

# Oral pathology

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## Oral mucosal lesions

The oral cavity is lined by a membrane composed of stratified squamous epithelium. This epithelium serves as a cover for the oral soft tissues as a barrier to the entry of external pathogenic factors. Depending on the intraoral site, the stratified squamous epithelium may be non-keratinized, orthokeratinized or parakeratinized. Knowledge of clinical aspects of oral mucosal diseases must be correlated with oral anatomy. E.g. recurrent aphthous stomatitis occurs primarily on the non-keratinized mucosa, whereas recurrent herpes simplex infections occur almost exclusively on the keratinized mucosa.

- Keratinized mucosa** (functional mucosa) e.g. gingiva and hard palate.
- Non –keratinized mucosa** (lining mucosa) e.g. floor of the mouth and buccal mucosa.
- Specialized mucosa** e.g. dorsal surface of the tongue .

In general, oral mucosal lesions could be divided into:

- **Oral infections**
  - Viral
  - Bacterial
  - Fungal
- **Ulcerative conditions**
- **Vesiculobullous diseases**
- **White lesions**

The clinician should be familiar with the following terms :

**Macule:** Focal area of color change which is not elevated or depressed in relation to its surroundings.

**Papule:** Solid, raised lesion which is less than 5 mm in diameter.

**Nodule:** Solid, raised lesion which is greater than 5 mm in diameter.

**Sessile:** Describing a tumor or growth whose base is the widest part of the lesion.

**Pedunculated:** Describing a tumor or growth whose base is narrower than the widest part of the lesion.

**Papillary:** Describing a tumor or growth exhibiting numerous surface projections.

**Verrucous:** Describing a tumor or growth exhibiting a rough, warty surface.

**Vesicle:** Superficial blister, 5 mm or less in diameter, usually filled with clear fluid.

**Bulla:** Large blister, greater than 5 mm in diameter.

**Pustule:** Blister filled with purulent exudate.

**Ulcer:** Lesion characterized by loss of the surface epithelium and frequently some of the underlying connective tissue. It often appears depressed or excavated.

**Erosion:** Superficial lesion. Often arising secondary to rupture of a vesicle or bulla, that is characterized by partial or total loss of the surface epithelium.

**Fissure:** Narrow, slit like ulceration or groove.

**Plaque:** Lesion that is slightly elevated and is flat on its surface.

**Petechia:** Round, pinpoint area of hemorrhage.

**Ecchymosis:** Nonelevated area of hemorrhage, larger than a petechia.

**Telangiectasia:** Vascular lesion caused by dilatation of a small, superficial blood vessel.

**Cyst:** Pathologic epithelium-lined cavity often filled with liquid or semi-solid contents.

### **Microscopic changes of oral mucosa:**

#### ❖ **Epithelial changes:**

**Hyperkeratosis:** refers to an increase in the thickness of stratum corneum, which yields a white appearance of the oral mucosa clinically. This hyperkeratinizations can occur in keratinized area or abnormally in non-keratinized area. When the nuclei are lost from the surface the condition is named (hyperorthokeratosis). When remnants of the nuclei persist the condition is named (hyperparakeratosis).

**Hyperplasia:** an increase in the thickness of the epithelium from surface to basal cell layer.

**Acanthosis :** An increase in the prickle cell layer is termed .

**Epithelial dysplasia** (dyskeratosis or epithelial atypia): an abnormal growth pattern of epithelial cells. Generally indicates a premalignant change.

**Acantholysis :** loss of adhesion between the cells of prickle cell layer (spinous cell layer) the cells appear to fall apart, which lead to vesicle formation, e.g. pemphigus vulgaris.

#### ❖ **Connective tissue changes:**

- Inflammatory infiltrate are common, as chronic inflammatory cells infiltration, e.g. gingivitis.

- Hyperplasia of connective tissue refers to an increase in the amount of collagen fibers.

- Ductal and glandular distension could be seen in many accessory mucous glands due to pressure and obstruction.

