WINSPERT

ORAL MEDICINE Part 1

H.O.T

HIGH-PRIORITY ORGANISED THEORY

NOTES

By Dr. Jigyasa Sharma

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Thank you for your understanding and continued dedication.

Best regards,
WINSPERT TEAM

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ORAL MEDICINE PART 1 H.O.T TOPICS

HIV ORAL MANIFESTATIONS

Reference: TG

Antiretroviral drugs interact with many commonly prescribed drugs—consult an HIV expert before prescribing any drug in a patient taking antiretroviral drugs. Unusual and rare adverse reactions (e.g. perioral paraesthesia) can occur with antiretroviral drugs. With currently available antiretroviral therapy, many patients with HIV are well managed and stable. However, patients with HIV infection, particularly smokers, are at increased risk of oral diseases, such as opportunistic infections, periodontal disease, necrotising periodontitis, oral hairy leucoplakia and oral squamous cell carcinoma. HIV-related salivary gland hypofunction can occur and increases the risk of oral candidiasis.

Other oral manifestations of HIV include recurrent aphthous stomatitis, intramucosal haemorrhages and hyperpigmentation of the oral mucosa.

Some conditions are particularly related to late-stage HIV, such as Kaposi sarcoma and oral hairy leucoplakia.

Oral diseases and opportunistic infections in patients with HIV infection should be managed in conjunction with an HIV expert. Referral to an oral medicine specialist may also be appropriate.

Reference: HIV and dentistry in Australia: clinical and legal issues impacting on dental care Article

Dentists are ideally positioned to identify, manage and treat HIV-associated oral manifestations and have a responsibility to themselves and to their patients to be up to date with the evolving area of HIV and related issues.

HIV is transmitted by exposure to HIV-infected bodily fluids or tissues by way of unprotected sex, re-using drug-injecting equipment and vertical transmission from mother-to-child. In Australia, male-to-male sex remains the dominant mode of transmission.

Transmission via medical procedures is uncommon as the risk of HIV transmission after percutaneous exposure to HIV-infected blood is 0.3%.

HIV is present in saliva, however it is not considered a risk factor for transmission because of the low levels of HIV and endogenous antiviral factors present in saliva. There is no evidence that HIV can be transmitted by contact with tears, sweat, urine or faeces.

Dentists can play a key part in the diagnosis and management of patients with HIV. Research has demonstrated that early identification of HIV is an important factor in maximizing positive outcomes for individual patients and for preventing ongoing transmission.

Unfortunately, indications for HIV testing are often missed. Dentists should not underestimate the contribution they can make in the diagnosis of HIV by way of its oral manifestations.

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HIV ORAL MANIFESTATIONS

Reference: TG

Oral lesions may be present at all stages of HIV infection. However, it should be emphasized that HIV-associated oral lesions are not pathognomonic as it is possible to find such conditions in immunocompetent people without HIV infection.

Treatment of HIV infection involves the use of combinations of antiretroviral medications.

The oral manifestations of HIV can be divided into five categories:

- **Microbiological infections (fungal, bacterial and viral)**
- **Oral neoplasms**
- **Neurological conditions**
- **Other oral conditions that may be associated with HIV infection**
- **Oral conditions associated with HIV treatment**

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1) Microbiological infections (fungal, bacterial and viral)

Fungal infections:

Mycoses or fungal infections are often the first and can be the most prevalent conditions affecting the oral mucosal surfaces of patients with HIV infection. The main fungal pathogen involved in oral disease is Candida albicans. Ninety per cent of patients with advanced HIV disease are affected with oral candidiasis at some point during their disease.

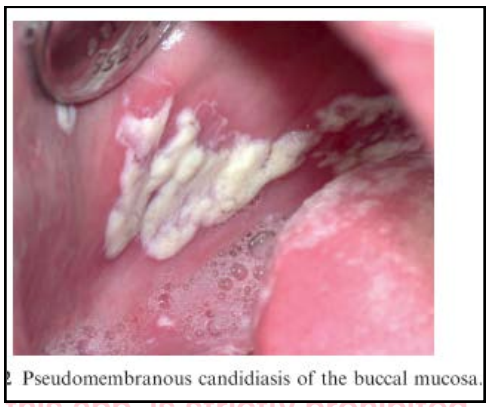
However, cART results in a significant reduction of oral candidiasis. cART has changed the prevalence patterns of oral lesions associated with HIV infection, so candidiasis is no longer the most common condition.

The classic forms of oral candidiasis described below include:

- Pseudomembranous Candidiasis
- Erythematous Candidiasis
- Angular Cheilitis
- Chronic Hyperplastic Candidiasis

Pseudomembranous candidiasis

- It is evident by the presence of creamy white or yellow plaques found on any of the intraoral surfaces which, when scraped, reveal an erythematous or bleeding mucosal surface.
- It may cause no symptoms or mild to moderate pain or burning and is usually intermittent, however may be chronic.
- The diagnosis is clinical, although when uncertain or there is a lack of response to treatment, microscopy, culture or biopsy may be needed.
- A periodic acid Schiff staining of a cytological smear may show candidal hyphae.



2 Pseudomembranous candidiasis of the buccal mucosa.

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Erythematous Candidiasis

- Causes patchy red or erythematous areas that may become atrophic.
- It is often associated with oral appliances, may become diffuse, and is commonly found on the hard palate, the dorsum of the tongue and occasionally on the buccal mucosa.
- Similar to pseudomembranous candidiasis, there may be no symptoms or mild to moderate pain or burning.
- Candidiasis is usually intermittent, how- ever it may become chronic, especially when related to dentures.
- A history and examination is sufficient for diagnosis, although in cases where there is an uncertain diagnosis or poor response to treatment, sampling for microscopy and culture or biopsying may be necessary.

It should be noted antifungals commonly interact with other medications. An important interaction is the potentiation of warfarin by miconazole.

Angular Cheilitis

- Angular cheilitis, found at the labial commissures, causes an erythematous lesion with red or white fissures or ulcers.
- It is asymptomatic or only causes mild to moderate pain/burning, of intermittent duration but may become chronic.
- Diagnosis is clinical however occasionally, taking a swab for microscopy and culture, or a biopsy, may be appropriate if there is an uncertain diagnosis or the lesion does not respond well to therapy.

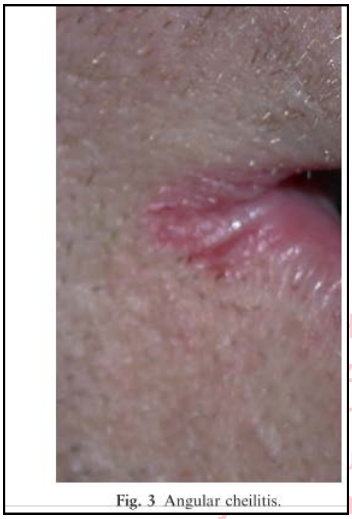

Fig. 3 Angular cheilitis.

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Chronic Hyperplastic Candidiasis

- Chronic hyperplastic candidiasis is associated with smoking, local factors, blood group antigen secretor status and nutritional deficiencies (iron and vitamins A, B1 and B2).
- The lesions are generally considered premalignant and may demonstrate dysplasia.
- Clinically, they may appear as speckled or homogenous rough white patches that are irregular, unable to be wiped off and are indistinguishable from a leukoplakia.
- Chronic hyperplastic candidiasis is long-standing and most commonly found on the buccal mucosa, near the labial commissures, with less frequent involvement of the palate or tongue where it can be confused with oral hairy leukoplakia.
- It is usually asymptomatic, although speckled lesions are more likely to cause discomfort.
- Lesions can be clinically diagnosed but due to their premalignant status and the similarity in appearance to malignant lesions, a biopsy to define and characterize the lesion is ideal.
- A definitive diagnosis should be sought and ongoing monitoring is necessary. Depending on the histopathology, further treatment or referral may be necessary.
- Topical or systemic antifungals and surgical therapies are treatment options.

Treatment for oral candidiasis with topical antifungals can be initiated by a general dentist, however prescribing systemic antifungal therapy is beyond the scope of practise for a general dentist.

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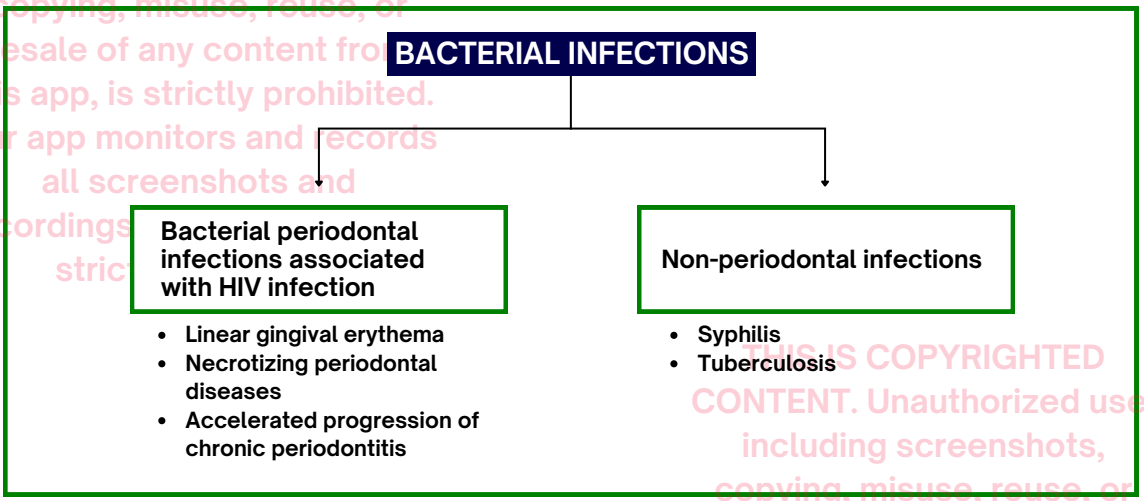
Bacterial infections:

There is a wide range of bacterial pathogens that cause oral disease in patients with HIV infection. This section considers bacterial periodontal infections associated with HIV infection, as well as syphilis and tuberculosis, which are bacterial, non-periodontal infections.

For dentists, one of the most significant oral manifestations of HIV-associated bacterial infections is periodontal pathology.

This pathology falls into three groups:

- **Linear Gingival Erythema**
- **Necrotizing Periodontal Diseases**
- **Accelerated Progression of Chronic Periodontitis**



Linear gingival erythema is primarily a fungal disease. However, it is worth mentioning here as linear gingival erythema may represent a precursor condition to necrotizing ulcerative periodontal diseases associated with HIV infection.

The necrotizing diseases of the periodontium include necrotizing ulcerative gingivitis, periodontitis and stomatitis. The prevalence of necrotizing periodontal diseases associated with HIV infection has reduced with the introduction of modern antiretroviral therapy. Particularly spirochetes, but the usual periodontal pathogens, are believed to be important in the pathogenesis of necrotizing ulcerative gingivitis and periodontitis.

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Necrotizing ulcerative gingivitis

presents with pain, ulceration and gingival bleeding. The lesion does not involve the alveolar bone and is characterized by punched out, ulcerated and erythematous interdental papillae covered by a greyish necrotic slough. It is found on the gingival tissues, particularly the interdental papillae and causes moderate to severe pain, bleeding and fetor oris. Systemic features such as fever, malaise and lymphadenopathy may be present. Necrotizing ulcerative gingivitis has a sudden onset and short duration, although may be progressive in some cases.

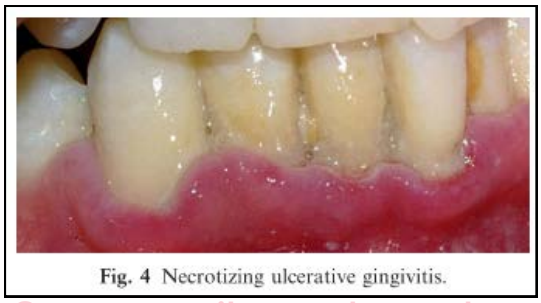

Fig. 4 Necrotizing ulcerative gingivitis.

Necrotizing ulcerative periodontitis

Presents similarly to necrotizing ulcerative gingivitis, except the lesion involves the alveolar bone, and potentially, gingival recession and tooth mobility. Onset is sudden and the condition can be rapidly progressive. It causes ulcerated and erythematous gingival and periodontal tissues. The interdental papillae are covered by a greyish necrotic slough. Symptoms are the same as those for necrotizing ulcerative gingivitis.

Fig. 5 Necrotizing ulcerative periodontitis.

Necrotizing ulcerative stomatitis

Involves an extensive area of oral ulceration, tissue necrosis and erythema that extends from the gingivae into the adjacent mucosa and may involve bone, leading to osteonecrosis and sequestration. It is evident on the periodontal tissues and may extend into the maxillary or mandibular bone, with the same symptoms and duration as necrotizing ulcerative periodontitis.

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Management of periodontal infection in HIV patients

Currently, there is inadequate information to provide dentists with evidence-based guidelines for the management of periodontal infections in people with HIV. Periodontal diseases should be treated as they would in people without HIV infection, with removal of plaque, calculus and necrotic tissue. Excellent home oral hygiene should be encouraged, and smoking cessation recommended.

Adjunctive therapies and the prescription of systemic antibiotics should be considered on a case-by-case basis. Adjunctive therapies include irrigation and rinsing with 10% povidine iodine or alternatively 15 mL chlorhexidine 0.12%–0.2% mouthwash, rinsed in the mouth for one minute, 8 to 12 hourly. Chlorhexidine should only be used on a short-term basis as prolonged periods of use may cause discolouration of teeth and restorations. Narrow spectrum antibiotics, such as metronidazole (400 mg orally [or for a child; 10 mg/kg up to 400 mg], 12 hourly for five days), are preferable to broad spectrum antibiotics, to reduce the likelihood of overgrowth of commensals and antibiotic resistance.

If a dentist feels inadequately equipped to manage periodontitis in people with HIV or if they fail to control the disease, then referral to a periodontist, a general practice dentist with an interest in HIV infection or a specialized clinic at a major hospital, is required.

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Viral infections:

There are seven groups of viruses that commonly cause oral lesions. Patients co-infected with HIV and with any of these viruses are at increased risk of developing oral conditions.

The viral groups include:

- herpes simplex virus (HSV 1 and 2)
- varicella zoster virus (VZV)
- cytomegalovirus (CMV)
- human papilloma virus (HPV many subtypes)
- Epstein-Barr virus (EBV)
- molluscum contagiosum virus 2 (MCV2)
- human herpesvirus (HHV8)

The oral manifestations of viral co-infections with HIV are discussed below. Treatment of viral infections, on the whole, should be done by a medical practitioner or dental specialist.

Herpes simplex virus (HSV 1 and 2)

HSV has two main types, type 1 and type 2. HSV, when it appears on the lips, is known as herpes labialis or a cold sore. Primary HSV infection may be very severe, whereas recurrent infections are usually less severe.

Herpes labialis presents as multiple small vesicles or ulcers on the lips and may include adjacent skin. Intraoral HSV infection presents as small, round vesicles that rupture, leaving shallow ulcers that can coalesce. The lesions are superimposed on an inflamed, erythematous base. In people with HIV infection, recurrent HSV infection is common.

Lesions occur on the lips and anywhere in the oral cavity. In the mouth, HSV is commonly found on keratinized epithelium, including hard palate, gingiva and dorsum of the tongue, but in people with HIV infection it can sometimes be found on non-keratinized epithelium. Prodromal symptoms may be present before the rapid onset of lesions, persisting for 7–14 days causing mild to severe pain. They may be localized or widespread, involving the entire oral cavity and lips. Fever, lymphadenopathy and other symptoms may occur, especially with a primary infection. A diagnosis can be made from history and examination, or if uncertain, a swab for PCR analysis can be performed. No treatment for HSV is required if symptoms are mild, although treatment of symptoms may be necessary. Severe or recurrent infections should be treated by a medical practitioner or dental specialist with topical and/or oral antiviral medications such as aciclovir, famciclovir or valaciclovir.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

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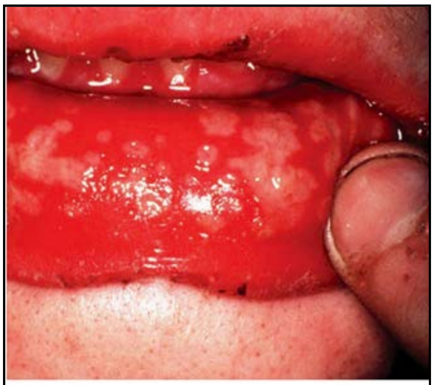

Fig 1. Primary herpetic gingivostomatitis consists of vesicles of the tongue, cheek mucosa and gingivae that break down to form clusters of small round or irregular superficial ulcers with a yellowish base and a red margin.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

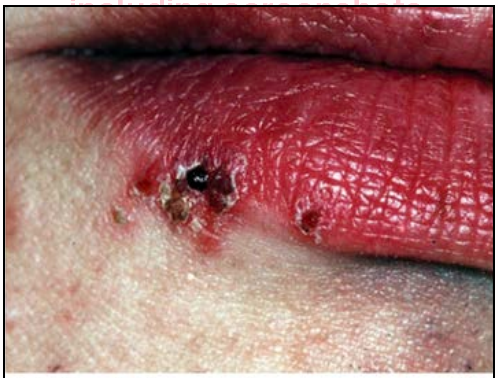

Fig 2. Herpes labialis occurs on the mucocutaneous junction of the lip or on the skin adjacent to the nostril. These lesions are often preceded by a prickling sensation before blisters form, which then enlarge, coalesce, rupture and become crusted before healing.

REFERENCE: HIV and dentistry in Australia: clinical and legal issues impacting on dental care ARTICLE

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Varicella Zoster virus (VZV)

VZV is the herpes virus that causes the primary infection known as chicken pox and can reactivate from a latent state to cause herpes zoster (HZ) or shingles. The pattern of VZV infection is generalized in primary infection, however HZ infection is usually unidermatomal but can be multidermatomal or disseminated. VZV infection can be recurrent.

Intraorally, VZV presents as a roughly linear eruption of herpetiform vesicles or bullae that ulcerate and may coalesce. Extraorally, the vesicles can ulcerate and form a crust or scab.

There may be prodromal symptoms present and the rash causes mild to severe pain. The duration of VZV is usually 10–14 days, although infections can become chronic and leave scarring. A diagnosis may be made on clinical findings, although a swab for PCR analysis may be performed if diagnosis is uncertain.

In the setting of immunocompromise, urgent review of VZV for consideration of treatment is required. Antiviral medications, such as aciclovir, famciclovir or valaciclovir, can reduce illness severity and complications.

HZ in the ophthalmic (V1) distribution of the trigeminal nerve requires ophthalmological referral to minimize ocular complications.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

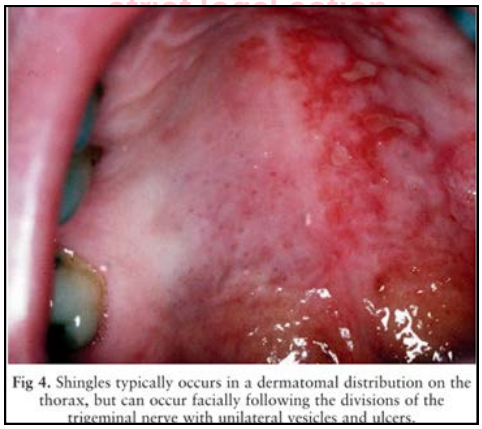

Fig 4. Shingles typically occurs in a dermatomal distribution on the thorax, but can occur facially following the divisions of the trigeminal nerve with unilateral vesicles and ulcers.

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Oral lesions may be present at all stages of HIV infection. However, it should be emphasized that HIV-associated oral lesions are not pathognomonic as it is possible to find such conditions in immunocompetent people without HIV infection. Treatment of HIV infection involves the use of combinations of antiretroviral medications.

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Reference: TG

Human papilloma virus (HPV many subtypes)

Reference: TG

- **Human papilloma viruses (HPV) can cause a wide range of oral mucosa lesions. The virus is usually transmitted by direct contact with a lesion. Squamous papilloma is the most common oral HPV lesion, appearing as a protruding growth with small finger-like projections.**
- **Sexually transmitted HPV infections can cause oral HPV lesions called condyloma acuminata. Verruca vulgaris—the common wart—is also caused by HPV infection and may present in the oral cavity.**
- **Both condyloma acuminata and verruca vulgaris can be clinically similar to squamous papilloma.**
- **Oncogenic types of HPV are now recognised as a cause of some squamous cell carcinoma, particularly of the posterior tongue, tonsillar region and oropharynx. These appear to be a distinct entity, separate to the oral cancers associated with alcohol and tobacco use.**
- **Refer patients with suspected HPV lesions to an appropriate specialist for biopsy and management.**

REFERENCE: HIV and dentistry in Australia: clinical and legal issues impacting on dental care ARTICLE

A variety of benign mucocutaneous lesions are induced by the HPVs, including:

- **verruca vulgaris**
- **condyloma acuminatum**
- **focal epithelial hyperplasia (Heck's Disease)**
- **squamous papilloma**

Verruca vulgaris

Also known as the common wart. There may be single or multiple cauliflower-like growths with a white or pink surface. Lesions of varying diameter, either sessile or pedunculated, are found anywhere in the oral cavity but more commonly seen on the labial mucosa. Usually asymptomatic, lesions may be present for years. Commonly a clinical diagnosis is sufficient, however a biopsy is definitive.

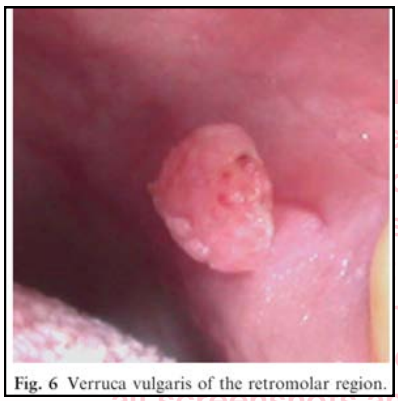

Fig. 6 Verruca vulgaris of the retromolar region.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

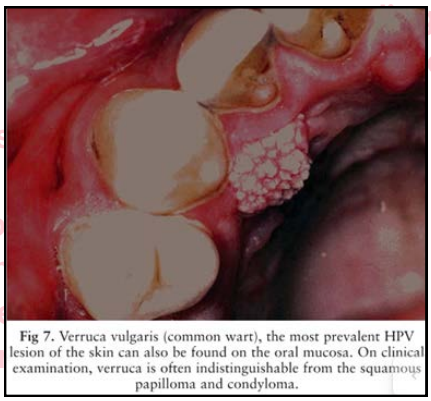

Fig 7. Verruca vulgaris (common wart), the most prevalent HPV lesion of the skin can also be found on the oral mucosa. On clinical examination, verruca is often indistinguishable from the squamous papilloma and condyloma.

REFERENCE: HIV and dentistry in Australia: clinical and legal issues impacting on dental care ARTICLE

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Condyloma acuminatum

Also known as the venereal wart, is characteristically found on anogenital mucosa, however warts may also be seen on oral mucosa and may present as multiple, large and disfiguring lesions in association with HIV infection. Condyloma acuminatum may present as single or multiple lesions of varying sizes, which are soft and have a pink to grey appearance. Often multiple nodules coalesce to form pedunculated or sessile papillary growths. They can be found on any mucosal surface, particularly the ventral tongue, gingiva, labial mucosa and palate. Although the lesions are normally asymptomatic, the condition is often chronic. Diagnosis is made on clinical findings, however for a definitive diagnosis, biopsy is recommended.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

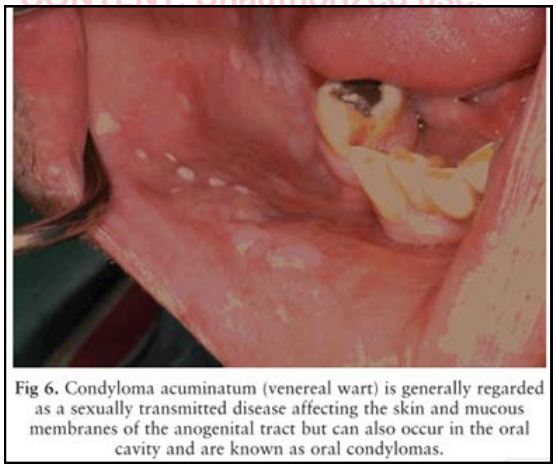

Fig 6. Condyloma acuminatum (venereal wart) is generally regarded as a sexually transmitted disease affecting the skin and mucous membranes of the anogenital tract but can also occur in the oral cavity and are known as oral condylomas.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE

Squamous Papilloma

Squamous cell papilloma (SCP) is a relatively common benign tumour of the oral epithelium, representing about half of all soft tissue tumours. On gross appearance, oral papilloma is characterized by small finger-like projections, resulting in a lesion with a rough or cauliflower-like verrucous surface. Oral papilloma are benign lesions.

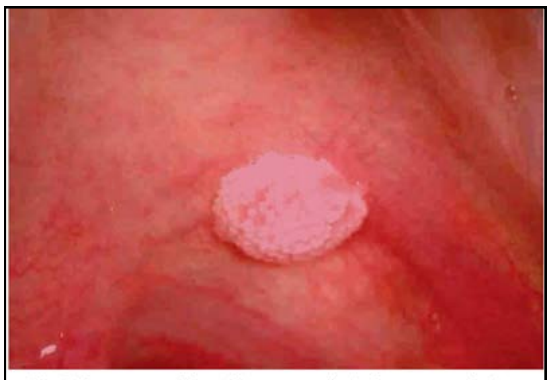

Fig 5. Squamous cell papillomas are relatively common benign tumours of the oral epithelium, representing about half of all soft tissue tumours.

REFERENCE: HIV and dentistry in Australia: clinical and legal issues impacting on dental care ARTICLE

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HIV ORAL MANIFESTATIONS

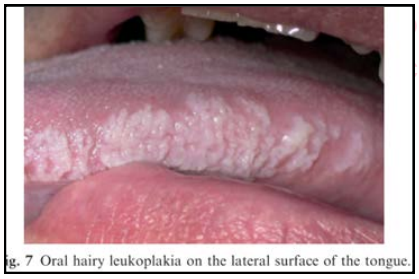
Reference: TG

Epstein-Barr virus (EBV)

EBV has been connected to infectious mononucleosis, Burkitt's lymphoma, non-Hodgkin's lymphoma and nasopharyngeal carcinoma. The chief manifestation of EBV in people with HIV infection is oral hairy leukoplakia (OHL), and so EBV and OHL will be discussed together. EBV has been linked to oral ulceration in patients with advanced HIV infection.

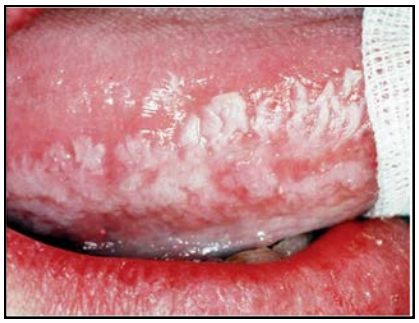
OHL has rarely been reported in immunocompetent people without HIV infection and may be considered a marker of disease progression. As CD4 cell counts fall in the context of HIV infection, OHL is increasingly found and it is common when the CD4 count drops below 150 cells/lL.

OHL lesions present as whitish, elevated, non-removable patches of variable size. Characteristically, the surface of the lesion has vertical ridges, but smooth lesions can occur. Lesions are found on the lateral borders of the tongue and sometimes they may extend onto the ventral and dorsal surfaces of the tongue and occasionally on the buccal mucosa.



g. 7 Oral hairy leukoplakia on the lateral surface of the tongue.

Source: Oral viral infections and the therapeutic use of antiviral agents in dentistry ARTICLE



REFERENCE: HIV and dentistry in Australia: clinical and legal issues impacting on dental care ARTICLE

OHL is a chronic condition and diagnosis is possible with clinical findings and/or biopsy. Specific treatment is not indicated due to the benign and asymptomatic nature of OHL.

OHL usually resolves following the introduction of effective antiretroviral therapy, although it may recur after stopping treatment.

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HIV ORAL MANIFESTATIONS

Reference: TG

2) Oral neoplasms

There are two common malignancies associated with HIV infection that may have oral involvement:

- Kaposi's sarcoma (KS)
- non-Hodgkin's lymphoma (NHL)

At present there is insufficient evidence to establish a direct relationship between oral squamous cell carcinoma and HIV infection.

KS was the most common malignancy associated with HIV infection; however, rates have significantly decreased with cART.

Kaposi's sarcoma (KS)

KS presents initially as a symptomatic red macule which enlarges to form a red-blue plaque. These plaques may grow into lobulated nodules that potentially ulcerate and some-times cause pain. The lesions can be red, purple, blue or brown in colour and range from flat macules to ulcerated nodular masses. KS can be found on the skin or mucous membranes and, in the mouth, KS most commonly involves the hard palate, followed by the gingiva and buccal mucosa.

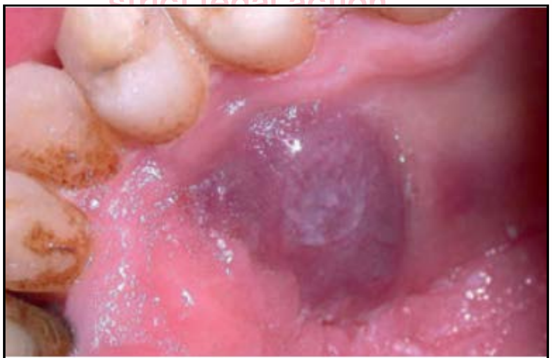

Fig. 8 Kaposi's sarcoma.

Biopsy carries a significant risk of haemorrhage as the lesions are extremely vascular and this should be avoided by the general dental practitioner. Often commencement of cART can lead to spontaneous resolution of these lesions. Systemic chemotherapy, intra-lesional chemotherapy and radiotherapy are treatment modalities that may be employed.

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HIV ORAL MANIFESTATIONS

Reference: TG

3) Other oral conditions that may be associated with HIV infection

- Hyperpigmentation,
- Aphthous Ulceration
- Xerostomia

have been documented as occurring in the context of HIV infection.

4) Oral conditions associated with HIV treatment

HIV medications can cause:

- Taste disturbance
- Dry lips
- The cracking and crusting of the lips (Protective creams designed for use on the lips, such as papaya-based lip ointments can be helpful in alleviating this condition)
- Xerostomia (the most important factor in the development of dental caries)
- oral ulceration
- erythema multiforme (Stevens–Johnson syndrome)
- lichenoid reactions
- hyperpigmentation

There is the potential that many drugs prescribed by dentists may interact with cART, therefore medications should always be prescribed in consultation with the patient's medical practitioner(s)

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HIV ORAL MANIFESTATIONS

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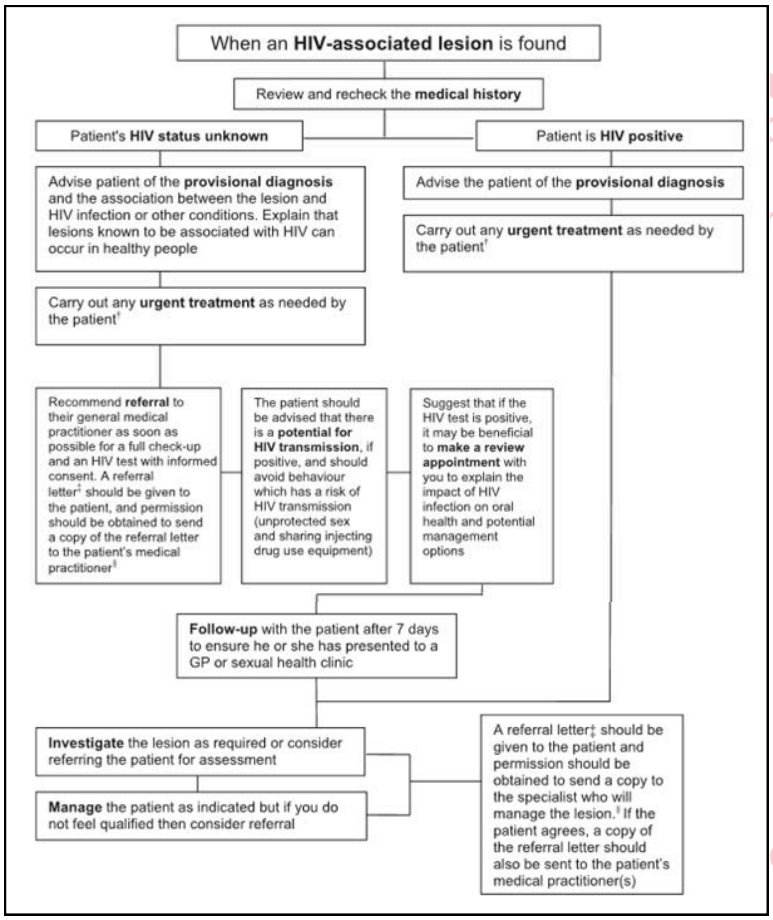
Algorithm for the management of oral manifestations indicative of HIV infection



- Any treatment should be initiated in consultation with the patient's general medical practitioner and/or with specialists as necessary.
- A referral letter should contain patient details, reason for referral, examination findings, diagnosis, and details of treatment.
- If a patient declines referral for follow-up or to have a referral sent to their general medical practitioner or specialist, then it is necessary to consult with a senior colleague and obtain a medicolegal opinion about the necessary steps that should be undertaken to follow-up the patient. There may be circumstances, although rare, where a breach of confidentiality is legally permissible in order to have the patient adequately followed up.

