

## LESSON № 2 Caries and its complications/ pulpitis, periodontitis/ Oral mucosa diseases.

### Dental caries

Dental caries is a disease that damages tooth structures, resulting in what is commonly called tooth decay or cavities, which are holes in the teeth. This damage first affects the hard tissues of the teeth (enamel, dentin and cementum). As the destruction progresses, these tissues begin to break down, which can eventually lead to holes in the teeth. If left untreated, the disease can lead to pain, tooth loss, infection, and, in severe cases, death. There is a long history of dental caries: over a million years ago, hominids such as Australopithecus suffered from cavities. However, the incidence of cavities was very low well into the Paleolithic and Mesolithic periods. The largest increases in the prevalence of caries have been associated with dietary changes. Today, caries remains one of the most common diseases throughout the world.

There are numerous ways to classify dental caries. Although the presentation may differ, the risk factors and development among distinct types of caries remain largely similar. Initially, it may appear as a small chalky area but eventually develop into a large, brown cavitation. Though sometimes caries may be seen directly, radiographs are frequently needed to inspect less visible areas of teeth and to judge the extent of destruction.

Tooth decay is caused by certain types of acid-producing bacteria (specifically Lactobacillus species, Streptococcus mutans, and Actinomyces species) which cause damage in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose.<sup>[6][7][8]</sup> The resulting high levels of acidity from lactic acid in the mouth affect teeth because a tooth's special mineral content causes it to be sensitive to low pH. Specifically, a tooth (which is primarily mineral in content) is in a constant state of back-and-forth demineralization and remineralization between the tooth and surrounding saliva. When the pH at the surface of the tooth drops below 5.5, demineralization proceeds faster than remineralization (i.e. there is a net loss of mineral structure on the tooth's surface). This results in the ensuing decay. Depending on the extent of tooth destruction, various treatments can be used to restore teeth to proper form, function, and aesthetics, but there is no known method to regenerate large amounts of tooth structure. Instead, dental health organizations advocate preventive and prophylactic measures, such as regular oral hygiene and dietary modifications, to avoid dental caries.

### History



An image from 1300s (A.D.) England depicting a dentist extracting a tooth with forceps. Archaeological evidence shows that dental caries is an ancient disease dating far into prehistory. Skulls dating from a million years ago through the neolithic period show signs of caries, excepting those from the Paleolithic and Mesolithic ages. The increase of caries during the neolithic period may be attributed to the increase of plant foods containing carbohydrates. The beginning of rice cultivation in South Asia is also believed to have caused an increase in caries. A Sumerian text from 5000 BC describes a "tooth worm" as the cause of caries. Evidence of this belief has also been found in India, Egypt, Japan, and China.

Unearthed ancient skulls show evidence of primitive dental work. In Pakistan, teeth dating from around 5500 BC to 7000 BC show nearly perfect holes from primitive dental drills. The Ebers Papyrus, an Egyptian text from 1550 BC, mentions diseases of teeth. During the Sargonid dynasty of Assyria during 668 to 626 BC, writings from the king's physician specify the need to extract a tooth due to spreading inflammation. In the Roman Empire, wider consumption of cooked foods led to a small increase in caries

prevalence. The Greco-Roman civilization, in addition to the Egyptian, had treatments for pain resulting from caries.

The rate of caries remained low through the Bronze and Iron ages, but sharply increased during the Medieval period. Periodic increases in caries prevalence had been small in comparison to the 1000 AD increase, when sugar cane became more accessible to the Western world. Treatment consisted mainly of herbal remedies and charms, but sometimes also included bloodletting. The barber surgeons of the time provided services that included tooth extractions. Learning their training from apprenticeships, these health providers were quite successful in ending tooth pain and likely prevented systemic spread of infections in many cases. Among Roman Catholics, prayers to Saint Apollonia, the patroness of dentistry, were meant to heal pain derived from tooth infection.

There is also evidence of caries increase in North American Indians after contact with colonizing Europeans. Before colonization, North American Indians subsisted on hunter-gatherer diets, but afterwards there was a greater reliance on maize agriculture, which made these groups more susceptible to caries.

