

Oral Medicine Lecture

Oral ulceration and vesiculobullous lesions

Many ulcerative or vesiculobullous disease of the mouth have a similar clinical appearance. The oral mucosa is thin, causing vesicles and bullae to break rapidly into ulcers; ulcers are easily traumatized from teeth and food, and they become secondarily infected by the oral flora. These factors may cause lesions that have a characteristic appearance on the skin to have a non specific appearance on the oral mucosa. Therefore, a careful and detailed history and clinical examination should be obtained to reach the diagnosis. The diagnosis of oral lesions requires knowledge of basic dermatology because many disorders occurring on the oral mucosa also affect the skin and many frequently terms used to describe the clinical appearance of the skin as well as the oral mucosa lesions are:



Macule



Papule

Macule: flat and well-demarcated lesion of any size, characterized by color change in contrast to the surrounding skin. It is generally caused by alteration of melanin pigment. A good example in the oral cavity is the melanotic macule

Papule: elevated, solid and circumscribed lesion, usually 1 cm or less in diameter. e.g. hyperplastic candidiasis often presents as yellow-white papules, papular form lichen planus.



Plaque



Erosions

Plaque: elevated, flat-topped, firm and superficial lesion, they are large papules.usually greater than 1 cm in diameter; may be coalesced papules.

Erosions. These are red lesions often caused by the rupture of vesicles or bullae or trauma to the mucosa and generally moist on the skin, eg. erosive form lichen planus, erosion from chemical, thermal or trauma irritation.



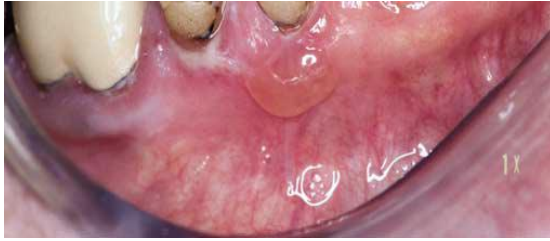
Nodules



Vesicle

Nodules. These lesions are present deeper in the dermis or mucosa. The lesions may also protrude above the skin or mucosa but are generally wider than they are high. A good example of an oral mucosal nodule is the irritation fibroma.

Vesicle: elevated, thin-walled lesion; filled with serous (clear) fluid, less than 1 cm in diameter. e.g.clinical eruptions of viral infections of skin and oral mucosa.



Bulla



Pustule

Bulla: elevated lesion filled with clear fluid, greater than 1 cm in diameter. e.g. pemphigus vulgaris.

Pustule: These are blisters containing purulent material.



Purpura



Ulcer

Purpura. These are reddish to purple bruises caused by blood from vessels leaking into the connective tissue. These lesions do not blanch when pressure is applied and are classified by size as petechiae (less than 0.5 cm) or ecchymoses. e.g. blood diseases (clotting disorders).

Ulcers. These are well-circumscribed, often depressed lesions with an epithelial defect that is covered by a fibrin clot, causing a yellow-white appearance. A common example is aphthous ulcers.

Classification of oral ulceration and vesiculobullous lesions

Three pieces of information in particular help the clinician rapidly categorize a patient's disease and simplify the diagnosis:

- a. Solitary (single) and multiple lesions
- b. Acute and chronic lesions
- c. Recurrent and non recurrent lesions

The Recurring Oral Ulcers

Aphthous Ulcers (also termed canker sores).

Recurring oral ulcers are among the most common problems seen by clinicians. The lesions are confined to the oral mucosa and begin with prodromal burning any time from 2 to 48 hours before an ulcer appears. During this initial period, a localized area of erythema develops. Within hours, a small white papule forms, ulcerates, and gradually enlarges over the next 48 to 72 hours. The individual lesions are round, symmetric, and shallow (similar to viral ulcers), but no tissue tags are present from ruptured vesicles. These ulcers occur periodically and heal completely between attacks. In the majority of cases, the individual ulcers last about 7-10 days, and ulceration episodes occur 3–6 times per year. Most appear on the non-keratinizing epithelial surfaces in the mouth (i.e. anywhere except the attached gingiva, the hard palate and the dorsum of the tongue), although the more severe forms, which are less common, may also involve keratinizing epithelial surfaces. Symptoms range from a minor nuisance to interfering with eating and drinking. The severe forms may be debilitating, even causing weight loss due to malnutrition.

the incidence ranges from 5 to 50% affecting about 20% of the general population to some degree. The onset is often during childhood or adolescence in the 2nd decade of life, and the condition usually lasts for several years before gradually disappearing. There is no cure, and treatments aim to manage pain, reduce healing time and reduce the frequency of episodes of ulceration.

