent a delayed hypersensitivity reaction to constituents derived from dental materials or flavoring agents in foods and other ingested substances.

Oral Lichen Planus

- ✓ The etiology is not known
- ✓ It has become evident that the **immune system** has a primary role in the development of this disease.
- ✓ This is supported by the histopathologic characteristics of a subepithelial band–formed infiltrate dominated by **T lymphocytes** and **macrophages** and **the degeneration of basal cells** known as **liquefaction degeneration**

- ❑ Cytotoxic CD8+ T lymphocytes are responsible for apoptosis of the keratinocytes in the basal cell layer. These lymphocytes are a major component of the subepithelial inflammatory infiltrate in the mucosal lamina propria.
- ✓ These features can be interpreted as an expression of the cell-mediated arm of the immune system being involved in the pathogenesis of OLP through **T-lymphocyte cytotoxicity directed against antigens expressed by the basal cell layer**.
- ✓ It is **not** possible to identify a single etiologic factor behind OLP.
- ✓ Other factors, such as **stress**, may also be of importance to establish this inflammatory process. It is not unusual that patients report that they have been exposed to negative social events **months before** the onset of the disease.
- ✓ Altogether, this makes the etiology behind OLP a multifactorial process



histopathologic characteristics of lichen planus

- During recent years, an association between OLP and hepatitis C virus (HCV) has been described in small proportions, possibly geographically based;
- OLP prevalence is also enhanced in those with thyroid diseases.

- Prevalence OLP have been reported and vary from 0.5 – 2.2%
- Among referred patients, **women** is higher than that of men,
- The condition usually occurs in people older than 40 years, the mean age of onset being 53 years.
- It is very rarely encountered in children
- Does not seem to have a hereditary predisposition.

Clinical Findings

Some have long periods of remission, while others have frequent exacerbations and are not very responsive to treatment.

OLP may contain both red and white elements which can be a part of the following clinical types:

•• Reticular •• Papular •• Plaque-like•• Bullous••Erythematous ••Ulcerative

OLP confined to the **gingiva** may be entirely erythematous, with **no reticular or papular** elements present, and this type of lesion has to be confirmed by **a biopsy**.

The explanation of the different clinical manifestations of OLP is related to the magnitude of the **subepithelial inflammation**.

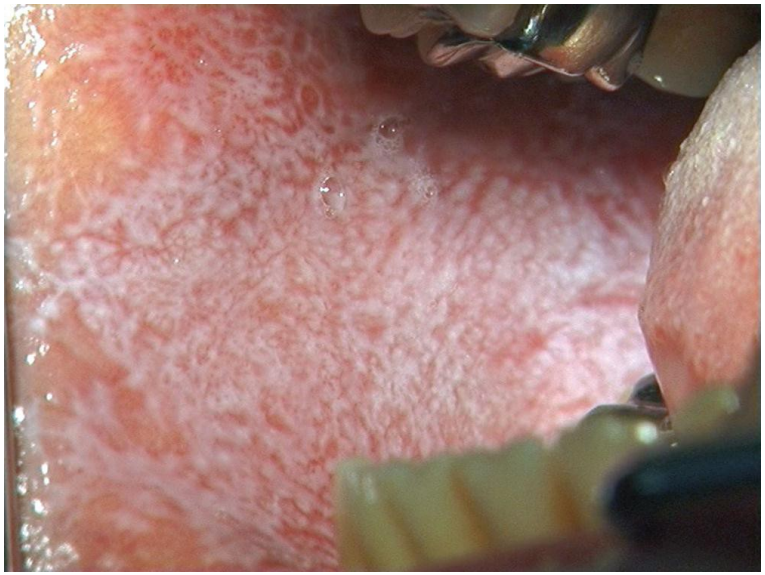
A mild degree of inflammation provoke the epithelium to produce hyperkeratosis, whereas more intense inflammation will lead to partial or complete deterioration of the epithelium.

- Typically, OLP is **bilateral** (symmetric) and can appear both white and red, depending on disease activity
- Hyperkeratotic white striations **Wickham's striae**, which are a hallmark of the condition.

