Lec. 17 ***Dr. Ali H. Murad***

WHITE LESIONS OF ORAL MUCOSA

These are groups of lesions which characterized clinically by being white or having some component that is white.

White lesions are visibly striking because they standout against the normal pink red mucosa, more over they are important because a white lesion may be premalignant (increased risk of malignant transformation) or malignant.

The color of normal oral mucosa depends on the amount of blood vessels in the underlying CT, the covering epithelium and the surface keratin. When the covering epithelium and the keratin layer is increased in thickness, this will mask the red color of the underlying CT. Therefore, the oral mucosa will appear as a white lesion in that area.

White lesions of oral mucus membrane appear thus because

(1) One or more of the epithelial layer is thickened or

(2) An extrinsic or intrinsic pseudo-membrane is adherent to the surface mucosa.

In general, white lesions are color changes in the mucous membrane oral cavity due to many diseases. varying from simple, moderates to severe.

In light skinned individuals, the oral mucous membrane normally exhibits a uniform pink coloration.

Individuals who are dark-skinned will often exhibit some pigmentation of the oral mucous membrane, such intra oral pigmentation has been termed physiologic or racial pigmentation and conspicuous on the labial gingiva but is also observed on the buccal and labial mucosa.

Multiple local and systemic factors can affect the oral epithelium and its normal clinical and microscopical appearances.

local physical irritants such as tobacco smoke act on the thin, normally non-keratinized sq. epithelium to induce excessive thickening of the keratin and or spinous cell layer. Additionally, constituents of tobacco smoke can stimulate the melanocytes to induce both local and generalized pigmentation of the oral mucosa. In older patients, there are many oral color changes because they chew on the mucosa.

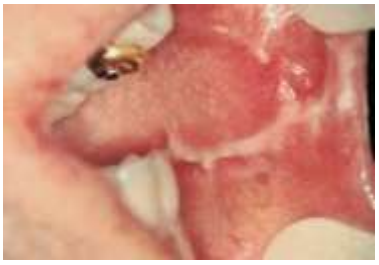
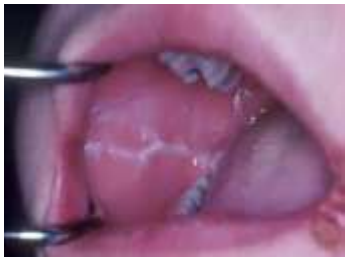
The patient that has bruxism may have white line near the occlusal plane; this is not serious condition due to continuous check biting.

Linen Alba

Definition: Linen Alba is relatively common alteration of the buccal mucosa.

Etiology: Pressure, sucking from the buccal surface of the teeth.

Clinical features: It presents as an asymptomatic, bilateral, linear elevation with a slightly whitish color at the level of the occlusal line of the teeth.

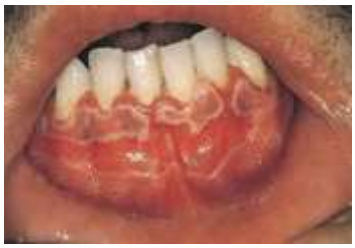
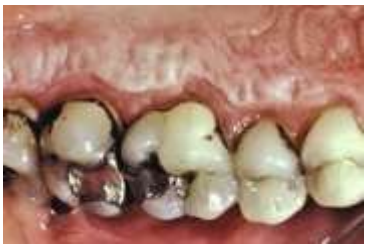
It has a normal consistency on palpation. The diagnosis is based on clinical grounds alone.

Treatment: No treatment is required.

Materia Alba of the gingiva

Definition and etiology: Materia Alba results from the accumulation of food debris, dead epithelial cells, and bacteria. It is common at the dentogingival margin. Rarely, materia Alba may be seen along the vestibular surface of the attached gingiva in patients with poor oral hygiene.

Clinical features: It presents as a soft, whitish plaque that is easily detached after slight pressure.

