A clinical approach to white patches in the mouth

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White patches are common findings in the oral cavity and may affect any surface.

Such lesions are often an incidental finding on routine examination. Depending on the underlying aetiology, however, symptoms may or may not be present. White patches may be isolated or involve multiple areas and have variable presentations including linear patterns, plaque like lesions, diffuse patches and mixed white and erythematous areas.

Lesions appear white in the oral cavity due to the constant bathing of hyperkerototic tissue in saliva, analogous to the palms of the hands and soles of the feet when immersed in water for long periods. These areas may be benign or malignant or have malignant potential. Therefore it is important to investigate the lesion with a thorough history, examination and the appropriate investigations. This article briefly reviews common lesions which may present as a white patch in the oral cavity and their management.

Trauma and friction

White lesions due to trauma are most often due to physical insult but occasionally may follow chemical irritation by ingestion or application of caustic substances.

Hyperkeratosis typically results from a chronic insult such as friction from a rough dental restoration or chronic and habitual cheek or lip biting. This tends to be more insidious in nature and the patient may not present unless the area becomes symptomatic or infected.

Management involves identifying a cause of injury, addressing any underlying or associated behaviours, good oral hygiene and follow up in two weeks.

If there is a suspicion that dental restorations, tooth tissue or dental prostheses are the causative factors then a referral a general dental practitioner is appropriate.

We do not support the use of standard commercial mouthwashes for these lesions as the supposed antiseptic benefits are questionable. Further-

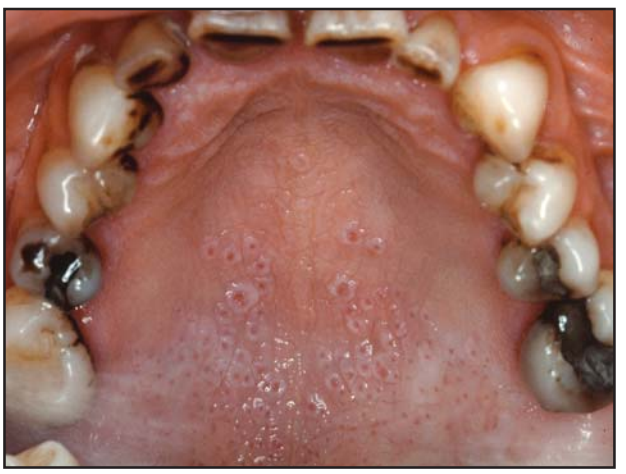

Figure 1. Nicotinic stomatitis showing characteristic distribution of hyperkeratosis on the palatal mucosa punctuated by inflamed orifices of the minor salivary glands.

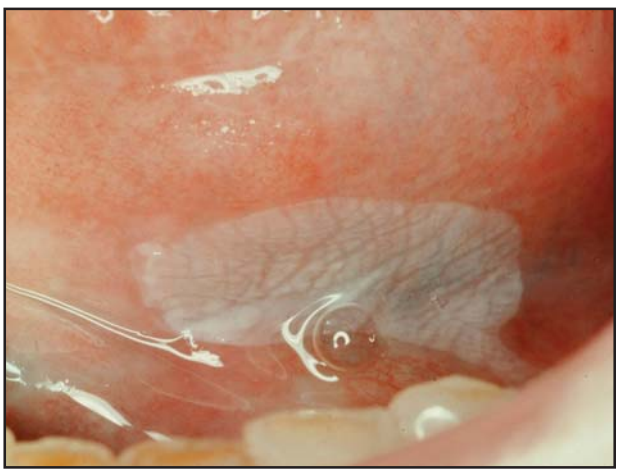

Figure 2. Leukoplakia on the ventral surface of the tongue. The lesion is flat and homogenous with a dense white appearance.

