A GUIDE TO COMMON ORAL LESIONS FOR DENTAL OFFICERS

Oral Health Programme
Ministry of Health Malaysia
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FOREWORD

It gives me great pleasure to introduce this illustrated book comprising a wide range of common mucosal lesions seen in Ministry of Health's facilities. It is indeed a commendable effort by these authors to compile the first Oral Medicine atlas by Dental Specialists in the Ministry of Health.

In recent years, the field of Oral Pathology and Oral Medicine in Malaysia has shown positive growth not only in terms of the number of centres and specialists but also the range of oral pathology and oral medicine cases detected. The multitude of oral mucosal diseases encountered and the importance of recognising variable clinical presentation must be highlighted when formulating the correct diagnosis. Ensuring the best possible management requires good diagnostic skills and hence the necessity to understand the correct clinical criteria for diagnosis is paramount.

This atlas was written to serve as a guide for dental officers and trainee specialists in recognising mucosal lesions encountered in daily practice. I am certain that clinical criteria and differential diagnoses listed would be particularly useful to assist our dental officers in recognizing the differences between lesions with similar features. The Oral Health Programme of the Ministry of Health has long recognised the need to standardise and train dental officers to detect potentially malignant oral mucosal lesions. This atlas not only complements but further strengthens our oral cancer screening programme in line with our efforts to improve the detection of oral cancer in Malaysia.

I truly hope that this book would be beneficial to all our practitioners in providing the best service to the public.

Principal Director of Oral Health
Ministry of Health Malaysia

PREFACE

The aim of writing this book is to provide a pictorial guide to commonly encountered oral mucosal lesions seen among Malaysians. Recognising and identifying oral mucosal lesions is an important aspect of daily clinical oral examinations. The possibility of presence of potentially malignant lesions in the oral cavity makes it crucial that these lesions are recognised and treated early as the oral cavity can be easily examined. Sometimes lesions of the oral cavity is the first clinical presentation of a more serious systemic disease such as HIV/AIDS, as such diagnosis is very important.

Systematic oral mucosal examination are often overlooked in a busy clinic. The first section emphasizes the importance of carrying out systematic examination due to the reasons as stated above. A pictorial guide on how to carry out mucosal examination is clearly presented here.

The second section of this book concentrates on the various lesions and they are grouped according to the clinical presentation to help officers in making a more logical differential diagnosis. The final section provides a quick guide in managing cases especially oral potentially malignant lesions.

We hope this book would be a useful tool for dental officers, specialty trainees and others in their daily practice especially in formulating a diagnosis of oral mucosal lesions. This book could also serve as a guide for officers involved in oral cancer screening programme.

The authors would like to acknowledge the support given by our Principal Director of Oral Health, Dr Nomah binti Taharim, Head of Specialty (Oral Pathology & Oral Medicine), Dr Lau Shin Hin and immediate past Principal Director of Oral Health, Datuk Dr Noor Aliyah binti Ismail. We would also like extend our acknowledgement to all Oral Pathology & Oral Medicine specialists for their advice, Dr Ahmad Khairuddin and dental officers from Dental Specialist Clinic, Sarawak General Hospital for their contribution rendered during the preparation of this atlas.

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1.1 Systematic Oral Examination

A systematic approach to oral examination is very important to ensure that no oral site is missed during oral examination. Any removable prostheses or appliances should be removed prior to examination. A good light source is essential for complete visualisation of the areas. Any food debris should be wiped away or rinsed off prior to examination. Examination should start from the lips, followed by labial mucosa, buccal mucosa, floor of the mouth and ventral tongue, dorsal and lateral surface of tongue, hard and soft palates, gingivae and teeth **(Figure 1a-j)**.

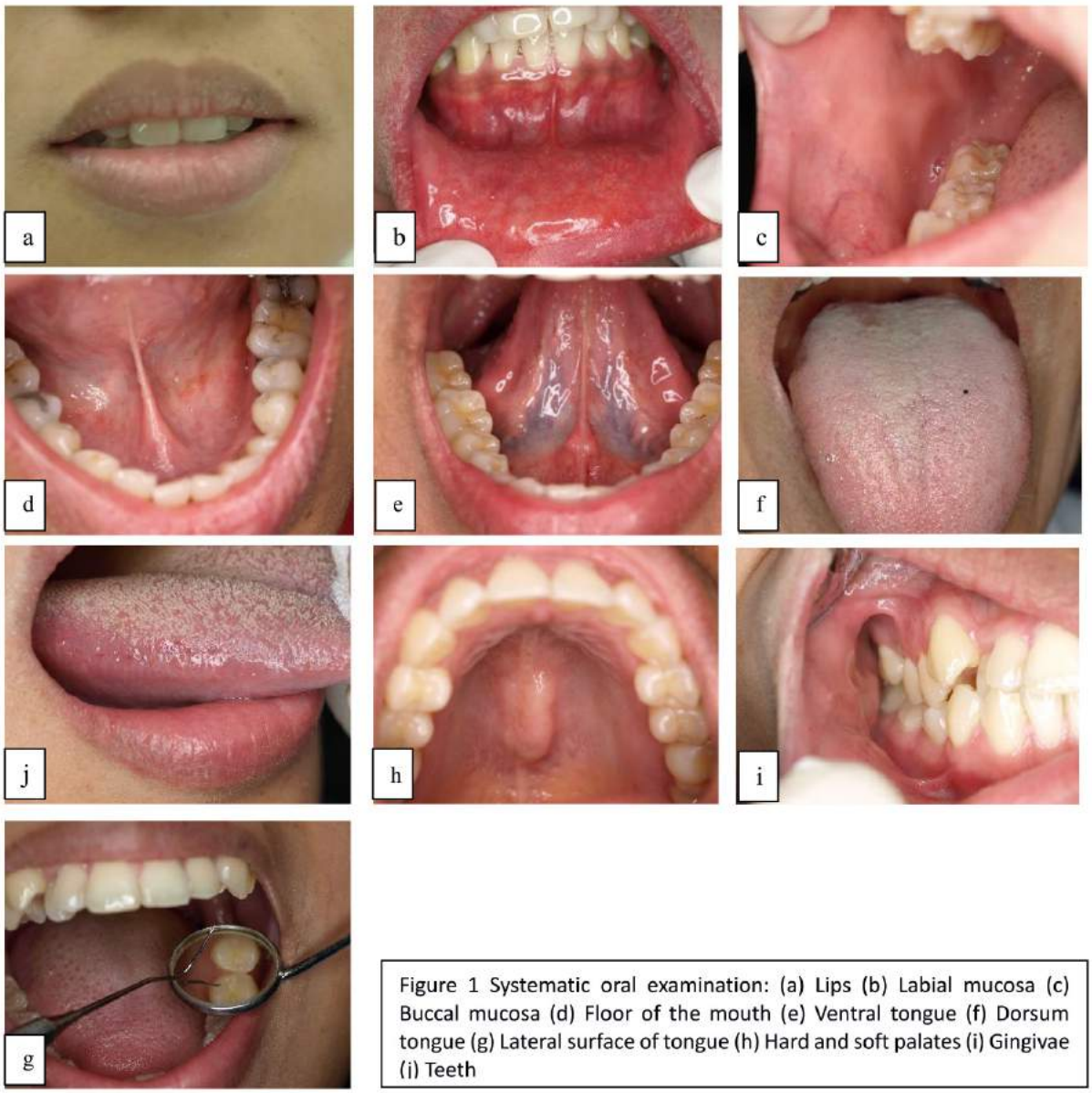


Figure 1 Systematic oral examination: (a) Lips (b) Labial mucosa (c) Buccal mucosa (d) Floor of the mouth (e) Ventral tongue (f) Dorsum tongue (g) Lateral surface of tongue (h) Hard and soft palates (i) Gingivae (j) Teeth

2.0 SECTION 2

2.1 EROSIVE & ULCERATIVE LESIONS

An ulcer is a break in the mucous membrane with loss of surface tissue. It involves damage to both the epithelium and lamina propria with the ulcer base located at a deep level in the submucosa or even within muscle or periosteum.

An erosion is a superficial breach of the epithelium, with little damage to the underlying lamina propria. Only the superficial epithelial cells of the epithelium are lost and the lesion can reach the depth of the basement membrane.

The most common causes of ulcerative oral lesions are trauma and aphthous stomatitis. An important differential diagnosis for oral ulcer especially non-healing ulcers is oral cancer.

(a) Traumatic Ulcers

Description: As the oral mucosa is thinner than skin, it is easily damaged by local trauma. The shape and size of traumatic ulcers **(Figure 2.1a-b)** are variable. Ulcers present as localized single ulcer or sometimes multiple ulcers depending on the causative factor. They are usually painful and of short duration. However, the ulcers can become chronic if the irritation is persistent.

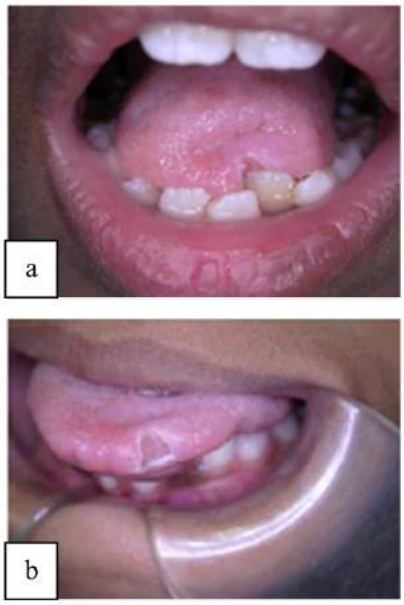


Figure 2.1 Traumatic ulcers

Aetiology: Oral ulcers can occur secondary to physical, thermal, chemical/ electrical or irradiation trauma. The most common cause of mechanical trauma is accidental biting due to sharp or non-aligned tooth. Irritation from ill-fitting dentures can also cause ulcers.

Treatment: Treatment is to remove the causative factor if known. Use of topical agents such as chlorhexidine or benzydamine hydrochloride mouthwashes and hyaluronic acid such as Gengigel® may relief the pain.

Prognosis: Ulcers should heal if the cause is removed. An ulcer which does not heal within 2-3 weeks should be biopsied to rule out malignancy.

Differential Diagnosis: Traumatic ulcers must be differentiated from recurrent aphthous stomatitis, squamous cell carcinoma, infections (viral, bacterial & fungal) and other causes of ulcerative mucosal lesions.

(b) Recurrent Aphthous Stomatitis (RAS)

Description: It is one of the commonest oral diseases and cause of oral ulcers. They appear as painful single or multiple ulcers. Size range from small lesions (≤5mm) known as minor aphthae **(Figure 2.2)** to larger lesions (≥5mm) known as major aphthae. Herpetiform aphthae **(Figure 2.3)** are less common and present as multiple pinpoint ulcers which coalesce to form large irregular areas. There is positive history of recurrent episodes. Any age group can be affected, ranging from the very young to old with young adults and females more commonly affected. Ulcer begins as red macule which soon ulcerates to produce round or oval lesion with yellowish white centre and erythematous peripheral halo.