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TUBERCULOSIS AND DENTAL MANAGEMENT

Tuberculosis

Reference: ADA article : https://www.ada.org/resources/ada-library/oral-health-topics/tuberculosis-overview-and-dental-treatment-conside

Tuberculosis is caused by infection with Mycobacterium tuberculosis (M. tuberculosis). Infection occurs through inhalation of airborne droplets containing viable M. tuberculosis, which then travel to the alveoli of the lungs. Only people with active disease can spread the infection.

M. tuberculosis is transmitted through infectious airborne particles, known as “droplet nuclei,” which can be generated when people with pulmonary or laryngeal tuberculosis sneeze, cough, speak or sing. These small particles (1 to 5 micrometres in diameter) can stay suspended in the air for hours. Non-coughing individuals who are suspected of having tuberculosis cannot be presumed to be non-infectious because M. tuberculosis transmission may still occur ithout the presence of coughing. According to one study, up to 77% of respiratory bio-aerosol samples from newly diagnosed patients may contain M. tuberculosis organisms.

If a susceptible person inhales aerosolized droplet nuclei containing M. tuberculosis, infection may begin if the organisms reach the alveoli. Within two to 12 weeks, the body’s immunological response to M. tuberculosis usually prevents further multiplication and spread. The mycobacterium can live in the lungs of an infected person for years, even a lifetime, without the person exhibiting any symptoms; this state is called latent infection. A person with latent tuberculosis is generally asymptomatic and not infectious to others but the infection can develop into active tuberculosis in the future and usually exhibits a positive reactive tuberculin skin test. Most people who have latent tuberculosis infection never develop active disease, but if they do not receive treatment for latent infection, about 10 percent of people with latent infections can develop active disease over a lifetime. This can happen when the person’s immune system is weakened, allowing the mycobacteria to cause active tuberculosis infection (e.g., individuals with HIV, diabetes, certain hematologic disorders such as leukemias and lymphomas, prolonged corticosteroid use, and other conditions).

Only a person with active tuberculosis can transmit the disease. People with active tuberculosis infection generally have symptoms (e.g., persistent, productive cough; night sweats, fever, weakness or fatigue; weight loss; pain in the chest); and can have a positive tuberculin skin test reaction.
Respiratory tuberculosis may be present in any patient with symptoms including coughing for more than three weeks, loss of appetite, unexplained weight loss, night sweats, bloody sputum or haemoptysis, hoarseness, chest pains, fever, fatigue or presence of persistent lesions of the oral mucosa that are non-responsive to therapy.

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TUBERCULOSIS AND DENTAL MANAGEMENT

There are multiple tests for tuberculosis (TB), including:

- TB blood test: Also known as an Interferon Gamma Release Assay (IGRA), this test involves drawing a small amount of blood and sending it to a laboratory. TB blood tests are more accurate than TB skin tests.
- TB skin test: Also known as the Mantoux tuberculin skin test, this test involves injecting a small amount of fluid under the skin on the lower inner forearm. A small bump will form where the fluid was injected.
- Chest x-ray: A chest x-ray can help determine if you have TB.
- Sputum smear and culture: These laboratory tests can help determine if TB germs are present.
- Drug resistance tests: These laboratory tests can help determine drug resistance.

Other tests for TB include:

- Acid-fast microscopy
- Molecular assays
- X-ray, ultrasound, or CT scan
- Biopsy

Key Points:

- Although the risk of transmission of tuberculosis in dental settings is low, the Centres for Disease Control and Prevention (CDC) recommends dental health care personnel include protocols for tuberculosis infection control in their offices' written infection control program.
- Infection occurs through inhalation of small airborne droplets containing Mycobacterium tuberculosis, which then travel to the alveoli of the lungs; only people with active disease can spread the infection.
- A person with latent tuberculosis is not infectious; he or she can be treated in the dental office under standard infection control precautions.
- However, for a person with active tuberculosis, standard precautions are insufficient to prevent transmission of the bacterium.

Reference: https://www.health.state.mn.us/diseases/tb/basics/factsheets/tst.html

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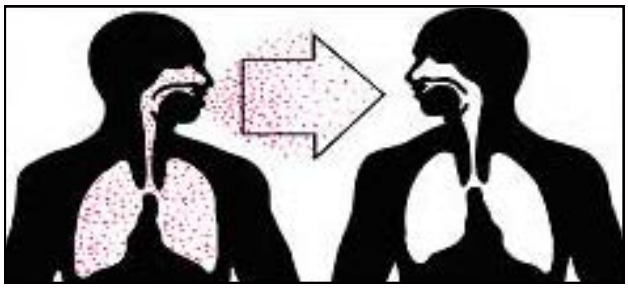
The tuberculosis (TB) skin test, sometimes called a "Mantoux," is a simple, harmless way to find out if you have latent TB infection.



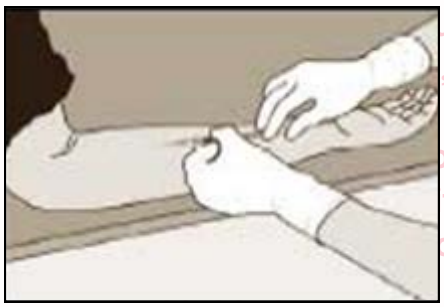
What is latent TB infection?

There are two phases of TB. Both phases can be treated with medicine. When TB germs first enter your body, they cause latent TB infection. Without treatment, latent TB infection can become active TB disease. Anyone can get TB because it spreads from one person to another through the air.

Phase 1 – Latent TB Infection	Phase 2 – Active TB Disease
TB germs are "asleep" in your body. This phase can last for a very long time – even many years.	TB germs are active and spreading. They are damaging tissue in your body.
You don't look or feel sick. Your chest x-ray is usually normal.	You usually feel sick. Your doctor will do special tests to find where TB is harming your body.
You can't spread TB to other people.	If the TB germs are in your lungs, you can spread TB to other people by coughing, sneezing, talking, or singing.
Usually treated by taking one medicine for 9 months.	Treated by taking 3 or 4 medicines for at least 6 months.

How can I tell if I have latent TB infection?

A TB skin test ("Mantoux") can show if you have latent TB infection. You could have latent TB infection if you have ever spent time close to someone with active TB disease (even if you didn't know they were sick). Your health care provider will use a small needle to inject some harmless testing fluid (called "tuberculin") under the skin on your arm.



Your health care provider MUST check your arm 2 or 3 days after the TB skin test, even if your arm looks OK to you.

If you have a reaction to the test, it will look like a raised bump. Your health care provider will measure the size of the reaction. If there is a bump, it will go away in a few weeks.

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TUBERCULOSIS AND DENTAL MANAGEMENT

How do I take care of my arm after the TB skin test?

- Don't cover the spot with a bandage or tape.
- Be careful not to rub it or scratch it.
- If the spot itches, put a cold cloth on it.
- You can wash your arm and dry it gently.

What if my TB skin test is negative?

The test is "negative" if there is no bump (or only a very small bump) at the spot where the fluid was injected. A negative TB skin test usually means that you don't have TB.

In some situations, you may need to have another TB skin test later.

What if my TB skin test is positive?

The test is "positive" if there is a bump of a certain size where the fluid was injected. This means you probably have TB germs in your body. Most people with a positive TB skin test have latent TB infection. To be sure, your doctor will examine you and give you a chest x-ray. You may need other tests to see if you have active TB disease.

You should have a TB skin test if:

- you have had frequent close contact with someone who has active TB disease,
- you have lived in a country where many people have TB,
- you work or live in a nursing home, clinic, hospital, prison, or homeless shelter, or
- you have HIV infection or certain other health problems.

What if I've had BCG vaccine?

Even if you have had BCG vaccine, you can have a TB skin test.

- People who have had BCG vaccine still can get latent TB infection and active TB disease.
- BCG vaccine may help protect young children from getting very sick with TB. This protection goes away as people get older.
- BCG vaccine sometimes causes a positive TB skin test reaction. But if you have a positive reaction to the TB skin test, it probably is from TB germs in your body - not from your BCG vaccine.

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Dental Management

Reference: TG

Infection by airborne transmission of respiratory secretions can occur with pulmonary tuberculosis. Tuberculosis is spread by droplets or by direct contact and has been transmitted as a result of dental procedures. Patients with these diseases should have their dental treatment deferred until they are no longer infectious and have reached the end of any mandatory quarantine period.
Where treatment cannot be deferred (e.g., facial swelling), transmission-based precautions must be used for provision of dental treatment. These are described below.

- **Schedule the patient to be seen as the last patient of the day.**
- **Have the patient use a suitable antimicrobial pre-procedure mouth rinse (e.g., chlorhexidine gluconate, essential oil mouth rinse, hydrogen peroxide, povidone iodine or ozonated water).**
- **Wear high-filtration surgical masks that are adapted well to the face. Use of surgical respirators (N95 or N99) is optional; this would apply only to staff who have been fitted (i.e. fit tested) and trained properly in how to wear these respirators, with proper fit checking before use.**
- **Consider the use of barriers for high-risk items (optional).**
- **For restorative dentistry, use a dental dam and high-velocity evacuation to reduce the formation of aerosols. For other procedures, use techniques that minimise the production of splashes of fluids and generation of aerosols.**
- **At the end of the appointment, undertake the surface cleaning process twice (i.e., one full additional cycle of surface cleaning). This could be detergent followed by disinfectant, or two cycles using a product that combines detergent and disinfectant.**

Reference: ADA article : https://www.ada.org/resources/ada-library/oral-health-topics/tuberculosis-overview-and-dental-treatment-conside

Environmental Controls

- **Use an airborne infection isolation room to provide urgent dental treatment to patients with suspected or confirmed infectious TB**
- **In settings with high volume of patients with suspected or confirmed TB, use high-efficiency particulate air filters or ultraviolet germicidal irradiation**

A person with non-infectious latent tuberculosis may be treated in the dental office under standard infection control precautions.

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TUBERCULOSIS AND DENTAL MANAGEMENT

Any patient with symptoms suggestive of active tuberculosis disease should be removed from the area of other patients or staff, instructed to wear a surgical or procedure mask, assessed for the urgency of their dental care and promptly referred for medical care. Standard precautions are insufficient to prevent transmission of the bacterium. Elective dental treatment should be deferred until the patient has been declared non-infectious by a physician. Urgent dental care for a person with suspected or active tuberculosis should be provided in a facility that has the capacity for airborne infection isolation and has a respiratory protection program in place.
Standard surgical face masks are not adequate to protect against tuberculosis transmission; however, appropriate respiratory protection (e.g., fitted, disposable N95 respirators) provide protection when treating a patient with active disease.

Reference: CARRANZA

The patient with tuberculosis should receive only emergency care. If the patient has completed chemotherapy, the patient's physician should be consulted regarding infectivity and the results of sputum cultures for Mycobacterium tuberculosis. When medical clearance has been given and the sputum culture results are negative, these patients may be treated normally. Any patient who gives a history of poor medical follow-up (e.g., lack of yearly chest radiographs) or shows signs or symptoms indicative of tuberculosis should be referred for evaluation. Adequate treatment of tuberculosis requires a minimum of 18 months, and thorough posttreatment follow-up should include chest radiographs, sputum cultures, and a review of the patient's symptoms by the physician at least every 12 months.

TB in HIV patients

Reference: CARRANZA

Tuberculosis (TB) is caused by Mycobacterium tuberculosis. The atypical mycobacteria are beyond the scope of this document. People with HIV infection may develop TB via primary infection, reactivation of latent infection and re-infection with new strains. TB is rare in Australian-born people but much more common in those born, or who have lived, in countries of high TB prevalence.
Occasionally, TB may present as chronic ulcers with a grey-yellow slough in the mouth which, when examined histologically, demonstrate the presence of granulomas or granulomatous inflammation. There may also be lymphadenopathy in the head and neck. TB can occur at any CD4 cell level; however, the frequency and severity of disease is inversely proportional to the CD4 cell level. TB presents significant infection control issues. If the patient has active disease then transmission-based precautions are necessary and, if possible, dental treatment should be postponed. If a patient has suspected TB, referral to a medical practitioner for investigation and management is necessary.

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BURNING MOUTH SYNDROME

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Burning mouth syndrome (BMS) is characterized by burning pain in the tongue or other oral mucous membrane.

Often associated with symptoms such as

- **subjective dryness of the mouth**
- **paraesthesia**
- **altered taste**

for which no medical or dental cause can be found. The difficulty in diagnosing BMS lies in excluding known causes of oral burning.

Burning mouth syndrome (BMS) is defined by the International Association for the Study of Pain 1 as burning pain in the tongue or other oral mucous membrane associated with normal signs and laboratory findings lasting at least four to six months.

Table 1. Local, systemic and psychosocial factors which may be responsible for oral burning

Local	Systemic	Psychosocial
Dry mouth • hyposalivation • xerostomia	Haematinic disorders • vitamin B group • iron • folate • zinc	Psychological disorders • depression • anxiety • somatization
Taste alterations	Autoimmune type connective tissue • Sjögren's syndrome • sicca • systemic lupus erythematosis	Personality profiles • neurotisicism • extraversion • openness • conscientiousness
Oral infection • fungal • bacterial • viral	Gastroesophageal reflux disease	
Oral mucosal diseases • lichen planus • benign migratory glossitis • hairy tongue • fissured tongue	Endocrine-related disorders • diabetes • thyroid disorders • hormone deficiencies	
Oral parafunction	Medication side effects • tricyclic antidepressants • ACE inhibitors	
Oral galvanism	Central nervous system disorders • multiple sclerosis • Parkinson's disease • trigeminal neuralgia	
Poorly designed dentures	Idiopathic focal conditions • oro-cervical • uro-genital	
Allergic reactions • dental products • food products		

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BURNING MOUTH SYNDROME

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Local Factors

There are a number of local factors that can cause oral burning which must be excluded prior to making a diagnosis of primary BMS.

1) Dry Mouth

Xerostomia and hyposalivation are both conditions that can cause dry mouth, but they are distinct:

Xerostomia: The subjective feeling of having a dry mouth
Hyposalivation: An objective reduction in the amount of saliva produced, also known as salivary gland hypofunction. Measured by sialometry. (stimulated salivary flow rate= 1-2ml/min; unstimulated salivary flow rate = 0.3 to 0.4 mL/min; An unstimulated salivary flow rate of 0.1 to 0.2 mL/min and stimulated flow rate of 0.7 mL/min or less is considered indicative of salivary gland hypofunction)

A lack of lubrication with saliva predisposes the oral mucosa to friction and pain often of burning quality.
Twenty-five per cent of BMS patients report dry mouth which may either be idiopathic or secondary to medication use such as tricyclic antidepressants and benzodiazepines.

2) Taste alteration

Taste disturbances, such as an alteration in taste perception (dysgeusia) and/or a persistently altered taste are often reported by BMS patients.
Persistent taste reported included bitter (33 per cent), metallic (27 per cent) or combination (10 per cent) which decreased in 60 per cent of subjects after rinsing with distilled and deionized water.

3) Oral infection

- Infections involving the oral cavity have been reported as a cause of oral burning.
- Oral candidiasis is a common fungal infection implicated in BMS and must be ruled out. Of concern, is the high prevalence of candida species in BMS patients, therefore making it difficult to discern its specific role in causing oral mucosal burning.
- Typically, the presence of fungal infection is often associated with the findings of atrophy, erythema and ulceration of the oral mucosa which may be the cause of burning pain.
- Patients often report an increased pain upon eating suggestive of candida-induced burning and likely due to irritation of the mucosa.

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BURNING MOUTH SYNDROME

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

- On the contrary, a decrease or abortion of the pain while eating is commonly found in BMS patients.
- Bacterial infections involving spirochetes, fusiform, Enterobacter and klebsiella species and helicobacter pylori have been suggested as causative of BMS.
- Due to the often-described rapid onset of BMS and dysesthesia, viral (herpes viruses) causes have been considered

4) Oral mucosal diseases

Oral mucosal diseases such as:
(have been proposed as causative of BMS)

- Lichen Planus

  Atrophic and ulcerative forms of lichen planus are known to have a burning pain particularly during periods of exacerbation.
- Benign migratory glossitis

  Benign migratory glossitis is usually painless but burning may occur in areas of depapillation which may be exacerbated by spicy foods, alcohol or stress.
- Hairy tongue
- Fissured tongue

  Fissured tongue is also usually painless unless grooves and fissures become inflamed or infected due to accumulation of debris resulting in a burning sensation.

These oral mucosal diseases are all associated with visual clinical findings, yet in BMS patients, the oral mucosa appears normal.

5) Oral parafunction

Parafunctional oral habits such as:

- Clenching
- Bruxing
- tongue posturing
- lip trapping
- sucking
- licking
- mouth breathing

have been proposed as causative in BMS. To date, studies do not support the assertion that parafunctional habits may cause BMS.

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6) Oral galvanism

The role of oral galvanism due to electrochemical potential differences between dissimilar metals (restorations and metal prosthesis) as a cause of BMS is rare, but has been reported in the literature.

7) Poorly designed dentures

Poorly designed dentures have been implicated as causative for BMS. Correction of tongue space deficiency because of a lingually positioned occlusal table or incorrect vertical dimension may benefit some patients.

8) Allergic reactions

- **Dental products**

  Allergic reactions to polymethylmethacrylate, epoxy curing agent, chromium, cobalt, nickel, cadmium, amalgam (mercury), gold, potassium, palladium and related materials in dental products.

- **Food producs**

  Food related products such as sorbic acid, propylene glycol, fragrance mix (eugenol, cinnamic aldehyde), benzoic acid, mint and cinnamon may cause allergic contact stomatitis (type IV hypersensitivity reaction) but are rarely implicated in BMS.

  Once again, there is a lack of clinical oral mucosa irritation in BMS patients. In cases that are confirmed by patch testing, cessation of exposure to these materials may result in improvement of burning symptoms.

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Systemic Factors

1) Haematinic disorder

Blood disorders associated with anaemias, including vitamin B group, iron and folate deficiencies are associated with a variety of oral manifestations including oral dryness, tongue papillary changes and burning pain.
Zinc deficiency has also been associated with burning oral mucosa.
Blood studies could be utilized to rule out these factors as the cause of the oral burning symptoms.

2) Autoimmune type connective tissue

Similarly, autoimmune type connective tissue disorders such as Sjogren's syndrome, sicca and systemic lupus erythematosus are associated with oral dryness and increased risk of candida infections that may cause oral burning.

3) Gastroesophageal reflux disease

Gastroesophageal reflux disease must be considered in any patient complaining of oral burning.

4) Endocrine-related disorders

Endocrine-related disorders, especially uncontrolled diabetes and thyroid disorders, along with hormonal deficiencies and alterations at menopause have also been associated with oral burning.

5) Medication side effects

Medications that may cause hyposalivation such as tricyclic antidepressants have been implicated, but the angiotensin converting enzyme (ACE) inhibitors, namely captopril, enalapril and lisinopril have been particularly associated with oral burning pain.

6) Central nervous system disorders

Central nervous system changes associated with conditions such as multiple sclerosis, Parkinson's disease and trigeminal neuralgia may be associated with oral neuropathic pain that may assume a burning nature. The prevalence of BMS has been suggested to be greater in patients with Parkinson's disease than in the general population, suggesting a role of dopaminergic pathways.

7) Idiopathic focal conditions

BMS has been linked with other "dynias", a group of idiopathic focal conditions with a predilection for the oro-cervical and uro-genital regions such as vulvodynia.

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BURNING MOUTH SYNDROME

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Psychosocial Factors

- The findings of high levels of psychological disturbances involving depression, anxiety, somatization and personality disorders are not unusual or unique to BMS patients.
- These are common findings in the chronic pain population and may contribute to the cause, intensity or urgency of complaint or may be the result of the constant pain.
- Furthermore, many of the medications used to treat these psychological conditions and personality disorders can cause side effects such as dry mouth and taste alterations that may induce or exacerbate oral burning symptoms.
- Therefore, the question remains whether psychological disturbances and personality disorders are aetiologically related to BMS or if chronic oral burning sensations initiate or exacerbate psychosocial disorders.

Differences between secondary BMS from primary BMS when a cause for the burning pain is illusive. it is difficult to draw definitive conclusions for a pragmatic approach for differentiating primary and secondary BMS. It is prudent for practitioners treating BMS to recognize possible local, systemic and psychological factors that may be responsible for oral burning and in turn manage the patient's symptoms appropriately.

Take away points:

TRUE BMS (primary/idiopathic

- Taste alteration: Taste disturbances, such as an alteration in taste perception (dysgeusia) and/or a persistently altered taste are often reported by BMS patients.
- Reduced pain while eating: a decrease or abortion of the pain while eating is commonly found in BMS patients.
- Lack of clinical oral mucosal irritation
- Oral mucosa appears normal

Reference: TG

- Burning mouth syndrome is an oral sensory disorder without a detectable cause; it is diagnosed when other conditions that can cause an oral burning sensation have been excluded.
- Burning mouth syndrome is more common in women, with the highest prevalence reported in women older than 70 years.
- The onset of burning mouth syndrome may be sudden following a specific event (e.g. dental treatment, a significant increase in personal stressors) or gradual and unrelated to any obvious event. Burning O mouth syndrome is often poorly diagnosed and managed.

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- Although symptoms of burning mouth syndrome vary, the characteristic symptom is a burning sensation of the tongue and, less frequently, the coincident hard palate and mucosal aspect of the lips.
- Symptoms can cause minor inconvenience or, in severe cases, prevent patients from conducting normal daily activities. In extreme cases, patients may exhibit suicidal tendencies.
- Most commonly, the burning sensation is mild in the morning and increases in intensity as the day progresses; this presentation has the best prognosis.

Other signs and symptoms associated with burning mouth syndrome include

- Parafunctional habits (e.g. Unconsciously rubbing the tongue against the adjacent teeth and the hard palate, which can cause traumatic abrasion of the filiform papillae on its dorsal surface)
- Dry mouth
- Halitosis
- Dysgeusia (most commonly a metallic taste)

Diagnosis and management of burning mouth syndrome

If burning mouth syndrome is suspected, the initial work-up is extensive and requires a detailed clinical history, including a dental, medical and medication history.

Because burning mouth syndrome is a diagnosis of exclusion, other causes of the patient's symptoms must be ruled out, such as:

- local causes (e.g. mucocutaneous conditions, fungal infections, rough dental surfaces)
- systemic causes
- hypersensitivity in patients who feel the problem is prosthesis-related (hypersensitivity can be identified with skin patch testing, but this is rarely required)
- drugs (e.g. drugs that cause sensory neuropathy, taste aberrations or salivary gland hypofunction)

The management of burning mouth syndrome is complex. The most important component of management is helping the patient to understand the condition (i.e. that burning mouth syndrome is a chronic neuropathic pain syndrome, irrespective of the likely initial triggers). Some patients may improve with discussion and counselling alone.

The most important component of managing burning mouth syndrome is helping the patient to understand the condition.

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The most important component of managing burning mouth syndrome is helping the patient to understand the condition.

- **lifestyle changes to modify a patient's response to external stressors (e.g. relaxation therapy, time management, exercise, community group participation)**
- **pharmacological management—topical or systemic use of psychotropic drugs (e.g. tricyclic antidepressants, antiepileptic drugs, clonazepam).**

Pharmacological management is the treatment chosen by most patients and requires specialist referral.

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PRE-ECLAMPSIA & PREGNANCY - ORAL MANIFESTATION & MANAGEMENT

Reference: Oral Health in Pregnancy

Pregnancy, the period from conception to birth, is characterised by profound hormonal changes. Fluctuation in hormones, particularly female steroid hormones (oestrogens and progesterone), influences many tissues in the body.

The tissues supporting the teeth, including the periodontium and especially the gingiva, are also affected. There are several reasons why dental professionals should focus on oral health in pregnant women.

The United States Surgeon General suggested (US Department of Health and Human Services 2000) that oral health treatment during pregnancy was an important strategy to:

- **maintain good oral health**
- **prevent the development of inflammatory diseases (gingivitis and periodontitis) and thus minimise any possible link to pre-term low birthweight infants, pre-eclampsia and gestational diabetes**
- **decrease oral bacteria colonisation, thus minimising transmission of bacteria to the child and decreasing the prevalence of Early Childhood Caries**

Common oral Problems in Pregnancy

- **Caries**
  Behaviours that may occur and may impact on caries risk are craving for and eating sugary foods and frequent ingestion or use of carbonated drinks to alleviate nausea.
- **Gingivitis**
  The accumulation of dental plaque may result in gingivitis, characteristically beginning in the 2nd month of pregnancy and increasing up to the 8th month, after which it declines. The effect of these changes on the periodontal tissues results in increased gingival swelling. Increased bleeding on probing may be seen in clinical examinations during pregnancy.
- **Periodontitis**
  Periodontitis is a multifactorial disease, with microbial dental plaque being the initiator. The initiation and progress of periodontal disease depend on the immunological response of the individual to the infection.
  The most important risk factors for development of periodontal disease are:
  - **Cigarette/cannabis smoking**
  - **age**
  - **stress**
  - **diabetes mellitus**
  - **high plaque levels**

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Reference: Oral Health in Pregnancy

Common oral Problems in Pregnancy

- **Pyogenic granuloma**
  Occasionally, localised gingival inflammatory enlargement, known as the pregnancy tumour, or pyogenic granuloma, can be found in up to 5% of pregnant women. Pyogenic granulomas bleed easily due to their highly vascular nature and may be painful. Smaller lesions sometimes regress with extra oral hygiene measures such as scaling and meticulous cleaning. However, if the lesion is causing problems due to size or discomfort, as long as there is no medical contraindication it can be excised. However, the patient would need to be warned of the risk of recurrence during the rest of the pregnancy again, meticulous oral hygiene would reduce the risk.
- **Erosion**
  Nausea and vomiting are the commonest symptoms consistently experienced in early pregnancy. Persistent vomiting may have an erosive effect on tooth structure, and pregnant women should be advised to have a drink of milk or water following a vomiting episode and not to brush their teeth immediately after vomiting.

Oral health and general health in pregnancy: emerging issues

- preterm birth
- low birth weight
- gestational diabetes
- Pre-eclampsia
- foetal growth restriction

Prevention of oral disease in pregnancy

- Standard preventive measures such as drinking of fluoridated water, twice daily use of fluoridated toothpaste and a low-sugar diet should be recommended for pregnant women.
- Fluoride supplements are not recommended in pregnancy as there is no evidence of effectiveness.
- A visit to the dentist is recommended for all pregnant women to check on periodontal conditions, as well as to minimise cariogenic oral flora through treatment of existing dental caries and advice on oral hygiene habits.
- Plaque control through meticulous oral hygiene is suggested for minimisation of gingivitis and to reduce the load of oral bacteria.
- Smoking cessation advice should be part of a preventive strategy for periodontal disease and for the range of conditions with which periodontal disease has been associated.

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Reference: Oral Health in Pregnancy

Dental treatment in Pregnancy

- Oral treatment during pregnancy is an important strategy to improve both maternal and infant oral health.
- However, dentists may be reluctant to provide dental treatment for a pregnant woman for a number of reasons, including;
  i. concern about tetragenicity of various medications such as anaesthetics and antibiotics.
  ii. concerns about treatment in early pregnancy because of the possibility of spontaneous abortion and premature labour in later pregnancy may be barriers to effective care.
- A recent clinical trial by Michalowicz et al. (2008) found that providing dental treatment between 13- and 21-weeks' gestation was not associated with any adverse pregnancy outcomes. The treatment provided included scaling and root planning with local anaesthesia, and emergency dental treatment including restorative and surgical care.
- This supports work by Daniels et al. (2007), who found that dental care during pregnancy, including amalgam fillings, was not associated with birth outcomes or language development.
- This is somewhat in contrast to the precautionary recommendation from the National Health and Medical Research Council (NHMRC) (1999) that 'During pregnancy it is prudent to minimise exposure to all foreign substances including materials used in dental restorations. This indicates that placement or replacement of dental amalgam restorations should be avoided, especially during the first trimester'.
- Routine dental treatment can be undertaken quite safely in the second trimester.
- Pregnant women may be uncomfortable lying on their backs for long periods in the third trimester. Emergency treatment can be undertaken at any time with appropriate precautions.

Reference: Periodontitis and Preeclampsia in Pregnancy: A Systematic Review and Meta-Analysis

Localized periodontal inflammation spreads systemically during pregnancy inducing adverse pregnancy outcomes. Periodontitis appears as a significant risk factor for preeclampsia, which might be even more pronounced in lower-middle-income countries.

Reference: Periodontal diseases and adverse pregnancy outcomes: Shihoko Komine-Aizawa1, Sohichi Aizawa1,2 and Satoshi Hayakawa1

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Reference: Oral Health in Pregnancy

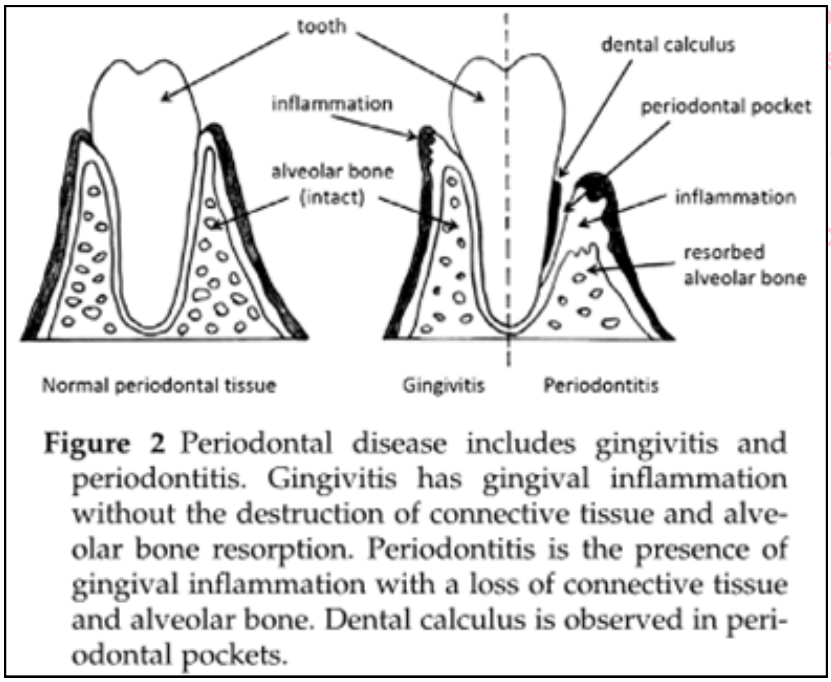

Figure 2 Periodontal disease includes gingivitis and periodontitis. Gingivitis has gingival inflammation without the destruction of connective tissue and alveolar bone resorption. Periodontitis is the presence of gingival inflammation with a loss of connective tissue and alveolar bone. Dental calculus is observed in periodontal pockets.

Reference: Periodontitis and Preeclampsia in Pregnancy: A Systematic Review and Meta-Analysis

Periodontitis has independently been linked to several pregnancy complications such:

- preterm birth
- low birth weight
- gestational diabetes
- Pre-eclampsia
- foetal growth restriction

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PRE-ECLAMPSIA & PREGNANCY - ORAL MANIFESTATION & MANAGEMENT

Reference: Oral Health in Pregnancy

Pre-Eclampsia

Preeclampsia **is the onset of pregnancy-related hypertensive disorder and proteinuria arising most commonly after 20 weeks of gestation, which could lead to eclampsia and induce maternal and perinatal morbidity, and mortality.**

Reference: Oral Health in Pregnancy

There are multiple risk factors for pre-eclampsia including pre-existing diabetes and first pregnancy.
It was found that women were at higher risk of pre-eclampsia if they had severe periodontal disease or progression of periodontal disease during pregnancy. It was hypothesised that periodontal disease contributes to placental inflammation. It is unclear whether the relationship between periodontal disease and pre-eclampsia is an association that is due to factors related to both conditions independently, and whether there is a causal linkage. Other studies have not found this relationship.

Reference: Periodontitis and Preeclampsia in Pregnancy: A Systematic Review and Meta-Analysis

Higher levels of some periodontal pathogens such as P.gingivalis and F. nucleatum were found in placenta of patients with preeclampsia.
Ananth et al. has reported the association between intrauterine growth restriction and maternal periodontitis. Since severe and early onset preeclampsia were associated significantly with foetal growth restriction, this could contribute to the mechanism underlying the association between preeclampsia and periodontitis.
Recently, some evidence has indicated that pathogenesis of preeclampsia involves maternal gut microbiota, specifically, high-fibre diet which promote short chain fatty acid production and are associated with reduced risk of preeclampsia.
Similarly, high-fibre foods such as fruit and grains have been linked to the reduction of the progression of periodontal disease, suggesting the role of dietary intake in the potential relationship between preeclampsia and periodontal disease.
Pregnant women in low socioeconomical areas should be given access to oral healthcare services and encouraged to have their periodontal health checked and treated during pregnancy to potentially lower the risk of preeclampsia and other pregnancy complications.
Jeffcoat et al. reported non-surgical periodontal therapy could significantly reduce the medical costs for pregnant women by 73.7%.