RAS is classified according to clinical characteristics: into:

Minor aphthous ulceration

This is the most common type of aphthous stomatitis, accounting for about 80–85% of all cases. This subtype is termed minor aphthous ulceration (MiAU), or minor recurrent aphthous stomatitis (MiRAS). The lesions themselves may be referred to as minor aphthae or minor aphthous ulcers. These lesions are generally less than 10 mm in diameter (usually about 2–3 mm), and affect non-keratinized mucosal surfaces (i.e. the labial and buccal mucosa, lateral borders of the tongue and the floor of the mouth). Usually several ulcers appear at the same time, but single ulcers are possible. Healing usually takes seven to ten days and leaves no scar. Between episodes of ulceration, there is usually an ulcer-free period of variable length.

Major aphthous ulceration (Sutton disease, periadenitis mucosa necrotica recurrens),

This subtype makes up about 10% of all cases of aphthous stomatitis. It is termed major aphthous ulceration (MaAU) or major recurrent aphthous stomatitis (MaRAS). Major

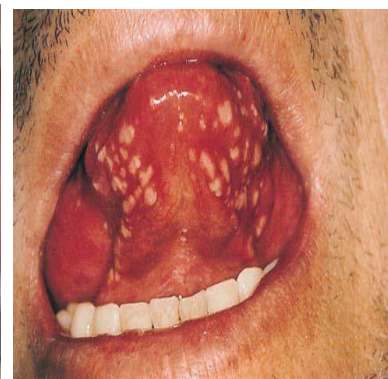
aphthous ulcers (major aphthae) are similar to minor aphthous ulcers, but are more than 10 mm in diameter and the ulceration is deeper. Because the lesions are larger, healing takes longer (about twenty to thirty days), and may leave scars. Each episode of ulceration usually produces a greater number of ulcers, and the time between attacks is less than seen in minor aphthous stomatitis. Major aphthous ulceration usually affects non keratinized mucosal surfaces, but less commonly keratinized mucosa may also be involved, such as the dorsum (top surface) of the tongue or the gingiva (gums). The soft palate or the fauces (back of the throat) may also be involved, the latter being part of the oropharynx rather than the oral cavity. Compared to minor aphthous ulceration, major aphthae tend to have an irregular outline.



Minor Aphthous



Major Aphthous



Herpetiform Aphthous

Herpetiform ulceration

Herpetiform ulcers (also termed stomatitis herpetiformis or herpes-like ulcerations) is a subtype of aphthous stomatitis so named because the lesions resemble a primary infection with herpes simplex virus (primary herpetic gingivostomatitis). However, herpetiform ulceration is not caused by herpes viruses. As with all types of aphthous stomatitis, it is not contagious. Unlike true herpetic ulcers, herpetiforme ulcers are not preceded by vesicles (small, fluid filled blisters). Herpetiforme ulcers are less than 1 mm in diameter and occur in variably sized crops up to one hundred at a time. Adjacent ulcers may merge to form larger, continuous areas of ulceration. Healing occurs within fifteen days without scarring. The ulceration may affect keratinized mucosal surfaces in addition to non keratinized. Herpetiform ulceration is often extremely painful, and the lesions recur more frequently than minor or major aphthous ulcers. Recurrence may be so frequent that ulceration is virtually continuous. It generally occurs in a slightly older age group than the other subtypes, and females are affected slightly more frequently than males.

Treatment

The vast majority of people with aphthous stomatitis have minor symptoms and do not require any specific therapy. The pain is often tolerable with simple dietary modification during an episode of ulceration such as avoiding spicy and acidic foods and beverages. Many different topical, analgesics /anesthetics / antiseptics and anti-inflammatory Orabase (often combined with cortisone such as triamcinolone

agents and systemic medications have been used for treatment. Intralesional injections of cortisone is indicated for chronic frequently erupting painful ulcer.

