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ORAL MANIFESTATIONS OF MICROBIAL DISEASES

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INTRODUCTION

THE ORAL MUCOSA and alveolar bone respond to a wide variety of generalised diseases, including viral, bacterial and mycotic infections. In the past decade, a considerable body of new ideas about oral manifestations of microbial diseases has emerged. This paper summarises the major conditions which might produce local signs and symptoms in the oral cavity and should be of interest to general practitioners and specialists alike.

VIRAL INFECTIONS

These may produce a characteristic clinical picture providing an easy or spot-diagnosis or may require histopathological and laboratory aids to arrive at the correct diagnosis. The virus may be confined to the oral mucosa exclusively, as in Herpes simplex infections, or may involve the oral mucosa as part of a widespread involvement of skin and other mucous membranes, as in chicken-pox or measles. The typical lesion produced, in the oral cavity, by viruses as an erythema and a vesicular eruption, and it is on a knowledge of the extent and distribution of these lesions that the clinician arrives at a correct diagnosis. Cases that still defy diagnosis may even require elaborate investigations like viral and immunological studies. The important viral infections are summarised below.

Localised viral infections

Herpes simplex infections – These are caused by Herpes simplex I virus and usually occur in older children and young adults exposed to the virus after the protection conferred by maternal antibodies has waned off. There is a prodormal phase lasting 24 to 48 hours. In primary herpetic infections, there is initially an acute gingivitis, and the gums are red and swollen but do not undergo necrosis. The tongue shows non-specific coating probably resulting from the associated pyrexia. Scattered vesicles may appear in any area of the oral cavity and rarely do they fuse to produce bullae. They rupture after 24 hours leaving shallow ulcers which have no zone of erythema surrounding them, and heal after 12 to 18 days without any scarring. The diagnosis is essentially clinical and rarely is cytological study required. Characteristic cytological features seen are the presence of giant-cells with large, hyperchromatic nuclei displaying nuclear inclusion or elementary bodies. Herpes simplex virus is notoriously known to produce recurrent infections which manifest clinically as Herpes labialis or "cold sores". The virus resides latent in the oral epithelium and may be activated by such precipitating factors as fever, trauma, psychological tension, exposure to sunlight or even prolonged kissing. Groups of vesicles surrounded by a zone of erythema and oedema appear beyond the vermillion border of the lips, producing intense pain, irritation and pruritis. The lesions usually rupture after 24 to 48 hours leaving behind shallow ulcers which generally heal after 2 to 10 days.

Herpangina – This condition is caused by Coxsackie Group A, Type IV virus. Typically, there is an intense inflammation of the oropharynx and posterior areas of the oral cavity like the soft palate, tonsillar area, and rarely the posterior third of the tongue. Small vesicles may appear but may not become apparent due to the intense erythema. Recurrent

herpangina is unusual and is seen in patients undergoing treatment with immuno-suppressive drugs like corticosteroids.

Generalised viral infections

Hand-foot-and mouth disease – This is a rare condition produced by Coxsackie Group A (A-5, A-10, A-16) virus. There is patchy erythema of all areas of the oral mucosa, but the palate, oropharynx and gingivae are spared. A vesicular eruption appears later and again small ulcers result from rupture of these lesions. The diagnosis becomes apparent when the characteristic vesiculo-papular lesions with purpuric borders appears on the palms, soles, heels, knees and legs.

Chicken-pox (varicella) – This common condition is produced by primary exposure to varicella-zoster virus. Oral lesions accompany or may rarely preceed the more pronounced skin eruptions. There may be a generalised erythema in addition to vesicles which usually rupture producing ulcers which heal in 10–14 days. In severe cases, bullous lesions and large areas of ulceration can occur.

Herpes zoster (Shingles) – This condition is produced by recurrent infection with varicella-zoster virus. If the trigeminal ganglion is involved, a unilateral, linear, vesicular eruption appears on the buccal mucosa, gingiva, palate or tongue. The ulcers secondarily produced heal without scarring. In some patients, post-herpetic neuralgic pain may be an unpleasant sequealae. Herpes zoster affecting the geniculate ganglion of the facial nerve (Ramsay-Hunt Syndrome) produces vesicular eruptions in the anterior two-thirds of the tongue or in the ear accompanied by a facial palsy.