Reference: Periodontal diseases and adverse pregnancy outcomes: Shihoko Komine-Aizawa1, Sohichi Aizawa1,2 and Satoshi Hayakawa1

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Reference: Oral Health in Pregnancy

Pathogenic Mechanisms of Periodontal Diseases and APO (adverse pregnancy outcome)

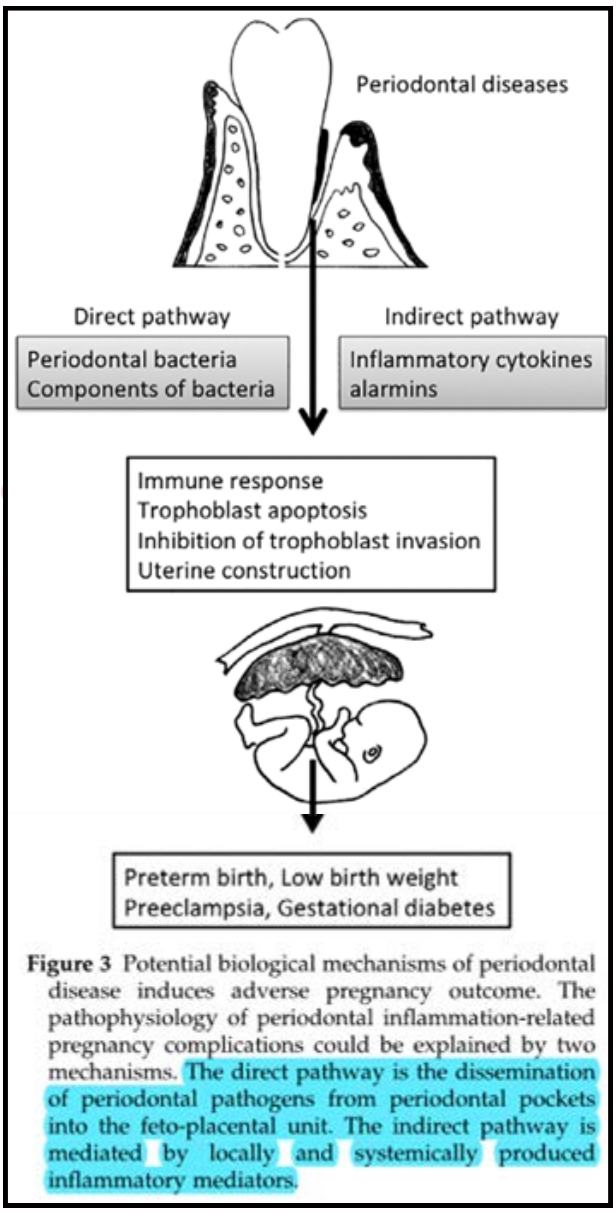

Figure 3 Potential biological mechanisms of periodontal disease induces adverse pregnancy outcome. The pathophysiology of periodontal inflammation-related pregnancy complications could be explained by two mechanisms. The direct pathway is the dissemination of periodontal pathogens from periodontal pockets into the feto-placental unit. The indirect pathway is mediated by locally and systemically produced inflammatory mediators.

Currently, two mechanisms are considered: the direct and indirect pathways (Fig. 3).

The oral microbiome and pathogenic periodontal bacteria are considered to disseminate to the feto-placental unit. Therefore, the direct roles of oral bacteria on APO have been investigated using experimental animal models and in vitro models. Another possible mechanism is that inflammatory mediators produced by infected periodontal tissue affect the feto-placental unit and myometrium.

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PRE-ECLAMPSIA & PREGNANCY - ORAL MANIFESTATION & MANAGEMENT

Reference: Oral Health in Pregnancy

Conclusion

Multiple factors are associated with the incidence of APO, and periodontal disorders are an independent risk factor, according to epidemiological and experimental studies.
Periodontal bacteria, especially P. gingivalis, and their components can injure the trophoblast morphologically and functionally.
Moreover, inflammatory mediators from periodontal pockets might elicit an inflammatory immune response at the feto-placental unit.
However, periodontal treatment during pregnancy seems to have little effect on the prevention of APO incidence.
Trophoblast cells migrate into the uterine myometrium and reconstruct the uteroplacental sinus during the early period of pregnancy, and the placental structure is completed in the first trimester.
Therefore, although dental care is effective in curing periodontal diseases, dental care during pregnancy may occur too late to reduce pregnancy complications.

Reference: Pregnancy, parity and periodontal disease EL Morelli, JM Broadbent, JW Leichter, WM Thomson

Pregnancy, parity and periodontal disease

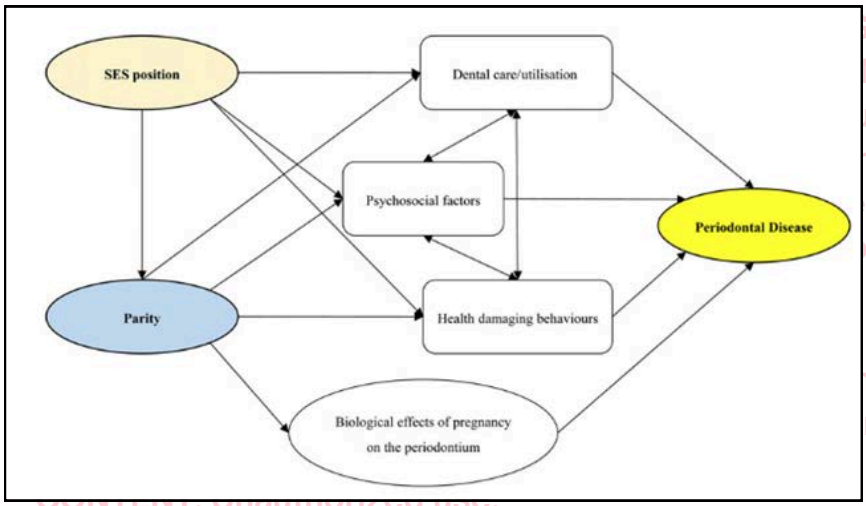

Fig. 1 Proposed theoretical model of the association between parity and periodontal disease. SES = socioeconomic status. Adapted from Russell et al. (2008).

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DRY MOUTH

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Xerostomia and hyposalivation are both conditions that can cause dry mouth, but they are distinct:

- **Xerostomia: The subjective feeling of having a dry mouth**
- **Hyposalivation: An objective reduction in the amount of saliva produced, also known as salivary gland hypofunction. Measured by sialometry. (stimulated salivary flow rate= 1-2ml/min; unstimulated salivary flow rate = 0.3 to 0.4 mL/min; An unstimulated salivary flow rate of 0.1 to 0.2 mL/min and stimulated flow rate of 0.7 mL/min or less is considered indicative of salivary gland hypofunction)**
- **A lack of lubrication with saliva predisposes the oral mucosa to friction and pain often of burning quality.**

Reference: TG

The subjective feeling of dry mouth (xerostomia) is a relatively common condition that may or may not occur in the context of salivary gland hypofunction (an objective reduction in the quantity and the quality of saliva).
Many physiological and pathological conditions and drugs can cause salivary gland hypofunction or dry mouth.

Common causes of dry mouth include:

- **dehydration**
- **alcohol**
- **anxiety**
- **mouth breathing**
- **drugs**

If common causes have been excluded, investigate for less common medical conditions associated with dry mouth (e.g. Sjogren syndrome).

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Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Drugs frequently associated with dry mouth

- anticholinergic drugs
- antihistamines
- drugs to lower blood pressure
  -angiotensin converting enzyme inhibitors
  -angiotensin II receptor blockers
  -alpha blockers
  -beta blockers
  -diuretics
- inhaled bronchodilators
  -beta2 agonists (e.g. salbutamol)
  -muscarinic antagonists (e.g. tiotropium)
- opioids
- psychotropic drugs
  -antidepressants
  -antipsychotics
  -illicit drugs (e.g. marijuana, cocaine)
  -psychostimulants (e.g. amfetamines)
- urinary antispasmodics

Dry mouth is likely to be more severe if these drugs are used in combination.

Dry mouth is a debilitating adverse effect of head and neck radiotherapy with the degree of salivary flow reduction dependent on the dose and region of the radiation.

Chronic dry mouth can have a profound effect on the oral environment and can contribute to:

- tooth decay and erosion
- periodontal disease
- oral mucosal disease
- oral candidiasis
- difficulty with the retention of dentures
- difficulty with chewing, swallowing and speech
- altered sense of taste.

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Management of dry mouth

Encourage patients to have a dental review and any necessary dental treatment before starting a drug that can cause dry mouth.
Review the patient's medications and, in conjunction with the prescriber, stop any non-essential medications that can cause a dry mouth.

Management strategies for patients with dry mouth include:

- ensuring adequate hydration
- ensuring good oral hygiene
- regular dental examination and treatment every 3 to 6 months
- topical remineralising agents to prevent tooth decay
- symptomatic relief.
- Options for the symptomatic relief of dry mouth include:
- artificial salivary products or other oral lubricants (e.g. bicarbonate mouthwash) (however, effects may be too transient to be of significant benefit)
- products that stimulate saliva, such as throat lozenges or chewing gum (however, many products are acidic or have a high sugar content, which can cause further tooth decay).

<u>If symptomatic measures are inadequate, or if dry mouth is a symptom of systemic disease, refer patients to an appropriate specialist.</u>

Practical advice for patients with dry mouth

To manage your dry mouth:

- ensure you are adequately hydrated—drink at least 1.5 litres of tap water a day
- chew food thoroughly before swallowing because chewing stimulates saliva flow
- chew sugarless gum or suck sugarless sweets (avoid fruit flavours)
- avoid smoking cigarettes
- avoid acidic foods
- limit your caffeine and alcohol intake, especially in the evening
  -add milk to tea or coffee to reduce the drying effect
- avoid mouthwashes and other oral preparations that contain alcohol
- trial various over-the-counter dry mouth products or bicarbonate mouthwash
  -a bicarbonate mouthwash can be made by adding half a teaspoon of bicarbonate powder to a glass of warm water. Rinse with mouthwash on waking and at any time during the day.

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To prevent oral and dental consequences of dry mouth:

- ensure you have good oral hygiene
- have regular dental examinations
- avoid acidic beverages (e.g. wine, fruit juices, soft drinks, sports drinks) or limit their consumption to meal times
- limit your sugar intake and avoid sugary snacks.

Reference: Oral care: xerostomia (dry mouth) and oral drug-induced effects

Dry mouth is also a common condition in patients receiving palliative care.

Dry mouth is also a common adverse effect of drugs such as marijuana and heroin. Many people with drug dependency have severe dental disease with discoloured blackish teeth and do not prioritise any action for dental treatment.
In addition, injecting drug users have a higher prevalence of hepatitis C infection than the general community. Hepatitis C also causes dry mouth.

Common diseases states that may cause dry mouth

1) Sjogren syndrome

- It is an autoimmune disease of unknown origin associated with inflammatory infiltration of the exocrine glands, particularly the salivary and lacrimal glands, leading to secretory gland dysfunction and, usually severe sicca symptoms of dry eyes and or dry mouth (sicca is a term used to refer to dry eyes and mouth).
- Sjögren's syndrome affects many organ systems in the body.
- However, for dental practitioners it is important to recognize the many oral and dental manifestations that are associated with the syndrome. In addition dental practioners should also recognise the systemic nature of the disease.
- In severe cases, the dryness can cause salivary gland enlargement and calculus formation.
- It can also affect the trachea causing dry cough and a hoarse voice. Rarely, loss of gastrointestinal exocrine function can cause pancreatic dysfunction or pancreatitis, and atrophic gastritis.
- Sjogren syndrome may be primary or secondary.
- Patients with primary Sjogren syndrome often have fatigue, arthralgia and a non-erosive arthritis, as well as Raynaud phenomenon.Primary SS does not occur with any other systemic autoimmune disease.
- However, secondary Sjogren, occurs in association with other autoimmune disorders such as rheumatoid arthritis, systemic lupus erythematosus (SLE) or systemic sclerosis. Most commonly with rheumatoid arthritis.

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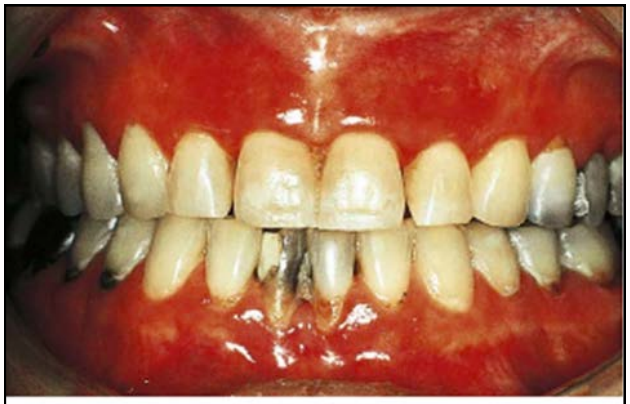
Epidemiology:

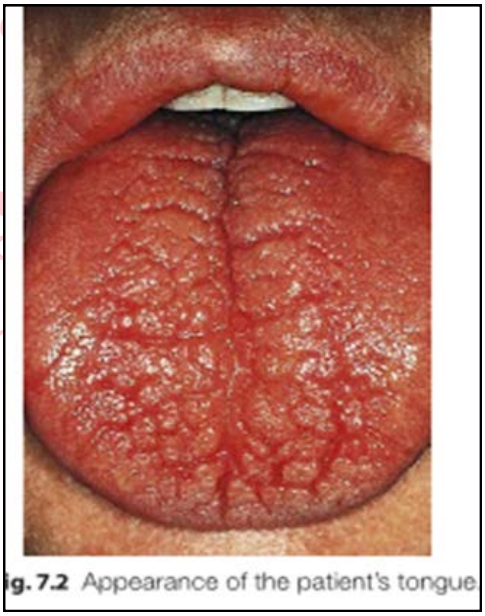
- **It can affect all ages but mostly seen in menopausal women in the fourth and fifth decades of their life.**
- **SS is more prevelant in women than man (9:1) predilection**

> undiagnosed.[9] Early onset SS (onset less than 35 years of age) has been associated with more severe signs and symptoms.[4] Interestingly, approximately one-third of patients with some form of autoimmune disorder concurrently suffer from SS.[1,10]

- **Its exact aetiology and pathogenesis is still unclear but it is well established that it is multifactorial.**
- **The aetiology though not completely understood the series of 4 events is what occurs mostly in all SS patient:**
  a) **Initiation by exogenous factors**
  b) **Disruption of salivary gland epithelial cells**
  c) **T lymphocyte migration and lymphocytic infiltration of exogenous glands**
  d) **B lymphocyte hyperactivity and production of rheumatoid factor and antibodies to Ro (SS-A) and La (SS-B)**



• **Fig. 7.1** Appearance of the patient's anterior teeth.



ig. 7.2 Appearance of the patient's tongue

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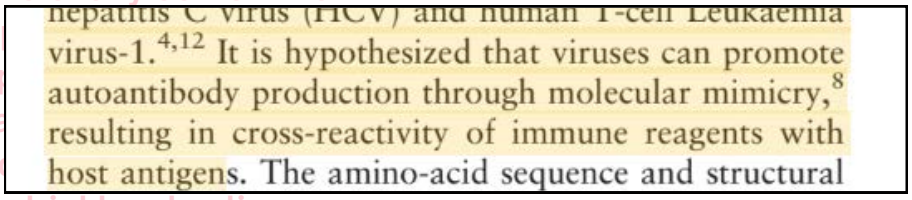
Risk factors for sjogrens syndrome:

1. Genetic predisposition:

- It has been suggested by multiple reports that there is genetic predisposition to the syndrome where 2-3 members of the same family are affected by the syndrome.
- So, a family history of the disease puts a person at increased risk of developing SS.

2. Viral initiating factor:

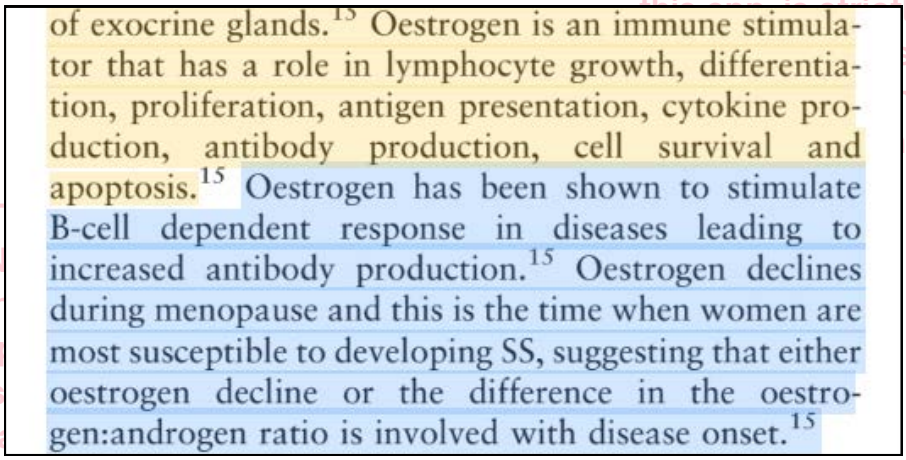
- Viruses potentially involved in the etiology of SS are:
  i. Epstein Barr virus
  ii. Hepatitis C virus (HCV)
  iii. Human T cell leukemia virus-1



hepatitis C virus (HCV) and human T-cell Leukaemia virus-1.[4,12] It is hypothesized that viruses can promote autoantibody production through molecular mimicry,[8] resulting in cross-reactivity of immune reagents with host antigens. The amino-acid sequence and structural

3. Sex hormones:

- The high percentage of females with SS when compared to men suggests that immune regulatory properties of sex hormones are involves in its development.
- The ratio of androgen to oestrogen modulates the cellular immune response which is involved in destruction of exocrine glands.



of exocrine glands.[15] Oestrogen is an immune stimulator that has a role in lymphocyte growth, differentiation, proliferation, antigen presentation, cytokine production, antibody production, cell survival and apoptosis.[15] Oestrogen has been shown to stimulate B-cell dependent response in diseases leading to increased antibody production.[15] Oestrogen declines during menopause and this is the time when women are most susceptible to developing SS, suggesting that either oestrogen decline or the difference in the oestrogen:androgen ratio is involved with disease onset.[15]

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Pathogenesis of Sjogren syndrome:

- Auto antibodies are found in the serum of both primary and secondary SS
- Most common are Ro/SS-A and La/SS-B that are found 60-70%in primary SS.
- The presence of autoantibodies is linked with earlier onset of the disease, increased disease severity, longer duration of the disease, recurrent parotid gland enlargement and extra glandular manifestation.
- Hence, the presence of autoantibodies can be used as a predictor of disease severity in newly diagnosed patient.

> newly diagnosed patients. Patients with younger onset SS demonstrated higher serum levels of Ro/SSA and La/SSB antibodies and higher rheumatoid factor. This was associated with more severe clinical symptoms in the younger onset group of patients.[7]

- Chronic infiltration of lymphocyte and subsequent infiltration of salivary gland acini. A classic sign of Sjogren is focal lymphocytic infiltration of salivary gland.

Diagnosis of SS

- The diagnosis of SS is not straightforward as many symptoms are subjective and vague, which can be dismissed as other conditions or effect of medication.
- The estimated average of interval between initial symptoms and diagnosis of disease is 6-10 years due to difficulty in diagnosis due to vague symptoms.
- The common vague symptom that patients mostly initially present with are arthralgia, fatigue and extra glandular complications.
- Differential diagnosis of SS includes:

Table 1. Differential diagnosis of Sjögren's syndrome[3]

Drug therapy	Anticholinergic drugs
Past treatments	Past head and neck radiation treatment
Systemic disease	Sarcoidosis
	Hep C
	HIV/AIDS
	Graft-versus-host disease
	Pre-existing lymphoma
	Rheumatoid arthritis
	Systemic lupus erythematosus
	Scleroderma
	Primary biliary cirrhosis
	Diabetes mellitus
	Cytomegalovirus and other herpes viruses
	Ectodermal dysplasia

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Diagnosis of SS

- **The current method of diagnosis is the set of criteria that has been established which is the gold standard for diagnosis of SS**

Table 2. Revised International Classification Criteria for Sjögren's syndrome[2,10]

Criteria	
I	Ocular symptoms: at least one of the following 1. Daily dry eyes for >3 months 2. Persistent sensation of sand or gravel 3. Use of tear substitutes >3 times daily
II	Oral symptoms: at least one of the following 1. Dry mouth daily >3 months 2. Recurrent salivary gland swellings 3. Use of liquid to aid in swallowing food
III	Ocular signs: at least one of the following 1. Schirmer's I test (≤5 mm in 5 min) 2. Rose Bengal score (≥4)
IV	Histopathology: focal lymphocytic sialoadenitis with focus score ≥1 per 4 mm^2 of tissue
V	Salivary gland involvement: at least one of the following 1. Unstimulated salivary flow ≤1.5 ml/15 min 2. Abnormal parotid sialography 3. Abnormal salivary scintigraphy
VI	Autoantibodies: presence of Ro(SSA) or La(SSB) or both, in serum

- **METHOD of diagnosis of Four objective criteria are**

Table 3. Diagnostic tests for Sjögren's syndrome[2,3,10,14,21,23]

Testing for opthalmic involvement
Schirmer's I test: quantitative measure of tear production over a specific period of time
Rose Bengal eye stain: reveals breaks in the corneal-epithelial surface to evaluate ocular surface irritation
Patient history of opthalmic symptoms
Testing for oral involvement
Salivary sialometry: low salivary flow is defined as less than 1.5 ml of saliva per 15 minutes
Labial minor salivary gland biopsy: showing lymphocytic sialoadenitis with a focus score of ≥1 per 4 mm^2 of tissue
Examination for salivary gland enlargement: parotid and/or submandibular
Patient history of oral symptoms
Systemic tests
Presence of Ro/SSA and La/SSB autoantibodies in patients serum
Presence of rheumatoid factor in patients serum

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Investigations for Sjogren's include:

TABLE 7.1 Investigations for Patients with Sjögren's Syndrome

Sample	Test	Relevance
Saliva	Whole salivary flow rate	See above; differentiates false from true xerostomia.
	Culture for candidal count	To exclude superimposed candidosis.
	Stimulated parotid flow	Accurate estimation of maximum possible parotid salivary flow.
Blood	Full blood picture	Mild anaemia is common in all autoimmune conditions and may require treatment.
	Erythrocyte sedimentation rate (ESR)	Relatively nonspecific, but raised in inflammatory conditions, and when there are raised immunoglobulins, especially IgG in Sjögren's syndrome, useful for monitoring their activity after treatment.
	Immunoglobulin (Ig) levels	Often raised in autoimmune disorders and may be markedly raised in primary Sjögren's syndrome.
	Autoantibody screen	Autoantibodies are a frequent finding in autoimmune disease. This appears to be a partly nonspecific effect, and many different autoantibodies may be seen. The exact combination in routine screening varies among centres but usually includes rheumatoid factor, antinuclear, antithyroid, antiparietal cell and antimitochondrial antibodies. Additional autoantibodies that may be seen in Sjögren's syndrome are anti–salivary gland duct antibody and ssA and ssB autoantibodies (anti-Ro and anti-La) directed against extractable nuclear antigens. None of these antibodies is individually helpful in diagnosis, but the presence of more than one is typical. They may aid in the diagnosis of connective tissue disease in secondary Sjögren's syndrome, and ssA and ssB may indicate patients at risk of specific complications. Anti–salivary gland duct antibody is not related to either the periductal infiltrates seen on biopsy or the pathogenesis of the disease.
Urine	Glucose	Occasionally useful to exclude unsuspected diabetes as a cause of dehydration.
Salivary gland	Ultrasonography Sialography	In established disease, ultrasonography almost always shows characteristic changes. Ultrasonography has almost replaced sialography for in investigating Sjögren's syndrome. However, sialography is still useful if salivary stones or strictures are suspected because of additional swelling during eating.
	Other imaging techniques	Pertechnetate scintigraphy is a complex but useful test of secretion from individual glands. It is useful if sialography is not possible but involves a significant dose of radiation. Magnetic resonance imaging is useful to delineate the extent of salivary gland swelling, if present.
	Minor salivary gland biopsy	The histological appearances of salivary glands are characteristic in established disease. Biopsy of major glands is difficult, but the same changes may be seen in the minor glands of the lips and cheeks, provided a sufficient sample is removed (6–8 glands).
	Parotid gland biopsy	Biopsy of the tail of the parotid is possible without significant risk to the branches of the facial nerve. It provides an excellent sample and may be useful when other techniques have failed or when other conditions need to be excluded. It may also be helpful in the diagnosis of lymphoma in swollen parotid glands. Core biopsy taken under imaging guidance is performed more easily compared with an open biopsy, which is rarely undertaken unless indicated for clarifying whether another disease such as IgG4 disease or a lymphoma is present.
Eye	Schirmer test	This measures lacrimal secretion. Narrow filter paper strips are placed with one end under the lower eyelid and the length wetted is recorded after 5 minutes. In practice, the test is not very reproducible. (It is also uncomfortable and may cause corneal abrasions when the eye is very dry and, for this reason, is no longer recommended.) Ophthalmological examination is preferable, but the Schirmer test remains widely used.
	Ophthalmological examination	An ophthalmologist uses a slit lamp to detect conjunctival splits and Lissamine Green staining identifies dried tear secretion on the front of the eye. Although these changes are rarely helpful in diagnosis, examination and follow-up are required to prevent long-term complications of dry eyes.

Reference: Oral care: xerostomia (dry mouth) and oral drug-induced effects

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Complication associated with Sjogren's syndrome:

- SS is associated with many systemic conditions like Raynaud's Phenomenon, lymphadenopathy, peripheral neuropathies, but the severity varies between patients.
- The most serious and well-established complication of SS is the increased incidence of malignant lymphoma (lympho- proliferative disease) 44 times higher than general population.
- Other symptoms that are associated with pts with SS are:

a. Dryness of nose, skin and vagina
b. Recurrent sinusitis
c. Chronic cough from dryness of trachea
d. Recurrent oral candidiasis from salivary gland hypofunction
e. Chronic Keratoconjunctivitis sicca leading to irregular surface of cornea causing deteriorating vision and increased risk of recurrent infections.
f. SS may also have extraglandular symptoms that can affect kidney, liver, CNS, and joints

Table 4. Symptoms associated with extraglandular manifestations of Sjögren's syndrome[26]

Malaise	Peripheral neuropathy	Primary biliary cirrhosis
Fatigue	Autoimmune thyroiditis	GI symptoms
Fibromyalgia	Renal tubular acidosis	Respiratory diseases
Fever	Myositis	Psychosis
Arthralgia	Chronic hepatitis	Lymphadenopathy
Synovitis	Purpura	Splenomegaly
Raynaud's phenomenon	Vasculitis	Lymphoma

g. 45% of patients with SS have associated thyroid dysfunction

about 33% of cases.

Respiratory disease may occur early in the course of SS but these diseases often have a long, chronic clinical course.[5] Cough is the most common symptom and this is usually indicative of a dry trachea as the exocrine gland secretions have been adversely affected.[9]

Raynaud's phenomenon is a vascular condition where there is fluctuating vasoconstriction of the blood vessels in the extremities such as fingers and toes[1] and is seen in approximately 13% of patients with SS, but is also commonly associated with scleroderma. This suggests that SS and scleroderma need to both be considered when this phenomenon occurs and care taken to explore all differential diagnoses.[1]

Glomerulonephritis is seen commonly in SS, with one study showing a prevalence of 55%.[26] It is, therefore, a serious complication of SS as it can lead to an increased risk of mortality. Glomerulonephritis has also been

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Complication associated with Sjogren's syndrome:

g. • **There is also increase incidence of psychological disorders and depression in patients with SS.**

> The incidence of psychological disorders and depression are high in patients with SS.[25] This may be related to the downturn in quality of life elicited by the symptoms of the disease. Xerostomia has a severe impact as patients find eating, speaking, swallowing and sleeping difficult. Patients with oral removable prostheses may have difficulty retaining dentures. The development of oral ulcers and mucosal inflammation is common. The incidence of debilitating fatigue in approximately 50% of patients, may also contribute to the depression commonly seen in patients with SS, particularly the elderly.[9] Patients often struggle with social withdrawal, economic burden and poor nutrition as a result of difficulty eating.[4]

Oral Complications of SS:

- **SS is a progressive disease that shows deteriorating lacrimal and salivary secretions over time.**

1. Decreased Salivary Flow:

- **It has many effects on oral cavity.**
- **In SS the gingiva and mucosa are not protected by salivary mucins, leading to less lubrication of tissues. This can cause signs such as oral mucosal inflammation, mucosal sloughing, erythematous mucosa and traumatic ulcers.**

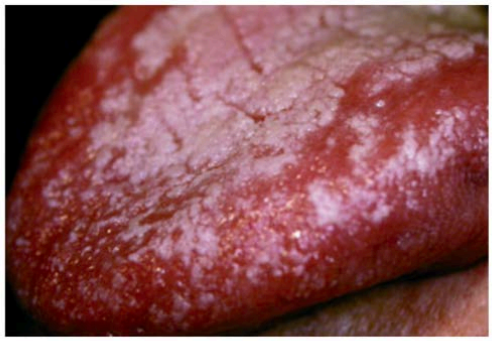

Fig 1. Erythematous mucosa in a patient with Sjögren's syndrome.

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Oral Complications of SS:

1. Decreased Salivary Flow:

- Depapillation of tongue in advanced disease cases may be seen.

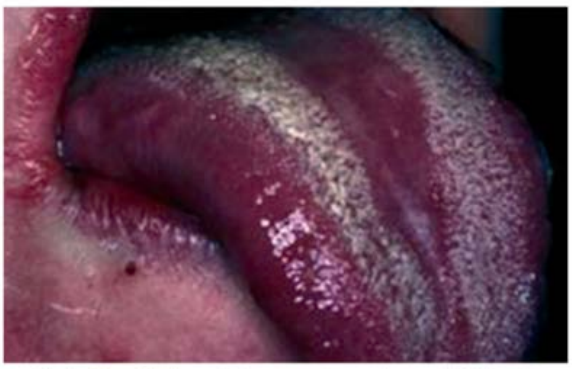

Fig 2. Depapillation of the tongue in a patient with Sjögren's syndrome.

- The decrease in salivary flow can lead to an inflammatory response of gingiva , manifesting as oedema, inflammation or recession as a result of poor oral lubrication and less clearance of food and plaque particles by saliva.
- Denture retention is a problem due to dry mouth.
- Opportunistic injections such as Candidiasis is increased, as the protective molecules of saliva is decreased.

2. Candida infection

- It is often present as atrophic or erythematous candidiasis and is associated with burning mouth and is reported by approx., 1/3rd of patients with SS.
- The prevalence of candida albicans is more than 68% in patients with SS whereas only 23- 68 % in general population

3. Smooth surface caries:

- The incidence of caries is increased with decreased salivary flow.

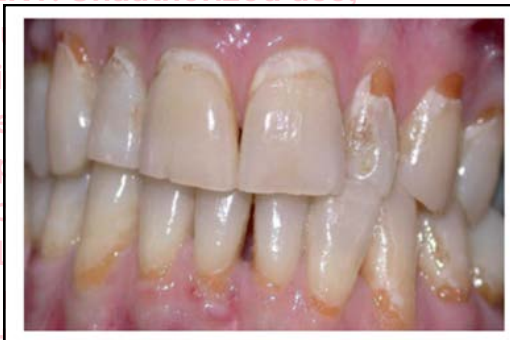

Fig 3. Smooth surface caries resulting from salivary gland hypofunction.

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Management of SS and treatment options:

- **As a general dentist, it is essential to treat oral symptoms and prevent the oral disease which is prevalent in SS patients.**
- **An individualized treatment regime is important to best manage patient's oral problems.**

Suggestions by various reports are:

Table 5. Management of salivary deficit[2,3,8,18]

Regular sipping of water
Artificial saliva substitutes
Sugar-free chewing gum
Avoidance of anticholinergic drugs if possible
Pharmacological treatments (not yet available in Australia) including pilocarpine and cevimeline

Table 6. Prevention of oral disease in Sjögren's syndrome patients[2,8]

Regular examinations and recalls
Professional topical fluoride applications
Home fluoride regimes tailored to individual patient
Monitoring and reinforcement of oral hygiene instruction
Regular water intake
Sugar-free chewing gum

- **As the patients are at increased risk of caries, they need to be seen more regularly for examination and preventive treatments such as home fluoride regimens**
- **Patients also need to follow excellent oral hygiene measure which should be regularly reinforced by the dentist.**

regularly reinforced by the dental practitioner.[8] Patients should be advised to drink water regularly to: (a) help lubricate the oral tissues; (b) increase saliva flow where possible; and (c) aid in balancing the oral pH to lower caries risk. Artificial saliva substitutes may be used to enhance oral comfort and aid in tissue lubrication.[8]

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DRY MOUTH

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Management of SS and treatment options:

- **Candida should be managed by topical antifungal treatments (as per TG)**
- **Patients should be advised to avoid anticholinergic agents if possible.**
- **Pilocarpine and cevimeline are drugs that have shown subjective and objective improvement in both saliva and tear flow rate.**

Pilocarpine mimics acetylcholine and acts as a messenger between cholinergic neural cells and glandular acinar cells[18] and may prevent apoptosis.[9] It was reported that the effect of pilocarpine lasts for approximately two to three hours when taken orally. Another study found that using a pilocarpine mouthwash increased salivary flow for 75 minutes.[3] The mouthwash is considered a better option as it is a topical application and its absorption systemically is much reduced from the oral form. This then reduces the possible systemic side effects such as increased motility of the gastrointestinal tract and increased smooth muscle tone.[3] However, the action of pilocarpine relies on there being residual unaffected portions of the exocrine glands, therefore it is most effective in the early stages of disease when fibrosis of the acinar cells has just begun.

