Review Article

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Cardiovascular Diseases and Periodontal Diseases: Review and Update

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Abstract

Accumulated evidence has strongly suggested that the long-term effects of periodontal diseases can be linked to more serious systemic conditions such as cardiovascular diseases (CVD) and diabetes. Especially a prevalence of coronary heart disease is found to be significantly increased in patients with periodontitis after adjusting the risk factors such as smoking, diabetes, and blood pressure. Furthermore, various studies have shown that *Porphyromonas gingivalis*, a major periodontal pathogen is able to excerbate atherosclerosis following oral-hematogenous spread due to the bacteremia, caused by *P. gingivalis*, endothelial cells activate various adhesion molecules, thus increasing the likelihood of macrophage diapedesis and subsequent conversion to foam cells and further atheroma progression. These findings indicate the tight relationship between periodontal pathogens and CVD.

Key words: Atherosclerosis, Cardiovascular, Infection, Periodontitis, Risk factor

INTRODUCTION

The periodontal diseases are highly prevalent and can affect up to 90% of the worldwide population.[1] Gingivitis, the milder form of periodontal disease is caused by the bacterial biofilm (dental plaque). The association of coronary heart disease and periodontal disease may be due to an underlying response trait, which places an individual at high risk for developing both periodontal disease and atherosclerosis. It was suggested that periodontal disease, once established provides a biological burden of endotoxin (lipopolysaccharide) and inflammatory cytokines, especially thromboxane A2, prostaglandin E2,[2] interleukin (IL)1L = 1α, and tumor necrosis factor-beta, which serve to initiate and excerbate atherogenesis and thrombembolic events.[1]

International Classification of Diseases, 9th Revision defined diseases of the circulatory system as follows: (1) Ischemic heart diseases, (2) cerebrovascular diseases, (3) diseases of arteries, arterioles and capillaries (known as peripheral vascular disease), arterial septal vascular disease (ASVD) affect the heart and blood vessels; which is a major component of the cardiovascular system (CVS).[2] It is a chronic process over many years but it can cause acute clinical events including acute coronary syndrome (ACS), myocardial infarction (MI), and strokes.[1]

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CARDIOVASCULAR DISEASE (CVD)

It is frequent nowadays. Many patients with heart disease require dental treatment. Various surveys have suggested that dental disease may possibly contribute to the development of atherosclerosis and MI. Anxiety or pain during dental treatment can cause an outpouring of adrenaline which can both greatly increase the load on heart and also precipitate dangerous dysrhythmia.

PERIODONTAL DISEASE

There are evidences that dental infection, particularly periodontal disease, is possible a risk factor for atherosclerosis coronary artery diseases. Patients who have valvular defects (congenital or acquired as result of post-rheumatic fever) or some other congenital defects such a septal defects or who have prosthetic valves, should receive antibiotic therapy

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as prophylaxis before dental extraction (high-risk group), scaling or periodontal surgery. These are likely to release a significant number of bacteria from the gingiva, particularly periodontal pockets. The role of periodontal infection/inflammation is a risk factor for atherosclerosis.[3]

These observations are further corroborated in animal studies that demonstrates that oral infection of atherosclerosis-prone (apolipoprotein E-deficient) mice with P gingivalis resulted in accelerated atherosclerosis and the concomitent presence of *Porphyromonas gingivalis* DNA in their aortic tissue (Lalla *et al.*, 2003).[4]

A recent review of the epidemiologic pattern of periodontitis report a range in prevalence of severe periodontitis from 1% among 20-29 years to 39% among individuals more than 65 years of age.[5]

Poor periodontal status was significantly associated with increased C-reactive protein (CRP) and fibrinogen levels. Another group investigated the association between periodontitis and subclinical atherosclerosis, commonly measured by means of carotid artery intima-media thickness (cIMT) assesments.[6] Increased cIMT has been documented to be directly associated with increased risk of MI and stroke (O' Leary *et al.*, 1999). Becker *et al.* (2001) provided the first evidence that periodontitis may be linked to subclinical atherosclerosis.[7]

Several studies have shown that periodontal diseases are associated with heart disease. While a cause and effect relationship has not yet been proven. Patient at risk for infective endocarditis may require antibiotic before a dental procedure. Cardiologist and periodontist will decide if the existing heart condition requires the use of antibiotics before dental procedure.[8] Additional studies have pointed out to a relationship between periodontal disease and stroke. In one study that looked out the cause relationship of oral infection as a risk factor for stroke, people diagnosed with acute cerebrovascular ischemia were found more likely to have an oral infection when compared to those in the control group.[9]

BASIC PERIODONTAL ANATOMY

Teeth are supported by a connective tissue attachment apparatus (periodontal ligament) that is partly inserted into the outer layer of the root surface (root cementum) and partly into bone that surrounds the teeth. Teeth and gingival epithelium that surround teeth form several different ecological environments, each suitable for colonization by a distinct group of microorganism. The gingival sulcus is a niche that is readily colonized by oral bacteria that form a biofilm, or dental plaque.

