ACTA SCIENTIFIC DENTAL SCIENCES

Volume 9 Issue 1 January 2025

Review Article

Periodontitis and Cardiovascular Diseases- An Enigma Resolved

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Received: November 27, 2024

Published: December 06, 2024

Abstract

Cardiovascular disease is one of the most common diseases which are more prevalent in India. CVD cases are rising day by day. Periodontitis is one of the inflammatory conditions which is strongly associated with CVD. This article summarizes and gives the overall idea about the relationship status between CVD and periodontitis.

Keywords: Periodontitis; Cardiovascular Diseases; Enigma Resolved

Introduction

Oral health offers suggestions about general health. Periodontal disease is an infection and chronic inflammation of the gum tissues and bone that hold your teeth in place. The early sign of periodontal disease is called gingivitis. If not managed with proper diurnal oral care, it may worsen and become periodontitis. Periodontitis is a disease that involves connective tissue destruction, loss of bone, and ultimate tooth loss. Recent studies have suggested that chronic inflammatory conditions can increase the risk of cardiovascular disease. In this article, we will be analyzing the correlation between periodontal disease and cardiovascular disease.

Cardiovascular disease

One of the major causes of death occurring in the world is due to cardiovascular diseases (1/3rd of global death) [1]. The major etiologies of CVD are obesity and diabetes [1]. Major cardiovascular diseases include coronary artery disease, stroke, atrial infiltration, and atherosclerosis [2]. Gene functional annotation and pathway enrichment analysis [1] shows medical etiology between CVD and periodontitis. Other factors include increased age, male when compared to female, Africans and Asians, Native Americans, Alaskan natives, dyslipidemia, hypertension, and abnormal fluctuating glucose metabolism [3]. Atherosclerosis is a common condition where all other cardiovascular complication arises [4].

Classification of CVD

Lesion type	Description
1	An initial lesion with foam cells (intimal xanthoma or fatty streak)
2	Fatty streak with multiple foam cell layers
3	Pre-atheroma with extracellular lipid pools
4	Atheroma with a confluent extracellular lipid core
5	Fibro-atheroma
6	Complex plaque with possible surface defect or hemorrhage or thrombus or some combination
7	Calcified plaque
	Fibrotic plaque without lipid core [5].

Table 1

Atherosclerosis is the most common form of cardiovascular disease (CVD) characterised by accumulation of atheromatous plaque in the arterial lumen with persistant inflammation. As the lesion progresses, blood flow in the lumen reduces by >50% leading to Angina. Lesions may rupture, particularly if there is persistant inflamation. A clot in the coronary artery can completely obstruct the blood flow leading to Myocardial Infarction. Alternatively, the clot can also travel to distant sites, particularly to brain leading to stroke. Periodontitis and atherosclerosis share common pathways as they both are associated with persistant inflammation [6].

Role of bacteria in relation to CVD and periodontitis

Oral bacteria such as Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis have been involved in the formation of atheroma lesions [3] and these bacteria are involved in the formation of periodontitis. Porphyromonas gingivalis is shown to accelerate atherosclerosis in murine models. It shows fatty materials in rabbit [3] aorta and causes aortic and coronary lesions [3]. Other bacteria such as Treponema denticola, and Tannerella forsythia [4] also plays an important role.

Bacteria that are habituated in the subgingival biofilm can reach the blood streams and enter distant organs (bacteremia) [7], mostly occur during extraction, normal habitual functions like chewing, and usage of dental floss [7].

Actinomycetemcomitans are found more often in blood flow before and after scaling, and root planing [7]. There is also the indirect route of transmission which is by the invasion of phagocytic and dendritic cells by the oral bacteria. They release leukotoxins, proteases, and collagenases. Most of the bacteria are detected from heart valves, aortic valves, and coronary circulation which induces platelet aggregation [4].

Periodontitis and CVD: A possible link

Studies of inflammation have shown that proinflammatory cytokines can activate leukocytes, lymphocytes, and macrophages to produce a local inflammatory response, resulting in smooth muscle cell proliferation and extracellular matrix deposition. This results in instability in the fibrous cap leading to plaque rupture and myocardial infarction (MI).

A direct relationship between periodontitis and atherosclerotic CVD has not been established, it is hypothesized that periodontitis can trigger an inflammatory cascade in the oral cavity and could lead to a similar systemic cascade due to transient bacteremia. Recent studies support this hypothesis. Some data suggest increased CVD in those with significant pocket depth. In a study on Swedish population, 805 patients with cardiovascular disease underwent dental examination and panoramic x-ray, and dental health was classified as healthy (≥ 80% remaining bone), mild to moderate periodontal disease (PD) (66-79%) and severe (<66%) showed risk of CVD [8].

Etiopathogenesis

Atherosclerosis is characterized by an alteration of the vascular endothelium and the formation of atherosclerotic plaques that decrease the lumen of blood vessels. Lipid deposition is a key phenomenon in atherogenesis and plaques are formed by an LDL cholesterol core and a fibrous capsule.

For understanding the etiopathogenesis of atherosclerosis we must know about the anatomy of blood vessels.

They are built of three layers: tunica intima (internal layer), media (middle layer) and externa or adventitia (external layer).

