ORAL MEDICINE

INFECTIOUS DISEASES OF ORAL MUCOSA

A- VIRAL INFECTIONS

1- Primary Herpetic Stomatitis

Primary infection is caused by *Herpes simplex* virus, usually type I, which in the non-immune person (not exposed to the virus previously), can cause an acute vesiculating stomatitis. However, most primary infections are subclinical.

The two major types, HSV1 and HSV2, can be distinguished serologically. Classically, HSV1 causes a majority of cases of oral and pharyngeal infection, meningoencephalitis, and dermatitis above the waist; HSV2 is implicated in most genital infections.

Although this distinction applies to a majority of cases, changing sexual habits are making that distinction less important. Both types can cause primary or recurrent infection of either the oral or the genital area, and both may cause recurrent disease at either site.

Clinical features

The early lesions are vesicles which can affect any part of the oral mucosa, the hard palate and dorsum of the tongue are favored sites.

The vesicles are dome-shaped and usually 2-3 mm in diameter. Rupture of vesicles leaves circular, sharply defined, shallow ulcers with yellowish or grayish floors and red margins.

The ulcers are painful and may interfere with eating. The gingival margins are frequently swollen and red, particularly in children, and the regional lymph nodes are enlarged and tender.

There is often fever and systemic upset, sometimes severe, particularly in adults. Oral lesions usually resolve within a week to ten days, but malaise can persist so long that an adult may not recover fully for several weeks.

Diagnosis and treatment

The clinical picture is usually distinctive. A smear showing virus-damaged cells is additional diagnostic evidence. A rising titre of antibodies reaching a peak after 2-3 weeks provides absolute but retrospective confirmation of the diagnosis.

Acyclovir (200-400 mg(five times daily) , for seventh days) is a potent anti-herpetic drug and is life-saving for potentially lethal herpetic encephalitis or disseminated infection. Acyclovir suspension used as a rinse and then swallowed should accelerate healing of severe herpetic stomatitis if used sufficiently early.

Bed rest, fluids and soft diet may sometimes be required, local anaesthetic before meal helped children to eat the foods. Systemic non-

steroidal anti-inflammatory drug needed to control the fever and pain and reduce the exacerbations of inflammation of this disease. Metronidazole helped to control the secondary infections such as the gingivitis.

2- Herpes Labialis

After the primary infection, the latent virus can be reactivated in 20-30% of patients to cause cold sores (fever blisters).

Triggering factors include the common cold and other febrile infections, exposure to strong sunshine, menstruation or, occasionally, emotional upsets or local irritation, such as dental treatment. Neutralizing antibodies produced in response to the primary infection are not protective.

Clinically, changes follow a consistent course with prodromal paraesthesia or burning sensations, then erythema at the site of attack. Vesicles form after an hour or two, usually in clusters along the mucocutaneous junction of the lips, but can extend onto the adjacent skin. The vesicles enlarge, coalesce and weep exudates. After two or three days they rupture and crust over but new vesicles frequently appear for a day or two only to scab over and finally heal, usually without scarring.

The whole cycle may take up to 10 days. Secondary bacterial infection may induce an impetiginous lesion which sometimes leaves scars.

Treatment

In view of the rapidity of the viral damage to the tissues, treatment must start as soon as the premonitory sensations are felt. Acyclovir cream is available without prescription and may be effective if applied at this time.

This is possible because the course of the disease is consistent and patients can recognize the prodromal symptoms before tissue damage has started. However, pencyclovir applied 2-hourly is more effective.

3- Herpetic Whitlow

Both primary and secondary herpetic infections are contagious and cause herpetic cross-infections called *Herpetic whitlow* which is a recognized uncommon hazard to dental surgeons and their assistants. Herpetic whitlows, in turn, can affect patients and have led to outbreaks of infection in hospitals and among patients in dental clinics. Now that gloves are universally worn when giving dental treatment, such cross-infections should no longer happen.

In immunodeficient patients, such infections can be dangerous but acyclovir has dramatically improved the prognosis in such cases and may be given on suspicion. Mothers applied antiherpetic drugs to children's lesions should wear gloves.

4-Coxsackievirus Infections

Coxsackieviruses are ribonucleic acid (RNA) enteroviruses and are named for the town in upper New York State where they were first discovered. Coxsackieviruses have been separated into two groups, A and B. There are 24 known types of coxsackievirus group A and 6 types of coxsackievirus group B.

These viruses cause hepatitis, meningitis, myocarditis, pericarditis, and acute respiratory disease. Three clinical types of infection of the oral region that have been described are usually caused by group A coxsackieviruses: **1-herpangina**, 2-**handfoot- and-mouth disease**, and 3-**acute lymphonodular pharyngitis.**

Types of coxsackievirus A have also been described as causing a rare mumpslike form of parotitis.