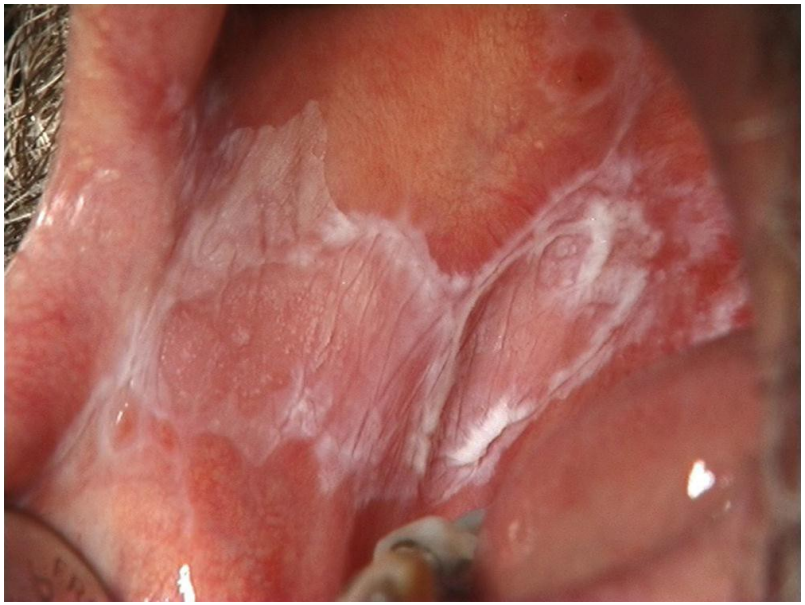
- Typically, the **reticular, papular, and plaque-like** are **asymptomatic**, although the patient may experience a **feeling of roughness**.
- The **bullous** form is very unusual but may appear as bullous structures surrounded by a reticular network.
- **Erythematous** (atrophic) OLP is characterized by a homogeneous red area. when this type of OLP is present in the buccal mucosa or in the palate, striae are frequently seen in the periphery of the lesion.
- **Ulcerative** lesions are the most disabling form of OLP. Clinically, the fibrin-coated **ulcers** are surrounded by an erythematous zone with white striae in the periphery.



A reticular



Papular



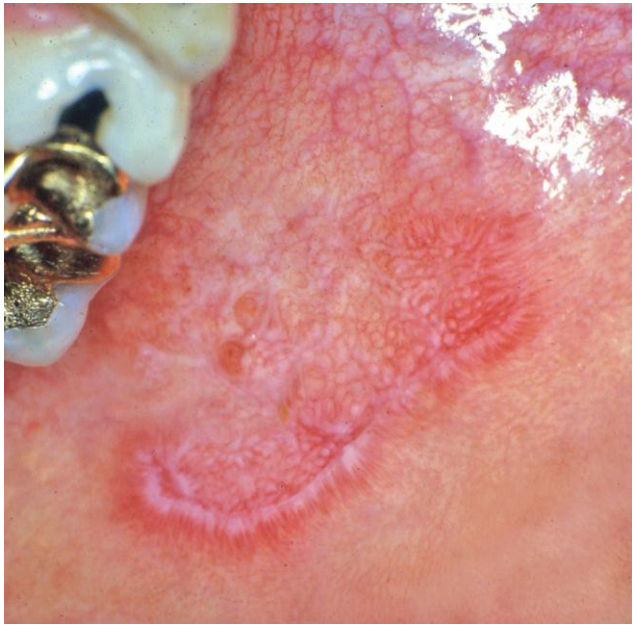
A plaque-like



Erythematous



Ulcerative



Discoid lupus erythematous



Cutaneous lichen planus on the flexor side of the fore arm.

Clinical Manifestation

- Cutaneous lesions may be seen in approximately **15%** of patients with OLP.
- The classic appearance of skin lesions consists of pruritic erythematous flat topped.

The predilection **sites** are the trunk and flexor surfaces of arms and legs

- The most frequent extra-oral mucosal site involved is the genital mucosa; 20% of women presenting with OLP also have genital involvement.
- Symptoms including burning, pain, vaginal discharge
- Following intense scratching of the lesions, **trauma** may aggravate the disease, which is referred to as a **Koebner phenomenon** (appearance of new skin lesions on previously unaffected skin secondary to trauma).

This phenomenon may also be of relevance for **OLP**, which is continuously exposed to physical trauma during mastication and brushing.

Lichenoid contact reactions to dental materials, which are most often detected on the buccal mucosa and the lateral borders of the tongue. OLP, on the other hand, usually displays a more general involvement.

