

3. LESIONS LESS LIKELY TO BECOME CANCERS

Introduction

Apart from being causally related to oral cancer and precancerous lesions, tobacco use is also responsible for an array of oral lesions that are not considered "precancerous". Of course, this fact does not mean that oral cancer will not develop amongst individuals with "other tobacco-related lesions"; only that the excess risk has not been demonstrated as significant. If clinicians themselves are unfamiliar with such lesions, patients may be given incorrect information possibly leading to cancer phobia. A thorough knowledge and understanding of "other tobacco-related lesions" is obviously essential for differential diagnosis as well as for patient management.

PRELEUKOPLAKIA

Preleukoplakia is a definite entity with specific diagnostic criteria and behavior. It is characterized as a low-grade or mild reaction of the mucosa, appearing as a gray or grayish-white, but never completely white, lesion, with a slight lobular pattern and indistinct borders blending into an adjacent normal mucosa (Fig. 1). The prevalence of preleukoplakia in India varies from 0.5% to 4.1%. Preleukoplakia is strongly associated with tobacco smoking and is considered as a

precursor to leukoplakia. Over a 10-year observation period, 15% of the preleukoplakias progressed to leukoplakia and 0.4% to oral cancer. Malignant transformation can occur directly from preleukoplakia, or the lesion may develop first into leukoplakia and then progress to cancer.

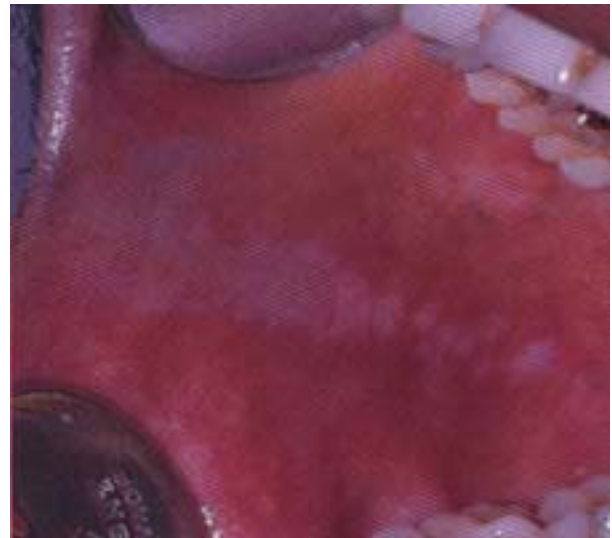


Fig. 1. Preleukoplakia in the right commissure and the buccal mucosa of a bidi smoker. Note the grayish appearance of the lesion and indistinct borders.

LEUKOEDEMA

Leukoedema is a chronic mucosal condition in which the oral mucosa has a gray, opaque

appearance as though a grayish film were hanging over it (Fig. 2). When the mucosa is stretched, leukoedema disappears, only to reappear when it is relaxed. This condition is mostly observed among *bidi* smokers. The prevalence of leukoedema in India varies from 0.02% to 0.3%. Unlike leukoplakia leukoedema does not present a keratinized surface. It develops due to the piling up of spongy cells. Over a 10-year observation period, 64% leukoedemas remained stationary and 36% regressed.

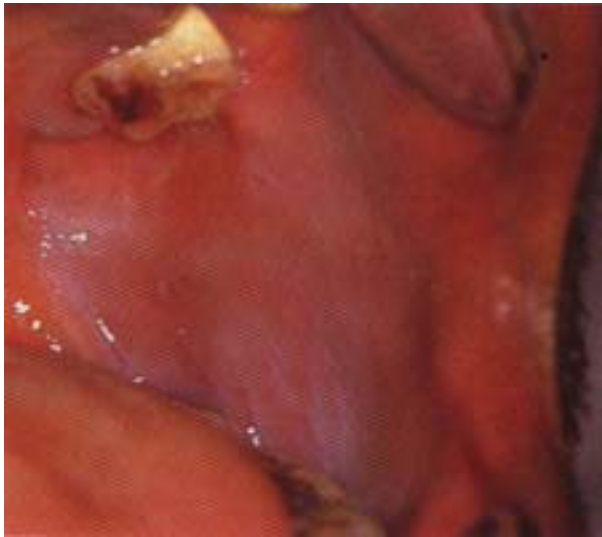


Fig. 2. Leukoedema in the left buccal mucosa of a young *bidi* smoker.

SMOKER'S PALATE

Smoker's palate, also known as leukokeratosis nicotina palati, is a common reaction of the palatal mucosa to smoking. This lesion consists of a diffused white palate with numerous excrescences having central red dots, corresponding to the orifices of the minor salivary glands (Fig. 3). The prevalence of smoker's palate in India varies from 0.4% to 9.5%.