Cevimeline is a newer drug that is an acetylcholine analogue stimulating muscarinic M3 receptors on lacrimal and salivary glands. Studies show that cevim-

- **Early diagnosis of SS is essential so that oral complications can be minimised and appropriate management initiated both medically and dentally.**
- **Cooperative approach between all health professionals involved in patients care is essential for appropriate management of such cases.**

(REFERENCE: Oral care: xerostomia (dry mouth) and oral drug-induced effects)

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DRY MOUTH

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

Common diseases states that may cause dry mouth

2) Radiotherapy:

Dry mouth may also be a direct effect of head and neck radiotherapy, with the degree of salivary flow reduction dependent on the dose and region of the radiation.

3) Rheumatoid arthritis

Patients may also suffer from dry mouth. These patients may also suffer with dry eyes, nose and skin.

4) Scleroderma patients and other inflammatory connective tissue diseases:

These patients may be affected by dry eyes and/or dry mouth.

5) Sleep apnoea

It is a common cause of dry mouth. In addition, the use of the continuous positive airway pressure (CPAP) machines may cause dry mouth. Regular use of artificial saliva products can be of value. Dry mouth in patients using CPAP may also indicate significant mouth leak and chin straps should be trialled. Humidification of the CPAP machine will not relieve dry mouth while a mouth leak exists. Reduction in CPAP pressure should be considered.

6) Palliative care:

Dry mouth is a common condition in palliative care. It is exacerbated by mouth breathing due to anxiety, as well as medications. Prolonged dry mouth will result in increased dental decay with pain and loss of teeth, significant worsening of periodontal disease and any underlying mucosal disease, increased risk of oral candidiasis, and significant difficulty with the retention of dentures. There may also be difficulty with chewing, swallowing and speech.

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DRY MOUTH

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

TABLE 2: **SOME MEDICINES CAUSING DRY MOUTH**[2-10]

DRUG CLASS	EXAMPLES
Alcohol	
Antidepressants	Tricyclic antidepressants, some SSRIs (e.g. citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine), some SNRIs (e.g. duloxetine) and other antidepressants (e.g. mirtazapine).
Antihistamines (first generation with anticholinergic effects)	Brompheniramine, chlorpheniramine, cyclizine, cyproheptadine, dexchlorpheniramine, dimenhydrinate, diphenhydramine, pheniramine, promethazine, trimeprazine, triprolidine
Antipsychotic medicines (due to anticholinergic effects)	Amisulpride, aripiprazole, asenapine, chlorpromazine, clozapine, haloperidol, lurasidone, olanzapine, paliperidone, quetiapine, risperidone, ziprasidone
Benzodiazepines	Class effect, e.g. diazepam
Betablockers	e.g. atenolol
Caffeine	In coffee, also combined with paracetamol
Medicines with anticholinergic effects (plus first generation antihistamines)	Aclidinium, amantadine, amitriptyline, atropine, belladonna alkaloids, benzhexol, benztropine, biperiden, chlorpromazine, clomipramine, cyclopentolate, darifenacin, disopyramide, dothiepin, doxepin, glycopyrronium, homatropine, hyoscine (butylbromide or hydrobromide), imipramine, ipratropium, mianserin, nortriptyline, orphenadrine, oxybutynin, pericyazine, pizotifen, prochlorperazine, propantheline, solifenacin, tiotropium, tolterodine, tropicamide, umeclidinium
Methadone	Pharmacotherapy replacement programs. Dry mouth leading to significant salivary hypofunction and development of dental caries
Nicotine	Nicotine replacement therapy, smoking
Opioids	Class effect, e.g. buprenorphine, codeine, fentanyl, hydromorphone, methadone (see above), morphine, oxycodone, tapentadol, tramadol
Others	Acitretin, bupropion, clonidine, dexamphetamine, domperidone, entacapone, isotretinoin, methylphenidate, pramipexole, pseudoephedrine

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DRY MOUTH

Reference: Separating oral burning from burning mouth syndrome: unravelling a diagnostic enigma ARTICLE

TABLE 2: **SOME MEDICINES CAUSING DRY MOUTH**[2-10]

PRODUCT	COMMENTS
ANTIPLAQUE/ANTIBACTERIAL PREPARATIONS	
Chlorhexidine 0.2% mouthwash, alcohol free	• Dilute 5mL mouthwash with 5mL water, rinse twice daily • Can be used by people having concurrent chemotherapy and by people with chemotherapy-induced mucositis • Limits exposure to water-borne pathogens • May assist with cleansing of mucositis and shifting of mucous plaques on mucosal surfaces • Oral hygiene adjunct
Chlorhexidine 0.2% gel, alcohol-free	• Apply when needed to all mucosal surfaces and gingival margins • Oral hygiene adjunct: provides lubrication and eases discomfort
ANTI-INFLAMMATORY AND PAIN-RELIEF PREPARATIONS	
Benzydamine hydrochloride 0.15% solution (Difflam)	• Rinse 10–15ml and spit out, 4–6 times daily • Provides some pain relief • May reduce frequency and severity of oral mucositis in patients with head and neck cancer receiving radiotherapy
Lignocaine 2% viscous (Xylocaine Viscous)	• Rinse 10–15mL and spit out, 4-hourly • Provides some pain relief
LUBRICATION PREPARATIONS	
Lip balm with chlorhexidine	• Apply as necessary, useful for lip mucositis
Artificial saliva products (e.g. Aquae spray or gel; Biotene oral balance gel, Oral-7 Gel, Oralube spray)	• Use as required • Transient relief of oral dryness up to 4 hours • pH neutral
DentaMed Gel	• Use in place of tooth paste • Hydrates the oral mucosa • Best results if used regularly at night • pH neutral
Oral rinses (e.g. Biotene moisturising oral rinse, Biotene dry mouth oral rinse, Oral-7 mouth wash)	• Can be used up to five times a day • Soothes and moisturises for up to 4 hours. • Alcohol-free and sugar-free • pH neutral

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SALIVA STIMULANTS	
Pilocarpine (e.g. Xylitol chewing gum, Recaldent chewing gum)	• Side effects of pilocarpine may include increased sweating, nausea, dizziness • Need some saliva flow to be present
TOOTHPASTES	
Biotene dry mouth toothpaste – Gentle mint and Freshmint Oral-7 toothpaste	• Alcohol-free and sugar-free

TABLE 4: **NON-DRUG SUGGESTIONS FOR MANAGING DRY MOUTH**[2,4,11]

- Avoid dry and heated air
- Avoid cigarette smoke
- Ensure adequate hydration
- Drink (rather than sip) adequate amounts of water
- Eat chewy foods to stimulate saliva flow
- Chew food thoroughly before swallowing
- Chew sugarless gum or suck sugarless sweets
- Chew celery
- Limit caffeine and alcohol intake, and avoid cigarettes
- Avoid astringent foods and drinks (e.g. black tea and coffee)
- Avoid alcohol-containing mouthwashes
- Use bicarbonate mouthwashes—decreases acidity. (A bicarbonate mouthwash can be made up by adding approximately half a teaspoon of bicarbonate powder to a glass of warm water. Rinse with mouthwash on waking and at any time during the day.)

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ORAL MUCOSAL DISEASE

Reference: TG

Oral mucosal lesions are common. They can be due to:

- Physiological changes
- local disease
- an oral manifestation of a skin condition
- an adverse drug reaction or systemic disease (e.g. gastrointestinal disease)

Successful management of an oral mucosa' disease requires an accurate diagnosis. Assessing an oral mucosal lesion involves taking a full patient history (Including a medication history), performing a thorough extraoral and Intraoral examination and using diagnostic investigations where appropriate.

Failure to respond to initial treatment, an unclear diagnosis or the presence of any suspicious features ("RED FLAG" features) could indicate malignancy. Early referral to an appropriate specialist is required.

'Red Flag' features of oral mucosal disease:

1. oral ulcers that have lasted for more than 2 weeks
2. orals ulcers that recur
3. nontraumatic oral ulcers in children
4. pigmented lesions on the oral mucosa
5. red, white or mixed red and white lesions on the oral mucosa of unknown origin or with features of potentially malignant disease, such as:
   a. induration
   b. ulceration with rolled margins
   c. fixation to underlying tissues
   d. lesions in high-risk sites (e.g. lateral tongue, floor of mouth)
6. facial or oral paraesthesia
7. persistent oral mucosal discomfort with no obvious cause
8. lumps or swellings, including lymphadenopathy
9. swelling, pain or blockage of a salivary gland, suggestive of salivary gland disease
10. suspected allergy or adverse reaction to dental materials (e.g. oral lichenoid lesion)
11. dry mouth that is not adequately relieved with artificial salivary products and nonpharmacological methods
12. dry mouth caused by systemic disease
13. suspected oral manifestations of systemic disease (e.g. syphilis, Behget syndrome, HIV, inflammatory bowel disease, lichen planus, pemphigoid)
14. lesions occurring in immunocompromised patients (e.g. patients with neutropenia or HIV infection)

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ORAL MUCOSAL DISEASE

Reference: TG

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Oral potentially malignant disorders include:

- oral leucoplakia
- oral erythroplakia
- chronic hyperplastic candidiasis
- actinic cheilitis
- oral lichen planus
- oral submucous fibrosis
- discoid lupus erythematosus
- dyskeratosis congenita
- epidermolysis bullosa

The following conditions can be managed in general practice:

- recurrent aphthous ulcerative disease
- traumatic oral ulcers
- oral candidiasis
- angular cheilitis
- oral mucocutaneous herpes simplex virus
- dry mouth
- oral mucositis
- amalgam tattoo
- geographic tongue
- hairy tongue

There are physiological causes of oral mucosal discolorations (e.g. Fordyce spots [ectopic sebaceous glands], leukoedema), which do not require active management.

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ORAL MUCOSAL DISEASE

Reference: TG

Oral Cancer

Oral cancer is associated with significant morbidity and mortality. Early presentations of oral cancer are usually asymptomatic, whereas late presentations include pain, discomfort, reduced mobility of the tongue, Increased mobility of the teeth or an inability to wear dentures.

Oral cancer can mimic many other oral mucosal diseases, so early specialist referral is required for investigation and biopsy of any suspicious lesion.

Squamous Cell Carcinoma

It is the most common oral malignancy, which arises from the epithelium of the oral cavity. Oral squamous cell carcinoma can affect any part of the oral mucosa; however, it most commonly occurs on the lateral 'surfaces of the tongue, the floor of the mouth or the gingivae.
Genetic susceptibility, environment, occupation and diet may also contribute to the development of oral squamous cell carcinoma.
Cancers originating from the salivary glands and supporting nonepithelial tissues are less common than squamous cell carcinoma.
Metastatic cancers to the oral soft tissues and jawbones commonly originate from primary malignancies in the breast, prostate, kidneys or lungs. Leukaemia and lymphoma may also present in the oral cavity.

Risk factors for oral squamous cell carcinoma include:

- advanced age
- male gender
- smoking or tobacco use
- alcohol use
- infection by oncogenic viruses (e.g. human papillomavirus)
- personal or family history of squamous cell carcinoma of the head and neck
- history of cancer therapy
- prolonged immunosuppression
- areca nut (betel quid) chewing

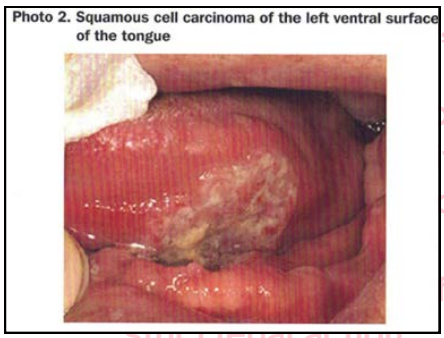
Photo 2. Squamous cell carcinoma of the left ventral surface of the tongue

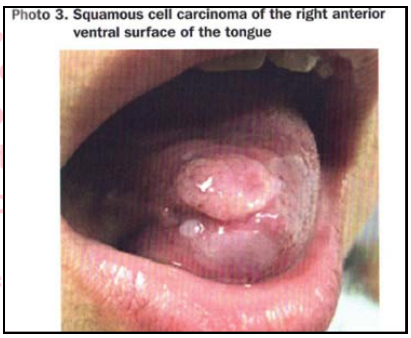
Photo 3. Squamous cell carcinoma of the right anterior ventral surface of the tongue

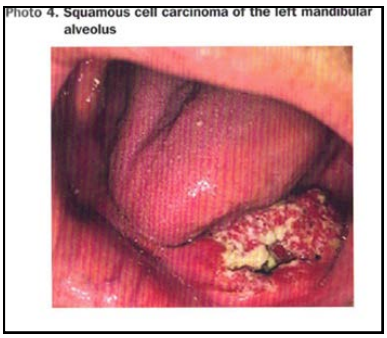
Photo 4. Squamous cell carcinoma of the left mandibular alveolus

Reference: Odell

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ORAL MUCOSAL DISEASE

Reference: TG

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Photo 2. Squamous cell carcinoma of the left ventral surface of the tongue

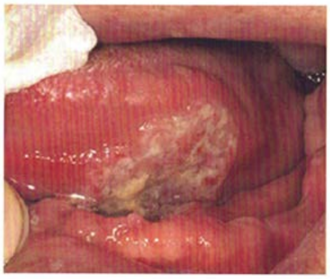


Photo 3. Squamous cell carcinoma of the right anterior ventral surface of the tongue

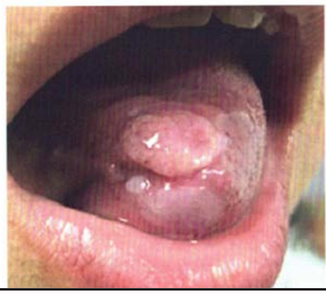
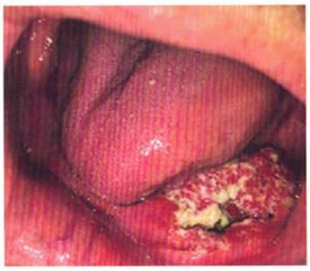


Photo 4. Squamous cell carcinoma of the left mandibular alveolus



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◆ **What are the Causes of Mixed Red and White Patches in the Mouth?**

The causes of white patches are discussed more fully in Case 45. Several may also be associated with red areas.

Cause	Red and White Lesion(s)
Trauma	Chemical burn Cheek biting
Infection	Thrush (acute hyperplastic candidosis) Chronic hyperplastic candidosis (candidal 'leukoplakia')
Lichen planus and similar conditions	Lichen planus Lichenoid reaction (topical and systemic) Lupus erythematosus
Idiopathic or smoking	Idiopathic keratosis (leukoplakia) including: Sublingual keratosis Smoker's keratosis Speckled leukoplakia Stomatitis nicotina (smoker's palate)
Neoplasia	Squamous cell carcinoma

◆ **What Features Might Indicate That This Lesion is Already Malignant? Which are Early Signs, and Which are Late Signs?**

Feature	Early	Late
Red or speckled areas	*	*
Nonhealing ulceration	*	*
Rolled everted ulcer margin		*
Induration of surrounding tissues		*
Bleeding from the surface		*
Fixation of the tissues		*
Destruction of adjacent bone		*
Enlarged hard lymph nodes		*
Size	Small carcinomas are probably diagnosed early, but there is great variation in the rate of growth, and this is only an assumption	
Pain	Unpredictable, often absent and sometimes the presenting complaint	

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ORAL MUCOSAL DISEASE

Reference: TG

Oral Leucoplakia

Leucoplakia is a clinical term for a nonremovable white lesion that is not easily recognisable as any particular condition and therefore requires further investigation.
Oral leucoplakia may be homogenous (uniform lesion often with a fissured surface), or nonhomogeneous (with surface irregularity and textural or colour variation [e.g. speckled).
Some oral leucoplakia lesions show histologic evidence of dysplasia, carcinoma in situ or invasive squamous cell carcinoma. The malignant transformation rate for oral leucoplakia is variably reported, but ranges between 0.13 to 34%, with a mean annual transformation rate of 3.8% per year.
Refer patients with oral leucoplakia to an appropriate specialist for biopsy and monitoring.

Biopsy of a persistent undiagnosed oral white patch is required to exclude epithelial dysplasia, carcinoma in situ and squamous cell carcinoma.

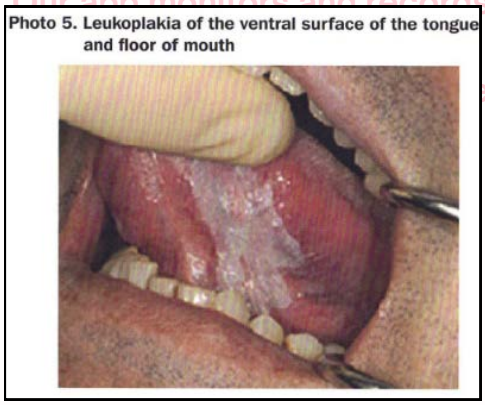

Photo 5. Leukoplakia of the ventral surface of the tongue and floor of mouth

Reference: Odell

TABLE 45.1 Causes of White Patches in the Mouth

Type of Lesion	White Lesion(s)
Normal mucosal variants	Leukoedema Fordyce spots/granules
Inherited epithelial disorders	White sponge naevus Pachyonychia congenita
Traumatic lesions	Frictional keratosis Cheek and tongue biting Chemical burn
Infections	Thrush (acute hyperplastic candidosis) Chronic hyperplastic candidosis (candidal 'leukoplakia') Chronic mucocutaneous candidosis Hairy leukoplakia Syphilitic leukoplakia
Lichen planus and similar conditions	Lichen planus Lichenoid reaction (topical and systemic) Lupus erythematosus Graft versus host disease (GVHD)
Unknown	Leukoplakia (homogenous/verrucous/nodular/speckled)
Smoking-related	Smoker's keratosis Stomatitis nicotina (smoker's palate)
Neoplastic	Squamous cell carcinoma

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ORAL MUCOSAL DISEASE

Reference: TG

TABLE 45.3 Features Associated with Risk of Malignant Transformation

Feature	Risk of Malignant Transformation
Dysplasia	The degree of dysplasia is the best predictor, and it may change, either progressing or regressing, with time.
Genetic status	Aneuploidy and loss of heterozygocity indicate increased risk.
Site	White lesions in the floor of the mouth, posterior and lateral tongue and retromolar area carry the highest risk. Those on the hard palate and dorsum of tongue carry no significant risk except in some cases where plaque-like lichen planus has been present for many years and becomes verrucous. Verrucous lesions on the gingivae can also be high risk for malignant transformation.
Colour	Development of red areas or speckling carries a high risk and is usually associated with severe dysplasia histologically.
Surface	Development of verrucous, nodular areas or ulceration indicate high risk.
Tobacco use	Smoking increases the risk. However, smoking also causes many white patches with no dysplasia, and so statistically, patches in nonsmokers carry the higher risk.
Age	The risk of malignant transformation rises with age.
Gender	Female patients are at higher risk (despite the fact that oral carcinoma is more common in men).
Size	Larger lesions have a higher risk of malignant transformation.
Duration	Patches present for a longer time have a higher risk of malignant transformation.
Multiple areas of leukoplakia	Patients with multiple areas of leukoplakia and those who have had a previous oral cancer are at higher risk.
Family history of carcinoma in upper aerodigestive tract	Indicates increased risk.
Candidal infection in presence of dysplasia	Indicates a small increase in risk.
Change in clinical appearance	Changes apart from that in colour such as changes in size or nodularity or the development of a verrucous surface, indicate a higher risk.
Underlying conditions	Conditions that predispose to oral carcinoma such as submucous fibrosis, raise the relative risk of malignant transformation.

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ORAL MUCOSAL DISEASE

Reference: TG

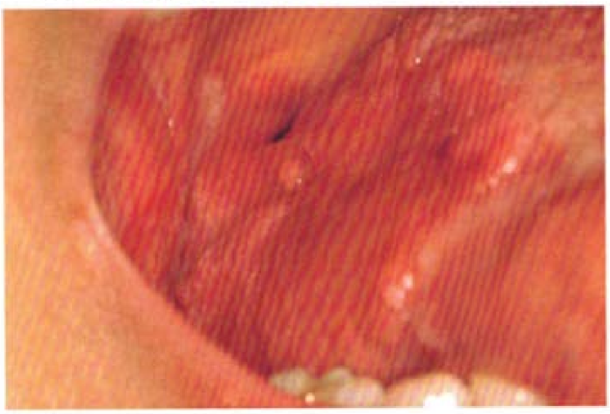
Oral Erythroplakia

Erythroplakia is a clinical term for a potentially malignant fiery red lesion that cannot be attributed to any particular condition. Lesions are usually asymptomatic and isolated, and commonly appear on the floor of the mouth, tongue, soft palate and buccal mucosa. Lesions may appear as smooth, velvety, granular or nodular plaques, often with clear margins. Oral erythroplakia most commonly affects middle-aged and elderly men.

Approximately 70 to 90% of oral erythroplakia lesions are carcinoma in situ or squamous cell carcinoma upon presentation.

Urgent referral to a specialist for biopsy of oral erythroplakia lesions is essential because approximately 70 to 90% are carcinoma in situ or squamous cell carcinoma upon presentation.
Periodic review and repeated biopsy by the managing specialist is recommended for all patients with oral erythroplakia, because malignant transformation is common.

Photo 6. Erythroplakia of the right postero-lateral surface of the tongue



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ORAL MUCOSAL DISEASE

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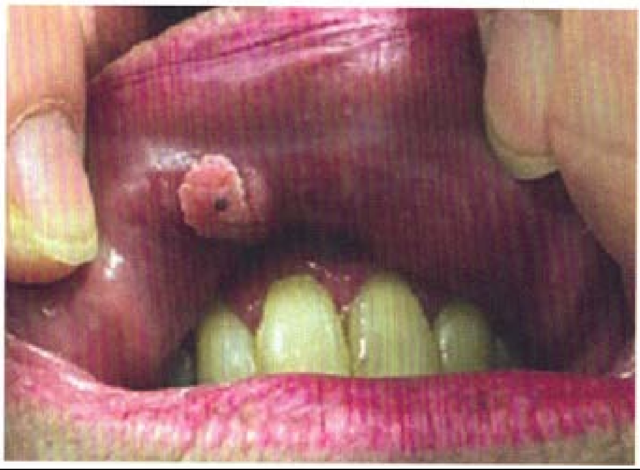
Human papilloma virus-related oral lesions

Human papilloma viruses (HPV) can cause a wide range of oral mucosa lesions. The virus is usually transmitted by direct contact with a lesion.

Squamous papilloma is the most common oral HPV lesion, appearing as a protruding growth with small finger-like projections.

Photo 7. Papilloma of the right maxillary labial mucosa



Sexually transmitted HPV infections can cause oral HPV lesions called condyloma acuminata.

Verruca vulgaris—the common wart—is also caused by HPV infection and may present in the oral cavity.

Both condyloma acuminata and verruca vulgaris can be clinically similar to squamous papilloma.

Oncogenic types of HPV are now recognised as a cause of some squamous cell carcinoma, particularly of the posterior tongue, tonsillar region and oropharynx. These appear to be a distinct entity, separate to the oral cancers associated with alcohol and tobacco use.

Refer patients with suspected HPV lesions to an appropriate specialist for biopsy and management.

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ORAL MUCOSAL DISEASE

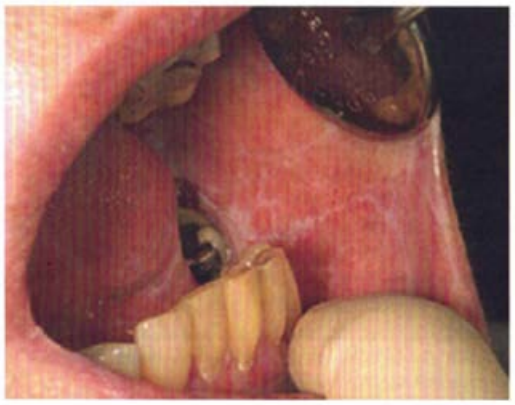
Reference: TG

Oral lichen planus

Lichen planus is an uncommon idiopathic immune-mediated condition that can affect the skin, hair, nails, and oral and genital mucosae.

Oral lichen planus typically occurs on the buccal mucosa, tongue and gingivae. In the nonerosive form of the disease, the lesions consist of a characteristic reticular pattern of white striations or plaques.

Photo 8. Oral lichen planus of the left buccal mucosa showing characteristic white striations



Erosive oral lichen planus presents as erythematous, ulcerated or eroded areas of mucosa, which are often painful. Symptoms include stinging or burning, especially with spicy or acidic food.

Oral lichen planus is associated with an increased risk of oral squamous cell carcinoma.

Refer patients with suspected oral lichen planus to a specialist for biopsy, definitive diagnosis and management.
Differential diagnosis should exclude oral lichenoid lesions.
If lichen planus occurs on the gingival tissues, management includes improving oral hygiene and periodontal health.
Patients with oral lichen planus require ongoing review by an oral medicine specialist because of the chronic nature of the condition and the potential for malignant transformation.

biopsy-proven oral lichen planus becomes symptomatic, treat with:

- betamethasone dipropionate 0.05% cream or ointment topically to the lesions, twice daily after meals, until symptoms resolve.
- Advise patients to stop using topical corticosteroids once symptoms have resolved.
- If the patient's symptoms have not improved after 3 weeks of topical corticosteroids, the symptoms change or the appearance or texture of the lesion changes, advise patients to return to their treating specialist.

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

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Oral lichen planus

There are six recognized oral presentations of lichen planus:

- reticular
- papular
- plaque-form
- atrophic
- ulcerative(erosive) and rare
- bullous form

These latter three forms can be associated with significant discomfort requiring either topical and/or systemic immunosuppressive therapy.

The cause(s) of the various oral lichenoid lesions, ranging from idiopathic oral lichen planus (OLP) to the "contact" lesion, is not understood, but all the lesions are characterized histologically by a typical "lichenoid tissue reaction" featuring a band-like lymphohistiocytic infiltrate within the lamina propria and liquefaction degeneration of the basal keratinocytes. These reactions may be the result of several diverse possible triggers, but all culminate in a common pathologic process, that of T-lymphocyte directed, immune-mediated, damage to the oral epithelial basal cells.

OLP most frequently presents in women, by a ratio of approximately 3:1 to 3:2 compared with men, aged 40 years and above.

Mucosal lesions are usually multiple and almost always have a bilateral, symmetrical distribution. They commonly take the form of minute white papules that gradually enlarge and coalesce to form either a reticular, annular, or plaque-like pattern. A characteristic feature is the presence of slender white lines (Wickham's striae) radiating from the papules. In the reticular form, there is a lace-like network of slightly raised white lines, often interspersed with papules or rings. The plaque- like form may be difficult to distinguish from leucoplakia.

Involvement of the gingivae is described clinically as desquamative gingivitis, but is not unique to OLP and may feature in the presentation of other oral dermatoses, especially pemphigoid and pemphigus.

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ORAL MUCOSAL DISEASE

Reference: TG

Oral lichenoid lesion

Lichenoid mucosal reactions can be caused by:

- contact hypersensitivity to dental restorations
- hypersensitivity reactions to drugs, particularly:
  a. Drugs that lower blood pressure (e.g. beta blockers, angiotensininhibitors, diuretics [particularly hydrochlorothiazide)
  b. nonsteroidal anti-inflammatory drugs (NSAIDs)
  c. drugs that treat thyroid disorders
- Medical conditions:
  a. hepatitis C infection, particularly in patients with the human leukocyte antigen HLA-DR6 allele (which is common in people of Mediterranean descent)
  b. thyroid disorders
  c. chronic graft-versus-host disease.

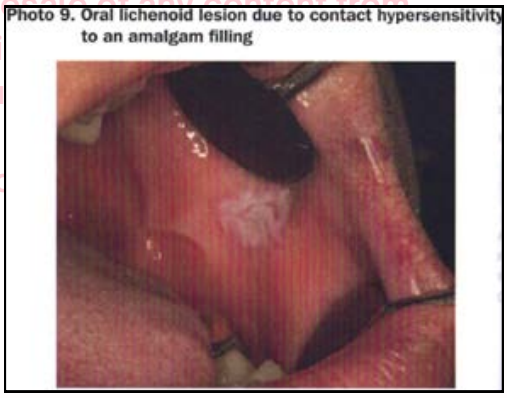

Photo 9. Oral lichenoid lesion due to contact hypersensitivity to an amalgam filling

Refer patients with a suspected oral lichenoid lesion to an appropriate specialist for definitive diagnosis and management.
In the case of contact hypersensitivity to an amalgam filling, replacement of the implicated amalgam filling may result in partial or full resolution of the lesion.

However, removal of all amalgam fillings is not recommended.

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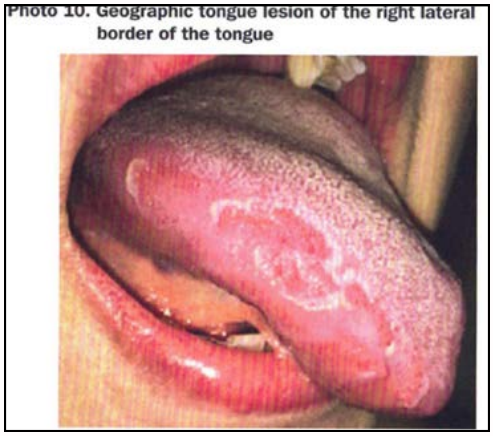
Geographic tongue/ erythema migrans/ migratory glossitis

It is a benign condition affecting up to 5% of the population.
It manifests as migratory red lesions and usually involves the dorsal surface of the tongue, but sometimes extends to the floor of the mouth and buccal mucosa.
The red patches have a central atrophic and depapillated zone, which, in the most common presentation, is surrounded by elevated white or cream margins.
Occasionally the central red patch is sensitive, but not painful. If pain or burning is present, investigate for other causes or seek specialist advice.

Photo 10. Geographic tongue lesion of the right lateral border of the tongue



The cause of geographic tongue is unknown, but there may be a family history of the condition. Some patients have atopic allergies, or can relate the lesions to particular foods or stress.
Histologically, the lesions are psoriasiform, but geographic tongue is not related to a specific condition.
Management of geographic tongue is not required beyond correct diagnosis and reassurance. If any 'red flag' features of oral mucosal disease are present, refer to an appropriate specialist.

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ORAL MUCOSAL DISEASE

Reference: TG

Amalgam tattoo

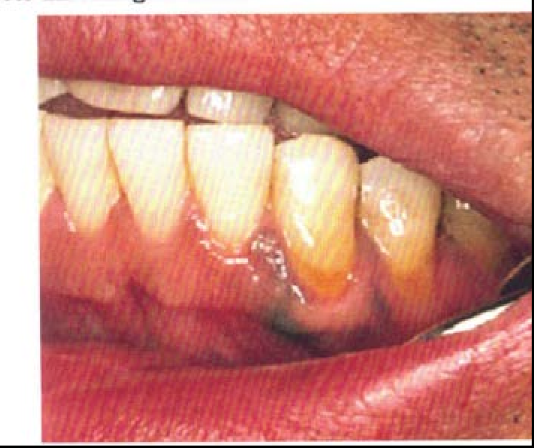
Amalgam tattoos are a common cause of exogenous oral discolouration.

They result from the iatrogenic mucosal implantation of amalgam particles during the course of a dental procedure.

They are usually small, macular and blue-grey to black in colour.

Amalgam tattoos are usually found in close proximity to amalgam-restored teeth or where such teeth were previously present.

oto 11. Amalgam tattoo



Amalgam tattoos are benign and do not require treatment, beyond correct diagnosis. To confirm the diagnosis, metallic amalgam particles may be evident on X-ray.

If the diagnosis is not confirmed or if any 'red flag' features of oral mucosal disease are present, refer patients to an appropriate specialist.

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Hairy tounge

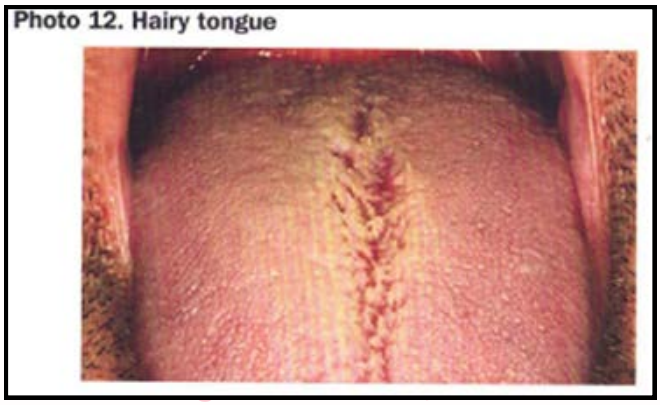
Photo 12. Hairy tongue

Hairy tongue occurs when excessively long and hyperkeratinised filiform papillae of the tongue become stained by an accumulation of epithelial cells, exogenous material or chromogenic microorganisms.