Behçet's Disease (Behçet's Syndrome)

Behçet's disease (BD) was initially described by the Turkish dermatologist Hulusi Behçet as a triad of symptoms including recurring oral ulcers, recurring genital ulcers, and eye involvement.

Nearly all patients present with some form of painful oral mucocutaneous ulcerations in the form of aphthous ulcers or non-scarring oral lesions.^[3] The oral lesions are similar to those found in inflammatory bowel disease and can be relapsing.^[3] Painful genital ulcerations usually develop around the anus, vulva, or scrotum and cause scarring in 75% of the patients. Additionally, patients may present with erythema nodosum, cutaneous pustular vasculitis.

A new set of diagnostic criteria was developed that includes recurrent oral ulceration occurring at least

three times in one 12-month period plus two of the following four manifestations:

1. Recurrent genital ulceration
2. Eye lesions, including uveitis or retinal vasculitis
3. Skin lesions, including erythema nodosum, pseudofolliculitis, papulopustular lesions, or acneiform nodules in postadolescent patients not receiving corticosteroids
4. A positive pathergy test, which is performed by placing a 20-gauge needle 5 mm into the skin of the forearm. The test is positive if an indurated papule or pustule greater than 2 mm in diameter forms within 48 hours.

The management of BD depends on the severity and the sites of involvement. Patients with sight-threatening eye involvement or central nervous system lesions require more aggressive therapy with drugs, with a higher potential for serious side effects. Azathioprine and other immunosuppressive drugs combined with prednisone have been shown to reduce ocular disease as well as oral and genital involvement.

Necrotizing Ulcerative Gingivitis (NUG) and Periodontitis (NUP):

These are acute ulcerative-inflammatory conditions associated with polymicrobial infection have strong associations with immune suppression (especially AIDS), debilitation, smoking, stress, poor oral hygiene, local trauma, and contaminated food supply. Diabetes may also be a risk factor. Microbes involved include *Treponema species*, fusospirochetal organisms are common in the periodontal tissues. NUG and NUP. Periodontitis may or may not be associated with fever and malaise, although submandibular lymphadenopathy is usually present

Oral Manifestations;

NUG has a rapid and acute onset. The first symptoms include excessive salivation, a metallic taste, and sensitivity of the gingiva. This rapidly develops into extremely painful and erythematous gingiva with scattered punched-out ulcerations, usually on the interdental papillae, although any part of the marginal gingiva may be affected. There is accompanying malodor, and there may be gingival bleeding. Because of the pain associated with the gingivitis, there is usually abundant build-up of dental plaque around the teeth because it may be too painful to perform effective oral hygiene. In patients in whom there is severe immunodeficiency or malnutrition, NUG may progress to noma. Noma; The overlying skin becomes discolored, and perforation of the skin is followed. The orofacial lesions are cone-shaped, with the base of the cone within the oral cavity and the tip at the skin aspect. There is sloughing of the oral mucosa followed by sequestration of the exposed, necrotic bone and teeth. Without treatment, the mortality rate is 70 to 90%

Definitive treatment of NUG and NUP consists of gentle débridement to remove as much of the debris and plaque as possible; this is best accomplished with topical anesthesia during the first few visits. The use of chlorhexidine digluconate mouthrinse led to resolution in >90% of cases. Patients with more extensive disease and/or systemic symptoms may require antibiotics active against gram-negative anaerobes. Interestingly, metronidazole, which has little activity against spirochetes, also is effective, suggesting that resolution can occur without treatment of the entire microbial complex. Once the acutely painful episodes have resolved, scaling and root planing to completely remove all residual plaque and calculus are indicated.

Solitary chronic non recurrent ulcers

Traumatic Injuries Causing Solitary Ulcerations

The most common cause of single ulcers on the oral mucosa is trauma. Mucosal ulcers may be caused by direct physical/mechanical, thermal, or chemical trauma to the mucosa. The dentist must reexamine

all patients with single ulcers for significant healing in 1 to 2 weeks; if healing is not evident in this time, a biopsy should be taken to rule out cancer .