HERPANGINA

Unlike herpes simplex infections, which occur at a constant rate, herpangina frequently occurs in epidemics that have their highest incidence from June to October. The majority of cases affect young children ages 3 through 10, but infection of adolescents and adults is not uncommon.

Clinical Manifestations. After a 2- to 10-day incubation period, the infection begins with generalized symptoms of fever, chills, and anorexia. The fever and other symptoms are generally milder than those experienced with

primary HSV infection. The patient complains of sore throat, dysphagia, and occasionally sore mouth. Lesions start as punctate macules, which quickly evolve into papules and vesicles involving posterior pharynx, tonsils, faucial pillars, and soft palate.

Lesions are found less frequently on the buccal mucosa, tongue, and hard palate . Within 24 to 48 hours, the vesicles rupture, forming small 1 to 2 mm ulcers. The disease is usually mild and heals without treatment in 1 week.

Herpangina may be clinically distinguished from primary HSV infection by several criteria:

1. Herpangina occurs in epidemics; HSV infections do not.

2. Herpangina tends to be milder than HSV infection.

3. Lesions of herpangina occur on the pharynx and posterior portions of the oral mucosa, whereas HSV primarily affects the anterior portion of the mouth.

4. Herpangina does not cause a generalized acute gingivitis like that associated with primary HSV infection.

5. Lesions of herpangina tend to be smaller than those of HSV.

Treatment. Herpangina is a self-limiting disease, and treatment is supportive, including proper hydration and topical anesthesia when eating or swallowing is difficult. Specific antiviral therapy is not available.

Hand-Foot-and-Mouth Disease

This common, mild viral infection which often causes minor epidemics among school children, is characterized by ulceration of the mouth and a vesicular rash on the extremities. Hand-foot-and-mouth disease is usually caused by strains of Coxsakie A virus. It is highly infectious, frequently spreads through a classroom in schools and may also infect a teacher or parent. The incubation period is probably between 3-10 days.

Clinical features

The rash consists of vesicules, sometimes deep-seated, or occasionally bullae, mainly seen around the base of fingers or toes, but any part of the limbs may be affected. The rash is often the main feature and such patients are unlikely to be seen by dentists. In some outbreaks either the mouth or the extremities alone may be affected.

Regional lymph nodes are not usually enlarged and systemic upset is typically mild or absent.

Lymphonodular pharyngitis is considered a variant of herpangina and is associated with CVA10. Patients report a sore throat, but rather than

presenting with vesicles that break down to ulcers, patients develop diffuse small nodules in the oropharynx.

Treatment

CV infections are self-limiting (unless complications arise or the patient is immunocompromised), and management is directed toward control of fever and mouth pain, supportive care, and limiting contact with others to prevent spread of the infection. Effective antiviral agents for CV are not available.

5- Herpes Zoster (Trigeminal area)

Zoster (shingles) is characterized by pain, a vesicular rash and stomatitis. The varicella zoster virus caused chickenpox in the non-immune persons (mainly children), while reactivation of the latent virus causes zoster (shingles), mainly in the elderly.

Unlike herpes labialis, repeated recurrences of zoster are very rare, occasionally, there is an underlying immunodeficiency.

Herpes zoster is a hazard in organ transplant patients and can be an early complication of some tumours, particularly Hodgkin's disease, and increased in AIDS cases and potentially lethal.

Clinical features

Herpes zoster usually affects adults of middle age or over but occasionally attacks even children. The first signs are pain, irritation and tenderness in the dermatome (skin and mucosa) corresponding to the affected ganglion.

Vesicles, often confluent, form on one side of the face and in the mouth up to the midline. The regional lymph nodes are enlarged and tender. The acute phase usually lasts about a week. Pain continues until the lesions crust over and start to heal, but secondary infection may cause suppuration and scarring of the skin.

Malaise and fever are usually associated. Patients are sometimes unable to distinguish the pain of trigeminal zoster from severe toothache,

Management

Herpes zoster is an uncommon cause of stomatitis, but readily recognizable. According to the severity of attack, oral acyclovir (5 mg/kg three times daily for 7 days) should be given at the earliest possible moment, together with analgesics. For immunocompromised patients this dose of acyclovir is doubled.

The addition of oral corticosteroids such as prednisolone may accelerate relief of pain and healing. In immunodeficient patients, intravenous acyclovir is required and may also be justified for the elderly in whom this infection is debilitating.

Post-herpetic neuralgia mainly affects the elderly and is difficult to relieve, and the pain is more variable in character and severity than trigeminal neuralgia. It is typically persistent rather than paroxysmal. Post-herpetic neuralgia is remarkably being resistant to treatment. Nerve or root resection are ineffective and the response to drugs of any type, including carbamazepine (tegretol), is poor.