PATHOPHYSIOLOGY

Three clinical parameters are typically recorded in epidemical studies of Parkinson's disease (PD) to asses prevalence: (1) Bleeding on probing which reflects the presence of an inflammatory infiltrate in gingival tissue, (2) Pocket depth which describes the deepening of gingival sulcus from which dental plaque biofilm can propagate, and (3) Clinical attachment level, which reflects the amount of periodontal tissue loss. Bleeding on probing and increased pocket depth indicates current pathology, whereas attachment levels provide a cumulative measure of loss of support caused by aggregate effects of pathogenetic factors such as PD and trauma.

Markers of periodontitis include evaluation of subgingival microbial colonization by selected periodontal organism and level of serum immunoglobulin (Ig)G and IgA antibodies to selected periodontal bacteria.[10] In some cases, high titers likely suggest the presence of a protective adaptive response, whereas, in others, they reflect the severity of periodontitis.[11]

MICROBIOLOGY OF PD

A newly cleaned surface of the tooth is rapidly covered with a glycoprotein deposit referred as a pellicle. The microbial composition of dental plaque differs above and below the gingival margin. Factors that influence the distinct pattern of microflora include specific local surface receptors for bacterial adherence. In the presence of gingivitis, Gram-negative anaerobic bacilli predominate in the subgingival flora. Subgingival microflora in gingivitis represents a transition between that associated with health and periodontitis.[12]

Initial (primary) supragingival colonizers have particularly affinity for constituents of pellicle. These colonizers include *Streptococcus sanguis*, *Streptococcus oralis*, *Streptococcus mutans*, *Actinomyces naeslundii,* and *Actinomyces odontolyticus*. The primary colonizer is followed by adherence of secondary colonizers such as *Fusobacterium nucleatum*, which in turn coaggregate with later colonizers. Within a short time complex communities of Gram-positive and Gram-negative bacilli and cocci become embedded in an extracellular matrix.[13]

RISK FACTORS FOR PD AND CVD

Risk factor associated with the development of PD includes local, systemic, and genetic factors. Although several bacterial species are currently recognized as casually associated with periodontitis, mere colonization of the

gingival niche by these species is not sufficient for disease to occur.[14] Instead PD is thought to be evolved from the stage of gingivitis, a local inflammatory process without loss of periodontal tissue support, that likely represent a stable, largely protective host response to periodontitis, a condition characterized by loss of connective tissue attachment and alveolar bone, influenced by environmental exposures and specific genetic predisposition.[15]

Contributors to ASVD are similarly multifactorial and include a complex interplay between genetic, environment and lifestyle factors. Many prevalent risk factor with well-documented impact are shared by ASVD and PD and could confound a relationship between increasing age, smoking, alcohol abuse, race/ethnicity, education and socioeconomic status, male sex, diabetes mellitus, and overweight are all factors associated with both ASVD and PD.[2] Although smoking is a major risk factor for both periodontal and CVD recent evidence seems to indicate that the observed association between PD and ASVD may be independent of smoking. It has been shown both in cross-sectional[16] and in longitudinal studies,[17] that PD and ASVD are associated in never smoker as well.

PATHOGENIC MECHANISM PROPOSED AS LINKS BETWEEN CVD AND PD

There are several pathways which have been proposed as a potential link between CVS and PD.

Indirect Mechanisms: Systemic Inflammation

Atherosclerosis may begin during childhood with initial infiltration of the endothelium with fatty substances and progress over many decades. Plaques that contain a soft atheromatous core are unstable, and their rupture will expose highly thrombogenic contents to blood, with activation of thrombosis and ensuring ACS, MI or stroke.[18]

The link between ASVD and inflammatory mediators in blood is well-established, with consistent associations between levels of systemic inflammatory markers and increases in clinical events, such as MI and nonhemorrhagic stroke, and in surrogate markers such as increased cIMT.[19]

A well-studied inflammatory marker is CRP. Many studies of individuals with no prior history of ASVD have demonstrated that a single nonfasting measure of CRP is a predictor of future vascular events, including MI, stroke, peripheral arterial disease, and sudden cardiac death.[20]

Additional inflammatory markers associated with CVD include lipoprotein-associated phospholipase A2[21] and tissue inhibitor of matrix metalloproteinase,[22] myeloperoxidase, and fibrinogen.