Differential diagnosis:

Candidiasis, chemical burn, leukoplakia.

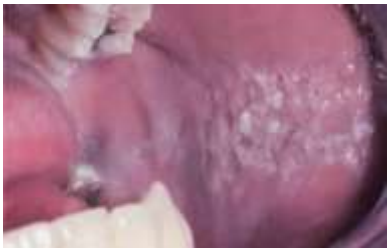
Treatment: Good oral hygiene.

Chronic Biting

Definition and etiology:

Mild chronic biting of the oral mucosa is relatively common in nervous individuals. These patients consciously bite the buccal mucosa, lips, and tongue, and detach the superficial epithelial layers.

Clinical features: The lesions are characterized by a diffuse irregular white area of small furrows and desquamation of the epithelium. Rarely, erosions and petechia may be seen.



The diagnosis is made clinically.

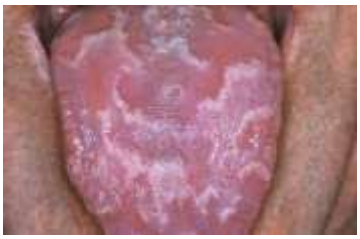
Differential diagnosis: Candidiasis, lichen planus, leukoplakia, hairy leukoplakia, white sponge nevus, leukoedema.

Treatment: Recommendation to stop the habit.

Geographic Tongue

Definition: Geographic tongue, or erythema migrans, is a relatively common benign condition, primarily affecting the tongue and rarely other oral mucosa sites (geographic stomatitis).

Etiology The exact etiology remains unknown. It may be genetic.



Uremic Stomatitis

Definition: Uremic stomatitis is a rare disorder that may occur in patients with acute or chronic renal failure.

Etiology: Increased concentration of urea and its products in the blood and saliva. The pathogenesis of oral lesions is not clear. It usually appears when blood concentration of urea exceeds 30 mmol/L.

The degradation of oral urea by the enzyme urease forms free ammonia, which may damage the oral mucosa.

Clinical features: Four forms of uremic stomatitis are recognized:

(a) The ulcerative form,

(b) The hemorrhagic form,

(c) The nonulcerative, pseudomembranous form

(d) The hyperkeratotic form.

The last two forms appear as white lesions.

The hyperkeratotic form presents as multiple painful white hyperkeratotic lesions with thin projections

The tongue, and the floor of the mouth are more frequently affected. Xerostomia, uriniferous breath odor, unpleasant taste, and burning sensation are common symptoms.

Candidiasis, viral and bacterial infections are common oral complications.

The diagnosis is based on the history, the clinical features, and urinalysis and blood urea level determination.

Differential diagnosis: Candidiasis, cinnamon contact stomatitis, hairy leukoplakia, white sponge nevus, drug reactions.

Treatment: The oral lesions usually improve after hemodialysis. A high level of oral hygiene, mouthwashes with oxygen release agents, and artificial saliva are suggested. Antimycotic, antiviral, and antimicrobial agents if necessary.

Cinnamon Contact Stomatitis

Definition: Cinnamon contact stomatitis is a relatively common oral mucosal reaction to continuous contact of substances with cinnamon.

Etiology: Artificial cinnamon flavoring especially in the form of chewing gum, candies, toothpaste, drops, etc.

Clinical features:

The condition is characterized by erythema of the oral mucosa, usually in association with desquamation and erosions.

The white lesions may be subdivided in to group on the basis of pathogenesis or etiological factors:

1. Developmental white lesions: are generally bilateral in distribution and most of these white lesions are genetically inherited e.g.: Leukoedema, White spongy nevus.

2. White lesions associated with physical or tobacco product irritation are grouped under the heading leukoplakia.

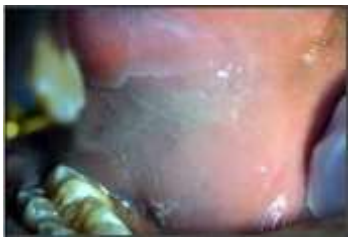
3. White lesions of nonhereditary disease may coexist with skin lesions. e.g.: Lichen planus, Lupus erythematosus.

