بسم الله الرحمن الرحيم
Normal oral mucosa
Pale colour of normal mucosa

It results from an interplay between four factors:

1. Vascularity
2. Epithelium thickness
3. Melanin pigment
4. Keratinization
White lesions
White lesions

**Def.**

White-appearing lesions of the oral mucosa, obtained their characteristic appearance from the scattering of light through an altered surface, e.g. such alterations may be the result of a thickened layer of keratin that may be due to:

1. Chronic physical trauma.
3. Tobacco use.
4. Inflammatory reactions.
5. Genetic abnormalities.
Colour of White lesions results from

1. Hyperkeratosis (thickened layer of keratin)
2. Acanthosis (epithelial hyperplasia as the thickened spinous layers masks the normal vascularity (redness).
3. Intracellular epithelial edema
4. Reduced vascularity of subjacent connective tissue
5. Fibrous exudate covering an:
   - ulcer
   - fungal colonies
   - Submucosal deposits
   - surface debris
Classification of white lesions

1. Hereditary
   - White sponge nevus
   - Leukodema
   - Follicular keratosis
   - Hereditary benign intraepithelial dyskeratosis

2. Reactive
   - Nictinic stomatitis
   - Hairy tongue
   - Hairy leukoplakia

3. Preneoplastic and neoplastic
   - Leukoplakia
   - Candidal leukoplakia
   - Squamous cell carcinoma
   - Actinic cheilitis

4. Other white lesions
   - Lichen planus
   - Lupus erythematosus
   - Geographic tongue

5. Non epithelial
   - Candidiasis
   - Mucosal burns
   - Oral submucous fibrosis
   - Foerdyce's granules
   - Parulis
White lesions

Classification

a. Hereditary conditions
   - Leukoedema
   - White spongy nevus
   - Hereditary benign intraepithelial dyskeratosis
   - Follicular keratosis

b. Reactive lesions
   - Focal (fractional) hyperkeratosis
   - White lesions associated with smokeless tobacco
   - Nicotine stomatitis
   - Hairy leukoplakia

c. Preneoplastic and neoplastic lesions
   - Actinic Chelitis
   - Idiopathic leukoplakia

d. Other white lesions
   - Geographic tongue
   - Lichen planus
   - Lupus erythematosus

e. Non-epithelia white yellow lesions
   - Candidiasis
   - Mucosal burns
   - Submucous fibrosis
   - Fordyce's granules
   - Ectopic lymphoid tissue
   - Gingival cyst
   - Parulis
   - Lipoma
Hereditary conditions

1. Leukoedema
2. White spongy nevus
3. Hereditary benign intraepithelial dyskeratosis
4. Follicular keratosis
Leukoedema
Leukoedema

Def.

Accumulation of fluid within the epithelial cells of the buccal mucosa

Etiology

unknown
Leukoedema

Clinical features
Site: buccal mucosa
Age: childhood
S&S:
1. Asymptomatic.
2. Bilateral in buccal mucosa.
3. It is a gray-white, diffuse
4. Filmy or milky surface.

With stretching of buccal mucosa, opaque changes will dissipate, except in more advanced cases.
Leukoedema

*Histopathologic features*

1. Parakeratosis
2. Acanthosis
3. Marked intracellular edema of spinous cells.
4. Small pyknotic nuclei with clear cytoplasm.
Hydropic Change - Leukoedema

Oral epithelium - microscopic

Source: TUSDM

(c) 2017, Michael A. Kahn, DDS
Leukoedema

Differential diagnosis
1. White spongy nevus
2. leukoplakia,
3. hereditary benign intraepithelial dyskeratosis.
4. Response to chronic cheek biting.

Overall thickness of these lesions. Their persistence upon stretching, and specific microscopic features help separate them from leukoedema.

Treatment and prognosis
No treatment is essential and no malignant potential
White spongy nevus
**White spongy nevus**

*Def.*

It is an autosomal dominant transmitted condition that is often mistaken for leukoplakia.
White spongy nevus

Clinical features

Site: buccal mucosa, tongue, conjunctival mucosa, vaginal valva and esophageal mucosa.

Age: Appears early in life, before puberty.