In the medieval Islamic world, Muslim physicians such as al-Gazzar and Avicenna (in The Canon of Medicine) provided the earliest known treatments for caries, though they also believed that it was caused by tooth worms like what the ancients believed. This was eventually proven false in 1200 by another Muslim dentist named Gaubari, who in his Book of the Elite concerning the unmasking of mysteries and tearing of veils, was the first to reject the idea of caries being caused by tooth worms, and he stated that tooth worms in fact do not even exist. The theory of the tooth worm was thus no longer accepted in the Islamic medical community from the 13th century onwards.

During the European Age of Enlightenment, the belief that a "tooth worm" caused caries was also no longer accepted in the European medical community. Pierre Fauchard, known as the father of modern dentistry, was one of the first to reject the idea that worms caused tooth decay and noted that sugar was detrimental to the teeth and gingiva. In 1850, another sharp increase in the prevalence of caries occurred and is believed to be a result of widespread diet changes. Prior to this time, cervical caries was the most frequent type of caries, but increased availability of sugar cane, refined flour, bread, and sweetened tea corresponded with a greater number of pit and fissure caries.

In the 1890s, W.D. Miller conducted a series of studies that led him to propose an explanation for dental caries that was influential for current theories. He found that bacteria inhabited the mouth and that they produced acids which dissolved tooth structures when in the presence of fermentable carbohydrates. This explanation is known as the chemoparasitic caries theory.<sup>1</sup> Miller's contribution, along with the research on plaque by G.V. Black and J.L. Williams, served as the foundation for the current explanation of the etiology of caries.

In 1921, Major Fernando E. Rodriguez Vargas, DDS of the Army Dental Corps discovered the bacteria which causes dental caries. According to his investigations, three types of the Lactobacillus species, during the process of fermentation, are the causes of cavities. In December 1922, he published an original and fundamental work on the specific bacteriology of dental caries. His findings were published in the December issue of the Military Dental Journal titled "The Specific Study of the Bacteriology of Dental Cavities". Rodríguez Vargas also developed the techniques and methods of analysis. On September 28, 1928, Rodriguez Vargas published in the "Journal of the American Medical Association" his findings in the effectiveness of Iodine and other chemical agents as disinfectants of the mucous membranes of the mouth. Since then, other scientists have used the findings of his investigations as the basis in the study of the bacteriology of dental caries.

## **Epidemiology**

Worldwide, most children and an estimated ninety percent of adults have experienced caries, with the disease most prevalent in Asian and Latin American countries and least prevalent in African countries. In the United States, dental caries is the most common chronic childhood disease, being at least five times

more common than asthma. It is the primary pathological cause of tooth loss in children. Between 29% and 59% of adults over the age of fifty experience caries.<sup>1</sup>

The number of cases has decreased in some developed countries, and this decline is usually attributed to increasingly better oral hygiene practices and preventive measures such as fluoride treatment. Nonetheless, countries that have experienced an overall decrease in cases of tooth decay continue to have a disparity in the distribution of the disease. Among children in the United States and Europe, twenty percent of the population endures sixty to eighty percent of cases of dental caries. A similarly skewed distribution of the disease is found throughout the world with some children having none or very few caries and others having a high number. Australia, Nepal, and Sweden have a low incidence of cases of dental caries among children, whereas cases are more numerous in Costa Rica and Slovakia.

The classic "DMF" (decay/missing/filled) index is one of the most common methods for assessing caries prevalence as well as dental treatment needs among populations. This index is based on in-field clinical examination of individuals by using a probe, mirror and cotton rolls. Because the DMF index is done without X-ray imaging, it underestimates real caries prevalence and treatment needs.

## **Classification**

Caries can be classified by location, etiology, rate of progression, and affected hard tissues. These classification can be used to characterize a particular case of tooth decay in order to more accurately represent the condition to others and also indicate the severity of tooth destruction.