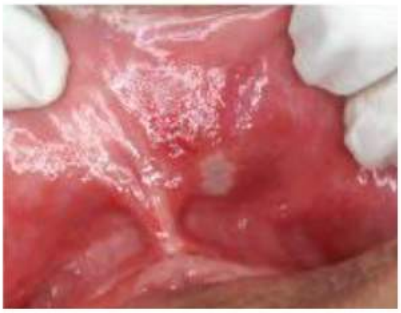


Figure. 2.2 Minor aphthae

Aetiology: Generally unknown and may not be due to a single condition, but rather may be manifestation of a group of disorders. A variety of predisposing factors have been implicated in RAS. In one-third of cases, there may be genetic predisposition as shown by positive family history. Stress and cessation of smoking may exacerbate RAS. Haematinic deficiencies (iron, folic acid or vitamin B12) may be relevant in some patients. In some women, RAS may be associated with fall in oestrogen level during menstrual cycle. Food allergies occasionally underlies RAS. Sodium lauryl sulphate (SLS), a detergent common in toothpaste may act as an irritant to produce oral ulcers.

Figure 2.3 Herpetiform aphthae

Treatment: Correct any predisposing factors if any. Use of topical analgesics or anti-inflammatory agent such as benzydamine hydrochloride may relief pain. Topical corticosteroids can often control RAS and is particularly suitable for frequently recurring or severe ulcerations. Mild agents such as triamcinolone acetonide may be effective but a higher potency corticosteroid may be required in severe cases. The use of topical hyaluronic acid such as Gengigel® may help in pain control and healing. Maintenance of good oral hygiene and use of SLS-free toothpaste maybe helpful to reduce the ulcer duration.

Prognosis: Ulcers are rarely cured. Remission and control of condition may be achieved through the various treatment strategies. Minor RAS generally heals in 7-10 days. Herpetiform and major RAS may take longer time to heal. Major RAS may heal with scarring.

Differential Diagnosis: RAS must be differentiated from ulceration due to primary herpes simplex infection. Recurrent intraoral herpes commonly occurs on the hard palate which is rarely involved in RAS. RAS are indistinguishable with aphthous-like ulcers associated with a range of systemic conditions such as Behcet's syndrome, Reiter's syndrome, Crohn's disease and coeliac disease.

(c) Herpes Simplex Virus (HSV) Infections

Description: In the oral cavity, HSV infection manifest as primary herpetic stomatitis **(Figure 2.4)** and secondary (recurrent) herpes labialis (cold sores) or intraoral ulcers. Primary herpetic infection is quite often sub-clinical. The virus is contracted early in life and seen mainly in children and adolescent. However, it is increasingly seen in older patients and this may be attributed to HSV-2 transmitted sexually. Transmission is through contact with infected bodily fluid such as saliva. There is generalized mucosal involvement in primary herpetic infection with initial presentation of red, swollen gingiva followed by vesicles which breaks down to form painful ulcers elsewhere in the mouth. Fever and/or malaise and lymphadenopathy often accompanies the oral symptoms. Secondary herpes labialis **(Figure 2.5)** present as localised crops of vesicles at the vermillion border. Lesions may be preceded by tingling or burning pain. Occasionally recurrent herpes can present as localised multiple pinpoint intraoral ulcers on the palate. Diagnosis is largely based on clinical presentation.

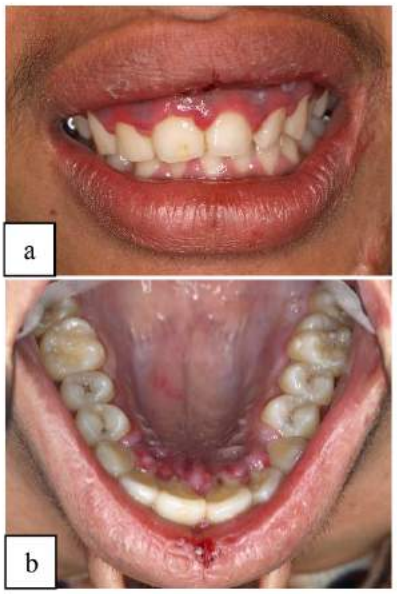


Figure 2.4 Primary Herpetic Stomatitis (a) red swollen gingiva (b) Ulcer on gingiva and palate

Aetiology: Oral and oropharyngeal herpetic infections are mainly cause by HSV-1 virus whereas HSV-2 mainly affects the genital or anal region. Recurrent infections occur when the virus is reactivated in individuals who have had contracted HSV-1 or HSV-2 infections.

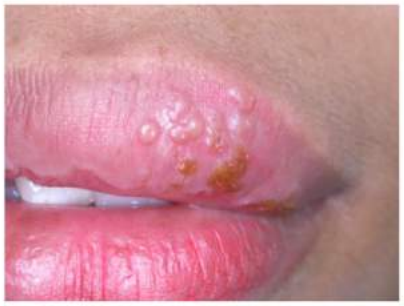


Figure 2.5 Herpes Labialis

Treatment: Most individuals will have spontaneous remission within 7- 10 days. Treatment is aimed to reduce fever and pain. Antipyretics/ analgesics such as paracetamol may relieve fever and pain. Adequate fluid intake is also important especially in children. Use of local antiseptic mouthwash such as chlorhexidine may aid resolution of oral intraoral lesions. Use of antiviral drugs have been shown to reduce the duration of the disease. However, antivirals are most effective if started early. Systemic antivirals are useful in neonates, pregnant women and immunocompromised patients.

Prognosis: Although primary infection usually resolves within a week, the virus, however, remains latent thereafter in the ganglion. Recurrent infection may occur if the virus is reactivated. Herpes labialis is the most common form of recurrence and presents as vesicular lesions on the lip. Intraorally, recurrent herpetic infection can present as unilateral small crop of ulcers usually on the palate.

Differential Diagnosis: Primary infection may resemble erythema multiforme, herpangina and hand, foot and mouth disease. Recurrent intraoral herpetic ulcers may mimic herpes zoster and RAS.

(d) Oral Tuberculosis

Description: Tuberculosis, a widespread infectious disease has afflicted the world's population since the earliest centuries. Despite the improvement in public health measures, tuberculosis still remains as a major health concern. Tuberculosis in the oral cavity can occur either as primary or secondary infections. Secondary lesions are more common than primary lesions and are often associated with pulmonary disease **(Figure 2.7)**. Intra-orally, these granulomatous lesions present as red and granular or ulcerated **(Figure 2.6)** with raised margins resembling primary oral carcinoma. Oral lesions can occur on soft tissue and supporting bone, extraction sockets and may also affect the tongue and floor of mouth.

Figure 2.6 Oral tuberculous ulceration

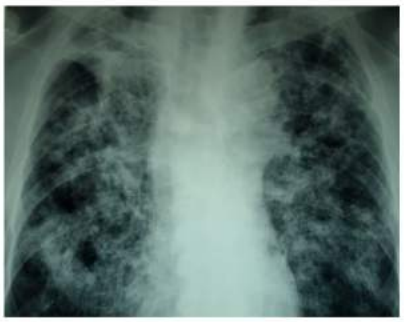


Figure 2.7 Pulmonary tuberculosis

Aetiology: The main causative organism is mycobacterium tuberulosis. The tuberculous bacilli are thought to invade the mucosa through small break in the surface epithelium. Poor oral hygiene, local trauma and irritation are local factors which may facilitate bacillus entry. Self-inoculation through infected sputum, haematogenous or lymphatic spread may cause oral involvement.

Treatment: As oral tuberculous lesions may resemble carcinoma or other mucosal lesions incisional biopsy is required. Presence of caseating granulomas and positive staining to acid-fast bacilli warrants a full work-up for tuberculosis. Other investigations include chest x-ray and/or sputum culture. Urgent referral to physician is recommended.

Prognosis: Dental personnel must be aware that infectious respiratory diseases has transmissible potential in the dental operatory. Adherence to cross-infection control procedures should be strictly followed.

Differential diagnosis: Squamous cell carcinoma, other ulcerative disorders such as major aphthous stomatitis, vesiculobullous disorders.

(e) Erythema Multiforme (EM)

Description: EM is an acute hypersensitivity reaction affecting the mucocutaneous tissues. It is characterized by widespread mouth ulcerations with swollen, bleeding and crusted lips **(Figure 2.8a)**. Oral lesions are often more pronounced in the anterior mouth. Other mucosae and skin **(Figure2.8b-c)** may also be affected. In most cases, only the oral mucosa is affected. Rarely, widespread involvement (Stevens-Johnson syndrome and toxic epidermal necrolysis) may occur and is life threatening. Diagnosis of EM relies mainly on clinical signs.

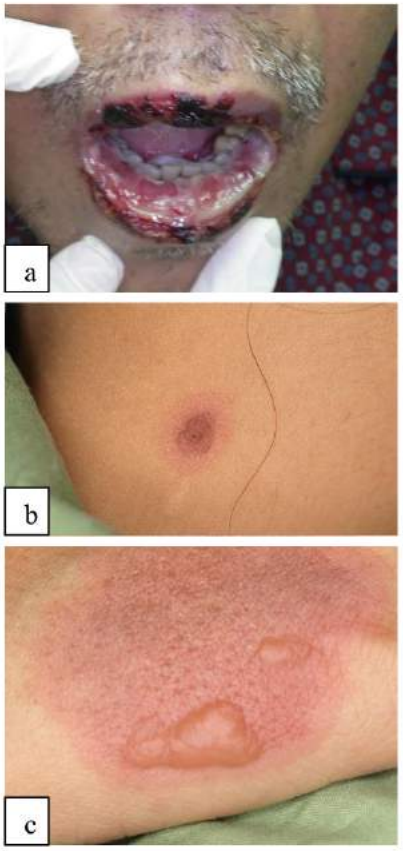


Figure 2.8 Erythema multiforme; (a) Oral ulcers and crusted lip (b) Target lesion on skin (c) Bullae

Aetiology: Herpes simplex virus, drugs especially NSAIDS, anti-convulsants and anti-microbials, food additives and immune conditions such as BCG or hepatitis B immunization have been identified as potential triggers. Most cases of widespread Stevens-Johnson syndrome/ toxic epidermal necrolysis is assumed to be caused by drugs with allopurinol, sulphonamide antibiotics, cephalosporins and carbamazepine being "high risk" triggers.