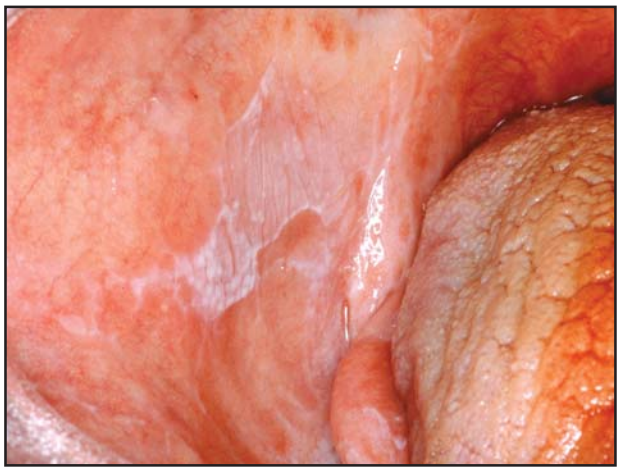

Figure 3. Leukoplakia involving the buccal mucosa and anterior fauces. This lesion is more diffuse in nature and less homogenous in the distribution of hyperkeratosis.

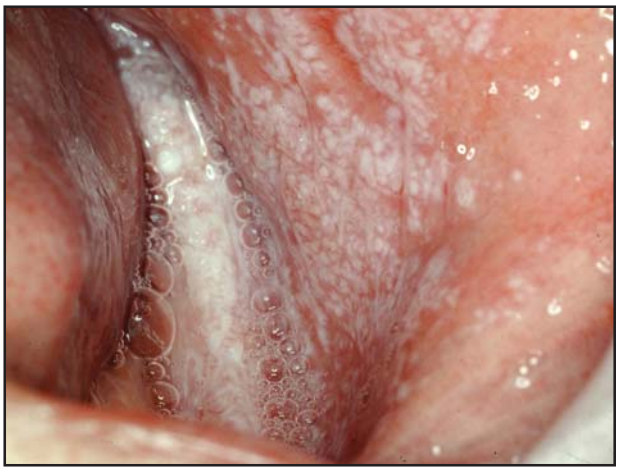

Figure 4. Leukoplakia of the buccal mucosa and posterior alveolar ridge. This presentation is more patchy on the buccal mucosa and less plaque-like. The alveolar ridge appears to have a thick layer of hyperkeratosis.

more, the majority contain around 12% alcohol, which would hardly be conducive to mucosal healing.

Tobacco-related

A common form of tobacco-related white patch in the oral cavity is nicotinic stomatitis or 'smoker's palate'.

This is attributed to pipe-smoking in particular but can occur with any form of tobacco smoking. The hard palate is characteristically affected with a widespread white appearance punctuated by red spots that represent the orifices of irritated minor salivary glands. The soft palate may also be involved along with the palatal gingivae around the maxillary dentition. This appearance is due to chronic irritation of the mucosa producing a hyperkeratosis. The lesion itself is essentially benign.

Another form of irritative hyperkeratosis not commonly seen in New Zealand is that associated with chewing tobacco. The predominant sites are the buccal vestibules or the vestibule of the lower lip where the tobacco is held.

The management of patients with these lesions is difficult, as it must address life-style changes and education.

The obvious recommendation is the cessation of tobacco usage, making the patient aware of the overall health benefits as well as decreasing

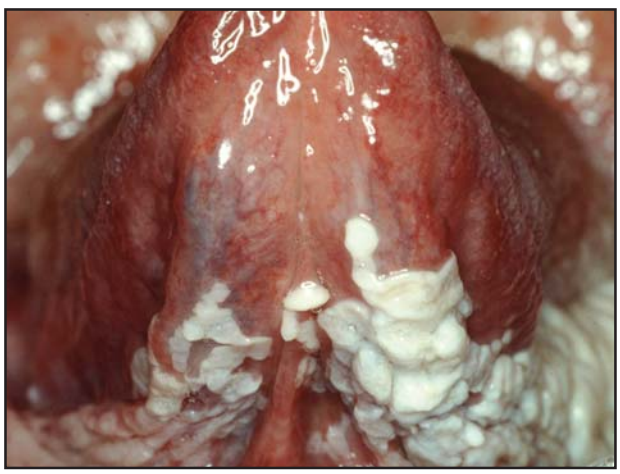

Figure 5. Leukoplakia of the ventral tongue and floor of mouth. On initial observation an impression of acute pseudomembranous candidosis can easily be confused. However, the white areas were unable to be wiped off and is a good example of gross leukoplakia. There was minimal epithelial dysplasia histologically.