S&S:
1. Bilateral
2. Asymptomatic
3. Deeply folded white or gray.
4. Spongy in consistency.
White spongy nevus

**Histopathologic features**

- Thick epithelium (acanthosis).
- Parakeratosis.
- Within stratum spinosum, marked hydropic or clear cell change.
- Pyknotic nuclei and eccentric in location.
- This oedematous cells giving (basket weavy appearance).
White spongy nevus
White spongy nevus

An eosinophilic condensation may be noted under (E/M) in the perinuclear of the cells of superficial layers of the epithelium, this feature can be known as *keratin tonofilaments*.
White spongy nevus

Differential diagnosis
1. Hereditary benign intraepithelial dyskeratosis
2. Hypertrophic lichen planus
3. Frictional keratosis
5. Leukoedema

Treatment
No treatment since it is a benign asymptomatic condition.
Hereditary benign intraepithelial dyskeratosis (Hbid) (Witkop's disease)
**Hereditary benign intraepithelial dyskeratosis**

**Def.**

It is actually a syndrome

**Etiology**

Hereditary autosomal dominant transmitted condition.
Hereditary benign intraepithelial dyskeratosis

Clinical features
Age: Early in life(within the first year).

S&S: 1. Asymptomatic condition
   2. White
   3. Folded plaques of spongy mucosa.

Site: Buccal and labial mucosa,
      labial commissures, floor of the mouth,
      lateral surface of the tongue, gingival
      and papillae.
Hereditary beginn intraepithelial dyskeratosis

Histopathologic features
1. Acanthosis.
2. Hydropic degeneration of spinous cells.
3. Enlarged, hyline, waxy eosinophilic cells (which is the dyskeratotic elements).
4. Dyskeratotic cells may be surrounded by adjacent cells producing (cell within cell).
5. Inflammatory cell infiltrate is minimal.
Hereditary benign intraepithelial dyskeratosis
Hereditary benign intraepithelial dyskeratosis

Differential diagnosis

1. White spongy nevus
2. hypertrophic lichen planus
3. frictional keratosis.
Follicular keratosis
(Darier's Disease)
<table>
<thead>
<tr>
<th>Parameters</th>
<th>Etiology</th>
<th>Clinical picture</th>
<th>features Histopathologic</th>
<th>Differential diagnosis</th>
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</thead>
<tbody>
<tr>
<td><strong>Follicular keratosis</strong></td>
<td>Autosomal, dominant mode of inheritance.</td>
<td>1. Oral mucosal sites include keratinized regions such as: attached gingiva, hard palate.</td>
<td>.1. Vertical clefts 2. acantholytic epithelial cells. Microscopically, these may be represented under the term &quot;warty dyskeratonia&quot;.</td>
<td></td>
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<tr>
<td>(Darier's Disease)</td>
<td>• 50% of.</td>
<td>2. Lesions may extend to oropharynx and pharynx.</td>
<td>3. Skin (symmetrically distributed over the face and trunk).</td>
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<tr>
<td></td>
<td></td>
<td>3. Skin (symmetrically distributed over the face and trunk).</td>
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<tr>
<td>Site</td>
<td>Childhood or adolescence.</td>
<td></td>
<td></td>
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<tr>
<td>Age</td>
<td>Thickening of the skin of palms and soles.</td>
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<tr>
<td>S&amp;S</td>
<td>• Papular lesions on the skin.</td>
<td></td>
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<td></td>
<td>• Localized lesions may follow sunburns, especially on the legs.</td>
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<td></td>
<td>• Oral lesions are in the form of papules</td>
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Reactive lesions
Reactive lesions

1. Focal (fractional) hyperkeratosis
2. White lesions associated with smokeless tobacco
3. Nicotine stomatitis
4. Hairy leukoplakia
5. Hairy tongue
Focal (frictional) hyperkeratosis
Focal (frictional) hyperkeratosis

Def.

It is a white lesion that is often classified under the general term "leukoplakia".

The tissue response represents a protective action against low-grade, long-term trauma.
Focal (frictional) hyperkeratosis

**Etiology**

1. Self-evident

2. Chronic rubbing or friction of an oral mucosal surface

**Clinical features**

*Site:*

areas that are commonly traumatized such as lips, buccal mucosa along occlusal line, & edentulous ridges

*S&S:*

Friction-induced hyperparakeratosis or leukoplakia

**Histopathologic features**

1. Hyperkeratosis

2. chronic inflammatory cells on connective tissue.
White lesions associated with smokeless tobacco
White lesions associated with smokeless tobacco

** Causes **

Chewing tobacco or snuff

** Clinical features **

alterations of oral cavity

** Histopathology **

1. hyperkeratosis
2. chronic inflammatory cells on connective tissue

** Differential diagnosis **

1. leukoplakia

*If the etiology of a white lesion is in doubt, it should be regarded as idiopathic leukoplakia.*
Nicotine stomatitis
**Def.**

One of the more common oral forms of keratosis.

