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The Role of The Human Papilloma Virus in Head and Neck Squamous Cell Carcinoma

Human Papilloma Virüsünün Baş Boyun Skuamöz Hücreli Karsinomundaki Rolü

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Head and neck squamous cell carcinoma (HNSCC) including oral squamous cell carcinoma (OSCC) is the sixth most common cancer worldwide.[1,2] Despite advances in diagnosis and treatment of the disease, the mortality rates still remain high, leading to more than 200,000 deaths per year.[2] Even though early stage OSCC can often be cured with surgery and adjuvant radiation therapy, the 5-year survival of patients with locally advanced disease is 48-50% and even lower for metastatic disease (26%).[2]

Most common risk factors of OSCC are tobacco use and alcohol consumption, but infection by high-risk genotypes of human papilloma virus (HPV) has been presented recently as a new risk factor, more specifically for the lesions in younger adults which are located at the oropharynx, including the base of tongue, soft palate, tonsils, and pharyngeal wall.[1,2,3] The prevalence of HPV infection in oropharyngeal SCC (OPSCC) has been approximated to vary between 45 to 90% [2], increasing the prevalence of HPV associated HNSCC in the United States, Western Europe, Canada and Australia.[2,3]

It's revealed that oral mucosa has a prevalence of HPV ranging from 0.6% to 81% and it may act as a source both for HPV infections and recurring HPV-associated lesions. [1] HPV is a non-enveloped, double-stranded, circular DNA virus with a double-stranded DNA molecule. [1] The open reading frames located at the early region of the genome encodes proteins E1, E2, E4, E5, E6, and E7 both to provide replication and cellular transformation, and to control viral transcription.[1] When HPV DNA enters into the host cell genome, E6 and E7 genes are transcribed to stimulate cell cycle progression during productive infection. [2] Since the E6 and E7 proteins are also highly effective in blocking the negative cell cycle regulators such as p53, Rb1 and Rb2, they are attributed to cancer development in high risk papillomavirus. [2] Genotypic variations in the DNA base sequences of E6 and E7 determine the type of HPV and establish the oncogenic phenotype into high and low-risk types: high risk includes HPV-16, 18, 31, 33, 35, 45, 51, 52, 56, 58, 59 and low risk contains HPV 6, 11, 42, 43, 44. [1] High risk HPV 16 is the dominant subtype that has been identified in OPSCC, and is thought to represent 90% of OPSCC cancers that are HPV positive .[2]

The HPV transmission to the oral cavity can be via sexual contacts, perinatal transmission of the neonate during birth [1] and a personal history of cervical HPV infection causing autoinfection. [3] Patients with HPV-related OSCC are more commonly younger males, have a higher socioeconomic status compared to HPV-unrelated OSCC patients [3] and mostly present at a higher stage, with large metastatic lymph nodes. [1,4]

HPV-positive tumors in head and neck area present

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better prognosis compared with those that are HPV-negative [1,2,4] and have improved response to both radiation therapy and chemotherapy. [2] Specifically, HPV associated OPSCC is associated with a reduced risk of dying and of local regional recurrence (26% and 49%, respectively).[2] Unfortunately, recurrence is observed up to 30% of HPV-related HNSCC patients. [3] All of this information is indicative that HPV associated OPSCC is a distinct clinical entity and currently efforts are being made to develop novel screening methodologies and therapeutics to target HPV associated OPSCC. [2]

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