Fig.3. Smoker's palate in a *bidi* smoker. Note the few excrescences with central red dots on a whitish palate.

In an early stage, smoker's palate may consist of a grayish palatal mucosa, either with a few (Fig.4) or without any



Fig.4. Smoker's palate that shows a grayish palate and excrescences in a *bidi* smoker.



Fig.5. Excrescences with a mosaic pattern on a grayish palatal mucosa in a conventional dhumti smoker from Goa.

excrescences. Protection of the palate against the smoke, or discontinuance of smoking, results in regression of smoker's palate.

Smoker's palate in a dhumti smoker : The intensity of smoker's palate depends on the extent and type of smoking. Smoker's palate associated with conventional dhumti smoking in Goa is relatively more intense (Fig. 5) as compared to the lesion associated with *bidi* smoking. Overall, smoker's palate remains stationary in 66% of the cases, and 34% regress spontaneously.

PALATAL ERYTHEMA

Palatal erythema is a common lesion strongly associated with *bidi* smoking. It consists of diffused erythema on the hard palate (Fig. 6), that occasionally extends to the soft palate. Because of the intense redness in most lesions, palatal erythema can be misdiagnosed as erythroplakia. Microscopically, however, palatal erythema shows an inflammatory reaction with superimposed candidal hyphae, sometimes with minimal epithelial dysplasia.

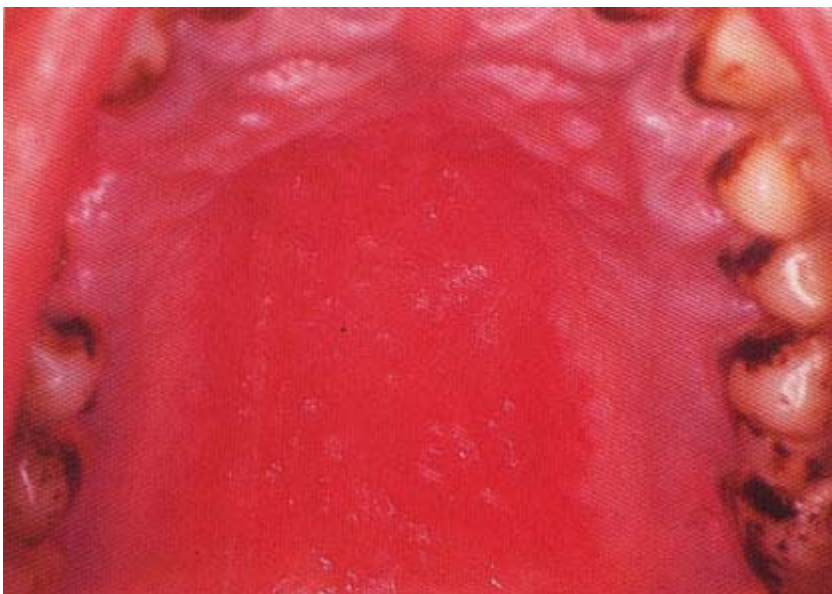


Fig.6. Palatal erythema in a *bidi* smoker. Note slight papillary hyperplasia.



Fig.7. A less intense palatal erythema, but with papillary hyperplasia in a moderate smoker.

Palatal erythema with papillary hyperplasia: Occasionally, the redness in a palatal erythema is less intense (Fig.7). Palatal erythema predominantly (87%) occurs among males,

consistent with the widespread smoking habit among men. About 25% of palatal erythemas are associated with central papillary atrophy of the tongue and bilateral commissural leukoplakias.



Fig.8. Papillary hyperplasia of the palate in a *bidi* smoker who never wore a denture.



Fig. 9. Denture stomatitis type III which is characterized by a hyperemic mucosa with a granular and nodular appearance. The granular hyperplasia is generally localized to the center of the palate. The patient was a nonsmoker.