Treatment: No specific treatment is available but specialist care is warranted. Mild EM may respond to improvement oral hygiene (0.2% chlorhexidine mouthwash) and use of topical steroids although systemic steroids may sometimes be required. Severe forms require urgent referral and hospital admission. Any known triggers should be addressed accordingly.

Prognosis: Recurrences can occur in 25% of cases. For severe EM aggressive supportive care is very important as conditions are they considered medical emergencies.

Differential diagnosis: Vesiculobullous disorders such as pemphigus, mucous membrane pemphigoid.

(f) Oral Cancer

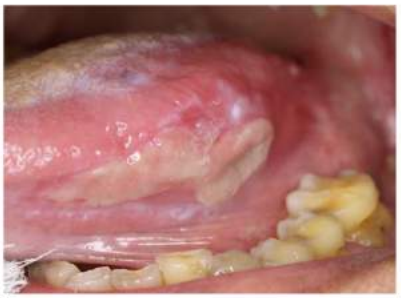


Figure 2.9 Oral Squamous cell carcinoma – ulcer with rolled borders

Description: The most common type of cancer in the oral cavity is Oral Squamous Cell Carcinoma (OSCC). Buccal mucosa and tongue are common sites but OSCC can affect other mucosa such as gum, palate, floor of mouth and others. It is predominantly seen in older patients (over 65 years) but prevalence among younger adults (<45 years) are increasing. Males are more commonly affected. However, the difference between the gender are decreasing. There is geographic variation in the incidence of OSCC and there is now accumulating evidence of ethnic variations as well. It may arise de novo or from pre-existing potentially malignant lesions such as leukoplakia or erythroplakia. Clinically OSCC can present as an exophytic or endophytic ulcer **(Figure 2.9)**.

Aetiology: Lifestyle factors especially use of tobacco and alcohol have been attributed to be major risk factors for OSCC. Betel quid chewing habits have also been implicated in the development of OSCC. Diet, genetic and immunological factors may also play a role in the development of OSCC as not all cases of OSCC have risk habits as mentioned above.

Treatment: Management of OSCC requires a multidisciplinary team approach. Surgery with or without post-operative radiotherapy is the main mode of treatment. For advanced tumour stage, chemotherapy may be beneficial. Newer treatment modalities such as targeted therapies may also be of benefit in certain cases.

Prognosis: OSCC is an epithelial malignancy with a potential to cause local invasion, regional nodal spread and distant metastasis. Stage 1 and 2 OSCC have better prognosis than late stages. Morbidity and mortality rates are poorer for late stage tumours. Hence, early diagnosis is essential. Any ulcers, red or white lesions which are persistent (>3 weeks) after removal of any potential local causes should be biopsied to rule out OSCC.

Differential Diagnosis: Traumatic ulcers, tuberculosis, major RAS.

2.2 WHITE LESIONS

There are many lesions affecting the oral mucosa which can present as a white lesion. As infections, reactive and benign conditions can present clinically as white lesions, it is important to differentiate these from potentially malignant or malignant lesions.

(a) Leukoedema

Description: It appears as a diffuse grayish-white, oedematous film which is present bilaterally on the buccal mucosa **(Figure 2.10a)**. The delicate whitish folds disappear on stretching the mucosa **(Figure 2.10b)** and reappear upon release. There is no pain symptom associated.

Aetiology: Aetiology is not clear.

Treatment: No active treatment required.

Prognosis: There is no malignant potential.

Differential Diagnosis: Other causes of white patches such as leukoplakia, frictional keratosis.

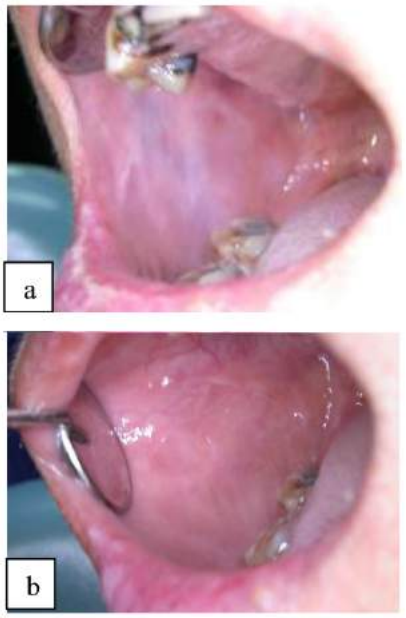


Figure 2.10 Leukoedema (a) Delicate folds on buccal mucosa (b) folds disappear on stretching.

(b) Frictional Keratosis

Description: Frictional keratosis **(Figure 2.11)** is common. It appears as whitish pale translucent lesions in the early stages which later becomes dense and white with rough surface. It is commonly seen on the buccal mucosa (unilateral/bilateral), margins of the tongue and sometimes on the edentulous ridge or gingiva.

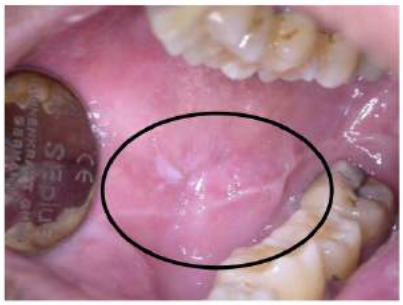


Figure 2.11 Frictional keratosis (circled)

Aetiology: Repeated trauma such as cheek biting, sharp teeth edges, rough food, toothbrushing or ill-fitting appliances. Rarely self-mutilating habits as seen in psychiatric disorders or some rare syndromes may present with frictional keratosis.

Treatment: Diagnosis is based on clinical history and findings. It is important to differentiate the frictional keratosis from other predominantly white lesions such as white sponge naevus, lichen planus and leukoplakia. Besides removal of any possible traumatic factors and advise stopping any habits, no active treatment is required.

Prognosis: The lesion has no malignant potential. However, a biopsy may be warranted if either the patient or clinician have any concerns.

Differential Diagnosis: Other causes of white patches such as leukoplakia, lichen planus, chronic hyperplastic candidosis.

(c) Acute Pseudomembranous Candidosis ("Thrush")

Description: Acute pseudomembranous candidosis or "thrush" **(Figure 2.12)** is the white form of candidal infection clinically characterized by white flecks, papules or plaques which can be rubbed off to leave erythematous, raw mucosal base. It is categorized as an opportunistic infection and thus more common in immunocompromised individuals and also neonates who have not yet developed mature immune status.

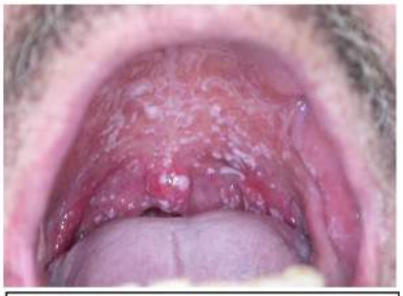


Figure 2.12 Acute pseudomembranous candidosis

Aetiology: The main causative organism is C. albicans although other candidal strains can also cause thrush. There are multiple predisposing factors which can either be due to local changes or systemic immunity. The use of broad-spectrum antibiotics and corticosteroids, decreased in salivation secondary to head & neck radiotherapy, immune defects such as HIV or patients on immunosuppressive treatment are a few common predisposing factors.

Treatment: Diagnosis is based mainly on clinical findings. A smear to identify hyphae and spores or culture to support the diagnosis can be carried out if necessary. Any possible predisposing factors should be identified and dealt with. Treatment with topical antifungals such as nystatin, miconazole or fluconazole may be indicated.

Prognosis: Recurring infection may occur if underlying predisposing factors are not fully investigated and managed.

Differential Diagnosis: Other causes of white patches such as leukoplakia, frictional keratosis.

(d) Chronic Hyperplastic Candidosis

Description: Also known as candidal leukoplakia **(Figure 2.13)**, this type of candidosis present as persistent, discrete raised lesion having white or speckled red/white appearance. Usually occur on the buccal mucosa on one or both sides at the commissure region. Unlike acute pseudomembraneous candidosis, the lesion is not readily rubbed off.

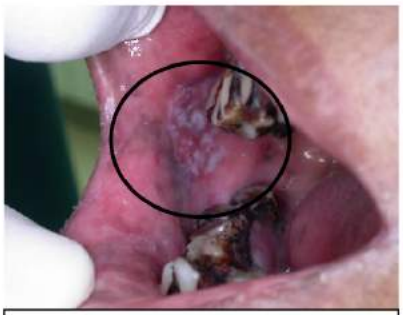


Figure 2.13 Chronic hyperplastic candidosis (circled)

Aetiology: C. albicans is by the far the most common species isolated from these lesions. In a minority of the patients, smoking, iron and folate deficiencies and defective cell-mediated immunity may be possible predisposing factors.

Treatment: Chronic hyperplastic candidosis is clinically indistinguishable from other leukoplakias. A biopsy is required to demonstrate the presence of Candidal hyphae and to distinguish it from other non-candidal white lesions. Histopathological examination is also necessary to assess the presence or absence of dysplasia. Antifungals should be used in treating this lesion. In some cases, use of systemic fluconazole or itraconazole may be required. Surgical excision is indicated if dysplasia is reported to be more than mild.

Prognosis: 9-40% of chronic hyperplastic candidosis may develop into carcinoma. Candidal biotypes associated with chronic hyperplastic candidosis have been shown to have higher nitrosation potential, thus indicating a possible role in malignant transformation of these lesions. Factors which may influence the prognosis include tobaccco/alcohol use and the degree of dysplasia.

Differential Diagnosis: Other causes of white patches such as leukoplakia, frictional keratosis, squamous cell carcinoma.

(e) Hairy Leukoplakia

Description: Persistent, white, corrugated lesion usually seen on one or both lateral border of the tongue **(Figure 2.14)**. The lesion is typically asymptomatic.

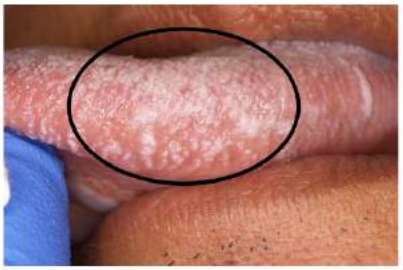


Figure 2.14 Hairy Leukoplakia (Circled)

Aetiology: Associated with Epstein Barr virus. Seen mainly in HIV/AIDS patients and other immunocompromising states.

Treatment: Diagnosis is largely clinical. Treatment is often not required. Use of acyclovir or other antiviral therapy may resolve the lesion.

Prognosis: No malignant potential. In HIV/AIDS cases, presence of the lesion is a predictor of poor prognosis.

Differential Diagnosis: Other causes of white patches such as leukoplakia, frictional keratosis.