Oral lichenoid drug eruptions

- Have the same clinical and histopathologic characteristics as OLP.
- The patient's disease history may give some indication as to which **drug** is involved, but lichenoid drug eruptions **may not start when the drug was first introduced**.
- **Withdrawal** of the drug **are the most reliable ways** to diagnose lichenoid drug eruptions
- An OLDE may not develop for several months after a new drug is started.
- It may also take **several weeks** before an OLDE disappears following withdrawal
- The delay between exclusion of the offending drug and regression of the lesion indicates that the molecule has sensitized the epithelium, which may persist regardless of drug withdrawal.

Oral GVHD has the same clinical appearance as OLP, but the lesion is usually more generalized.
The **lichenoid reactions** are frequently seen simultaneously with other characteristics, such as xerostomia and the presence of localized skin involvement and liver dysfunction.

Examples of drugs which have been associated with lichenoid reactions.

1. Angiotensin-converting enzyme inhibitors
2. Antimalarials
3. Barbiturates
4. Colchicine
5. Dapsone
6. Gold
7. Hydroxychloroquine
8. Metformin
9. Nonsteroidal anti-inflammatory drugs
10. Penicillamine
11. Phenothiazines
12. Phenytoin
13. Sulfonamide
14. Tetracyclines

Oral and genital lichen planus

- No relationship seems to exist between the degree of severity in the oral and genital sites.
- Genital has been reported in males, but the association with OLP is not as frequent as for women.
- Esophageal lichen planus has been described to occur simultaneously with OLP in some patients, the main complaint being **dysphagia**.

Diagnosis

- Papules or reticular components have to be present in order to establish a correct clinical diagnosis.
- These pathognomonic components may exist together with plaque-like, erythematous, bullous, or ulcerative lesions
- **In patients with gingival erythematous lesions**, it may be difficult to find striae or papules.
- A biopsy is usually required for an accurate diagnosis of this type of OLP.

- It is important that the biopsy is taken as far as possible from the **gingival pocket** to avoid inflammatory changes due to periodontal disease.

Oral mucosal lesions that do not belong to the group of lichenoid reactions may sometimes comprise a differential diagnostic problem

1. **Discoid lupus erythematosus (DLE)**

shows white radiating striae sometimes resembling those of **OLP**. The striae present in DLE are typically more prominent, with a more marked hyperkeratinization, and the striae may abruptly terminate against a sharp demarcation

- Histopathologic criteria for lupus erythematosus (LE) have been reported to discriminate against OLP

2. **Plaque-like OLP** is discriminated from **homogeneous oral leukoplakia** as

the latter is not featured with papular or reticular elements.

3.**Erythematous OLP of the gingiva exhibits** a similar clinical presentation as **mucous membrane pemphigoid**.
In pemphigoid lesions, the epithelium is easily detached from the connective tissue by a probe or a gentle searing force (Nikolsky's phenomenon).
A biopsy for routine histology and direct immunofluorescence are required for an accurate differential diagnosis.

4.**Ulcerating conditions** such as **erythema multiforme** and **adverse reactions to non-steroidalanti-inflammatory** drugs (NSAIDs) may be difficult to distinguish from **ulcerative OLP**.
The former lesions, however, do not typically appear with reticular or papular elements in the periphery of the ulcerations.

Management

Since the etiology behind OLP is unknown, basic conditions for development of preventive therapies are lacking.

Current therapies are directed against:

(1) Immune mechanisms using immunosuppressives;

(2) The cellular inflammatory response using anti-inflammatories; and

(3) Reducing or eliminating symptoms

- Careful oral hygiene to reduce biofilm-associated supplementary Inflamation is extremely important in OLP patients with symptoms.
- Several topical drugs have been suggested, including steroids, retinoids, and ultraviolet phototherapy.
- Among these, topical steroids are widely used and accepted as the **primary** treatment of choice very **potent steroids** as **clobetasol propionate** in favor of **intermediate steroids** such as **triamcinolon acetonide**.
- **Cyclosporine** may be considered a **second choice**, although the efficacy has been questioned.
- **Tacrolimus** should **only be used** by experts when symptomatic OLP lesions are recalcitrant to topical steroids.

Topical steroids

- Are preferably used as a mouth rinse or a gel.
- These formulas are often easier for the patient to administer than a paste.

A reasonable approach may be to apply the drug two to four times a day for one to two months, followed by tapering during the following eight weeks until a maintenance dose of two to three times a week is reached.

