



## **A Case Series-Smokeless Tobacco Induced Oral Premalignant Lesions**

**Pooja B. Muley<sup>1\*</sup>, Lata Kale<sup>1</sup>, Sonia Kaur Sodhi<sup>1</sup> and Vishwas Kadam<sup>1</sup>**

<sup>1</sup>Department of Oral Medicine and Radiology, C.S.M.S.S. Dental College and Hospital, Aurangabad, India.

### **Authors' contributions**

*This work was carried out in collaboration between all authors. Author PBM designed the study, performed the statistical analysis, wrote the protocol and first draft of the manuscript. Author LK managed the analyses of the study. Authors SKS and VK managed the literature searches. All authors read and approved the final manuscript.*

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**Case Study**

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### **ABSTRACT**

Wide spectrum of oral mucosal lesions can be caused by tobacco in any form. Tobacco use causes an array of changes in the oral cavity, from mucosal pigmentation to thickening, ulceration of the epithelium. Oral cancer and other oral mucosal lesions such as tobacco induced keratosis, leukoplakia, and submucous fibrosis are strongly associated with tobacco consumption. The prevalence and severity of lesions demonstrate a dose-response relationship, which is best predicted by the amount, frequency and duration of smokeless tobacco use.

**Keywords:** Tobacco induced keratosis; leukoplakia; erythroplakia; submucous fibrosis.

### **1. INTRODUCTION**

One of the most common habits seen in India is tobacco use. Tobacco was introduced in India by

the Portuguese nearly 400 years ago and since then it rapidly became a part of socio-cultural milieu in various communities [1]. India is the second largest producer and consumer of

\*Corresponding author: E-mail: [poojamuley.31@gmail.com](mailto:poojamuley.31@gmail.com);

tobacco next only to China [2]. The prevalence of tobacco use among Indian adults is 35% [3]. Tobacco is used in chewable as well as smokable forms. Smokeless tobacco is used in various forms like betel quid which is a mixture of areca nut, slaked lime, and flavoring agent wrapped in betel leaf and tobacco commercial preparations such as gutka, zarda, khaini, mishri, etc., contain the pieces of areca nut coated with powdered tobacco, sweetening and flavoring ingredients in addition to other spices such as saffron, cardamom, etc. which are very popular and highly addictive [4].

Smokeless tobacco used in India is mostly of the species *Nicotianarustica* which contains higher concentrations of tobacco-specific nitrosamines and hence high carcinogenicity, while most smoking tobacco is *Nicotianatabacum*. Sun/air-cured CT in unprocessed, processed, or manufactured form is the cheapest and is used in different parts of India. In Karnataka, it is available as bundles of long strands of leaves (hogesoppu) or as powdered sticks (kaddipudi) which can be used with lime, areca nut, or in a betel quid [5].

Tobacco in any form can cause wide spectrum of oral mucosal lesions. The type and location of these lesions in turn depends on the type, duration of frequency of use. Tobacco use causes an array of changes in the oral cavity, from mucosal pigmentation to thickening, ulceration of the epithelium. Oral cancer and other oral mucosal lesions such as tobacco induced keratosis, leukoplakia, and submucous fibrosis (SMF) are strongly associated with tobacco consumption. In comparison to Western populations, in which oral cancer represents about 3% of malignancies, it accounts for over 30% of all cancers in India; this difference can be attributed to regional variation in the prevalence and pattern of tobacco habits [6].

Precancerous Lesion can be defined as a benign lesion with morphologically altered clinical or histopathological tissue which has greater than normal risk of containing microscopic focus of cancer or of transforming into malignant lesion after diagnosis at a later date. Precancerous Condition can be defined as a disease or patient habit which does not necessarily alter the clinical appearance of local tissue but is known to have a greater than normal risk of precancerous lesion or cancer development [7].

Therefore this case series reviews various patients with oral premalignant mucosal lesions associated with smokeless tobacco reported in CSMSS Dental College, Kanchanwadi.

## 2. CLINICAL PRESENTATIONS

Over a period of 2 years, numerous patients with habit of chewable tobacco reported to the Dept of Oral Medicine & Radiology, CSMSS Dental College, Kanchanwadi. Among them 8 patients were selected who were between the age of 20 to 45 years. All of them were chronic tobacco users in the form of gutkha, paan or zarda

### 3. CASE 1

A 38 year old truck driver reported to the department with a chief complaint of missing tooth. Patient had habit of gutkha chewing since 10 years for 4 to 5 times per day and quid placement was in lower labial vestibule for 15 to 20 minutes. On examination of mucosa, a white hyperkeratotic patch of size approximately 5x4 cm, extending from labial aspect of gingiva of 32,31,41,42,43 to the vermilion border of lower lip. Patch was nontender and scrapable on the peripheral area. Associated severe gingival recession was seen on labial aspect of 32,31,41,42,43. Exposed roots of the associated teeth were stained with dark brown colour due to the prolonged contact with gutkha. On taking punch biopsy the histopathological report revealed thick hyperkeratotic epithelium with no signs of dysplasia. So the diagnosis of tobacco pouch keratosis was given.



**Fig. 1. Tobacco pouch keratosis on lower labial vestibule**

### 4. CASE 2

A 40 years old patient reported to the OPD with the complaint of painless white patch on right and left buccal mucosa since 5 months. Patient had a

habit chewing commercial preparations of tobacco since 15 years with a frequency of 3 to 4 times a day. Along with these the patient also consumed alcohol occasionally.