### **Location**

Generally, there are two types of caries when separated by location: caries found on smooth surfaces and caries found in pits and fissures. The location, development, and progression of smooth-surface caries differ from those of pit and fissure caries. G.V. Black created a classification system that is widely used and based on the location of the caries on the tooth. The original classification distinguished caries into five groups, indicated by the word, "Class", and a Roman numeral. Pit and fissure caries is indicated as Class I; smooth surface caries is further divided into Class II, Class III, Class IV, and Class V. A Class VI was added onto Black's classification and also represents a smooth-surface carious lesion.

### **Etiology**

Rampant caries as seen here may be due to methamphetamine use.

In some instances, caries are described in other ways that might indicate the cause. "Baby bottle caries", "early childhood caries", or "baby bottle tooth decay" is a pattern of decay found in young children with their deciduous (baby) teeth. The teeth most likely affected are the maxillary anterior teeth, but all teeth can be affected. The name for this type of caries comes from the fact that the decay usually is a result of allowing children to fall asleep with sweetened liquids in their bottles or feeding children sweetened liquids multiple times during the day. Another pattern of decay is "rampant caries", which signifies advanced or severe decay on multiple surfaces of many teeth. Rampant caries may be seen in individuals with xerostomia, poor oral hygiene, methamphetamine use (due to drug-induced dry mouth<sup>1</sup>), and/or large sugar intake. If rampant caries is a result of previous radiation to the head and neck, it may be described as radiation-induced caries. Problems can also be caused by the self destruction of roots and whole tooth resorption when new teeth erupt or later from unknown causes.

### **Signs and symptoms**

Dental explorer used for caries diagnosis.

A person experiencing caries may not be aware of the disease. The earliest sign of a new carious lesion is the appearance of a chalky white spot on the surface of the tooth, indicating an area of demineralization of enamel. This is referred to as incipient decay. As the lesion continues to demineralize, it can turn brown but will eventually turn into a cavitation ("cavity"). Before the cavity forms, the process is reversible, but once a cavity forms, the lost tooth structure cannot be regenerated. A lesion which appears brown and

shiny suggests dental caries was once present but the demineralization process has stopped, leaving a stain. A brown spot which is dull in appearance is probably a sign of active caries.

As the enamel and dentin are destroyed, the cavity becomes more noticeable. The affected areas of the tooth change color and become soft to the touch. Once the decay passes through enamel, the dentinal tubules, which have passages to the nerve of the tooth, become exposed and cause the tooth to hurt. The pain may worsen with exposure to heat, cold, or sweet foods and drinks. Dental caries can also cause bad breath and foul tastes. In highly progressed cases, infection can spread from the tooth to the surrounding soft tissues. Complications such as cavernous sinus thrombosis and Ludwig's angina can be life-threatening.

## Diagnosis

Primary diagnosis involves inspection of all visible tooth surfaces using a good light source, dental mirror and explorer. Dental radiographs (X-rays) may show dental caries before it is otherwise visible, particularly caries between the teeth. Large dental caries are often apparent to the naked eye, but smaller lesions can be difficult to identify. Visual and tactile inspection along with radiographs are employed frequently among dentists, particularly to diagnose pit and fissure caries.<sup>[48]</sup> Early, uncavitated caries is often diagnosed by blowing air across the suspect surface, which removes moisture and changes the optical properties of the unmineralized enamel. This produces a white 'halo' effect detectable to the naked eye. Fiberoptic transillumination, lasers and disclosing dyes have been recommended for use as an adjunct when diagnosing smaller carious lesions in pits and fissures of teeth) A small spot of decay visible on the surface of a tooth. (B) The radiograph reveals an extensive region of demineralization within the dentin (arrows). (C) A hole is discovered on the side of the tooth at the beginning of decay removal. (D) All decay removed.

Some dental researchers have cautioned against the use of dental explorers to find caries. In cases where a small area of tooth has begun demineralizing but has not yet cavitated, the pressure from the dental explorer could cause a cavity. Since the carious process is reversible before a cavity is present, it may be possible to arrest the caries with fluoride and remineralize the tooth surface. When a cavity is present, a restoration will be needed to replace the lost tooth structure.

