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Case Report

TRAUMA INDUCED ORAL MALIGNANT ULCER: A CASE REPORT

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Abstract: Oral cancer is one of the sixth most frequently occurring cancers. Oral squamous cell carcinoma (OSCC) accounts for 90% of all oral cancers. SCC is a malignant neoplasm of mucosal origin. The most common site is lateral border and ventral surface of tongue. The etiology of OSCC is multifactorial. The use of tobacco and betel quid, heavy alcoholic drinking, diet low in fresh fruits and vegetables, viruses, trauma from dental structures and genetics are considered as possible risk factors. Early diagnosis plays an important role in improving prognosis and reducing morbidity and mortality associated with OSCC. It is managed by surgery, radiotherapy, chemotherapy singularly or in combination, but regardless of treatment modality, the 5-year survival rate is poor at about 50%. This can be attributed to the fact that about two-thirds of OSCC already have a large lesion at the time of diagnosis. This present case high lights a case of OSCC induced by a sharp tooth with no deleterious habits history and confirmed by histopathology.

Key words: Non-Healing Ulcer, Oral Squamous Cell Carcinoma, Sharp Tooth, Trauma

Introduction: Oral cancer represents 2%-5% of all cancers, being one of the 10 most frequent ones. Although its incidence is lower than for other types of cancer, it is a major poblational health problem because of its mortality and severe morbidity.[1] The most important risk factors for OSCC includes consumption of tobacco or betel quid and regular drinking of alcoholic beverages. However infection with a high-risk human papillomavirus (HPV) genotypes and a diet low in fresh fruits and vegetables,[2] syphilis, sunlight exposure and intrinsic factors (systemic or generalized disorders such as malnutrition, iron deficiency anemia)[3,4] have also recently been implicated in the aetiopathogenesis of OSCC. The highest incidence and prevalence of OSCC is found in Indian subcontinent where the risk of developing OSCC is increased by very prevalent

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habits of chewing tobacco, alcohol, betel quid or areca nut. The mutagenic effects of these deleterious substances are dose, duration and frequency of consumption dependent and are accelerated and exaggerated by the concurrent use of two or more of these agents. However, as not all persons who practice these high risk habits will develop OSCC and it may develop idiopathic and environmental factors may either afford protection against OSCC development or may predispose to or even promote OSCC development.[2] Apart from oral cancer risk factors already described in literature, other emerging factors such as chronic irritation from dental factors has also been proposed.[1]

Chronic trauma to oral mucosa (CTOM) is the result of repeated mechanical irritative action of an intraoral injury agent. Defective teeth (malpositioned or with sharp or rough surfaces because of decay or fractures), ill fitting dentures and/or para functional habits acting individually or together, could all be responsible of this mechanical irritation. CTOM could generate lesions on a healthy mucosa or intensify previous oral disease.[1]

The oral cavity is more accessible to complete examination. But either due to ignorance or inaccessibility of medical care, the disease gets detected in later stages. Thus, there is a need for improvement in early detection of OSCC as in initial stages, treatment is more effective and morbidity is minimal.[5] Along with conventional method of early diagnosis of OSCC that includes gold standard biopsy, oral brush cytology and toludines blue and Lugol's iodine staining other advanced diagnostic aids are also helpful as adjunct to conventional aids like optical coherence tomography, fluroscence endoscopy, lab-on-a-chip, DNA ploidy, chemiluminescence vizilite, velscope system, salivary and protein biomarkers.[6,7]

We report a case of OSCC of left buccal mucosa with chronic trauma as precipitating factor.

