Preventive Medicine

Subgingival Microbiota and Chronic Periodontal Disease: Culpability in Causation of Systemic Pathologies

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Abstract

Oral care is critical from the standpoint of systemic health. No health preventive strategy is complete without taking into cognizance the significance of subgingival microbiota in causation of a number of systemic conditions. Clinical studies and etiopathogenetic models substantiate a distinct connect between chronic periodontitis and a host of cardiovascular, respiratory, endocrine, musculoskeletal, and reproductive system pathologies. A potent source of contagion, subgingival microbiota associated with periodontitis may act as a significant risk factor in atherosclerotic coronary disease, carotid atheromas, chronic obstructive pulmonary disease, rheumatoid arthritis, systemic osteoporosis, diabetes mellitus, premature labor and birth of preterm babies, head and neck squamous cell carcinomas, and a myriad of other conditions.

Key words: Oral health, periodontitis, systemic diseases

INTRODUCTION

Several recent studies have suggested a connect between chronic periodontitis (CP) and a number of systemic diseases.1-4 CP has been found to be a significant risk factor for cardiovascular diseases, cerebrovascular diseases, peripheral arterial disease, respiratory diseases, and preterm births.5 In addition, it has also been incriminated in the pathogenesis of obesity, insulin resistance, diabetes mellitus, rheumatoid arthritis, osteoporosis, and a host of complications in pregnancy.6,7 It may also trigger pyogenic liver abscess.8

The relationship between periodontitis and systemic diseases is, however, bi-directional. Some of the systemic conditions have also been found to increase the incidence and severity of periodontal disease by modifying the immune response.6

Several mechanisms could link oral infections with secondary systemic effects. The subgingival microbiota could enter the systemic circulation and reach the target organs as a result of transient bacteremia; the virulent toxins produced by the subgingival microbiota could induce toxic insult; or subgingival microbiota may excite immunological injury and produce metastatic inflammation.4-6,7

A large and constantly expanding body of literature substantiates this connect between oral and systemic health, yet, the awareness among health care professionals of the iniquitous nexus between CP and systemic ill-health is, at best, scarce.

CHRONIC PERIODONTITIS

CP is an inflammatory condition [Figure 1] associated with a number of subgingival microbiota, including Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Bacteroides forsythus, Prevotella intermedia, Campylobacter rectus, Treponema denticola, and Fusobacterium nucleatum.8,9 Prevalent globally, CP can occur at all ages and is more common in the older individuals. A host of factors such as systemic conditions, for example, diabetes mellitus, alcohol consumption, cigarette smoking, stress, aging, and depression increase the susceptibility to CP.8,9

In its early quiescent stage, CP is relatively asymptomatic. Generally, individuals with early CP, therefore, do not report for treatment. This results in steady proliferation of subgingival microbiota and gives ground for their systemic influences. Characterized by cycles of transitory remission and exacerbation, CP produces a continual and progressive destruction of gingival tissue, periodontal ligament, cementum, and alveolar bone.8,9 This process of tissue destruction directly relates to damage caused by plaque bacterial products and also to indirect damage caused by bacterial induction of the host inflammatory and immune responses.10

The classic presenting signs and symptoms of CP include plaque accumulation, calculus formation, gingival swelling,
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Figure 1: Chronic Periodontitis. Generalized gingival inflammation, subgingival calculus, clinical attachment loss and irreversible bone resorption producing bucco-lingually mobile / apically depressible teeth. Further ingress of oral microbiota through periodontal pockets would produce systemic spread and exfoliation of vital teeth.

A poorly modulated systemic inflammatory response also serves as predictors for likelihood of developing serious cardiovascular disease. Oral bacteria have also been found in carotid atheromas and it has been postulated that these bacteria have the ability to promote platelet aggregation, resulting in potentially fatal thrombotic events. 

Respiratory system

The oral cavity is a potential reservoir for respiratory pathogens. Constant aspiration of oral bacteria can produce pneumonia of the lungs. Alternatively, the colonization of dental plaque by respiratory pathogens can be followed by its bolus aspiration. Alteration of the mucus surface by salivary enzymes in periodontitis could also lead to an increase in adhesion and colonization of respiratory pathogens; destruction of salivary pellicles on pathogenic bacteria by periodontal disease-associated enzymes; and alteration of respiratory epithelium by cytokines from periodontal disease, facilitating the infection of the epithelium by respiratory pathogens.

Systematic review of literature exploring the association between oral health and respiratory diseases reveals that improved oral hygiene can reduce the occurrence and progression of respiratory diseases among the high-risk elderly living in nursing homes, especially those in intensive care units. A potential association may exist between periodontitis and chronic obstructive pulmonary disease. Lung function can decrease with increasing periodontal attachment loss. In subjects with poor oral hygiene, a fivefold increase in chronic respiratory diseases has been reported.