# Oral infections

## I-Bacterial Infections

### Tonsillitis and Pharyngitis

Tonsillitis and Pharyngitis are extremely common and may be caused by many different organisms. Group A streptococci is responsible for 20% to 30% of acute pharyngitis cases in children and 5% to 15% of cases in adults. Spread is typically by person-to-person contact through respiratory droplets or oral secretions, with a short incubation period of 2 to 5 days.

#### **Clinical Features**

Although the infection can occur at any age, the greatest prevalence occurs in children 5 to 15 years old, with most cases in temperate climates arising in the winter or early spring.

The signs and symptoms of tonsillitis and pharyngitis vary from mild to intense. Common findings include sudden onset of sore throat, temperature of 101° to 104° F, dysphagia, tonsillar hyperplasia, redness of the oropharynx and tonsils, palatal petechiae, cervical lymphadenopathy, and a yellowish tonsillar exudate that may be patchy or confluent.

Systemic symptoms, such as headache, malaise, anorexia, abdominal pain, and vomiting, may be noted, especially in younger children.



### **Treatment :**

Streptococcal pharyngitis usually is self-limited and resolves spontaneously within 3 to 4 days after onset of symptoms.

The oral antibiotic of choice for group A streptococci is either penicillin V or amoxicillin. Other choices for penicillin-allergic patients include azithromycin, clindamycin, cephalosporins and macrolides (such as, erythromycin or clarithromycin).

### **Scarlet Fever (Scarlatina)**

Scarlet fever is a systemic infection produced by group A,  $\beta$ -hemolytic streptococci. The disease begins as a streptococcal tonsillitis with pharyngitis in which the organisms elaborate an erythrogenic toxin that attacks the blood vessels and produces the characteristic skin rash.

### **Clinical Features:**

Scarlet fever is most common in children from the ages of 3 to 12 years. The tonsils, soft palate, and pharynx become erythematous and edematous, and the tonsillar crypts may be filled with a yellowish exudate. In severe cases, the exudates may become confluent and can resemble diphtheria. Scattered petechiae may be seen on the soft palate in up to 10% of affected patients. During the first 2 days, the dorsal surface of the tongue demonstrates a white coating through which only the fungiform papillae can be seen; this has been called white strawberry tongue.

By the fourth or fifth day, red strawberry tongue develops when the white coating desquamates to reveal an erythematous dorsal surface with hyperplastic fungiform papillae.

Classically, in untreated cases, fever develops abruptly around the second day. The patient's temperature peaks at approximately 103° F and returns to normal within 6 days. Abdominal pain, headache, malaise, nausea, and vomiting frequently are present.

The exanthematous rash develops within the first 2 days and becomes widespread within 24 hours. The classic rash of scarlet fever is distinctive and often is

described as a “sunburn with goose pimples.” Pinhead punctate areas that are normal in color project through the erythema, giving the skin of the trunk and extremities a sandpaper texture. The rash is more intense in areas of pressure and skin folds. In contrast, the skin of the face usually is spared or may demonstrate erythematous cheeks with circumoral pallor. The rash usually clears within 1 week, and then a period of desquamation of the skin occurs.



### **Treatment:**

The oral antibiotic of choice for group A streptococci is either penicillin V or amoxicillin. Other choices for penicillin-allergic patients include azithromycin, clindamycin, cephalosporins such as, cefadroxil or cephalexin), and macrolides (such as erythromycin or clarithromycin). Ibuprofen can be used to reduce the fever and relieve the associated discomfort.

## Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis is a relatively rare specific infectious gingival disease of young persons. *Fusobacterium nucleatum*, *Treponema vincentii*, and probably other bacteria play an important role. Predisposing factors are emotional stress, smoking, poor oral hygiene, local trauma, and HIV infection.

### **Clinical features:**

The characteristic clinical feature is painful necrosis of the interdental papillae and the gingival margins, and the formation of craters covered with a gray pseudo-membrane. Spontaneous gingival bleeding, halitosis, and intense salivation are common. Fever, malaise, and lymphadenopathy are less common. Rarely, the lesions may extend beyond the gingiva (necrotizing ulcerative stomatitis).

### **Treatment:**

Systemic metronidazole and oxygen-releasing agents topically are the best therapy in the acute phase, followed by a mechanical gingival treatment.



