Risk Factors Associated with Periodontal Diseases: A Review

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ABSTRACT

Periodontal diseases are a group of chronic infections caused by pathogenic bacteria colonizing the periodontium. Initiation and progression of periodontal infections are affected by both local and systemic conditions. The local factors include dental plaque and plaque retentive areas such as dental calculus and defective restorations. Systemic risk factors include poorly controlled diabetes mellitus and tobacco smoking. Systemic conditions associated with immunodeficiency state such as neutropenia, AIDS/HIV infections are also important risk factors. Recent studies have revealed several potentially important periodontal risk indicators. These include stress and coping behaviors and osteopenia associated with estrogen deficiency. There are also demographic factors associated with periodontal disease including gender, and hereditary factors.

Key words: Diabetes mellitus, Periodontitis, Risk factors, Smoking, Systemic factors.

INTRODUCTION

Periodontitis is one of the most ubiquitous diseases and is characterized by the destruction of connective tissue and dental bone support following an inflammatory host response secondary to infection by periodontal bacteria. Severe periodontitis, which may result in tooth loss, is found in 5–20% of most adult populations worldwide. Children and adolescents can have any of the several forms of periodontitis such as aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases. It is now generally agreed that almost all forms of periodontal disease occur as a result of mixed microbial infections within which specific groups of pathogenic bacteria coexist. Evidence is reviewed on the potential roles of modifiable and non-modifiable risk factors associated with periodontal disease. An understanding of risk factors is essential for clinical practice¹.

Risk factor is defined as an environmental, behavioral, or biologic factor confirmed by temporal sequence, usually in longitudinal studies, which if present, directly increases the probability of a disease occurring, and if absent or removed, reduces the probability. Risk factors are part of the causal chain or expose the host to the causal chain. They can be both modifiable or non-modifiable. Once disease occurs, removal of a risk factor may not result in a cure²,³.

This overview discusses various risk factors associated with periodontal diseases and dental considerations while managing them. Restorative dentistry, which encompasses conservative dentistry, endodontics and prosthodontics also have their own set of factors that play an important role, and that are to be considered before and during treatment of periodontal diseases.
Periodontal Factors
Modifiable risk factors

Smoking
Cross-sectional and longitudinal data provide strong support for the statement that the risk of developing periodontal disease as measured by clinical attachment loss and alveolar bone loss increases with increased smoking. Studies have shown that smoking does not reduce the amount of plaque present and in fact, smokers may experience less gingival bleeding than nonsmokers with lower plaque indexes. It has been suggested that this reflects an alteration of the caliber of the blood vessels perfusing the gingival tissues. It has also been suggested that reduced bleeding reflects an underlying disruption of the immune response and that this may account for the increased loss of clinical attachment and alveolar bone. It has been demonstrated that there are differences in the oxygen saturation of hemoglobin in the gingiva of smokers. Risk factors for periodontal disease Mehta 2 and non-smokers, suggesting that smokers have functional impairments in the gingival microcirculation. Furthermore, it has been shown that smoking has significant adverse effects on the immune system, which include the modification of the humoral and cellular immune systems, and cytokine and adhesion molecule network.

Diabetes mellitus
It is proven beyond doubt that diabetes (poorly controlled) can lead to aggravation of periodontal infection and exaggerated bone loss and vice versa is also true i.e. poorly controlled diabetes can be due to chronic periodontitis. The increase in blood glucose level is associated with periodontitis in diabetic patients. Studies have shown there is reduction in glycated hemoglobin after periodontal treatment. All these evidences suggest that control of periodontal infection is not only important for oral health, but also for general health in diabetic patient.

Microorganisms and periodontal disease
Of all of the various microorganisms that colonize the mouth, there are three, Porphyromonas gingivalis, Tannerella forsythia (formerly Bacteroides forsythus), and Actinobacillus actinomycetemcomitans which have been implicated as etiologic agents in periodontitis.

