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Review Article

Periodontitis and Systemic Disorders: A Comprehensive Review

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***ABSTRACT*:**

Literature from the recent past have revealed possible link between periodontitis and different systemic diseases. The periodontitis-systemic disease relationship constitutes an important part of clinical periodontal research.Confounding remains the most challenging issue in the interpretation of the associations found between diabetes, obesity, other systemic disorders and periodontal disease. Hence; we planned the present review to highlight some of the important aspects of the relationship between periodontitis and systemic disorders.

Key words: Periodontitis, Review, Systemic disorders.

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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.[1- 3]

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 practice.[4]

CARDIOVASCULAR SYSTEM

Chronic periodontitis is associated with the incidence of coronary heart disease (CHD) among younger men, independent of established cardiovascular risk factors. Cumulative evidence supports a causal association between periodontal infection and artherosclerotic cardiovascular disease or its sequelae.[5- 7]The possible link may involve direct and indirect effects of the periodontal infection; an alternative pathway may be related to genetic and other host factors that increase the susceptibility to both atherosclerosis/thrombosis and chronic periodontitis. Studies have shown that periodontitis results in higher systemic levels of C-reactive protein, interleukin (IL)-6, and neutrophils. These elevated inflammatory factors may increase inflammatory activity in atherosclerotic lesions, potentially increasing the risk for cardiac or cerebrovascular events. These systemic markers of inflammation are also said to serve as predictors of present and future cardiovascular events and disease. In addition, oral bacteria have been found in carotid

atheromas and it is reported that some oral bacteria may be associated with platelet aggregation, an event important for thrombosis. Evidence that suggests an association between chronic oral infections and myocardial infarction had also been presented.[8, 9]

Etiologically, the chronic presence of periodontal microbes can lead to atherogenesis via two pathways: (1) direct invasion of the arterial wall and (2) the release, in response to infection, of systemic inflammatory mediators with atherogenic effects. These pathogens, especially P. gingivalis, have demonstrated the ability to interact with the endothelial surface and to induce smooth-cell proliferation, causing damage and impairing the vasomotor functionality of the endothelial cells. Serum C-reactive protein (CRP) plays a role in endothelial dysfunction, and elevated levels of CRP provide insight into the linking of periodontal disease and CVD. In patients with periodontal disease who have elevated plasma levels of both fibrinogen37 and TNF-alpha, there is an association with increased carotid intima-media thickness (IMT).38 IMT and left ventricular mass (LVM) are alternative, yet valuable tools in measuring carotid atherosclerosis. However, our understanding of the mechanism linking these inflammatory markers with atherosclerosis progression is unclear.[10]

Recent studies have shown that CRP may directly interfere with endothelial nitric oxide (NO) availability, by both decreasing the expression of NO synthase and simultaneously increasing the production of reactive oxygen, which inactivates NO. Elevated CRP serum levels are the signal feature of the transition from stable coronary artery disease to the formation of a platelet-rich thrombus following plaque rupture or erosion. These findings shed light on the fact that endothelial activity, associated with elevated CRP serum levels, is characterized by the impaired systemic bioavailability of NO in coronary artery disease patients. Further investigation of this hypothesis (i.e., the rote of CRP on NO) has led to the discovery that CRP serum levels are important in predicting the availability of NO in the systemic circulation in coronary artery disease patients.[11, 12]

DIABETES

Diabetes mellitus comprises a group of metabolic diseases in which the characteristic phenotype is loss of control of glucose homeostasis, resulting from defects in insulin secretion, insulin action or both. In 2015, there were an estimated 415 million people worldwide with diabetes and this is projected to rise to 642 million by 2040. In 2015, more than 10% of adults in 25 states of the USA had diabetes. It is widely accepted that there is a bi-directional association between periodontitis and diabetes. It is well documented that periodontal disease is more prevalent and severe in those with diabetes, but most studies supporting this observation have been cross-sectional precluding the ability to firmly demonstrate the direction of the association. However, there have been prospective studies and a good baseline for the current review is a large study in the USA which suggested diabetes control, but not aetiology (type 1 or type 2 diabetes), was associated with accelerated periodontal attachment loss progression. This was reinforced by Taylor and colleagues, who suggested it may be the level of metabolic control and duration of diabetes that influence periodontal disease risk, with a significant heterogeneity among diabetic individuals.[13]

METABOLIC SYNDROME

Metabolic syndrome (MetS) relates to a cluster of disorders including excess body fat around the waist and abdominal area, increased blood pressure, elevated plasma glucose, elevated serum triglycerides and reduced serum high-density lipoprotein. The most recent definition from the International Diabetes Federation is the presence of central obesity, defined as a waist circumference exceeding ethnic specific guidelines, plus two from four of the other factors. It has been highlighted that 25% of the world's adults have MetS and as such, they have a fivefold greater risk of developing T2DM. The baseline for MetS and periodontitis is the systematic review and meta-analysis completed by Nibali and colleagues which used data from 19 cross-sectional and only one longitudinal study [18]. MetS was associated with the presence of periodontitis with an OR of 1.71 (95% CI 1.42–2.03); however, there was evidence of heterogeneity.[14]

CLINICAL IMPLICATIONS

Systematic reviews suggest that the presence of PD may be independently associated with a risk of multiple systemic diseases. Retrospective epidemiological studies and animal studies indicate a connection with biological plausibility. However, the clinical proof of causality will be extremely difficult in this setting due to multiple reasons. First, the initiating factor for systemic disease could be overlooked, as disease in the early phase is usually asymptomatic. Second, the proposed infection and inflammation role in causing systemic disease could be originating from sites other than the oral cavity. Lastly, most of the conducted studies are generating mixed results, which requires deeper evaluation of study design method.[15]

The available evidence supporting the association between PD and systemic diseases is relatively immature at this point. The best available evidence today indicates that infection and inflammatory reaction associated with PD may contribute toward systemic disease. Further studies are needed for confirmation. To summarize, this is what is known so far: 1) Strong evidence suggests that PD is associated with systemic diseases; 2) The available epidemiological and animal studies indicate PD increases the risk for systemic disease; 3) No causality between PD and systemic is confirmed so far, although it may exists based on one of the proposed mechanisms mentioned earlier; 4) Periodontal therapy overall has a favorable effect on systemic disease subclinical markers, despite heterogeneous response; 5) Interventional studies are needed to confirm the biological plausibility of PD to directly or indirectly cause systemic disease.[16]

CONCLUSION

It is significant to appreciate the etiologic aspects and the pathogenesis of periodontal pathologies to identify and appreciate the associated risk factors. As periodontal disease is multifactorial, effective disease management requires a clear understanding of all the associated risk factors. Therefore; future studies are recommended.

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