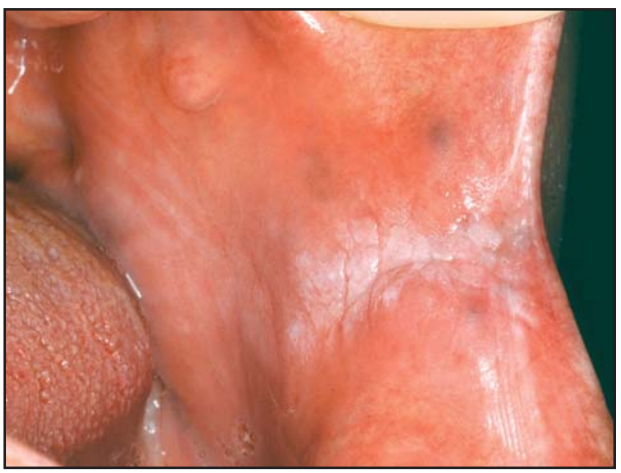

Figure 6. White lesion in the internal commissure of the buccal mucosa. Differential diagnoses include chronic cheek biting or a leukoplakia. Moderately severe dysplasia was present despite the inocuous appearance.

the potential for oral malignancy. Areas that appear ulcerated, roughened or erythematous should be viewed with heightened suspicion and referred for biopsy.

A logical choice of referral is to an oral and maxillofacial surgeon who is experienced and comfortable dealing with intra-oral lesions. They would also possess appropriate equipment, good lighting and surgical assistance.

Candidosis

Candida is a commensal organism often present in the oral cavity and is of no significance. When disturbances in the micro-flora occur, for example after administration of a broad spectrum antibiotic, candida may proliferate opportunistically. It may also infest the mucosa in the presence of chronic damage or a compromised immune response. Candidosis has many different forms and does not accurately represent a disease in its own right. When it presents, it is appropriate to seek an underlying aetiology.

Thrush (pseudomembranous candidosis) is particularly common in infants, in the elderly and immunocompromised patients such as those with HIV, diabetes mellitus or medication induced immunosuppression. Patients on long-term corticosteroids or chronic broad spectrum antibiotics are also particularly vulnerable. A characteristic pattern may develop in individuals using steroid inhalers for control of asthma, where candida colonises the soft palate and dorsum of the tongue.

Thrush presents as a creamy, white plaque that is easily wiped off, leaving an erythematous base. Although it may appear anywhere in the oral cavity, it is predominantly seen on the buccal mucosa, palate and tongue. The clinical picture invariably gives the diagnosis but further investigations may include taking a smear for candidal hyphae: a swab is of negligible value as over half of the population will harbour candida in their mouth as part of the normal commensal flora.

Management should address the predisposing factor(s), topical antifungals can be considered.

Chronic mucocutaneous candidosis is rare and is associated with a possible genetically based immune defect predisposing these individuals to candidal infections not only in the mouth, but also involving the nails and skin.

The oral presentation is similar to thrush but perhaps slightly more diffuse. These white patches are generally more persistent and may ne-

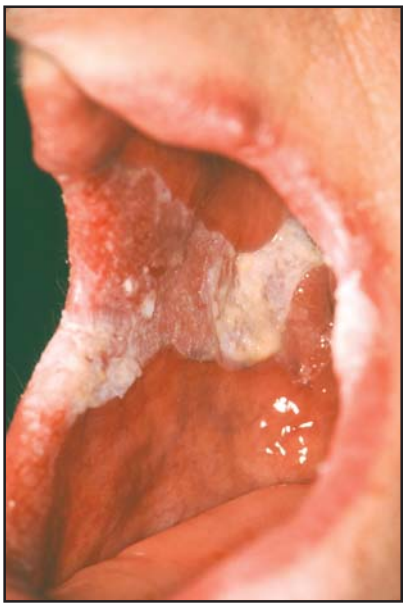

Figure 7. Mixed white and red lesion of the internal commissure of the buccal mucosa. This is squamous carcinoma and contrasts with the otherwise less aggressive looking lesion as seen in Figure 6 in a similar area.