- When potent topical steroids are used, a fungal infection may emerge, and a parallel treatment with antifungal may be necessary.
- Since **half** of OLP lesions exhibit candidal infection, antifungal treatment may be used **before treatment with steroids.**

- However, no randomized clinical trials exist where different formulas, strengths, and classes of topically applied steroids have been compared

- For more widespread lesions, **betamethasone mouthwash** is more useful.
- In the case of widespread lesions, dexamethasone 0.5 mg/5 mL rinse can be applied three times a day for three minutes, gradually decreasing the number of applications following the improvement.

- **Adhesive pastes** consisting of pectin, gelatin, and carboxymethyl cellulose containing topical steroids (e.g., triamcinolone) have been formulated for use on moist oral mucosal surfaces.

- **Ointments** containing steroids can also be effective, but as they are hydrophobic they require drying of the mucosal surface before application.

Topical application of **cyclosporine and tacrolimus** has been suggested as a substitute topical therapy in OLP patients who develop candidiasis regardless of antimycotic therapy.

- Cyclosporine has been reported to be less effective than clobetasol propionate.
- No adverse effects related to these two drugs have been reported, except for a temporary burning sensation following the use of cyclosporine

Systemic Therapy

Although topical steroids are usually able to keep OLP patients free of symptoms, systemic steroids used to control symptoms from recalcitrant lesions.

- A dose of 0.5–1 mg/kg prednisolone daily for **seven** days has been suggested, followed by a reduction of 5 mg each subsequent day.
- Maintenance dose with topical steroids may be commenced during tapering of systemic steroids.
- However, before switching to systemic steroids, the use of peri- or intralesional steroids should be considered.
- Usually, 0.2–0.5 mL of 40 mg/mL methyl prednisolone or triamcinolone suspension is injected around and under the erosive lesion of OLP.

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Erythematous OLP of the gingiva

To be successful, it is critical to remove both **sub- and supragingival plaque and calculus**.

- If a **microbial plaque–induced gingivitis** make the lesion more resistant to pharmacologic treatment.
- Thus, oral hygiene should be optimized prior to the beginning of steroid treatment.

Once the hygiene treatment is complete, some patients experience a decrease in or even elimination of symptoms and steroid treatment is no longer justified.

- If symptoms persist, **steroid gels** in prefabricated **plastic trays** may be used for **30 minutes** at each application to increase the concentration of steroids in the gingival tissue.

Management

Drug-Induced Lichenoid Reactions

- ✓ Are not usually seen in conjunction with severe life-threatening reactions such as toxic epidermal necrolysis.
- ✓ Discontinuance of the drug and symptomatic treatment with topical steroids are often sufficient.
- ✓ The patient should be properly educated about the responsible drug to prevent future REACTION.

Lichenoid Reactions of GVHD

- ❑ The major cause of GVHD is **allogeneic hematopoietic cell**
- ❑ Oral lichenoid reactions as part of GVHD may be seen both in acute and **chronic** GVHD transplantation.
- ❑ Clinically are indistinguishable from OLP, that is, reticulum, erythema, and ulcerations, but lichenoid reactions associated with GVHD are typically associated with a **more widespread** involvement.
- ❑ It is not possible to distinguish between OLP and oral GVHD based on clinical and histopathologic features.

- ❑ The same treatment strategy as for OLP may be used for chronic oral GVHD, that is, topical steroid preparations, such as fluocinonide and clobetasol gel.
- ❑ Opportunistic infections such as candidiasis should always be considered in immunosuppressed patients.
- ❑ The development of **secondary malignancies** has been recognized as a potentially serious complication of GVHD.
- ❑ Patients with a history of oral GVHD should therefore be examined for oral malignancies as part of the medical follow-up

Malignant transformation of OLP

- A recent systematic review pooled 7806 OLP patients from 16 follow-up studies on the risk of malignant transformation showed a wide variations of **(0.03% to 1.3%).**
- Patients thus need to be realistically informed about a **small** but **increased risk** of developing oral carcinoma.
- **No** clear relationship with the clinical phenotype has emerged, and the site as well as the severity appears to be important OLP.