4. Inflammatory lesions that may produce thickened epithelium and or a surface pseudomembrane will appear white and in these instances the surface coating may be rubbed a way with gauze e.g.: Candidacies, Koplik spots of measles, Mucous patches of syphilis, Chemical burns, and miscellaneous white spots and papules e.g. Fordyce's granules.

***LEUKOEDEMA*:**

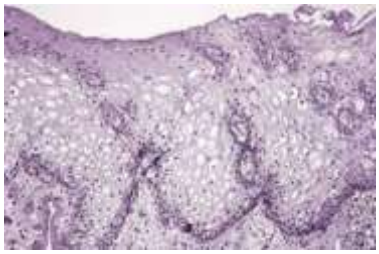
Is a common oral mucosal condition of unknown cause, it occurs more commonly in blacks than in white persons. Ethnic or racial groups are affected and is best regarded as a variation of normal.

Clinically characterized by a diffuse, grayish, white milky appearance of the mucosa, the surfaces frequently appear folded, resulting in wrinkles or whitish streaks. The lesions don't rub off. It occurs bilaterally on the labial mucosa.

Histologically: Biopsy specimens of leukoedema demonstrate an increase in thickness of the epithelium with intra cellular edema of the spinous layer and appear

vacuolated and contain considerable quantities of glycogen. These vacuolated cells appear large and have pyknotic nuclei, the epithelium surface is frequently Para keratinized and the rete ridges are broad and elongated.



Leukoedema is a benign condition and no treatment is required, leukoedema can be easily diagnosed clinically because the white appearance disappear when the cheek is everted and stretched and this help us to distinguish it from other white lesions.

WHITE SPONGE NEVUS: (ORAL EPITHELIAL NAEVUS)

It is a genetically determined skin disorder that is inherited as an autosomal dominant trait. Patients usually have the lesion at birth or in early childhood.

Clinically

1. Any part of the oral mucosa may be involved, the edge of the lesion is not well defined but gradually merge with normal mucosa.

2. Other mucosal surfaces (nasal, pharyngeal and anogenital) may be affected.

3. The condition is not painful

4. Other family members often have the condition.

5. The clinical features and history are diagnostic.

Histologically, the epithelium shows parakeratosis and acanthosis with spongiosis. Individual cell keratinization may be seen in the spinous cell layer.

The condition is benign and require no treatment.

TRAUMATIC KERATOSIS:

A- Mechanical:

Friction to oral mucosa lead to two types of lesion.

1. In acute frictional response ulceration will occur.

2. In chronic frictional response epithelial thickening (hyperplasia) and hyperkeratinization will occur, i.e. a white lesion.

Sharp teeth, cheek biting, prolonged wearing of ill-fitting dentures are examples of causes of chronic friction.

The lesions produced are known as frictional keratosis (focal keratosis) which in time become white, to diagnose frictional keratosis the source of chronic irritation must be identified which fits the size and shape of the lesion and the lesion must resolve (disappear) when the source of irritation is removed.

Histologically, frictional keratosis show hyperkeratinization which may be accompanied by some acanthosis but there is no dysplasia.

B- Chemical: Chemical Burn:

Definition: This is an injury to the oral mucosa caused by topical application of caustic agents.

Etiology: Caustic agents include aspirin, hydrogen peroxide, phenol, alcohol, silver nitrate, trichloro acetic acid, acid etching liquid, and varnishes of tooth cavities.

chemical materials may produce a variety of reaction in the oral mucosa. A severe form of chemical trauma such as that produced by topical use of aspirin (aspirin burn) is likely to produce epithelial necrosis, sloughing and ulceration. While a low grade chronic chemical trauma result in hyperkeratosis.

