Dental Trauma – A Risk Factor For OSCC

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Abstract:

Oral cancer is the 8th most frequent cancer in the world among males and 14th among females. The most common type of oral cancer is squamous cell carcinoma (SCC). SCC is a malignant neoplasm of epithelial origin. In India, buccal mucosa and gingivobuccal sulcus are frequent sites of oral SCC (OSCC) but in recent times increasing number of tongue SCC have been reported. The etiology of OSCC is multifactorial with both extrinsic and intrinsic factors playing important role. A definite causal association has been found between OSCC and smoking and/or alcohol consumption. But over time, OSCC case reports in a non-smoking non-drinking (NSND) population are also emerging. This case report presents a case of OSCC induced by chronic mechanical irritation with no deleterious habit history.

Introduction

Oral and pharyngeal cancers, grouped together are the sixth most common cancers in the world[1]. The incidence rate of cancers of the oral cavity in both males and females in all urban cancer registries of India are among the highest in the world[2]. In parts of India, oral cancer can represent more than 50% of all cancers. In certain other countries also, such as Sri Lanka, Pakistan, and Bangladesh, oral cancer is the most common cancer. In developed countries, oral cancer is less common but is the eighth most common form of cancer overall[3]. The global incidence of lip, oral cavity, and pharyngeal cancers of 529,500 cases, corresponding to 3.8% of all cancer cases, is predicted to rise by 62% to 856,000 cases by 2035 because of changes in demographics[4].

The morbidity and mortality rates of this disease are further increased due to a tendency for invasion and metastasis as well as the propensity to develop either second primary tumors or second field tumors[5]. Annually, 130,000 people succumb to oral cancer in India, which translates into approximately 14 deaths per hour. In the US, oral cancer represents approximately 13% of all cancers thereby translating into 30,000 new cases every year[6]. However, in comparison with the U.S. population, where oral cavity cancer represents only about 3% of malignancies, it accounts for over 30% of all cancers in India. The variation in incidence and pattern of oral cancer is due to regional differences in the prevalence of risk factors[7].

Almost 90% of oral cancers are squamous cell carcinomas (SCC)[8]. SCC is a malignant neoplasm of mucosal origin, which is predominantly found in middle-aged and older persons[9,3]. But it is also observed over time that there is nearly five-fold increase in incidence of OSCC in patients under the age of 40 years, many with no known risk factors[10]. OSCC affects males more frequently than females, although the ratio is equalizing[3]. In recent times, a distinct clinical subgroup of elderly female nonsmoking (NS) and nondrinking (ND) patients with OSCC has been reported, which may represent an increasing proportion of OSCC patients over time. NSND population represents 13–35% of the OSCC population[11]. Most studies from India report buccal mucosa as the most common sub-site, followed by tongue, the least frequent site being lip unspecified[12]. But a surprising increase in oral tongue cancer has been observed in females[13,14].

The etiology of oral cancer is multifactorial with both extrinsic and intrinsic factors playing important role. Extrinsic factors include the agents such as tobacco, excess consumption of alcohol, betel quid usage, chronic irritation, poor oral hygiene, syphilis, and sunlight (vermilion cancers only). Intrinsic factors include systemic or generalized disorders such as malnutrition, immunosuppression and iron-deficiency anemia[15].

In this paper, we present a case report of OSCC caused by chronic dental trauma.

Case Presentation

A 60-year-old male presented with the complaint of pain and burning sensation in relation to right lateral border of tongue since 4 months. The patient was apparently asymptomatic 4 months back and then noticed a small growth that had been slowly enlarging. The patient was a lifelong non-smoker, non-alcoholic and also did not have history of chewing betel quid and spicy food habits. On intra-oral examination, an ulcero-proliferative growth at the right lateral border of tongue was seen [Fig 1], which was 3 × 2 cm in size, firm, and sessile with indurated margin. On palpation, right submandibular lymph nodes were found to be enlarged, firm and nontender. The previous dental history revealed that the patient sustained chronic irritation from the ragged margins of adjacent teeth, extraction of carious 46 and cuspal grinding in relation to 47 was performed by a private dentist 2 months back but there was no change in the growth pattern of the lesion. Patient had no significant medical history. Based on the clinical findings, a provisional diagnosis of SCC of right lateral border of tongue was made.

