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RESEARCH ARTICLE

ORAL SQUAMOUS CELL CARCINOMA - CASE REPORT

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Abstract

Most head and neck cancers are derived from the mucosal epithelium in the oral cavity, pharynx andlarynx and are known collectively as head and neck squamous cell carcinoma (HNSCC). Oral cavity cancers are generally associated with tobacco consumption, alcohol abuse,exposure to environmental pollutants and infection with viral agents, namely HPV and EBV or both, whereaspharynx cancers are increasingly attributed to infection with humanpapillomavirus (HPV), primarilyHPV-16. Despiteevidence of histological progression from cellular atypia through various degrees of dysplasia,ultimately leading to invasive HNSCC, most patients are diagnosed with late-stage HNSCC without a clinically evident pre malignant lesion.

Introduction:-

The worldwide updated cancer prevalence reported an increase in the incidence of oral cancer[1]. Oral cavity cancer (OCC) is categorized under head and neck cancer (HNC)[2] and regarded as the sixth most common cancer worldwide[3]. Oral squamous cell carcinoma accounts for 90% of the histologic type of oral cancer[4]and may or may not be preceded by oral potentially malignant disorders. OCC is defined as malignant neoplasia of the oral cavity and includes buccal mucosa, floor of mouth, anterior tongue, alveolar ridges, retro-molar trigone, hard palate, and inner part of lips[5]. More than 90% of OCC originates from the squamous tissues, hence widely known as oral cavity squamous cell carcinoma (OCSCC) [6]. The contributing factors for the development of OCC are the consumption of tobacco products in smoke or smokeless form. Moreover, low socioeconomic status, self-negligence, and lack of awareness are the key factors for OCC to occur[7]. The association of viruses and cancer was well documented. More than 20% of cancer prevalence worldwide could be related toinfectious agents, including viruses, bacteria, and parasites. Over 15% of these cases are associated with viruses[8]. Human papillomavirus (HPV) is an epitheliotropic oncogenic DNA virus rarely found in oral mucosa, probably due to saliva clearance[9]. Nevertheless, more than 200 genotypes were detected in the oral mucosa[10]. HPV infection of the oral mucosa may result in benign or malignant disease. The role of HPV as an independent risk factor in oral carcinogenesis was well recognized in the literature[4]. HPV positivity claimed to override traditional prognostic indicators such as tumorgrade and histological subtype[11]. However, other studies reported a non-significant association between HPV and oral cancer, suggesting its presence to be merely an incidental finding[12].

OCC is generally observed in people aged above 40 years compared to younger ones. Worldwide, a higher prevalence of OCC occurs in males than females, 5.8 vs. 2.3 per 100,000 [13].

Case presentation

A 55 years old female patient, with a chief complaint of pain and burning sensation while having food for a week reported to theDepartment of Oral Medicine and Radiology in Divya Jyoti College of Dental Sciences and Research,

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Modinagar. Patient was edentulous and was wearing an ill fitted denture for past 5 years. Her medical history was unremarkableexcept for smoking tobacco for last 20 years. Intraoral clinical examination revealed a white cauliflower like surface with irregular boundaries and induration about 1.5 mm X 2mm in its maximum dimensions involving the anterior region of floor of the mouth opposite extending from canine to premolar region(figure 1). Bilateral suspicious lymph nodes were tender and fixed.

Excisional biopsy was done to rule out an oral squamous cell carcinoma of floor of mouth.

Figure 1:- Pre-operative pic.

Histopathological examination in H&E stained section shows proliferating epithelial islands and nests invading the underlying connective tissue. These tumour cells are large in size demonstrating anaplastic features like altered N/C ratio, nuclear and cellular pleomorphism, individual cell keratinisation, vesicular nuclei with prominent and multiple nucleoli. Many atypical mitotic figures are also seen scattered between these tumour cells. Keratin pearls of varying size and shape surrounded by anaplastic cells are also seen. There is florid lymphocytic infiltration of the connective tissue. Blood vessels of varying size and shape many engorged with RBC's can also be appreciated.Above features were indicative of moderately differentiated Oral Squamous cell carcinoma.(figure 2). Hence the final diagnosis of Oral Squamous Cell carcinoma is established.

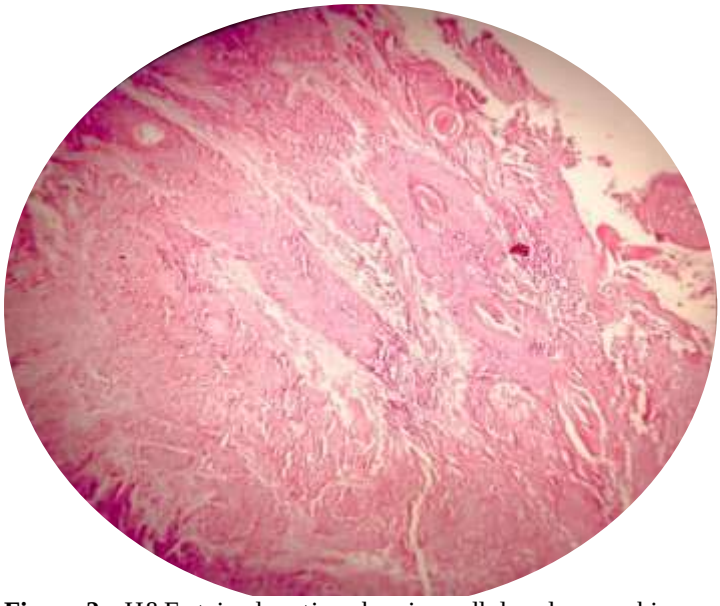

Figure 2:- H&E stained section showing cellular pleomorphism.