Bite injuries, an example of direct physical or mechanical trauma. Traumatic injuries may also result from sharp teeth or cusps, malocclusion, ill-fitting dental prostheses, overzealous tooth brushing and flossing and self-injurious habits. Traumatic ulcer is usually occurs on the cheek or the lateral borders of the tongue due to sharp edges of the teeth. It is single deep base with rolled margins has the impression of the traumatic cusps. chronic ulcer with crater margins should be biopsied if did not disappear two weeks after the removal of the traumatic cause to exclude malignancy.

Thermal injuries include burns occur on the palate from ingesting hot foods and beverages (such as hot pizza or coffee).

Chemical trauma is caused by placing caustic substances directly on the mucosa. Mouthwashes or other oral care products with high alcoholic content, hydrogen peroxide, or phenols used too frequently or undiluted, aspirin can cause mucosal ulcerations.

TB ulcer

Tuberculous oral lesions are a relatively rare occurrence. Oral tuberculous lesions may be either primary or secondary in occurrence. Primary lesions are uncommon, seen in younger patients and present as single painless ulcer with regional lymph node enlargement. The secondary lesions are common, often associated with pulmonary disease, usually present as single, indurated, irregular or star shape with undermined margins mostly on the posterior dorsal tongue. painful ulcer covered by inflammatory exudates in patients of any age group but relatively more common in middle aged and elderly patients.

Diagnosis is by tuberculin test, Chest X ray and Lab investigation of the microbial swab of the sputum or base of the ulcer and stain with Ziehl–Neelsen stain, also known as the acid-fast stain.

treatment require systemic antibiotic therapy for the infection.



Traumatic ulcer

TB Ulcer (tongue)

Chancre (Syphilis) of upper lip

Syphilitic ulcer (chancre)

Primary syphilis is characterized by a chancre appearing between 10 and 90 days after infection. Syphilitic chancre is a solitary, painless, indurated, reddish ulcer, accompanied by regional lymphadenopathy, which is localized at the site of *Treponema pallidum* (TP) inoculation and usually resolves after approximately one month. It is commonly found in the genital area. Extra genital syphilitic chancre are, mainly involving the oral mucosa. solitary ulcer usually of the lip or, more rarely the tongue. The upper lip is more commonly affected than the lower in males, while the opposite occurs in females. The ulceration is usually deep, with a red, purple, or brown base and an irregular raised border. There is usually an accompanying cervical lymphadenopathy. The ulceration of primary syphilis may be confused with other solitary ulcerative disorders, most notably traumatic ulceration, squamous cell carcinoma, and non-Hodgkin's lymphoma.

The diagnosis of primary syphilis may be aided by detailed analysis of the sexual and/or social lifestyle. Dark field microscopy used but with less accuracy due to contamination. VDRL is mostly -ive at this stage.

Treatment require systemic therapy for the treatment of syphilis.

Recurrent Vesicular lesions of the oral mucosa:

Herpes Simplex Virus (HSV) Infection

infections above the waist are caused by HS V-1 and those below the waist by HS V-2, although with changing sexual practices, it is not uncommon to culture HS V-2 from oral lesions and vice versa.

The primary infection, which occurs on initial contact with the virus, is acquired by inoculation

of the mucosa, skin, and eye with infected secretions. The virus then travels along the sensory nerve axons and establishes chronic, latent infection in the sensory ganglion (such as the trigeminal ganglion). Extraneuronal latency (ie, HSV remaining latent in cells other than neurons such as the epithelium) may play a role in recurrent lesions of the lips. Recurrent HS V results when HSV-1 reactivates at latent sites and travels centripetally to the mucosa or the skin, where it is directly cytopathic to epithelial cells, causing recrudescence HSV infection in the form of localized vesicles or ulcers.⁶ The most common sites of infection are the oral and genital mucosa and the eye. HS V infection of the cornea (keratitis) is a major cause of blindness in the world. HS V-1 or -2 may cause herpes whitlow, an infection of the fingers when virus is inoculated into the fingers through a break in the skin. This was a common occupational hazard (including within the dental profession) before the widespread use of gloves.