Tobacco habits generally (smoking, chewing snuff and betel-nut) are considered as chronic chemical irritation. These habits produce epithelial thickening (hyperplasia and hyperkeratosis).

The diagnosis should be made on the basis of the clinical features and history.

Differential diagnosis: Necrotizing ulcerative gingivitis and stomatitis, materia alba, candidiasis, mechanical trauma, bullous diseases.

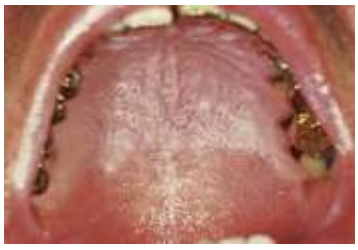
Treatment: Treatment is symptomatic.

C: Thermal:

white lesions in the anterior parts of the cheek, tongue and palate may be seen in regular smokers of cigarettes, cigars and pipes. Thermal and chemical effect of the smoke are involved in the development of hyperkeratinization.

Local keratosis at the same site of the source of smoke cigarettes and cigars (lip) and in pipe smokers, the lesion is seen on dorsum of the tongue and palate.

Nicotinic stomatitis of the palate is a clinical condition which may develop in association with any type of smoking, but particularly in pipe smokers.

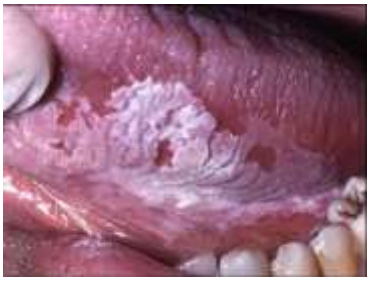
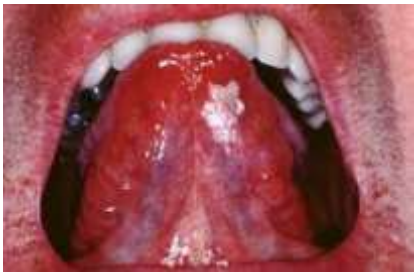


The lesion appears as grayish-white with scattered red spots (representing the orifices of minor salivary glands).

Histologically, nicotinic stomatitis shows hyperkeratosis and acanthosis of palatal epithelium. The condition is usually reversible if smoking is stopped.

***LEUKOPLAKIA*:**

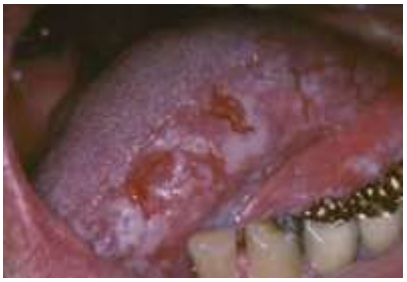
It is a clinical term define by W.H.O. as a white patch on the oral mucosa that cannot be scraped off and that cannot be classified as any other diagnosable diseases. A small percentage of leukoplakia are premalignant and some may be invasive sq.cell.ca at the time of presentation. About 5% of oral sq.cell.ca appear clinically as white lesions(leukoplakia).

It is impossible to predict which lesions are likely to become malignant, but certain clinical presentation may be associated with premalignancy such as:

I. Sublingual keratosis i.e. white lesion in the floor of the mouth and ventral surface of the tongue.

2. Speckled leukoplakia, i.e. white keratotic lesion with red patches or cracks.



Etiology: The etiology is varied and many of the causative factors are the same as for carcinoma. They include:

1. Tobacco: The habit of tobacco smoking appears most closely associated with leukoplakia development more than 80% of patients with leukoplakia are smokers, heavy smokers have greater numbers of lesions and larger lesions than do light smokers.

2. Alcohol.

3. Chronic irritation such as mal occlusion, bad prosthesis and sharp tooth.

4. Galvanism caused by metal dental restoration such as gold and amalgam.

5. Syphilis.

6. Vit deficiency especially Vit A and Vit B complex.

7. Hormones especially sex hormones.

8. Candidiasis, many biopsy specimen of leukoplakia show presence of candida albicans, but the relationship between the two is still uncertain.

