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RESEARCH ARTICLE

PERIODONTAL DISEASE AS A POTENTIAL RISK FACTOR FOR SYSTEMIC DISEASES

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Abstract

Periodontitis is a local inflammatory process mediating the destruction of periodontal tissues. Advances in science and technology over the last century have greatly expanded our knowledge of pathogenesis of periodontal diseases. The oral cavity is a window into the health of the body. Periodontal diseases comprise a number of infectious and inflammatory conditions brought about by the interaction between supragingival and subgingival biofilms and the host inflammatory response. The oral cavity serves as the 'port of entry' for the most diverse array of foreign antigenic challenges of a bodily orifice. These challenges are transient and maybe in the form of, medication or killed bacterial antigens contained within food and drink. Oral diseases such as dental caries, periodontal diseases and other oral mucosal lesion have a profound effect on general health and quality of life.

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INTRODUCTION

In recent years, there has been intense interest in potential associations between periodontal disease and various chronic systemic diseases and conditions. Periodontal Medicine as suggested by Offenbacher[1] is a rapidly emerging branch of periodontology focusing on relationship between periodontal and systemic health. This means a two-way relationship in which periodontal disease in an individual may be a powerful influence on an individual's systemic health or disease as well as the more customarily understood role that systemic disease may have in influencing an individual's periodontal health or disease. For the new diagnostic and treatment strategies in periodontal medicine it is essential to understand the pathophysiology of the systemic diseases associated with periodontal disease.

EFFECT OF PERIODONTAL INFECTION ON CARDIOVASCULAR DISEASE

Periodontal infection may affect the onset or progression of atherosclerosis and coronary heart disease through certain mechanisms. Periodontitis and atherosclerosis both have complex etiologic factors, combining genetic and environmental influences. In addition to smoking, periodontitis and atherosclerosis share many risk factors and have distinct similarities in basic pathogenic mechanisms.

Ischemic heart disease is associated with the process of atherogenesis and thrombogenesis. Increased viscosity of blood may promote major ischemic heart disease by increasing the risk of thrombus formation. Fibrinogen is probably the most important factor in promoting this hypercoagulable state and is the precursor to fibrin. An increased fibrinogen level results in increased blood viscosity. Increased plasma fibrinogen is the recognized risk factor for cardiovascular events and peripheral vascular disease. Elevated white blood cell count is

also a predictor of heart disease. Circulating leucocyte may promote occlusion of blood vessels. Coagulation factor VIII or von willebrand factor (VWF) has been associated with the risk of ischemic heart disease.

Fibrinogen levels and White Blood Cell counts are often increased in patients with periodontal disease. Individuals with poor oral health may also have significant elevations in coagulation factor VIII or von willebrand factor antigen, increasing the risk of thrombus formation. Thus, periodontal infection may also promote increased blood viscosity and thrombogenesis, leading to an increased risk for central and peripheral vascular disease.[2]

ROLE OF PERIODONTAL DISEASE IN CEREBRAL INFARCTION

The role of periodontitis in case of cerebrovascular disease is the gram negative bacteria and the associated Lipopolysaccharides that cause infiltration of inflammatory cells into the arterial wall, proliferation of arterial smooth muscle and intravascular coagulation. These changes are identical to those seen in naturally occurring atheromatosis. Low level bacteremia may initiate host responses that alter coagulability, endothelial and vessel wall integrity and platelet function resulting in atherogenic changes and possible thromboembolic events.

The presence of systemic infection before stroke resulted in significantly greater ischemia and a more severe post ischemic neurologic defect than did stroke not preceded by infection. Also, stroke patients with a preceding infection had slightly higher levels of plasma fibrinogen and significantly higher levels of C - reactive protein than those without infection.[3]

Most cases of stroke are caused by thromboembolic events. Platelet aggregation plays a major role in thrombogenesis. Platelets selectively bind some strains of Streptococcus sanguis a common component of supragingival plaque and Porphyromonas gingivalis a pathogen closely associated with periodontitis. Aggregation of platelets is induced by the platelet aggregation associated protein (PAAP) expressed on some strains of these bacteria. Subsequently bacteremia from PAAP positive bacterial strains from the supragingival and subgingival plaque can increase platelet aggregation contributing to thrombus formation and subsequent thromboembolism, the leading cause of stroke. Furthermore, periodontal infection may stimulate a series of indirect systemic effects such as elevated production of fibrinogen and C - reactive protein which serve to increase the risk of stroke.[3]

MECHANISMS BY WHICH PERIODONTAL DISEASE MAY INFLUENCE DIABETES

Periodontal diseases may induce or perpetuate an elevated systemic chronic inflammatory state. Acute bacterial and viral infections are known to increase insulin resistance in people without diabetes, a condition which often persists for several weeks after clinical recovery from the illness. Such illnesses and resultant increases in insulin resistance in people with diabetes greatly aggravate glycemic control. Chronic gram negative periodontal infection may also result in increased insulin resistance.

Periodontitis patients particularly those colonized by gram negative organisms such as P. gingivalis, Tannerella forsythensis, and prevotella intermedia have significantly higher serum markers of inflammation such as C-reactive protein, Interleukin-6, and fibrinogen. Systemic dissemination of these organisms or their products may induce a bacteremia or endotoxemia, including an elevated inflammatory state and stimulating increased levels of serum inflammatory markers. The potential impact of elevated systemic proinflammatory mediators in subjects with diabetes is tremendous.