At times, pit and fissure caries may be difficult to detect. Bacteria can penetrate the enamel to reach dentin, but then the outer surface may remineralize, especially if fluoride is present. These caries, sometimes referred to as "hidden caries", will still be visible on x-ray radiographs, but visual examination of the tooth would show the enamel intact or minimally perforated.

## Causes

There are four main criteria required for caries formation: a tooth surface (enamel or dentin); caries-causing bacteria; fermentable carbohydrates (such as sucrose); and time. The caries process does not have an inevitable outcome, and different individuals will be susceptible to different degrees depending on the shape of their teeth, oral hygiene habits, and the buffering capacity of their saliva. Dental caries can occur on any surface of a tooth which is exposed to the oral cavity, but not the structures which are retained within the bone.

## Pulpitis

**Pulpitis** is inflammation of the dental pulp resulting from untreated caries, trauma, or multiple restorations. Its principal symptom is pain. Diagnosis is based on clinical findings and is confirmed by x-ray. Treatment involves removing decay, restoring the damaged tooth, and, sometimes, performing root canal therapy or extracting the tooth.

Pulpitis can occur when caries progresses deeply into the dentin, when a tooth requires multiple invasive procedures, or when trauma disrupts the lymphatic and blood supply to the pulp. It begins as a reversible condition in which the tooth can be saved by a simple filling. It becomes irreversible as swelling inside

the rigid encasement of the dentin compromises circulation, making the pulp necrotic, which predisposes to infection.

Infectious sequelae of pulpitis include apical periodontitis, periapical abscess, cellulitis, and osteomyelitis of the jaw. Spread from maxillary teeth may cause purulent sinusitis, meningitis, brain abscess, orbital cellulitis, and cavernous sinus thrombosis. Spread from mandibular teeth may cause Ludwig's angina, parapharyngeal abscess, mediastinitis, pericarditis, empyema, and jugular thrombophlebitis.

### **Symptoms, Signs, and Diagnosis**

In reversible pulpitis, pain occurs when a stimulus (usually cold or sweets) is applied to the tooth. When the stimulus is removed, the pain ceases within 1 to 2 sec.

In irreversible pulpitis, pain occurs spontaneously or lingers minutes after the stimulus is removed. A patient may have difficulty locating the tooth from which the pain originates, even confusing the maxillary and mandibular arches (but not the left and right sides of the mouth). The pain may then cease for several days because of pulpal necrosis. As infection develops and extends through the apical foramen, the tooth becomes exquisitely sensitive to pressure and percussion. A periapical (dentoalveolar) abscess elevates the tooth from its socket and feels "high" when the patient bites down.

Diagnosis is based on the history and physical examination, which makes use of provoking stimuli (application of heat, cold, percussion). X-rays help determine whether inflammation has extended beyond the tooth apex and help exclude other conditions.

### **Treatment**

In reversible pulpitis, pulp vitality can be maintained if the tooth is treated, usually by caries removal, and then restored.

Irreversible pulpitis and its sequelae require endodontic (root canal) therapy or tooth extraction. In endodontic therapy, an opening is made in the tooth and the pulp is removed. The root canal system is thoroughly debrided, shaped, and then filled with gutta-percha. After root canal therapy, adequate healing is manifested clinically by resolution of symptoms and radiographically by bone filling in the radiolucent area at the root apex over a period of months. If the patient has systemic signs of infection, such as fever, an oral antibiotic is prescribed (penicillin VK 500 mg q 6 h or, for patients allergic to penicillin, clindamycin

## **Periodontitis**

Early-stage periodontal disease (gingivitis) is seldom painful and causes relatively minor signs, such as red, swollen and bleeding gums. But untreated gingivitis can progress to periodontitis, a serious infection that destroys the soft tissue and bone that support your teeth, and eventually may cause tooth loss.

What's more, long-term periodontitis can lead to even more-serious problems, including higher blood sugar levels and an increased risk of heart attack and stroke. Gum disease may even affect your unborn child. Pregnant women with periodontitis are much more likely to give birth to premature babies than are women with healthy gums.

