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Human Papilloma Virus and Oropharyngeal Cancer-West and the East

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Head and neck squamous cell carcinoma is among the common malignancies, particularly in developing countries. Carcinogens in the tobacco are considered as the most common etiology for this disease, with synergistic action of alcohol in some cases. Over the past two decades, there is an increase in incidence of oropharyngeal squamous cell carcinoma in Europe and America, while the incidence of oral, laryngeal and hypopharyngeal cancer is showing a downward trend[1]. This rise is commonly seen in otherwise healthy young individuals who are non-smokers. Epidemiological and molecular studies have confirmed that human papilloma virus (HPV) is the cause of such cancers. Gillison described the causal association of HPV with oropharyngeal squamous cell carcinoma[2].

HPV is an epitheliotropic double stranded DNA virus with more than 120 genotypes identified. HPV-16 is most common genotype associated with Oropharyngeal cancers. Other high risk genotypes which can cause malignant change are HPV-18, HPV-31 and HPV-33. Expression of the viral oncoproteins E6 and E7 causes degradation and inactivation of p53 and retinoblastoma (Rb) proteins, respectively. The E7-induced downregulation of Rb leads to the overexpression of p16 in an unsuccessful attempt to stop cell proliferation. Expression on p16 is a reliable surrogate marker for HPV associated carcinoma. Studies on HPV associated Head and Neck cancers are based on immunohistochemistry for p16 expression or identification HPV genome by Polymerase chain reaction and In situ hybridization[3]. p16 specifically identifies transcriptionally active HPV in the Tumours. Studies from the west show a significantly high prevalence of HPV associated oropharyngeal carcinoma, whereas there are very limited studies from rest of the world[1]. Studies from India and Japan have shown p16 positivity in the range of 20% to 30% only[3,4]. However, even in these countries, the rates appear to be increasing over the years. In the presence of high consumption of tobacco products, tobacco induced promoter hypermethylation of p16 gene silences the function and prevents overexpression of p16. Hence p16 positivity becomes less reliable as a surrogate marker of HPV infection in the presence of high tobacco burden[3].

HPV associated oropharyngeal cancers, in the west, generally occur in younger healthy individuals with no or very less exposure to tobacco. Lymphoepithelial sites, tonsil and base of tongue are the common sites of these tumours. They generally present early when the tumour is small, but with large neck secondaries. Response to treatment is excellent in these tumours, and these patients have a better prognosis than Non-HPV associated cancers. Those patients with HPV associated oropharyngeal carcinoma are less likely to have a second primary. Considering the excellent prognosis of patients with HPV associated Oropharyngeal carcinoma, even when presented with large neck secondaries, American Joint Committee on Cancer has modified the staging for these tumours in the 8th edition of the manual, with effect from 2018[5]. There is a need for improved surveillance for HPV in squamous cell carcinoma of the oropharynx, so that the treatment may be de intensified in HPV positive cases.

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