Have A Sharp Tooth? A Link to Oral Cancer-A Case Series

Dr. Rajni Aggarwal[1]*, Dr. Basavaraj T. Bhagawati[2], Dr. Deepika Chaudhary[3], Dr. Manoj Kumar Garg[4]

[1]*Professor& HOD, Department of Periodontology & Oral Implantology, Surendera Dental College and Research Institute, Sriganganagar Rajasthan, India Email: rajniperio@gmail.com
[2]Professor& HOD, Department of Oral Medicine & Radiology, Surendera Dental College and Research Institute, Sriganganagar Rajasthan, India Email: drbasavarajb@yahoo.com
[3]Post Graduate Student, Department of Periodontology & Oral Implantology, Surendera Dental College and Research Institute, Sriganganagar Rajasthan, India Email: dr.deepikachoudhary9@gmail.com
[4]Assistant Professor (Plastic Surgeon), Department of General Surgery, Dr. S.S Tantia Medical College, Sriganganagar Email: manoj19jan@gmail.com

***Corresponding Author:** Dr. Rajni Aggarwal

*Professor& HOD, Department of Periodontology & Oral Implantology, Surendera Dental College and Research Institute, Sriganganagar Rajasthan, India Email: rajniperio@gmail.com
DOI: 10.47750/pnr.2022.13.S05.421

Abstract

Squamous cell carcinoma of the mouth (OSCC) accounts for 90% of all oral malignancies. It is a mucosal neoplasm that is malignant. The lateral border and ventral surface of the tongue are the most typical locations. Though the cause of OSCC is unknown, tobacco and betel quid usage, heavy alcoholic drinking, a diet lacking in fresh fruits and vegetables, infections, and heredity are all thought to be common risk factors. Dentist need to be aware that oral trauma and irritations such as – sharp teeth or over-hang restorations may also lead to OSCC within the short period of time. So, early detection of OSCC in such conditions is critical for improving prognosis and lowering morbidity and mortality. As a result, we offer a case series of three patients with a white lesion who were diagnosed with OSCC caused by a sharp tooth and verified by histopathology.

Keywords: Squamous cell carcinoma of the mouth, Oral cancer, Chronic mechanical irritation, Sharp Tooth

INTRODUCTION:

In humans, carcinogenesis is multifactorial. Tobacco and alcohol are frequently cited as primary causes of oral cancer.[1] However, they may not be the cause of all cancers, and people who are not exposed to those variables may develop cancerous lesions. This means that oral cancer is caused by other variables, one of which has been mentioned: chronic mechanical irritation (CMI). The oral mucosa is injured repeatedly by the mechanical action of an intraoral irritational agents, resulting in CMI. Any mechanical irritation could be caused by defective teeth (malpositioned or with sharp or rough surfaces due to decay or fractures), ill-fitting dentures (sharp or rough surfaces, lack of retention, stability, or overextended flanges), and/or parafunctional habits (e.g. oral mucosa biting or sucking, tongue interposition or thrusting), acting individually or in combination. CMI may cause alterations in the healthy mucosa or exacerbate existing oral illnesses. Some publications have identified defective teeth as a prevalent feature in OC patients. This case series demonstrates various instances of OSCC caused by a sharp tooth with no history of harmful practices and confirmed by histology.

CASE DESCRIPTION

Case 1

A 45-year-old female patient presented to the Department of Periodontology with the chief complaint of a non-healing ulcer on the posterior right lateral border of the tongue that had been present since one month, was gradual in onset and rapid in progression. It was associated with mild pain on mastication. There was no bleeding, pus discharge, or any other constitutional symptoms. Patient did not reveal any history of smoking, chewing tobacco or areca nut or consuming alcohol any time in her life. Patient gave history of trauma from adjacent sharp tooth since 1 months while chewing food. Intraoral examination revealed lingually placed sharp cusp of right lower second premolar, first and second molar (45,46,47)causing obvious trauma to tongue (Figure 1), fractured cusp of right lower second premolar. Clinical examination revealed a solitary elevated, sessile ulcerated, irregularly shaped lesion extending from posterior lateral border of tongue to floor of the mouth and lower right first molar to retromolar area anteroposteriorly,. On the basis of history and clinical examination a provisional diagnosis of oral leukoplakia was given with differential diagnosis of a chronic traumatic ulcer and inflammatory hyperplasia. An incisional biopsy (Punch biopsy) was performed to confirm the diagnosis. (Fig-2) Histopathology report revealed hyperkeratosis with bulbous rete ridges basal call hyperplasia with mild N:c ratio and chronic inflammation mainly lymphocytes in stroma. No granulation and malignancy seen. Overall features were in favour of leukoplakia with non-specific inflammation. (Fig.-3)