**Etiology**

It is associated with pipe and cigar smoking.
Nicotine stomatitis

Clinical features

Site: Palatal mucosa.

S&S:
1. Erythematous type reaction
2. Red dots may be noted on the posterior portion of the hard palate.
3. These dots may be surrounded by a white keratotic ring
4. Elevated dots.
5. These dots represent inflammation of the ductal elements of the underlying minor salivary glands.
Histopathologic features

1. Orthokeratosis.

2. Acanthosis (Thick epithelium)

3. In minor salivary glands, excretory ducts may show squamous metaplasia

4. The glandular elements contain chronic inflammatory cells.
Hairy leukoplakia
1. An opportunistic infection relates to *Epstein-Barr virus*.

2. It is related to *AIDS* patients and in patients with other forms of immunosuppression.

3. *male homosexuals.*
**Clinical feature**

*Site*: along lateral margins of tongue and may extend into dorsal surface.

*S&S*: - asymptomatic
- It may unilateral or bilateral
- folded flat
- plaque like lesion
- corrugated hairy like projections
1. Epithilium: hyperplastic, marked hyperparakeratosis and acanthosis with formation of thin keratotic projections (surface irregularities)

2. Appearance of an area of vacuolated koilocyte-like cells in the upper layers of the epithelium (nuclear alterations created by extensive EBV replication that replaces the chromatin into viral inclusions).

3. Heavy candidal infection of the parakeratin layer

4. Ballooning degeneration in upper spinous layer

5. Connective tissue with no inflammatory reactions

6. No epithelial dysplasia
Diagnosis

1. Immunohistochemical staining technique using anti-viral antibodies.
2. Ultrastructural study using electron microscope

Differential Diagnosis

1. Idiopathic leukoplakia
2. lichen planus
3. leukoplakia associated with tobacco use
4. frictional keratosis
5. chronic hyperplastic candidiasis

Treatment
antiviral drugs such as Acyclovir
Preneoplastic and neoplastic lesions
White lesions

Preneoplastic and neoplastic lesions

1. Actinic Chelitis

2. Idiopathic leukoplakia.
Actinic cheilitis
Def.

Actinic cheilitis

It represents accelerated tissue degeneration of the lips, especially the lower lip.

Etiology

Secondary to regular and prolonged exposure to sunlight.
Actinic cheilitis

Clinical Features

Site: vermilion portion of lips.

S&S:
1. atrophic
2. pale, glossy appearance
3. Fissuring
4. wrinkling at right angles to cutaneous vermilion junction.

FIGURE 7-41
Angular cheilitis (perlèche). Severe fissures in the corners (angles) of the mouth in a patient with adult onset diabetes mellitus.
Actinic cheilitis

**Histopathologic features**
1. Atrophic epithelium
2. Hyperkeratosis.
3. Basal cells are generally hyperchromatic in nature.
4. Basophilic change of submucosa (lamina propria).
5. Telangiectasia

**Prognosis**
Development of carcinoma at this site.

**Treatment**
- No treatment.
Other white lesions
White lesions

Other white lesions

1. Geographic tongue

2. Lichen planus

3. Lupus erythematosus
Geographic tongue
Geographic tongue

**Def.**

An asymptomatic, elongated, erythematous patch of atrophic mucosa of the mid-dorsal surface of the tongue because of a chronic *C. albicans* infection.

**Etiology**

1. Emotional stress
2. Fungal infection
3. Bacterial infection.
4. It is associated with several conditions as: psoriasis, seborrheic dermatitis, and Reiter's syndrome.
Clinical features

Age: Children

Sex: Females > males

Site: Tongue

S&S:

1. small, round &-irregular areas of de-keratinization & desquamation of filiform papillae
2. The desquamated areas become red
3. Slightly tender
4. Elevated margins showing a white-to-slightly yellowish-white rim
5. Lesions move across the dorsum of tongue.
6. As healing occurs on one area, the process extends to adjacent areas.
7. Symptomatic condition
Histopathologic features

1. Reduced in number of filiform papillae
2. Hyperkeratosis and some acanthosis of the margins
3. Closer to the central portion of the lesion, corresponding to the erythematous areas, there is often loss of superficial parakeratin, with significant migration of polymorphic leukocytes and lymphocytes into epithelium.
4. The leukocytes noted within micro-abscesses near the surface.
5. An inflammatory cell infiltrate within the underlying lamina propria, consisting chiefly of neutrophils, lymphocytes and plasma cells.
Geographic tongue

Differential diagnosis

- Candidiasis.
- Leukoplakia.
- Lichen planus.
- Lupus erythematosis.