## Noma

Noma, also known as cancrum oris and gangrenous stomatitis, is a devastating disease of malnourished children that is characterized by a destructive process of the orofacial tissues. The condition is rare in developed countries. Necrosis of tissue occurs as a consequence of invasion by anaerobic bacteria such as *Fusobacterium necrophorum*, *Prevotella intermedia* and spirochaetes in a host whose systemic health is significantly compromised.



### ***Clinical Features :***

It typically affects children. also can occur in adults with a major debilitating disease (e.g., diabetes mellitus, leukemia, lymphoma, or HIV infection).The initial lesion of noma is a painful ulceration, usually of the gingiva or buccal mucosa, which spreads rapidly and eventually becomes necrotic. Denudation of involved bone may follow, eventually leading to necrosis and sequestration. Teeth in the affected area may become loose and may exfoliate. Penetration of organisms into the cheek, lip, or palate may also occur, resulting in fetid necrotic lesions.





**Treatment:** Therapy involves treating the underlying predisposing condition, as well as the infection itself. Therefore fluids, electrolytes, and general nutrition are restored, along with the introduction of antibiotics.

## Syphilis

Syphilis is a relatively common sexually transmitted disease Caused by *Treponema pallidum*.

### ***Clinical features:***

Syphilis may be acquired (common) or congenital (rare). Acquired syphilis is classified as primary, secondary and tertiary. The characteristic lesion in the primary stage is the chancre that appears at the site of inoculation, usually three weeks after the infection. Oral chancre appears in about 5–10% of cases, and clinically presents as a painless ulcer with a smooth surface, raised borders, and an indurated base. Regional lymphadenopathy is a constant finding.



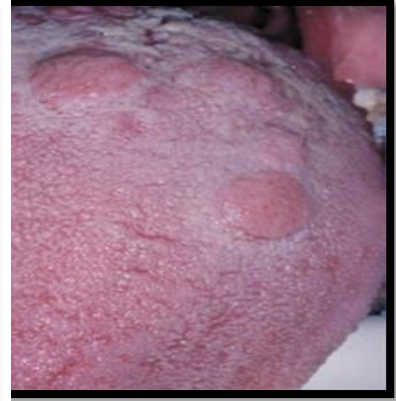
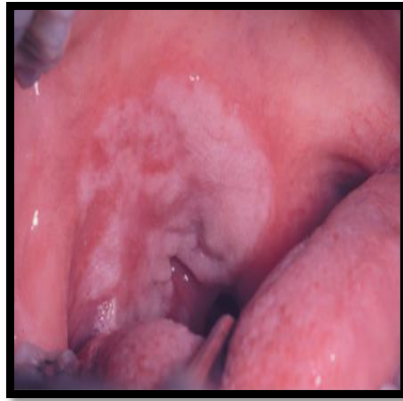
Oral Chancre

The secondary stage begins 6–8 weeks after the appearance of the chancre, and lasts for 2–10 weeks. Oral lesions are mucous patches (common), macular syphilids, and condylomata lata (rare). Constitutional symptoms and signs (malaise, low-grade fever, headache, lacrimation, sore throat, weight loss, myalgias and multiple arthralgias, generalized lymphadenopathy) as well as

cutaneous manifestations (macular syphilids, papular syphilids, condylomata lata, nail involvement, hair loss, atypical rash, etc.) are constant findings.



mucous patches



condylomata lata

Tertiary syphilis begins after a period of 4–7 years. Oral lesions are gumma, atrophic glossitis, and interstitial glossitis.