Application of transcutaneous electrical nerve stimulation (TENS) to the affected area by the patient himself for several times a day, is sometimes effective, by persistent stimulation the sensory pathways, so may prevent perception of pain centrally.

Ramsay Hunt syndrome

Ramsay Hunt syndrome is reactivated zoster infection in the facial nerve. Virus is latent in the geniculate ganglion, which houses both sensory and motor fibers. On reactivation, patients develop facial paralysis, loss of taste on one side of the anterior tongue and vesicles on the tongue, hard palate and in the external auditory canal. It must be differentiated from Bell's palsy. Treatment is as for zoster of the trigeminal nerve, but the chances of full recovery are lower than for Bell's palsy.

6- Cytomegalovirus-Associated Ulceration

Cytomegalovirus (CMV) is a member of the herpes virus group. Up to 80% of adults show serological evidence of CMV infection without

clinical effects but it is a common complication of immunodeficiency. Particularly AIDS.

In the latter it can be life-threatening. Oral ulcers in which CMV has been identified are sometimes clinically indistinguishable from recurrent aphthae, other have raised, minimally rolled borders. Generally, the ulcers are large, shallow and single, and affect either the masticatory or non-masticatory mucosa. Sometimes oral ulcers are associated with disseminated CMV infection.

The virus present in oral lesions may merely be a passenger, but their causative role is suggested by reports of response to gancyclovir.

B- BACTERIAL INFECTIONS

1- Tuberculosis

Oral tuberculosis is rare and a complication of pulmonary disease with infected sputum. Those with HIV infection are an important group of victims, but *oral tuberculosis is occasionally seen in immune-competent persons* who are usually elderly men with pulmonary infection that has progressed unrecognized or who have neglected treatment.

The typical oral lesion is an ulcer on the mid-dorsum of the tongue, lips or other parts of the mouth are infrequently affected. The ulcer is typically *angular*, with *over-hanging edges* and a *pale floor*, but can be *ragged* and *irregular*. It is *painless* in its early stages and *regional lymph nodes* are usually not *affected.*

Management

Diagnosis is confirmed by biopsy, chest radiography and a specimen of sputum. Mycobacterial infection is confirmed by culture or PCR (polymerize chain reaction).

Oral lesions subside rapidly if vigorous multidrug antibiotics is given for the pulmonary infection. *No local treatment is needed.*

2- Syphilis

Congenital syphilis is syphilis present in uterus and at birth, and occurs when a child is born to a mother with secondary syphilis. Some infants with congenital syphilis have symptoms at birth, but most develop symptoms later.

Blunted upper incisor teeth known as Hutchinson's teeth , mulberry molars, saddle nose (collapse of the bony part of nose) and hard palate defect all are sign that could been seen in congenital syphilis.

Acquired Syphilis:

Oral lesions have recently been reported in Britain but some may pass unrecognized. There are three types of syphilis which are:-

I. Primary syphilis

An oral *chancre* appears 3-4 weeks after infection and may form on the *lip,* tip of the *tongue* or rarely other oral sites. It consists initially of a firm nodule about a centimeter across. The surface breaks down after a few days, leaving a rounded ulcer with raised indurated edges. This may resemble a *carcinoma*, particularly if on the *lip*. A chancre is typically painless but regional lymph nodes are enlarged, rubbery and separate. *Serological reactions are negative at first*, so the diagnosis depends on finding of *Treponema pallidum* by dark-ground illumination of a smear from a chancre.

II. Secondary syphilis

The secondary stage develops 1-4 months after infection. It typically causes mild fever with malaise, headache, sore throat, and generalized lymphadenopathy, soon followed by a rash and stomatitis.

Oral lesions, which rarely appear without the rash mainly affect the tonsils, lateral borders of the tongue, and lips. They are usually flat ulcers covered by grayish membrane and may be irregularly linear (*snail's track ulcers*) or coalesce to form well-defined rounded area (*mucous patches*). Diagnosis is performed by serological reactions which are *positive* at this stage.

Condylomata lata are raised mucous patches that resemble large flat papilloma may also see in this stage.

III. Tertiary syphilis

Late stage syphilis develops in many patients about three or more years after infection. The onset is insidious and during the latent period the patient may appear well. A characteristic lesion is the *gumma*, which clinically may affect the *palate, tongue, and tonsils*. The gumma can vary from one to several inches in diameter. It begins as a swelling, sometimes with a yellowish centre which undergoes necrosis, leaving a painless indolent deep ulcer. The ulcer is rounded, with soft, punched out edges. The floor is depressed and pale in appearance. It eventually heals with severe *scarring* which may distort the *soft palate* or *tongue,* and the complication are perforate the *hard palate* or *destroy the uvula*.

Management

Antibiotics, particularly penicillin, are the mainstay of treatment, but tetracycline and erythromycin are also effective. Treatment should be by a specialist and must be continued until non-specific serological reactions are persistently negative.