Measles (Rubeola) – Twenty-four hours prior to the appearance of the skin rash, there is an intense erythema of the oral mucosa, and this is the significant oral manifestation of measles (Shklar and McCarthy, 1976). The so-called Koplik's spots are rarely seen and are supposed to be white spots on a background of inflammed buccal mucosa.

Infectious Mononucleosis – This is characterised by the presence of a low-grade fever, fatigue, lymphadenopathy and a sore-throat. The oropharynx is inflammed and an exudate with scattered superficial erosions may be present. In severe cases, extensive necrosis and sloughing occurs. This condition can produce a false-positive serological test for syphilis.

Mumps (Epidemic Parotitis) – This viral infection generally involves the parotid gland unilaterally or bilaterally, and only rarely are the sublingual and submandibular glands affected. The patient may complain of an unusually dry mouth (xerostomia) due to diminished production of saliva, and on examination, the papilla at the orifice of the parotid duct, in the vestibule opposite the crown of the upper second molar tooth, may be intensely inflammed.

Vaccination (Vaccinia) – After successful vaccination against smallpox the child may rub the lips or cheeks against the vaccination site, thus secondarily innoculating the virus in these areas. A large raised area with a necrotic centre, surrounded by erythema and oedema appears. Though alarming in appearance, the lesion heals within a few days. Smallpox (Variola) itself produces insignificant oral involvement, in the form of vesicles and ulcers, compared to the extensive cutaneous involvement.

BACTERIAL INFECTIONS

The oral mucosa is relatively resistant to entry by bacteria. There is a protective mechanism provided by lymphocytes and plasma cells present in the lamina propria of the oral cavity. The oral mucosa also harbours some nonpathogenic bacteria, like *Borrelia vincenti,* which only become invasive in times of lowered tissue resistance produced by other systemic diseases or physical and emotional stress. Oral lesions of nonbacterial origin, like ulcers produced by trauma or following rupture of vesicles caused by viral infections, may become secondarily involved by bacterial infections.

Localised bacterial infections

Vincent's Stomatitis (Acute necrotising gingivitis) – This infection is caused by *Borrelia vincenti* and *Bacillus fusiformis* which form part of the normal flora of the oral cavity. The organisms become invasive in times of lowered resistance produced by trauma, malnutrition, leukemia, severe systemic infections and the use of immuno-suppressive and antimetabolic drugs. The gingivae, on their unattached margins only, are inflammed and the gingival papillae between teeth undergo necrosis, producing a "punched-out" appearance. Later, ulceration occurs on the unattached gingival margins, producing a grayish-white slough or pseudo-membrane which, if removed, leaves a raw bleeding surface. The necrosis and suppuration produce a foul taste in the mouth and the patient transmits a fetid odour. In severely debilitated patients, the buccal and labial mucosa may undergo necrosis, destroying the lips or cheeks and producing the clinical condition known as Noma (cancrum oris or gangrenous stomatitis).

Generalised bacterial infections

Syphilis – The organism causing this disease is the *Treponema pallidum* and may infect the foetus transplacentally producing congenital syphilis, or be innoculated during sexual intercourse producing acquired syphilis. In acquired syphilis, the incubation period is approximately 3 weeks but may vary from 12–90 days. The typical lesion is a chancre at a site where the organisms enter via a breach of the mucous membrane. In extra-genital areas, this may be on the lips or the tongue. A papule which progressively enlarges and eventually ulcerates and is characteristically painless, is found. The regional lymph nodes would be palpably enlarged. Untreated, the lesion heals in 3–6 weeks.