Yet periodontitis is both preventable and treatable. Although factors such as smoking, heredity, medications and lowered immunity make you more susceptible to gum disease, the most common cause is poor oral hygiene. Daily brushing and flossing and regular professional cleanings can greatly reduce your chances of developing periodontitis.

**Symptoms** In the earliest stages, periodontal disease causes few signs or symptoms, and you may not be aware of a problem until your gums become soft and bleed slightly when you brush your teeth. As the disease progresses, you may notice more-serious changes, including:

- Swollen, bright red or purple gums

- Gums that feel tender when touched
- Gums that pull away from your teeth (recede), making your teeth look longer than normal
- New spaces developing between your teeth
- Pus between your teeth and gums
- Persistent breath odor or a bad taste in your mouth
- Loose teeth or a change in the way your teeth fit together when you bite

Because several types of periodontitis exist, you may experience problems that are unique to a particular form of the disease. For instance, aggressive periodontitis, which affects otherwise healthy young people, causes an unusually rapid deterioration of their teeth and gums. The condition can also occur episodically, with periods of severe disease alternating with periods when signs and symptoms improve or even seem to disappear.

Other types of periodontitis and their characteristics include:

- Chronic periodontitis. This most common type of gum disease is characterized by progressive loss of the bone and soft tissues that surround and support your teeth. The damage usually develops more slowly than it does in aggressive periodontitis.
- Periodontitis as a manifestation of systemic disease. This usually develops at a young age and occurs in conjunction with another health problem, such as diabetes.
- Necrotizing periodontal disease. A severe form of periodontitis, this causes the death of gum tissue, tooth ligaments and even bone. People suffering from malnutrition or living with HIV/AIDS are especially vulnerable.

## **Causes**

Periodontitis begins with plaque. This invisible, sticky film forms on your teeth when starches and sugars in food interact with bacteria normally found in your mouth. Although you remove plaque every time you brush your teeth, it re-forms quickly, usually within 24 hours.

Plaque that stays on your teeth longer than two or three days can harden under your gumline into tartar (calculus), a white substance that makes plaque more difficult to remove and that acts as a reservoir for bacteria. Unfortunately, brushing and flossing can't eliminate tartar — only a professional cleaning can remove it.

The longer plaque and tartar remain on your teeth, the more damage they can do. Initially, they may simply irritate and inflame the gingiva, the part of your gum around the base of your teeth. This is gingivitis, the mildest form of periodontal disease. But ongoing inflammation eventually causes pockets to develop between your gums and teeth that fill with plaque, tartar and bacteria. In time, the pockets become deeper and more bacteria accumulate, eventually advancing under your gum tissue. These deep infections cause a loss of tissue and bone. If too much bone is destroyed, you may lose one or more teeth.

Although the destructive cycle that starts with the accumulation of plaque is the most common cause of periodontal disease, a number of other factors can contribute to or aggravate the condition. These include:

- Tobacco use. Smoking is the most significant risk factor for periodontal disease. Chewing tobacco also contributes to periodontal disease. Tobacco use in any form damages your immune system, putting you at greater risk of periodontal infection. It also creates a favorable environment for harmful bacteria and interferes with the normal mechanisms for limiting bacterial growth in your mouth. Even exposure to secondhand smoke appears to contribute to periodontal disease. And because smoking impairs healing, smokers are less likely to respond to treatment than nonsmokers are.
- Heredity. Sometimes you may do everything right and still develop periodontal disease. In that case, you — along with close to one-third of the population — may have inherited a predisposition to gum problems.