Herpes Whitlow



Primary Herpetic Gingivostomatitis

Primary Herpetic Gingivostomatitis: Most cases of primary HS V-1 infections are subclinical and generally occur in children and teenagers. There is a 1- to 3-day viral prodrome of fever, loss of appetite, malaise, and myalgia that may also be accompanied by headache and nausea. Oral pain leads to poor oral intake, and patients may require hospitalization for hydration. The disease is self-limiting in otherwise normal patients and resolves within 10 to 14 days, typical for a viral illness.

The oral lesions, within a few days of the prodrome, erythema and clusters of vesicles and/or ulcers appear on the keratinized mucosa of the hard palate, attached gingiva and dorsum of the tongue, and the nonkeratinized mucosa of the buccal and labial mucosa, ventral tongue, and soft palate. Vesicles break down to form ulcers that are usually 1 to 5 mm and coalesce to form larger ulcers with scalloped borders and marked surrounding erythema. The gingiva is often fiery red, and the mouth is extremely painful, causing difficulty with eating. Pharyngitis causes swallowing difficulties. Primary HS V infection in adults follows a similar pattern.

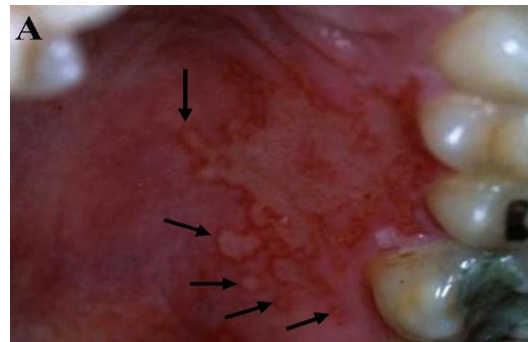
Treatment, the disease is self-limiting, the child may need supportive treatment such as antipyretic analgesic to relieve pain and antibiotic to prevent secondary infections.

The use of acyclovir at 15 mg/kg five times a day in children reduces the duration of fever, reduces HSV shedding, halts the progress of lesions, improves oral intake, and reduces the incidence of hospital admissions.

Recrudescence Oral HSV Infection: Reactivation of HSV may lead to asymptomatic shedding of HSV, in the saliva and oral secretions, an important risk factor for transmission; it may also cause ulcers to form. A symptomatic shedding of HSV is not associated with systemic signs and symptoms and occurs in 8 to 10% of patients following dental treatment. The term *recrudescence HSV* should be used to refer to the actual ulcerations caused by reactivated virus. Fever, ultraviolet radiation, trauma, stress, and menstruation are important triggers for reactivation of HSV. Recrudescence HSV on the lips is called recurrent herpes labialis (RHL) and occurs in 20 to 40% of the young adult population. These are associated with a prodrome of itching, tingling, or burning approximately 50% of the time, followed in succession by the appearance of papules, vesicles, ulcers, crusting, and then resolution of lesions. Pain generally is present only within the first 2 days. There is a suggestion that patients who do not experience a prodrome develop lesions from extraneural latent HSV within the epithelium and these lesions are less responsive to topical therapy.



Recurrent herpes labialis (RHL)



Recurrent intraoral HSV (RIH) infection

Intraoral recrudescence HSV in the immunocompetent host occurs chiefly on the keratinized mucosa of the hard palate, attached gingiva, and dorsum of the tongue. Such lesions are called recurrent intraoral HSV (RIH) infection. They present as 1 to 5 mm single or clustered painful ulcers with a bright erythematous border. One common presentation is the complaint of pain in the gingiva 1 to 2 days after a scaling and prophylaxis or other dental treatment.

Lesions appear as 1 to 5 mm painful vesicles but more often ulcers on the marginal gingiva.

Laboratory Diagnosis

- HSV isolation by cell culture
- polymerase chain reaction (PCR) to detect HSV antigen three to four times more often than culture

- HSV can be identified from scrapings from the base of lesions (especially vesicles) smeared onto glass slides. These can be stained with Wright, Giemsa (Tzanck preparation), or Papanicolaou stain.

Treatment is supportive by topical anesthetics, antiseptic mouth washes to relieve pain.