(f) Oral Lichen Planus

Description: Lichen planus (LP) is common mucocutaneous disorder, usually seen between the ages 30-65 years and have a slight female predominance. Oral LP (OLP) may occur in isolation or may precede/ accompany other cutaneous sites. Clinically, it presents mainly as a white lesion but often appears as mixed white/red areas. There are 6 clinical subtypes of OLP. Reticular OLP **(Figure 2.15 a-b)** appears as a white reticular network of raised white lines termed striae. The other predominantly white subtypes are papular (multiple white papules) and plaque-like (white patches resembling leukoplakia). The white striae may appear less prominent in papular and plaque-like subtypes. OLP which consists of only white lesions are often asymptomatic. Burning sensation is the most common complaint in symptomatic cases. (see next section for other subtypes of OLP)

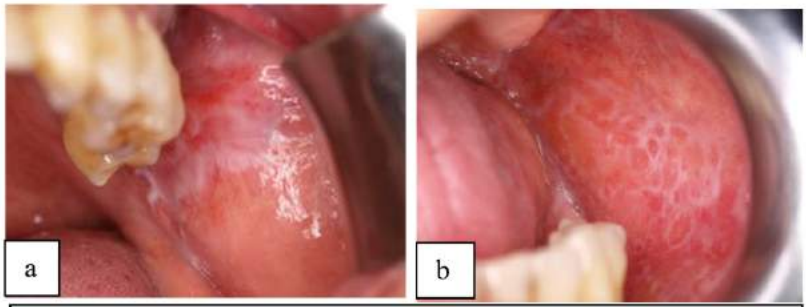


Figure 2.15 (a,b) Lichen Planus - reticular

Aetiology: OLP is an immunologically mediated disorder involving cytotoxic T cells resulting in epithelial damage. Familial cases have been reported suggesting a genetic basis for OLP. Although stress has been widely said to be an important aetiological factor, very few studies have objectively examined it.

Treatment: Treatment is indicated for symptomatic OLP to reduce the discomfort and control the symptoms. This include topical medication such as benzydamine hydrochloride (0.15%) spray or mouthwash and corticosteroids such as triamcinolone acetonide.

Prognosis: OLP has a small malignant potential of 1.1 – 1.37%[1,2] especially in the non-reticular OLP subtypes. Leukoplakia and lupus erythematosus need to be considered in the differential diagnoses as these lesions show similar clinical presentations.

Differential Diagnosis: Other causes of white patches such as leukoplakia, frictional keratosis.

[1] Aghbari SMH et al. 2017. Malignant transformation of oral lichen planus & oral lichenoid lesions: A meta-analysis of 20095 patient data. Oral Oncol. 68:92-102.
[2] Richards D. 2018. Malignant transformation rates in oral lichen planus. Evid Based Dent 19(4):122

(e) Leukoplakia

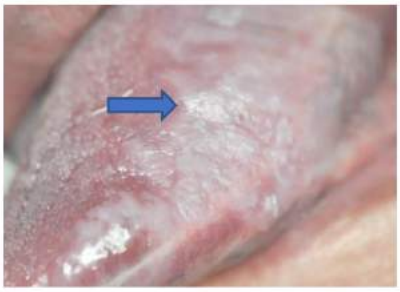


Figure 2.16 Homogenous Leukoplakia (arrow)

Description: Leukoplakia is defined by WHO as "clinical patches that cannot be wiped off and cannot be classified clinically or microscopically as another specific disease entity" [3]. Thus, leukoplakia is a clinical diagnosis only and can only be made by exclusion. Leukoplakia varies in size and can be small, focal or more widespread. It can be classified into homogenous and non-homogenous type. Homogenous leukoplakia **(Figure 2.16)** appears as uniformly white patches/plaques which maybe flat, wrinkled, pumice-like or corrugated. Commonly seen on the buccal mucosa but may occur at any oral site. Soft palate, ventrolateral tongue and floor of the mouth are considered high risk sites. (see next section for non-homogenous leukoplakia)

Aetiology: The exact aetiology is unknown. However, presdisposing factors include habits such tobacco, alcohol and betel quid use, trauma and vitamin A deficiency.

Treatment: Leukoplakia is a potentially malignant disorder with 1-2%[4] reported malignant change. The treatment depends on the histological finding and the extent of the lesion. Initial approach is to eliminate known risk factors. Surgical excision is beneficial in persistent leukoplakia or lesions where dysplasia is reported as carcinomatous foci may be present. If malignancy is reported histologically, then the leukoplakia has to be managed as treatment of oral squamous cell carcinoma.

Prognosis: Regular monitoring is necessary as recurrence has been reported. Even clinically homogenous leukoplakia may contain areas of dysplasia and some studies have even shown carcinoma in-situ/ invasive carcinoma in up 5-10% of leukoplakia where the initial incisional biopsy shows little or no dysplasia.

Differential Diagnosis: Other causes of white patches such as lichen planus, frictional keratosis, squamous cell carcinoma.

[3] Warnakulasuriya S, Newell WJ, van der Waal I. 2007. Nomenclature and classification of potentially malignant disorders of the oral mucosa. J Oral Pathol Med 36:575-80

[4] Petti S. 2003. Pooled setimate of world leukoplakia prevalence: a systematic revies. Oral Oncol 39:770-80

2.3 MIXED RED AND WHITE LESIONS

(a) Geographic Tongue

Description: Geographic tongue **(Figure 2.17)** is a harmless condition affecting the surface of tongue. It is also known as benign migratory glossitis and erythema migrans. It occurs in 1-2 % of adults and may be seen at any age. Geographic tongue typically involves the dorsum of the tongue, sometimes the ventrum, and rarely other areas on the oral mucosa.

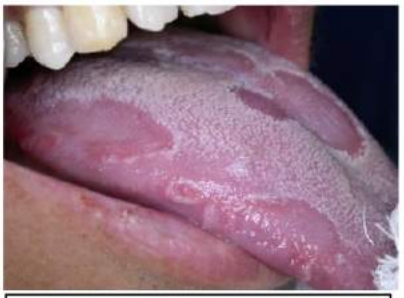


Figure 2.17 Geographic Tongue

The tongue is normally covered with papillae which are short, fine, hair like projection. With geographic tongue, patches on the surface of the tongue are missing the papillae and appear as smooth, red 'islands' often with slightly raised border. These patches (lesions) give the tongue a map like, or geographic appearance. The lesions often heal in one area and then move (migrate) to a different part of the tongue. Sign and symptoms of geographic tongue may include:

i. Smooth, red, irregularly shaped patches
ii. Frequent changes in location, size and shape of lesions
iii. Discomfort, pain or burning sensation in some cases

Aetiology: The cause of geographic tongue is unknown. A relationship between geographic tongue and psoriasis have been reported. Hereditary association have been reported, suggesting the involvement of genetic factors in the aetiology. However more research is need to better understand the relationship.

Treatment: In most cases, there is no need for treatment. Occasionally geographic tongue may cause a burning sensation. When symptoms are reported, topical anaesthetic may be used to obtain temporary relief. Other suggested treatment include antifungal, baking soda, antihistamine and topical steroids.

Prognosis: Geographic tongue is a benign condition and has no malignant potential.

Differential Diagnosis: The clinical features of this lesion are quite characteristic and histopathologic confirmation is rarely needed. Very similar lesions may be seen in psoriasis and Reiter syndrome. The clinical differential diagnosis might include glossitis, candidiasis, leukoplakia, lichen planus and lupus erythematosus.

(b) Smoker's Keratosis

Description: Smoker's keratosis is also called as nicotinic stomatitis It is refers to a diffuse white patch on the hard and soft palate **(Figure 2.18)**, usually caused by tobacco smoking. The palate may appear grey or white with papules or nodules that are slightly elevated with central red dots. These red dots represent the duct openings of minor salivary glands which have become inflamed by heat.

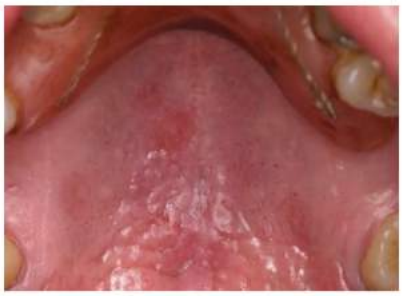


Figure 2.18 Smoker's keratosis

Aetiology: The cause of smoker's keratosis is thought to be heat induced keratosis rather than an effect of tobacco. The severity of the changes correlates with the frequency of the habit.

Treatment: This condition is painless. Lesion is usually completely reversible, once the smoking habit is discontinued.

Prognosis: The condition is not known to be potentially malignant.

Differential diagnosis: The diagnosis is based upon the clinical appearance and history. The differential diagnosis is with other causes of white lesions such as leukoplakia, Darier's disease, discoid lupus erythematosus, oral candidiasis and oral lichen planus.

(c) Lichen Planus

Description: Lichen planus is a chronic mucocutaneous disease. Most patients with lichen planus are middle aged adults. The oral lesions predominantly affect the buccal mucosa, tongue and gingivae, although other sites are occasionally involved. The patient may also have skin or genital involvement that can assist in the diagnosis of the oral lichen planus.

The oral lesions typically present as bilateral white lesions, occasionally with associated erosion or ulcers, Generally, there are two forms of oral lesions: reticular and erosive. The erosive form of lichen planus **(Figure 2.19a)** appears as red or erythematosus areas with central ulceration of varying degree and bordered by fine white striae. The attached gingival may involve, producing the reaction pattern called desquamative gingivitis **(Figure 2.19b)**. Erosive form is not as common as the reticular form, but it is more significant for the patient because the lesions are usually symptomatic.

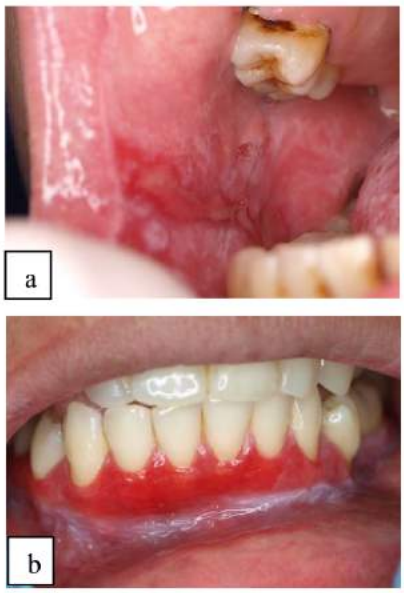


Figure 2.19 Erosive Lichen Planus
(a) Erosive area with striae
(b) Desquamative gingivitis

Aetiology: The aetiology of oral lichen planus is unknown. Current data suggest that oral lichen planus is a T cell mediated autoimmune disease in which autocytotoxic CD8 T cells trigger the apoptosis of the oral epithelial cells

Treatment: Erosive form is usually symptomatic. Symptoms can often be controlled with topical medication such as topical corticosteroid. Antifungals may help especially when there is candida superinfection. In severe cases systemic corticosteroid may be used. Other therapies for LP include azathioprine, cyclosporin, retinoids and dapsone.

