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Review Article

Oral cancer cause and concern- A review

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ABSTRACT

The most common type of cancer in the head and neck area and a global health concern is oral cancer. Oral cancer's precise cause is uncertain. Iron deficiency anemia, alcohol, tobacco, immunologic sensitivity, gene mutations, epithelial cell proliferation, suppressor proteins, and chemotherapy-related diseases, such as lymphoma and leukemia, are among the many risk factors for cancer. There have also been reports of viruses playing a part in the etiology. In certain locations, dietary variables including high fat and low fiber may contribute to carcinogenesis. Because alcohol consumption has been linked to an increased risk of oral cancer, a high prevalence of alcoholism is significant. It has also been suggested that other signaling factors play a part.

1. Introduction

Neoplasms are self-replicating new growths. They develop on their own and are not constrained by the same biological factors that govern the formation of other cells. They always have a negative impact and kill the host. The two main categories of neoplasms are benign and malignant.[1] Any cancer that develops from oral tissues is referred to as "oral cancer".[2] The greatest type of cancer that falls within the head and neck category is oral cancer. It is a global health issue, with over 640000 new cases reported.[3] With 10% of the estimated 644,600 new malignancies that arise in all areas of the body each year, oral cancer is a serious health issue in India. Oral cancer ranks first to sixth among all cancers in India based on the annual age-adjusted incidence rate. Depending on the kind and degree of tobacco use, oral cancer is more common in males than in women, with the maximum incidence occurring in the sixth decade. Tobacco use is responsible for 90% of mouth cancer cases in Southeast Asia, according to the WHO.[4] The survival rate of mouth cancer is significantly increased by early identification, which occurs when lesions are less than 3 cm in size and do not exhibit signs of deep invasion or metastasis. Unfortunately over the past few decades there has been only small improvement in the early diagnosis of oral cancer, over 50% of all cancer cases are extensive, late stage malignancies at the time of diagnosis/Hence treatment is aggressive which increases morbidity and result only in slight improvement in survival rate. Therefore early diagnosis of a lesion during the localized stage combined with adequate treatment appears to be the most effective way to further improve oral cancer control.[5] Of the total oral cancer, carcinomas account for 96%and sarcoma 4%. Oral tongue squamous cell carcinoma is one of the most common sites of head and neck cancer. Its normal epidemiology shows a persistent trend of an increase in incidence among young people in some regions of the world.[6]

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2. Discussion

2.1. Etiology and predisposing factors

Oral cancer's precise cause is uncertain. Differences in exposure to carcinogenic initiators or promoters may have an impact on the incidence rate variation among various groups or populations. Alcohol, tobacco, immunologic sensitivity, gene mutations, development of epithelial cells, suppressor proteins, and chemotherapy-related diseases, such as lymphoma and leukemia, are among the many risk factors for cancer.[7] One of the earliest locations for the signs and symptoms that lead to the diagnosis of these conditions may be the mouth. It is unclear if the difference in some immunological variation causes or results from oral cancer in people in the general population who do not otherwise have or are at risk of immunodeficiency.[8]

2.2. Tobacco

In addition to its impact on mortality, smoking raises the rate of morbidity, making it a global endemic that causes severe illness and other health issues. In addition to being addictive, tobacco is known to have other negative health effects. Nicotine, the primary addictive agent, is found in alkaloids found in both smoked and smokeless tobacco. They also include thousands of chemical compounds that are carcinogenic in addition to being irritants and poisons. Tobacco-specific nitrosamine polycyclic aromatic hydrocarbons, among many others, are the most specific carcinogens.[9]

2.3. Tobacco habit in India

There are many diverse forms of tobacco use, many of which are unique to particular regions of India. Tobacco was introduced for a variety of reasons. Tooth-related complaints account for 48.1% of the most common reasons, with peer group influence coming in second at 38%. For women, tooth-related problems accounted for 92.1% of the most common reasons, whereas for men, peer group influence was more significant at 58.1%.[10]

2.4. Oral cancer risk and smoking

Epidemiologic studies that show that the number of smokers among patients with mouth cancer is more than double that of the control group have solidly proven the link between cigarette smoking and oral carcinoma. At the University of California, San Francisco, a study of 403 individuals with oral and pharyngeal cancer who were monitored for an average of 5.1 years revealed that 72.1% smoked and 58.1% smoked more than one pack per day. The follow-up analysis revealed that nearly one patient (17.7%) acquired second primary oral and oro-pharyngeal cancer in a mean of five years, indicating the extremely high risk to tobacco users. There are several types of smoked tobacco, including cigarettes, bidis, chuttas, reverse chuttas, dhuntis, and hooklis. Smokeless tobacco is in the following form pan, pan masala, main puri, mava, tobacco lime preparation.[10]

2.4.1. Alcohol

Because alcohol consumption has been linked to an increased risk of oral cancer and a higher than predicted mortality, a high rate of alcoholism is significant. A study conducted more than 30 years ago found that excessive alcohol use was a major contributing factor to the development of oral cancer.In this study, compared to 12% of the control group, one-third of the 543 male patients drank more than 7 ounces of whisky daily. One team of researchers discovered that there was clear proof of alcoholic cirrhosis in 44% of 108 patients with tongue cancer and 59% of 68 patients with tonsilar fossa, palate, and floor of mouth cancer.[11] Most heavy drinkers are also smokers and their predisposing factors probably work in combination rather than independently.[12]