In this case, oral hygiene reinforcement, scaling, root planning and grinding of offending teeth were carried out. The patient was referred to the plastic surgeon in general hospital for opinion. They decides to resect the lesion (excisional biopsy) and sent for the biopsy. Histopathology report of excisional biopsy confirmed the diagnosis of Squamous cell carcinoma (Figure 4). TNM staging was done for tumor which was T2N1MX.

Figure-1: A sharp teeth induced solitary elevated, sessile ulcerated growth extending from posterior lateral border of tongue and floor of mouth.

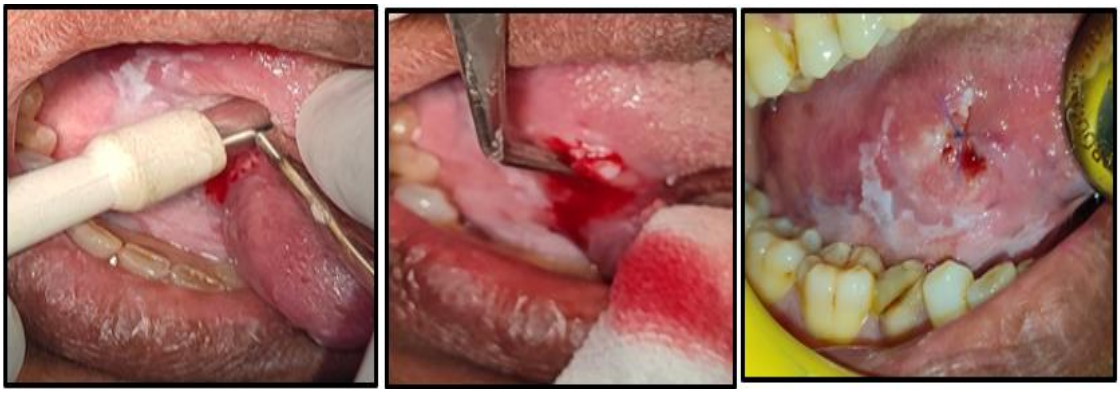

Figure- 2: An incisional biopsy (Punch biopsy) was performed on lateral border of tongue and vicryl 2-0 suture placed.

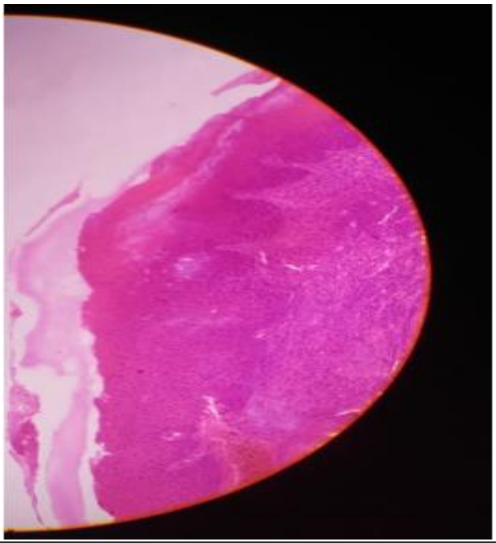

Figure-3: Histopathology report revealed hyperkeratosis with bulbous rete ridges basal call hyperplasia with mild N:c ratio and chronic inflammation mainly lymphocytes in stroma. No granulation and malignancy seen.

