What is the Risk of a White or Red Lesion in the Mouth Being Malignant?

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The oral mucosa, like any other epithelial surface, can vary in appearance of health. In disease the variation from the normal can sometimes be quite subtle or very obvious. Recognising these differences is nowhere more important than in those changes that define a premalignant or malignant lesion. The majority of premaligant lesions will most likely be seen by a patient's regular dentist on routine examination. These lesions will often have few symptoms, but if they occur the patient may complain of an alteration in surface texture or soreness. If patients present to their doctor with symptoms the referral may be to ENT rather than oral physicians or oral and maxillofacial surgeons (OMFS) for assessment. It is necessary therefore to be able to recognise mucosal change and even more importantly the significance of that change.

Premalignant lesions

A premalignant lesion is defined as a benign, morphologically altered tissue that has a greater risk of malignant transformation. Most such lesions present as white, red and white or red lesions.

Leukoplakia

Leukoplakia is defined by the World Health Organisation (WHO) as a white patch that cannot be rubbed off and cannot be characterised clinically or histologically as any other disease nor can it be associated with any physical or chemical causative agent except the use of tobacco. It is thus a clinical description, a diagnosis of exclusion and where the histology is hyperkeratosis +/- epithelial dysplasia.

The presentation of such leukoplakic lesions may be variable. Figure 1 shows a homogenous, thickly hyperkeratotic patch to the left of the midline in the floor of mouth. In Figure 2 the white patch is bilateral in the floor of mouth where anteriorly it is homogeneous and folded in appearance, but more posteriorly it is less well defined. Notice also the hyperkeratosis within the labial mucosa of the lower lip which is often seen where there has been a long standing tobacco habit. Hyperkeratosis within the floor of mouth is associated with tobacco. This is an anatomical site where saliva pools and where tobacco products in solution will lie. The site is of especial risk of malignant change and when alcohol consumption is added to the tobacco habit the risk is magnified. The risk of malignant change in the floor of the mouth is at least 25% and may be as high as 40%, whereas the risk of malignant change in leukoplakic lesions elsewhere in the oral mucosa is between 4 and 6%. Any such white lesion in the floor of mouth requires biopsy and the histology will inform the treatment. All patients need counselling in smoking cessation.

In Figure 3 the leukoplakia is on the right lateral border of the tongue. Anteriorly it is thickly white; on the ventral surface it is thinner, but still uniform in appearance. At the posterior lateral surface there appears to be linear area of erythema. This within an area of leukoplakia is of concern as it usually denotes increased cellular activity, also the more posterior the lesion the poorer the prognosis. Squamous cell carcinoma represents 90% of all oral malignancies and the lateral margin of the tongue is a site of high predilection. Biopsy of this erythematous area within the white patch is essential.

Speckled leukoplakia

Red and white lesions are often described as speckled leukoplakias. In Figure 4 the area shown on the buccal mucosa is erythematous and within it there appears to be white dots of varying size but of uniform density. This is classic specked leukoplakia and should be considered a high risk lesion. Biopsy of the speckled area and the erythematous region will show dysplastic change. Figure 5 is also of speckled leukoplakia, but here the lesion which lies on the alveolar and buccal mucosa has a high degree of keratosis with erythema both within and around it. Note also the continuation of the lesion within the lingual sulcus adjacent to the posterior edge of the tongue. This area, often known as Coffin's corner, is one which like the anterior floor of mouth is where saliva containing tobacco products pool and thus of necessity is high risk of malignant change (25%-40%). Biopsy from both areas and EAU is required.

