Viruses, An Eitiology of Periodontitis?

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Introduction

A complex disease is a disease in which various biological factors lead to a similar clinical manifestation. Periodontitis is a common, complex, inflammatory disease characterized by destruction of tooth-supporting soft and hard tissues of the periodontium and eventually may result in loss of teeth. It affects 30-50% of adult population with 7-15% of adults suffering from its severe form and relatively few patients receive adequate treatment. Though bacterial plaque is considered to be the most common etiological factor in development of periodontitis, periodontitis is multifactorial where in microbial species, host immune responses and ecosystem based factors are responsible for its development. Viruses are well known to cause diseases in oral cavity,however their role as a causative agent for periodontitis still remains unclear.

Even though specific infectious agents are key in the development of periodontitis, it is unlikely that a single agent or even a small group of pathogens are the sole cause or modulator of this complex disease. It is necessary to give up bacteria as a single cause of periodontitis in order to answer many questions regarding the etiopathogenesis of periodontitis. There are some questions that remain unanswered when it comes to periodontitis, as to why in some individuals only few teeth are affected despite the omnipresence of periodontopathic bacteria, why some teeth show alveolar bone loss upto apical area whereas the destruction is minimal in adjacent teeth and how some form of disease have bilaterally symmetrical pattern and although subjects with poor oral hygiene develops gingivitis, not every gingivitis progress to attachment loss.

Periodontitis frequently affects individuals who are immunocompetent, genetically or environmentally predisposed. Since many bacterial infections occur as superinfection to viral diseases, the studies in viruses as an etiological factor for periodontitis has led to unlocking a new door on the pathway of periodontal research which otherwise till recently was focussed on the bacterial etiology. Herpes viruses, especially herpes simplex virus (HSV), Epstein Barr virus (EBV) and human cytomegalovirus (HCMV) are the most frequently researched viruses in periodontology.

Key Words : Viruses, Periodontitis.

Review of Literature

Polymerase chain reaction (PCR) studies have been used for detection of virus in periodontitis, targeting different genomic regions of target nucleic acids. However false negative results may be obtained if the virus may be absent during sampling procedures.

Para and Slots in 1999 investigated 30 patients with advanced periodontitis and 26 patients with gingivitis for presence of human cytomegalovirus (HCMV), Herpes Simplex Virus (HSV), Human Papilloma virus (HPV), and Human immunodeficiency virus (HIV) in Gingival crevicular fluid (GCF) samples using PCR studies and found that HCMV was detected in 60% of periodontitis patients while only 31% of gingivitis group. EBV in 30% , HSV in 20%, HPV in 17% and HIV in 2% of periodontitis patients were detected positive, however they were not detected in gingivitis group.

Contreas and Slots in 1996 investigated 27 patients with chronic periodontitis. GCF samples were taken from same patient with periodontitis sites and gingivitis sites and found high prevalence of HCMV (59%), EBV-1 (37%), EBV-2 (22%), HSV (26%), HIV (4%) in periodontitis sites compared to HCMV (18%), EBV-1 (22%), EBV-2 (18%), HSV (7%) and HIV (0%) in gingivitis sites. They further investigated 6 chronic periodontitis and 3 localized periodontitis cases for detection of HCMV and found that HCMV were present in 89% of diseased sites.

Ting et al in 2000 investigated patients with localized juvenile periodontitis for the detection of HCMV. He hypothesized that primary cytomegalovirus infection at the time of root development of permanent incisors and first molars may affect the developing periodontium of these teeth resulting in cemental dysplasia. During puberty, this virus may be reactivated as a result of hormonal changes which may suppress antibacterial immune defences leading to overgrowth of specific genotypes of actinobacillusactinomycetemcomitans at the sites which harbour latent HCMV infection resulting in attachment loss at specific sites on teeth.

Kamma at el in 2001 investigated 16 subjects with juvenile periodontitis and found that HCMV, EBV, EBV-CMV co-inhabitation were significantly associated with disease active sites. Also periodontal CMV exhibited close association with P. gingivalis. Most of the sites with EBV-CMV co-infection showing P. gingivalis-D.pneumosintes dual infection revealed bleeding on probing.

Saygun et al in 2001 investigated 21 healthy controls and 30 chronic periodontitis patients and found that plaque samples showed HCMV (44.3%), EBV-1 (17.7%), HSV (6.7%) compared to healthy sites with HCMV (14.3%), EBV-1 (14.3%) and HSV (0%).

Kubar et al 2004 investigated plaque samples and found that patients with aggressive periodontitis had HCMV (68.8%) compared to HCMV (0%) in healthy controls.

According to **Teughels et al in 2007**, cytomegalovirus can enhance the adherence of Aggregatibacteractinomycetemcomitans (previously Actinobacillusactino-mycetemcomitans) to primary epithelial cells of periodontal pockets. These viruses can alter the function of immune cells resulting in abnormalities in adherence, chemotaxis and phagocytosis.

Imbrouito et al in 2008 found increased HCMV detection (57%) compared to healthy controls (40%).

Billichodmath et al in 2009 investigated for herpes virus in chronic and aggressive patients found association of herpes virus of 100% in chronic periodontitis compared to 57% in aggressive periodontitis patients.

Conclusion and Future Perspectives

According to currently accepted hypothesis on the etiopathogenesis of periodontitis, bacteria are imperative for the development of periodontitis. However bacterial-host interaction do not suffice to explain the localized distribution and the periods of exacerbations and quiescence during tissue breakdown.

It is hypothesized that an active herpes virus infection can initiate periodontal tissue breakdown. It triggers a release of proinflammatory cytokines that have potential to activate osteoclasts and matrix-metalloprotienases and impair antibacterial immune mechanisms causing an up-growth of periodontopathic bacteria. Herpes virus can extent direct cytopatic effects on cells such as fibroblasts, keratinocytes and endothelial cells.

Recent researches suggest that high load of EBV and CMV are statistically associated with aggressive periodontitis and HSV with chronic periodontitis. High counts of EBV and HCMV and their presence in proportion with severity of underlying disease render them unlikely to be mere bystanders in the pathogenesis of periodontitis. The ability of herpes virus to alter the immune responses may increase the severity of periodontitis.

Frequent occurrence of herpes virus in various types of severe periodontal disease makes the participation of herpes virus species in the etiology of periodontitis as a possibility. Decrease in the counts of viruses in treated sites with periodontal disease can indicate the quantification of herpes virus in periodontal sites as a prognostic importance. Also ongoing research on anti-herpes virus vaccination may offer hope for prevention of periodontitis in large group of population.

The detection of virus within periodontal pockets by various studies proves that virus do have a role to play in periodontitis. However, there is 'what came first, hen or egg?' situation when it comes to considering viruses for periodontitis. The virus infection may be primary infection causing bacterial periodontitis as a superinfection or it may be the bacterial host response resulting in reactivation of latent virus then affecting the severity and progression of periodontitis.

References

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