It is usually black, but may be other colours and can occur with the use of chlorhexidine mouthwash, after a course of antibiotics or in patients who have limited oral intake (e.g. with percutaneous endoscopic gastrostomy [PEG] feeding). If any 'red flag' features of oral mucosal disease are present, refer to an appropriate specialist.

Management of hairy tongue primarily involves identifying and addressing the cause. Other strategies include improving oral hygiene, brushing the tongue gently with a toothbrush and using sodium bicarbonate mouthwash.

A sodium bicarbonate mouthwash can be made by adding half a teaspoon of sodium bicarbonate powder to a glass of warm water. The mouthwash can be rinsed in the mouth on waking and at any time during the day.

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Traumatic Oral Ulcers

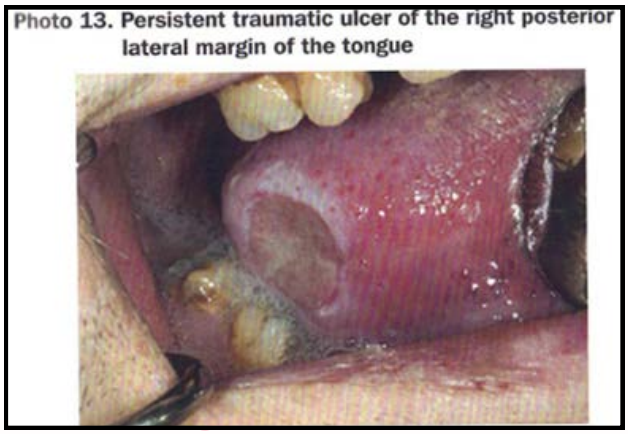

Photo 13. Persistent traumatic ulcer of the right posterior lateral margin of the tongue

Oral ulceration due to trauma is common and can be associated with:

- eating rough, sharp or hot foods
- sharp broken teeth or dental restorations
- toothbrushing
- oral prostheses or orthodontic appliances
- chemical burns (e.g. following incorrect use of tooth-bleaching products)

Address causes of trauma, including changing oral hygiene practices, smoothing sharp edges of teeth or restorations, adjusting prostheses or placing wax on orthodontic appliances.

Most traumatic ulcers resolve spontaneously if the cause of the trauma has been adequately addressed.

However, if any 'red flag' features of oral mucosal disease are present, refer to an appropriate specialist.

A salt water mouthwash is antiseptic and may provide symptomatic relief.
If temporary pain relief is required, apply a topical anaesthetic or analgesic to the ulcer, such as:

- benzydamine 1% gel (adult and child 6 years or older) topically to the ulcer, 2- to 3-hourly as necessary.

Persistent ulcers (lasting more than 2 weeks despite addressing the cause of trauma) or recurrent ulcers require investigation; refer to an appropriate specialist.

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ORAL MUCOSAL DISEASE

Reference: TG

Recurrent aphthous ulcerative disease

Recurrent aphthous ulcerative disease is the most common cause of nontraumatic ulcers of the oral mucosa. The disease has an immune-mediated pathogenesis and is characterised by the periodic eruption of painful ulceration of the oral 'mucosa.

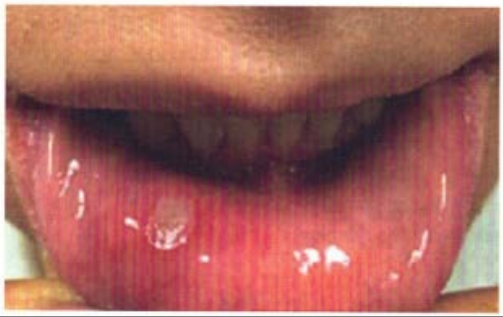
Aphthous ulcers can occur acutely with smoking cessation, but these usually resolve with time, or can be triggered by trauma (e.g. toothbrushing, orthodontic appliances)

The ulcers usually occur on the mucosa of the cheek, lip and floor of the mouth, but can occasionally affect the mucosa of the gingivae and hard palate.

Three forms of aphthous ulcers are recognised:

Minor aphthous ulcers	Major aphthous ulceration	Herpetiform aphthous ulceration
• most common form • presents as smaller lesions (usually 2 to 4 mm in diameter) • occur a few at a time • heal within 7 to 10 days.	• less common form • presents as larger lesions (10 mm or more in diameter) • can persist for up to 6 weeks • (and occasionally months) • heal with submucosal scarring.	• rare • presents as recurrent crops of non-vesicular small ulcers (1 to 2 mm in diameter) that coalesce to form larger ulcers • heal within 1 to 2 weeks not caused by the herpes virus, so do not have a cluster pattern.

Photo 14. Minor aphthous ulcer of the mandibular labial mucosa



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Reference: TG

TABLE 19.1 Features of Ulcers

Feature	Details
Site of ulcer	Recurrent aphthous stomatitis (RAS): Typically nonkeratinized mucosa. Erythema multiforme: Vermilion border of lip, buccal and labial mucosa. Traumatic ulceration: Usually recurs at the same site, often close to a sharp tooth. Crohn's disease: Typically affects buccal sulcus.
Size of ulcer	Minor RAS: 3–8 mm in diameter. Major RAS: greater than 1 cm in diameter (up to 3–4 cm). Herpetiform RAS: 0.2–3 mm in diameter.
Duration of each ulcer	Minor RAS: 7–10 days. Major RAS: May take weeks to months. Ulcers heal with scarring. Herpetiform RAS: 7–10 days. Erythema multiforme: Variable (10–21 days).
Number of ulcers	Minor RAS: Single ulcers or small crops of 2–5 ulcers Major RAS: One or two ulcers at a time. Herpetiform RAS: 30–100 ulcers at a time, which can coalesce such that it becomes difficult to count individual ulcers.
Frequency of attacks	RAS: Variable frequency; some patients may have ulcers continuously, whilst others experience ulcers just once per year. Erythema multiforme: Ulcers may recur at 6–8 week intervals in severe cases; other patients experience one-two attacks per year. Nutritional deficiencies/inflammatory conditions: Patients may have continuous ulceration.
Shape of ulcer	RAS: Usually round/oval and sharply defined; may become more irregular with healing. Herpetiform ulcers coalesce to form irregular shapes. Erythema multiforme: Irregular and ragged, merging with inflamed surrounding mucosa. Those on the lips are often covered by bloody fibrin sloughs.
Are the ulcers preceded by vesicles?	The presence of vesicles indicates possible viral infection or immunobullous disease. This fact may be helpful in the differential diagnosis of herpetiform ulcers, which resemble viral ulcers but are not preceded by vesicles.
Age of onset	RAS: Usually before or around adolescence. Erythema multiforme/inflammatory bowel disease: Typically second or third decade. Immunobullous disease: Typically, fourth decade onwards.
Family history	RAS: May have family history of ulceration. Coeliac disease/inflammatory bowel disease: May have positive family history. Erythema multiforme: Typically no family history. Traumatic ulceration: No family history.
Exacerbating or relieving factors	Ulcers can develop at a site of minor trauma in both RAS and immunobullous disease. Stress and menstruation can precipitate attacks of RAS. Erythema multiforme may be triggered by a drug, viral or other infection, although often no trigger is identified.

Reference: Odell

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Assessment of aphthous ulcers involves taking a thorough history and examination; see also 'red flag' features of oral mucosal disease.

In children, investigate for systemic causes of nontraumatic ulceration. In adults who have additional symptoms, investigate for a systemic cause. Systemic causes of aphthous ulcers include:

- iron, vitamin B12, folate or zinc deficiency
- coeliac disease
- ulcerative colitis
- Bechet syndrome
- PFAPA (periodic fever, aphthous stomatitis, pharyngitis, cervical adenitis) syndrome in children

Deficiencies should be treated only on laboratory confirmation.

Management of recurrent aphthous ulcerative disease

If an aphthae-like ulcer occurs in a child, refer for further investigation because it could be a sign of systemic disease.

Topical corticosteroid treatment can produce rapid healing of minor aphthous ulcers, particularly if used in the prodromal or pre-ulcerative stage.

The aim is to treat the lesion rather than prevent further outbreaks; for adults, use:

- hydrocortisone 1% cream or ointment topically to the lesions, 2 to 3 times daily after meals.
- If pain relief is required for minor aphthous ulcers in adults, apply a topical anaesthetic or analgesic to the ulcers, such as:
  -benzydamine 1% gel topically to the lesions, 2- to 3-hourly as necessary.
- Lidocaine viscous solution is an alternative topical anaesthetic for hospital settings (but be aware of the higher cost); for adults, use:
  -lidocaine 2% viscous solution, use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours.

Ulcers that are not improving after 2 weeks are potentially malignant—refer to a specialist for management and biopsy. Seek specialist advice for patients with major or herpetiform aphthous ulceration or immunocompromised patients with neutropenic ulceration.

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ORAL MUCOSAL DISEASE

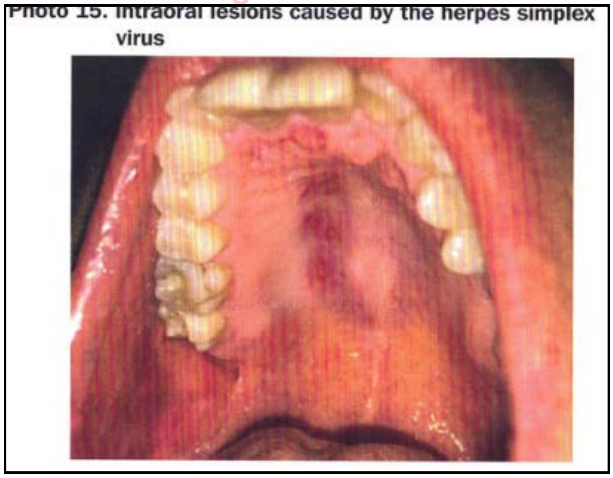
Reference: TG

Oral mucocutaneous herpes

Primary oral mucocutaneous herpes

Primary oral mucocutaneous herpes simplex virus (HSV) infection (herpetic gingivostomatitis) often occurs in childhood with fever, painful intraoral lesions, systemic symptoms (e.g. malaise, lethargy) and cervical lymphadenopathy.
Intraoral herpes simplex virus lesions begin as blisters an ulcerate rapidly. Healing occurs within several day;
in infants, but can take up to 2 weeks in older children. During this time, it may be difficult to eat and drink and hospital admission may be required.
Herpetic gingivostomatitis is rare in adults, but can be severe and present with dehydration due to severe odynophagia.
While herpes simplex virus is the most common virus to cause mouth ulcers, other viruses (e.g. varicella zoster virus, coxsackie virus, cytomegalovirus) may be the cause. Intraoral herpes simplex virus lesions may resemble those seen in necrotising gingivitis.
However, necrotising gingivitis is rare in children and is confined to the gingival tissues, while herpetic gingivostomatitis lesion: are widespread and affect all soft tissues in the mouth.

Photo 15. Intraoral lesions caused by the herpes simplex virus



To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

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ORAL MUCOSAL DISEASE

Reference: TG

Management

- Treat minor primary oral mucocutaneous herpes with supportive management (i.e. oral fluids, antipyretic drugs and analgesia).
- Apply a topical anaesthetic or analgesic, such as bezadymine .
- If this is not available, lidocaine viscous solution is an alternative topical anaesthetic for hospital settings (but be aware of the higher cost):
  - -lidocaine 2% viscous solution adult: use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours
  - -child 3 years or older: use the lowest dose necessary up to 0.2 ml./kg (maximum 5 mL), rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 4 doses in 24 hours
  - -child younger than 3 years: use the lowest dose necessary up to 0.2 ml/kg (maximum 1.25 mL), applied to the affected areas with a cotton swab, 3-hourly as necessary; maximum 4 doses in 24 hours.
- lithe pharynx is affected in adults and children older than 12 years, lidocaine viscous solution can be gargled and swallowed.

Management of primary oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- patients with severe presentations
- immunocompromised patients
- patients with HIV

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Recurrent oral mucocutaneous herpes

Recurrent oral mucocutaneous herpes simplex virus (HSV) infection follows latent reactivation of the virus. Lesions usually occur on the lips (herpes simplex labialis or cold sores), but can also occur on the intraoral mucosa or other areas of skin.
Lesions are usually preceded by the prodromal stage, lasting several hours to days, which features pain, burning, tingling or itching.
Recurrences are usually mild and infrequent, and their frequency can be minimised with sun protection. Herpes simplex virus reactivation may be complicated by erythema multiforme.
A herpes simplex virus lesion affecting the oral mucosa cannot be differentiated from an aphthous or traumatic ulcer using microbiological testing—most adults will have positive serology for the herpes simplex virus from previous exposure, and viral DNA may be detected on swabs of aphthous or traumatic ulcers. Instead, diagnosis requires a thorough history and clinical examination.
To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

Management:

For a minor recurrence of oral mucocutaneous herpes, episodic antiviral therapy may reduce its duration; use:

- aciclovir (adult and child older than 3 months) 5% cream topically, 5 times daily (every 4 hours while awake) for 5 days, started at the first sign of recurrence or during the prodromal stage

  OR
- famciclovir (adult) 1500 mg orally, as a single dose, taken at the first sign of recurrence or during the prodromal stage.

Using aciclovir cream for longer than recommended has no benefit.

Management of recurrent oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- patients with severe recurrences of herpes (with systemic signs and symptoms, or if the patient has difficulty eating or swallowing)
- patients with generalised or chronic herpes infection (with crusted lesions and ulceration)
- immunocompromised patients
- patients with HIV.

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Mucous membrane pemphigoid

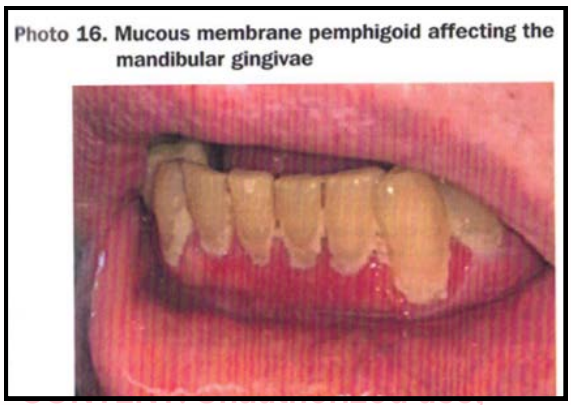
Photo 16. Mucous membrane pemphigoid affecting the mandibular gingivae

Mucous membrane pemphigoid is an uncommon autoimmune vesiculobullous disorder that affects stratified squamous epithelium. It occurs predominantly on the gingivae and palate. Mucous membrane pemphigoid presents as large, painful and persistent erosions, and is characterised by subepithelial splitting, with bulla or vesicle formation. The lesions heal with variable amounts of scarring. Differential diagnosis includes pemphigus vulgaris.

Refer patients with suspected mucous membrane pemphigoid to an appropriate specialist for biopsy and definitive diagnosis. Management usually requires long-term use of immunosuppressive therapy. Ophthalmologist review is necessary because there is a risk of blindness with mucous membrane pemphigoid.

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

Immune-mediated sub-epithelial blistering diseases characterized by vesicles or bullae that break down to leave ragged ulcers that affect epithelial surfaces, particularly the mouth.

Historically, pemphigoid was broadly subdivided into only two main conditions:

- **"bullous pemphigoid" that predominantly affects the skin, and rarely, mucosa;**
- **"benign mucous membrane pemphigoid" also known as cicatricial pemphigoid, but now more simply as "mucous membrane pemphigoid" (MMP), that conversely, overwhelmingly affects mucosa and infrequently the skin.**

Occasional drug-induced MMP has been reported, associated with penicillamine (D-Penamine), a heavy-metal chelator used in rheumatoid arthritis and the loop diuretic agent, frusemide (Lasix, Urex)

MMP is predominantly a disease of women, with a mean age at onset of 51–62 years. 32 Children are rarely affected.

The most common areas of involvement are the oral cavity (85%) and conjunctivae (64%).32 The oral mucosa is often the initial site of MMP lesions. The term "oral mucous membrane pemphigoid" (OMMP) is often used when MMP is limited to the oral cavity with no other mucosal involvement and "ocular cicatricial pemphigoid" (OCP) is used when MMP is limited to the conjunctivae.

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Oral mucosal lesions

Patients present with bleeding, pain, dysphagia or desquamation of the oral mucosa. Vesicles or bullae may occur anywhere on the oral mucosa and there may be a positive "Nikolsky sign", this sign is positive in pemphigus, where firm sliding pressure with a finger separates normal-appearing epithelium from the underlying lamina propria, resulting in the immediate formation of a vesicle or erosion.

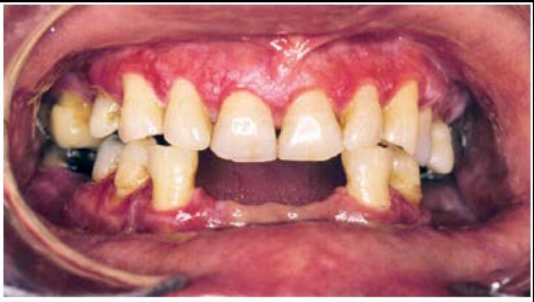

Fig 3. "Desquamative gingivitis", a clinical presentation (not a condition) seen with oral lichen planus, and on occasion, mucous membrane pemphigoid.

Reference: Odell

Main Causes of Desquamative Gingivitis

- Lichen planus
- Mucous membrane pemphigoid
- Pemphigus vulgaris

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

Ocular lesions

Ocular manifestations have been reported to occur in 3 to 48% of patients with oral lesions. Ocular involvement usually begins as chronic conjunctivitis with symptoms of burning, irritation, photophobia ich excess tearing.

Special investigations

- Biopsy (histopathological and DIF investigations)

Definitive diagnosis is based on biopsy of perilesional tissue with histological and direct immunofluorescence (DIF) examination.

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Reference: TG

TABLE 41.1 Causes of Oral Blistering

Mucous membrane pemphigoid (MMP)	An autoimmune disease with autoantibodies targeting various components of the basement membrane such as the bullous pemphigoid antigens BP180 and BP230. Autoantibody binding activates complement and attracts neutrophils and eosinophils, resulting in degradation of the basement membrane and separation of the epithelium from connective tissue. Oedema fluid collects in the space to form a blister.
Pemphigus vulgaris (PV)	An autoimmune disease with immunoglobulin G (IgG) autoantibodies directed against desmoglein 1 and 3 which are adhesion molecules in the desmosomes that hold the epithelial cells together. Autoantibody binding activates complement, the cells detach from each other (acantholysis) and spaces in the epithelium fill with fluid to form blisters. Mucosal lesions are associated with a predominance of autoantibodies against desmoglein 3 and cutaneous lesions with a predominance of autoantibodies against desmoglein 1.
The bullous subtype of lichen planus (LP)	A T cell–mediated reaction, possibly autoimmune or possibly directed against haptens or extrinsic antigens in the basal cells of the epithelium. Basal cells are killed, and because they maintain the basement membrane, the attachment of the epithelium to the connective tissue is weakened. Usually, the epithelium becomes thin and ulcerates because the dividing cells have been lost, but in the bullous subtype, the weak basement membrane separates, fluid accumulates and a blister forms.
Erythema multiforme (EM) and Stevens-Johnson syndrome	These hypersensitivity reactions, usually triggered by infections such as herpes simplex or mycoplasma (in EM) or drug hypersensitivity (in Stevens-Johnson syndrome/toxic epidermal necrolysis) cause oedema and fluid accumulation below and within the epithelium, raising blisters.
Angina bullosa haemorrhagica (ABH)	The mechanism of blister formation in ABH is unknown, but assumed to be a weakened basement membrane.
Viral infections	Oral viral infections such as herpes simplex and herpangina are 'lytic' infections – that is, the infected epithelial cells lyse to release the virus particles. Clusters of epithelial cells burst beneath the intact surface layers to produce vesicles or small blisters, which later rupture to form ulcers.
Epidermolysis bullosa	A heterogeneous group of inherited defects in the components of the epithelial cytoskeleton or of the basement membrane. Mechanical weakness in the tissue allows fluid filled spaces to open up under mild trauma or spontaneously.
Dermatitis herpetiformis	Autoantibodies against dietary gluten are thought to cross-react with epithelial cell enzymes, bind near the basement membrane, activate complement and cause small foci of separation of the epithelium. The blisters are only small vesicles, not usually clinically evident as blisters.

Reference: Odell

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ORAL MUCOSAL DISEASE

Reference: TG

Pemphigus Vulgaris

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

It is a group of autoimmune diseases characterized by intra-epithelial blistering, resulting in superficial vesicles or bullae that easily rupture, resulting in ulceration of mucosal and/or cutaneous sites.

Pemphigus vulgaris (PV) is the most common and clinically the most aggressive variant, being associated with significant morbidity and mortality, composing 70% of all reported cases. Pemphigus vulgaris commonly and initially affects the oral mucosa and then the skin. Other mucosal sites may also be involved, including the mucosa of the conjunctivae, nose, oesophagus, pharynx and larynx, and genitalia.

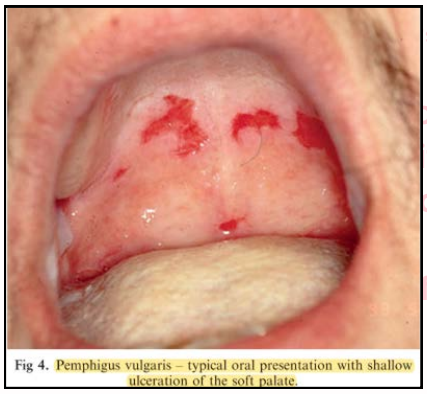

Fig 4. Pemphigus vulgaris – typical oral presentation with shallow ulceration of the soft palate.

Some drugs have been reported to induce PV, including the anti-mycobacterial antibiotic, rifampicin. More recently, associations have been reported with penicillamine as well as the anti-hypertensive ACE (angiotensin-converting enzyme) inhibitor, captopril, and other thiol-containing compounds.

Due to their fragile nature, being intra-epithelial (in contrast to the sub-epithelial nature of the blisters seen in pemphigoid), intact blisters are uncommon in PV, rupturing quickly to produce painful erosions. Patients can be Nikolsky sign positive on examination of the mucosa and the skin.

Special investigations

Clinical suspicion that a patient has one of the vesiculobullous diseases warrants biopsy of perilesional tissue, with histological and DIF examination being essential in establishing the diagnosis.

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ORAL MUCOSAL DISEASE

Reference: TG

Epidermolysis Bullosa Acquisita

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

Epidermolysis bullosa acquista (EBA) is a very rare non-inheritable, mechano-bullous condition characterised by the development of autoantibodies that target the Type VII collagen found in the basement membrane.In contrast, epidermolysis bullosa (EB) is an inherited form of the disease. Both diseases are characterized by the problem that even the slightest mechanical irritation, or trauma of the skin and/or the mucosal surfaces of the oral cavity and upper aerodigestive tract, results in the formation of blisters that only heal with scarring and severe atrophy of the affected tissues. In EB the defect is a genetically- induced molecular derangement of the keratin filaments, hemidesmosomes and anchoring filaments and fibrils that attach the epidermis to the deeper dermis. Clinically, EBA manifests very similarly to the inherited, but more severe and devastating, EB, but has later onset, usually in early adulthood, and so can be confused clinically with other blistering conditions, especially pemphigoid and its variants.

Treatment is usually successful with high-dose corticosteroids often required in combination with other immunosuppressant agents and/or dapsone.

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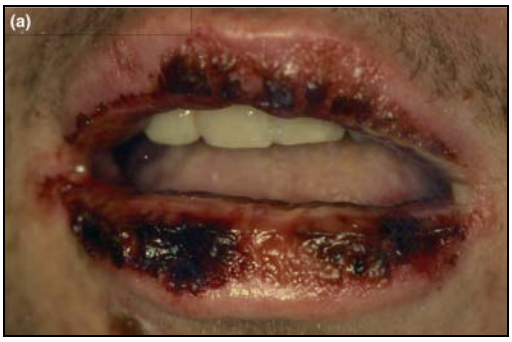

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Erythema multiforme (EM) is part of a spectrum of complex, immune-mediated, reactive, muco-cutaneous disorders that often presents with oral, especially, labial mucosal erythema, blistering and ulceration.

In EM major, the oral mucosa is the most commonly involved mucosal site, but any mucosa can be involved, including the epithelial lining of the trachea, bronchi, and gastrointestinal tract, as well as the genitalia. Erythematous macules precede vesiculobullous formation of the mucosa that rupture, leaving irregular superficial painful ulcers, with a marked erythematous halo, or develop into broad areas of white, desquamating, necrotic mucosa. The lip lesions are characteristically, markedly haemorrhagic.

SJS is a more severe disease, characterized by significant oral mucosal involvement, plus conjunctival and/or genital mucosal involvement, and more extensive skin involvement that generally follows several days later. The muco-cutaneous lesions last up to six weeks and reflecting the more significant disease process can heal with scarring, so resulting in laryngeal, conjunctival and vaginal strictures.

TEN presents with the oral lesions typical for EM major, but the skin involvement is extensive, with consequently up to 30% of the body surface denuded, and resembling second degree burns. Indeed, optimal care for patients with TEN is in a specialized burns unit, with attention to the critical issues of fluid loss, electrolyte disturbance and secondary infection.

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Reference: TG

Special investigations

There are no tests that are pathognomonic for EM and its variants. Biopsy for histopathology and direct immune-fluorescence are only useful in excluding conditions that can present similarly.
Laboratory investigations are directed in determining any recent infection that may have triggered the EM. Human Herpes Viruses 1 and 2 (HSV-1 and 2) are a very common trigger, preceding the presentation of EM major by up to 14 days, reportedly in some 70% of cases. The other commonly reported infectious trigger is Mycoplasma pneumoniae infection. For SJS and TEN, drugs, particularly anticonvulsants such as carbamazepine (Tegretol), phenytoin (Dilantin), phenobarbitol and sodium valproate are frequently implicated.

Treatment

There are no systematic reviews detailing the best treatment for EM major, or its more severe variants. The key aspects of care are firstly, identification and, if practical, treatment of the infectious trigger, or identification (from the history) and withdrawal of the suspected causative drug. Secondly, the use of immune-modulating therapy of which historically the mainstay has been systemic corticosteroid therapy. However, the use of systemic corticosteroids is mired in controversy, with some authors claiming a higher rate of adverse outcomes for patients placed on systemic corticosteroids, particularly in SJS and TEN.

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ORAL MUCOSAL DISEASE

Reference: TG

Oral candidiasis and Candida -associated lesions

Candida species are a commensal organism of the oral cavity. Oral candidiasis is an opportunistic infection that is uncommon in healthy individuals; however, it occurs relatively commonly in neonates.

If any 'red flag' features of oral mucosal disease are present, refer to an appropriate specialist.

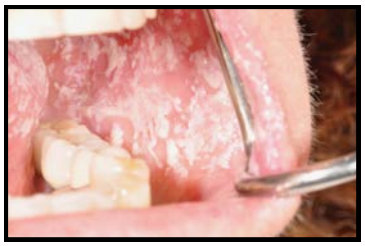
Management of oral candidiasis in immunocompromised patients requires specialist advice. Patients with undiagnosed HIV infection may present initially with oral candidiasis.

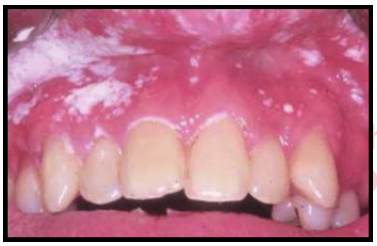
Common risk factors for oral candidiasis

Local Factors	Systemic Factors
• dentures • salivary gland hypofunction • corticosteroid inhalers • poor oral hygiene • smoking	• immune compromise leg poorly controlled diabetes) • drugs (e.g. systemic corticosteroids, antibiotics)

Overview of oral candidiasis and Candida associated lesions

Pseudomembranous candidiasis





Clinical features:

- creamy white curd, papules and plaques that are sometimes removable
- red, raw and often bleeding base
- generally asymptomatic
- may affect the oropharynx
- if the dorsal tongue is affected, autoinoculation of the palate may occur

Management:

- address predisposing factors
- use topical antifungal therapy for oral candidiasis
- if the infection affects the oropharynx, refer for specialist management

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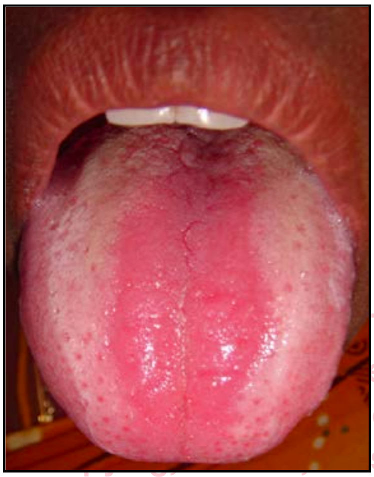
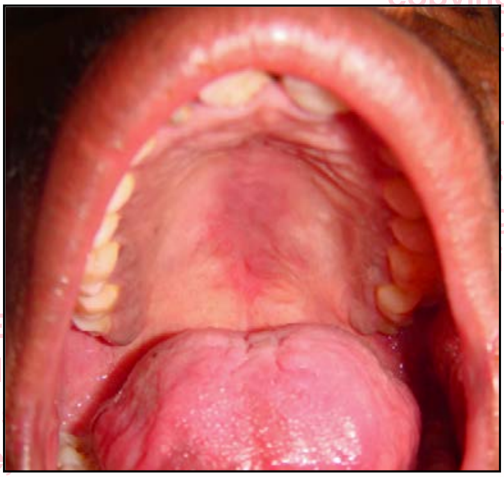
Erythematous candidiasis





Clinical features

- sensitive red lesions commonly affecting the palate and tongue
- the tongue may appear depapillated and smooth

Management

- address predisposing factors
- use topical antifungal therapy for oral candidiasis

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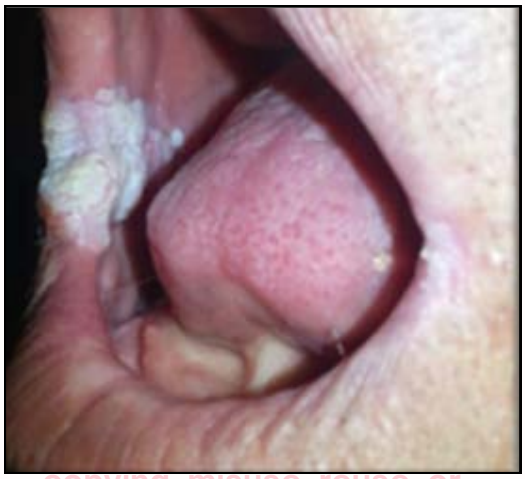
Hyperplastic candidiasis



Clinical features

- asymptomatic, nonremovable white plaques that may appear nodular
- usually affects the retro-commissures, anterior buccal mucosa and lateral tongue
- may be bilateral
- may resemble oral leucoplakia or oral cancer

Management

- address predisposing factors
- may be associated with epithelial dysplasia—refer to a specialist for biopsy and management

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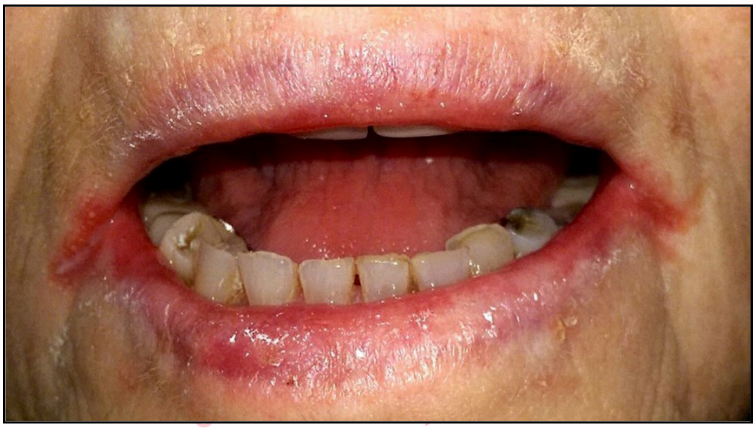
Angular cheilitis (angular stomatitis)



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Clinical features

- painful erythema and fissuring of the corners of the mouth
- usually caused by a mixed infection of Candida, Staphylococcus aureus and Streptococcus species
- often associated with intraoral candidiasis

Predisposing factors

- deep skin folds around the mouth (associated with worn down teeth, ill-fitting dentures or not wearing dentures)
- iron, folate or vitamin B12 deficiency
- Crohn disease
- granulomatous disease
- atopic and seborrhoeic dermatitis

Management

- dental review to assess dental or denture-related causes
- address predisposing factors
- use topical antifungal therapy for angular cheilitis
- treat oral candidiasis if present, with topical antifungal therapy for oral candidiasis

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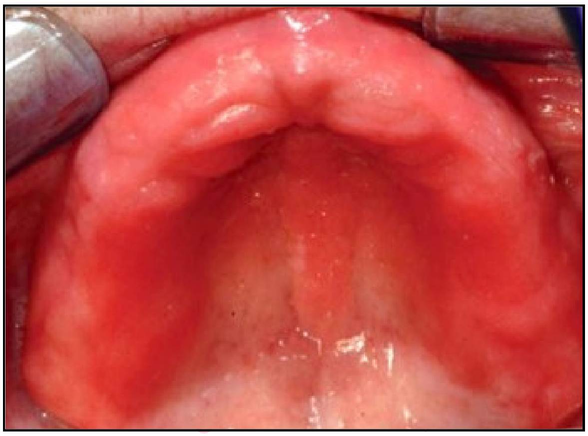
Denture-associated erythematous stomatitis (denture stomatitis)



Clinical features

- sensitive erythematous lesions confined to denture bearing areas, particularly the palate
- may appear punctate, or smooth and red
- nodular hyperplasia may be observed

Predisposing factors

- ill-fitting dentures
- suboptimal oral and denture hygiene
- dietary factors

Management

- advise patient to optimise denture hygiene and to remove dentures at night, clean them, then store them dry overnight
- dental review to assess fit of dentures
- if symptoms do not resolve after 1 month of optimal oral and denture hygiene, use topical antifungal therapy for oral candidiasis, applied inside the mouth and to the dentures

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ORAL MUCOSAL DISEASE

Reference: TG

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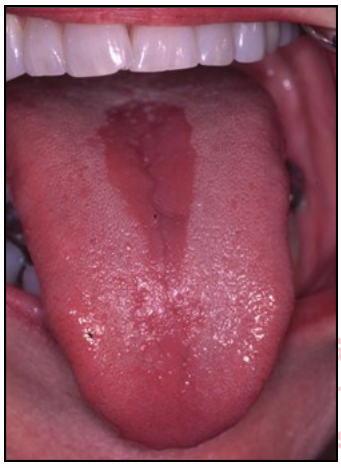
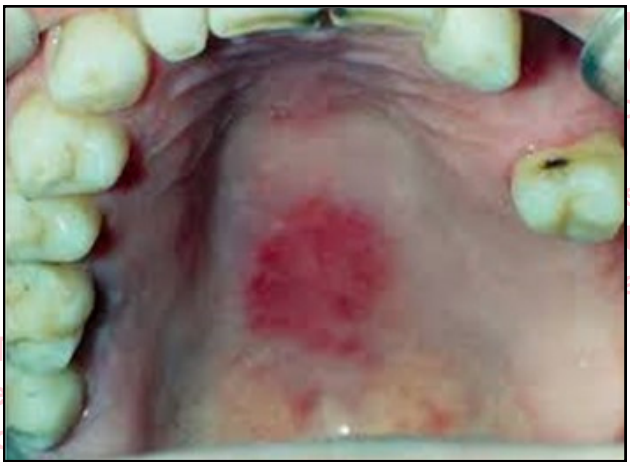
Median rhomboid glossitis





Clinical features

- rhomboid area of depapillation and erythema in the midline of the dorsal tongue
- may be fissured or nodular
- autoinoculation of the palate may occur
- usually asymptomatic although it may sting or burn

Management

- address predisposing factors
- use topical antifungal therapy for oral candidiasis

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ORAL MUCOSAL DISEASE

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Oral mucositis

Oral mucositis presents as painful inflammation, redness, swelling and ulceration of the oral mucosal surfaces caused by radiotherapy, chemotherapy or other drugs.
Oral mucositis is a type of stomatitis (an inflammatory condition of the oral tissues).
Other causes of stomatitis include salivary gland hypofunction and vitamin deficiencies.
Take a thorough history and examination to identify the cause.
Oral mucositis can lead to significant problems with eating, drinking an adherence to medication. Patients undergoing treatment of cancer who develop mucositis have an increased risk of systemic infection and require longer hospital admissions.
Patients should be dentally fit before starting chemotherapy or head and neck radiotherapy, particularly if the treatment will result in severe mucositis and reduced salivary flow.