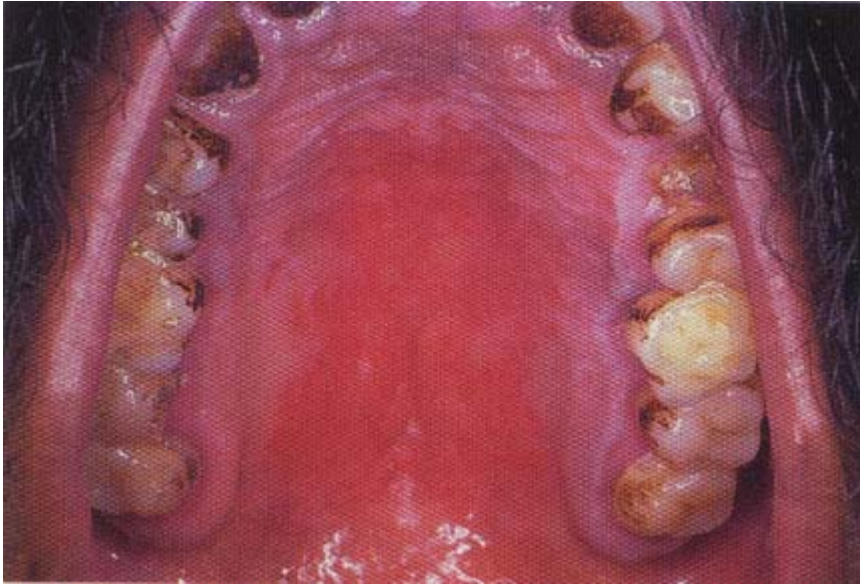


Fig.10. palatal erythema in a bidi smoker. Note the diffuse erythema of the palate in an individual who had a full compliment of teeth.

About 10% of individuals with palatal erythema also show papillary hyperplasia that consists of many red, ovoid or spherical, smooth nodules, 2-3 mm in size (Figs. 7 & 8). A similar lesion is seen among dentureusers who are nonsmokers and it is termed as chronic erythematous candidiasis or denture stomatitis (Fig. 9).

Natural history: Palatal erythema may remain stationary, regress spontaneously, or may be transient. Reduction or cessation of the smoking habit results in higher regression rates of palatal erythema (Figs. 10 & II). In a sample of 69 individuals with palatal erythema, who were subjected to tobacco cessation programs, the lesion regressed

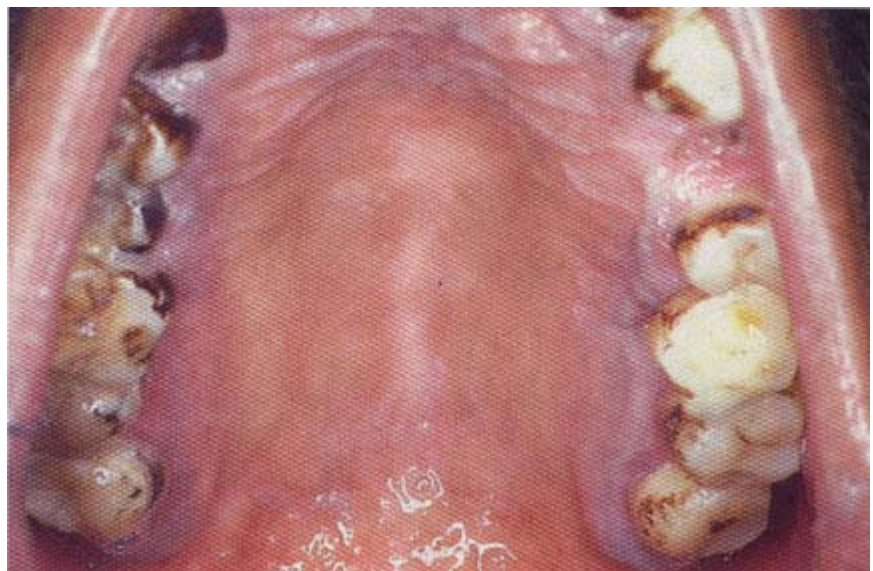


Fig.11. Regression of the palatal erythema shown in Fig. 10, following the disconuation of smoking. Note the loss of pigmentation.

spontaneously, i.e., without a change in tobacco use in 36%, and regressed more frequently, (75%), when the smoking habit was stopped or reduced substantially. The regression of the lesion was often marked by the loss of palatal pigmentation. Papillary hyperplasia, if present, however, does not regress.

CENTRAL PAPILLARY ATROPHY OF THE TONGUE

Central papillary atrophy of the tongue, also described as median rhomboid glossitis, consists of a well-defined, oval, pink area in the center of the dorsum of the tongue which is devoid of lingual papillae (Fig. 12). The lesions are generally smooth, but there may be a wide range in their size, shape and appearance. The prevalence of central papillary atrophy in India varies from 0.04%