Varicella Zoster (VZV) infection

Primary infection with VZV, an α -herpesvirus, leads to varicella (chicken pox). As with all herpesviruses, the virus then becomes latent, usually in the dorsal root ganglia or ganglia of the cranial nerves. Reactivation produces herpes zoster infection (HZI), commonly called shingles. this virus is cytopathic to the epithelial cells of the skin and mucosa, causing blisters and ulcers. Transmission is usually by the respiratory route, with an incubation period of 2 to 3 weeks.

HZI of the skin (shingles) is more common in adults and starts with a prodrome of deep, aching, or burning pain. There is usually little to no fever or lymphadenopathy. This is followed within 2 to 4 days by the appearance of crops of vesicles in a dermatomal or “zosteriform” pattern. This pattern describes the unilateral, linear, and clustered distribution of the vesicles, ulcers, and scabs in a dermatome supplied by one nerve. Thoracic/lumbar dermatomes are the most frequently involved, followed by the craniofacial area. Lesions heal within 2 to 4 weeks, often with scarring and hypopigmentation.

the ophthalmic division of the trigeminal nerve is the cranial nerve most often affected. Midface, with upper and lower lips involvement, patients experience a prodrome of pain, burning, and tenderness, usually on the palate on one side. This is followed several days later by the appearance of painful, clustered 1 to 5 mm ulcers (vesicles break down quickly) on the hard palate or even buccal gingiva, in a distinctive unilateral distribution. An uncommon complication of HZI involving the geniculate ganglion is *Ramsay Hunt syndrome*. Patients develop Bells palsy, vesicles of the external ear, and loss of taste sensation in the anterior two-thirds of the tongue.

treatment is by anti viral drug (Valacyclovir or famciclovir) for 7 days is effective in treating HZI and should be started within 72 hours of disease onset. These drugs also reduce the incidence of postherpetic neuralgia when compared with acyclovir.



VZ infection of Midface



VZV infection of Palate

Coxsackievirus (CV) Infection

Herpangina

The word *herpangina* derives from *herpes*, meaning “vesicular eruption,” and *angina*, meaning “inflammation of the throat.” Coxsackie V type A are the most common viruses isolated from this disease. But CV type B echoviruses (EV) have also been identified in this condition. As with all CV infections, children under 10 are usually afflicted and outbreaks usually occur in epidemics in summer. Patients develop fever, headache, and myalgia that usually last only 1 to 3 days.

The first oral symptoms of herpangina are sore throat and pain on swallowing. There may be erythema of the oropharynx, soft palate, and tonsillar pillars. Small vesicles form, but these rapidly break down to 2 to 4 mm ulcers. These persist for 5 to 10 days. Lymphonodular pharyngitis is considered a variant of herpangina and is associated with CV type A. Patients report a sore throat, but rather than presenting with vesicles that break down to ulcers, patients develop diffuse small nodules in the oropharynx.

hand-foot-and-mouth Disease (HFMD)

HFMD disease usually afflicts children younger than 10 years in summer. Patients have a low-grade fever and sore mouth; 75 to 100% of patients have a skin rash, especially on the hands and feet (dorsa, palms and soles) and 30% on the buttocks. The rash is first red and macular and then becomes vesicular.

The patients are febrile and complain of a sore mouth and throat. Lesions orally begin as erythematous macules that become vesicles and quickly break down to ulcers. Lesions are usually located on the tongue, hard and soft palate, and buccal mucosa but can present on any oral mucosal surface.

Treatment of the viral infections is supportive, however in severe form in immunosuppressed patients, antiviral medications may be required.

Bullous lesions of the oral mucosa;

Erythema multiforme;