Discussion:-

The higher risk of carcinoma reported in elderlypatients can be related to genetic susceptibility contributing to thephenotype[14]. The multifactorial etiology of OSCC includes tobacco, alcohol, and betel quid with and without added tobacco as the major risk factors[15]. In addition to these known risk factors, several associated risk factors have been suggested for OSCC. These include microbes, diet, socio-economic status, and occupational carcinogenic substances, etc. Factors having a controversial role with limited and in-consistent evidence in OSCC etiology include ethnicity and race, oral hygiene and dentition, environmental, genetic, marijuana smoking, khatchewing, nicotine replacement therapy, HIV infections, and alcohol in mouthwashes[16]. Among these, much importance is given to microbes, while the other potential risk factors remain relatively unexplored. One such poorly-explored factor is chronic mechanical irritation (CMI). There have been cases reported with OSCC developing in an oral site with a history of CMI. The role of inflammation in carcinogenesis is often underplayed, despite its inclusion as the 7th hallmark of cancer since 2009[17]. CMI of the oral mucosa is the result of repeated, low-intense action of an oral deleterious agent such as sharp teeth, ill- fitting dentures, and functional alterations, separately or in combination causing sustained trauma[18]. There are three types of CMI factors: Dental (malpositions, sharp/broken teeth, and/or rough or defective restorations); prosthetic (ill-fitting dentures, rough/sharp/overextended flanges, andlack of retention/stability); and functional (swallowing, occlusaland other dysfunctional disorders) [19]. Thus, it is plausible that the chronic inflammation caused by intra-oral factors such as ill-fitting dentures, sharp teeth could be associated with increased risk of oral cancer. CMI with or without associated factors such as tobacco and alcohol was found to exhibit a significant correlation with OSCC[20]. Considering the effect of tobacco on oral mucosal immunity, Johnson et al. in their in-vitro study have reported that nicotine increases the secretion of inflammatory cytokines IL-1, IL-6, IL-8, TNF and McP-1 gingival keratinocytes and hGFs[21]. Furthermore, in the epithelial cells, tobacco causes reduction in epithelial barrier function, reduction ofmucosa secretion, alteration of cytokine production, alteration of several receptor ligand expression reduces barrier function, increases mucus production, modifies cytokine/chemokine production, alteration of receptor/ligand, reduction in phagocytic activities, increased inflammation, and lymphocyte function[22]. Thus, it can be inferred that tobacco exposure on a chronicallyirritated mucosa tobacco can aggravate inflammation further thereby promoting carcinogenesis. Chronic irritation of oral mucosa may interfere with the oral microbiome, thus causing an imbalance in oral homeostasis. Pang et al. have reported the link between the oral microbiome, the epithelial barrier, the immune system, and chronic inflammation in an oncogenic parallelogram[23]. Furthermore, the altered oral microbiomedrives the chronic inflammation that may precede OSCC, and alters host cell response. The dental factors such as sharp teeth, malocclusion, and prosthetic factors such as ill-fitting denture and lack of stability had a strong association with OSCC[24]. Lockhart et al.[24] reported that all intra-oral malignancies arose at the areas in contact with teeth and/or appliances. Studies by Jain et al.[25]and Vaccarezza et al.[26] reported the association of OSCC with ill-fitting dentures. Furthermore, they reported the association between recurrent sores and OSCC. The narrative review by Piemonte and Lazos used the Bradford Hill Criteria to provide an in-depth assessment of the association between CMI and OSCC. Considering whether inflammation as a result of CMI could be an independent risk factor in oral carcinogenesis, it has to be understood that in a multifactorial disease setting, a promoter carcinogen could be considered a potentiating rather than a triggering cause. Furthermore, as previously described in multistep carcinogenesis process, initiation alone does not produce OSCC, and requires promotion subsequent to initiation. Therefore, the triggering factor could be the promoter factor, with the initiating factor being a predisposing cause. Thus, CMI can also be considered an effect modifier or enhancer, and hence controlling this factor in high risk population can reduce the incidence, morbidity and mortality rates of OSCC.

Conclusion:-

Considering the high morbidity and mortality rates of OSCC, strategies to prevent the disease is the need of the hour. Carcinomas associated with tobacco-related habits can be prevented by counselling and de-addiction programs, however, the other risk factors such as microbes, chronic trauma can be prevented by routine dental examination and adequate prophylactic measures.

Close observation, multiple biopsies,early detection, precise diagnosis, and a multidisciplinary teamapproach are all of paramount importance.

Consent-

Patient was explained about the lesion, and informed consentwas taken.

Conflicts of interest –
The authors declare that they have no conflicts of interest.

