



Link Between Periodontal Diseases and Coronary Artery Diseases: A Systemic Review

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ABSTRACT

This article aims to review the link between periodontal diseases and Coronary Artery Diseases. Periodontitis is a bacterially induced, localized chronic inflammatory disease destroying both the connective tissue and the supporting bone of the teeth. In the general population, severe forms of the disease demonstrate a prevalence of almost 5%, whereas initial epidemiological evidence suggests an association between periodontitis and Coronary Artery Disease (CAD). To which extent each of these periodontitis-mediated components contribute to vascular damage still remains uncertain. By contrast, oral hygiene including periodontal health might contribute to the overall well being and healthy lifestyle and hence as might at least partially contribute to cardiovascular prevention. This article will throw light on the link between periodontal disease and CAD.

Keywords: Periodontitis, Coronary Artery Disease, Epidemiological, Vascular damage.

INTRODUCTION

A high concentration of plasma cholesterol, especially low density lipoprotein in cholesterol is the principal multifactorial pathogenesis of atherosclerosis.¹ Coronary Artery Disease is unquestionably the most important manifestation of atherosclerosis.² The development of coronary artery disease can result from genetic and several environmental risk factors such as age, abnormal serum lipids, diabetes, smoking and hypertension.³ The word 'periodontium' means structures surrounding the teeth (i.e) 'peri'- 'around' and 'odontos'- 'teeth'.⁴ Periodontitis is a chronic 'infectious/inflammatory' disease of multifactorial etiology.⁵ There is growing evidence that poor dental health especially the presence of periodontal diseases increases the risk of occurrence of coronary heart disease.³

The objective of this review was to investigate the possible association between periodontal health and coronary artery diseases.

Pathway Linking Periodontitis and Cardiovascular Diseases

Various pathogenic mechanisms have been proposed to explain the association between periodontal disease and CAD. These mechanisms may individually or combined, may contribute to a plausible association.

1. Direct effect of periodontal bacteria on platelets: *Porphyromonasgingivalis* and *Streptococcus sanguis* have been shown to stimulate platelet aggregation and thrombosis *in vitro* and *in vivo*.^{6,7}
2. Invasion or uptake of periodontal bacteria in endothelial cells and phagocytes: specific periodontal pathogens and their components have been identified in human atheromatous tissue.⁸ For

instance: *Porphyromonasgingivalis* can invade aortic and heart endothelial cells via its fimbriae.⁹

3. Systemic inflammation: Endocrine like effects of pro inflammatory mediators-there is an up regulation of mediators in vascular tissue. C-reactive protein and fibrinogen levels are elevated.¹⁰⁻¹³
4. Macrophages incubated *in vitro* with *Porphyromonasgingivalis* and low density lipoprotein take up bacteria intracellularly and are capable of transforming into foam cells.¹⁴
5. Autoimmune reaction/molecular mimicry: Cross reacting antibodies to periodontal bacteria and human heat shock proteins (HSPs) including HSP 60 on endothelial cells have been recognized. These autoimmune responses could provoke endothelial damage and atherosclerosis.¹⁵

Unique Features of Periodontal Infection^{4,16,17}

1. Periodontitis is a polymicrobial infection.
2. It is a long standing chronic infection that is asymptomatic most of the times.
3. Unusual anatomic features-the tooth is partially exposed to external environment and partially embedded within periodontal pocket.
4. Microorganisms that initiate periodontal disease reside within a protective environment (i.e) the biofilm.
5. Presence of teeth enhances the complexity of host parasite relationship.

Overview in Stages of a Atherosclerosis^{4,18,19}

A number of risk factors closely relate to development of atherosclerotic disease and risk for cardiovascular events



(myocardial infarction and stroke), including age, gender, hypertension, diabetes mellitus, smoking and low serum levels of high density lipoprotein (HDL) cholesterol.

The stages in development of atherosclerosis involve a series of events that include development of fatty streak, progression to complex plaque and plaque rupture.

1. Fatty streak: Accumulation of monocytes in the vessel intima takes place/this is the initial event in development of early atherosclerotic lesions. They migrate across endothelial cells and mature into macrophages that express scavenger receptors and engulf modified lipoproteins.
2. Progression of complex plaque: leads to accumulation of fibrous tissue in vessel. Certain growth factors from endothelial cells stimulate smooth muscle cells to produce interstitial collagen.
3. Plaque rupture: Thrombus formation is a result of plaque rupture. In case of non rupture plaque, the fibrous cap protects blood from non lipid core of plaque. Inflammation interferes with the integrity of fibrous cap through blocking the creation of new collagen fibers and stimulating the destruction of existing collagen by action of matrix metalloproteinases (MMPs).

Management of Periodontal Diseases in Coronary Artery Disease Patients¹⁸⁻²⁰

1. Patients with moderate to severe periodontitis should be advised that they may have a higher risk of CAD than periodontally healthy people.
2. Cardiovascular risk factors such as hyperlipidaemia, hypertension, smoking and metabolic syndrome must be controlled by dental and medical practitioners.
3. Standard treatment including plaque and gingivitis control and oral hygiene should be provided to manage periodontal disease in patients with CAD.

CONCLUSION

As periodontitis continues to have high prevalence within the population and the fact that CAD remains a major cause of human death in developed countries, in light of these associations we can state that oral health has an influence on systemic health in general.

Although the inflammation pathway provides a plausible explanation for periodontal diseases and CAD, further research is needed to define the mechanism linking the two diseases and how patients with periodontitis should best be managed to reduce their risk for CAD.

Obesity, diabetes and other auto immune diseases can also contribute to the total burden of inflammation.

Overall it is an extremely complex interaction and no one mechanism is likely to provide the complete explanation. More research may be required to throw light on the

exact mechanism of periodontal diseases and its link with coronary artery disease.

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