Prognosis: Oral LP have a small premalignant potential and in non-reticular lesion (erosive form) a higher transformation rate has been reported [1].

Differential Diagnosis: The clinical features of lichen planus are characteristic but may not be specific, because other conditions such as lichenoid reaction, lupus erythematosus, chronic ulcerative reaction or graft versus host disease may also show a similar clinical pattern.

(d) Discoid Lupus Erythematosus

Figure 2.20 Discoid Lupus Erythematosus

Description: Lupus erythematosus is an immunological mediated condition. Patient with discoid lupus erythematosus usually have few or no systemic signs and symptoms, with lesions being limited to skin or mucosal surfaces. In most cases, the oral manifestation of DLE essentially appear clinically identical to the lesions of erosive lichen planus **(Figure 2.20)**. Characteristic features include an ulcerated or atrophic, erythematosus central zone, surrounded by white, fine radiating striae.

Aetiology: The aetiology is unclear, but drugs, hormones, and viruses may contribute in genetically predisposed patient.

Treatment: For patient with limited oral mucosa disease, topical corticosteroid is appropriate. Systemic steroid is used in severe cases of DLE.

Prognosis: The prognosis for patients with DLE is considerably better than that for patients with SLE. Transformation to SLE may be seen in approximately 5% of DLE patients.

Differential diagnosis: The oral manifestations of DLE may be difficult to differentiate from lichen planus lesions and lichenoid reaction. However palatal lesions are more common in DLE than in the lichen planus.

(e) Leukoplakia

Description: Leukoplakia occurs in about 0.1% of the population and predominantly in the middle aged and elderly. Some leukoplakia occur in combination with adjacent red patches. If the red and white areas are intermixed, the lesion is called erythroleukoplakia, leukoerythroplakia or speckled leukoplakia **(Figure 2.21)**. White lesions occurring inside the angles (commissure) of the mouth frequently have a speckled appearance and are usually associated with chronic candida infection (candidal leukoplakia).

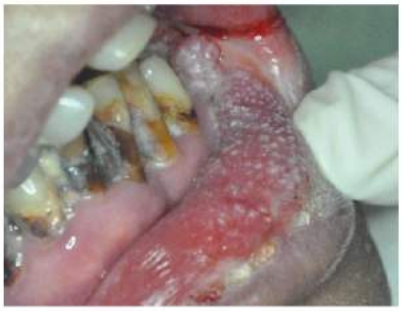


Figure 2.21 Speckled Leukoplakia

Aetiology: The cause of leukoplakia remains unknown. The risk factors that increase a person's chance of developing leukoplakia are heavy smoking, excessive alcohol use and chewing tobacco.

Treatment: Removal of known risk factors (tobacco, alcohol, betel and trauma) is mandatory. Candidal leukoplakia may respond to antifungals. If the lesion persists, it should be biopsied or removed.

Prognosis: Non-homogenous leukoplakia may be associated with an increased risk of malignant change[5]. The mean global malignant transformation rate of leukoplakia has been estimated to be 1-2%[4] . Leukoplakia on the floor of mouth, ventrolateral tongue and soft palate tend to have a greater potential for malignant transformation than in other sites.

Differential diagnosis: The clinical differential diagnosis might include frictional keratosis, candidiasis, lichen planus, lupus erythematosus and white sponge naevus. Leukoplakia is a diagnosis of exclusion.

[5]Holmstrup P, Vedtofte P, Reibel J, Stoltze K. 2006. Long-term treatment outcome of oral premalignant lesions. Oral Oncol 42:461-74.

(f) Oral Cancer

Description: More than 90% of oral cancer is oral squamous cell carcinoma (OSCC). OSCC is seen predominantly in elderly patients but intraoral cancer is increasing in younger adults. OSCC may present clinically as a mixed white and red lesion **(Figure 2.22)** with varying degree of ulcer or raised exophytic margins. An exophytic lesion typically has a surface that is irregular, fungating, papillary or verruciform. The lesion is usually fixed to deeper tissue and feels hard on palpation (indurated).

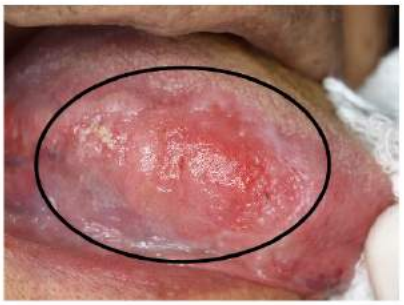


Figure 2.22 Squamous Cell Carcinoma with mixed red and white appearance (Circled)

Aetiology: A predisposition to OSCC has been attributed mainly to specific risk factors, such as tobacco (smoking and smokeless), chewing betel quid and alcohol. Dietary factor as well as the existence of genetic predisposition may also play a role.

Treatment: Carcinoma is usually treated by surgical excision.

Prognosis: The prognosis for survival from oral cancer depends on tumour stage.

Differential diagnosis: The differential diagnosis for a squamous cell carcinoma is essentially other malignant lesions (eg. lymphoma, salivary gland tumours and sarcoma) as well as potentially malignant lesion (eg. erythroleukoplakia, erythroplakia) and a few non-neoplastic lesions (eg. ulcer, growth, infection). The clinician's challenge is to differentiate cancerous lesions from multitude of other red, white or ulcerated lesions that also occur in the oral cavity. Most oral lesions are benign but may have appearance that may be confused with a malignant lesion. Conversely. some malignant lesions seen in an early stage may be mistaken for a benign entity. Any oral lesions that does not regress spontaneously or respond to usual therapeutic measures should be considered potentially malignant until histologically shown to be benign.

2.4 RED LESIONS

Red lesions can be either reactive, infective, benign or potentially malignant. Potentially malignant predominantly red lesions are less common than white lesions and has a higher risk of malignant transformation. Thus, it is important to differentiate a benign or reactive red lesion from lesion which is potentially malignant.

(a) Denture-related Stomatitis

Description: Denture-related stomatitis affects the palate **(Figure 2.23)** and appears as red sometimes spongy/ granular areas. The appearance is very characteristic and covers only the denture bearing area. The condition is generally asymptomatic but some patients may complaint of mild soreness. Disease is mainly seen among middle-aged and elderly.

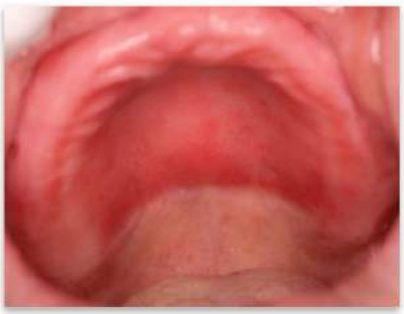


Figure 2.23 Denture-related

Aetiology: Poor denture hygiene and wearing of dentures at night are main predisposing factors. Smoking, diabetes, dry mouth occasionally predisposes while haematinic deficiencies and HIV are rarely an underlying factor. Allergy to dental material may be another factor. Accumulation of microbial plaque consisting of both bacteria and yeasts plays a role in the pathogenesis of this condition with Candida albicans the most frequently isolated organism. Denture-related stomatitis is considered as a form of erythematous or atrophic candidosis.

Treatment: Improving denture hygiene and storing denture in suitable antiseptic solution at night may improve the condition. Other possible predisposing factors should be addressed and this include cessation of smoking and treating dry mouth with oral moisturisers or mouthwash. Supportive treatment with Chlorhexidine mouthwash may be beneficial. Use of topical antifungal such as nystatin suspension may sometimes be required.

Prognosis: Uncommon complications include angular stomatitis and papillary hyperplasia of the palate.

Differential Diagnosis: Erythroplakia, granulomatous lesions.

(b) Median Rhomboid Glossitis

Description: A benign condition clinically characterized by red depapillated lesion at midline dorsum of the tongue **(Figure 2.24)** just anterior to the circumvallate papilla. It typically appears as oval or rhomboid shaped with smooth shiny surface. The lesion may less commonly appear as hyperplastic or lobulated and exophytic. The condition is rarely associated with soreness. Sometimes, an associated opposing red lesion is seen on the palate and this is known as "kissing lesion".

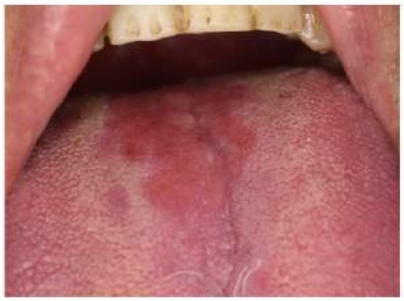


Figure 2.24 Median Rhomboid Glosssitis

Aetiology: Lesion was once thought to be a developmental defect. However, many believed median rhomboid glossitis is related to candida infection and culture from these lesions have shown presence of candida species. It is thought to be a type of chronic erythematous candidosis. Smoking, denture wearing, use of corticosteroid inhalers and HIV infection may predispose to the condition.

Treatment: Diagnosis is usually made based on clinical appearance and biopsy is not required. Treatment includes cessation of smoking and use of topical antifungal agents such as nystatin.

Prognosis: Usually resolution of the lesion after antifungal therapy but sometimes lesion may be resistant to complete resolution. Condition is benign and there is no malignant potential.

Differential Diagnosis: Erythroplakia, Lichen planus, granular cell tumour, squamous cell carcinoma.

(c) Erythroplakia

Description: It is defined by WHO as "any lesion of the oral mucosa that presents as bright red velvety plaques which cannot be characterized clinically or pathologically as any other recognizable condition"[6] **(Figure 2.25)**. Men are mostly affected. It is commonly seen on the soft palate, floor of the mouth and buccal mucosa.

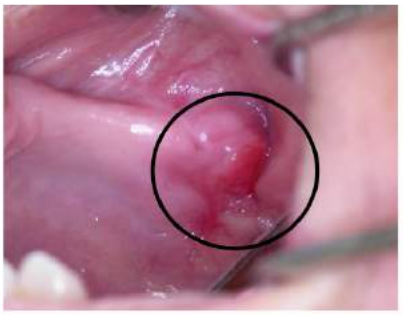


Figure 2.25 Erythroplakia (Circled)

Aetiology: Aetiology is unknown but tobacco, alcohol and betel use may be involved.

Treatment: Biopsy is mandatory to assess the degree of dysplasia and to differentiate from other inflammatory or atrophic lesions. Surgical excision is the main mode of treatment. Supportive treatment includes cessation of smoking, alcohol consumption and betel use. Regular monitoring by specialist is crucial.

Prognosis: Among the oral potentially malignant lesions, erythroplakia has the greatest predilection to develop to carcinoma. They often present with severe dysplasia or may have already develop into carcinoma in-situ / invasive carcinoma.