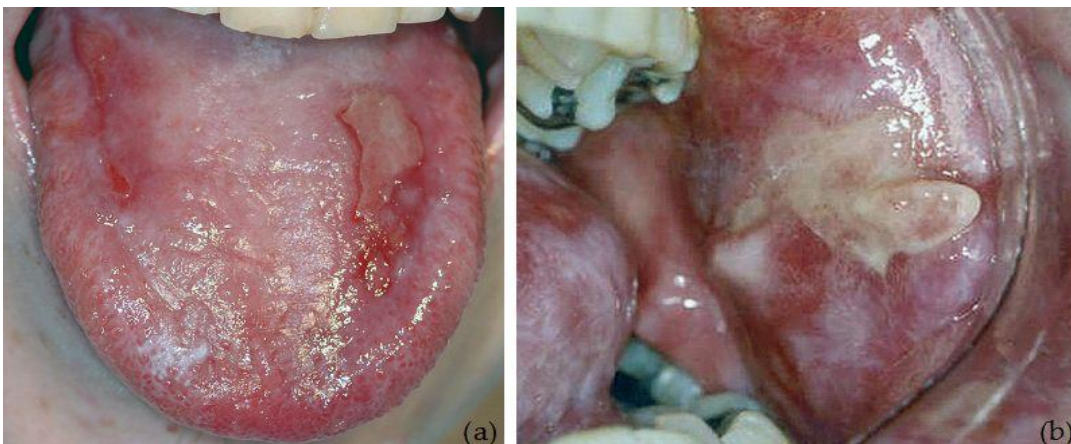
is an idiopathic disease that involves an immunologic abnormality. It may be triggered by infection, especially with herpes simplex virus, or drugs, such as antibiotics. It is characterized by the acute onset of blisters and ulcers of skin and oral mucosa. The appearance of the skin lesions is variable. “Target” or “iris” lesions of the skin are characteristic but not present in all cases, and consist of a blister surrounded by erythematous rings. Oral mucosal blisters and ulcers are present in multiple locations and are painful. Hemorrhagic crusting of the lips is often present. Fever, malaise, and pharyngitis may precede the lesions.

Stevens Johnson syndrome; is a severe form of erythema multiforme and demonstrates conjunctivitis and genital ulcers in addition to mucocutaneous lesions. Topical and/or systemic corticosteroids may be useful in treatment. Offending drugs should be discontinued. The prognosis of the disease is good, however fatal if not treated (topical and/or systemic steroid).



bullous lichen planus

Ulcerative lesions are the most disabling form of oral lichen planus (OLP). Clinically, the fibrin-coated ulcers are surrounded by an erythematous zone frequently associated with radiating white striae. This appearance may reflect a gradient of the intensity of subepithelial inflammation that is most prominent at the center of the lesion. When this type of OLP is present in the buccal mucosa or in the palate, striae are frequently seen in the periphery. the affected patient complains of a burning pain in conjunction with spicy and sore food intake. trauma may aggravate the disease, which is referred to as a *Koebner phenomenon*. The most frequent extraoral mucosal site involved is the genital mucosa. Genital lichen planus has also been reported in males, but the association with OLP is not as frequent as for women. OLP requires a histopathologic examination in order to arrive at a correct diagnosis. Treatment is by systemic and topical steroid depend on the severity or progression of the disease.



Pemphigus vulgaris (PV);

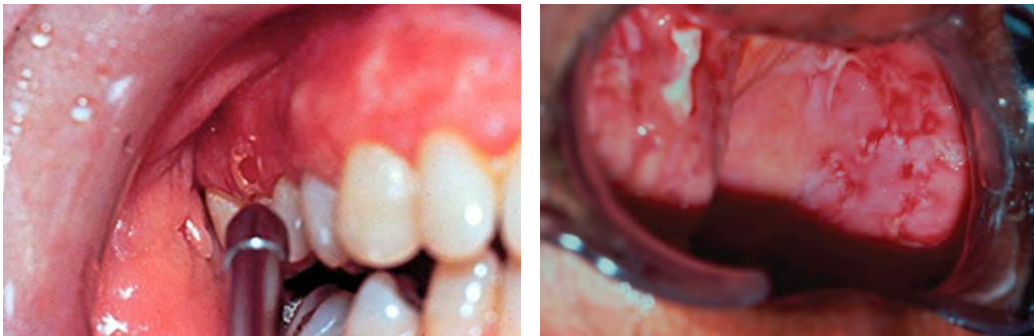
is a mucocutaneous lesion of chronic nature. The classic lesion of pemphigus is a thin-walled bulla arising on otherwise normal skin or mucosa. The bulla rapidly breaks but continues to extend peripherally, eventually leaving large areas denuded of skin. In patients with PV, the bulla enlarges by extension to an apparently normal surface. Another characteristic sign of the disease is that pressure to an apparently normal area results in the formation of a new lesion. This phenomenon, called the Nikolsky sign, results from the upper layer of the skin pulling away from the basal layer. The Nikolsky