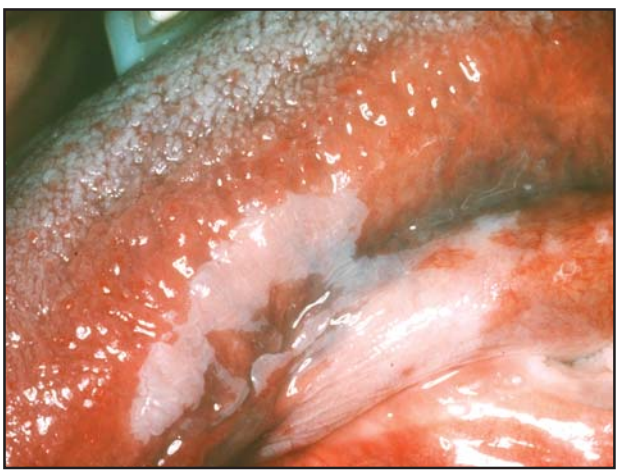

Figure 8. Leukoplakia of the lateral border and ventral areas of the tongue. The lesion is homogenous in nature although the anatomical site gives a higher risk of it being dysplastic or malignant. There are erythematous areas associated with the posterior ventral surface and floor of the mouth, which would further heighten clinical suspicion.

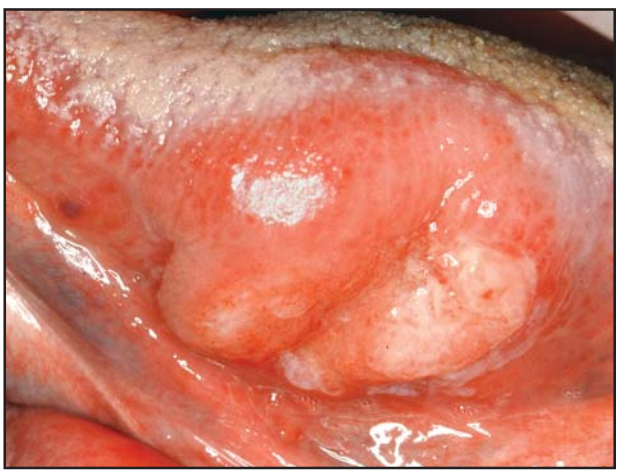

Figure 9. Squamous cell carcinoma of the tongue. Note the raised indurated borders of the lesion and distortion of the local tissue due to tumour invasion.

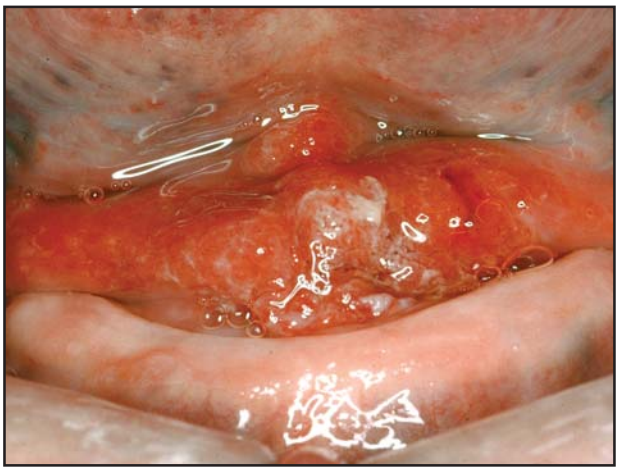

Figure 10. Squamous cell carcinoma of the anterior floor of mouth. The area is raised and would be firm on palpation.