If a topical analgesic is needed, use:

- benzydamine hydrochloride 0.15% solution 15 mL, rinsed in the mouth for 30 seconds then spat out, 1.5- to 3-hourly as necessary (use diluted with 15 mL of water if stinging occurs)

  OR

- lidocaine 2% viscous solution, use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours.

- If pain is not adequately managed with topical measures, systemic analgesics may be required—seek advice from the patient's multidisciplinary team.
- Establish preventive oral care regimens, and regularly assess the oral cavity. Patients with profound mucositis have difficulty performing oral hygiene measures effectively, though this should be encouraged nevertheless.
- Chlorhexidine limits exposure to water-borne pathogens, and can be used when pain and discomfort restrict oral hygiene practices; use:
  chlorhexidine 0.2% mouthwash alcohol-free, 10 mL rinsed in the mouth for 1 minute then spat out, 8- to 12-hourly (use diluted with 10 mL of water if stinging occurs).
- Chlorhexidine gel is an alternative oral hygiene adjunct to mouthwash, and may provide some lubrication and ease discomfort; use:
  chlorhexidine 0.5% gel alcohol-free, apply 2 to 3 times daily to all mucosal surfaces and gingival margins.
- Nutritional support for patients with mucositis is important. Encourage patients to avoid irritant foods (e.g. acidic, spicy, salty, dry or abrasive foods). Refer patients for specialist nutritional advice if mucositis is severe.

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Dry mouth

The subjective feeling of dry mouth (xerostomia) is a relatively common condition that may or may not occur in the context of salivary gland hypofunction (an objective reduction in the quantity and the quality of saliva).

Many physiological and pathological conditions and drugs can cause salivary gland hypofunction or dry mouth. Common causes of dry mouth Include:

- dehydration
- alcohol
- anxiety
- mouth breathing
- drugs

If common causes have been excluded, investigate for less common medical conditions associated with dry mouth (e.g. Sjogren syndrome).

Drugs frequently associated with dry mouth

- anticholinergic drugs
- antihistamines
- drugs to lower blood pressure
  - — angiotensin converting enzyme inhibitors
  - — angiotensin II receptor blockers
  - —alpha blockers
  - — beta blockers
  - — diuretics
- inhaled bronchodilators
  - — beta2 agonists (eg salbutamol)
  - — muscarinic antagonists (eg tiotropium)
- opioids
- psychotropic drugs
  - — antidepressants
  - — antipsychotics
  - — illicit drugs (e.g. marijuana, cocaine)
  - — psychostimulants (e.g. amfetamines)
- urinary antispasmodics

Dry mouth is likely to be more severe if these drugs are used in combination.

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Dry mouth is a debilitating adverse effect of head and neck radiotherapy with the degree of salivary flow reduction dependent on the dose and region of the radiation.

Chronic dry mouth can have a profound effect on the oral environment and can contribute to:

- tooth decay and erosion
- periodontal disease
- oral mucosal disease
- oral candidiasis
- difficulty with the retention of dentures
- difficulty with chewing, swallowing and speech
- altered sense of taste

Management of dry mouth

Encourage patients to have a dental review and any necessary dental treatment before starting a drug that can cause dry mouth.

Review the patient's medications and, in conjunction with the prescriber, stop any non-essential medications that can cause a dry mouth.

Management strategies for patients with dry mouth include:

- ensuring adequate hydration
- ensuring good oral hygiene
- regular dental examination and treatment every 3 to 6 months
- topical re-mineralising agents to prevent tooth decay
- symptomatic relief

Options for the symptomatic relief of dry mouth include:

- artificial salivary products or other oral lubricants (e.g. bicarbonate mouthwash) (however, effects may be too transient to be of significant benefit)
- products that stimulate saliva, such as throat lozenges or chewing gum (however, many products are acidic or have a high sugar content, which can cause further tooth decay).

If symptomatic measures are inadequate, or if dry mouth is a symptom of systemic disease, refer patients to an appropriate specialist.

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ORAL MUCOSAL DISEASE

Reference: TG

Practical advice for patients with dry mouth

To manage your dry mouth:

- ensure you are adequately hydrated—drink at least 1.5 litres of tap water a day
- chew food thoroughly before swallowing because chewing stimulates saliva flow
- chew sugarless gum or suck sugarless sweets (avoid fruit flavours)
- avoid smoking cigarettes
- avoid acidic foods
- limit your caffeine and alcohol intake, especially in the evening
  — add milk to tea or coffee to reduce the drying effect
- avoid mouthwashes and other oral preparations that contain alcohol
- trial various over-the-counter dry mouth products or bicarbonate mouthwash
  — a bicarbonate mouthwash can be made by adding half a teaspoon of
- bicarbonate powder to a glass of warm water. Rinse with mouthwash on waking and at any time during the day.

To prevent oral and dental consequences of dry mouth:

- ensure you have good oral hygiene
- have regular dental examinations
- avoid acidic beverages (e.g. wine, fruit juices, soft drinks, sports drinks) or limit their consumption to meal times
- limit your sugar intake and avoid sugary snacks

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

The clinical presentation of common oral viral infections encountered in the dental practice are discussed, including

- herpes simplex virus types 1 and 2 (HSV)
- Epstein-Barr virus
- varicella-zoster virus
- Coxsackie virus
- human papilloma virus (HPV)
- human immunodeficiency virus (HIV)

The diagnosis, principles of management and pharmacological agents available for the treatment of oral viral infections are also discussed.

Viruses are not self-reproducing. They need the presence of another organism or host to reproduce or replicate. The host possesses ribosomes which the virus itself cannot synthesize. Viruses contain only one type of nucleic acid, either DNA or RNA. They are reproduced solely from their nucleic acid, i.e., a virus never arises directly from a pre-existing virus.

Nearly all human viruses possess a protein shell (nucleocapsid). Some also have an envelope surrounding the capsid. This envelope is made from lipids which are derived from host cell membranes.

Four different types of nucleic acid genomes are found in human viruses: single stranded DNA (ssDNA), double-stranded DNA (dsDNA), single stranded RNA (ssRNA) and double stranded RNA (dsRNA).

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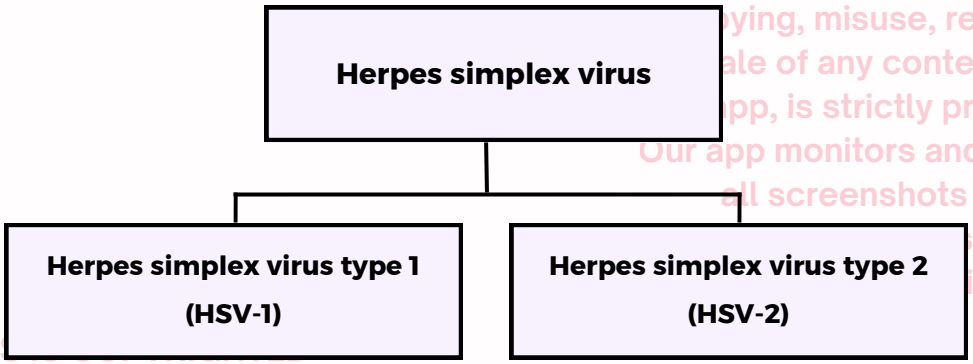
ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

1) Oral mucocutaneous herpes



- Primary herpetic gingivostomatitis
- Secondary herpes simplex lesions (Herpes labialis)

Primary oral mucocutaneous herpes

Reference: TG

Primary oral mucocutaneous herpes simplex virus (HSV) infection (herpetic gingivostomatitis) often occurs in childhood with fever, painful intraoral lesions, systemic symptoms (e.g. malaise, lethargy) and cervical lymphadenopathy.

Intraoral herpes simplex virus lesions begin as blisters an ulcerate rapidly. Healing occurs within several day; in infants but can take up to 2 weeks in older children. During this time, it may be difficult to eat, and drink and hospital admission may be required.

Herpetic gingivostomatitis is rare in adults but can be severe and present with dehydration due to severe odynophagia.

While herpes simplex virus is the most common virus to cause mouth ulcers, other viruses (e.g. varicella zoster virus, coxsackie virus, cytomegalovirus) may be the cause. Intraoral herpes simplex virus lesions may resemble those seen in necrotising gingivitis.

However, necrotising gingivitis is rare in children and is confined to the gingival tissues, while herpetic gingivostomatitis lesion are widespread and affect all soft tissues in the mouth.

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ORAL VIRAL INFECTIONS

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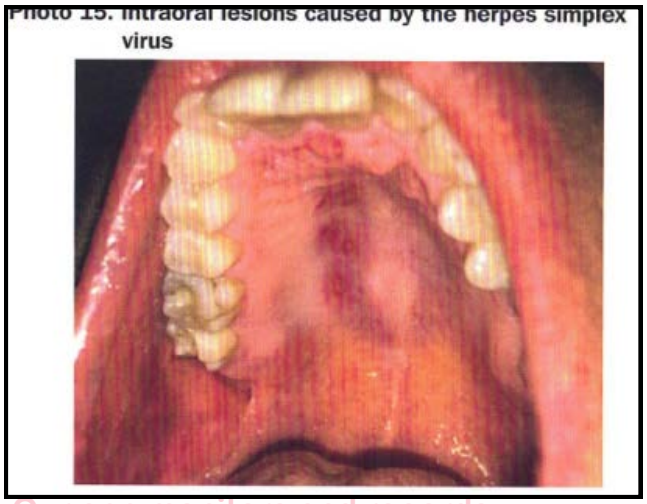
1) Oral mucocutaneous herpes

Primary oral mucocutaneous herpes

Reference: TG



To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry

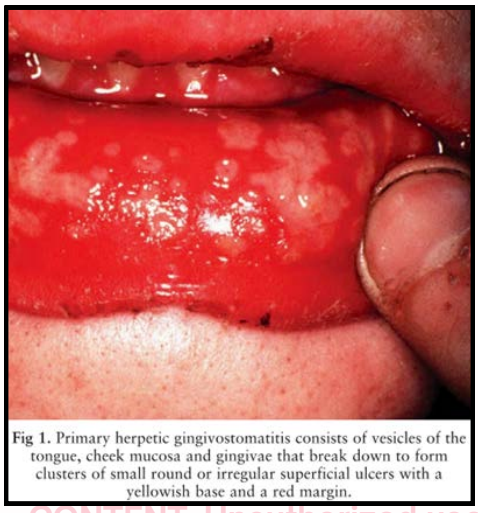


Fig 1. Primary herpetic gingivostomatitis consists of vesicles of the tongue, cheek mucosa and gingivae that break down to form clusters of small round or irregular superficial ulcers with a yellowish base and a red margin.

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1) Oral mucocutaneous herpes

Primary oral mucocutaneous herpes

Management:

- Treat minor primary oral mucocutaneous herpes with supportive management (i.e. oral fluids, antipyretic drugs and analgesia).
- Apply a topical anaesthetic or analgesic, such as:
  - benzydamine 1% gel (adult and child 6 years or older), topically to the lesions, 2-to 3-hourly as necessary.
- If this is not available, lidocaine viscous solution is an alternative topical anaesthetic for hospital settings (but be aware of the higher cost):
  - lidocaine 2% viscous solution adult: use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours
  - child 3 years or older: use the lowest dose necessary up to 0.2 ml./kg (maximum 5 mL), rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 4 doses in 24 hours
  - child younger than 3 years: use the lowest dose necessary up to 0.2 ml/kg (maximum 1.25 mL), applied to the affected areas with a cotton swab, 3-hourly as necessary; maximum 4 doses in 24 hours.
- lithe pharynx is affected in adults and children older than 12 years, lidocaine viscous solution can be gargled and swallowed.

Management of primary oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- patients with severe presentations
- immunocompromised patients
- patients with HIV.

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ORAL VIRAL INFECTIONS

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1) Oral mucocutaneous herpes

Recurrent oral mucocutaneous herpes (Herpes labialis)

Recurrent oral mucocutaneous herpes simplex virus (HSV) infection follows latent reactivation of the virus. Lesions usually occur on the lips (herpes simplex labialis or cold sores) but can also occur on the intraoral mucosa or other areas of skin.

Lesions are usually preceded by the prodromal stage, lasting several hours to days, which features pain, burning, tingling or itching.

Recurrences are usually mild and infrequent, and their frequency can be minimised with sun protection. Herpes simplex virus reactivation may be complicated by erythema multiforme.

A herpes simplex virus lesion affecting the oral mucosa cannot be differentiated from an aphthous or traumatic ulcer using microbiological testing—most adults will have positive serology for the herpes simplex virus from previous exposure, and viral DNA may be detected on swabs of aphthous or traumatic ulcers. Instead, diagnosis requires a thorough history and clinical examination.

To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

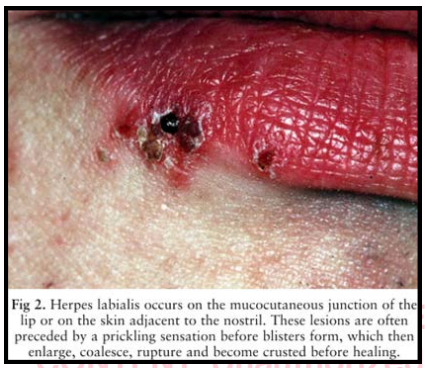

Fig 2. Herpes labialis occurs on the mucocutaneous junction of the lip or on the skin adjacent to the nostril. These lesions are often preceded by a prickling sensation before blisters form, which then enlarge, coalesce, rupture and become crusted before healing.

Secondary herpes simplex lesions (Herpes labialis) develop in susceptible people most often at the mucocutaneous junction of the lip or on the skin adjacent to the nostril. The development of these lesions is often preceded by a prickling sensation before blisters form. These blisters then enlarge, coalesce, rupture and become crusted before healing (Fig 2). This usually takes 7-10 days in healthy individuals, but in immunocompromised patients' secondary herpetic lesions can be widespread, very slow to heal and refractory to treatment.

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ORAL VIRAL INFECTIONS

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1) Oral mucocutaneous herpes

Recurrent oral mucocutaneous herpes (Herpes labialis)

Management:

For a minor recurrence of oral mucocutaneous herpes, episodic antiviral therapy may reduce its duration; use:

- **aciclovir (adult and child older than 3 months) 5% cream topically, 5 times daily (every 4 hours while awake) for 5 days, started at the first sign of recurrence or during the prodromal stage**

OR

- **famciclovir (adult) 1500 mg orally, as a single dose, taken at the first sign of recurrence or during the prodromal stage.**

Using aciclovir cream for longer than recommended has no benefit.

Management of recurrent oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- **patients with severe recurrences of herpes (with systemic signs and symptoms, or if the patient has difficulty eating or swallowing)**
- **patients with generalised or chronic herpes infection (with crusted lesions and ulceration)**
- **immunocompromised patients**
- **patients with HIV.**

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2) Epstein-Barr virus

Epstein-Barr virus (EBV) is involved in a diverse range of conditions such as

- **infectious mononucleosis**
- **oral hairy leucoplakia**
- **non-Hodgkin's lymphoma**
- **Burkitt's lymphoma**
- **nasopharyngeal carcinoma**

The first two of these conditions will be discussed below.

Infectious mononucleosis (glandular fever)

This is a relatively common disease affecting both genders equally and occurring predominantly in teenagers and young adults. There is a relatively long incubation time (35+days). Transmission is through salivary spread with resultant pharyngitis, lymphadenopathy, malaise, arthralgia and myalgia.

Oral hairy leucoplakia (OHL)

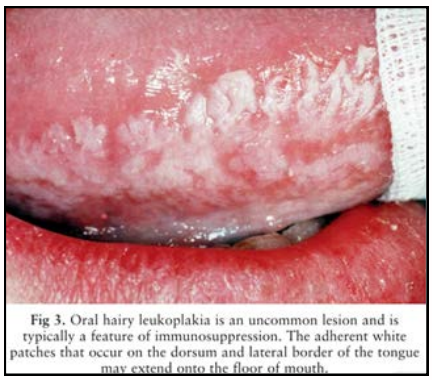

Fig 3. Oral hairy leukoplakia is an uncommon lesion and is typically a feature of immunosuppression. The adherent white patches that occur on the dorsum and lateral border of the tongue may extend onto the floor of mouth.

This is very uncommon and occurs equally in both genders. It is typically a feature of immunosuppression. It is characterized by adherent white patches, bilateral, on the dorsum and lateral border of tongue that may extend onto the floor of mouth. The cause of this condition is now thought to be an EBV-related epithelial proliferation that arises due to the associated immunosuppression.

Principally, this has been reported in

- **HIV disease**
- **cyclosporin-induced immunosuppression**
- **long-term high-dose corticosteroids use**
- **uncontrolled diabetes mellitus.**

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3) Varicella-zoster virus

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The primary infection of this virus is commonly a childhood illness: chicken pox. This disease has a prodromal illness with oral vesicles and ulcers typically on the palate and a skin rash that can be pruritic, papular and pustular with vesicles' that most often occur on the trunk. The disease is self-limiting, lasting 5-10 days and is usually contracted by direct contact.

Shingles is the secondary infection of varicella-zoster virus and tends to occur only in middle to late life in both genders. It occurs via viral reactivation and can be associated with immunodeficiency. Typically, this occurs in a dermatomal distribution on the thorax, but can occur facially following the divisions of the trigeminal nerve. When they occur, clinical oral features are unilateral vesicles and ulcers.

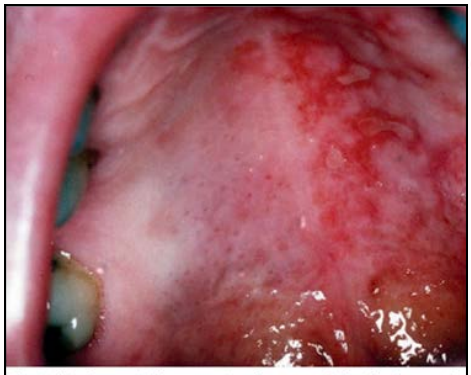

Fig 4. Shingles typically occurs in a dermatomal distribution on the thorax, but can occur facially following the divisions of the trigeminal nerve with unilateral vesicles and ulcers.

Ramsay Hunt Syndrome occurs when reactivation involves the chorda tympani, vesicles and ulcers of the external ear (otitis externa), anterior ⅔ tongue, soft palate and facial palsy (Ramsay Hunt Syndrome is rarely bilateral).

Postherpetic neuralgia occurs in approximately 30 per cent of patients with shingles and is localized, precipitated by light touch and can be very acute, sharp pain. The involved skin can be erythematous.

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ORAL VIRAL INFECTIONS

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4) Coxsackie virus

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

This virus causes two conditions that involve the oral mucosa.

a) Herpangina

Herpangina is a systemic infection, common in childhood. Fever and sore throat usually last for two days and are followed by the appearance of lesions in the oropharynx. These are numerous small vesicles, 1- 2mm in diameter, found mostly on the pillar of fauces, uvula, tonsils and palate. Unlike herpetic gingivostomatitis, the gingivae are not commonly affected. The oral ulceration will last for three or four days and, unlike herpes infections, there will be no recurrences of that particular type of Coxsackie virus.

a) Hand, foot and mouth disease

Hand, foot and mouth disease is another Coxsackie virus infection most commonly seen as an epidemic among young schoolchildren. It is characterized by the presence of small vesicles on the oral mucosa, palmar surfaces of the hands and plantar surfaces of the feet. The presence of extra-oral lesions helps distinguish it from herpetic gingivostomatitis.

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

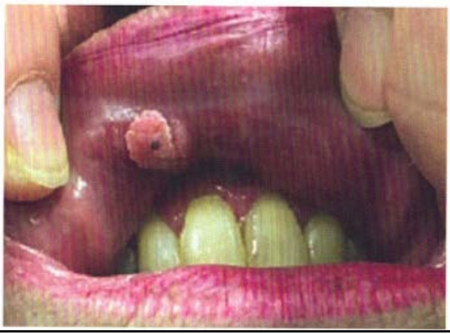
5) Human papilloma virus-related oral lesions

Reference: TG

Human papilloma viruses (HPV) can cause a wide range of oral mucosa lesions. The virus is usually transmitted by direct contact with a lesion.

Squamous papilloma is the most common oral HPV lesion, appearing as a protruding growth with small finger-like projections.

Photo 7. Papilloma of the right maxillary labial mucosa



Sexually transmitted HPV infections can cause oral HPV lesions called condyloma acuminata.

Verruca vulgaris—the common wart—is also caused by HPV infection and may present in the oral cavity.

Both condyloma acuminata and verruca vulgaris can be clinically similar to squamous papilloma.

Oncogenic types of HPV are now recognised as a cause of some squamous cell carcinoma, particularly of the posterior tongue, tonsillar region and oropharynx. These appear to be a distinct entity, separate to the oral cancers associated with alcohol and tobacco use.

Refer patients with suspected HPV lesions to an appropriate specialist for biopsy and management.

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

5) Human papilloma virus-related oral lesions

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry

Human papilloma virus (HPV) has been shown to be linked to a number of benign lesions of the oral mucosa, such as squamous cell papilloma, condyloma, verruca and focal epithelial hyperplasia (FEH).

Squamous cell papilloma (SCP) is a relatively common benign tumour of the oral epithelium, representing about half of all soft tissue tumours. On gross appearance, oral papilloma is characterized by small finger-like projections, resulting in a lesion with a rough or cauliflower-like verrucous surface. Oral papilloma are benign lesions.

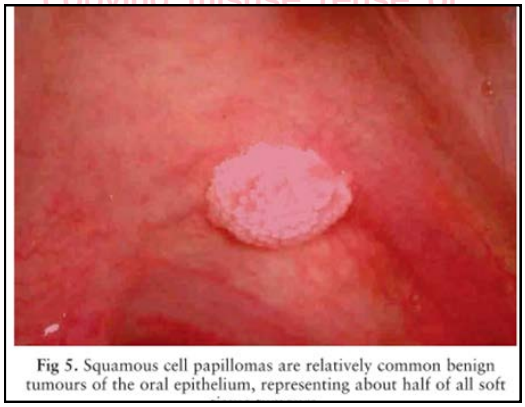

Fig 5. Squamous cell papillomas are relatively common benign tumours of the oral epithelium, representing about half of all soft

Condyloma acuminatum (venereal wart) is generally regarded as a sexually transmitted disease affecting the skin and mucous membranes of the anogenital tract. It is now accepted that oral condylomas can arise not only by oral sex but also by autoinoculation or as a result of maternal transmission.

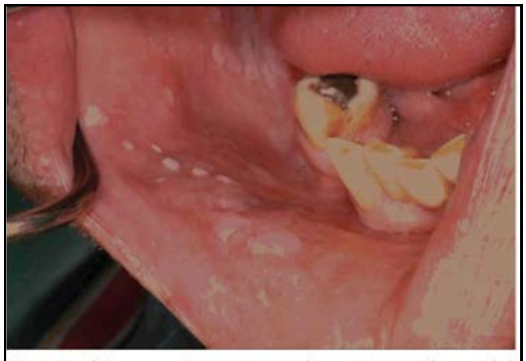

Fig 6. Condyloma acuminatum (venereal wart) is generally regarded as a sexually transmitted disease affecting the skin and mucous membranes of the anogenital tract but can also occur in the oral cavity and are known as oral condylomas.

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

5) Human papilloma virus-related oral lesions

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry

Verruca vulgaris (common wart) is the most prevalent HPV lesion of the skin, but is also found in oral mucosa. The most common locations are the mucosal areas in which keratinization of the epithelium resembles that of the skin, i.e., lip, hard palate and gingivae. It has been emphasized that the diagnosis of oral verruca should be preserved for lesions showing histological characteristics of verruca vulgaris of the skin. On clinical examination, verruca is often indistinguishable from SCP and condyloma. To confirm the diagnosis, cutaneous HPV types should be identified in oral verruca. So far, there are no follow-up studies on the natural history of oral verruca.

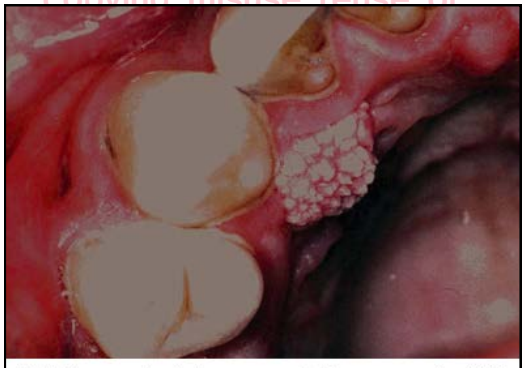

Fig 7. Verruca vulgaris (common wart), the most prevalent HPV lesion of the skin can also be found on the oral mucosa. On clinical examination, verruca is often indistinguishable from the squamous papilloma and condyloma.

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

6) Human immunodeficiency virus

The acquired immune deficiency syndrome (AIDS) is the most serious expression of disease resulting from infection with the human immunodeficiency virus (HIV). A diagnosis of AIDS implies that there has been some damage to the immune system resulting in opportunistic infections or secondary cancers. Infection with HIV causes a continuum of clinical conditions. These can range from the asymptomatic carrier state to mild-to-more severe AIDS-related conditions to the diseases of AIDS itself.

Mild-to-moderate states encompass a wide spectrum of disease, e.g., OHL, whilst AIDS itself is characterized by more life-threatening infections, neurological manifestations or secondary cancers. Throughout the course of HIV infection, the virus continues to replicate rapidly. CD4 cells, the major cells targeted by HIV, are killed and replaced in large numbers, until, finally, the capacity of the immune system to respond further is exhausted, resulting in severe immunodeficiency.

Oral manifestations of HIV infection are:

- OHL
- Oral candidiasis
- Kaposi's sarcoma (KS)

If it is suspected that patients exhibit any of these conditions and infection with HIV is suspected it is advisable to refer the patient. KS is a common neoplasm in AIDS. Oral involvement may be observed in up to 60 per cent of patients with KS; 45 per cent of patients have both, skin and oral lesions. Oral KS frequently involves the palate, the attached gingivae and the dorsum of the tongue. Clinically, a macular early lesion and a papulonodular form are recognized. The aetiology of AIDS-related KS has been extensively investigated, with human herpesvirus 8 (HHV8) being the key agent in the development of this lesion.

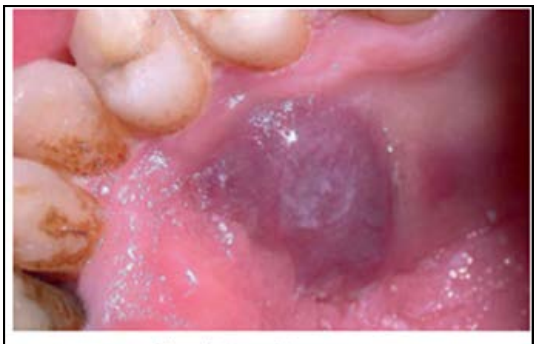

Fig. 8 Kaposi's sarcoma.

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Interactions with Antiretroviral Drugs and Drugs Commonly Used in Dental Practice

Reference: Odell

Drug	Antiretroviral Drug	Effect
Metronidazole	Atazanavir, darunavir, lopinavir, tipranavir, ritonavir	Antiretroviral formulations may contain alcohol, causing a disulfiram reaction.
Clindamycin	Ritonavir	Increase in clindamycin levels.
Erythromycin	Darunavir, fosamprenavir, indinavir, lopinavir, saquinavir, tipranavir, ritonavir	Large increase in erythromycin levels.
Diazepam, midazolam	Protease inhibitors, non-nucleoside reverse transcriptase inhibitors	Oral administration of many benzodiazepines is contraindicated because of their altered metabolism. This leads to an increase in their sedative effects. Oral midazolam particularly is to be avoided. Note that intravenous midazolam, as used in dentistry, is titrated to the patient's response, and this is not contraindicated. However, great care needs to be taken in the titration to avoid oversedation. Proceed slowly. There remains a possibility of prolonged sedation, although this does not appear to be a problem in clinical practice. Reversing the sedative effect with flumazenil may theoretically result in late re-sedation. The recovery time to discharge should be increased to monitor this.
Lidocaine (lignocaine)	Protease inhibitors, non-nucleoside reverse transcriptase inhibitors	There is a theoretical risk of impaired lidocaine (lignocaine) metabolism. The significance of this is unclear in the dental setting but it would be prudent to avoid approaching the accepted maximum dose.
Fluconazole	Tipranavir, nevirapine	Increase in levels of tipranavir, nevirapine.
Miconazole oral gel	Protease inhibitors, non-nucleoside reverse transcriptase inhibitors	Poorly absorbed but may be ingested with saliva. Risk of increase in antiretroviral levels in blood if used to excess.

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ORAL VIRAL INFECTIONS

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

7) Hepatitis C (HCV) infection

Reference: The increasing problem of hepatitis C virus infection

The widespread incidence of hepatitis C (HCV) infection throughout the community is of concern. Although many of those infected will not suffer significantly from their infection, up to one-third will have liver disease, fatigue and oral health problems. General dental practitioners need to be aware of the precautions necessary in treating people with severe liver disease. This paper discusses the issues associated with treating patients who have HCV infection including the importance of preventive programs to reduce dental pathology and maximise oral health.

Transmission of HCV to staff in the dental surgery

More than a decade ago, concern over transmission of HCV in the dental setting was identified as an important issue both for healthcare workers and their patients. The dominant mode of transmission is blood-to-blood contact, with quoted transmission rates of between 0-10 per cent (average: 1.8 per cent) in the situation where a healthcare worker sustains a sharps injury from an infected patient. While this is less than the comparable figure for hepatitis B virus (HBV), which has a transmission rate of 25-35 per cent, it should be remembered that healthcare workers can be immunised against HBV but not HCV.