Differential Diagnosis: Other inflammatory and atrophic lesions such as geographic tongue, lichen planus, denture-related stomatitis, betel chewers mucosa and other.

[6] Pinborg JJ et al. 1997. WHO International Histological Classification of Tumours. Histological typing of cancer & precancer of the oral muocsa. 2nd ed. Berlin, Germany:Springer.

(d) Haemangioma and Vascular Malformations

Description: Vascular lesions such as haemangioma **(Figure 2.26)** and vascular malformations **(Figure 2.27)** should be included in the differential diagnosis of red lesions in the oral cavity. They appear as well-demarcated flat or raised lesions usually deep red in colour and may also appear blue or purple. Portwine stains are common capillary malformations that occur among newborns. The lesions darken and becomes more nodular as the patient ages. Lesions normally blanched upon pressure.

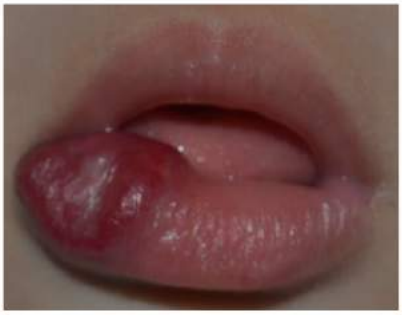


Figure 2.26 Haemangioma

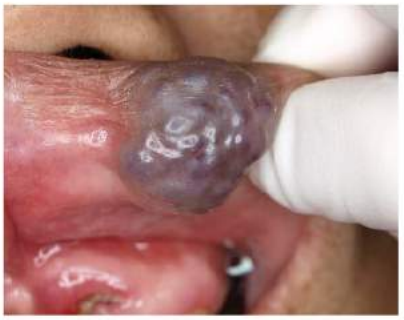


Figure 2.27 Vascular malformation

Aetiology: Traditionally the term haemangioma has been used to describe a variety of vascular developmental vascular anomalies. Currently, haemangiomas are considered benign tumours of infancy that displayed rapid growth of endothelial cell proliferation followed by gradual involution. Vascular malformations on the other hand are structural anomalies of blood vessels without endothelial proliferation. They are present at birth and persist throughout life.

Treatment: Most haemangiomas of infancy undergo involution and as such management involves mainly monitoring. Surgical intervention is warranted if the lesion is problematic or life-threatening otherwise a more conservative management option is adopted.

Prognosis: Vascular malformations of the jaws have a potential risk of severe bleeding which may occur spontaneously or during surgical manipulation. Cases of fatal haemorrhages have occurred following incisional biopsy and dental extractions.

Differential Diagnosis: Inflammatory oral lesions such as pyogenic granuloma, malignant vascular tumours such as Kaposi sarcoma, varices and others.

2.5 PIGMENTED LESIONS

Pigmented lesions are commonly found in the mouth. Such lesions represent a variety of clinical entities ranging from physiologic changes to manifestation of systemic illness and malignant neoplasms. Most pigmented lesions in the mouth are benign, however biopsy is often required to exclude sinister pathology.

(a) Amalgam Tattoo

Description: Amalgam tattoo **(Figure 2.28)** is one of the most frequent causes of exogenous pigmentation in the oral mucosa. Clinically, it presents as asymptomatic grey, blue or black, non-blanching macules in the oral mucosa. The borders can be well defined, irregular or diffuse. Amalgam tattoo usually occurs on the gingival and alveolar mucosa, but these lesions may also involve the buccal mucosa or floor of the oral cavity.

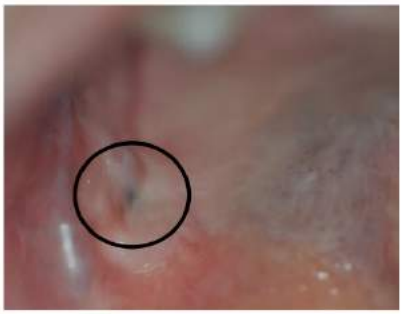


Figure 2.28 Amalgam Tattoo (Circled) – pigmented lesion on edentulous alveolar ridge

Aetiology: Amalgam tattoo is usually caused by amalgam splinters inadvertently implanted into the mucosa during dental restorations but may also be caused by diffusion through the teeth.

Treatment: No treatment is required since the lesion is entirely benign.

Prognosis: Painless and benign

Differential diagnosis: The amalgam tattoo should be differentiated from other pigmented lesions such as melanocytic nevus, focal melanosis, physiological pigmentation, thrombosed varix, malignant melanoma and implanted exogenous materials. To confirm the diagnosis of amalgam tattoo, the clinician can obtain radiographs of the areas of mucosal discoloration in an attempt to demonstrate the metallic fragments. If no metallic fragments are found and the lesion cannot be diagnosed clinically, biopsy may be needed to rule out the possibility of melanocytic neoplasia.

(b) Naevus

Description: Naevus **(Figure 2.29)** is a benign and focal proliferation of naevus cells that can be congenital or acquired. It is a hamartoma rather than true neoplasm. The most common mucosal type is the intramucosal naevus followed by the blue naevus. It can occur at any age but is most prevalent between the second and third decade of life. The palate is the most commonly affected site. Less common sites are the buccal mucosa, labial mucosa, gingiva, alveolar ridge and vermillion border. Clinically, naevus manifests as macules or slightly raised papules or plaques. They are usually small in size (<1 cm). The colour varies from light brown to dark brown and occasionally present as a non-pigmented lesion.

Figure 2.29 Naevus (Circled)

Aetiology: The aetiology and pathogenesis are poorly understood.

Treatment: If clinically indicated, conservative surgical excision is the treatment of choice.

Prognosis: Malignant transformation to malignant melanoma is rare but has been reported.

Differential diagnosis: Clinically, oral naevus can be indistinguishable from melanotic macule. Biopsy is usually required to establish the diagnosis and to rule out malignant melanoma.

(c) Racial Pigmentation

Description: The most usual cause of patchy or generalized brown oral mucosal pigmentation **(Figure 2.30)** is by melanin. This pigmentation may be seen in persons of any age. It usually develops during the first 2 decades of life but may not come to the patient's attention until later. Racial pigmentation is symmetric and persistent. The colour ranges from light to dark brown. The attached gingiva is the most common intraoral site of such pigmentation, where it appears as a bilateral, well demarcated, ribbon-like, dark brown band that usually spares the marginal gingiva.

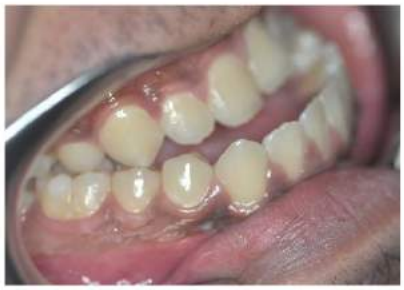


Figure 2.30 Racial Pigmentation

Aetiology: Racial pigmentation is due to greater melanocyte activity rather a greater number of melanocytes.

Treatment: The pigmentation is asymptomatic, and no treatment is required

Differential diagnosis: A clinical differential diagnosis would include smoking-associated melanosis, Peutz-Jeghers syndrome, Addison's disease and melanoma. Although racial pigmentation is usually clinically diagnostic, a biopsy may be indicated if clinical features are atypical.

(d) Malignant Melanoma

Description: Mucosal melanoma **(Figure 2.31)** is very rare but it is one of the most aggressive malignancies. The recorded incidence is up to 1 or 2 % of all melanomas. The most common site is the palate followed by the gingiva. Other oral mucosa sites may also be affected. Clinical signs of mucosal melanoma are usually dark brown, black or bluish-greyish plaques with irregular pigmentation and an asymmetrical, irregular border. Swelling, ulceration, bleeding, pain/discomfort are also common.

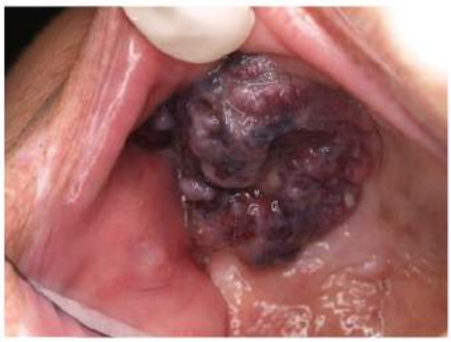


Figure 2.31 Malignant Melanoma

Aetiology: Melanomas may develop in or near a previously existing precursor lesion or in healthy-appearing skin. A malignant melanoma developing in healthy skin is said to arise de novo, without evidence of a precursor lesion. Many of these melanomas are induced by solar irradiation.

Treatment: Surgery is the definitive treatment for early-stage melanoma, with medical management generally reserved for adjuvant treatment of advanced melanoma.

Prognosis: Prognosis is uniformly described as poor and the overall 5-year survival rate is 15%. Differential diagnosis: Differentials to consider in the diagnosis of malignant melanoma include benign melanocytic lesions such as naevus, melanotic macules, oral melanoacanthoma and malignant tumour such as pigmented spindle tumour

2.6 LUMPS AND BUMPS

Swelling and lumps in the oral cavity are common. They are comprised of reactive lesions, benign growth or malignant tumours. Infections such as dental abscesses may also present as swelling. Normal anatomical structures such as tori, foliate, circumvallate and parotid papillae need to be differentiated from pathological lesions.

(a) Fibrous Epulis/ Fibrous Hyperplasia

Description: Present as a pedunculated or sessile mass **(Figure 2.32)** usually on the gingiva or buccal mucosa. They are one of the most common oral soft tissue lesions. Colour is similar to surrounding mucosa and is soft or mildly firm in consistency. Lesion is generally painless and very slowly progressive in nature.

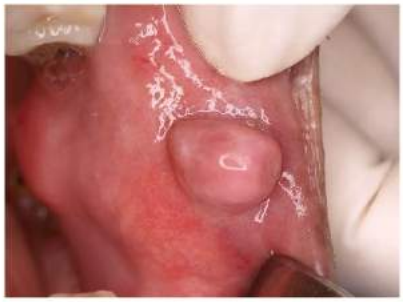


Figure 2.32 Fibroepithelial hyperplasia

Aetiology: Lesion is generally reactive in nature and chronic trauma or irritation to the mucosa is the main aetiological factor.

Treatment: Excision of the lesion is the mainstay of treatment. Any predisposing trauma or irritation should also be eliminated.

Prognosis: Prognosis is good and lesion rarely recur unless trauma or irritation is still not fully eliminated.

Differential Diagnosis: Other soft tissue lesions or tumours such as pyogenic granuloma, mucocele or benign or malignant tumours.

(b) Denture-induced Hyperplasia

Description: Lesion **(Figure 2.33a-b)** occurs as a flabby mass present at the buccal vestibule, alveolar ridge or sometimes on the hard palate and is associated with an ill-fitting denture. Generally asymptomatic unless ulcerated.