Intraoral Examination of right buccal mucosa revealed firm, nontender, non-scrapable, greyish white patch measuring 7x8 cm. Surface appears rough and slightly elevated which clinically resembled "cracked mud" in appearance. The lesion was not disappearing on stretching. Chair side investigation, toluidine blue staining was carried out to select the area of biopsy to be made. The selected area was then biopsied (incision). Histopathologic examination with H and E stained sections showed hyperkeratotic stratified squamous epithelium overlying a fibrous connective tissue. Epithelium was showing hyperplasia with no dysplastic features. Dense inflammatory infiltrate were also seen. So the diagnosis of homogenous leukoplakia was made.

### 5. CASE 3

A 58 years old farmer reported to the OPD with a chief complaint of pain in lower left back region of jaw since 4 months. Patient had a habit chewing gutka since 20 years with a frequency of 10 to 15 times a day. Intraoral Examination of left buccal mucosa revealed firm, non-tender, non-scrapable, red and white patch measuring 10x8 cm. Surface appears rough and irregular. The red component appeared velvety red with well demarcated margins. The lesion was not disappearing on stretching. Chair side investigation, toluidine blue staining was carried out to select the area of biopsy to be made. The selected area was then biopsied (incision). The histopathological report confirmed our diagnosis of erythroleukoplakia. Histopathologically, the surface of keratinization layer was seen to be thin and epithelial atrophy with mild dysplasia was seen.

### 6. CASE 4

A 42 years old carpenter reported to the department with a complaint of burning sensation on right buccal mucosa since 3 months. Patient had a habit chewing gutka since 20 years with a frequency of 10 to 15 times a day. On examination a mixed white and red patch seen right buccal mucosa extending on right commissural area, covering right retromolar area and extending on soft palate. Incisional biopsy was performed and the histopathological report revealed hyperparakeratotic stratified squamous

epithelium which is atrophic at places. Features of moderate epithelial dysplasia are evident in the present sections. Dysplastic features are involving half of epithelial thickness at places. The connective tissue core is fibrous with muscle tissue in the deeper areas. So the diagnosis of speckled leukoplakia was made (carcinoma in situ).

### 7. CASE 5

A 44 year old factory worker reported in the department with a chief complaint of restricted mouth opening since 3 years associated with burning sensation on eating spicy foods. Patient had a habit of gutkha chewing along with arecanut since 12 years for 10 to 12 times per day, quid placement in lower left buccal vestibule for 30 minutes. On examination reduced interincisal opening about 25 mm, blanching of right and left buccal mucosa, palate, retromolar area. There was reduced cheek flexibility and tongue protrusion. On palpation thick fibrous bands were palpable on right and left buccal mucosa and retromolar area. The punch biopsy revealed thin atrophic epithelium along with multiple fibroblasts and densely collagen fibers in connective tissue. Thus the diagnosis of oral submucous fibrosis was made.



**Fig. 2. Oral Submucous fibrosis with reduced mouth opening and blanching on palate and buccal mucosa**

### 8. DISCUSSION

The smokeless tobacco typically affects the oral mucosa where the product comes in contact

along with the adjacent periodontium. The lesions are usually well delineated from the normal mucosa. The affected area usually appears pale, white to gray or yellow to brown color and it may progress to a thickened and wrinkled appearance with increased usage of the tobacco. Histologically, the changes range from hyperparakeratosis to epithelial dysplasia. The prevalence and severity of lesions demonstrate a dose-response relationship, which is best predicted by the amount, frequency and duration of smokeless tobacco use [8].

Various studies suggest that smokeless tobacco use can cause oral cancer [9,10]. What is of importance to the dental practitioner is whether and when a tobacco-associated leukoplakia will transform into a malignancy. A review of the literature reveals two different opinions. In one view, the development of a carcinoma from smokeless tobacco use is definite but slow — typically, 20 to 50 years of use is required to cause the malignant changes in the oral lesions [11].

Numerous studies have examined the transformation rates of oral leukoplakias — that is, the time that elapses before a leukoplakic lesion shows malignant change. Pindborg and others [12] followed 248 patients for a mean observation period of 3.7 years; during this time, the period prevalence of malignant transformation in leukoplakia was 4.4%. In addition, a study by Roed-Petersen and others [13] found that the transformation rate for malignancy or epithelial dysplasia of oral leukoplakia was greater than 6%. In general, it seems well accepted that the annual malignant transformation rate of leukoplakia amounts 2%-3% for all clinical subtypes together, including the ill-defined and much debated entity of proliferative verrucous leukoplakia [14]. According to Reichart Prevalence figures of erythroplakia are only available from studies in South- and South East Asia and are as low as 0.02% [15]. The annual malignant transformation rate is actually unknown but is much higher than in leukoplakia. Malignant transformation rate of OSF was found to be in the range of 7–13%. According to Murti PR, Bhonsle RB and others long term followup studies a transformation rate of 7.6% over a period of 17 years was reported. [16]

When cancer is found in association with smokeless tobacco use, the two most common forms of cancer are verrucous carcinoma and

squamous cell carcinoma [17]. It has been observed that the presence of dysplastic changes cannot be predicted by the habit or by the clinical grading of the lesion. The most definitive method of determining if a lesion has progressed to cancer is to biopsy the most suspicious site.

## 9. CONCLUSION

Smokeless tobacco induced oral premalignant lesions are often undiagnosed due to lack of public awareness and due to lack of knowledge among medical professionals. Clinical appearance and diagnosis of a lesion is not adequate to determine its premalignant nature as not all white lesions turn malignant. Diagnostic biopsy and histopathological examination should be considered for any mucosal lesion that persists for more than 14 days after obvious irritants have been removed. Prognosis and patient survival is directly related to stage and grade of cancer at initial diagnosis.

## CONSENT

As per international standard or university standard, patient's consent has been collected and preserved by the authors.

## ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the authors.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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