Socio-economic status
Results from the National Health and Nutrition Examination Survey III in US population suggest that socioeconomic disparities are contributing to the oral health inequities. It was found that the prevalence of socioeconomic disparities is contributing to the oral health inequities. Low income and a rural residence were significant risk indicators for attachment loss. This and other studies suggest that measures of socioeconomic status, including income, education levels, and urban status are fairly good risk indicators for periodontal diseases. Groups with low socioeconomic status (low income and/or education) are at a higher risk of having periodontal diseases than groups with high socioeconomic status, and the increased risk level in this group seems to be attributed to behavioral and environmental factors.

Psychological factors
A hypothesis of an increased risk for destructive periodontal diseases due to psychological disorders has long been promoted. There is an increased focus on study of the cellular and molecular basis for an increased risk for periodontal tissue loss due to stress and other psychosocial factors, and the interaction between the immune system and the central nervous system, which mediates the effects of these factors in maintaining the host response to infection. It is well established that psychological stress can down-regulate the cellular immune response, and disrupt the homeostasis of the network of signals linking the nervous, endocrine, and immune systems thereby interfering with the communication between the central nervous system and the host immune system.

Stress
The term stress serves as a convenient description for complex and incompletely understood psychological and physiological phenomena. Anxiety, as well as other emotional or psychosocial stresses, produces well-characterized neuroendocrine and biochemical changes in experimental animals. The physiological consequences of these stress-mediated changes have been shown to have significant adverse effects on the proper functioning of the immune system.
system. It has been reported that periodontal disease is more widespread and severe in those with higher levels of stress. It has been shown that occupational stress may have a relationship to the progression of periodontitis.  

**Nutrition**

Nutrition can influence the growth, development and metabolic activities of the periodontium; the high rate of cell turnover in the periodontal tissues requires that essential nutrients are readily available. Iron deficiency has a deleterious effect on macrophages, cells that are intimately involved in the immune and inflammatory systems. Cells of the immune system have a high turnover rate, a high demand for the building blocks of DNA, and are vulnerable to folic acid deficiency. Zinc plays a significant role in the regulation of the inflammatory process and a deficiency could negatively influence host resistance to gingivitis.  

**Non-modifiable risk factors**

**Genetic factors**

Although bacterial infection is the etiologic agent in periodontal disease, studies of identical twins suggest 50% of the susceptibility to periodontal disease is due to host factors. Interleukin-1 (IL-1) gene polymorphisms have been linked to periodontal disease. Thus, specific IL-1 genotypes have been linked to the presence of pathogenic microorganisms, and to an increased risk of Mehta Risk factors for periodontal disease in non-smokers and smokers. Furthermore, the evidence suggesting possible interactions between IL-1 and smoking and diabetes suggest that there is interplay between genetic and environmental factors that results in periodontal disease. Evidence also suggests possible relationships between periodontal disease and formyl-methionyl-leucyl-phenylalanine and Fc receptor polymorphisms.  

**Osteoporosis**

Osteoporosis is a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration with a consequent increase in bone fragility and susceptibility to fracture. Several cross-sectional studies have shown that alveolar bone density is altered in osteoporotic individuals. In longitudinal studies, a relationship has been shown between osteoporosis and alveolar bone loss, but not between osteoporosis and clinical attachment levels.  

**Other systemic diseases**

Several deficiencies of neutrophil function have been related to periodontal disease. These include Chediak-Higashi syndrome, cyclic neutropenias, lazy leukocyte syndrome, agranulocytosis and leukocyte adhesion deficiency and Down syndrome and Papillon–Lefevre syndrome. Except for Downs’s syndrome, these diseases are exceedingly rare, so probable though not definitive relationships to periodontal disease have not been established.  

**Ageing**

Aging is associated with an increased incidence of periodontal disease. Periodontal disease is a chronic infection therefore symptoms of periodontitis such as attachment loss, bone loss, gingival recession can be the result of the cumulative effect rather than older age of a person.  

To conclude, it is important to understand the etiological factors and the pathogenesis of periodontal disease to recognize and appreciate the associated risk factors. As periodontal disease is multi-factorial, effective disease management requires a clear understanding of all the associated risk factors.

**REFERENCES**