Periodontal inflammation is associated with systemic markers such as CRP, tumor necrosis factor alpha, IL-1, IL-6, and IL-8.[23] Systemic inflammation is similarly associated with cellular activation that involves cellular adhesion molecules, toll-like receptors, matrix metalloproteinase, and nuclear factor-k beta activation. The resulting interplay between endothelium, monocytes and platelets might be proatherogenic,[24] contributing indirectly to atherogenesis or adverse cardiovascular outcome related to atheromatous plaque rupture in a subject with periodontitis.[25]

Indirect Mechanism: Mimicry

Molecular mimicry is thought to occur when sequence similarities between foreign and self-peptides produce cross-activation of autoreactive T or B cells that can lead to tissue pathology or autoimmunity.[26]

Expression of host protective heat shock proteins (HSPs) such as HSP60 on endothelial cells may be induced by a variety of factors, including cytokines and shear stress and antibodies to HSP60, which have been associated with higher morbidity and mortality from atherosclerotic ASVD.[27] Proponents of molecular mimicry as a link between PD and ASVD suggest that endothelial damage may be aggravated by an immune response to bacterial HSP, such as molecular chaperone GroEL present in *P. gingivalis* and other periodontopathic bacteria.[28]

Direct Mechanism: Bacteremia and Vascular Infection by Periodontal Pathogens

Bacteremia that originates from the oral cavity is a common event that can occur during chewing and tooth brushing. It potentially occurs multiple times per day in individuals with some degree of gingivitis and periodontitis.[29]

A comprehensive search of literature provides a list of more than 275 bacterial species that have been identified in blood cultures after routine daily events or dental procedures.[30] Viridans group streptococci represent a significant proportion of flora around teeth, particularly in the dental biofilm that grows about the gingival crest. From there periodontal organism circulates in the blood stream either within phagocytic cells or extracellularly and subsequently are deposited in an atheromatous plaque. Common PD pathogens including *P. gingivalis* adhere to and invade various human vascular cells in culture.[31]

Numerous studies have examined the effect of antichlamydial antibiotic therapy on outcomes in patients with coronary artery disease. Of note systemic antibiotics alone would not be expected to lead a long-term resolution of chronic

periodontitis, in which bacteria resides in a biofilm. One study found that antibiotic use was associated with reduced cardiovascular events but did not improve mortality.[32]

Observational Studies using Noninvassive Imaging/surrogate Markers of ASVD

A systemic review on the use of broad spectrum of such methods in patients with PD included screening computed tomography of coronary arteries, ultrasound of the carotid arteries, magnetic resonance imaging, microalbuminuria and other biochemical measures of kidney dysfunction, and flow-mediated vasodilation (FMD) of the brachial artery. These methods have proved useful in clinical investigations of specific ASVD manifestation in defined patients cohorts and have been applied to the question of a possible PD/ASVD link.

Association Periodontitis with Subclinical Carotid or Coronary Artery Disease

Increased cIMT has correlated with PD in several association studies, which demonstrated that severe periodontitis, high subgingival colonization concentrations by specific periodontal pathogen, and high serum IgG titers against individuals periodontal bacteria were significantly related to increase cIMT in adjusted analyses.

Detection of coronary artery calcium (CAC) by computed tomography has been promoted as a marker of risk for future ASVD events. The 2007 ACC/American Heart Association (AHA) expert consensus statement on CAC scoring by computed tomography judged that it may be reasonable to use CAC measurement in asymptomatic patient with intermediate CHD risk (between 10% and 20%, 10 years risk of estimated coronary events) on the basis of available evidence that demonstrates incremental risk prediction information in this selected patient group.[33]

Association of Periodontitis with Endothelial Dysfunction

Endothelial dysfunction may be the earliest vascular manifestation of ASVD and has been associated with traditional risk factors for ASVD including systemic inflammation, obesity, and physical inactivity among others.[34] Smoking cessation, use of statin therapy and anigiotensin converting enzyme inhibitors, improved endothelial function in clinical trials. A number of tools are used to asses endothelial function *in vivo*. More recently noninvasive methods such as high resolution ultrasound assessment of the brachial artery after FMD or nitroglycerine administration and digital pulse amplitude tonometry have been studied across a broad spectrum of patients populations.[35]

Association of Periodontitis with Systemic Inflammation

Periodontitis is associated with both local and systemic inflammation. Multiple cytokines and inflammatory markers, including IL-1, IL-6, IL-8, and tumor necrosis factors are abundantly produced locally in the gingiva of patients with periodontitis and can be recovered in gingival crevicular fluid samples obtained from involved tooth sites.[36] CRP has been linked to an incident of MI, stroke peripheral arterial disease, and sudden cardiac death in multiple prospective epidemiological studies.