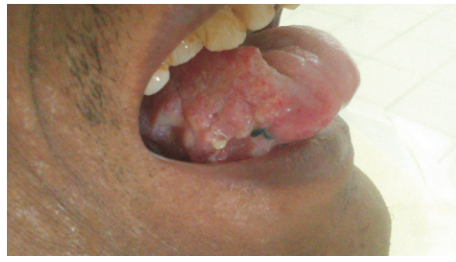

Fig 1: Picture showing proliferative growth on the Right lateral border of tongue.

Incisional biopsy was performed under local anaesthesia and the specimen was sent for histopathological examination. Gross specimen comprised of two soft tissues, measuring 5 x 5 x 3 mm and 3 x 4 x 2.5 mm in size, irregular in shape, whitish brown in colour and soft in consistency.

Histopathological features

On low magnification, highly cellular stroma was seen. On higher magnification, hyperparakeratinized dysplastic epithelium, with loss of stratification, clear cells with eccentrically placed nuclei were seen. Sheets of epithelial cells within the connective tissue stroma were seen. The cells were hyperchromatic, highly pleomorphic with altered nuclear-cytoplasmic ratio. The tumour cells were round, oval, or elongated in shape. Abnormal mitotic figures were seen dispersed throughout the stroma. Formation of keratin pearls, individual cell keratinisation were also evident. Dense inflammatory cells mainly comprising of lymphocytes and plasma cells, endothelial lined blood vessels and extravasated RBCs were seen within the stroma. A diagnosis of well differentiated OSCC was made [Fig 2 & Fig 3].

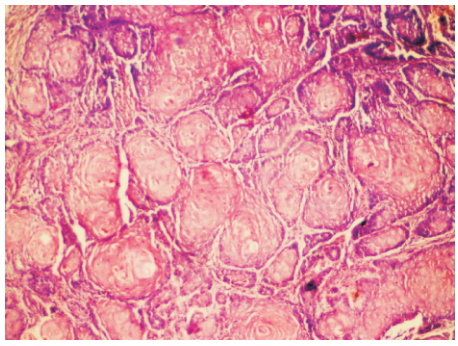

Fig 2: Picture showing sheets of epithelial cells invading into the underlying connective tissue and keratin pearls formation (H&E x 100)

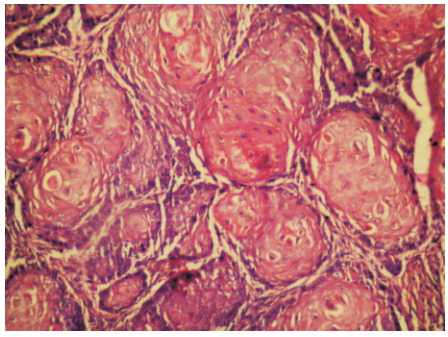

Fig 3: Picture showing keratin pearls, hyperchromatic large nuclei with prominent pleomorphism (H&E x 400)

Discussion

Tongue is the most common site for OSCC in the western world, which accounts for approximately 40% to 50% of all cases and is most often associated with consumption of alcohol and smoking[16,17]. In recent times, it is also observed in India, that there is a relative increase of glossal SCC in patients who do not have tobacco and/or areca nut chewing and alcohol drinking habits[13,18,19].

As with many carcinomas, the risk of intraoral cancer increases with increasing age, especially for males. In recent times, increased numbers of tongue SCC are reported in young adults also[14]. The age of the patient in this reported case was 60 years.

Early lesions are often asymptomatic and slow-growing[20]. OSCC often is preceded by leukoplakia or erythroplakia or they may appear as ulcers without adjacent white or red mucosal change[16]. As the lesion develops, the borders become diffuse and ragged, and induration and fixation ensue. If the mucosal surface becomes ulcerated, the most frequent oral symptom is that of a persistent sore or irritation, burning sensation while eating, and difficulty in speaking or swallowing. Lesions can extend to several centimeters in diameter if treatment is delayed; this delay permits large lesions to invade and destroy vital structures. Persons with OSCC are most often aware of an alteration in oral cancer site for 4–8 months before seeking professional help[20]. Patient delay can largely be attributed to lack of public awareness of the signs and symptoms of disease and oral symptoms being rarely attributed to cancer and frequently interpreted as minor oral conditions[21]. In the present case also, it was observed that patient was aware of the condition for last 4 months and considered it as minor condition arising out of dental trauma.