Case report: A 65 year old male patient reported to department of Oral Medicine and Radiology with chief complaint of a non-healing ulcer on left buccal mucosa since 8 months which was gradual in onset and rapid in progression which was not associated with bleeding or any pus discharge or constitutional symptoms and was associated with mild pain on mastication. Patient did not reveal any history of smoking, chewing tobacco or areca nut or consuming alcohol any time in his life. Patient gave history of trauma from adjacent sharp tooth since 8 months while chewing food. Intraoral examination revealed buccally placed with sharp cusp of 38 causing obvious trauma to area (Figure 1), root stumps with 36 and a solitary elevated, sessile ulcerated growth extending from distal aspect of 36 to retromolar area anteroposteriorly and from buccal surface of 27, 28 till vestibular depth of 37, 38 superoinferiorly with approximate size of 3x3.5 cm, irregularly shaped with well defined borders. Growth seems to be nodular and arise from left buccal mucosa and rests on mandibular left alveolar ridge along line of occlusion of sharp cusp of 34,35,36,37,38. Overlying surface appears erythematous and mildly tender to palpate, bleeds severely on provocation and is indurated in some areas with soft in consistency in other areas of nodule (Figure 1). A solitary mobile tender submandibular lymph node measuring approximately 1.5x2 cm, firm in consistency was palpable on left side. On the basis of history and clinical examination a provisional diagnosis of oral malignant ulcer was given with differential diagnosis of a chronic traumatic ulcer and inflammatory hyperplasia were made.

An orthopantamogram was made which revealed no obvious pathology relating to growth indicating that it is a soft tissue lesion without bony involvement (Figure 2). Incisional biopsy was performed which confirmed a diagnosis of Squamous cell carcinoma (Figure 3). TNM staging was done for tumor which was $T_2N_1M_X$.

Discussion: OSCC is considered a serious public health problem that causes great morbidity and mortality in the population.[8,9] Most frequent sites of occurrence includes tongue (27.6%) followed by oropharynx (22.8%), lip (16.5%), floor of mouth (14%), gingival (9.1%), hard palate (4.1%) and buccal

mucosa (3.5%). The risk of OSCC increases with increasing age. Prevalence is much higher in males with male to female ratio being 2:1. Early lesions are often asymptomatic and slow growing. Advanced lesions become diffuse with ragged borders and induration and fixation ensue. If mucosal surface becomes ulcerated, the most frequent oral symptom is persistent soreness or irritation. Patients may report numbness or difficulty in speaking or swallowing and lesions can extend to several centimeters in diameter if treatment is delayed which causes local invasion and destruction of vital and osseous structures. Since there is minimal pain during early growth phase hence this explains the delay in seeking professional care.[1,2,3,4,8]

The mechanism by which CTOM contributes is controversial. It has been proposed 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. The other mechanism involved could be because of chronic inflammation at the site affected by CTOM, through release of chemical mediators and oxidative stress. This could induce genetic, epigenetic changes, damage DNA, inhibiting its repair, altering transcription factors, preventing apoptosis and stimulating angiogenesis, therefore contributing in all stages of carcinogenesis.[1]

In the present case, patient did not report any deleterious habits. Past family and medical histories were also non-contributory. The elucidating factor was history of chronic trauma due to sharp edges of molars and particularly third molar which may be considered as one of the possible mechanism of buccal mucosa carcinoma.

Management of such patients include removal of chronic irritant followed by either surgical excision/resection of tumor, radiotherapy, systemic cytotoxic chemotherapy alone or in combination, blocking of epidermal growth factor receptor.[2,10]

Conclusion:OSCC arises from within a field of precancerized epithelium either from a potentially malignant disorder or de novo. Consequently early recognition of this entity with thorough knowledge of all even less frequent risk factors as well as a multidisciplinary management may help in better prognosis.

Figure 1 – solitary ulcerated nodular growth at left buccal mucosa with sharp adjacent teeth

Figure 2 – OPG showing no relevant pathology (red arrow showing no bony involvement)

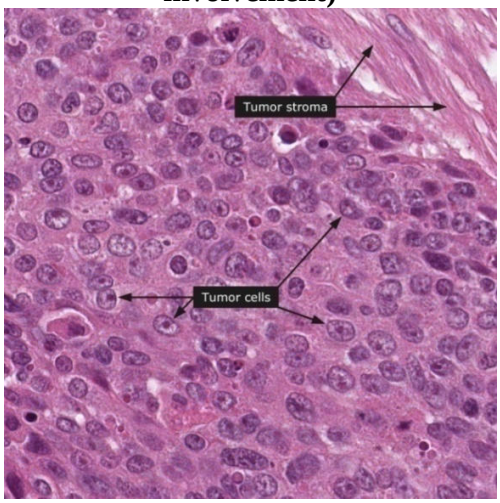

Figure 3 – H & E section showing dysplastic cells (10x)

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