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RESEARCHARTICLE

DETECTION OF HUMAN PAPILLOMA VIRUS IN ORAL SQUAMOUS CELL CARCINOMA

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ABSTRACT

Oral squamous cell carcinoma is the most common malignant neoplasm of oral mucosa, representing more than 90%. Tobacco and alcohol has been considered as the classical risk factors. Human papilloma Virus has been proposed as an etiological risk factor since 2007. Thirty five cases diagnosed with OC their ages and gender matched with controls were enrolled in this study. Fifty-five un-stimulated whole saliva samples (35 OC and 20 apparently health subjects) were collected. DNA was purified from exfoliate cells to amplify HPV-DNA using HPV-L1 gene sequence primers by polymerase chain reaction (PCR) method, the genotyping was performed using direct sequencing method. Mean age was 52.23±13.73 years in cases (range: 17-70 years) while in controls was 50.55±12.5 years (range: 24-74 years).Forty-six percent (16/35) of OC patients was positive for detection of HPV DNA (P<0.001). The most frequent type in patient group was HPV-18 type accounting for (31%) of cases (P<0.05). The prevalence rate of HPV was significantly higher among younger ages (<50 years) with P=0.042. In addition the prevalence of HPV was higher with other variables with no significant association: male, tongue tissue, and grade I differentiation, and squamous cell carcinoma (P=0.150, P=0.678, P=0.983, ad P=0.765 respectively).

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INTRODUCTION

Oral squamous cell carcinoma (OSCC) is the most common oral malignancy, representingmore than 90% of the malignant tumors, approximately 263,900 new cases and 128,000 deaths by cancer of the oral cavity are estimated to have occurred in the world in 2008 (Jemal *et al.*, 2011). The Established etiological factors of oral cancer (OC) included cigarette smoking and heavy alcohol abuse; however, a growing group of patients, including young adults and women, have no known tobacco or alcohol exposure have been emerged, therefore; possible viral etiologic factors such as oncogenic human papilloma virus (HPV) have been proposed (Rosebush *et al.*, 2011). High-risk HPV-16 and 18, as etiological agents of anogenital carcinomas, have been firmly established in the literatures and due to morphological similarities and epitheliotrophic nature of HPV as well as HPV's oncogenic potential, a link between OC and HPV seemed logical (Syrjänen, 2003).

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International Agency for Research on Cancer (IARC) has acknowledged HPV as an independent risk factor since 2007 and that 30-50% of OSCC has been associated with HPV-16 (IARC, 2007).All HPVs are small DNA viruses with a genome of around 8 kb that consists of double-stranded circular DNA, and which is enclosed in a 52-55 nm viral capsid. The genome is divided into three regions; the early and late regions, and the non-coding control region (NCCR). The early region encodes the E1-E2, E4-E7 proteins responsible for gene regulation, replication, pathogenesis and transformation (ZurHausen, 2006). In HR HPVs, E6 binding and degradation of p53, and E7 binding and inhibition of the retinoblastoma protein (Rb) result in deregulation of cell cycle control, the late region encodes for L1 and L2, the major and minor viral capsid proteins respectively (ZurHausen, 2006). Incidence of HPV(+) OSCC varies greatly worldwide from 25-80% and it is predicted to increase in the near future. This rise in incidence is mostly occurring in individuals aged 40-55 years, without environmental risk factors, and is associated with persistent infection with HR-HPVs (Chaturvedi *et al.*, 2011). HPV(+) OSCC patients tend to be younger than HPV(-) ones (Lajer *et al.*, 2010). HPV-16 is the most common genotype found in almost 90% of the HPV(+) oropharyngeal cancers.