The carcinogenic role of chronic trauma caused by fractured crowns or dental malocclusion is still controversial[22]. Recurrent dental trauma is not widely accepted as a carcinogen. Head and neck oncology generally gives little emphasis to chronic teeth or denture irritation as a causative factor. Thus, little attention is given to teeth or dental/denture trauma in the treatment of premalignant changes or cancers of the oral cavity presenting de novo. Clinicians rarely recognize, and even less frequently document, whether dental trauma was present before the development of cancer symptoms[23]. There may be 3 types of chronic mechanical irritation (CMI) factors: dental (malpositions, sharp/broken teeth, and/or rough or defective restorations); prosthetic (ill-fitting dentures, rough/sharp/overextended flanges, and lack of retention/stability); and functional (swallowing, occlusal, and other dysfunctional disorders)[24].

Sites of chronic dental trauma include the tongue edge and the buccal mucosa within the range of sharp teeth or a rough dental edge. The gingiva, floor of mouth, and buccal mucosa are the key sites of trauma from chronic denture rubbing. The edge of tongue is one of the most common sites of tumor occurrence in both smokers and nonsmokers, though it is much more common in non-smokers[23].

One explanation for this high incidence of edge of tongue cancers is chronic dental trauma and that is due to placement of teeth, which are more often turned inwards toward the tongue than outwards and also, the tongue, through chewing and talking, has more movement against the teeth. This irritant effect may play role in the development of SCC at the lateral border of tongue, in lifelong nonsmokers, in the absence of other major risk factors. Chronic dental trauma may enhance the absorption of other known oral cavity carcinogens, especially cigarette smoke and alcohol[23]. These topical carcinogens would have increased absorption through broken and ulcerated mucosa compared with absorption through intact lining. However, with the ubiquitous application of tobacco carcinogens throughout the oral cavity in smokers and drinkers, tumor location in smokers is considerably more diverse compared with non-smokers[23].

CMI per se may not be able to produce genetic mutations but may prompt epigenetic changes that ultimately inhibit DNA repair and apoptosis. This suggests that chronic irritation could at least play a role as promoter and progressor in oral carcinogenesis. Thus, if a cancer has eventually started from another cause, chronic irritation will probably hasten the process[24].

The suggestion that chronic irritation and inflammation can lead to the development of cancer elsewhere in the body is not a new concept. Multiple cancers in different sites have been shown to be a result of chronic trauma, irritation, or inflammation, e.g. laryngeal cancer in textile workers due to chronic irritation from textile fibers, esophageal cancer due to chronic gastro-esophageal reflux disease, colon cancer in inflammatory bowel disease etc[23].

Chronic trauma may lead to the development of Chronic Traumatic Ulcer (CTU). CTU is a relevant clinical finding because it represents the effect of a low-intensity and persistent CMI. CTU has been linked to malignant transformation mainly through case reports but several authors reject this possibility. But recurrent oral ulcerations have shown an increase of the risk of OSCC in nonsmokers and nondrinking individuals. Previous studies have shown a clear link between persistent inflammation and cancer, through the overexpression of genes regulating proliferation, angiogenesis, and immune evasion. So, CMI could also play a role promoting a continuous inflammatory state[24]. Chronic inflammation can induce immune suppression and contribute to the occurrence of OSCC[25]. Infiammation can contribute to multiple hallmark capabilities by supplying bioactive molecules to the tumor microenvironment, including growth factors that sustain proliferative signalling, survival factors that limit cell death, proangiogenic factors, extracellular matrix-modifying enzymes that facilitate angiogenesis, invasion, and metastasis, and inductive signals that lead to activation of epithelial-mesenchymal transition and other hallmark-facilitating programs. Infiammatory cells can release chemicals, notably reactive oxygen species, that are actively mutagenic for nearby cancer cells, accelerating their genetic evolution toward states of heightened malignancy[26].

Conclusion

Nonsmokers develop OSCC on the edge of the tongue at twice the rate of smokers. Dental trauma could be a significant risk factor for OSCC. CMI should be properly recorded in addition to habit history. Patients presenting with evidence of dental irritation or premalignant changes to oral mucosa must receive appropriate dental care to help prevent cancer development.

References

References are available on request at editor@healtalkht.com