Some investigators have sub classified oral leukoplakia in to:-

1. Frictional keratosis.

2. Smokers keratosis.

3. Galvanic lesion

4. Leukoplakia in association with candida.

5. Idiopathic leukoplakia.

Clinically leukoplakia has variable presentation from a small well circumscribed plaque to an extensive lesion involving a large area of the oral mucosa. Lesions may be white, yellowish white or grayish white. Lesions may have a homogenous or non-homogeneous surface. The surface of leukoplakia may be smooth or rough, some time it may be wrinkled or the surface may be crises-crossed by small cracks or fissures.

Non-homogenous leukoplakia may show areas of redness, producing a speckled appearance. In some cases, the lesion may take a prominent warty appearance described as verrucous leukoplakia.

Non-homogenous types of leukoplakia are more likely to be associated with epithelial dysplasia.

At present W.H.O. classify oral leukoplakia in to

(a) homogenous type

(b) non-homogenous type.

Using the W.H.O. system the non-homogenous type may be further sub divided in to

(1) erythro-leukoplakia

(2) erosive leukoplakia

(3) nodular leukoplakia

(4) verrucous leukoplakia.

Erythroplakia: is a bright red velvety plaque on the oral mucosa which cannot be categorized clinically or pathologically as being due to any other condition.

Erythroplakia may be homogenous represent severe epithelial dysplastic carcinoma in situ or even invasive sq. cell. carcinoma.

Histopathology:

The histologic aspect of leukoplakia may vary from hyperkeratosis either with or without epithelial dysplasia, to carcinoma in situ and even sq.cell.ca.

The histopathological alteration of dysplastic epithelial cell is similar to those of sq.cell.ca and include the following:

1. Enlarged nuclei and cell.

2. Large and prominent nucleoli.

3. Increased nuclear cytoplasmic ratio.

4. Hyperchromatic nuclei (dark staining).

5. Pleomorphic nuclei and cells (abnormally shaped).

6. Dyskeratosis (premature keratinization of individual cell).

7. Increased mitotic activity (excessive number of mitosis)

8. Abnormal mitotic figures (tripolar or star- shaped mitosis or mitotic above the basal layer).

In addition, histomorphologic alteration of the epithelium may be seen as:

1. Bulbous or teardrop shaped rete ridges.

2. Loss of polarity (lack of progressive maturation to word the surface).

3. Epithelial pearls.

4. Loss of a typical cellular cohesiveness.

When all these changes are present in all the layer of the epithelium then this is known as carcinoma in situ.

If only few changes are seen in a case. then this is known as mild dysplasia, more changes in the epithelium is called moderate or severe epithelial dysplasia.

When dysplastic cells penetrate the basement membrane and appear within the C.T., then the case is sq.cell.ca.

Treatment:

Generally speaking, the treatment of the disease is aimed at the elimination of any recognizable irritating factors such as smoking, alcohol, correction of mal occlusion etc.

Sometime the lesions regress, if not surgical removal must be done by total excision of the lesion, if small.

For large diffuse leukoplakia cryosurgery or treatment with laser beams can be good alternative to routine surgical removal.

Prognosis of leukoplakia:

A proportion undergo malignant transformation. Transformation times may vary from one to several years.

Dysplastic lesions (i.e. leukoplakia with epithelial dysplasia) carry an increased risk of malignant transformation.

The potential for malignant transformation is greater in high risk sites (sub lingual).