Inflammation is significantly elevated in presence of obesity, insulin resistance, hyperglycemia and diabetes. High serum levels of acute phase reactants C - reactive protein and fibrinogen are seen in patients with insulin resistance and obesity. Insulin resistance and obesity are recognized as chronic inflammatory states and share many of the pathophysiologic features of atherosclerosis. Obesity, atherosclerosis and insulin resistance are strongly linked to the actions of the proinflammatory cytokines Interleukin-6 and Tumor Necrosis Factor-α and there resultant stimulation of acute phase reactants production in the liver. Insulin resistance is strongly liked to obesity.[4]

ROLE OF PERIODONTITIS IN PREGNANCY

In the early 1990s, studies have hypothesized that oral infections, such as periodontitis, could represent a significant source of both infection and inflammation during pregnancy. Studies stated that periodontal disease is a Gram-negative anaerobic infection with the potential to cause Gram negative bacteremia in persons with periodontal disease. They hypothesized that periodontal infections, which serve as reservoirs for Gram negative anaerobic organisms, lipopolysaccharide (LPS, endotoxin) and inflammatory mediators including PGE2 and TNF-α may be a potential threat to the fetal-placental unit.

Women having Low Birth Weight (LBW) infants have higher levels of Actinobacillus actinomycetemcomitans, Tannerella forsythia, Porphyromonas gingivalis and Treponema denticola in their

subgingival plaque. Women having LBW infants have higher levels of gingival crevicular fluid (GCF) PGE_2 and IL-1. GCF levels of IL-1 and PGE_2 have been shown to correlate highly with intraamniotic IL-1 and PGE_2 levels. Thus women having LBW infants have a higher prevalence and severity of periodontitis, more gingival inflammation, higher levels of putative periodontal pathogens and an elevated subgingival inflammatory response compared with women having normal birth weight infants.[2]

ASSOCIATION BETWEEN OBESITY AND PERIODONTAL DISEASE

It has been suggested that Obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction. The first report on the relationship between obesity and periodontal disease appeared in 1977 by Perlstein et al. They observed histopathologic changes in the periodontium in hereditary obese Zucker rats. Using ligature induced periodontitis; they found alveolar bone resorption to be greater in obese animals compared with non-obese rats. However, in response to bacterial plaque accumulation, periodontal inflammation and destruction were more severe in obese animals. In obese and hypertensive rats, plaque accumulation caused even more pronounced periodontal destruction than in obese animals, suggesting that a combination of risk factors, such as those defined by the metabolic syndrome, elicit the most severe periodontal effects. The underlying biological mechanisms for the association of obesity with periodontitis are not well-known. However, adipose tissue derived cytokines and hormones may play a key role. Fat tissue is not merely a passive triglyceride reservoir of the body, but also produces a vast amount of cytokines and hormones, collectively called adipokines or adipocytokines, which in turn may modulate periodontitis.[5]

ORAL BACTERIA AS ETIOLOGIC AGENTS OF RESPIRATORY INFECTION

It is possible that the teeth can serve as a reservoir for respiratory infection. Oral bacteria can be released from the dental plaque into the salivary secretions, which are then aspirated into the lower respiratory tract to cause pneumonia.[6]

Several mechanisms can be envisioned to help explain how oral bacteria can participate in the pathogenesis of respiratory infection:

a) Aspiration of oral pathogens for example P. gingivalis, A. actinomycetemcomitans into the lung to cause infection.

b) Periodontal disease associated enzymes in saliva may modify mucosal surfaces to promote adhesion and colonization by respiratory pathogens.

c) Periodontal disease associated enzymes may destroy salivary pellicles on pathogenic bacteria.

d) Cytokines originating from periodontal tissues may alter respiratory epithelium to promote infection by respiratory pathogens.[7]

PERIODONTAL DISEASE AND OSTEOPOROSIS

Wactawski-Wende et al[8] found a significant relationship between alveolar crestal bone height as a measure of periodontitis and skeletal osteopenia. In addition there was a relationship between osteopenia at the hip and probing attachment loss. According to Third National Health and Nutrition Examination survey (NHANES III) osteopenia of hip was significantly associated with severity of periodontal disease in females and males, independently of the confounding effect of age, gender, smoking or intake of dietary calcium. This association was increased even further in postmenopausal women. [6]

CONCLUSION

Periodontal medicine is a rapidly emerging branch of periodontology focusing on establishing a strong relationship between periodontal health/disease. Until now there are many studies linking periodontal disease with systemic diseases including coronary heart disease, cerebrovascular disease, diabetes mellitus, pregnancy complications, respiratory diseases, renal diseases, osteoporosis, obesity, rheumatoid arthritis, cancer, Alzheimer's, anemia etc.

As depicted by a few studies that questioned the association, we need to keep in mind that those with adequately managed systemic diseases do not guarantee a healthy periodontium and vice versa. Treating periodontal infection may have promising practical advantages that translate into better management of systemic diseases.

Universal management protocol and guidelines may necessitate further exploratory studies into this interlink but it is evident that in battling systemic disease and periodontal disease, taking into account the contribution of each to one another, shall bolster our approach in bettering the overall health.

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