sign is most frequently associated with pemphigus but may also occur in other blistering disorders. Pemphigus vulgaris is a painful autoimmune disease in which the patient forms antibodies to a component of desmosomes located in the stratified squamous epithelium. This results in loss of adherence of epithelial cells and the formation of intraepithelial blisters. The blisters are fragile and quickly rupture forming painful ulcers or erosions which heal slowly. Large areas of skin and mucosa can be involved and may cause serious problems with infection.

Eighty to 90% of patients with PV develop oral lesions sometime during the course of the disease, and in 60% of cases, the oral lesions are the first sign. The oral lesions may begin as the classic bulla rapidly breaks leaving a denuded base. The lesions start on the buccal mucosa, often in areas of trauma along the occlusal plane. The palate and gingiva are other common sites of involvement. It is common for the oral lesions to be present for months before the skin lesions appear. These lesions must be biopsied to distinguish PV from subepithelial blistering diseases such as mucous membrane pemphigoid and erosive lichen planus. Immunofluorescence studies of biopsy material are necessary to make a definitive diagnosis of pemphigus.

An important aspect of patient management is early diagnosis, when lower doses of medication can be used for shorter periods of time to control the disease. Management varies according to several factors, including the severity of the disease and the speed at which the disease progresses.

The disease is treated aggressively with corticosteroids or other immunosuppressive drugs. Without treatment (systemic and/or topical steroid) the disease can be fatal because of septicaemia and dehydration.



Nikolsky Sign (peeling of epithelium by air pressure) Pemphigus vulgaris (irregular ulcers)

Mucous Membrane Pemphigoid (MMP) (Cicatricial Pemphigoid)

MMP is a chronic autoimmune subepithelial disease that primarily affects the mucous membranes of patients over the age of 50, resulting in mucosal blistering, ulceration, and subsequent scarring. The disease occurs twice as frequently in women. The primary lesion of MMP occurs when autoantibodies directed against proteins in the basement membrane cause a subepithelial split and subsequent vesicle formation. The subepithelial lesions of MMP may involve any mucosal surface, but they most frequently involve the

oral mucosa. The conjunctiva is the second most common site of involvement and can lead to scarring and adhesions. corneal damage is common, and progressive scarring leads to blindness in close to 15% of patients. Lesions may also affect the genital mucosa, causing pain and sexual dysfunction. Oral lesions occur in over 90% of patients with MMP. Desquamative gingivitis is the most common manifestation and may be the only manifestation of the disease appearing bright red. Since these desquamative lesions resemble the lesions of erosive lichen planus and pemphigus, all cases of desquamative gingivitis should be biopsied and studied with both routine histology and Direct Immunofluorescence to determine the correct diagnosis. Lesions may present as intact vesicles of the gingival or other mucosal surfaces, but more frequently they appear as nonspecific-appearing erosions. The erosions typically spread more slowly than pemphigus lesions and are more self-limiting.

Management of MMP depends on the severity of symptoms and site of involvement. When the lesions are confined to the oral mucosa, use of systemic corticosteroids should only be considered for short periods of time for severe outbreaks until less toxic forms of therapy can be substituted. Unlike pemphigus, MMP is rarely a fatal disease, and long-term use of systemic steroids for oral lesion involvement alone is seldom indicated. Patients with mild oral disease should be treated with topical and intralesional steroids. Desquamative gingivitis can often be managed with topical steroids in a soft dental splint



subepithelial bulla of BMMP



Desquamative gingivitis of BMMP

Bullous Pemphigoid

Bp, which is the most common of the subepithelial blistering. Diseases, occurs chiefly in adults over the age of 60 years. It is self-limited and may last from a few months to 5 years without treatment. The oral lesions are clinically and histologically indistinguishable from oral lesions of mucous membrane pemphigoid, but early remission of BP is more common Diagnosis is by routine histology and DIF of a biopsy specimen.

Treatment of autoimmune diseases in general, is by immune suppressant medications such as systemic and topical steroids.