Electrocardiogram Abnormalities Associated with Periodontitis

Abnormal EGC findings are often nonspecific and may be associated with atherosclerotic and nonatherosclerotic cardiac diseases, including by pertension, valvular, and infiltrative disorders. Studies of prevalence EGC changes in Indians and Japanese population have suggested that risk for EGC abnormalities increases with the severity of PD.[37]

Periodontal Intervention and ASVD Risk

Periodontal therapy consists of mechanical debridement of root surfaces accompanied by home based plaque control (tooth brushing and flossing) whether or not the treatment of PD modifies the risk for or complications of ASVD has yet to be established. The most recent available systemic review of 6 treatment studies investigating the effects of periodontal therapy or serum CRP levels concluded that there is modest evidence of a treatment-induced reduction in CRP.[38]

Existing evidence does not prove that treating periodontitis will prevent CVD. However, periodontitis causes inflammation inside the mouth and evidence shows that inflammation inside the body can help us ascertain how healthy the heart and blood vessels are, even in the early stages of CVS.[39] Several studies and randomized clinical trials have reported improvement of endothelial function and associated markers of inflammation among subjects with significant PD who have undergone nonsurgical periodontal therapy with or without systemic antibiotic.[40]

Recently a randomized controlled trial involving full mouth mechanical debridement by either surgical or nonsurgical approach, dictated by the patient's condition, completed within a single session and accompanied by the extensive application of local antibiotics in all deep periodontal pockets demonstrated a significant improvement in brachial artery FMD at a 6-month follow-up examination.[41]

A review of intervention studies that investigated the effect of periodontal therapy on plasma levels of inflammatory mediators revealed inconsistent findings. Patient treated by nonsurgical periodontal therapy displayed a significant increase in plasma tumor necrosis factor alpha, CRP, and IL-6 levels immediately after intervention, which suggests a systemic acute phase response, possibly caused

by massive bacterial inoculation in conjunction with the instrumentation of periodontal tissue.[42]

To date, only a single multicenter pilot study has examined the effects of periodontal therapy on the secondary prevention of cardiac events. The periodontitis and vascular events periodontitis and vascular events investigation.[43] Randomized patients with periodontitis and a history of CHD (angiographically proven coronary artery disease of recent MI or surgical or percutaneous coronary revasculaization) to either community care (generally consisting of supragingival debridement only, control group) or a study protocol that consisted of oral hygiene instruction and nonsurgical periodontal therapy. Over a 25 months follow-up period, adverse cardiovascular events occurred with similar frequency in the community and the periodontal treatment group.

CONCLUSION

It is now clear from the epidemiologic studies that a potential link exists between PD and CVD oral health care. Professionals can identify patients who are unaware of their risk of developing serious complications as a result of CVD and who are in need of CVD and those who need medical intervention.

Prospective interventional studies are required to determine the exact link between PD and CVD as well as to evaluate whether periodontal treatment may reduce the risk of developing CVD. However, the challenge remains whether PD can be considered one among the traditional risk factor for CVD as the link established from different studies is not limited to a recent CVD. PD Seems to be associated with no more than a modest increase (−20%) in cardiovascular risk in general population.

As the ongoing studies report and confirm the strength of the association between PD and CVD in the next two decades, the oral health-care professionals and medical professionals have to prepare for better planning of prevention programs. It seems from the scientific evidence gathered so far that interventional care remains invaluable not only for oral health but also general health as well.

We conclude that the current evidence supports the notion that the incidence of ACVD, as represented by incident CHD, cerebrovascular disease, and peripheral arterial disease is higher in subjects with PD and/or worse periodontal status, compared to subjects without PD or with better periodontal status, independent of many cardiovascular risk factors. However, this may not be the case in all groups of the population.

Periodontitis could bear a significant CVD risk since it is a long-term disease process with a high prevalence in the Western population and may not always respond to treatment. Observations that the risk is highest in individuals with periodontitis and elevated CRP concentration and serum antibody levels to periodontal pathogens may suggest that periodontitis increases CVD risk mostly in individuals who react to this infection by a systemic inflammatory and immune response.

The relation between PD and ASVD is potentially of great public health importance because of their high prevalence extensive review of literature indicates that PD is associated with ASVD independent of known confounders. This information comes mostly from observational studies, however, and therefore does not demonstrate that PD is a cause of ASVD, nor does it confirm the contention that therapeutic periodontal intervention prevents heart disease or stroke or modify the course of ASVD. Although a contribution of PD to ASVD is biologically possible, periodontal and CVD share multiple risk factors that are prevalent and powerful promoters of disease, including tobacco use, diabetes mellitus, and age.

Recommending periodontal treatment solely for the purpose of atherosclerosis CVD prevention is not warranted based on current scientific evidence. Periodontal treatment must be recommended on the basis of the value of its benefits for the oral health of patient recognizing that patients are not healthy without good oral health and taking into account AHA recommendations. However, the emergence of periodontal infections as a possible risk factor for CVD is leading to a convergence in oral and medical care. As dental, public health and medical researchers and practitioners reach across disciplines, a holistic approach to care only benefit the patients and public health as a whole.

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