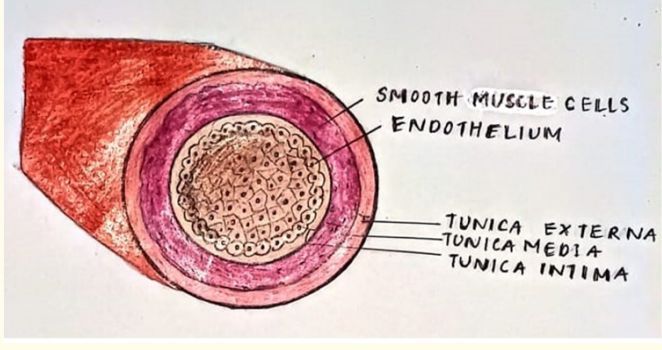

Figure 1: Layers of blood vessels.

The tunica intima consists of the endothelium and the basal elastic lamina, present in both arteries and veins. The tunica media (is composed of smooth muscle fibres, elastic fibres and collagen, depending on the type of artery. The tunica adventitia is formed by loose connective tissue with fibroblasts and collagen. The following is the etiopathogenesis.

- Accumulation of lipids [LDL] in the endothelial layer of the intima, where they become oxidised in the tissue, leading to formation of oxidized - LDL.

- Following this there is an initial inflammation, attracting monocytes, which migrate into the endothelium and differentiate into macrophages. The cardiovascular vessel becomes stiffer and less elastic.
- Transformation of macrophages into foam cells through phagocytosis of ox-LDL.
- Formation of a necrotic core within the early atheromatous lesion, consisting of necrotic macrophages, lipids and extracellular matrix. The inflammatory process and necrotic tissue leads to the production of cytokines and pro-inflammatory mediators, driving the inflammatory process.
- Migration of smooth muscle cells from the media into the intima and formation of a fibrous cap on the surface of the lesion.
- The atheroma can either eventually occlude the lumen of the blood vessel or rupture and exposition of underlying atherosclerotic plaque leading to thrombosis or blood clot formation that can migrate [9].

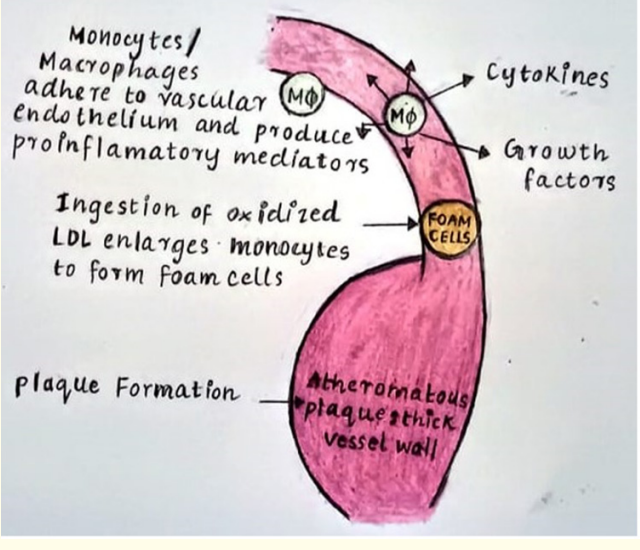

Figure 2: Pathogenesis of Cardiovascular Diseases

Periodontitis and atrial fibrillation

It is suspected that inflammation plays a major role in atrial fibrillation development, associated with coagulation and propagation of thrombosis. *In vivo* studies have shown that periodontitis was associated with enhanced immune activation in the atrial myocardium, which results in a change in the electrophysiological properties of the atrium [10].

Periodontal treatment and CVD

Periodontal treatment is shown to be effective in reducing the systemic markers of inflammation. Studies have shown a reduction in systemic markers such as IL-1, IL-6, and fibrinogen after non-surgical periodontal therapy. Vidal et al have observed a reduction in cardiovascular markers, such as a reduction in systolic and diastolic blood pressure after periodontal therapy [11].

Patients under antiplatelet therapy

Patients under dual antiplatelet therapy especially under acetylsalicylic acid (ASA) in combination with clopidogrel, have a risk of post-operative bleeding complications, and these hemorrhagic events can be managed with local hemostatic measures [3].

Patients under anticoagulant therapy

Patients taking vitamin K antagonists under dental extractions, and minor dental procedures do not seem to have an increased risk of bleeding compared to those discontinuing oral anticoagulants.

Recommendations for patients at dental office with CVD or at risk of CVD

The person should be aware that gum disease is a chronic condition that can aggravate CVD. Personalized advice provided by the dental health professional includes

- Brushing twice daily.
- Cleaning between teeth using interdental brushes or dental floss.
- Use of dentifrices and or mouth rinses with proven activity against plaque.
- Regular dental checkups as part of managing CVD.
- Patient should also suspect gum disease if noticing any red or swollen gums, bleeding from the gums, foul taste, loose teeth, increased space between teeth, or calculus on teeth.
- Gum disease can also able present without a sign if the patient smokes, annual dental check-ups are necessary [3].

Conclusion

The present review highlights the possible link between periodontitis and CVD. It is preferable for physicians who treat patients with CVD to assess the patient's oral health and symptoms such as tooth mobility, poor breath, gingival bleeding during brushing, flossing, or eating, or a terrible taste in the mouth, all of which are signs of an oral infection. If there is a suspicion of oral tissue inflammation or periodontal disease, the doctor must refer the patient

to a periodontist for further evaluation and treatment, which may include scaling, root planning, oral health education, and other complementary treatments such as local antibiotic therapy or antiseptic mouthwash.

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