These assessments may need to be reviewed in the light of increasing prevalence of HCV in the community at large, taking into account more recent data on the risk of HCV transmission from sharps injury. It is currently believed the risk of occupational acquisition of HCV infection is greater for healthcare workers than the general community, particularly those healthcare workers in contact with seropositive patients, however the risk is small. Current data are relatively limited, but it appears the prevalence of HCV infection is not notably higher in dentists than in the general community. There is, however, an increase in prevalence with increasing years of practice and occupationally acquired HCV has also occurred in dental students. Of note, HCV is more prevalent in oral surgeons than in general dentists and this may reflect not only exposure-prone procedures but also a higher rate of HCV infection in the patient groups treated. For example, in a survey of patients attending hospital oral surgery departments, 1.1 per cent of patients had anti-HCV antibodies, a considerably higher percentage than in the general community.

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Transmission to patients

An increased prevalence of HCV has been documented in some populations of dental patients, implying cross-infection from patient to patient or staff to patient in the dental setting. Hospitalisation and medical/dental care were implicated as risk factors for non-drug injecting people. Piazza et al27 demonstrated dental treatment was the only risk factor in 9 per cent of cases of acute HCV infection. Through the use of the polymerase chain reaction (PCR) assay, the same researchers demonstrated HCV genetic material could be recovered from dental equipment, handpieces, burs and workbenches and extensive contamination of dental surgeries occurred following treatment of HCV-infected patients. Clearly, if sterilisation and decontamination procedures are inadequate, transmission of HCV in the dental surgery is a real possibility. Flamm et al17 noted dental treatment may have been a risk factor for HCV before infection control precautions became commonplace.

Dental management of the HCV-infected patient

The most significant problem to be faced with a patient suffering cirrhosis will be the likelihood of prolonged bleeding following dental procedures. This bleeding is caused by a lack of coagulation factors and thrombocytopaenia (Fig 1, 2). Consequently, any invasive dental treatment (extractions, surgery and extensive periodontal treatment) should be undertaken after consultation with the appropriate medical specialists. Simple treatment may be carried out utilising agents to establish local control of bleeding (for example, topical tranexamic acid).

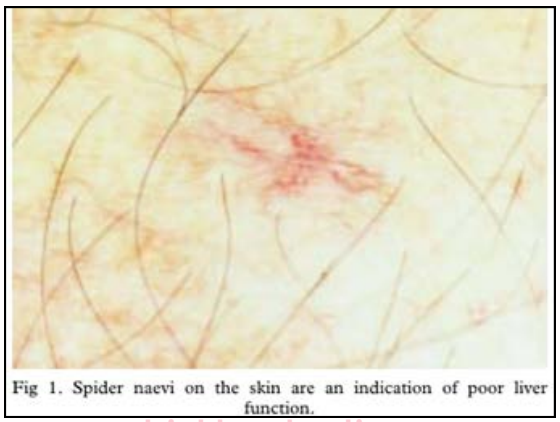

Fig 1. Spider naevi on the skin are an indication of poor liver function.

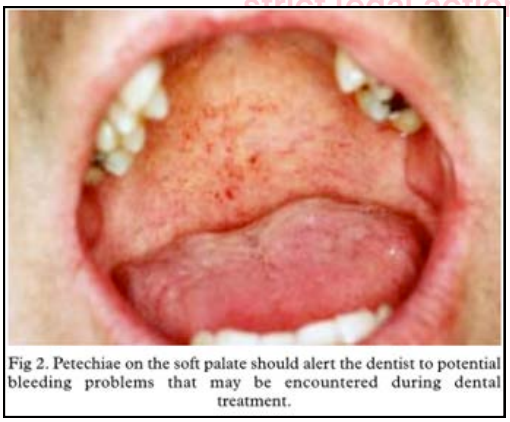

Fig 2. Petechiae on the soft palate should alert the dentist to potential bleeding problems that may be encountered during dental treatment.

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7) Hepatitis C (HCV) infection

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Dental management of the HCV-infected patient

There is a small but significant risk for a patient with severe cirrhosis that drug interactions and toxicity will burden an already stressed liver. The use of octapressin as a vasoconstrictor, for example, is contraindicated in someone with extensive liver dysfunction. Prescribing medications that are processed or excreted in the liver is also potentially hazardous. Drugs such as metronidazole, tetracyclines, erythromycin and paracetamol are contraindicated for people suffering liver failure.

In addition to medical complications arising from liver disease, problems in delivering dental treatment also exist for those undergoing HCV infection therapy. Drugs such as IFN, ribavirin and corticosteroids may lower resistance to infection and cause bleeding, so invasive dental treatment should be postponed until therapy has ceased. Urgent dental treatment needs to be undertaken in consultation with the appropriate medical specialists.

Recent data indicate people with HCV infection may be prone to extensive dental disease (Fig 3). This further complicates management for those who do suffer significant cirrhosis.

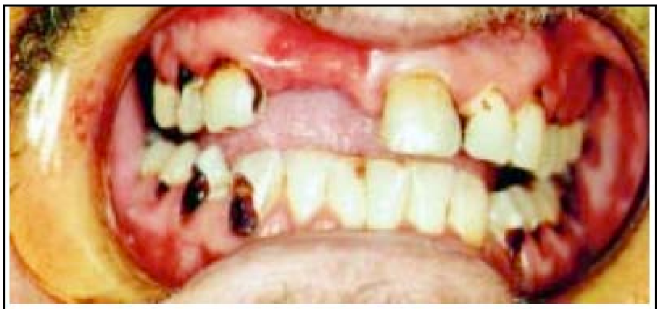

Fig 3. Rampant dental caries can be present in HCV infection and, as a result, appearance may be affected.

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Dental management of the HCV-infected patient

The contributing factors to the dental disease burden are varied, but xerostomia is a notable feature of HCV infection and oral health is impacted by this factor (Fig 4). Farrell3 reported a case of a man presenting with fatigue and irritability. Based on raised ALT and AST levels, HCV infection had been present undiagnosed for approximately 20 years. Clinically, the only symptom of disease was an enlarged parotid gland. Dentists need to be aware of the possible effects of a chronic viral infection on salivary gland function and institute appropriate preventive strategies to maintain dental health.

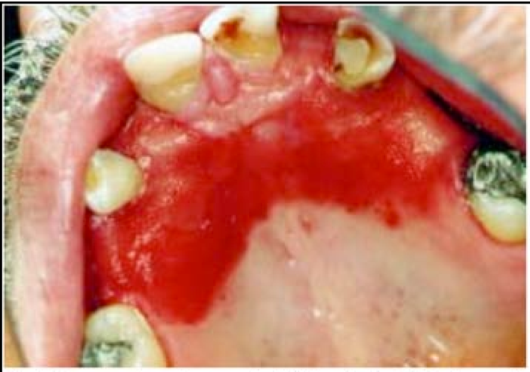

Fig 4. Xerostomia can increase the likelihood of oral infections such as candidiasis. In this patient, the candida infection is also related to the wearing of a partial denture.

Studies suggest a correlation between lichen planus and HCV infection. However, the reason for the correlation is unknown and although it has been proposed erosive lichen planus is an indication for serological testing for HCV infection, this would seem to be unwarranted at this stage.

Finally, alcoholism may be a problem for some patients and these individuals may show accelerated tooth wear from erosion.

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Conclusion

The widespread incidence of HCV infection is of concern. Although many of those infected will not suffer significantly from their infection, up to one-third will have liver disease, fatigue and oral health problems. Practitioners need to be aware of the precautions necessary in treating people with severe liver disease and the importance of preventive programs to reduce dental pathology and maximise oral health for individuals infected with HCV.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

This paper discusses the range of recurrent oral ulceration which affects the oral mucosa. Types of ulceration covered in this paper include:

- Traumatic
- Infective
- Aphthous
- ulceration related to the oral dermatoses
- drug-induced
- ulceration as a manifestation of systemic disease
- ulceration indicating malignancy

The diagnosis and management of the patient with recurrent oral ulceration requires a systematic approach based on the principles of taking an adequate history, clinical examination, investigations as appropriate, institution of management and, finally, review to allow for any necessary modifications of that management. It is worthwhile to begin with a definition of an ulcer: an ulcer is a complete breach of the epithelium. This becomes covered with a fibrin slough and appears as a yellow/white lesion surrounded by erythema.

Some types of recurrent oral ulceration have a typical onset in childhood or adolescence (such as recurrent aphthous ulceration/stomatitis). This pattern of oral ulceration can sometimes present in later life but a middle-aged or elderly patient presenting with recurrent oral ulceration should also raise other diagnostic possibilities such as lichen planus and vesiculobullous disorders.

A more typical pattern of recurrent oral ulceration will be characterized by periods of ulceration with remissions between bouts of ulceration. Some patients complain of an altered sensation prior to ulcer development which is known as a prodromal phase.

Because some patients with recurrent oral ulceration may have extraoral manifestations, questions should be directed to any skin involvement or other systems being affected such as the eyes or genital regions, which would raise a clinical suspicion of Behcet's syndrome. Some patients with recurrent oral ulceration may have a vesiculobullous disorder and questioning regarding any awareness of blistering before the ulcers appear should be pursued. At this stage it is reasonable to ask the patient whether they have any ulcers present at the time of the consultation. If this is not the case, then a presumptive diagnosis can be made at the end of the initial consultation and the patient reviewed when the ulcers next appear.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Medical history

Many patients with recurrent oral ulceration are in good health but some may have pre-existing medical problems which may be of relevance. These may include anaemia, blood dyscrasias, autoimmune disease and diabetes. The medical history will include ascertaining any medication taken by the patient. Some medications are associated with oral ulceration, e.g., methotrexate.

Dental History

Oral ulceration which appears after dental treatment can be an indicator of minor recurrent aphthous ulceration. Minor trauma to the tissues can precipitate ulcers in susceptible patients. Some patients may report a crop of ulcers at the same site in the mouth occurring after dental treatment. This may occur in the palate or buccal sulcus and would raise a suspicion of recurrent intraoral herpes simplex virus infection – effectively an oral "cold sore".

Examination

Extraoral examination should focus on general appearance including a crude measure of nutritional status. Assessment of skin and conjunctival pallor may assist in identifying anaemic patients. The regional lymph nodes should be palpated as these may be enlarged in the case of persistent or large ulcers.

Intraoral examination should assess the presence or absence of ulcers. The number, shape, size and location of the ulcers should be recorded. The presence or absence of scarring should be established. Minor recurrent aphthous ulceration will tend to present with several more or less circular ulcers on the buccal/labial mucosa and lateral and ventral surfaces of the tongue. In a patient with herpetiform ulceration, multiple pinpoint ulcers would typically be seen on the non-keratinized mucosa with the possibility of more ragged ulcers by virtue of adjacent ulcers enlarging and fusing. Major aphthous ulcers tend to be larger (>10 mm diameter) and are more commonly seen in the oropharynx; they heal with scarring.

A pattern of more ragged ulceration, perhaps with peeling of the adjacent epithelium, would raise the possibility of a vesiculobullous disorder, such as mucous membrane pemphigoid or pemphigus vulgaris. Clinical distinction between the two can be difficult but in pemphigus vulgaris the vesicles are short-lived and therefore infrequently seen, whereas in mucous membrane pemphigoid the blisters, by virtue of their full-thick- ness roof, can persist for longer. Bleeding, crusting and ulceration of the lips should raise a suspicion of erythema multiforme. Ulcers related to a denture margin may also come within the category of recur- rent oral ulceration. In such a case it may simply be a recurrent traumatic ulcer related to the denture.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Causes of oral ulceration

1) Trauma

Traumatic ulceration may be recurrent if the offending irritant is not removed. The irritant may be mechanical, thermal or chemical in nature.

2) Viral infection

Recurrent intraoral viral infection is usually limited to secondary herpes simplex virus. The recurrences are most commonly due to human herpes virus-1 (HHV-1) (which usually causes orofacial infections). Recurrent oral HHV-2 lesions (which are usually associated with genital infections) are rare. Clinically, the initial presentation is of fluid-filled vesicles which rapidly break down to form a cluster of small ulcers with ragged margins. The lesions usually resolve in about 7 to 10 days in healthy individuals, but in immunocompromised patients' secondary herpetic lesions can be widespread, very slow to heal and refractory to treatment.

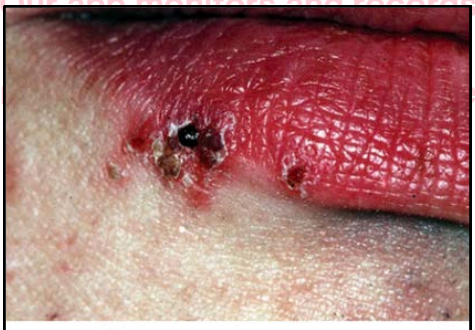

Fig 2. Herpes labialis occurs on the mucocutaneous junction of the lip or on the skin adjacent to the nostril. These lesions are often preceded by a prickling sensation before blisters form, which then enlarge, coalesce, rupture and become crusted before healing.

Reference: Oral viral infections and the therapeutic use of antiviral agents in dentistry

Secondary herpes simplex lesions (Herpes labialis) develop in susceptible people most often at the mucocutaneous junction of the lip or on the skin adjacent to the nostril. The development of these lesions is often preceded by a prickling sensation before blisters form. These blisters then enlarge, coalesce, rupture and become crusted before healing (Fig 2). This usually takes 7-10 days in healthy individuals, but in immunocompromised patients' secondary herpetic lesions can be widespread, very slow to heal and refractory to treatment.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Management

Reference: TG

For a minor recurrence of oral mucocutaneous herpes, episodic antiviral therapy may reduce its duration; use:

- **aciclovir (adult and child older than 3 months) 5% cream topically, 5 times daily (every 4 hours while awake) for 5 days, started at the first sign of recurrence or during the prodromal stage**
- **OR**
- **famciclovir (adult) 1500 mg orally, as a single dose, taken at the first sign of recurrence or during the prodromal stage.**

Using aciclovir cream for longer than recommended has no benefit.

Management of recurrent oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- **patients with severe recurrences of herpes (with systemic signs and symptoms, or if the patient has difficulty eating or swallowing)**
- **patients with generalised or chronic herpes infection (with crusted lesions and ulceration)**
- **immunocompromised patients**
- **patients with HIV.**

Reference: The patient with recurrent oral ulceration

Other viral infections occurring in the mouth are due to varicella-zoster virus and coxsackie virus. These infections may become recurrent if the patient is immunocompromised.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Varicella-zoster virus

Reference: : Oral viral infections and the therapeutic use of antiviral agents in dentistry

The primary infection of this virus is commonly a childhood illness: chicken pox. This disease has a prodromal illness with oral vesicles and ulcers typically on the palate and a skin rash that can be pruritic, papular and pustular with vesicles' that most often occur on the trunk. The disease is self-limiting, lasting 5-10 days and is usually contracted by direct contact.

Shingles is the secondary infection of varicella-zoster virus and tends to occur only in middle to late life in both genders. It occurs via viral reactivation and can be associated with immunodeficiency. Typically, this occurs in a dermatomal distribution on the thorax, but can occur facially following the divisions of the trigeminal nerve. When they occur, clinical oral features are unilateral vesicles and ulcers.

Ramsay Hunt Syndrome occurs when reactivation involves the chorda tympani, vesicles and ulcers of the external ear (otitis externa), anterior ⅔ tongue, soft palate and facial palsy (Ramsay Hunt Syndrome is rarely bilateral).

Postherpetic neuralgia occurs in approximately 30 per cent of patients with shingles and is localized, precipitated by light touch and can be very acute, sharp pain. The involved skin can be erythematous.

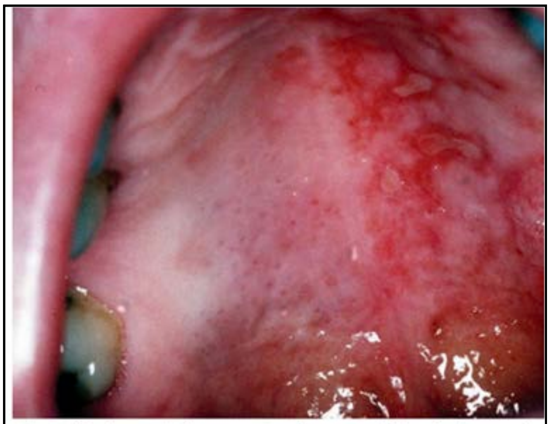

Fig 4. Shingles typically occurs in a dermatomal distribution on the thorax, but can occur facially following the divisions of the trigeminal nerve with unilateral vesicles and ulcers.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Coxsackie virus

Reference: : Oral viral infections and the therapeutic use of antiviral agents in dentistry

This virus causes two conditions that involve the oral mucosa.

a) Herpangina

Herpangina is a systemic infection, common in childhood. Fever and sore throat usually last for two days and are followed by the appearance of lesions in the oropharynx. These are numerous small vesicles, 1- 2mm in diameter, found mostly on the pillar of fauces, uvula, tonsils and palate. Unlike herpetic gingivostomatitis, the gingivae are not commonly affected. The oral ulceration will last for three or four days and, unlike herpes infections, there will be no recurrences of that particular type of Coxsackie virus.

a) Hand, foot and mouth disease

Hand, foot and mouth disease is another Coxsackie virus infection most commonly seen as an epidemic among young schoolchildren. It is characterized by the presence of small vesicles on the oral mucosa, palmar surfaces of the hands and plantar surfaces of the feet. The presence of extra-oral lesions helps distinguish it from herpetic gingivostomatitis.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Acute necrotizing ulcerative gingivitis (ANUG)

Reference: : The patient with recurrent oral ulceration

Ulceration due to bacterial infection, as in acute necrotizing ulcerative gingivitis (ANUG), may be recurrent. ANUG tends to be more prevalent in winter months and there is an association with smoking. Recurrences may be more likely if the patient has compromised general health.

Reference: : TG

Necrotising gingivitis (previously known as acute necrotising ulcerative gingivitis [ANUR affects the interdental papillae and gingivae; if not managed appropriately, it can spread to involve the bone. For management of necrotising gingivitis. Necrotising gingivitis most commonly occurs in young adult smokers and rarely occurs in children. Children thought to have necrotising gingivitis should be assessed for acute herpetic gingivostomatitis (for information on oral mucocutaneous herpes.

Management of necrotising gingivitis

Thorough debridement of plaque and necrotic debris is necessary for successful management of necrotising gingivitis. However, it may not be possible to complete debridement at the initial presentation because necrotising gingivitis can be associated with significant pain. Begin management of necrotising gingivitis with:

- **gentle removal of as much plaque and necrotic debris as possible, using local anaesthetics if necessary**
- **local irrigation with chlorhexidine 0.2% mouthwash or hydrogen peroxide 3% solution. Chlorhexidine mouthwash or hydrogen peroxide solution (as below) may also be used if pain limits the patient's ability to mechanically clean their teeth**
- **antibiotic therapy: metronidazole 400 mg orally, 12-hourly for 3 to 5 days.**
- **analgesics**
- **advice to stop smoking.**

Profoundly immunocompromised patients immune compromise or patients with severe cases of necrotising gingivitis require prompt referral for specialist management in addition to the management above.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Acute necrotizing ulcerative gingivitis (ANUG)

Reference: : The patient with recurrent oral ulceration

If pain and inflammation restrict oral hygiene practices, recommend short-term use of a mouthwash to reduce plaque formation; use:

- **1 hydrogen peroxide 3% solution 5 mL, mixed with 5 mL of warm water, rinsed in the mouth for 1 minute then spat out, 12-hourly until pain has reduced**

  OR
- **hydrogen peroxide 1.5% solution 10 mL, rinsed in the mouth for 1 minute then spat out, 12-hourly until pain has reduced**

  OR
- **2 chlorhexidine 0.2% mouthwash 10 mL rinsed in the mouth for 1 minute then spat out, 8- to 12-hourly until pain has reduced**

  OR
- **2 chlorhexidine 0.12% mouthwash 15 mL rinsed in the mouth for 1 minute then spat out, 8- to 12-hourly until pain has reduced.**

Review the patient in 48 to 72 hours; perform a periodontal examination end provide the patient with advice on oral hygiene. Perform thorough debridement as soon as possible to prevent recurrence.

A poor response to treatment or recurrence of symptoms is usually due to Inadequate debridement or a lack of improvement in oral hygiene, rather than an ineffective antibiotic regimen. If the infection has not responded to appropriate management (complete debridement, antibiotic therapy, improved oral hygiene) within 2 weeks, refer for specialist management.

Treatment failure is usually due to inadequate debridement or poor oral hygiene, rather than ineffective antibiotic therapy.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Reference: : The patient with recurrent oral ulceration

1) Nutritional deficiency

A nutritional deficiency such as a deficiency of iron, folate or vitamin B12 may predispose the patient to recurrent oral ulceration and it may aggravate RAU.

2) Haematological disorders

Haematological disease such as leukaemia, pancytopaenia, aplastic anaemia or agranulocytosis may present clinically with ulceration, but this ulceration is unlikely to be recurrent.

3) Medications

A number of medications, e.g., methotrexate may have a side-effect of oral ulceration (Fig 1). This side effect may be dose-related.

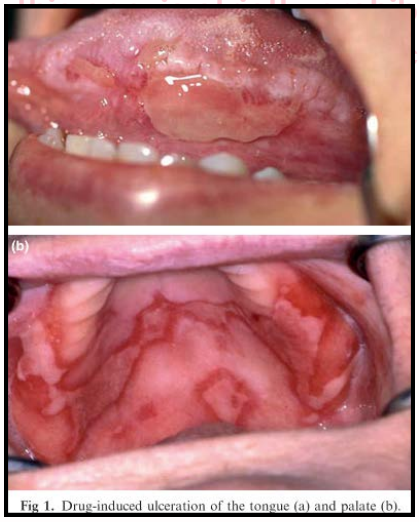

Fig 1. Drug-induced ulceration of the tongue (a) and palate (b).

4) Xerostomia

Xerostomia may predispose to recurrent oral ulceration, especially if dentures are worn. The xerostomia may be multifactorial in origin and may be due to autoimmune disease such as Sjogren's syndrome or the side effects of medications such as antidepressant medications.

5) Neoplastic disease

Although oral neoplastic disease may present with oral ulceration, the ulceration is persistent and progressive in nature.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Reference: : The patient with recurrent oral ulceration

6) Specific conditions to consider

Recurrent aphthous ulceration (RAU) is the most common form of recurrent oral ulceration, reportedly affecting up to 20% of the population. In most patients, the ulcers first appear in childhood or adolescence; there may be a slight female predisposition and in some patients, there is a family history of similar ulceration which suggests a genetic factor. The aetiology of the condition is not completely understood but is thought to be immunologically based.

Recurrent aphthous ulceration can occur in three forms:

i) Minor recurrent aphthous ulceration

This is the most common form, accounting for approximately 80–90% of cases. The ulcers are usually round or oval and occur on the non-keratinized oral mucosa. Thus, they tend to occur on the lip and cheek mucosa and lateral margins of the tongue, sparing the dorsum of the tongue, palate and gingivae. In the buccal or labial sulcus, the ulcers may be linear (Fig 2). One to five ulcers usually occur at a time and they are approximately 5 mm in diameter. The ulcers heal without scarring after 1 to 2 weeks and then recur, usually at intervals of a few weeks or months, although some patients are rarely without ulcers.

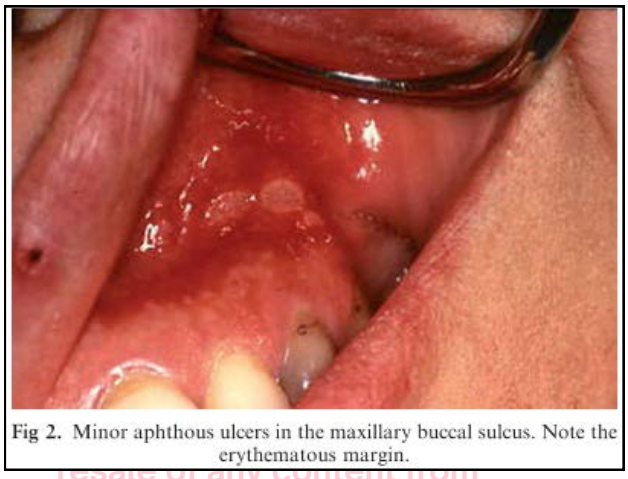

Fig 2. Minor aphthous ulcers in the maxillary buccal sulcus. Note the erythematous margin.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Reference: : The patient with recurrent oral ulceration

6) Specific conditions to consider

ii) Major recurrent aphthous ulceration

This form is much less common and accounts for about 5–10% of cases. The ulcers are similar to those of minor recurrent aphthous ulceration, but occur on any part of the oral mucosa including keratinized regions such as the hard palate and dorsum of the tongue as well as the oropharynx and can be larger than 10 mm in diameter (Fig 3). One or two ulcers generally occur at any one time. They tend to be persistent, lasting for at least one month, heal with scarring, and then recur.

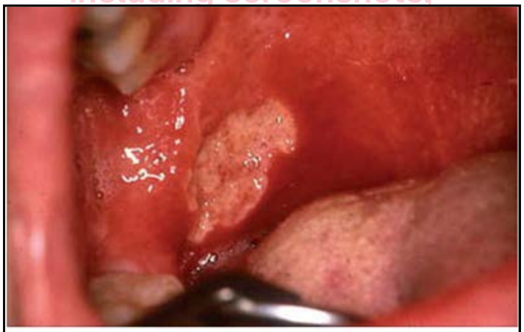

Fig 3. A major aphthous ulcer on the soft palate. The ulcer is large and irregular in shape.

iii) Herpetiform ulceration

This has a similar prevalence to major RAU. This form of ulceration begins as small round ulcers, approximately 1 mm in diameter (Fig 4), which are present in large numbers (up to 100). These coalesce to produce larger ulcers with irregular margins. They usually occur on the non-keratinized mucosa, but any part of the oral mucosa may be affected. The ulcers can take up to two weeks to heal (without scarring) and later recur.

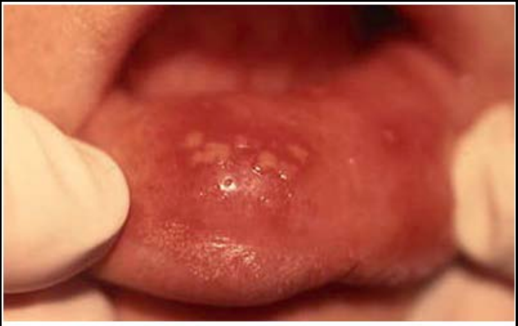

Fig 4. Herpetiform ulceration on the lower lip mucosa.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Some patients have ulceration which is intermediate between minor and major RAU, sometimes termed severe minor RAU. Although these ulcers commonly develop in childhood, some patients develop them later in life.
All forms of aphthous ulceration produce significant discomfort and patients with severe minor aphthae, major aphthae or herpetiform ulceration may have difficulty eating and talking.

Three forms of aphthous ulcers are recognised:

Reference: TG

Minor aphthous ulcers	Major aphthous ulceration	Herpetiform aphthous ulceration
• most common form • presents as smaller lesions (usually 2 to 4 mm in diameter) • occur a few at a time • heal within 7 to 10 days.	• less common form • presents as larger lesions (10 mm or more in diameter) • can persist for up to 6 weeks • (and occasionally months) • heal with submucosal scarring.	• rare • presents as recurrent crops of non-vesicular small ulcers (1 to 2 mm in diameter) that coalesce to form larger ulcers • heal within 1 to 2 weeks not caused by the herpes virus, so do not have a cluster pattern.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

TABLE 19.1 Features of Ulcers

Feature	Details
Site of ulcer	Recurrent aphthous stomatitis (RAS): Typically nonkeratinized mucosa. Erythema multiforme: Vermilion border of lip, buccal and labial mucosa. Traumatic ulceration: Usually recurs at the same site, often close to a sharp tooth. Crohn's disease: Typically affects buccal sulcus.
Size of ulcer	Minor RAS: 3–8 mm in diameter. Major RAS: greater than 1 cm in diameter (up to 3–4 cm). Herpetiform RAS: 0.2–3 mm in diameter.
Duration of each ulcer	Minor RAS: 7–10 days. Major RAS: May take weeks to months. Ulcers heal with scarring. Herpetiform RAS: 7–10 days. Erythema multiforme: Variable (10–21 days).
Number of ulcers	Minor RAS: Single ulcers or small crops of 2–5 ulcers Major RAS: One or two ulcers at a time. Herpetiform RAS: 30–100 ulcers at a time, which can coalesce such that it becomes difficult to count individual ulcers.
Frequency of attacks	RAS: Variable frequency; some patients may have ulcers continuously, whilst others experience ulcers just once per year. Erythema multiforme: Ulcers may recur at 6–8 week intervals in severe cases; other patients experience one-two attacks per year. Nutritional deficiencies/inflammatory conditions: Patients may have continuous ulceration.
Shape of ulcer	RAS: Usually round/oval and sharply defined; may become more irregular with healing. Herpetiform ulcers coalesce to form irregular shapes. Erythema multiforme: Irregular and ragged, merging with inflamed surrounding mucosa. Those on the lips are often covered by bloody fibrin sloughs.
Are the ulcers preceded by vesicles?	The presence of vesicles indicates possible viral infection or immunobullous disease. This fact may be helpful in the differential diagnosis of herpetiform ulcers, which resemble viral ulcers but are not preceded by vesicles.
Age of onset	RAS: Usually before or around adolescence. Erythema multiforme/inflammatory bowel disease: Typically second or third decade. Immunobullous disease: Typically, fourth decade onwards.
Family history	RAS: May have family history of ulceration. Coeliac disease/inflammatory bowel disease: May have positive family history. Erythema multiforme: Typically no family history. Traumatic ulceration: No family history.
Exacerbating or relieving factors	Ulcers can develop at a site of minor trauma in both RAS and immunobullous disease. Stress and menstruation can precipitate attacks of RAS. Erythema multiforme may be triggered by a drug, viral or other infection, although often no trigger is identified.

Reference: Odell

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

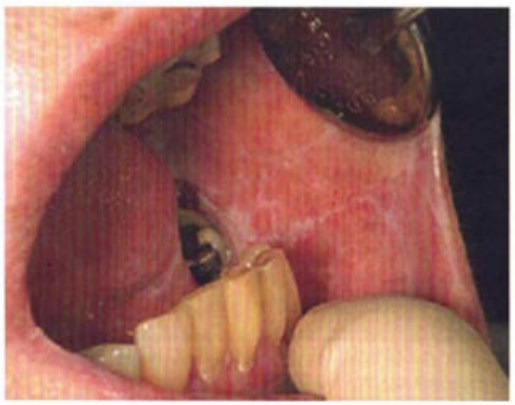
These conditions largely comprise lichen planus, mucous membrane pemphigoid, pemphigus vulgaris and erythema multiforme, although less common conditions such as dermatitis herpetiformis also affect the mucosa and may present clinically as recurrent oral ulceration.

Oral lichen planus

Lichen planus is an uncommon idiopathic immune-mediated condition that can affect the skin, hair, nails, and oral and genital mucosae.

Oral lichen planus typically occurs on the buccal mucosa, tongue and gingivae. In the nonerosive form of the disease, the lesions consist of a characteristic reticular pattern of white striations or plaques.

Photo 8. Oral lichen planus of the left buccal mucosa showing characteristic white striations



Erosive oral lichen planus presents as erythematous, ulcerated or eroded areas of mucosa, which are often painful. Symptoms include stinging or burning, especially with spicy or acidic food.

Oral lichen planus is associated with an increased risk of oral squamous cell carcinoma.

Refer patients with suspected oral lichen planus to a specialist for biopsy, definitive diagnosis and management.
Differential diagnosis should exclude oral lichenoid lesions.
If lichen planus occurs on the gingival tissues, management includes improving oral hygiene and periodontal health.
Patients with oral lichen planus require ongoing review by an oral medicine specialist because of the chronic nature of the condition and the potential for malignant transformation.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Oral lichen planus

biopsy-proven oral lichen planus becomes symptomatic, treat with:

- **betamethasone dipropionate 0.05% cream or ointment topically to the lesions, twice daily after meals, until symptoms resolve.**
- **Advise patients to stop using topical corticosteroids once symptoms have resolved.**
- **If the patient's symptoms have not improved after 3 weeks of topical corticosteroids, the symptoms change or the appearance or texture of the lesion changes, advise patients to return to their treating specialist.**

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

There are six recognized oral presentations of lichen planus:

- **reticular**
- **papular**
- **plaque-form**
- **atrophic**
- **ulcerative(erosive) and rare**
- **bullous form**

These latter three forms can be associated with significant discomfort requiring either topical and/or systemic immunosuppressive therapy.

The cause(s) of the various oral lichenoid lesions, ranging from idiopathic oral lichen planus (OLP) to the "contact" lesion, is not understood, but all the lesions are characterized histologically by a typical "lichenoid tissue reaction" featuring a band-like lymphohistiocytic infiltrate within the lamina propria and liquefaction degeneration of the basal keratinocytes. These reactions may be the result of several diverse possible triggers, but all culminate in a common pathologic process, that of T-lymphocyte directed, immune-mediated, damage to the oral epithelial basal cells.