Figures on facing page

1: *Leukoplakia in floor of mouth.*
2: *Leukoplakia of the floor of mouth and labial mucosa.*
3: *Leukoplakia of the right lateral border of the tongue.*
4: *Speckled leukoplakia of buccal mucosa.*
5: *Speckled leukoplakia of alveolar and buccal mucosa.*
6: *Erythroplakia of the buccal mucosa.*
7: *Erythroplakia of right soft palate and fauces.*
8: *Chronic hyperplastic candidosis of the right commissure.*
9: *Chronic hyperplastic candidosis of the left commissure.*
10: *Oral submucous fibrosis.*


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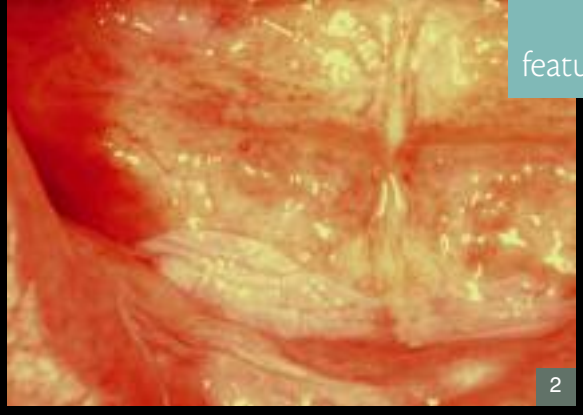
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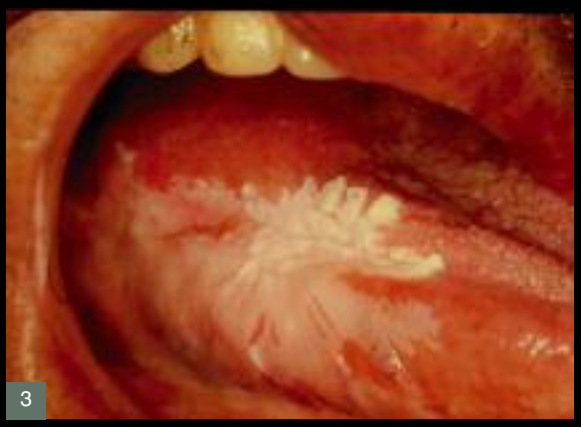