Secondary syphilis is produced by dissemination of the organism through the blood and lymphatic stream producing systemic manifestation like fever, lymphadenopathy and skin rashes. The oral lesions consist of a diffuse, erythematous pharyngitis; mucous patches which are elevated, erythematous areas with a grayish-white exudate; or erythematous macules and papules on the palate. Tertiary syphilis manifests itself in the oral cavity in the form of a gumma of the tongue or palate or as an interstitial glossitis. Gummatous lesions are necrotic or proliferative and in the palate may produce a perforation, and in the tongue, if multiple, may produce a lobulated tongue. Interstitial glossitis is characterised by an obliterative vasculitis producing atrophy of papillae ("bald-tongue") or muscular wasting ("wrinkled tongue"). Leucoplakia may eventually appear and precede a carcinoma of the tongue. Congenital syphilis used to be aptly summarised by the triad of Hutchinson consisting of blindness (caused by interstitial keratitis), deafness and tooth defects, but other lesions may also be present (Bradlaw, 1953). Gummas may occur on the palate, jaws or facial bones and the tongue may display interstitial glossitis. Linear scars extending from the corners of the mouth, known as rhagades, are also frequently seen. If the decidous teeth are involved, enamel hypoplasia usually results. Permanent central incisors usually display hypoplasia with an increase of antero-posterior dimension and mesio-distal narrowing of incisal surface with or without notching. The first molars usually display a narrow crown with several underdeveloped cusps (mulberry molars).

Tuberculosis – In primary infections, a nodule appears at the site of innoculation in the oral cavity and undergoes ulceration after 2–3 weeks. Regional lymphadonepathy is pronounced. If the resistance of the patient is high, the lesion usually heals.

Secondary tuberculous infection produces a wide range of clinical manifestations. Tuberculous ulcers of the tongue are irregular, or sharp and linear, with undermined borders and with a purulent exudate in the bases. Surrounding induration is minimal. Granulomas may be periapical or on the buccal mucosa. If the mandible is involved, suppuration may extend to the skin producing tracts and fistulae (Cawson, 1960). In secondary infections, the cervical lymph nodes are also involved producing hard masses.

Scarlet fever – The organism causing this condition is the Group A, hemolytic streptococcus. The oral signs include an acute pharyngitis, and the inflammation may extend to the tonsilar areas. Acute stomatitis is present at some stage of the condition and the tongue may have a non-specific white coating. The fungiform papillae usually stand out prominently producing the so-called "strawberry tongue".

Gonorrhoea – Like extragenital chancres, gonorrhoea in the oral cavity is produced by oro-genital contact. The oro-pharynx is red, ulcerated and necrotic and a pseudomembrane is generally seen (Schmidt *et al.*, 1961).

Leprosy – Oral manifestations of leprosy will be dealt with in a paper in the Malaysian Dental Journal (1978).

FUNGAL INFECTIONS

These are relatively rare, and the only one of any significance is thrush (oral moniliasis or candidiasis). The organism, *Candida albicans,* is a yeast normally resident in oral cavity, and becomes pathogenic when the normal bacterial flora of the oral cavity is destroyed by broad-spectrum antibiotic therapy, or in states of lowered resistance, like prematurity, debilitating diseases, diabetes, and the use of immunosuppressive drugs. Fungal infections may be classified as superficial or deep.

Superficial mycoses

Candidiasis (thrush) – The typical lesions are creamy, milk-curd exudates, scattered in the oral cavity, with a bright red base. They strip with difficulty and leave a raw bleeding surface. Diagnosis is essentially clinical and rarely are smears or cultures necessary.

Deep mycoses

Actinomycosis – This is caused by *Actinomyces israelli,* an obligatory anaerobe which thrives in devitalised tissue like apical dental lesions, deep periodontal pockets and area of trauma. A granulomatous lesion appears in the oral cavity and spreads

via subcutaneous planes to involve the skin where a reddish-purple nodule appears and breaks down to form sinuses. Yellowish sulfur granules emerge from the orifices of the sinuses, and microscopically these are seen to be teeming with the causative organism.

Other very rare deep mycoses like blastomycosis, histoplasmosis, muromycoses, sporotrichosis and coccidiomycosis produce ulcerative, granulomatous, or proliferative lesions.

CONCLUSION

In many cases, a thorough examination of the oral cavity is an essential step in arriving at a correct diagnosis of a systemic disease. This paper has only high-lighted the oral manifestations of microbial disease, but it must never be forgotten that systemic, immunological, neurological, metabolic, nutritional, and congenital diseases can have their own specific signs and symptoms displayed in the oral cavity.

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