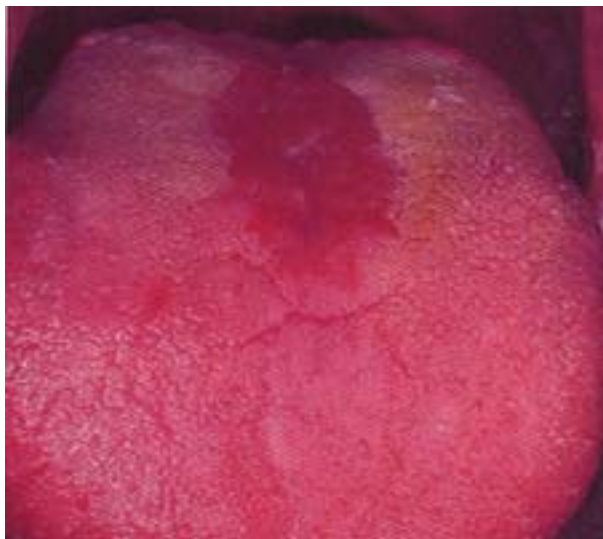


Fig. 12. A deep pink central papillary atrophy of the tongue.



Fig. 13. A rhomboid-shaped central papillary atrophy of the tongue with slightly lobulated and fissured surface.

to 0.1%. This lesion is strongly associated with bidi smoking; in a sample of 182 lesions, 82% occurred among bidi smokers and the rest in those who smoked and chewed tobacco. Nine of the 182 individuals with this lesion were women who smoked bidis.

Variation: The length of central papillary atrophy may vary from 0.8 to 5 cm and the breadth from 0.4 to 2.4 cm. The lesion may be oval, elongated, rhomboid, or irregular with smooth, fissured, or lobular surface (Fig. 13). Sometimes, the surface of the central papillary atrophy is strikingly lobular (Fig. 14) and this lobular appearance should be distinguished from the induration seen in oral cancer. About 69% of the central papillary atrophy lesions occur independently, 14% with palatal erythema, 8% with leukoplakia, and with palatal erythema, and commissural leukoplakia in 3% (triad). The remaining 6% occur along with other tobacco-related

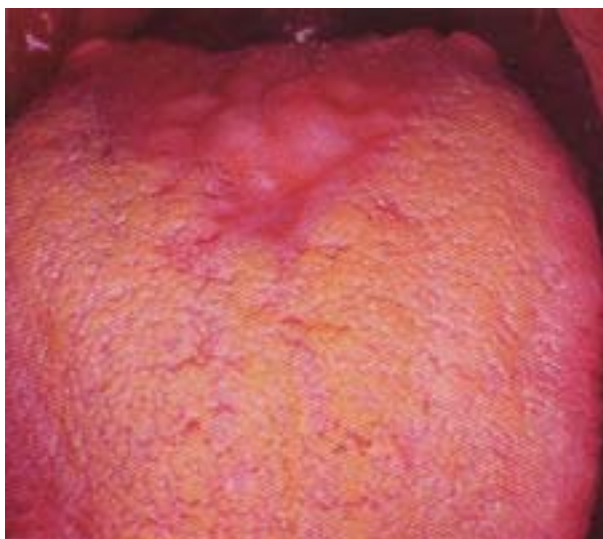


Fig. 14. Central papillary atrophy with a striking labular appearance.

lesions. About 95% of these lesions occur among men, generally in the age range of 35-44 years. Central papillary atrophy of the tongue seen in *bidi* smokers must

be distinguished from the atrophic candidiasis reported among people with human immunodeficiency viral infection (HIV).

Natural history: Central papillary atrophy may remain stationary, repapillate spontaneously, recur, or may be inconsistent in behavior. Reduction or cessation of smoking is associated with higher rates of repapillation (Figs. 15 & 16). For example, in a sample of 182 individuals with this lesion who were subjected to health education for stopping their tobacco-habits, 33% of the lesions repapillated spontaneously, and 64% repapillated when the smoking habit was reduced or stopped. On average, repapillation of central papillary atrophy occurs in a mean observation period of 1.1 years after discontinuation of the smoking habit, whereas, spontaneous repapillation may take a longer time (1.7 years).

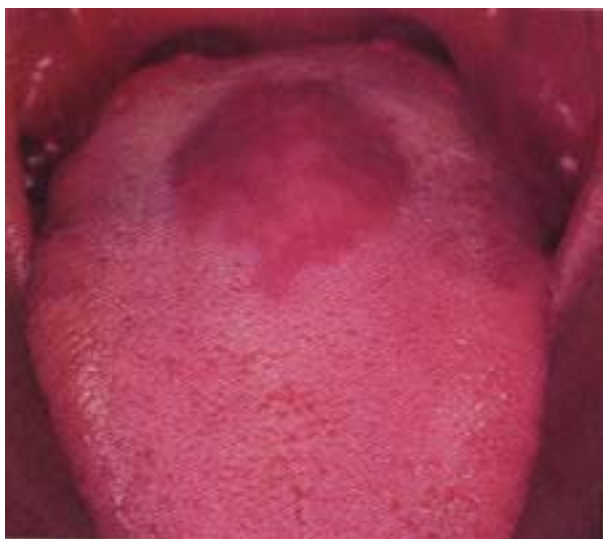


Fig. 15. A large, deep pink lesion in a female *bidi* smoker.