Treatment

- Not required as condition is self-limited.
- Re-assuring of the patient that this condition is totally benign
Lichen planus
Def.

It is chronic inflammatory mucocutaneous disease may be associated with malignancy, where it appears as either white reticular, plaque, or erosive lesions with a prominent T-lymphocyte response in the immediate underlying connective tissue.

Lichens are primitive plants composed of symbiotic algae and fungi. The term planus is Latin for flat.
Etiology

The etiology of LP is unknown but many factors have been implicated.
Etiology

Epithelial basal cells are the primary target in lichen planus (LP).

The mechanism of basal cell damage appears to be related to a cell-mediated immune process involving Langerhans' cell, T-lymphocytes, and macrophages.
**Pathogenesis of lichen planus**

Langerhans' cells contact and "recognize" an antigen

Langerhans' cells process and present appropriate antigenic determinants to T-lymphocytes

T-lymphocytes attracted to the area by Langerhans / macrophages lymphokines known as "interleukin-1" (IL-1).

- **Stimulates**
  - IL-1
  - T-lymphocytes

- **produce IL-2**

- **T-cell proliferation.**

Activated lymphocytes are subsequently cytotoxic for basal cells & secrete gamma-interferon.
Gamma-interferon induces keratinocytes to express the Class II histocompatibility antigens (HLA-DR) & Lymphocytes normally expressing HLA-DR antigens

HLA-DR increase rate of differentiation of keratinocytes with formation of a thickened surface (This latter feature is seen clinically as "white lesion") & explain the lymphocyte attraction and contact to the epithelium

During this contact, inappropriate epithelial antigenic information may be transferred from Langerhans' cells and macrophages to lymphocytes because of the HLA-DR linkage

With this mechanism, self-antigens may be recognized as "foreign", resulting in an "autoimmune" response

keratinocytes, demonstrating antigens on basal cell surface that are structurally similar to foreign antigens and are recognized by host T-lymphocytes
**Pathogenesis of lichen planus**

These T<sub>8</sub>-lymphocytes become cytotoxic for basal keratinocytes cells in a hyperimmune reaction.

Degeneration of the basal layer that might lead to liberation of an activated factor analogous to IL-1.

IL-1 leading to the stimulation and proliferation of T-lymphocytes.

These lymphocytes secrete, among other mediators, a lymphokine, "Tumor Necrosis Factor-p" (TNFp), which could destroy the epithelium.
Pathogenesis

Acanthosis

LC

KC death

Atrophy

Intraepithelial Tcells

Changes in basal KC and basement membrane

Cytokines

KC: Keratinocyte
LC: Langerhans cell
1. Langerhans cells and macrophages produce IL-1.
2. IL-1 attracts and stimulates T helper (CD4) cells to produce IL-2.
3. IL-2 causes proliferation and activation of T cytotoxic (killer) (CD8) cells.
4. Activated T cells secrete gamma-interferon which induces keratinocytes to express HLA-DR (class II histocompatibility antigens)
5. Normally lymphocytes express HLA-DR but now, keratinocytes expressing HLA-DR also
6. Linkage of these HLA-DR lead to improper epith antigenic information and basal epith cells recognized as foreign body by T cells and stimulate autoimmune response as T cells become cytotoxic for basal cells.
**Lichen planus**

**Clinical feature**

Signs and symptoms: oral lesions often precede skin lesions and may be the only one.

(2) **Oral lesions**

- **5 Forms**
  - Reticular
  - Plaque
  - Atrophic
  - Erosive
  - Bullous
Reticular form Lichen planus

Oral manifestations

The most common type

**Site:** 1. buccal mucosa.

2. tongue and less frequently on gingiva and the lips, or they may occur anywhere

**S&S:** - Presence of numerous interlacing keratotic lines or striae (the so-called "Wickham's Striae") that produce lacy pattern.