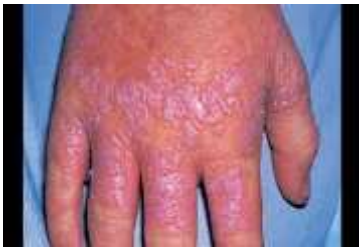
Dermatological causes of white lesions:

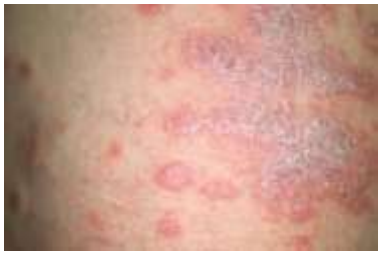
Lichen planus:

is a chronic inflammatory mucocutaneous disease of unknown etiology in which oral lesions either accompany, precede or follow skin lesions. It may also affect both sexes.

Skin lesions may appear before mucosal lesions or some time follows them. The typical lesions are found on the upper trunk, the flexor surface of the arm and legs. The characteristic clinical features of skin lesions are the appearance of purple,

polygonal, pruritic papules which may have whitish striae (Wickhams striae) on their surface. These lesions could be seen in any area of the skin, but the commonest area is the flexor surface of the wrist.

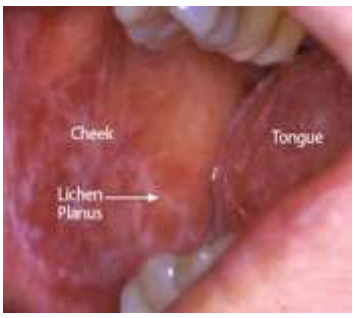


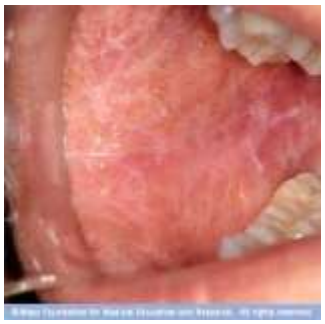


The skin lesion develops slowly and usually resolve within 18 months although recurrence may occur.

Finger nails are occasionally involved showing vertical ridges.

In contrast, oral lesions are much more chronic, in some patient's oral lesion may extend several years. The most common appearance of oral LP is a network of white lines in the buccal mucosa, bilaterally and posteriorly, this network appearance has been termed Wickhams striae.





A wide spectrum of clinical presentation may occur alone or in combination, these are:

Reticular.

Atrophic

Plaque-like

Papular

Erosive (ulcerative).

Bullous.

Reticular, plaque-like and popular patterns are usually symptom free.

Atrophic, erosive types have a red and glazed appearance with areas of superficial ulceration. These lesions are painful and cause discomfort to the patient. If the erosive component is severe, epithelial separation may occur, this result in the relatively rare presentation of bullous LP.

LP on the gingiva often presents as a desqumative gingivitis.

Etiology: Although the cause of LP is unknown, it is generally considered to be an immunologically mediated process that microscopically resembles a hypersensitivity reaction. It is characterized by an intense T-cell infiltrate (CD4 and especially CD8 cells) localized to the epithelium-connective tissue interface.

The severity of the disease commonly parallels the patient's level of stress. An association between LP and hepatitis C infection has been suggested. There appears to be no relationship between LP and either hypertension or diabetes mellitus, as previously proposed. Many of these cases likely represent lichenoid drug reactions to the medications used to manage conditions, which may mimic LP clinically.

Histopathology: The microscopic picture is characteristic

1. Hyper parakeratosis or orthokeratosis.

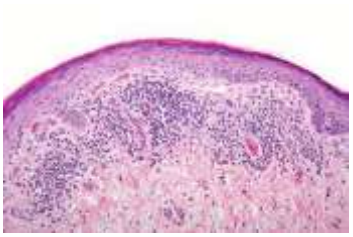
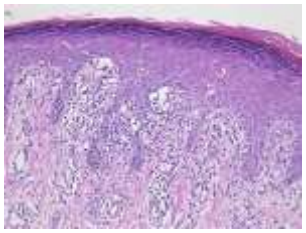
2. Thickening of the granular layer.

