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PREVENTION AND CHEMOPREVENTION IN ORAL CAVITY CANCER: AN INDIAN RADIATION ONCOLOGIST PROSPECTIVE

Oncology

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ABSTRACT

Recently enormous interest is being created in the prevention of oral cavity cancer in view of that cancer and cardiovascular disease is being clubbed by government of India about prevention and control program. Epidemiological studies shown that a diet rich in vegetables and fruits is directly associated with decrease risk of cancer and cardiovascular diseases, furthermore studies revels that use of antioxidants such as Ascorbic acid vitamin C , Tocopherol vitamin E, β carotene (Pro vitamin A), Selenium and other micronutrients play important role in prevention of oral cavity cancer .

KEYWORDS

Antioxidants, micronutrients, Oral cavity cancer.

DISCUSSION:

The Prevention is being divided in three categories Primary, secondary and tertiary prevention, Primary prevention of oral cavity cancer is all about avoiding known carcinogens, it has been seen that after stopping smocking and use of alcohol the risk of developing oral cavity typical squamous cell carcinoma reduces to great extent. After avoiding smocking smoking for 1-3 years, there has been 30% reduction in risk of developing typical head and neck cancer compared to those who continue smoking. Secondary Prevention of cancer involves early detection of cancer by means of screening methods on the population at the risk, hence there is a need for increased awareness about early signs and symptoms of potentially malignant oral cavity lesions, The American cancer society has recommended that doctors must examine the oral cavity and throat as a part of routine general check up of any patient. Tertiary prevention is the prevention in the patients already treated for malignancy, regular follow up is strongly recommended by National Comprehensive Cancer Network (NCCN) , for such patients after curative treatment particularly to detect early Recurrence , Second primary or for the complications after radical treatments like after radiotherapy , surgery and chemotherapy .

Criteria for Diagnosis of Multiple Cancer:

It was Warren and Gates who first introduced criteria to diagnose multiple primary cancer in oral cavity which was further modified by Hong at al , for diagnosis of multiple primary carcinomas , the criteria's are give below are:

1. Cancer must be distinct and anatomically separate; means cancer must be multi centric in nature.
2. It must have occurred 3 years after the first diagnosis or it should be separate from first tumor by at least 2cm from the normal healthy oral mucosa.

Definition of Synchronous and Metachronous Cancers:

Synchronous tumor refers in which the second primary cancer is diagnose within six month of diagnosis of primary tumor however Metachronous tumors define in which the second primary cancer diagnose after more than six months of diagnosis of first primary cancer .

Field Cancerization: The concept of field cancerization was introduced by Slaghuter at al in 1953, He has suggested that multiple primaries develop in the area receiving similar insult to oral cavity mucosa due to similar carcinogens exposure.

Chemoprevention: The interesting was presented by Michel B Sporn It refers to prevention of reversal, suppression, or transformation of a premalignant lesion to get converted into invasive carcinomas with the help of natural or synthetic chemicals. Chemoprevention for head and neck cancers denotes prevention of development of both synchronous and metachronous primaries for the upper aero digestive tract and lung as well. Different class of natural products and targeted agents such as retionoid receptors legends, selective cyclooxygenase, inhibitors, peroxisome, as well as epidermal growth receptor EGFR inhibitors all these agents have been studied in details.

Table: Inhibitors of carcinogen

Blocking agents	Suppressing agents
Phenols	Retinoids
1-Ellagic acid	Carotenoids
2-Caffeic acid	Selenium salts
3-Ferulic acid	Proteases inhibitors
4- p-hydroxycinnamic acid	Inhibitors of arachidonic acid metabolism
5-Indols	Cyanates and isocyanates
6-Aromatic isothiocyanates	Phenols

Retinoids: Retionoids are naturally occurring and synthetic vitamin A metabolites and analogs that binds to retinoic acid receptors and promote cell differentiations and decrease cell proliferation and apoptosis. The loss of nuclear RARβ is an early event observed in premalignant/dysplastic lesion lesions. Targeting the retinoid signaling pathways therefore could act as a strategy for chemoprevention.

Curcumin: Curcumin (Diferuloymethane) is a polyphenol compound which plays anticancer roles due to its inhibitory properties of tumor initiation and tumor promotion . curcumin act through many intracellular signal transduction pathways at different levels such as transcriptions factors and nuclear factor transcription .

Lycopene: Lycopene is an effective Antioxidant with property of modifying intercellular exchange junctions, and hence preventing Cancer.

Cyclooxigenase Inhibitors: Cyclooxigenase -2(COX2) is commonly seen to be over expressed in oral dysplasias and squmous cell carcinomas of oral cavity cancers . Some clinical studies indicate that COX2 inhibitors could have some chemopreventive role. But due to the cardiovascular toxicities of these COX-2 inhibitors do have disappointing results.

Green Tea: The polyphenol, Epigallocatevhin3 gallate (EGCG) is known antioxidant , has been found in green tea extract . EGCG can modulate multiple signaling pathways including inhibition of receptor tyrosine kinase and their downstream pathways like inhibition of NF kappa-B, and activation of p53 pathway.