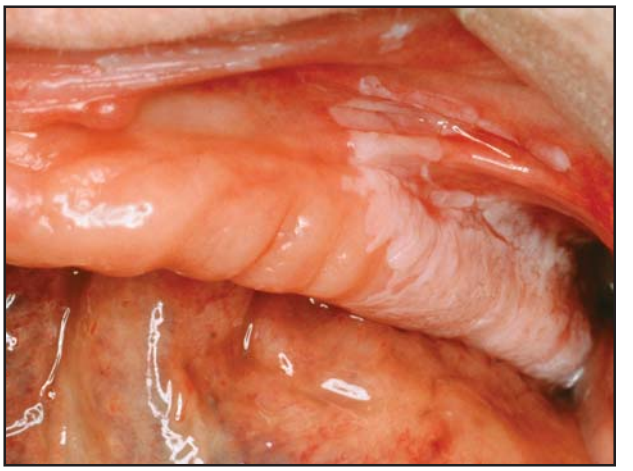

Figure 11. Leukoplakia involving the lateral aspect of the upper alveolar ridge. There is an erythematous area posteriorly that was highly dysplastic.

cessitate systemic antifugal treatment after topical therapy has failed.

Lichen planus

(see NZFP October 2001)

Lichen planus is a mucocutaneous disorder of unknown aetiology but is attributed to an underlying immunologic process. Associations with other autoimmune processes such as primary biliary cirrhosis, graft versus host disease, dermatomyositis and chronic active hepatitis have been described giving support for an autoimmune pathogenesis. A small number of individuals present with a lichenoid reaction due to a drug reaction or reactive process to certain dental materials such as the mercury in amalgam restorations. The presentation of a lichenoid reaction is clinically indistinguishable from lichen planus.

Lichen planus of the oral cavity may present as white striae, white plaques, erythematous atrophic areas, ulcerated areas or desquamative gingivitis.

The most common form of lichen is the striated pattern that may vary from simple linear patterns to intricate lacework or reticular networks. This usually occurs on the buccal mucosal surfaces or lateral margins of the tongue but occasionally can be found on any other surface. Next most common is the ulcerative form of oral lichen planus which present in similar areas and is characterised by a central area of ulceration with a white periphery that may have a reticular pattern associated with it.

The plaque form of lichen is less common and predominantly presents on the buccal mucosa or the dorsum of the tongue. Tongue lesions have a different character to them when compared to candidal overgrowth or poor oral hygiene. There is an irregular margin resembling the striated form of lichen and cannot be wiped off.

Oral lesions of lichen may be asymptomatic or present with a burning sensation. The ulcerative form can cause considerable discomfort.

Management should include specialist referral for confirmation and a biopsy if necessary. Lichen is thought to carry a malignant potential although the magnitude is unclear, probably less than 1%.

Leukoplakia

The word leukoplakia literally means 'white plaque'. It is defined by the World Health Organisation as *'a white patch or plaque that cannot be characterised clinically or pathologically as any other disease'*.

Leukoplakia may occur at any site of the oral cavity. The lesion may be totally white or have erythematous areas associated with it – known

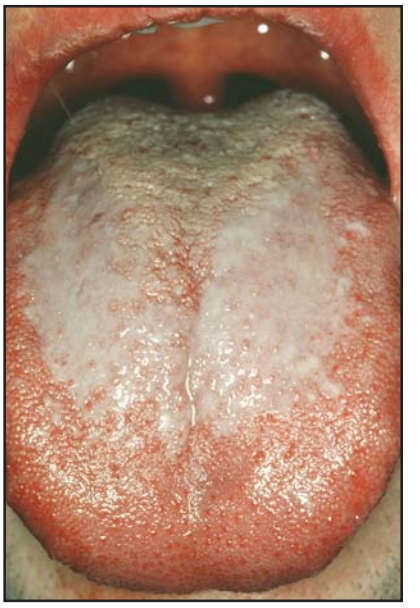

Figure 12. Extensive white lesion involving the dorsum of the tongue. This lesion could not be wiped off and was found to be lichen planus. Note the irregular margins of the lesion and the comparable appearance to the reticular form of lichen.

as speckled leukoplakia. Totally white lesions may be flat and plaque-like or have variations in thickness and texture such as corrugations or hypertrophic areas. Their size is also variable, ranging from under 1cm^2 to involving most of the mouth.