3. Acanthosis with intercellular edema of the spinous cells.

4. Saw tooth appearance of the rete ridges.

5. Liquefactive or necrosis of the basal cell layer.

6. Infiltration of lymphocyte in the sub-epithelial connective tissue, consisting mainly of T lymphocytes.


Immunofluorescence test show a positive fluorescence that outlines the basement membrane zone after reaction with anti-fibrinogen.

Treatment and prognosis:

There is no specific treatment for LP although corticosteroid has found to be helpful in reducing pain especially in the erosive type. There is a possibility of malignant change especially of the erosive type of LP. because LP is a chronic condition, patients should be observed periodically and should be offered education about the clinical course, rationale of therapy, and possible risk of malignant transformation.

***Lupus erythematosus*:**

LE presented as two main forms:

1. Chronic discoid LE. (chronic)

2. Systemic LE. (acute)

Both of which have oral manifestation.

A third form known as subacute LE.

Chronic discoid LE is a localized disease, less aggressive form, predominantly affecting the skin and rarely progressing to the systemic form. It may, however, be of great cosmetic significance because of its predilection for the face.

Systemic LE is a disseminated disease involving almost every organ of the body.

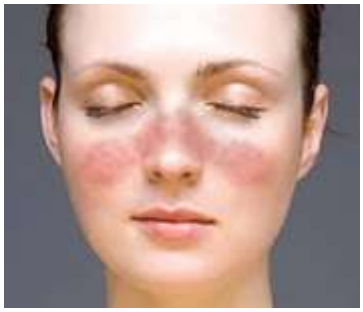
Subacute cutaneous LE, described as lying intermediate between SLE, and DLE, result in skin lesions of mild to moderate severity.

Etiology:

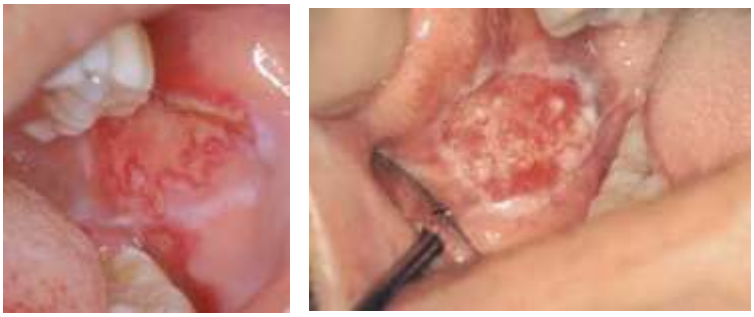
It is believed to be an autoimmune disease involving both the humoral and the cell mediated arms of the immune system.

Clinical features:

DLE: is characteristically seen in middle age, especially in women. Lesions commonly appear solely on the skin, most commonly on the face and scalp. Oral and vermilion lesions are also commonly seen, but usually in the company of cutaneous lesions. Skin lesions appear as scaly red patches (heal with scar). Sometimes these lesions have a symmetrical distribution over the nose and cheek, the so-called butterfly pattern.



Oral lesions, seen in about 25% of patients with cutaneous DLE. The buccal mucosa, gingiva, and vermilion are mostly affected. The most usual oral picture is a discoid area of erythema or ulceration surrounded by a white keratotic border sometimes with radiating striae similar to LP.



SLE: in SLE skin and mucosal lesions are relatively mild, and patient's complaints are dominated by multiple organ involvement. Systemic symptom of SLE may initially consist of fever, weight loss, and malaise. Oral lesions of SLE are generally similar to those seen in DLE.

The lesions of SLE include skin rashes, lymphadenopathy, also kidneys, liver, lung and nervous system are involved.

Histopathology:

The oral lesion show ortho or parakeratinized with areas of epithelial hyperplasia and atrophy. Subepithelial and deeply situated perivascular foci of lymphocytes are present in the C.T. and there may be liquefactive degeneration of basal cells similar to LP.

Treatment:

DLE is usually treated with topical corticosteroids.

Systemic steroid may be used in the treatment of SLE.