P53-Targeted Agents: p53 mutations founds in 47-62% of cancers of head and neck region.

EGFR Inhibitors: Epidermal growth factors receptor is known to be over expressed in malignant , premalignant and also normal tissues of patients of head and neck cancers , EGFR expression increases with the increasing degree of dysplasia and is profoundly increased in more than 90% cases of head and neck cancers as well as its sub site of oral cavity cancers . Erlotinib decreases the incidence of oral squamous cell carcinoma as seen in many studies.

CONCLUSIONS:

There is high incidence of oral cavity cancer in the countries like India especially where Tobacco, Pan-Masala, Areca nut chewing still

remains as customary in the larger populations, the high incidence of second primary almost 15% is seen in oral cavity cancer, Chemoprevention the use of natural or synthetic chemicals for reversal suppression or prevention for conversion of premalignant lesions of oral cavity into invasive cancer.

Conflict of Interest: None.

REFERENCES:

1. R. L. Siegel, K. D. Miller, and A. Jemal, "Cancer statistics, 2018," CA: A Cancer Journal for Clinicians, vol. 68, no. 1, pp. 7–30, 2018.
2. R. L. Siegel, K. D. Miller, and A. Jemal, "Cancer statistics," CA: A Cancer Journal for Clinicians, vol. 66, no. 1, pp. 7–30, 2016.
3. K. D. Miller, R. L. Siegel, and C. C. Lin, "Cancer treatment and survivorship statistics, 2016," CA: A Cancer Journal for Clinicians, vol. 66, no. 4, pp. 271–289, 2016.
4. P. Boscolo-Rizzo, C. Furlan, V. Lupato, J. Polesel, and E. Fratta, "Novel insights into epigenetic drivers of oropharyngeal squamous cell carcinoma: Role of HPV and lifestyle factors," Clinical Epigenetics, vol. 9, no. 1, p. 124, 2017.
5. C. Lindsay, H. Seikaly, and V. L. Biron, "Epigenetics of oropharyngeal squamous cell carcinoma: opportunities for novel chemotherapeutic targets," Journal of Otolaryngology - Head and Neck Surgery, vol. 46, no. 1, pp. 1–10, 2017.
6. J. Ferlay, E. Steliarova-Foucher, J. Lortet-Tieulent et al., "Cancer incidence and mortality patterns in Europe: estimates for 40 countries in 2012," European Journal of Cancer, vol. 49, no. 6, pp. 1374–1403, 2013.
7. G. Wright and M. Y. Morgan Dr., "Alcohol and tobacco misuse: Reducing aerodigestive cancer risk," World Journal of Hepatology, vol. 5, no. 8, pp. 452–457, 2013.
8. M. Hashibe, P. Brennan, S.-C. Chuang et al., "Interaction between tobacco and alcohol use and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium," Cancer Epidemiology, Biomarkers & Prevention, vol. 18, no. 2, pp. 541–550, 2009.
9. W. Smith and N. Saba, "Retinoids as chemoprevention for head and neck cancer: Where do we go from here?" Critical Review in Oncology/Hematology, vol. 55, no. 2, pp. 143–152, 2005.
10. T. Tanaka and R. Ishigamori, "Understanding Carcinogenesis for Fighting Oral Cancer," Journal of Oncology, vol. 2011, Article ID 603740, 10 pages, 2011.
11. D. V. Messadi and K. Sato, "Oral Cancer Chemoprevention: Current Status and Future Direction," J Calif Dent Assoc, vol. 44, no. 2, pp. 101–11, 2016.
12. J. A. Monteiro De Oliveira Novaes and W. N. William, "Prognostic factors, predictive markers and cancer biology: The triad for successful oral cancer chemoprevention," Future Oncology, vol. 12, no. 19, pp. 2379–2386, 2016.
13. E. L. Yanik, H. A. Katki, M. J. Silverberg, M. M. Manos, E. A. Engels, and A. K. Chaturvedi, "Leukoplakia, oral cavity cancer risk, and cancer survival in the U.S. elderly," Cancer Prevention Research, vol. 8, no. 9, pp. 857–863, 2015.
14. J. Reibel, "Prognosis of oral pre-malignant lesions: significance of clinical, histopathological, and molecular biological characteristics," Critical Reviews in Oral Biology and Medicine, vol. 14, no. 1, pp. 47–62, 2003.
15. H. M. Mehanna, T. Rattay, J. Smith, and C. C. McConkey, "Treatment and follow-up of oral dysplasia—a systematic review and meta-analysis," Head & Neck, vol. 31, no. 12, pp. 1600–1609, 2009.
16. W. N. William Jr., "Oral premalignant lesions: Any progress with systemic therapies?" Current Opinion in Oncology, vol. 24, no. 3, pp. 205–210, 2012.
17. L. W. Wattenberg, "Chemoprophylaxis of carcinogenesis: a review," Cancer Research, vol. 26, no. 7, pp. 1520–1526, 1966.