Human Herpes Viruses in Patients with Chronic Periodontitis and Aggressive Periodontitis

Mona Z Zaghloul

Microbiology Unit, Department of Clinical Pathology, Ain Shams University Hospitals, Cairo, Egypt

 Corresponding author: Mona Z Zaghloul, Microbiology Unit, Department of Clinical Pathology, Ain Shams University Hospitals, Cairo, Egypt, Tel: 02-24023494; E-mail: monazaki_810@hotmail.com

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Editorial

The pathogenesis of periodontal disease is a multifactorial process characterized by a complex interaction between the microbial and host factors and a variety of disease- modulatory environmental factors [1]. Recent microbiological researches on periodontal disease are focused on the possible involvement of human viruses in the etiology and pathogenesis of destructive periodontal diseases [2-5]. Herpes viruses are frequently found in periodontal pockets and may initiate or accelerate periodontal tissue destruction because of a virally mediated release of cytokines or even an impairment of the periodontal tissue defense mechanism, resulting in a heightened virulence of resident pathogenic bacteria [6-8]. Human herpes viruses are classified into eight distinct species: Herpes simplex virus (HSV) 1 and 2, Varicella zoster virus (VZV), Human Cytomegalovirus (HCMV), Epstein - Barr Virus (EBV) and human Herpes virus 6, human Herpes virus 7, and human Herpes virus 8 or Karpsoi's sarcoma-associated herpes virus [9]. Human cytomegalovirus (HCMV) and Epstein barr virus 1 (EBV-1) are involved in the pathogenesis of human periodontal disease. These viruses are capable of infecting and impairing Poly Morpho Nuclear Leukocytes (PMNs), macrophages and lymphocytes. Human cytomegalovirus may contribute to disease progression through the activation of IL1β gene transcription [10]. The reduced host defense by herpes virus-infected cells give rise to overgrowth of pathogenic bacteria that invade the periodontal tissue more efficiently [2].

Chronic periodontitis was defined when at least one of the four sites per tooth with Probing Pocket Depth (PPD) ≥ 3 mm or clinical attachment level (CAL) ≥ 2 mm [11]. While aggressive periodontitis was defined when the four sites per tooth with (PPD) ≥ 6 mm or (CAL) ≥ 5 mm with involvement of incisors and first molar teeth in subjects with age ranging from 18 to 35 years. HSV-1 is most commonly found in the oral cavity as a primary infection and establishes latency within the trigeminal ganglia [12]. Cultured epithelial cells and fibroblasts from clinically healthy human gingiva are susceptible to HSV infection [13] suggesting that those cells could be a reservoir for the latent virus. HSV-1 antigens could be detected in gingival biopsies from periodontally diseased patients using an indirect immunofluorescence assay [14]. Herpes viruses, including HCMV, EBV and HSV-1, can be reactivated from a latent infection either spontaneously or concurrent with another infection or with other stress factors affecting the host immune system [15,16].

References