- **Drugs.** Hundreds of prescription and over-the-counter antidepressants, cold remedies and antihistamines contain ingredients that decrease your body's production of saliva. Because saliva has a cleansing effect on your teeth and helps inhibit bacterial growth, this means that plaque and tartar can build up more easily. Other drugs, especially anti-seizure medications, calcium channel blockers and drugs that suppress your immune system, sometimes cause an overgrowth of gum tissue (gingival hyperplasia), making plaque much tougher to remove.
- **Diabetes.** A number of health problems can take a toll on your gums. One of the most significant of these is diabetes, which makes you more prone to many infections, including gum infections. But the relationship between diabetes and periodontal disease doesn't end there. Gingivitis and periodontitis impair your body's ability to utilize insulin, making diabetes harder to control. And because diabetes and periodontal disease may make you more susceptible to heart attack and stroke, having both conditions increases your risk of cardiovascular disease.
- **Hormonal changes.** Changes in hormone levels that occur during pregnancy, menopause or even menstruation can make your gums more susceptible to periodontal disease.
- **Nutritional deficiencies.** A poor diet, especially one deficient in calcium, vitamin C and B vitamins, can contribute to periodontal disease. Calcium is important because it helps maintain the strength of your bones, including the bones that support your teeth. Vitamin C helps maintain the integrity of connective tissue. It's also a powerful antioxidant that counters the tissue-destroying effects of free radicals — substances produced when oxygen is metabolized by your body.

## **Oral mucosa diseases**

### **Stomatitis**

**Stomatitis** is an inflammation of the mucosal surfaces in the mouth and on the tongue. HSV 1 and 2 viruses and *Candida albicans* are the most common causes of this inflammation. If inflammation includes the gingivae (gums) then it is called gingivostomatitis (common with herpes infections).

There are basically three different types of candidosis; pseudomembranous, erythematous, and hyperplastic. These types are further divided into 4 types;

- (1) Acute pseudomembranous candidosis (thrush),
- (2) Acute erythematous (atrophic) candidosis,
- (3) Chronic hyperplastic candidosis,
  - (a) Chronic oral candidosis (candidal leukoplakia),
  - (b) Endocrine candidosis syndrome,
  - (c) Chronic localized mucocutaneous candidosis
  - (d) Chronic diffuse candidosis.
- (4) Chronic erythematous (atrophic) candidosis.

Thrush (acute pseudomembranous candidiasis) is the term used for the multiple white-fleck appearance of acute candidiasis. Erythematous candidosis is the term used for the red lesions of candidiasis.

### **Etiology**

The most common causes of this infection are Herpes Simplex viruses 1 and 2 (gingivostomatitis or cold sores) and *Candida albicans* (oral candidosis). Other species of *Candida* (ex. *C. krusei*), have been isolated from severely immunocompromised patients with candidosis.

### **Epidemiology**

**Oral herpes-** is a very common infection affecting nearly 60% of the population by 15 years of age. Oral herpes is most commonly seen in young children however, it has also occasionally been observed in adults. HSV-1 and HSV-2 is transmitted person to person by touching infected saliva, mucous membranes, or skin and by sharing of cups or utensils. HSV-1 causes around 80% of the oral herpes infections. HSV-2 is usually transmitted to the oral cavity via oral sex. These viruses become latent and remain with the host for life. Recurrences in the mouth are uncommon. Asymptomatic shedding occurs periodically and even people without any herpetic lesions can infect others. The herpes simplex viruses infect humans only.

## Candidosis

**Candidosis** - a very common endogenous disease in patients with certain predisposing conditions. *Candida albicans* is a common inhabitant of the oral flora (50% of population) and any disturbance of this flora can result in overgrowth of the yeast. Is acquired during passage down the birth canal, by sharing cups and eating utensils, breast-feeding and sharing saliva. Candidosis may predispose individuals to esophageal spread. The percentage of carriers is higher in women than in men.

### **Predisposing factors:**

- § Infants and neonates
- § Patients on broad-spectrum antibiotic treatment
- § Patients taking steroids (inhaled and systemic; Children on inhaled steroids also have increased incidence of oral candidiasis.)
- § Patients with polyendocrine disorders (diabetes)
- § Smokers (30-70% more likely to carry *Candida*)
- § Denture wearers
- § Drugs with xerostomic adverse effects are associated with oral candidosis.
- § Xerostomia (dry mouth; Sjögren syndrome and after radiotherapy)
- § Patients with underlying immune dysfunction (HIV infection)

**Acute pseudomembranous candidosis (Thrush)**-Up to 37% of newborns develops thrush. It is usually a mild and self-limiting disease. It is uncommon during the first week of life. It is most commonly seen around the fourth week of life. Thrush is uncommon in infants older than 6-9 months. Thrush can occur, however, at any age in predisposed patients.

