



Significant Role of Viruses in Oral Squamous Cell Carcinoma Patients: A Review

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Abstract

Oral cancers are considered as the 6th most common predominant cancers in males. Some of the etiological components include viruses, alcohol and tobacco. Most common viruses include Hepatitis C virus, Epstein-Barr virus, Herpes simplex virus (HSV) and human papilloma virus (HPV). No doubt, the role of viruses is inevitable in progression and initiation of OSCC (Oral Squamous Cell Carcinoma). Rapid detection of these viruses can result in prevention and early treatment so that disease will not be progressed further.

Keywords: Oral Squamous Cell Carcinoma; Oral cancer; Herpes simplex virus; Hepatitis C

Introduction

OSCC (Oral Squamous Cell Carcinoma) is known to be the 6th most common cancer throughout the whole world. The disease is widespread between smokers in comparison with non-smokers [1]. Research shows that there have been seven times more smokers which have been suffered with oral cancer as compared to non-smokers [2,3]. OSCC is a disease predominant in males but the recent cancer ratio depicts that it is found in females as well. Oral cancers occur at the age of 40 years and above [4-8]. Typically, the disease occurs in floor of mouth and tongue [9-13].

Oral cancer is a multi-step procedure in pathogenesis which leads to normal regulatory pathways disruption that regulate normalized cellular roles including cell death, differentiation and cell division [14,15]. Even though chemical carcinogens such as alcohol and tobacco have been the main etiology. They tend to participate in producing mutations in P53 suppresser genes which lead to tumour progression and initiation. Numerous viruses cause alteration in DNA coding and oral epithelial which result in oral carcinomas [16].

Virus Found In Oral Squamous Cell Carcinoma

Human Papillomavirus (HPV)

HPV (Human papilloma virus) belongs to the family of Papovaviridae, un-wrapped dual standard spherical DNA viruses

[17]. They cause infections and particularly target basal cell of epithelium. Typically, HPV 6,11,16 and 18 are found in cancer epithelial cells [18]. Research indicates that prenatal transmission and oral sex cause infection [19].

The protein of HPV is comprised of a non-structural protein (genes encode E1-E7) and structural protein (late genes encodes L1-L2). Tumour transcription and replication transformation is regulated by early protein and final stages of viral cycle is carried out by late genes. HPV consisting of lower risk result in warts whereas HPV having high risk can cause malignant lesions [17,20,21,22,23]. P53 tumour suppressor gene (TP53) is degraded by the early protein E6 [24]. Q1 transition to S phase is regulated by TP53 in cell cycle [25]. Furthermore, this leads to damaging DNA, proliferation, increased cell and inadequacies can cause malignant transformation [26].

Herpes Simplex Virus (HSV)

HSV1 and HSV2 (Herpes simplex virus) cause infection to genital, ocular and oral areas. They stimulate the cellular protein expression and induce shock proteins [27]. These infections cause cell RNA and cell protein formation. Mechanism of Shut off becomes activated which is fundamentally an area of genome mtr1 of HSV1 and mtr2 of HSV2 respectively. Typically, there are two phases found. Phase 1 is more linked with viral activity which is followed

right after infections [28]. Phase 2 requires expressions of viral genes, causes cell transformation and removes synthesis of host protein [29].

Hepatitis C virus (HCV)

Johnson exposed one of the possible contributions of Hepatitis C virus in oral cavity besides liver which causes genetic factor mutation of cells [30]. Hepatitis C virus is a packed and single strand RNA virus which is infected by detoxification of humanoid factor VIII (Cho et al.) [31]. Viral poly protein breakdown by translation of RNA results in capsid formation, viral proteases (NS2, NS3) and glycoproteins (E1, E2) which seem essential for malignancy and replication of RNA [32].

Epstein-Bar Virus (EBV)

EBV virus is comprised of dual stuck DNA genome which encodes 90 proteins with 172,000 base pairs. The latent proteins of EBV are LMP2 and LMP1 [33]. LMP1 works in B cell destruction and cell transformation. LMP1 with various groups of carboxyl is attracted to act on TRAFs (Tumour Necrosis Factor Receptor) [33-35]. P38, jun, NF- κ B present higher activity in LMP1 due to this interaction representing B cells and epithelial cells [36-38]. It helps to activate apoptotic genetic factors such as fibroblast growth factor (FGF) and metalloproteinase-9 (MMP-9). LMP2 mimics signals of B cell receptor (P13K/AKT pathways) which results in abnormal cell development [39].

Conclusion

Oral cancer increases the risk of infections and causes immune suppression. Contact of viruses in people can be a causative component for progression and initiation of disease. Additional studies on viruses will work as a supporting tool for new therapies, diagnosis and early examinations.

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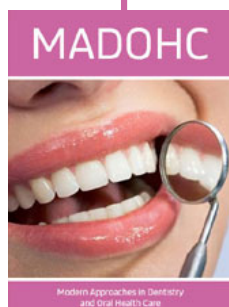
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