Oral lichen planus: is a persistent, complicated inflammatory illness. Both the skin and mucosa may be impacted. The most frequent site is the buccal mucosa. OLP has been linked in reports to malignant transformation. According to a UK study, 37% of 241 OLP patients experienced a carcinoma transition within 12 years. The author of this evaluation of research conducted since 1981 noted that cancers linked to oral lichen planus ranged from 0.4% to 5.6% in seven different nations.[13]

2.4.2. Denture wearing

Various studies show no difference between denture wearers and control group in the occurrence of oral cancer.[14]

2.4.3. Nourishment

In certain locations, dietary variables including high fat and low fiber may contribute to carcinogenesis. Antioxidant, vitamin, and nutritional deficiencies have been proposed as co-factors in carcinogenesis; in particular, low fruit and vegetable intake has been linked to cancer of the mouth, larynx, and esophagus. Flavonoids, rutin, isothiocynates, indoles, and phenolic antioxidants are active components found in fruits and vegetables that may have suppressory effects. Oral precancerous lesions and eventual cancer have been linked to low dietary and/or blood levels of vitamin A. Because beta-carotene, a precursor to vitamin A, has not proved successful in controlling pre-malignant oral lesions, this association has been established on the correlation between vitamin A deficits and hyperkeratosis.[15] In a recent study, it was found that consumption of fruits and vegetables is associated with a reduced risk of oral cancer.[16]

The Plummer-Vinson syndrome, which includes iron deficient anemia, has been linked to a higher risk of tongue cancer. Although high iron storage has been suggested as a potential sign or cause in cancer, carcinoma of the tongue,

iron deficiency has not been demonstrated to be a prevalent finding in people presenting with oral cancer. Although high iron storage has been suggested as a potential sign or cause in cancer 19, iron insufficiency has not been demonstrated to be a prevalent finding in patients presenting with oral cancer.[17]

2.4.4. Virus

There has long been conjecture regarding the involvement of viruses in the genesis of mouth cancer. The precise involvement of viruses remains somewhat speculative after years of clinical observation and investigation.

1. The herpes virus: The herpes simplex virus infects human tissue in eight different forms. Human herpes virus 6, 7, 8, (HHV-6, HHV-7, HHV-8) Varicella zoster virus (VZV), human cytomegalovirus (CMV), Epsteinbarr virus (EBV), and herpes simplex virus types 1 (HSV1) and 2 (HSV2).
2. Herpes simplex virus type 1: This virus is known to cause recurrent herpes labialis and primary herpes stomatitis. The virus can shift from cells to a malignant phenotype under some situations, despite the fact that it is extremely cytolytic and effectively kills infected cells.
3. Burkett lymphoma in Africa is most likely caused by Epstein-Barr virus (EBV), which is also closely linked to nasopharyngeal cancer in a number of Asian nations. EBV causes hairy leukoplakia everywhere in the world. It appears unlikely that EBV contributes in any way to the development of oral cancer because hairy leukoplakia does not seem to be associated with the development of oral cancer.
4. Human herpes virus 6: HHV6 may contribute to HIV since it can enhance the expression of multiple HIV activities. It first appeared in patients with acquired immunodeficiencysyndrome.
5. Human herpes virus 8: This is the most recent human herpes virus to be identified in Kaposi's sarcoma lesions linked to AIDS.
6. Papilloma virus: around 70 different types of this virus have been discovered. Types 16 and 18 are the main human papillomaviruses that pose a high risk.[18]

2.5. Other causes attributed to causes of cancer are

Repression of G protein coupled receptor family C group 5 member A associated with pathologic differentiation grade of oral squamous cell carcinoma.[19]

TGF-beta1, Smad-2/3, Smad-1/-5/=8 and Smad-4 signaling factors are expressed in ameloblastomas, adenomatoid odontogenic tumor, and calcifying cystic odontogenic tumor.[20]

Expression of human epidermal growth factor receptor (HER), especially EGFR and HER4 in odontogenic tissue suggesting that growth signals mediated by these receptor molecules contribute to cell proliferation ,survival and differentiation in both normal and neoplastic odontogenic epithelial tissues.[21]

Syndecan-1(SD 138) expression by the tumors stroma is considerd to be associated with poor prognosis of ameloblastoma, KCOT, and dentigerous cyst.[22]

Expression of Prox-1 in the neoplastic spindle cells supports the lymphatic differentiation in oral kaposi's sarcoma.[22]

Elevated level of serum growth differentiation factor 15 is associated with oral leukoplakia and oral squamous cell carcinoma.[23]

Increased alpha v beta 6 integrin and MMP-3 expression and collagen fiber changes in human oral squamous cell carcinoma and is related to unfavorable prognosis.[23]

3. Conclusion

Dentists must be aware of the main risk factors that are suitable for the community in order to prevent oral malignancies. Evidence on recognized and changeable risk factors is included in this review. It is crucial to take into account reported disagreements because treatments ought to be supported by solid scientific evidence. In order to enable dentists to ignore characteristics for which there is either insufficient or no suggestive epidemiological evidence, this review offers scientific information based on current evidence. In order to ensure that patients and the general public receive accurate information and to facilitate the development of prevention efforts, it is crucial to dispel several misunderstandings regarding factors deemed irrelevant to this illness. A multimodal strategy that integrates health education, tobacco and alcohol control, early detection, and early treatment is needed to reduce the burden of this eminently preventable cancer.

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5. Conflict of Interest

None.

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