Vulvovaginal-Gingival Syndrome

- The most frequent extraoral mucosal site involved is the genital mucosa
- Close to **25%** of women presenting with OL also have genital involvement, though not always symptomatic.
- A specific presentation of gingival, vulval, and vaginal lichen planus with genital and oral (usually buccal mucosa) scarring and loss of sulcular depth known as **vulvovaginal-gingival (VVG) syndrome.**

- Equivalent in men **is peno-gingival syndrome**
- Symptoms in female patients include burning, pain, vaginal discharge and dyspareunia(genital pain that can be experienced before, during, or after intercourse) which are frequently noted, especially with the erythematous or ulcerative forms of the disease.

- No relationship seems to exist between the degree of severity in oral and genital sites.

Investigations

- **Routine Investigations**

The presence of **bilateral** white and red areas of mucosa, often with the typical clinical presentation of some reticular and/or ulcerative component, is diagnostic for OLP.

- **Biopsy** is needed for histopathologic confirmation. Biopsy is also advised if red lesions persist despite treatment. biopsy samples should be taken from the nonulcerated adjacent full-thickness epithelium, including basement membrane and subepithelial connective tissue, so that pathologic changes around the basement membrane can be identified.
- **Special Investigations**

Direct immunofluorescence (DIF), otherwise essential for vesiculobullous diseases is rarely performed In OLP, as it is nonspecific. However, DIF helps in differentiating OLP from MMP or PV in cases of desquamative gingivitis, or it can help differentiate OLP from lupus.

- Epicutaneous patch testing

can be useful for identifying causative allergens if OLCR is suspected, which can then be avoided.

Hematological investigations

Are sometimes performed in OLP **NOT** to make a diagnosis, but **TO SCREEN** for possibly associated systemic diseases.

- There are reports of potentially relevant associations between OLP thyroid disease and also hepatitis B or C virus infection.
- Therefore, liver function tests—aspartate transaminase (AST), alanine transaminase (ALT), and gamma-glutamyl-transpeptidase (GGT)—and thyroid hormones are sometimes performed in OLP patients.

- **Photographic records** of lesions can be very helpful to assist in the determination of any response to therapy and can become part of the evidence of clinical outcomes.