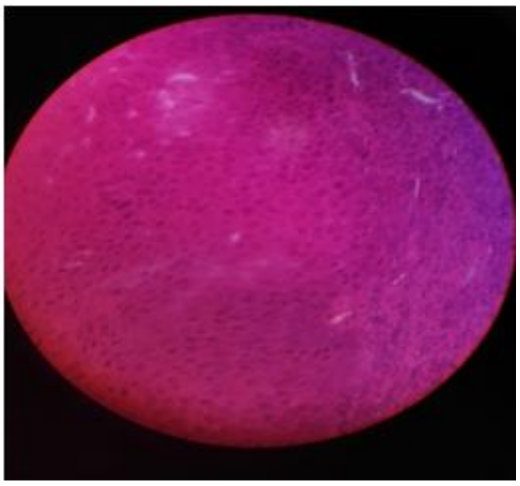

FIGURE- 4: Histopathology report confirmed a diagnosis of Squamous cell carcinoma.

Case 2

A 54-year-old male patient presented to the Department of Periodontology with the chief complaint of white patch on the posterior right ventral surface of tongue that had been present since one month. It was associated with pain and irritation but no other constitutional symptoms. Patient did not reveal any history of smoking, chewing tobacco or areca nut or consuming alcohol any time in her life. It was associated with trauma from adjacent sharp tooth since 1 months. Intraoral examination revealed sharp cusp of right lower second premolar causing trauma to tongue. (Figure 5) Clinical examination revealed a solitary elevated, sessile ulcerated lesion on posterior lateral border of tongue. Provisional diagnosis of oral leukoplakia was given with differential diagnosis of a chronic traumatic ulcer and inflammatory hyperplasia. An incisional biopsy (Punch biopsy) was performed to confirm the diagnosis. (Fig-6) Histopathology report showed epithelium with severe dysplastic features and break in the basement membrane and dysplastic/ atypical cells invading underlying connective tissue stroma. Numerous malignant epithelial islands with large amount of keratin pearl formation were seen in the connective tissue stroma. Histopathological features confirmed the diagnosis of well differentiated squamous cell carcinoma.

Figure-5 : A sharp tooth induced lesion on posterior right ventral surface of tongue.

FIGURE-6: Histopathology report confirmed a diagnosis of well differentiated Squamous cell carcinoma.

Case 3

A 39-year-old male patient presented to the Department of Periodontology with the chief complaint of traumatic ulcer on the posterior left ventral surface of tongue that had been present since two months. It was associated with pain but no other constitutional symptoms. Patient did not reveal any history of smoking, chewing tobacco or areca nut or consuming alcohol any time in her life. It was associated with trauma from adjacent sharp teeth in the left molars. Clinical examination revealed a solitary elevated, sessile ulcerated lesion on posterior lateral border of tongue. (Fig 7) An incisional biopsy was performed. Histopathology report showed para-keratinized stratified squamous epithelium overlying the connective tissue stroma. Epithelium showing features of dysplasia. The connective tissue stroma showed loosely arranged collagen fibers with sheets of chronic inflammatory cells beneath the basement membrane. The histopathology features were suggestive of epithelial dysplasia. (Fig 8)

Figure-7: A sharp tooth induced lesion on left lateral surface of tongue.

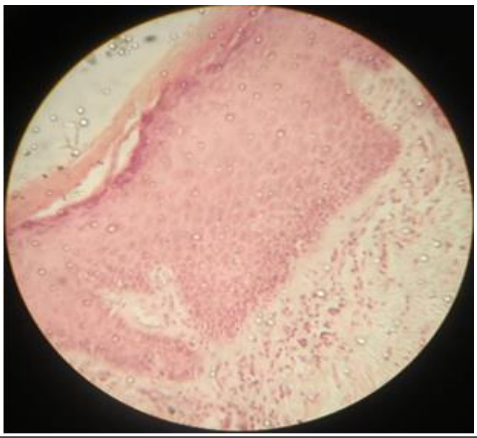

FIGURE-8: Histopathology report confirmed a diagnosis of well differentiated Squamous cell carcinoma.