- Symmetrical fashion
**Site:** 1. over the dorusm of tongue
2. buccal mucosa

**S&S:**
1. It resembles leukoplakia
2. Elevated plaques
3. smooth surface.
FIGURE 8-8
Lichen planus. Plaque form of LP on the buccal mucosa.
FIGURE 8-9
Lichen planus. Papular skin lesions distributed in a linear pattern on the flexor surface of the wrist.

FIGURE 8-10
Lichen planus. Papular lesions on the skin of the sole of the foot.
The "atrophic form" may be seen in conjunction with reticular or erosive variants.

**Site:** attached gingiva.

**S&S:** 1. whitish keratotic striae that are usually evident at margins of the atrophic zones radiating peripherally and blending into surrounding mucosa.

2. Symptomatic, with patients complaining of burning or pain in the area of involvement.
Erosive form lichen planus

*S&S: 1. Granular surface
   2. Brightly erythematous

Careful examination usually demonstrates a keratotic component, generally peripheral to the site of erosion, with either reticular or finely radiating keratotic striae.
**Bullous variant Lichen planus**

**Site:** Buccal mucosa, especially in the posterior and inferior regions adjacent to the second and third molars & lateral margin of the tongue. Rarely, gingiva and along the inner aspect of the lips.

**S&S:**

The bullae or vesicles range from a few millimeters to several centimeters in diameter.

2. Bullae are short-lived and, upon rupturing, leave an ulcerated, extremely painful surface
Skin lesions

1. Small, violaceous, polygonal, flat-topped papules with a predilection for the flexor surfaces.

2. Cutaneous lesions are noted in approximately 20 to 60% of patients presenting with oral lichen planus.
Lichen planus

Histopathologic features

reticular form

1. hyperorthokeratosis or hyperparakeratosis.
2. Variable degrees of acanthosis may be seen.
3. Liquefaction of basal layer to the extent of a near-total absence of basal cells.
4. Destruction of the epithelial-connective tissue interface is noted
5. lymphocytic band pattern found subepithelially in the lamina propria.
6. increased numbers of Langerhans' cells within the epithelium (as demonstrated with immunohistochemistry).
7. Discrete eosinophilic ovoid bodies representing necrotic keratinocytes are occasionally noted at the basal cell level or within the surrounding inflammatory cell infiltrate. Colloid (or the so-called "Civatte Bodies").
8. Eosinophilic band adjacent to the basement membrane zone, often between the lymphocytic infiltrate and the epithelial cells are also present.
**FIGURE 8-11**

**Lichen planus.** Microscopic features demonstrating the characteristic narrow dense band of T lymphocytes in the immediately adjacent connective tissue of reticular LP (A) and atrophic and erosive LP (B).
Histopathology

Discrete eosinophilic ovoid bodies representing the apoptotic keratinocytes (degenerate cells) are seen at the basal zone, the area of epith and connective tissue interface and have been termed colloid, hyaline or civatte bodies.
A thin band of eosinophilic condensation eosinophilic band may be seen at the junction of epith and connective tissue and it may contain IgM and fibrin.

Presence of a dense band of inflammatory cells lymphocytic band just beneath the epith / connective tissue junction.

The inflam cells are almost entirely lymphocytes (T-lymphocytes) and macrophages.

No significant degree of epith atypia.
Oral Lichen planus

Histopathology

lymphocytic band and basal cell degeneration
Oral Lichen planus

Histopathology

Atrophic lichen planus

Hyperplastic lichen planus
Oral Lichen planus

Histopathology

Ulcerative lichen planus
Oral Lichen planus

Histopathology

The basement membrane is thickened (esinophilic band)
Oral Lichen planus

Prognosis

- Almost all cases of oral lichen planus run a benign course but malignant transformation has been described in very small proportion and more likely occurs in the erosive and atrophic forms.

Treatment

- Corticosteroids
- Antifungal therapy to eliminate of 2ry candida albicans growth
- Systemic and topical vitamin A analogues (Retinoids)
Lichen planus

Differential diagnosis
1. Atrophic candidiasis
2. Leukoplakia
3. Squamous cell carcinoma
4. Drug eruption

N.B
Erosive atrophic lichen planus affecting the attached gingiva must be differentiated from "circatricial pemphigoid"

Treatment and prognosis
- Topical and systemic corticosteroids are useful.
- Vitamin A (retinoids) has been used.
Non-epithelial white yellow lesions
Mucosal burns
Etiology

Chemical burns

Topical application of chemicals such as aspirin or caustic agents to the mucosa.