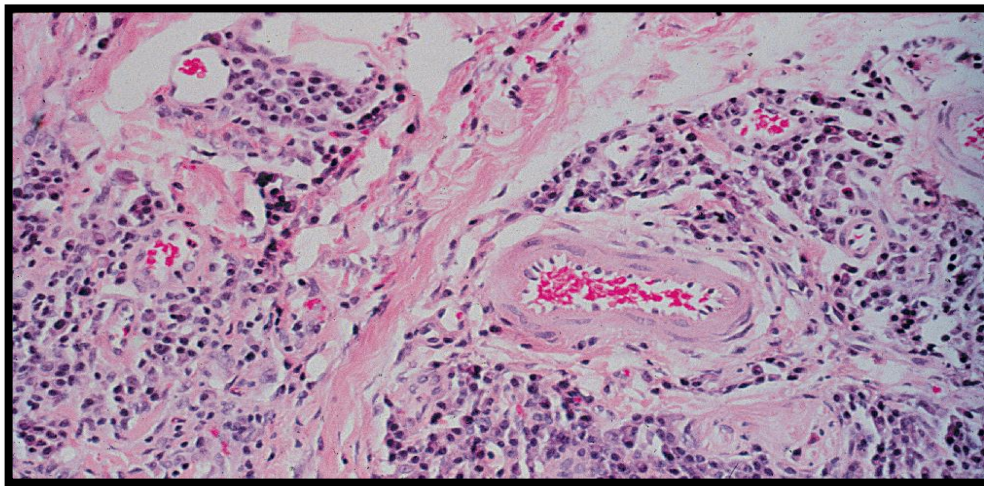


The most common oral lesions in congenital syphilis are a high-arched palate, short mandible, Hutchinson's teeth, and Moon's or mulberry molars.

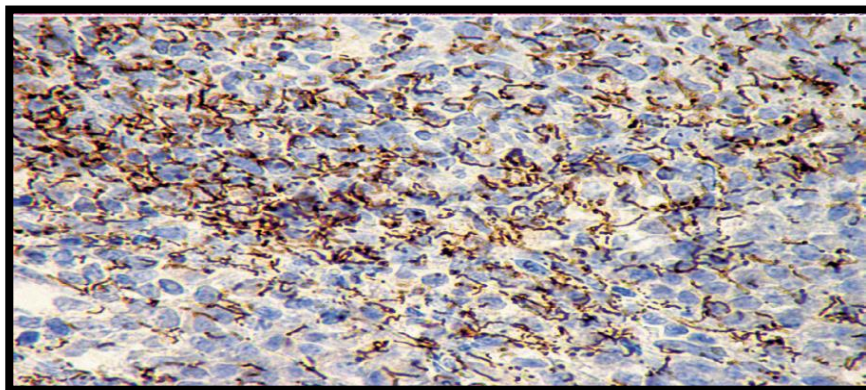


### ***Histopathology.***

The basic tissue response to *T. pallidum* infection consists of a proliferative endarteritis and infiltration of plasma cells. Spirochetes can be demonstrated in the tissues of various lesions of syphilis using silver stains, although they may be scant in tertiary lesions. Gumma may show necrosis and greater numbers of macrophages, resulting in a granulomatous lesion that is similar to other conditions, such as tuberculosis (TB).



A chronic perivascular inflammatory infiltrate of plasma cells and lymphocytes.



Immunoperoxidase reaction for *Treponema pallidum* demonstrating numerous spirochetes in the epithelium.

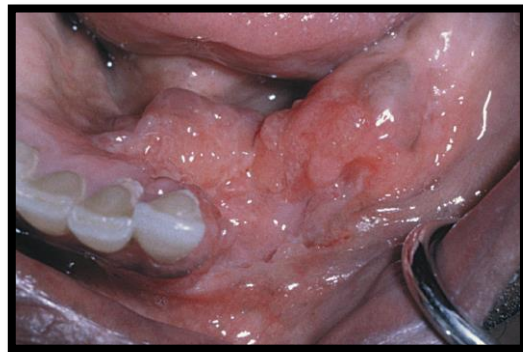
***Treatment.*** Penicillin is the antibiotic of choice. Erythromycin or cephalosporins are good alternatives.

## Tuberculosis

Tuberculosis is a chronic, granulomatous, infectious disease that primarily affects the lungs, caused by *Mycobacterium tuberculosis*.