Musculoskeletal system

Moderate-to-severe periodontitis can possibly increase the risk of rheumatoid arthritis. Periodontal disease can be a causal factor in the initiation and maintenance of the autoimmune inflammatory response that occurs in rheumatoid arthritis. A remarkable similarity exists in the pathogenesis of periodontal diseases and rheumatoid arthritis. A poorly modulated inflammatory response can drive both the diseases, resulting in oxidative stress-induced tissue injury. In addition, there has been an increasing interest in the interrelationship between systemic osteoporosis, oral bone loss, tooth loss, and risk factors for these conditions, and a positive correlation between systemic bone mass and oral bone loss had been shown.

Reproductive system

A significant association exists between preterm birth and/or low birth weight and periodontitis, irrespective of parity, race, and maternal age. Periodontitis appears to be an independent risk factor for poor pregnancy outcome and preliminary evidence suggests that periodontal intervention may reduce this adverse pregnancy outcome. Bacterial infection may result in the activation of cell-mediated immunity and the subsequent production of cytokines, interleukins such as IL-1β and IL-6, tumor necrosis factor alpha (TNF-α), and prostaglandins (PGE2), and these normal physiological mediators of parturition could induce preterm labor and result in low birth weight babies.

Subclinical infections such as periodontal disease may also contribute to premature labor and low birth weight through other mechanisms. Pathogenic microorganisms, or their microbial products such as lipopolysaccharide (LPS), could reach the uterus via the blood stream, induce cytokine release in the decidua or in the membranes, and result in

Associated Systemic Diseases

Cardiovascular system

CP is associated with an increased incidence of coronary heart disease (CHD) independent of established cardiovascular risk factors. Cumulative evidence supports a causal association between periodontal infection and atherosclerotic cardiovascular disease or its sequel. While majority of the research points to local and systemic inflammation as the possible link between the two conditions, an alternative pathway may be related to genetic and other host factors that increase the susceptibility to both atherosclerosis/thrombosis and CP.

Studies have shown that periodontitis results in higher systemic levels of C-reactive protein, interleukin (IL)-6, and neutrophils. Increased levels of these markers have been found to compound inflammatory activity in atherosclerotic lesions, and have been held culpable of increasing the risk for cardiac or cerebrovascular events. These systemic inflammatory markers also serve as predictors for likelihood of developing serious cardiovascular disease. Oral bacteria have also been found in carotid atheromas and it has been postulated that these bacteria have the ability to promote platelet aggregation, resulting in potentially fatal thrombotic events.

Figure 1: Chronic Periodontitis. Generalized gingival inflammation, subgingival calculus, clinical attachment loss and irreversible bone resorption producing bucco-lingually mobile / apically depressible teeth. Further ingress of oral microbiota through periodontal pockets would produce systemic spread and exfoliation of vital teeth.

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increased prostaglandin levels, inducing uterine muscle contraction.\textsuperscript{[10,38,39]}

Pregnant women with periodontal disease are also more likely to develop gestational diabetes mellitus than pregnant women with healthy gingiva.\textsuperscript{[40]}

**Endocrine system**

Diabetics are more prone to develop periodontal disease. Curiously, however, periodontal disease may also be a risk factor for diabetes.\textsuperscript{[40,41]} Periodontal disease can cause bacteria to enter the bloodstream and activate immune cells. These activated cells produce inflammatory biological signals (cytokines) that have a destructive effect throughout the body.\textsuperscript{[2,7,42]} In the pancreas, chronic high levels of cytokines can result in destruction or loss of beta cells and induce type 2 diabetes mellitus. This phenomenon has been reported in otherwise healthy individuals with no other risk factors for diabetes.\textsuperscript{[42-44]} Since periodontal disease contributes to the progression of impaired glucose tolerance, diabetes mellitus, and worsening of hyperglycemia in individuals with established diabetes, proactive, preventive dental care must form a major component of secondary prevention strategy.\textsuperscript{[45]}

**Malignancies**

CP is an independent clinical high-risk profile for head and neck squamous cell carcinoma (HNSCC), especially in the oral cavity, followed by the oropharynx and larynx.\textsuperscript{[46,47]} A significant association has been found between the history of periodontitis and risk of developing lung, kidney, pancreas, and hematological malignancies.\textsuperscript{[48]} These associations have been validated in a number of studies, after adjustment for major risk factors, including cigarette smoking and socioeconomic status.\textsuperscript{[49]} The risk is most substantive for oral and esophageal cancers and is followed by that for gastric and pancreatic cancers.\textsuperscript{[50,51]} The possible mechanism that links periodontitis and malignancy is still not clear, though lifetime cumulative infection exposure may play a major role.\textsuperscript{[52]}

**Conclusion**

Oral health carries significant impact on the systemic health. Health care professionals need to recognize this association. Spread of public awareness and regular, biannual dental examination with timely management of CP is critical from the standpoint of any preventive health strategy in the community.

**References**


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