### **Pathogenesis**

**Oral herpes**- the virus causes painful sores on lips, gums, tongue, roof of the mouth, and inside the mucous membranes lining the cheeks. It also can cause symptoms that include fever and muscle aches. Mouth sores most commonly occur in children aged 1-2 years, but it can affect people at any age and any time of the year.

After the herpes virus infection it has the ability to proceed to the following stages.

1. Primary infection: The virus enters the skin or mucous membrane and reproduces. During this stage, oral sores and other symptoms, such as fever, may develop. The virus may cause asymptomatic infections. Asymptomatic infection occur twice as often as symptomatic disease.
2. Latency: From the infected site, the virus moves to nerves in the region. There the virus reproduces and becomes inactive.
3. Recurrence: During certain stresses, emotional or physical, the virus may reactivate and cause new sores and symptoms.

### **Three different types of candidosis:**

**Acute pseudomembranous candidosis (Thrush)** - During some disturbance of the normal oral flora *Candida albicans* will overgrow causing various signs and symptoms described below. Overgrowth of the yeast on the oral mucosa leads to desquamation of epithelial cells and accumulation of bacteria, keratin, and necrotic tissue. This debris combines to form a pseudomembrane, which may adhere closely to the mucosa. Neonates are colonized when they pass down the birth canal. Neonates can also acquire this organism during breast-feeding and from improperly cleaned bottle nipples. The organism also resides in the lower GI tract. Candidal diaper rash is oftentimes observed in conjunction with thrush.

**Erythematous candidosis**- There are both acute and chronic conditions. It is an inflammatory response to *Candida* overgrowth without the pseudomembrane forming or is the infection post pseudomembrane shedding. May arise due to a number of different factors and local conditions:

- § Follows acute pseudomembranous candidosis after the white plaques have shed yet the infection persists;
- § *de novo* in AIDS patients;

- § in patient receiving prolonged drug therapy: topical steroids or broad spectrum antibiotics;
- § most commonly related to wearing dentures.

There are varying degrees of inflammation starting with localized pinpoint hyperemia (type 1), diffuse erythema and edema (type 2), and inflamed hyperplastic epithelium (type 3).

**Hyperplastic candidosis (candidal leukoplakia)**- Is characterized by areas in the mouth of parakeratinization, marked hyperplasia with candidal hyphae invading the parakeratinized layer at right angles and superficial to the surface. In some cases can develop into malignancies.

### Manifestations

**Oral herpes**- The incubation period is 2-12 days. Most people average about 4 days.  
Duration of illness: 2-3 weeks. Fever, tiredness, muscle aches, and irritability may occur.

Pain, burning, tingling, or itching occurs at the infection site before the sores appear. Then clusters of blisters erupt. The blisters break down rapidly and when seen, appear as tiny, shallow, gray ulcers on a red base.

Oral sores: The most intense pain caused by these sores occurs at the onset and make eating and drinking difficult. The sores may occur on the lips, the gums, the front of the tongue, the inside of the cheeks, throat, and the roof of the mouth. They may also extend down the chin and neck.

The gums may become red, mildly swollen and may bleed. Neck lymph nodes often swell and become painful. In people in their teens and 20s, herpes may cause a painful throat with shallow ulcers and a grayish coating on the tonsils.

### **The 3 basic types of candidosis:**

**Acute pseudomembranous candidosis (Thrush)** – Is characterized by the presence of creamy-white plaques (pseudomembranes) that consist of superficial mucosal cells, neutrophils and yeast cells. These plaques can be found on the tongue, soft palate, cheek, gingivae and/or pharynx. Lesions often start as tiny focal areas that enlarge to white plaques (patches). When scraped with a tongue blade, lesions are difficult to remove and leave behind an inflamed base that may be painful and may bleed.