Aetiology: Reactive lesion due to trauma from ill-fitting denture.

Treatment: Excision of the lesion and reduction of the denture borders or adjustment of any ill-fitting denture areas.

Prognosis: Good. Recurrence is rare.

Differential Diagnosis: Clinical appearance is generally highly characteristic of the lesion and differential diagnosis is not a problem.

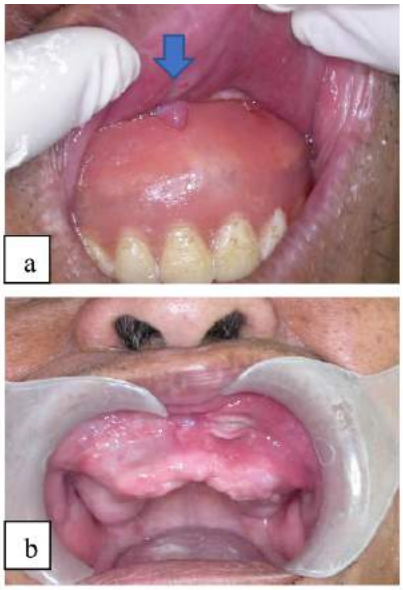


Figure 2.33 Denture-induced hyperplasia (a) Lesion with denture in-situ (b) Hyperplastic mass with ulceration

(c) Pyogenic Granuloma/ Pregnancy Epulis

Description: Reddish nodular overgrowth with surface ulceration, generally seen on the gingiva **(Figure 2.34)**, lips, tongue and buccal mucosa. The interdental papilla of the maxillary gingiva is the most common site. Because of the vascular nature of the lesion, lesions tend to bleed easily and mild pain may be experienced. Lesions commonly develop during pregnancy.

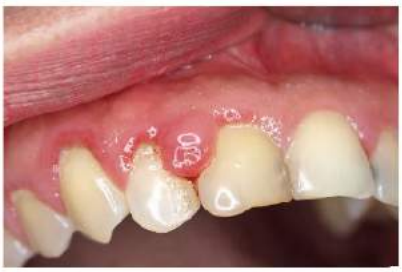


Figure 2.34 Pyogenic Granuloma

Aetiology: Chronic irritation or mild trauma may provoke the development of the lesions.

Treatment: Excision.

Prognosis: Good. Recurrence may occur if chronic irritation is still present.

Differential Diagnosis: Fibrous epulis, mucoceles, haemangioma or other vascular lesions.

(d) Mucoceles

Description: Present as a translucent or whitish dome-shaped or pedunculated lesion commonly seen on the lower lips **(Figure 2.35a)**, buccal mucosa and ventral tongue **(Figure 2.35b)**. Lesions are soft in nature and often burst and recur.

Aetiology: Mucocele is composed of a collection of mucin. The commonest histological type is mucous extravastion cyst where the cyst lining is devoid of epithelium and is made up of granulation tissue or compressed connective tissue. Traumatic biting causing severance of the salivary duct of the minor salivary glands leading extravasation and collection of mucin.

Treatment: Excision of the lesion and the adjacent affected minor glands.

Prognosis: Good. Recurrence may sometimes occur.

Differential Diagnosis: Other soft tissue growth such as pyogenic granuloma, salivary gland tumour, haemangioma and others.

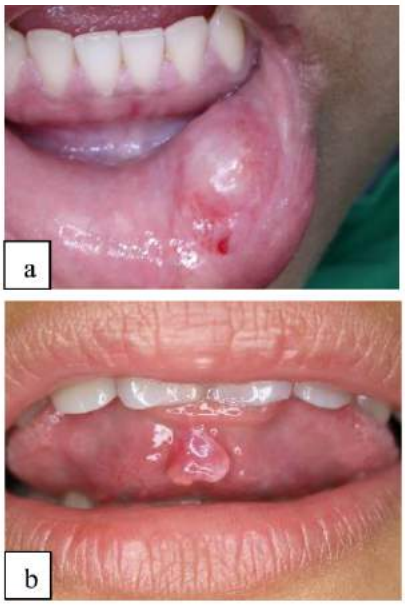


Figure 2.35 Mucocele at (a) Lower lip (b) ventral tongue

(e) Squamous Cell Papilloma

Description: Papilloma **(Figure 2.36)** appears as pedunculated or sessile white or pink growth with finger-like projections. Average size is less than 1.0 cm with most lesions being only a few millimetres in diameter. Most common site is the palate followed by the tongue and lips. Lesions are generally asymptomatic.

Aetiology: Squamous cell papilloma is a benign epithelial tumour of the oral mucosa. Human papilloma virus 6 and 11 have been implicated in development of this lesion.

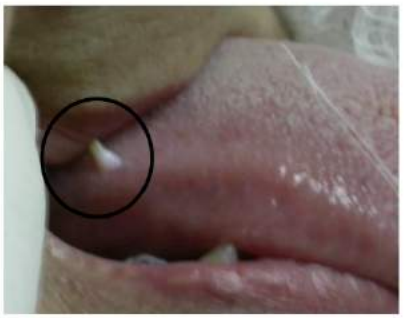


Figure 2.36 Squamous cell papilloma (circled)

Treatment: Excision.

Prognosis: Recurrence is rare. Lesion has no malignant potential.

Differential Diagnosis: Other viral warts such as verruca vulgaris and condyloma acuminatum. Larger lesion may resemble leukoplakia or verrucous carcinoma.

(f) Oral Squamous Cell Carcinoma

Description: Localised red/white swelling which is progressively increasing in size. The surface may appear nodular **(Figure 2.37)** or verrucal with/without ulceration. Can occur on any mucosal surface. Lesion on the alveolus may present with mobility of associated tooth and radiograph will show ill-defined radiolucency. Speech may be altered if the tongue is involved. Bleeding and pain may be a feature of advance disease state.

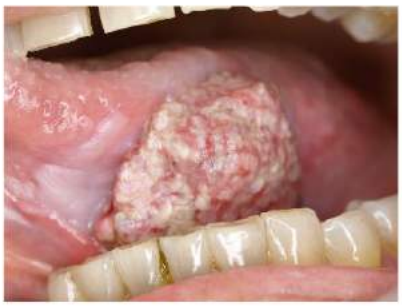


Figure 2.37 Nodular Squamous Cell Carcinoma

Aetiology: see previous section

Treatment: Oral cancer management protocol needs to be followed.

Prognosis: Prognosis depends on the stage of the disease, nodal status and presence of distant metastasis.

Differential Diagnosis: Early lesions may resemble reactive/ benign tumours. For advance lesions, other malignancies or metastatic tumours need to be considered.

(g) Metastatic Tumours

Description: Localised reddish, pink or normal coloured swelling on the gingiva or jaw swelling **(Figure 2.38)** which is rapidly increasing in size. Surface ulceration may or may not be present. Mandible is more frequently affected than the maxilla. Swelling on the jaw may be the first manifestation of the tumour. May present with associated symptoms of paraesthesia, tooth mobility and pathological jaw fracture. Radiograph will show ill-defined radiolucencies or radiopacities.

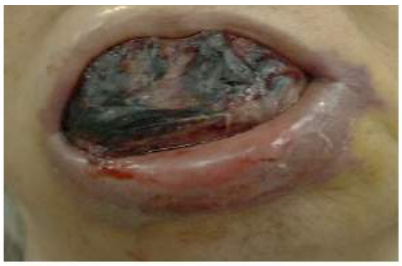


Figure 2.38 Metastatic tumour

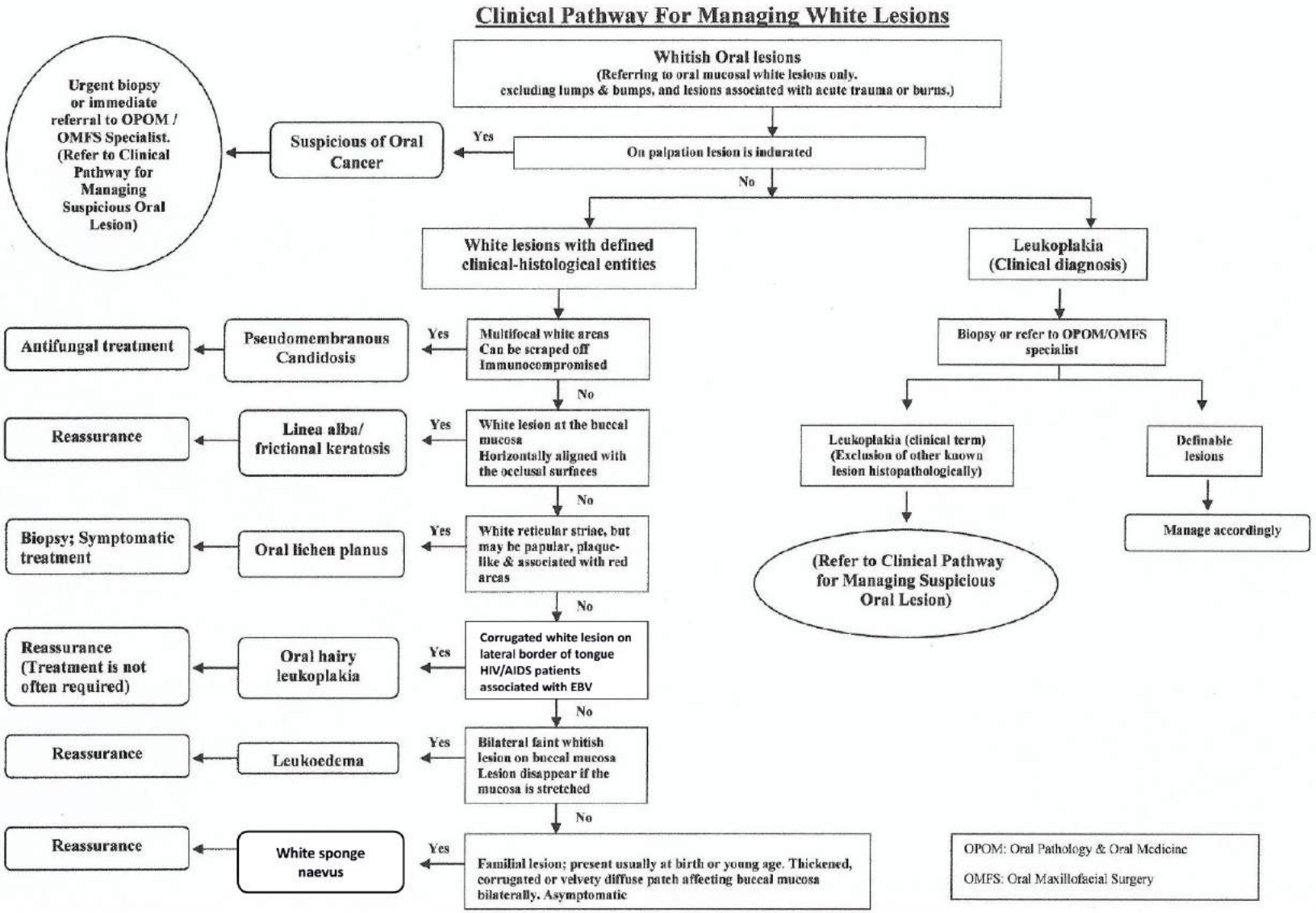
Aetiology: Most metastases to the jaw originate from the lungs, breast, prostate, thyroid, kidneys and gastrointestinal malignancies via lymphatic or haematogenous spread.