Thermal Burn

It associated with sticky hot foods that adhere to the palate.

Electric burn

Electric
**Clinical features**

**chemical burns**
1. localized mild erythema in case of short-term exposure to agent capable of inducing tissue necrosis.
2. Coagulation necrosis is more likely to occur as the concentration of the offending agent increases.
3. White slough or membrane.
4. Beneath the membrane, there will be a friable & painful surface that will bleed easily upon mobilization.

**Thermal Burn**
*Site*: hard palatal mucosa
*Features*: as chemical burn

**Electric burn**
1. It is potentially quite serious because they are more destructive.
2. Tissue damage, followed by scarring and reduction in the size of the oral opening.
Histopathologic features

Chemical burns
1. Epithelial component show "coagulation necrosis"
2. A fibrinous exudate is also evident
3. Intensely inflamed connective tissue.

Thermal burns
The same as chemical burns

Electrical burns
1. Deep extension of necrosis, often into muscles.
Mucosal burns

Differential diagnosis

- Acute necrotizing ulcerative gingivitis (ANUG).

In the absence of history of use of chemical or thermal offender, ANUG must be included.
Fordyace's granules
*Fordyce's granules*

**Def.**

It represents ectopic sebaceous glands or sebaceous choristomas (normal tissue in an abnormal site).

It is seen in approximately 80% of individuals.

**Etiology**

It is developmental in origin.
### Clinical features

**Site:** buccal mucosa, vermilion border of upper lip

**Age:** postpubertal.

**S&S:** 1. Asymptomatic.  
2. multiple  
3. symmetrically distributed

### Histopathologic features

- Superficial located lobules of sebaceous glands are aggregated around or adjacent to excretory ducts
- These ducts contain sebaceous and keratinous debris
Candidiasis
**BOX 7-4**

Classification of Basic Types of Oral Candidiasis

**ACUTE**
- Pseudomembranous (thrush)
- Atrophic (erythematous)

**CHRONIC**
- Hyperplastic (candidal leukoplakia)

**BOX 7-5**

Oral Lesions Associated with *Candida Albicans*

- Angular cheilitis (perlèche)
- Median rhomboid glossitis
- Chronic mucocutaneous candidiasis
**Acute pseudomembranous candidiasis (thrush)**

**Def.**

A clinical form of *C. albicans* infection that consists of creamy, loose patches of desquamative epithelium containing numerous matted mycelia over an erythematous mucosa that is easily removed, common in patients with more severe predisposing factors.
Classification of oral and perioral candidosis
Acute pseudomembranous candidosis (Thrush)

Predisposing factors
1. Local disturbance or systemic illness.
2. Antibiotic
3. Corticosteroid
4. Immunosuppressive drug therapy
5. Diabetes mellitus
6. Anaemia
7. Blood dyscrasias such as leukaemia
8. Advanced malignancy
9. Immunodeficiency states
Clinical features

**Age:** > 5% of newborn infants & 10% of elderly debilitated patients

**Site:** any mucosal surface of mouth

**S&S:** 1. Thick white coating (pseudo membrane) can be wiped away to leave a red, raw & bleeding base

2. Variation in size from small patches to confluent lesions covering a wide area.

**FIGURE 7.37**

*Acute pseudomembranous candidiasis (thrush).* Tongue exhibits a superficial layer of loose, cream-colored deposit with raised curdlike patches that can be removed, revealing an erythematous base.
Histological examination

1. Hyperplastic epithelium with the superficial layers infiltrated both by candidal hyphae and spores, and by inflammatory cells which are predominantly neutrophil leucocytes.

2. The neutrophils may accumulate to form micro-abscesses.

3. Intense inflammatory infiltrate and oedema at the junction between the superficial infected layer (the pseudomembrane) and the deeper epithelium.

4. Infiltration of the lamina propria by acute and chronic inflammatory cells.

5. The candidal hyphae appear as weakly basophilic, thread-like structures with haematoxylin and eosin staining, but are seen much more clearly after special staining such as in periodic acid Schiff (PAS) preparations.

6. Examination of a smear made of the pseudomembrane shows necrotic material and leucocytes with epithelial cells partly matted together by candidal spores and hyphae.

Acute pseudomembranous candidosis (Thrush)
FIGURE 7-38
Acute pseudomembranous candidiasis. Cytologic smear of whitish pseudomembrane stained with PAS demonstrating the branching hyphae and budding yeast spores superimposed on desquamated superficial squames.
Thank you