In neonates and infants, parents usually notice a white coating in the child's mouth. The child may have trouble feeding in severe cases. Candidal infection in the diaper area may accompany thrush. A thorough physical examination is important for patients with recurrent thrush infections and for older children.

**Erythematous candidosis**- these lesions consist of red areas of varying sizes that can occur on any part of the oral mucosa. If present on the tongue the lesions can be painful, fiery red and shiny with evidence of depapillation. This disease can be acute or chronic in nature.

**Hyperplastic candidosis (candidal leukoplakia)**- Usually an individual lesion on the oral mucosa of the cheek near the commissure, at the angles of the mouth, or on the surface of the tongue. They are chronic, discrete, raised lesions that may vary from a small, palpable, translucent, or whitish area to a large, dense, opaque plaque that is hard and rough to the touch. Homogeneous or speckled areas, which **do not rub off** (nodular lesions), can be seen. Speckled, red-white, areas account for 3-50% of the lesions and have a higher change of malignant transformation.

### Diagnosis

Herpes gingivostomatitis- culture for virus, serology, Tzanck test positive.

Candidosis- swab lesions for culture, scrap some of the lesions smear on slide and use periodic acid-Schiff (PAS), Gridley stain, or Gomori methenamine silver (GMS) stain to reveal the pseudohyphae of the yeast, biopsy hyperplastic conditions for histology. Remember pseudomembranous lesions are removable but hyperplastic lesions are not.

## **Treatment**

Herpes gingivostomatitis- usually is self-limiting and does not usually require treatment however if severe penciclovir or valacyclovir can be given. Treatment will not cure and recurrences can occur.

Candidosis- Fluconazole or Itraconazole

### **Angular Cheilitis**

(angular stomatitis) is an inflammation of the angles of the mouth.

**Etiology** Most common cause is *Candida albicans*. If yellow crusting appears then *Staphylococcus aureus* is likely to also be involved.

**Pathogenesis** Angular stomatitis describes erythema and maceration of the skin adjacent to the angle of the mouth. It is often seen in the elderly where it is predisposed to by sagging facial muscles and ill-fitting dentures, which produce a fold in the angle of the mouth. Angular stomatitis also may be seen in individuals with HIV infection.

Angular stomatitis commonly is an isolated initial sign of anemia or vitamin deficiency, such as vitamin B-12, and resolves when the underlying disease has been treated. Iron deficiency anemia and other vitamin deficiencies have been cited as other predisposing factors.

**Manifestations** Lesions affect the angles of the mouth; soreness, erythema, and fissuring are characteristic; diagnosis usually is associated with ill-fitting denture-induced stomatitis.

**Diagnosis** Usually done clinically however you can swab the lesion and culture the organisms.

**Treatment**- hydrocortisone 1%, clioquinol 3%

### **Oral Leukoplakia:**

The significant fact about oral leukoplakia is that a small percentage of these lesions show premalignant or malignant epithelial changes and a small percentage eventually could become malignant. Tobacco in any form (smoking, chewing) is thought to be associated with the development of some of these lesions. Factors like alcohol, trauma, *Candida albicans* infection and nutritional deficiencies may also contribute to the development of leukoplakia.

**Clinical** Usually affects middle-aged or older adults. Appears as a white patch that can not be rubbed off, typically in the cheek, sides of the tongue and floor of the mouth. Malignant transformation varies in different populations, nearly 5% are malignant at first biopsy and 5% develop into malignancies at a later stage.

### **Smoker's palate:**

Relatively common tobacco related white lesion seen in the palate of a pipe, cigar or a cigarette smoker. Unless the habit is particularly intense or the patient is a reverse smoker (burning end directed inside the mouth), risk for malignancy is quite low. The combination of tobacco smoke and heat combustion is believed to be important for this tissue change.

**Clinical** White opaque appearance of the palatal mucosa, sometimes showing red dots.