References:-

1. M. Du, et al., Incidence trends of lip, oral cavity, and pharyngeal cancers: global burden of disease 1990-2017, J. Dent. Res. 99 (2) (2020) 143–151
2. Global Cancer Observatory. https://gco.iarc.fr/ (accessed June 24, 2021).
3. D.M. Parkin, et al., Estimating the world cancer burden: Globocan 2000, Int. J.Cancer 94 (2) (2001) 153–156.
4. S. Marur, et al., HPV-associated head and neck cancer: a virus-related cancer epidemic, Lancet Oncol. 11 (8) (2010) 781–789.
5. Network NCC. NCCN Head and Neck Cancer Series: Oral cancer. NCCN Global Guidel 2018:169–73.
6. Rivera C. Essentials of oral cancer. Int J ClinExpPathol 2015;8:11884–94.https://doi.org/10.5281/zenodo.192487.
7. Reich M, Licitra L, Vermorken JB, Bernier J, Parmar S, Golusinski W, et al. Best practice guidelines in the psychosocial management of hpv-related head and neck cancer: Recommendations from the european head and neck cancer society's make sense campaign. Ann Oncol 2016;27(10):1848–54.
8. H. ZurHausen, The search for infectious causes of human cancers: where and why, Virology 392 (1) (2009) 1–10
9. G. Campisi, L. Giovannelli, Controversies surrounding human papilloma virus infection, head & neck vs oral cancer, implications for prophylaxis andtreatment, Head Neck Oncol. 1 (2009) 8.A. Psyrri, D. DiMaio, Human papillomavirus in cervical and head-and-neck cancer, Nat. Clin. Pract. Oncol. 5 (1) (2008) 24–31.
10. N. Husain, A. Neyaz, Human papillomavirus associated head and neck squamous cell carcinoma: controversies and new concepts, J. Oral Biol. Craniofac. Res. 7 (3) (2017) 198–205.
11. N. Husain, A. Neyaz, Human papillomavirus associated head and neck squamous cell carcinoma: controversies and new concepts, J. Oral Biol.Craniofac. Res. 7 (3) (2017) 198–205.
12. R.E. Marx, D. Stern, Oral and Maxillofacial Pathology: a Rationale for Diagnosis and Treatment, Quintessence Pub. Co., Hanover Park, IL, 2012.
13. Nocini R, Lippi G, Mattiuzzi C. Biological and epidemiologic updates on lip andoral cavity cancers. Ann Cancer Epidemiol 2020;4:1.
14. W. Liu, et al., Malignant transformation of oral verrucousleukoplakia: a clinicopathologic study of 53 cases, J. Oral Pathol. Med. 40 (4) (2011) 312–316
15. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Personal Habits and Indoor Combustions. IARC Monographs on the Evaluation of Carcinogenic. Vol. 100E . Risks to Humans; 2012. p. 46-167.
16. Conway DI, Purkayastha M, Chestnutt IG. The changing epidemiology of oral cancer: Definitions, trends, and risk factors. Br Dent J 2018;225(9):867.
17. Singhvi HR, Malik A, Chaturvedi P. The role of chronic mucosal trauma in oral cancer: A review of literature. Indian J Med PaediatrOncol 2017;38(1):44-50.
18. Grinspan D. Diseases of the Mouth: Semiology, Pathology, Clinical and Therapeutic of the Oral Mucosa. Mundi, Buenos Aires 1970.
19. Piemonte ED, Lazos JP, Brunotto M. Relationship between chronic trauma of the oral mucosa, oral potentially malignant disorders and oral cancer. J Oral Pathol Med 2010;39(7):513-7.
20. Lazos JP, Piemonte ED, Lanfranchi HE, BrunottoMN.Characterization of chronic mechanical irritation in oral cancer. Int J Dent 2017;2017:6784526 [42] Almasri A, Wisithphrom K, Windsor LJ, Olson B. Nicotine and
21. lipopolysaccharide affect cytokine expression from gingival fibroblasts. J Periodontol 2007;78(3):533-41.
22. Jaspers I. Cigarette smoke effects on innate immune mechanisms in the nasal mucosa: Potential effects on the microbiome. Ann Am ThoracSoc 2014;11(Suppl 1):S38-42.
23. Pang X, Tang YJ, Ren XH, Chen QM, Tang YL, Liang XH. Microbiota, epithelium, inflammation, and TGF-β signaling: An intricate interaction in oncogenesis. Front Microbiol 2018;9:1-10.
24. Lockhart PB, Norris CM, Pulliam C. Dental factors in the genesis of squamous cell carcinoma of the oral cavity. Oral Oncol 1998;34(2):133-9.
25. Jain P, Jain M, Prasad BV, Kakatkar GS, Patel M, Khan J. A case-control study for the assessment of correlation of denture-related sores and oral cancer risk. J Contemp Dent Pract 2016;17(11):930-3.
26. Vaccarezza GF, Ferreira Antunes JL, Michaluart-Júnior P. Recurrent sores by ill-fitting dentures and intra-oral squamous cell carcinoma in smokers. J Public Health Dent 2010;70(1):52-7.