DISCUSSION:

Squamous cell carcinomas account for more than 90% of all mouth cancers.[2] SCC of the tongue is the most common cancer (27.6%), followed by oropharyngeal cancer (22.8%), lip cancer (16.5%), mouth floor cancer (14%), gingiva (9.1%), hard palate cancer (4.1%), and buccal mucosa cancer (4.1%). (3.5 percent).[3] The tongue, particularly the posterior lateral and ventral sides, is the most prevalent site of intraoral cancer involvement.[2] we report the cases of SSC on the posterior lateral border of the tongue. Males have a substantially higher prevalence than females, with a male to female ratio of 2:1.[4] Early lesions are typically asymptomatic and sluggish to grow. Advanced lesions develop diffuse borders with ragged edges, induration, and fixation. The most common mouth symptom of an ulcerated mucosal surface is persistent discomfort or irritation. If treatment is delayed, patients may experience numbness, trouble speaking or swallowing, and lesions can grow to be several centimeters in diameter, causing local invasion and loss of important and osseous tissues. In our cases, the patients did not disclose any harmful practices. Past medical and familial histories were likewise irrelevant. Chronic trauma caused by sharp edges of premolars and molars, was the elucidating factor, which could be one of the mechanisms of buccal mucosa carcinoma. Though the mechanism by which chronic trauma to the mucosa contributes is controversial. It has been suggested that the wound of oral mucosa may facilitate the absorption of other carcinogens. One mechanism suggests abnormal mitosis due to chronic trauma increases repair tissue injury which put cells at risk of DNA damage by other agents, initiating carcinogenesis.[5]

When a patient complains of intraoral pain and dysfunction, it is widely accepted that SSC of the oral cavity is easy to diagnose. Dentists have the highest chance of detecting early oral cavity lesions. They are responsible for thoroughly inspecting the oral cavity and referring patients with suspected lesions for adequate assessment and biopsy.[6] In addition, the patients first described the lesion as a dental trauma, but histopathologic investigation revealed it to be a biopsy-proven SCC. As a result, early identification of this entity and interdisciplinary care may aid in the prognosis of these instances. In the case of patients who see the dentist first, dentists should have sufficient awareness of the disease. Taking care of such patients include chronic irritant removal followed by surgical excision/resection of tumor, radiation, systemic cytotoxic chemotherapy alone or in combination, and epidermal growth factor receptor blocking.[7, 8]

CONCLUSION:

Oral Squamous Cell Carcinoma develops within a field of precancerized epithelium, either as a result of a potentially malignant condition or as a result of a spontaneous mutation. As a result, early recognition of this entity, as well as full understanding of all risk factors, including those that are less common, and multidisciplinary therapy, may aid in a better prognosis.

REFERENCES:

1. Radoï L, Menvielle G, Cyr D, Lapôtre-Ledoux B, Stücker I, Luce D, et al. Population attributable risks of oral cavity cancer to behavioral and medical risk factors in France: results of a large population-based case-control study, the ICARE study. BMC Cancer. 2015;15:827.
2. Neville B. Oral and maxillofacial pathology. 1st ed. Philadelphia: WB Saunders Co; 1995. p. 295–304.
3. National Cancer Institute. Surveillance epidemiology and end results program (SEER), 1983–1993.
4. Spiessl B, Hermanek P, Scheibe O. TNM. Atlas Illustrated guide to the TNM/p TNM-classification of malignant tumors. 2nd ed. New York, NY: Springer-Verlag; 1985. p. 18–24.
5. Kumar M, Singh PP, Saxena D, Singla N. Chronic trauma as precipitating factor of squamous cell cartcinoma of tongue – 3 case reports. IJDS. Oct 2014;6(4):29-31
6. Rosai J. Ackermans surgical pathology. 8th ed. Philadelphia: Mosby Co; 1989. p. 223–57.
7. Feller L, Lemmer J. Oral squamous cell carcinoma: Epidemiology, clinical presentation and treatment. JCT. 2012;3:263-8.
8. Spencer KR, Ferguson JW, Wiesenfeld D. Current concepts in the management of oral squamous cell carcimoma. ADJ. 2002;47(4):284-9.