OLP most frequently presents in women, by a ratio of approximately 3:1 to 3:2 compared with men, aged 40 years and above.

Mucosal lesions are usually multiple and almost always have a bilateral, symmetrical distribution. They commonly take the form of minute white papules that gradually enlarge and coalesce to form either a reticular, annular, or plaque-like pattern. A characteristic feature is the presence of slender white lines (Wickham's striae) radiating from the papules. In the reticular form, there is a lace-like network of slightly raised white lines, often interspersed with papules or rings. The plaque- like form may be difficult to distinguish from leucoplakia.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Oral lichen planus

Involvement of the gingivae is described clinically as desquamative gingivitis, but is not unique to OLP and may feature in the presentation of other oral dermatoses, especially pemphigoid and pemphigus.

Fig 6. Lichen planus presenting as a desquamative gingivitis.

Mucous membrane pemphigoid

This autoimmune disease is uncommon and is often limited to the oral mucosa but may also affect other mucosal surfaces including the conjunctiva to produce scarring (and sometimes blindness), hence the term cicatricial pemphigoid.

Clinical features of mucous membrane pemphigoid:
It is more common in older patients, with some evidence of a female predilection. The vesicles may sometimes present as blood blisters. Although the vesicles are more robust than in pemphigus vulgaris, they tend to rupture within 24 hours to produce ulceration (Fig 7), which may heal with scarring.

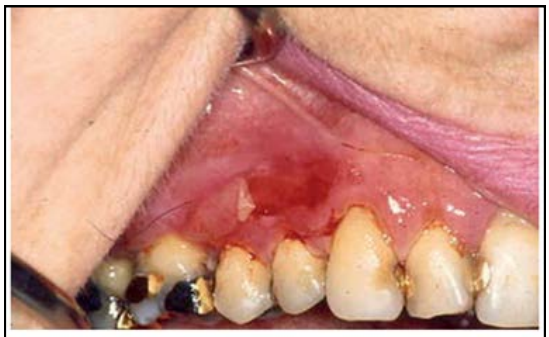
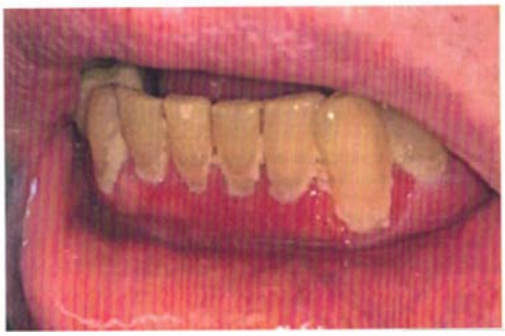


Fig 7. A collapsed vesicle on the maxillary alveolar mucosa in a patient with mucous membrane pemphigoid. This has recently ruptured but will soon become an ulcer covered by a fibrin slough.

Photo 16. Mucous membrane pemphigoid affecting the mandibular gingivae



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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Mucous membrane pemphigoid

Mucous membrane pemphigoid is an uncommon autoimmune vesiculobullous disorder that affects stratified squamous epithelium. It occurs predominantly on the gingivae and palate. Mucous membrane pemphigoid presents as large, painful and persistent erosions, and is characterised by subepithelial splitting, with bulla or vesicle formation. The lesions heal with variable amounts of scarring. Differential diagnosis includes pemphigus vulgaris.

Refer patients with suspected mucous membrane pemphigoid to an appropriate specialist for biopsy and definitive diagnosis. Management usually requires long-term use of immunosuppressive therapy. Ophthalmologist review is necessary because there is a risk of blindness with mucous membrane pemphigoid.

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*

Immune-mediated sub-epithelial blistering diseases characterized by vesicles or bullae that break down to leave ragged ulcers that affect epithelial surfaces, particularly the mouth.

Historically, pemphigoid was broadly subdivided into only two main conditions:

- **"bullous pemphigoid" that predominantly affects the skin, and rarely, mucosa;**
- **"benign mucous membrane pemphigoid" also known as cicatricial pemphigoid,**

but now more simply as "mucous membrane pemphigoid" (MMP), that conversely, overwhelmingly affects mucosa and infrequently the skin.

Occasional drug-induced MMP has been reported, associated with penicillamine (D-Penamine), a heavy-metal chelator used in rheumatoid arthritis and the loop diuretic agent, frusemide (Lasix, Urex)

MMP is predominantly a disease of women, with a mean age at onset of 51–62 years. 32 Children are rarely affected.

The most common areas of involvement are the oral cavity (85%) and conjunctivae (64%).32 The oral mucosa is often the initial site of MMP lesions. The term "oral mucous membrane pemphigoid" (OMMP) is often used when MMP is limited to the oral cavity with no other mucosal involvement and "ocular cicatricial pemphigoid" (OCP) is used when MMP is limited to the conjunctivae.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Oral mucosal lesions

Patients present with bleeding, pain, dysphagia or desquamation of the oral mucosa. Vesicles or bullae may occur anywhere on the oral mucosa and there may be a positive ''Nikolsky sign'', where firm sliding pressure with a finger separates normal-appearing epithelium from the underlying lamina propria, resulting in the immediate formation of a vesicle or erosion.

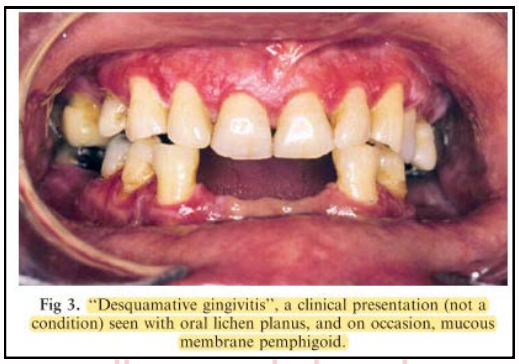

Fig 3. "Desquamative gingivitis", a clinical presentation (not a condition) seen with oral lichen planus, and on occasion, mucous membrane pemphigoid.

Reference: Odell

Main Causes of Desquamative Gingivitis

- Lichen planus
- Mucous membrane pemphigoid
- Pemphigus vulgaris

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

Ocular lesions

Ocular manifestations have been reported to occur in 3 to 48% of patients with oral lesions. Ocular involvement usually begins as chronic conjunctivitis with symptoms of burning, irritation, photophobia ich excess tearing.

Special investigations

- Biopsy (histopathological and DIF investigations)

Definitive diagnosis is based on biopsy of perilesional tissue with histological and direct immunofluorescence (DIF) examination.

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7) Oral dermatoses

Pemphigus vulgaris

This is a relatively uncommon autoimmune disease, reported to have a greater prevalence in Ashkenazi Jews. In a significant number of cases, oral mucosal lesions are the first presentation of the disease.

Clinical features of pemphigus vulgaris:

However, patients are often unaware of blistering because of the rapid breakdown to form ulcers (Fig 8).

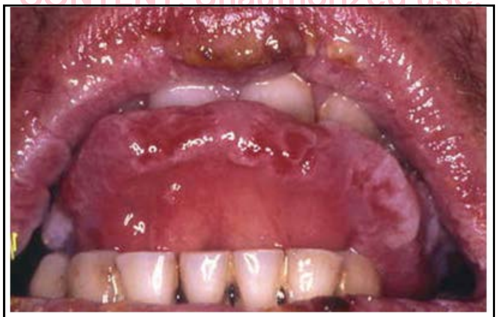

Fig 8. Ulceration of the tongue mucosa in a patient with pemphigus vulgaris

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*)

It is a group of autoimmune diseases characterized by intra-epithelial blistering, resulting in superficial vesicles or bullae that easily rupture, resulting in ulceration of mucosal and/or cutaneous sites.

Pemphigus vulgaris (PV) is the most common and clinically the most aggressive variant, being associated with significant morbidity and mortality, composing 70% of all reported cases. Pemphigus vulgaris commonly and initially affects the oral mucosa and then the skin. Other mucosal sites may also be involved, including the mucosa of the conjunctivae, nose, oesophagus, pharynx and larynx, and genitalia.

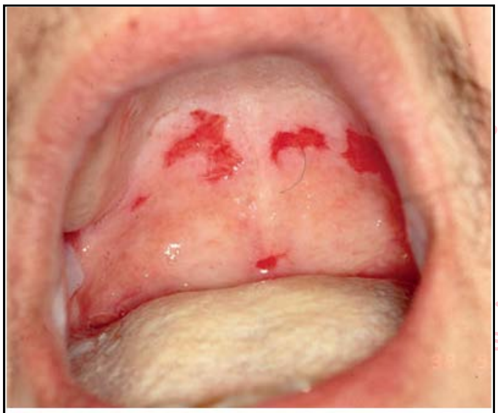

Fig 4. Pemphigus vulgaris – typical oral presentation with shallow ulceration of the soft palate.

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Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Pemphigus vulgaris

Some drugs have been reported to induce PV, including the anti-mycobacterial antibiotic, rifampicin. More recently, associations have been reported with penicillamine as well as the anti-hypertensive ACE (angiotensin-converting enzyme) inhibitor, captopril, and other thiol-containing compounds.

Due to their fragile nature, being intra-epithelial (in contrast to the sub-epithelial nature of the blisters seen in pemphigoid), intact blisters are uncommon in PV, rupturing quickly to produce painful erosions. Patients can be Nikolsky sign positive on examination of the mucosa and the skin.

Special investigations

Clinical suspicion that a patient has one of the vesiculobullous diseases warrants biopsy of perilesional tissue, with histological and DIF examination being essential in establishing the diagnosis.

Erythema multiforme

The condition is seen most commonly in adolescents and young adults and may be drug-induced or associated with an infection, commonly Herpes simplex or Mycoplasma pneumoniae.

Clinical features of erythema multiforme:

The lips are often swollen, ulcerated and crusted with blood – this is regarded as a sine qua non for diagnosis by some people (Fig 9). Stevens-Johnson syndrome is a more severe and generalized form of erythema multiforme, which involves the skin, oral mucosa, conjunctival and genital mucosa. The patient is febrile and unwell and requires hospital admission. Toxic epidermal necrolysis (Lyell syndrome) may represent the most severe end of the spectrum where epithelial necrosis is the predominant feature.

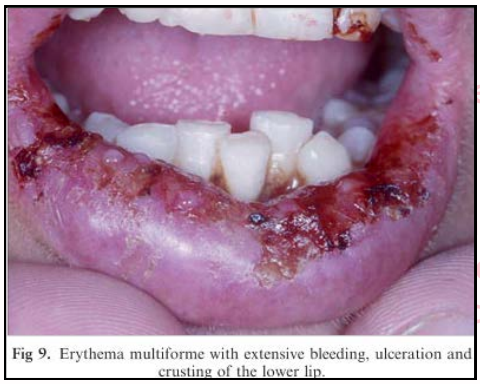

Fig 9. Erythema multiforme with extensive bleeding, ulceration and crusting of the lower lip.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

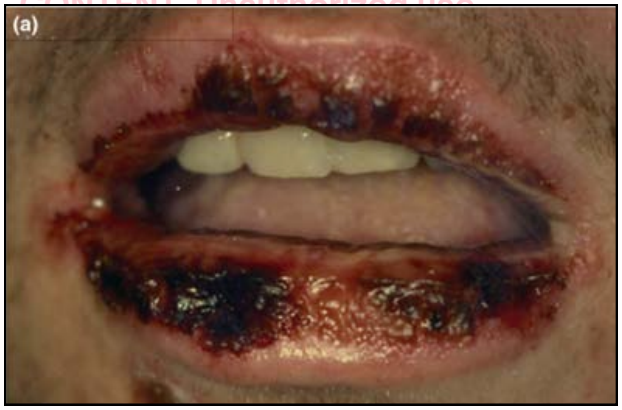
7) Oral dermatoses

Erythema multiforme

Diagnosis of erythema multiforme

Clinical features (particularly crusting and bleeding of the lips) and, in the case of recurrent episodes, associated events (such as recent medication or herpes labialis) will help establish a diagnosis. Histopathological examination with immunofluorescence will help to exclude other vesiculobullous disorders but is rarely diagnostic in itself.

Reference: Oral mucosal diseases: the inflammatory dermatoses M Schifter,* S-C Yeoh,H Coleman,* A Georgiou*



Erythema multiforme (EM) is part of a spectrum of complex, immune-mediated, reactive, muco-cutaneous disorders that often presents with oral, especially, labial mucosal erythema, blistering and ulceration.

In EM major, the oral mucosa is the most commonly involved mucosal site, but any mucosa can be involved, including the epithelial lining of the trachea, bronchi, and gastrointestinal tract, as well as the genitalia. Erythematous macules precede vesiculobullous formation of the mucosa that rupture, leaving irregular superficial painful ulcers, with a marked erythematous halo, or develop into broad areas of white, desquamating, necrotic mucosa. The lip lesions are characteristically, markedly haemorrhagic.

SJS is a more severe disease, characterized by significant oral mucosal involvement, plus conjunctival and/or genital mucosal involvement, and more extensive skin involvement that generally follows several days later. The muco-cutaneous lesions last up to six weeks and reflecting the more significant disease process can heal with scarring, so resulting in laryngeal, conjunctival and vaginal strictures.

TEN presents with the oral lesions typical for EM major, but the skin involvement is extensive, with consequently up to 30% of the body surface denuded, and resembling second degree burns. Indeed, optimal care for patients with TEN is in a specialized burns unit, with attention to the critical issues of fluid loss, electrolyte disturbance and secondary infection.

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7) Oral dermatoses

Erythema multiforme

Special investigations

There are no tests that are pathognomonic for EM and its variants. Biopsy for histopathology and direct immune-fluorescence are only useful in excluding conditions that can present similarly.

Laboratory investigations are directed in determining any recent infection that may have triggered the EM. Human Herpes Viruses 1 and 2 (HSV-1 and 2) are a very common trigger, preceding the presentation of EM major by up to 14 days, reportedly in some 70% of cases. The other commonly reported infectious trigger is Mycoplasma pneumoniae infection. For SJS and TEN, drugs, particularly anticonvulsants such as carbamazepine (Tegretol), phenytoin (Dilantin), phenobarbitol and sodium valproate are frequently implicated.

Treatment

There are no systematic reviews detailing the best treatment for EM major, or its more severe variants. The key aspects of care are firstly, identification and, if practical, treatment of the infectious trigger, or identification (from the history) and withdrawal of the suspected causative drug. Secondly, the use of immune-modulating therapy of which historically the mainstay has been systemic corticosteroid therapy. However, the use of systemic corticosteroids is mired in controversy, with some authors claiming a higher rate of adverse outcomes for patients placed on systemic corticosteroids, particularly in SJS and TEN.

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Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Discoid lupus erythematosus

The first indication of this systemic disease can be the occurrence of oral mucosal lesions, but oral lesions in lupus are relatively uncommon. Lupus erythematosus (LE) is an autoimmune disease in which autoantibodies are directed against nuclear components. Rare cases of drug associated LE have been reported.

Clinical oral features of lupus erythematosus:

LE is most commonly seen in older patients, with a female predilection. When it involves the oral mucosa, it may have an appearance identical to that of oral lichen planus. It may appear as a relatively nondescript ulcer with an irregular outline, sometimes depressed below the level of the surrounding mucosa, and surrounded by erythematous mucosa, perhaps bordered by radiating white striae or white papules.

Management if recurrent oral ulceration

1) Diagnosis

Ensure an accurate diagnosis has been made on the basis of a typical history and clinical appearance.

2) Trauma

Eliminate or control possible sources of mucosal trauma, e.g., cheek or lip biting, overly vigorous brushing of teeth or using a hard toothbrush, sharp teeth/dental prostheses or ingestion of sharp/rough foods.

3) Diet

Consider possible dietary factors and food sensitivities. 3 In occasional patients' certain foods (e.g., oranges, eggs, wheat or dairy products) and other agents (e.g., cosmetics) can initiate or exacerbate RAU. A food diary may be helpful in identification of specific precipitating foods.

4) Hormones

In some female patients, RAU episodes appear to be related to their menstrual cycle. However, the evidence for a hormonal basis is inconsistent. Nevertheless, those patients who do report such an association may benefit from suitable hormone therapy.

5) Psychological factors

Psychological factors may be an important factor as some patients notice that their ulcers become worse in periods of illness, stress or extreme fatigue. Some form of stress management counselling may be considered in some of these cases.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

7) Oral dermatoses

Discoid lupus erythematosus

Management if recurrent oral ulceration

6) Medications

- Many treatments have been advocated for recurrent aphthous ulceration. These may be based upon anti- septics, antibiotics, corticosteroids, immunosuppressants, antirheumatics, anti-inflammatories, hormone therapy, antivirals, colchicine, thalidomide, pentoxifylline, sodium cromoglycate, interferon, hyaluronic acid, helicobacter eradication, zinc, various acids, gastric ulcer treatments, ultrasound, laser, cautery, cryo- therapy, bio adhesives, herbal remedies, homeopathy, vitamins, lactobacillus as well as sundry other management strategies and combinations of various medications. Systemic treatment may be appropriate for more severe and resistant cases. It should be made clear to the patient that the objective of treatment is symptomatic and that the ulcers cannot be ‘‘cured’.
- They reported that chlorhexidine could reduce ulcer severity and/or duration but not incidence, steroids could reduce ulcer duration and may reduce pain. They reported that chlorhexidine could reduce ulcer severity and/or duration but not incidence, steroids could reduce ulcer duration and may reduce pain.
- Lignocaine 2% gel or mouthwash can be used for pain relief. This is especially helpful with extensive ulceration and major aphthous ulceration.
- A corticosteroid mouthwash may be helpful for widespread oral ulceration. This can be made from a 5 mg tablet of prednisolone crushed into 10 mL of warm water (or 1 mL of Redipred or Predmix in 10 mL water) or one dexamethasone 0.5 mg tablet in 10 mL water, the solution washed around the mouth and then expectorated. The immunosuppressant pimecrolimus 1% cream applied to lesions twice daily may be an effective alternative to other topical treatments. Intralesional injections of triamcinolone acetonide 10 mg/mL are used by some clinicians for lesions of lichen planus.
- A short course of systemic corticosteroids may occasionally be necessary in the management of major RAU and oral dermatoses.
- Topical corticosteroids used from the time of the earliest indication of prodromal symptoms provide symptomatic relief and reduce the duration of minor RAU and localized oral dermatoses.

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ORAL RECURRENT ULCERATIONS

Reference: The patient with recurrent oral ulceration

Conclusions

It is essential to review the patient to assess their progress and response to any treatment instituted. It is important that patients are aware of the limitations of treatment. For example, patients with RAU need to be advised (and sometimes reminded) that a cure is not possible but that treatment is intended to reduce symptoms. This caution is also appropriate for lichen planus and the vesiculobullous disorders. Depending upon the response to treatment, alternatives could be trialled.

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OSTEORADIONECROSIS OF THE JAW (ORNJ)

Reference: Predictive factors for osteoradionecrosis of the jaws: A retrospective study

Survival rates of patients with head and neck squamous cell carcinoma (HNSCC) have improved with the combined use of surgery and radiotherapy with/without chemotherapy. However, there remains significant morbidity associated with this treatment.

Osteoradionecrosis of the jaw (ORNJ) is considered as one of the more serious long-term complications of radio- therapy in the craniofacial region. The most widely used definition of ORNJ is based on clinical findings; where in the absence of tumour recurrence, irradiated bone becomes devitalized and exposed without healing for a period of 3-6 months.

The presentation of ORNJ is variable both in patient- reported symptoms and in clinical appearance. Chronic pain, nonhealing wounds, orocutaneous fistulas, and pathologic fractures have been reported. Patients with small initial lesions may remain asymptomatic, which could explain why ORNJ is often not detected earlier. Although improvements in management have occurred, the condition remains difficult to treat causing considerable morbidity and costs.

Identification of predictive features would determine patients at greater risk of developing ORNJ. Appropriate measures can then be put in place both to reduce the risk of developing ORNJ and expedite management if it were to occur.

For this study, ORNJ was defined as exposure of irradiated bone that had become nonvital and failed to heal over a period of at least 3 months and occurs in the absence of other diseases in a region that received radiotherapy.

Extraction sockets take a considerable amount of time to heal, hence, tooth removal in common with any other form of surgical manipulation of bone is associated with higher risk of developing ORNJ. This is particularly the case when DAS (dentoalveolar surgery) is performed too close to commencement of radiotherapy or too soon after radiotherapy. However, there remains disagreement whether a safe timing of dental extractions exist.

DAS (dentoalveolar surgery) performed after radiotherapy produced a much higher risk of developing ORNJ than when performed before radiotherapy. The reason that postradiotherapy DAS is associated with a much higher risk of developing ORNJ is possibly because radiotherapy has already significantly altered the underlying supporting structures of the teeth.

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OSTEORADIONECROSIS OF THE JAW (ORNJ)

Reference: Predictive factors for osteoradionecrosis of the jaws: A retrospective study

Most of the ORNJ cases occurred at sites of peri-resective surgery. Jaw surgery has been thought to predispose patients to development of ORNJ. Such surgery may be performed either to remove a tumour or gain surgical access. Any bone damage, surgical or otherwise, that has not completely healed before radiotherapy could predispose the patient to the development of ORNJ.

In the past, it has been the practice for surgical defects created by marginal/segmental procedures to be reconstructed using advancement flaps. This procedure causes undue soft tissue tension over the bone, potentially leading to dehiscence predisposing to development of ORNJ.

Tobacco usage is considered a risk factor for ORNJ due to its detrimental effects on wound healing. This is due to its vasoconstrictive properties and presence of chemicals that traumatize the oral mucosa and contaminate surgical wounds.

Nicotine causes platelet aggregation and vasoconstriction, increasing the risk of microvascular thromboses and decreasing micro-perfusion. Carbon monoxide competitively inhibits the binding of oxygen to haemoglobin, leading to cellular hypoxia. These toxic substances are able to undermine the conditions required for wound healing, exacerbating pre-existing tissue compromise.

Diabetes mellitus type 2 (DM2) has an adverse effect on the cardiovascular and immune systems. DM2 has been proven to predispose patients to peripheral vascular disease and microangiopathy, causing impairment of blood supply that is detrimental to wound healing. The effects of DM2 are further amplified by surgery and radiotherapy. DM2 also impairs the immune system, predisposing the patient to infections by affecting neutrophil chemotaxis and target killing.

Studies reporting the relationship between the risk of developing ORNJ and increasing radiation doses. Mean total radiation dose received by those who developed ORNJ was 65.8 Gy (63.6 Gy), however, there were patients who received doses higher than 66 Gy and did not develop ORNJ. This suggests that total radiation dose alone does not predict absolute risk. Nevertheless, high radiation doses exceeding 65 Gy does play a contributory role in the development of ORNJ.

In our study, chemotherapy by itself was not a significant predictor for the development of ORNJ. There is still some amount of controversy concerning the influence or role of chemotherapy in ORNJ, with some studies reporting a higher incidence of ORNJ when chemotherapy was used in conjunction with radiotherapy, whereas others have found no difference when chemotherapy was included. However, the authors of a recent systematic review concluded that the addition of chemotherapy agents does not seem to increase the risk of patients developing ORNJ.

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OSTEORADIONECROSIS OF THE JAW (ORNJ)

Reference: Predictive factors for osteoradionecrosis of the jaws: A retrospective study

There were 18 cases of ORNJ that occurred at the mylohyoid ridge region (a site with morphologically sharp/prominent bone) without any identifiable traumatic cause. This region is also commonly associated with medication-related osteonecrosis of the jaws (ONJs) as well as lingual mandibular sequestration in healthy individuals.

Each of these cases had radiation doses of 70 Gy or more. Although radiation dosage of 70 Gy or more is believed to cause “spontaneous” ORNJ due to increased rate of cell death brought about by the high radiation dose, not all such dosages led to ORNJ, implying the existence of other contributory factors in the development of the so-called spontaneous ORNJ.

It has been established that ORNJ has a predilection for the mandible over the maxilla. This is thought to be due to differences in the blood supply, anatomy, and morphology between these 2 structures. The mandible is much denser with a higher mineral content than the maxilla and as such absorbs and scatters more radiation.

A major percentage of ORNJ (88.1%) occurred within the first year after radiotherapy. This is consistent other studies about the timing of ORNJ postradiotherapy. Immediately after radiotherapy, irradiated tissues are compromised and still very vulnerable to further insult. Healing of these tissues will take months and, during the early stages of healing, the tissues are more prone to injury. What would be a minor insult to nonirradiated tissue would have more significance in irradiated tissue and should not be considered trivial. The efficiency of wound healing also differs between individuals, being influenced by systemic as well a treatment-related factors.

Before making a diagnosis of ORNJ, it is important to ensure that tumour recurrence/metastasis, medication-related ONJ, and osteomyelitis are ruled out, as these conditions can either mimic ORNJ or occur synchronously. Simple radionecrosis of mucosa can also present with exposed bone. However, with the improved understanding of the pathophysiology of ORNJ as well as availability of medical management there is no basis to justify waiting for 3-6 months to confirm the diagnosis of ORNJ. With that in mind, the following modified clinical definition for ORNJ is proposed:

“Clinical/radiological evidence of nonvital bone, regardless of size, in an irradiated area failing to exhibit any sign of healing for 1 month or more with/without clinical intervention in the absence of osteomyelitis, tumour recurrence, metastasis, and medication-related ONJ. Purely radiologic findings should be supported by clinical and/or histopathological findings.”

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OSTEORADIONECROSIS OF THE JAW (ORNJ)

Reference: Predictive factors for osteoradionecrosis of the jaws: A retrospective study

Although some authors have suggested that ORNJ can be diagnosed solely through radiographic findings suggestive of bone necrosis under intact mucosa, such findings need to be supported by clinical and histopathological findings. This is because many other intraosseous pathologies may confuse the diagnostic picture, and routine imaging at every review visit is impractical. However, any incidental radiologic findings suggestive of bone necrosis should alert the clinician for further clinical assessment followed by a biopsy to rule out malignancy or confirm ORNJ.

A 1-month period was chosen as oral wounds usually show some signs of repair and healing within 2 weeks in a healthy individual, if factors such as trauma, infection, and inflammation are controlled. This duration is expected to be longer in a compromised oral environment postradiotherapy; hence, the 1-month duration. Bone sequestrum that is noticed on a routine review appointment and is asymptomatic may have already been present for an indeterminate period. Thus, waiting for a further 3 months to reassess the situation cannot be considered good clinical practice. Exposed bone can also traumatize adjacent soft tissue, as is frequently the case at the mylohyoid ridge region with the lateroventral surface of the tongue often being traumatized.

Based on our findings, we propose several recommendations to reduce/prevent the occurrence of ORNJ, specifically, that

- the duration of bone exposure before diagnosis of ORNJ be shortened to 1 month;
- smoking cessation be strongly advocated
- educating patients on the value of glycaemic control in DM2;
- pre-radiotherapy dental assessment by a suitably trained dental specialist be performed at least 1 month before radiotherapy so that all necessary DAS can be completed at least 2-3 weeks before radiotherapy;
- patients who received DAS should be assessed by the dental team 2 weeks after DAS to ensure that the surgical site is healing well;
- these postradiotherapy patients should be followed up for life by a suitably trained dental team.

Reference: Clinical utility of hyperbaric oxygen therapy in dentistry

Hyperbaric oxygen therapy (HBOT) facilitates the transfer of oxygen to the tissues of the human body. By doing so, it promotes healing of wounds and minimizes the typical recovery time for patients. At this juncture, strictly within dental medicine, HBOT indicates the distribution of comprehensive oxygen at pressures greater than 1.4 atmosphere absolute (ATA), often in a series of treatments. This treatment requires the patient to stand within a hyperbaric chamber with pressure greater than ambient. It has many uses such as patient care, and wound care within standard medicine and dental medicine.

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Uses in Dentistry

- While it has various uses specific to dental medicine, HBOT is most often used for prevention of complications during radiation therapy.
- It is known to effectively increase tissue oxygenation and, moreover, expedite the healing wounds.

Table 1: Dental cases treated with hyperbaric oxygen therapy

Clinical condition	Number of cases	Mean time since radiotherapy	Time range	Number of hyperbaric oxygen therapy sessions (mean)	Time range
Osteoradionecrosis	3	6.7 months	3 to 10 months	34	12 to 38 months
Post-radiotherapy cases	3	54 months	8 to 60 months	11	10 to 14 months
Mandibular osteomyelitis	5	N/A	N/A	28	17 to 40 months

Osteoradionecrosis

Osteoradionecrosis of the jaw is commonly acknowledged as death of the jaw bone and bone within the head and neck region because of the decreased oxygen tension-hypotension hypocellularity and hypovascularity.

Osteoradionecrosis is a nonhealing, nonseptic lesion of the bone in which bone volume and density cannot be maintained by the hypocellular, hypo vascular, hypoxic tissue, which cannot adequately meet its metabolic demands.As the soft tissue decays, the bone begins to become exposed. Saliva and other foreign entities within the oral cavity will prompt cross-contamination, thus leading to significance in infection and further complications. Symptoms of osteoradionecrosis include mouth pain, jaw swelling, poor smelling breath, mouth sores, and difficulty opening the jaw.

Osteoradionecrosis often yields an array of serious side effects. These include facial deformity, pain, pathological fracture, sequestration of devitalized bone, and orocutaneous fistulas. Dysgeusia, paraesthesia, bone exposure, gingival ulceration, tooth fracture, pathologic mandibular fracture, xerostomia, and orocutaneous fistula are common within the oral cavity. All of the following induce extreme irritation and pain, and often lead to more severe complications if not treated in a timely manner.

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OSTEORADIONECROSIS OF THE JAW (ORNJ)

Reference: Predictive factors for osteoradionecrosis of the jaws: A retrospective study

Osteoradionecrosis

HBOT increases the oxygen tension in the region and promotes angiogenesis, the development of new blood cells.

↓

The development of new blood cells directly leads to wound healing.

↓

When a wound is beginning to heal, the angiogenic capillaries sprout and invade the fibrin-rich would clot.

↓

the clot is invaded, the alpha v beta 3 receptors are targeted, which are necessary for wound healing.

Osteoradionecrosis is often diagnosed in three different stages:

- Spontaneous
- from trauma preceding radiotherapy
- and due to trauma post-radiotherapy

In the spontaneous stage, the high dose of radiation during treatment can directly lead to death of the bone cells, destroying the bone. Radiation of > 700 cGy is considered enough to immediately kill the bone cells after one full year.

In trauma preceding radiotherapy, if radiation therapy is within 21 days of a tooth extraction, often times this leads to radiotherapy for oral cancers or mandibulotomy.

Trauma post-radiotherapy is very common, especially after dental extractions. Tooth extractions normally follow severe infection and inflammation of gum tissue surrounding the tooth. As the radiation affects progress, the tissue cannot handle the increase in vascular nutrition and oxygen demands that are required for healing. If the tissue cannot heal, osteoradionecrosis will develop.

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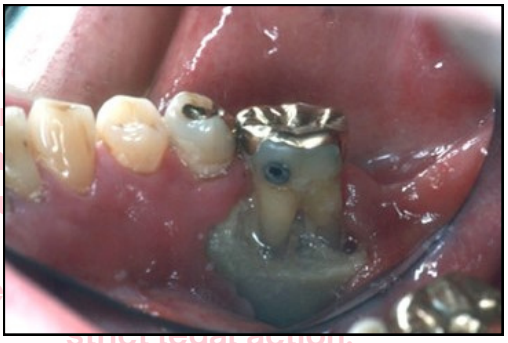
Osteoradionecrosis

Stage 1 Prior to Radiotherapy	Stage 2 During Ratiation	Stage 3 Post Radiation
• All the needful treatment should be done. • Restoration, Extraction, everything should be completed. • Fluoride application.	• Extraction should be absolutely contraindicated • Minor restorations can be done • Only I case of emergency pulp therapy should be done.	• Extractions can be done. But always try to avoid as much as possible. • Extraction should be performed under: a) Hypebaric O2 Rx with b) Primary closure with suture and with c) Antibiotic coverage

Head and neck radiotherapy

Reference: TG

- Patients who require head and neck radiotherapy should be reviewed by a dentist experienced in cancer management, as part of a multidisciplinary team.
- Radiotherapy can cause oral pain, mucositis, reduced salivary flow, oral infection, trismus and altered taste. Reduced salivary flow can increase the risk of periodontal disease and dental caries.
- Good oral hygiene can reduce the incidence, severity and duration of adverse effects associated with radiotherapy. Ensure optimal oral health; if possible, any necessary dental treatment should be completed before starting radiotherapy.
- If extractions are performed, allow adequate time for wound healing (usually 10 days to 3 weeks) before starting radiotherapy, if possible.
- Patients who have had head and neck radiotherapy are at increased risk of osteoradionecrosis. Encourage regular dental review and seek advice from the patient's multidisciplinary team before performing tooth extractions that are within the field of radiotherapy.
- If possible, choose conservative dental treatment options (e.g. periodontal treatment, restorations, endodontic treatment [root canal], fluoride application). Neutral fluoride products are better tolerated than acidulated products.
- Management of osteoradionecrosis is difficult and requires specialist management. Do not extract teeth from a patient who has had head and neck radiotherapy without consulting the patient's multidisciplinary team.



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