### *Clinical features:*

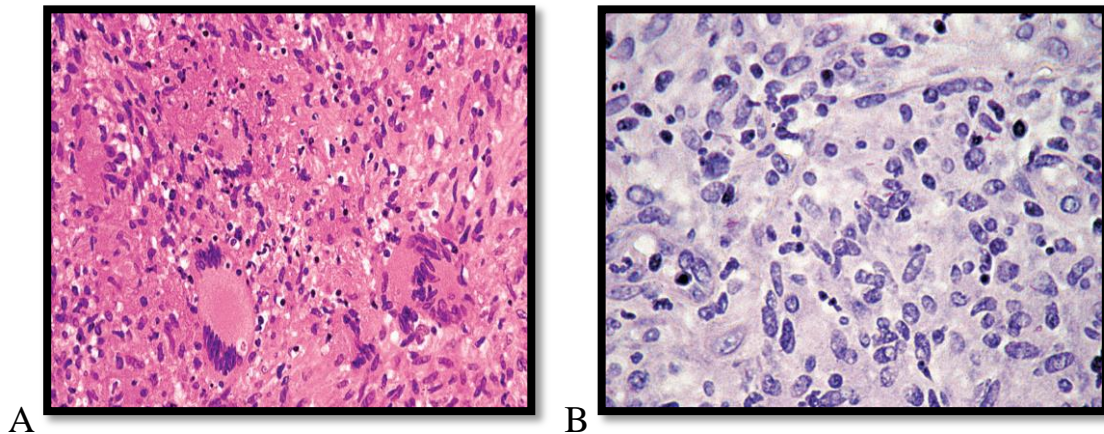
The oral lesions are rare, and usually secondary to pulmonary tuberculosis. The tuberculous ulcer is the most common feature. Clinically, the ulcer is painless and irregular, with a thin undermined border and a vegetating surface, usually covered by a gray-yellowish exudate. The surrounding tissues are inflamed and indurated. The dorsum of the tongue is the most commonly affected site, followed by the lip, buccal mucosa, and palate. Osteomyelitis of the jaws, periapical granuloma, regional lymphadenopathy, and scrofula are less common oral manifestations.



### ***Histopathology.***

The basic microscopic lesion of TB is granulomatous inflammation, in which granulomas show central caseous necrosis. In tissues, *M. tuberculosis* incites a characteristic macrophage response, in which focal zones of macrophages become surrounded by lymphocytes and fibroblasts. The macrophages develop an abundant eosinophilic cytoplasm, giving them a superficial resemblance to epithelial cells; for this reason, they are frequently called epithelioid cells. Fusion of macrophages results in the appearance of Langerhans giant cells, in which nuclei are distributed around the periphery of the cytoplasm. As the granulomas age, central necrosis occurs; this is usually referred to as caseous necrosis because of the gross cheesy texture of these zones.

A Ziehl-Neelsen or Fite stain must be used to confirm the presence of the organism in the granulomas, because several infectious and noninfectious conditions may produce a similar granulomatous reaction.



(A) Sheets of histiocytes are intermixed with multinucleated giant cells and areas of necrosis. (B) Acid-fast stain exhibiting scattered mycobacterial organisms presenting as small red rods.

## Actinomycosis

Actinomycosis is a chronic bacterial disease caused by *Actinomyces israelii*, an anaerobic, gram-positive bacterium. Infection usually appears after trauma, surgery, or previous infection.

### *Clinically:*

It typically presents as swelling of the mandible that may simulate a pyogenic infection. The lesion may become indurated and eventually may form one or more draining sinuses, leading from the medullary spaces of the mandible to the skin of the neck. The clinical course ranges from acute to chronic. The skin lesions are indurated and are described as having a “woody hard” consistency. Pus draining from the chronic lesion may contain small yellow granules, known as sulfur granules, which represent aggregates of *A. israelii* organisms. Radiographically, this infection presents as a lucency with irregular and ill-defined margins.



### *Histopathology.*

A granulomatous inflammatory response with central abscess formation is seen in actinomycosis. At the center of the abscesses, distinctive colonies of gram-positive organisms may be seen. Radiating from the center of the colonies are numerous filaments with clubbed ends.

### *Treatment.*

Long-term, high-dose penicillin or penicillin analogs are the required antibiotic regimen for actinomycosis.