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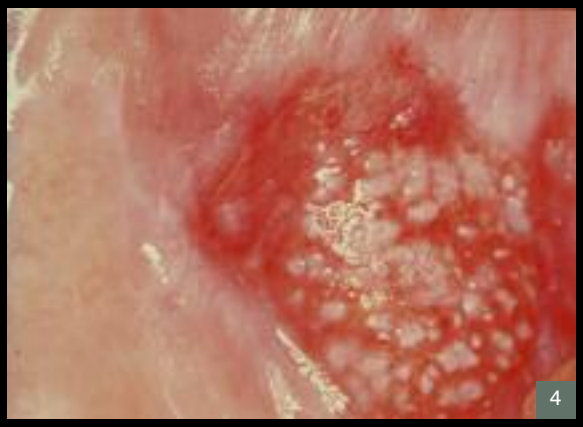
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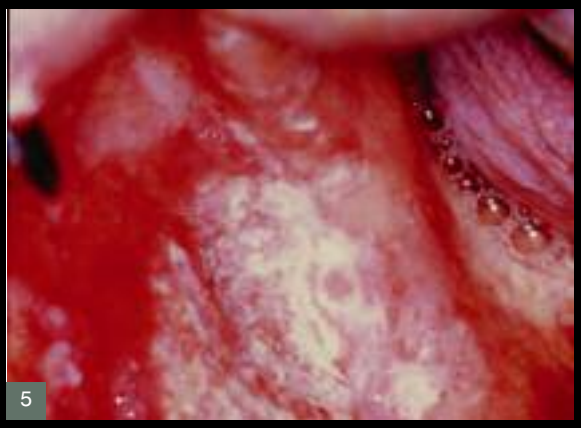
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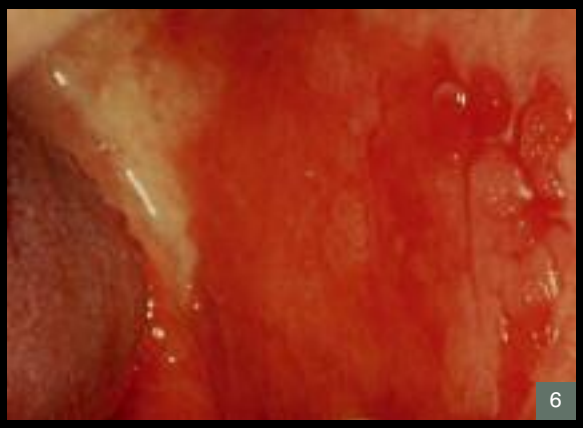
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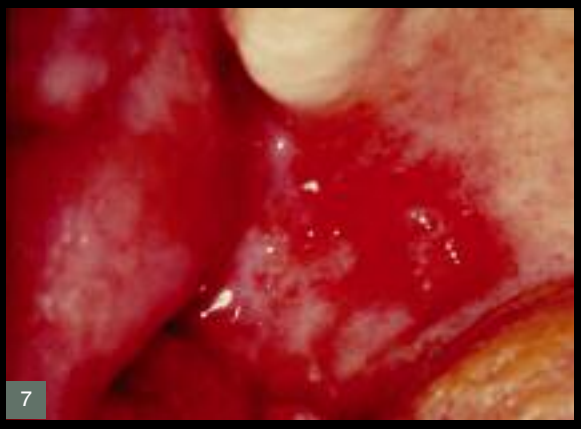
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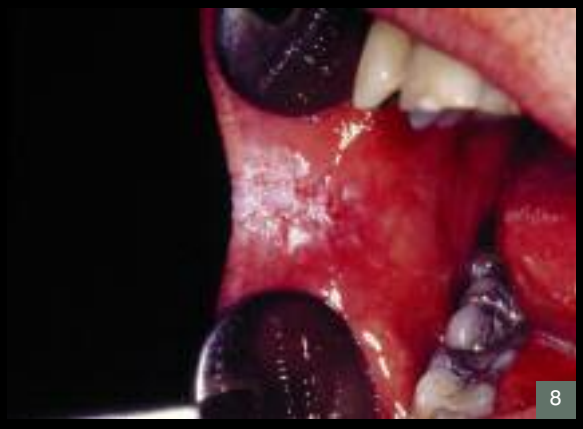
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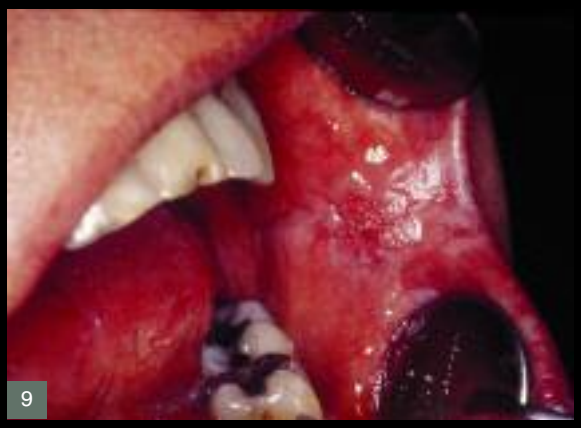
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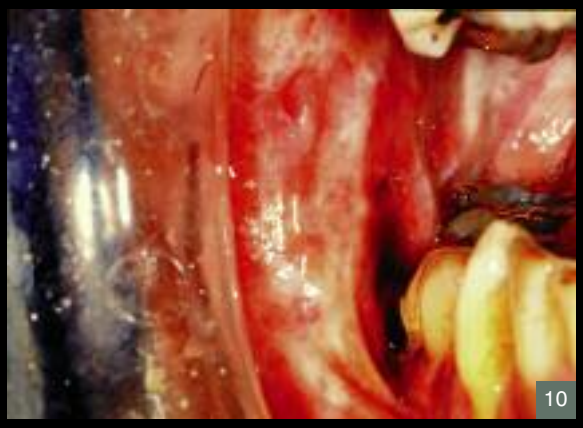
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Table 1: Sites of risk of malignant change