Fig. 16. The patient shown in Fig.15 discontinued the smoking habit and there was repapillation after eight months following the cessation of the smoking habit.

TOBACCO-LIME USER'S LESION

The habit of using a mixture of tobacco-lime (*khaini*) (see Appendix I) often produces a well-defined, thick, yellowish – white plaque



Fig.17. A tobacco-lime user's lesion in the premolar region of the right buccal mucosa of a Maharashtrian male *khaini* user.



Fig.18. A bright red *pan* stain in the left buccal mucosa of a betel-quid chewer.

at the site of placement of the mixture (Fig. 17). Occasionally, loose tags of tissue may be seen in the lesion. The tobacco-lime user's lesion resembles a leukoplakia and can be misdiagnosed as such. Unlike leukoplakia, however, tobacco-lime user's lesion can be scraped off with a piece of gauze. In Maharashtra, this lesion is much more common (2.9%) than leukoplakia (0.67%). Tobacco-lime user's lesion has a characteristic microscopic appearance that includes parakeratin-like material on the epithelial surface with ballooning cells and regimented acanthosis of the epithelium. It is hypothesized that the alkaline pH (8.3) of the tobacco-lime mixture exerts a caustic effect on the oral mucosa, causing an abortive keratin formation and clinically resulting in a yellowish-white plaque. Discontinuation of *khaini* use results in the clearance of this lesion.

PAN (BETEL QUID) STAIN

Pan stain is a red coloration of the oral mucosa in betel-quid chewers. It is transient in occasional chewers, but in habitual and heavy betel-quid chewers, the mucosa is perpetually stained (Fig. 18). The bright red color produced during the chewing of *pan* is due to the formation of o-quinone from the water-soluble polyphenols, notably leucocyanidins, at the alkaline pH of 8-9 via secondary reactions. Refraining from this habit or repeated washing generally clears the stain.

PAN ENCRUSTATION

In heavily addicted *pan* chewers, a thick brownish-black encrustation occurs at the site



Fig. 19 . Pan chewer's encrustation in the right buccal mucosa of a heavy betel-quid chewer.

of placement of *pan* (Fig. 19). This encrustation can be scraped off with a piece of gauze. It seems to be the *pan* chewer's counterpart of the tobacco-lime user's lesion. The microscopic features of *pan* encrustation

are also akin to the tobacco-lime user's lesion. This lesion does not seem to progress to leukoplakia. In about 26%, the lesions persist, 45% clear spontaneously, and 29% recur. Discontinuation of the betel-quid chewing leads to nearly complete clearance of the encrustation.

ORAL LICHEN PLANUS-LIKE LESION

Oral lichen planus-like lesion consists of white, wavy, parallel, nonelevated striae that do not crisscross as in lichen planus. The striae seen in lichen planus-like lesion are fine, somewhat like finger prints (Fig. 20). Betel-quid chewing is strongly associated with this lesion, so much so that it almost exclusively occurs at the site of placement of betel quid such as the mandibular groove and the buccal mucosa. Some 89% of the lesions occur among betel-quid chewers, and 11% in those individuals who chewed pan and smoked tobacco. The prevalence of oral lichen planus-like lesion in Ernakulam District is 0.7%.

Fig. 20. An oral lichen Planus-like lesion with fine finger print-like striae in the right buccal mucosa of a betel -quid (*pan*) chewer.





Fig. 21. An oral lichen planus-like lesion in the right buccal mucosa in which the striae radiate from a fibrin covered erythematous area.

Sometimes the striae in oral lichen planuslike lesion radiate from a central fibrin covered area corresponding to the site of placement of the betel quid (Fig. 21). The oral lichen planus-like lesion occurs predominantly (74%) among women in Ernakulam District, consistent with the widespread betel-quid chewing habit among women in that area. A vast majority of these

lesions occur in the 35-54 year age range. Microscopically, there is a slight preponderance of plasma cells in the lesion compared to oral lichen planus. Over a 6year observation period, 79% of the lesions remained stationary, and 21% regressed spontaneously. However, if the betel-quid chewing habit is discontinued, most of the lesions regress.