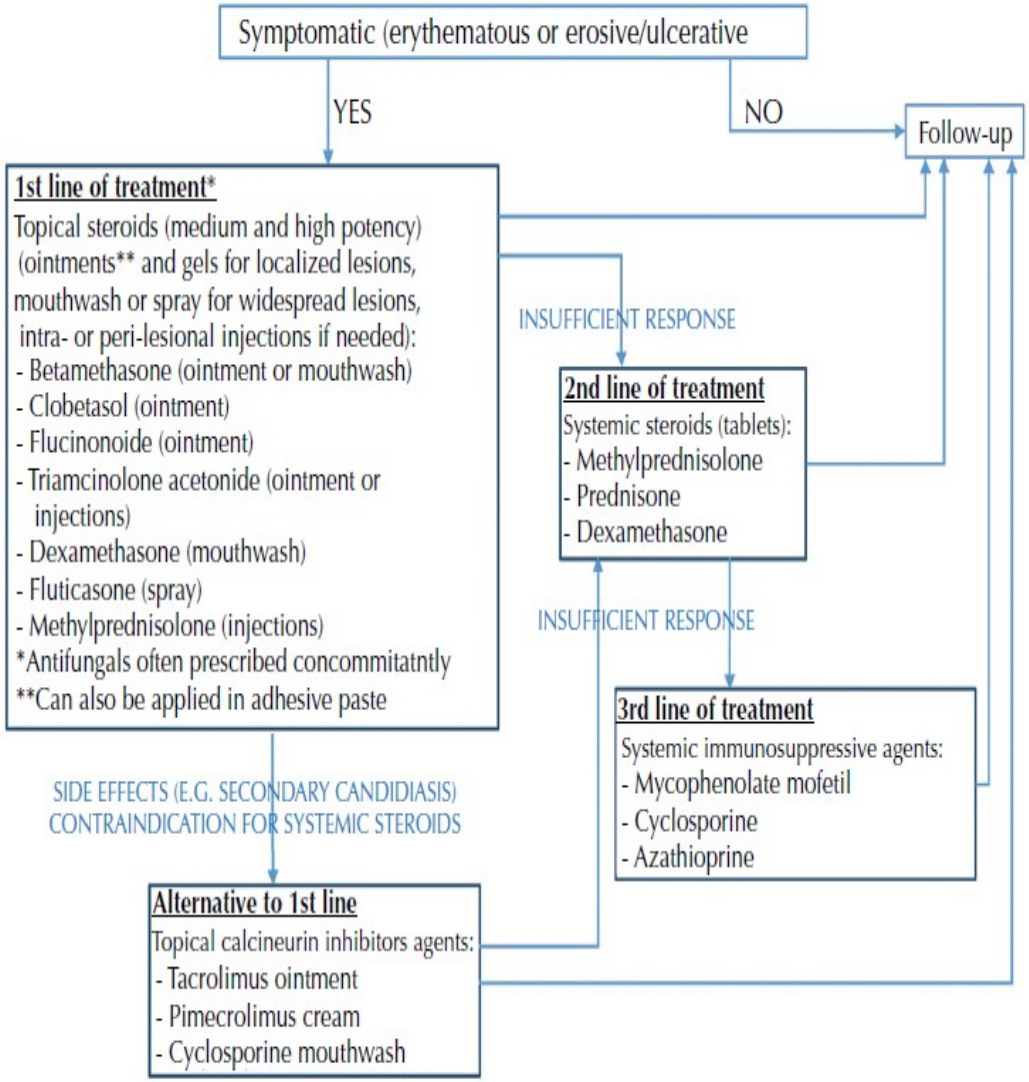


Figure 4-43 Algorithm for the management of oral lichen planus. Treatment is dependent on symptoms, the oral disease severity score, and previous response to therapy.

- The **oral lesions** observed in SLE and discoid lupus erythematosus are similar in their characteristics, both clinically & histopathologically.

- The typical clinical lesion comprises white striae with a radiating orientation, and these may sharply terminate toward the center of the lesions, which has a more erythematous appearance.
- The most affected sites are the hard palate, buccal mucosa, and gingiva.
- The tongue also can be involved, lesions in the palatal mucosa can be dominated by erythematous lesions and white structures may not be observed.
- The typical oral DLE lesion is a well-demarcated lesion with a mixed center and with a brush border of fine striae around the lesion. They are usually asymmetric or scattered, in contrast to OLP.
- **Oral mucosa lesions compatible with LE may be the first sign of the disease.**
- The classic categorization of LE into SLE and DLE has during recent years been supplemented with acute cutaneous lupus erythematosus and subacute cutaneous lupus erythematosus.

Diagnosis

A SLE diagnosis requires that **Four or more** of the **diagnostic criteria** should be present at each time point of the disease.
The typical DLE diagnosis comprises well-demarcated cutaneous lesions with round or oval erythematous plaques with scales and follicular plugging.
These lesions may form butterfly-like rashes over the cheeks and nose, known as a **malar rash**.

American college of rheumatology criteria for SLE

1 Malar rash
2 Discoid lesions
3 Photosensitivity
4 Presence of oral ulcers
5Non-erosive arthritis of two joints or more
6 Serositis
7 Renal disorder
8 Neurologic disorder (seizures or psychosis)
9Hematologic disorder (hemolytic anemia, leukopenia, lymphopenia, or thrombocytopenia)
10.Immunologic disorder (anti-DNA, anti-Smith, or. antiphospholipid antibodies).

Management

- ✓ The oral lesions may respond to systematic treatment used to for the disease
- ✓ When symptomatic intraoral lesions are present, topical steroids should be considered
- ✓ The treatment may begin with applications two to three times a day followed by a tapering during the next six to nine weeks
- ✓ such as **clobetasol** propionate gel 0.05%, **betamethasone** dipropionate 0.05%, or **fluticasone** propionate spray 50 µg aqueous solution are usually required
- ✓ Opportunistic oral infections can also originate the immunologic defects, which are part of the pathogenesis.
- ✓ Another complication of the drugs used in treatment of LE is mucosal ulceration caused by frequent exploitation of NSAIDs.

Oral mucosal lesions often mirror the disease activity.
They may regress spontaneously but can also persist for months or even years.

The overall objective is to use a minimum of steroids to obtain relief

Clinical Findings

- Oral Lichenoid Contact Reactions are considered to be a type of delayed hypersensitivity reaction to constituents derived from dental predominantly amalgam fillingsmaterials,

- Clinically, LCRs display the same reaction patterns as seen in OLP, that is, reticulum, papules, plaque, erythema, and ulcers.

- The most apparent clinical difference between OLP and LCR is the **extension of the lesions**.
- The majority of LCRs are confined to sites that are regularly in contact with dental materials, such as the **buccal mucosa** and the **border of the tongue**
- The majority of this type of LCR resolve following treatment with chlorhexidine