Transition of Oral Leukoplakia into Squamous Cell Carcinoma: A Case Report with Literature of Review

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The oral cancer is major health problem worldwide with the incidence of 500,000 new cases per year and considered as 4th most common cancer in world and 1st in India. Among all oral squamous cell carcinoma (SCC) is reported to be the most common malignant tumor involving oral cavity which is commonly associated with tobacco chewing habit in India and other parts of Asia and smoking habit in western countries. Oral leukoplakia is a white, non-scrapable, keratotic lesion in the oral mucosa which represents the most prevalent precursor lesion of oral SCC hence clinical diagnosis of oral leukoplakia associated with tobacco habit is very important for the prevention of oral cancer. Here, we report a case of oral SCC, which was reported to be transformed from existing oral leukoplakia lesion.

Keywords: Leukoplakia, Oral Squamous cell carcinoma, Premalignant lesion, SCC

INTRODUCTION

Oral cancer has always been the major worldwide health predicament having the approximate incidence of 5 lakh new cases per year which is almost 3% of all malignancies. It is considered the most serious agony and reported as one of the most common cancers in India. Oral squamous cell carcinoma (SCC) is reported to be the most common malignant tumor affecting oral mucosal structures. Oral cancer is most commonly associated with tobacco chewing habit with or without betel nut habit in India and other parts of Asia, whereas cigarette smoking and alcohol consumption are considered to be main risk factors in Western countries. Tobacco chewing with betel nut increases exposure to carcinogenic nitrosamines (which is approximately 1000 mg/day in tobacco chewers compared to nearly 20 mg/day in patients with smoking habit) and also to areca nut alkaloids derived nitrosamines as compared to tobacco smoke, which contains carcinogenic pyrolysis products for example polycyclic aromatic hydrocarbons, aldehydes, and nitrosamines. Similar to the other SCC the risk of oral SCC increases with increasing age especially in males. Persons with oral SCC almost have been aware of an alteration in that site for 4-8 months before seeking professional help. During the early growth phase, the lesion is usually painless, and this may be the reason for delay in seeking professional care. The most common sites involved are the tongue, oropharynx, lips, floor of the mouth, gingiva, hard palate, and buccal mucosa in decreasing order. The initial noticeable morphologic alterations of oral cancer are the manifestation of the precancerous lesion at the site of which the most frequent ones are leukoplakia and erythroplakia. Leukoplakia is considered to be the most common precursor lesion for oral SCC hence clinical diagnosis of oral leukoplakia associated with tobacco habit is very important for the prevention of oral cancer. Clinical stages (tumor node metastasis [TNM]) of OSCCs at diagnosis have an important influence on the survival and prognosis of patients. Here, we reported a case of oral SCC involving left posterior buccal vestibule and mucosa manifested as a non-healing chronic ulcer which progressed from precancerous white leukoplakic lesion.

CASE REPORT

A 47-year-old male patient reported to our institute with the chief complaint of mobility of teeth on the mandibular left posterior region since 1 month. He was experiencing a slight dull type of pain over that region. On dental examination, a dentist made him aware of a white non-scrappable patch on the left buccal mucosa. The patient was having habit of tobacco chewing since 20 years. He was advised to get
extracted the mobile teeth and also to stop the habit of chewing tobacco. Since then, patient reduced the frequency of chewing tobacco but did not stopped the habit completely. Extraorally; single, enlarged, roughly oval, approximately 1 cm × 2 cm in size, left submandibular lymph node was palpable and tender which was fixed and stony hard in consistency (Figure 1). On intraoral examination, Grade III mobility noted with 36, 37, and 38. A solitary, large, deep, irregular ulcer seen of approximately 2 cm × 4 cm in size with red erythematous everted edges seen in buccal vestibule in the region of 36, 37, and 38 (Figure 2). On palpation, tender indurated deep ulcer with rolled out margins noticed. There was a grayish white non-scrapable leukoplakic patch of approximately 1 cm × 2 cm in size seen on posterior aspect of the ulcer (Figure 2). By all clinical findings and history, provisional diagnosis of malignant ulcer transitioned from oral leukoplakia was considered. Intraoral periapical radiograph of the region showed the irregular bone loss extending till lower 1/3rd region of roots of 36 and 37 suggestive of bone infiltration of the tumor cells (Figure 3). On further radiographic investigations such as in panoramic radiograph (Figure 4) and computed tomography showed the irregular bone loss with resorption of both buccal and lingual cortical plates (Figures 5-7). Incisional biopsy of the lesion carried out under local anesthesia and the specimen (Figure 8), sent for the further histopathological examination which microscopically showed tumor composed of polygonal cells arranged in invasive clusters in fibrous stroma; concludes to the picture of well-differentiated SCC, (Figure 9). So, the final diagnosis of well-differentiated SCC involving mandibular left buccal mucosa (TNM - T2 N1 M0) was made. The left sided hemi-mandibulectomy along by radical neck dissection was planned. The patient is still under our follow-up and the postsurgical radiotherapy.

**DISCUSSION**

It is well recognized that oral SCC occurs as a result of a number of molecular and biochemical cellular changes and alterations in the underlying fibrovascular stroma including neovascularization. In addition to alterations in cellular structure, some clinical changes in affected epithelial tissues are also observed, known as precancerous lesions.
The important clinical significance of these lesions lie in its association with oral SCC. Oral leukoplakia is a white, non-scrappable, keratotic lesion in the oral mucosa which represents the most prevalent precursor lesion of oral SCC (prevalence ranges from 0.1% to 0.5%). As seen in our case, the tumor was transformed from previous oral leukoplakia lesion on left buccal mucosa and remaining leukoplakic lesion was also present on a posterior aspect of the malignant ulcer. Multiple clinical and socio-demographic variables affect the time required for the diagnosis like reluctance of the patient to consult the physician due to lack of health care, specially in case of patients with lower socio-economic status as well as health care professional delay in diagnosing and treating the disease. In our case too, the patient was advised to quit the habit and made aware for the white lesion before the occurrence of the tumor, but the patients negligence toward that leads to malignant transformation of the precancerous leukoplakic lesion which can be prevented by stopping the habit and early histopathological examination of the lesion. The most
margins. Use of tobacco and alcohol are considered the major etiological factors, however, nutritional deficiency, occupation, viral infection, and chronic dental irritation are the other risk factors. As in our case, habit of tobacco chewing along with lime was the major etiological factor. Chronic persistent leukoplakia or oral submucous fibrosis are considered as early predictors of transformation into oral SCC where the rate of transformation ranges from 0.13% to 6% and risk is increasing to 14% with dysplastic lesions. In our case, we reported the previous leukoplakia lesion which was transformed into well-differentiated SCC because of the negligence of the patient toward stoppage of habit.

**CONCLUSION**

Early diagnosis of precancerous lesions associated with tobacco habit and awareness of the patients toward the stoppage of habit to avoid its malignant transformation is very crucial for the oral physician.

**REFERENCES**


How to cite this article: Khairnar SA, Rajendra B, Sanjeev O, Swapnali C, Abhay K. Transition of Oral Leukoplakia into Squamous Cell Carcinoma: A Case Report with Literature of Review. IJSS Case Reports & Reviews 2016;2(11):7-10.

Source of Support: Nil, Conflict of Interest: None declared.