↓	Lateral margins of tongue
↓	Floor of mouth
↓	Soft palate and fauces
↓	Gingiva
↓	Buccal mucosa
↓	Labial mucosa
↓	Hard palate

Table 2: Oral premalignant conditions

Chronic hyperplastic candidosis
Submucous fibrosis
Actinic cheilitis
Mucosal lichen planus
Discoid lupus erythematosis
Paterson-Kelly / Plummer-Vincent Syndrome
Sjögren's syndrome

Erythroplakia

The WHO defines erythroplakia as a red patch that cannot be characterised clinically or histologically as due to any other condition. It, like leukoplakia, is a diagnosis of exclusion.

The incidence of leukoplakia in Western Europe is around 3%-4% of oral lesions. The prevalence of erythroplakia is unknown but it occurs much less frequently than leukoplakia. It has a much greater potential for malignant change than leukoplakia and on biopsy will commonly show severe epithelial dysplasia or carcinoma in situ. It should be considered malignant unless proven otherwise.

Figure 6 shows a classic area of erythroplakia of the buccal mucosa. The lesion is uniformly red and has a smooth almost velvety surface. The adjacent mucosa appears unaffected. In Figure 7 the erythema is affecting the right soft palate and fauces. This is a common site for erythroplakic lesions and unlike leukoplakia may occur unrelated to tobacco and alcohol usage.

The risk of a red lesion becoming malignant is far greater than speckled leukoplakia and greater indeed than a purely white lesion. In all cases of leukoplakia the site of the lesion also informs the level of risk of malignant transformation. The sites of greatest risk (in descending order of frequency) are shown in Table 1.

A number of other factors help define increased malignant potential when assessing leukoplakic lesions. A nodular surface appears to be more problematic than one that is homogeneous and where candida is present within the tissue this too increases the risk of malignant change.

Premalignant conditions

A number of premalignant conditions occur related to the mouth. These are listed in Table 2.

Of these premalignant conditions a number show a high rate of malignant change and it is the first two within Table 2 which are the most significant.

Chronic hyperplastic candidosis

This condition classically occurs within the commissures, is triangular in shape with the apex pointing backwards. Commonly it presents as speckled leukoplakia and Figures 8 and 9 show this affecting the left and right commissure in a man of middle life and who had a significant tobacco and alcohol habit over many years. The condition is usually bilateral, but may present, as in this case, slightly differently on each site. It is advisable to biopsy each site. The lesion on the left is much more speckled than that on the right and the histology reflected this in that the right commissure was severe epithelial dysplasia, whilst that on the left was mild to moderate dysplasia. Stopping smoking and reduction in alcohol intake is the lifestyle change required and as the incidence of malignant change is 25 to 50% removal of the altered tissue using laser ablation is the treatment of choice. Review within a specialist clinic is appropriate for at least 3-5 years and will depend on the life style change being maintained.

Submucous fibrosis

This condition occurs within populations of Indian and South East Asian origin, but may also be seen in Europeans who have adopted their dietary lifestyle and habits. The condition is probably genetically determined as not all will be affected. The aetiological factor is the areca nut which is incorporated within pan and chewed or chewed on its own. The generation of the condition may also be influenced by lack of certain dietary factors linked to religious practices, for example a vegan diet. Figure 10 shows the classic white appearance of the mucosa as a result of fibrosis below the epithelial surface. This tissue becomes rigid and the mouth opening is restricted. Palpation of the buccal mucosa identifies vertical banding not unlike that of systemic sclerosis. Notice also, in the figure, the staining of the teeth which signifies a concurrent tobacco habit. This condition is highly premalignant. Biopsy of the mucosa is required, but it is not easy as the wound will gape as the fibrotic tissue retracts on incision. It is essential that dietary habits involving the areca nut are stopped and correction of any haematinic deficiency made together with smoking cessation if it occurs. Long-term follow-up is a prerequisite of management.

All cases involving premalignant lesions and conditions where follow-up is required because of biopsy proven dysplasia require careful record keeping with clinical photographs of the initial lesions, following successful treatment and if further clinical deterioration should occur. Once lifestyle habits have been successfully changed and no recurrence has occurred discharge is indicated. By and large the dentist is the person who will probably review these patients for life and will carefully observe changes in behaviour requiring re-referral. They, because of their training in dental school, will refer to oral physicians or oral and maxillofacial surgeons for management. Currently this lifetime of regular dental care may no longer be a guarantee for some within the community and thus education of all medical practitioners in accurate oral examination is essential. ■

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