Treatment: Referral to the attending primary medical team for management.

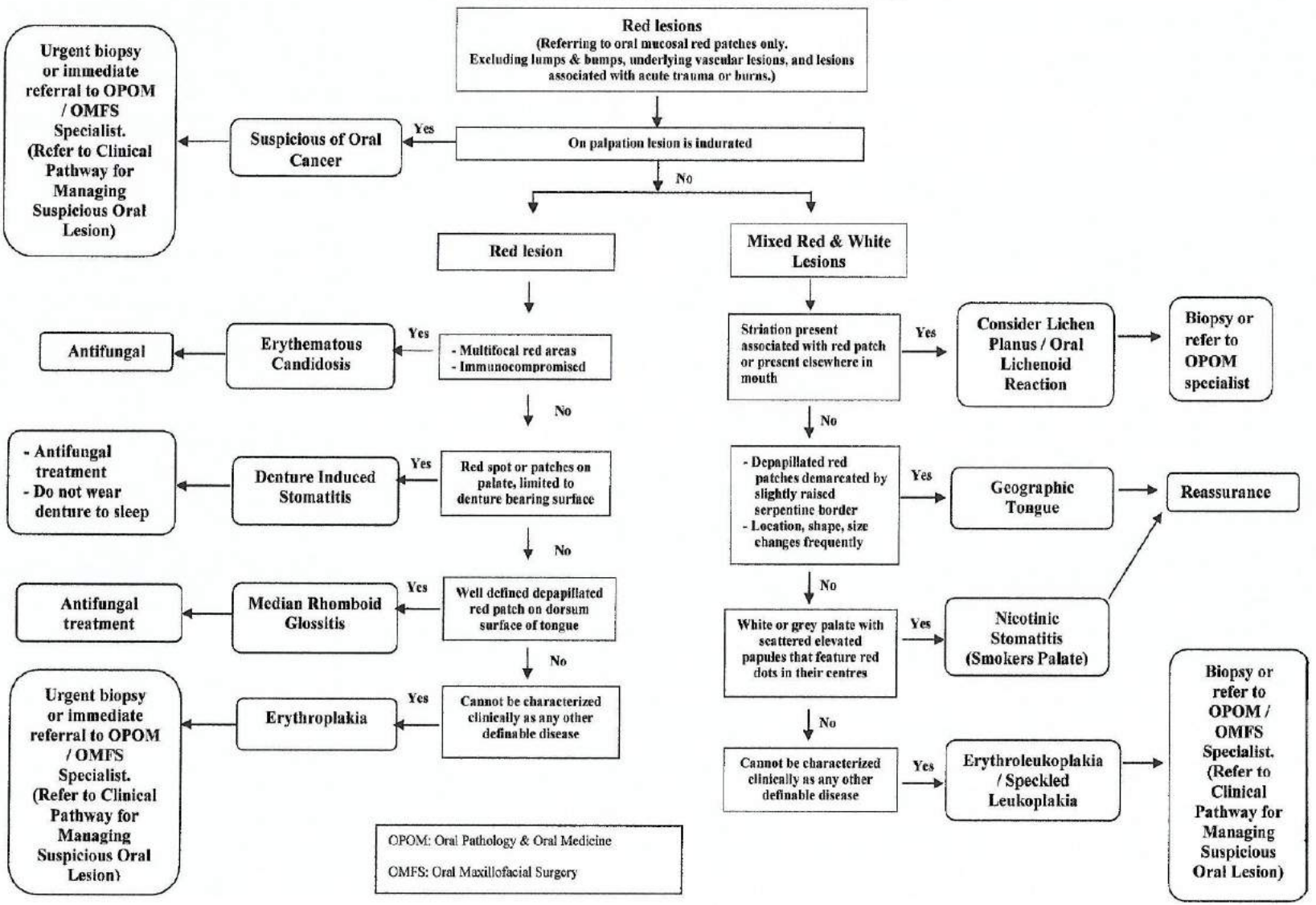
Prognosis: Presence of distant metastatic spread to the jaw is a sign of poor prognosis.

Differential Diagnosis: Squamous cell carcinoma, hyperplastic gingival swellings.

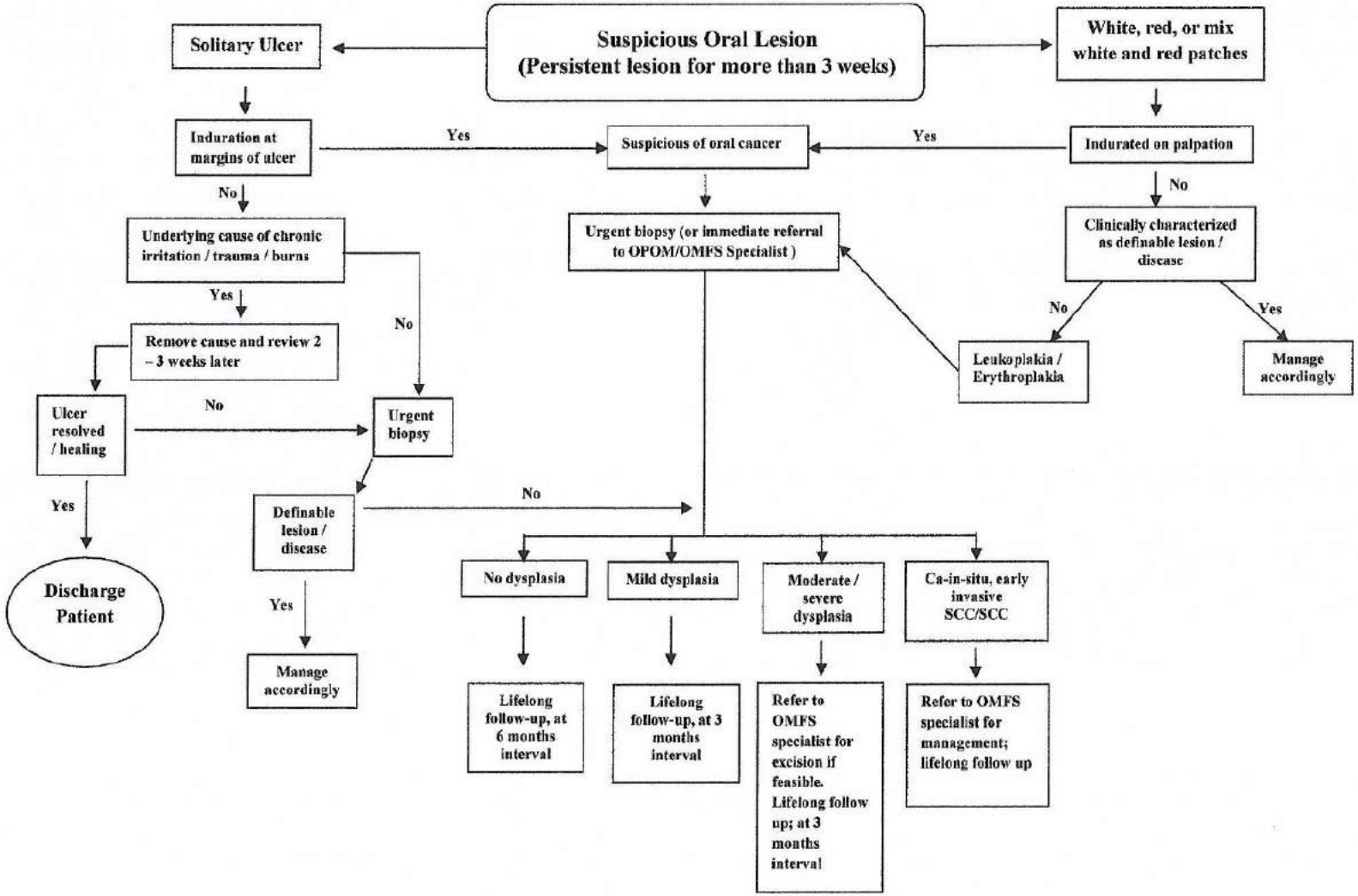
3.0 SECTION 3
CLINICAL PATHWAYS FOR MANAGING:
WHITE LESIONS
RED LESIONS
SUSPICIOUS ORAL LESIONS


Clinical Pathway For Managing White Lesions
Whitish Oral lesions
(Referring to oral mucosal white lesions only.
excluding lumps & bumps, and lesions associated with acute trauma or burns.)
On palpation lesion is indurated
Yes
No
Suspicious of Oral Cancer
Urgent biopsy or immediate referral to OPOM / OMFS Specialist. (Refer to Clinical Pathway for Managing Suspicious Oral Lesion)
White lesions with defined clinical-histological entities
Leukoplakia (Clinical diagnosis)
Multifocal white areas
Can be scraped off
Immunocompromised
Pseudomembranous Candidosis
Antifungal treatment
White lesion at the buccal mucosa
Horizontally aligned with the occlusal surfaces
Linea alba/ frictional keratosis
Reassurance
White reticular striae, but may be papular, plaque-like & associated with red areas
Oral lichen planus
Biopsy; Symptomatic treatment
Corrugated white lesion on lateral border of tongue
HIV/AIDS patients
associated with EBV
Oral hairy leukoplakia
Reassurance (Treatment is not often required)
Bilateral faint whitish lesion on buccal mucosa
Lesion disappear if the mucosa is stretched
Leukoedema
Reassurance
Familial lesion; present usually at birth or young age. Thickened, corrugated or velvety diffuse patch affecting buccal mucosa bilaterally. Asymptomatic
White sponge naevus
Reassurance
Biopsy or refer to OPOM/OMFS specialist
Leukoplakia (clinical term)
(Exclusion of other known lesion histopathologically)
Definable lesions
Manage accordingly
(Refer to Clinical Pathway for Managing Suspicious Oral Lesion)
OPOM: Oral Pathology & Oral Medicine
OMFS: Oral Maxillofacial Surgery

Clinical Pathway For Managing Red Lesions



Clinical Pathway for Managing Suspicious Oral Lesions



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