The aetiology of leukoplakia is unknown although tobacco and alcohol usage are associated with higher incidences of developing leukoplakia. UV exposure to the lips is another predisposing factor.

There is a male preponderance and the incidence increases after the age of 40 years. The lesions are invariably asymptomatic.

Leukoplakia is considered as a premalignant condition, however not all leukoplakias show epithelial dysplasia and there is no prognostic index as to whether a leukoplakia will undergo malignant transformation into a squamous carcinoma or not.

Generally speaking, those leukoplakias which exhibit epithelial dysplasia may have a 5–25% overall chance of undergoing malignant transformation, depending on the site. Those on the ventral surface of the tongue and floor of the mouth are considered to have the highest potential.

The speckled form of leukoplakia may have an even higher incidence of malignant transformation.

To compound the uncertainty, the time period associated with malignant transformation is also variable ranging from months to several decades. However, long standing leukoplakias have a greater risk of malignancy.

Clinical assessment is totally inadequate for determining the degree of dysplasia and smears are also of no value. Biopsy is mandatory at the earliest opportunity and may involve repeated biopsies over time due to the unpredictable nature of the lesion.

Life style changes involving the modification or cessation of tobacco and alcohol usage is most important and the use of sunscreens and wide brimmed hats to prevent further UV associated damage to the lips is prudent.

Once histopathological assessment of the epithelium has been performed, the further management of the condition depends on the presence or absence of epithelial dysplasia.

Dysplastic lesions should be excised surgically or with CO_2 laser whereas non-dysplastic leukoplakias may be managed conservatively with regular follow up and as mentioned earlier, repeated biopsies over time, especially if there is a change in appearance of a long standing lesion. However, removal is the preferred option whenever possible.

Not all patients or areas of the mouth are amenable to surgery and many advances have been made in medical therapy. Topical agents such as retinoids and cytotoxic drugs have been investigated and show some promise. However, further research and longitudinal data must be available before widespread acceptance is gained.

Another entity bears mention due to its unusual nature: hairy leukoplakia.

Hairy leukoplakia has become one of the most specific oral manifestations of HIV infection and presents as corrugated or hairy white patches usually affecting the lateral borders of the tongue and occasionally the dorsum of the tongue.

It is currently thought to be caused by repeated direct infection of the superficial epithelial cells by Epstein-Barr virus and not reactivation of the latent virus as previously believed. Unlike other leukoplakias, HIV related hairy leukoplakia is not generally though to have malignant potential.

Key Points

- Lesions appear white in the oral cavity due to the constant bathing of hyperkerototic tissue in saliva, analogous to the palms of the hands and soles of the feet when immersed in water for long periods.
- Candidosis has many different forms and does not accurately represent a disease in its own right.
- The aetiology of leukoplakia is unknown although tobacco and alcohol usage are associated with higher incidences of developing leukoplakia.
- There is no place for blindly prescribing antifungals or the various commercial mouthwashes.

Recommendations

White lesions of the oral cavity are common and often incidental findings on examination. Their causative factors are varied and range from local trauma to systemic illness.

Due to a premalignant potential in some of these lesions, white patches should be investigated fully and not be treated superficially.

There is no place for blindly prescribing antifungals or the various commercial mouthwashes.

A thorough history and examination should be performed including regional lymph nodes.

Causative factor(s) should be identified and underlying issues addressed. The patient should see a general dental practitioner if local factors are suspected.

Referral for biopsy of the lesion is appropriate to a clinican who deals with intra-oral conditions regularly and has the necessary equipment and setting for the biopsy procedure.