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Oral Infections and Cardiovascular Disease: An Evolving Perspective

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Abstract

Atherosclerotic cardiovascular disease (ACVD) is an inflammatory disease of the coronary arteries associated with atheroma formation, which can cause disability and often death. Periodontitis is ranked as the sixth most prevalent disease affecting humans affecting 740 million people worldwide. In the last few decades, researchers have focused on the effect of periodontal disease (PD) on cardiovascular disease. The aim of this review was to investigate the association between these two diseases. PD is a potential risk factor that may initiate the development, maturation, and instability of atheroma in the arteries.

Oral infections are the most common diseases of mankind. Numerous reports have implicated oral infections, particularly periodontitis, as a risk factor for atherosclerotic cardiovascular disease. In this review we examine the epidemiology and biologic plausibility of this association with an emphasis on oral bacteria and inflammation. Longitudinal studies of incident cardiovascular events clearly show excess risk for CVD in individuals with periodontitis. It is likely that systemic exposure to oral bacteria impacts upon the initiation and progression of CVD through triggering of inflammatory processes. Given the high prevalence of periodontitis, any risk attributable to future CVD is important to public health. Unraveling the role of the oral microbiome in CVD will lead to new preventive and treatment approaches.

Introduction

Periodontal disease (PD) is an inflammatory disease primarily initiated in response to a specific group of bacteria and characterized by a complex host-biofilm interaction. According to the World Health Organization, the severe form of periodontitis causes tooth loss in about 5-15% of the population worldwide, and it is considered the sixth most common disease affecting humans [1]. Aberrant immune–inflammatory responses determine a patient's susceptibility to developing periodontitis, which may be modified by a range of risk factors. The transition from gingivitis to periodontitis initiates when the population and activity of a specific group of periodontal pathogens, predominantly Gram-negative anaerobic bacteria such as Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Tannerella forsythia, Treponema denticola and spirochetes, increase in the subgingival biofilm.

Cardiovascular disease (CVD) remains a leading cause of mortality worldwide. Despite extensive research and advancements in preventive and therapeutic strategies, certain cases of CVD remain unexplained. In recent years, scientists have revisited the infection hypothesis, focusing on the potential link between oral infection and cardiovascular health [2]. This article aims to explore the emerging evidence supporting the connection between oral infections, particularly periodontal disease, and the development and progression of cardiovascular disease.

The oral-systemic connection

Oral health is not limited to the mouth alone; it has been increasingly recognized as an essential component of overall health and well-being. The oral cavity harbors a diverse array of microorganisms, some of which can cause infections and inflammations [3]. Poor oral hygiene and the presence of specific pathogens can lead to the development of periodontal disease, a chronic inflammatory condition affecting the supporting structures of the teeth.

The infection hypothesis

The infection hypothesis was introduced in 1823 by Rayer, who compared the calcification observed in arteries to that observed in inflammatory conditions elsewhere. Others disputed the pathogenic significance of these observations and discussions of the infection hypothesis were abandoned for a long period in the nineteenth century. Osler, often cited as the first to postulate the causal role of the infection hypothesis in arteriosclerosis, wrote in 1908 about the existence of four great factors in the causation of arteriosclerosis - normal wear and tear of life, the acute infections, the intoxications, and those combinations of circumstances that keep blood tension high [4]. Osler described fatty streaks in atheroma as well as their high prevalence in children. It was evident that the authors distinguished between other types of aortitis, as seen in conditions such as syphilis.

Periodontal disease and inflammation

Periodontal disease is characterized by the destruction of the gums, periodontal ligament, and alveolar bone. It results from the long-term accumulation of dental plaque and the subsequent inflammatory response triggered by bacteria present in the biofilm [5]. This chronic inflammation releases various pro-inflammatory mediators, such as cytokines and acute-phase reactants, which can enter the bloodstream and exert systemic effects.

Inflammation and atherosclerosis

Atherosclerosis, the buildup of fatty plaques within the arteries, is a key underlying cause of cardiovascular disease. Chronic inflammation plays a pivotal role in the initiation and progression of atherosclerosis. Inflammatory mediators released from periodontal infections can contribute to endothelial dysfunction, leading to the disruption of the arterial wall and the infiltration of immune cells [6]. This sets the stage

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for the formation of atherosclerotic plaques.

Bacterial translocation and endothelial dysfunction

One proposed mechanism linking oral infection to cardiovascular disease is the translocation of oral bacteria into the bloodstream. Several studies have detected oral pathogens, such as Porphyromonas gingivalis and Streptococcus mutans, within atherosclerotic plaques. These bacteria can adhere to endothelial cells, induce endothelial dysfunction, and stimulate an immune response, exacerbating the inflammation within the arterial wall [7].

Immune response and plaque rupture

The immune response triggered by the presence of oral pathogens within atherosclerotic plaques can further contribute to plaque instability and rupture. Activated immune cells release enzymes that degrade the fibrous cap, which normally stabilizes the plaque [8]. Once the cap ruptures, a blood clot can form, leading to partial or complete occlusion of the artery, potentially resulting in a heart attack or stroke.

Clinical evidence and observational studies

Numerous observational studies have provided evidence supporting the association between periodontal disease and cardiovascular disease. These studies have demonstrated an increased risk of CVD, including coronary artery disease, stroke, and peripheral arterial disease, in individuals with periodontal disease. Furthermore, interventions targeting periodontal health have shown promising results in improving cardiovascular outcomes, suggesting a potential causal relationship.

Clinical significance of the link between PD and ACVD for dental practitioners and cardiologists

As detailed previously, a substantial body of evidence supports the relationship between PD and ACVD [9]. Although many studies have reported that periodontal therapy significantly increases surrogate markers of ACVD within a short time, followed by improvement in systemic inflammation and endothelial function, invasive dental procedures including periodontal treatment have not been associated with increased risk of MI. Furthermore, hemoglobin A1c has been found to decrease after periodontal therapy, which is of clinical relevance [10].

Dental practitioners have to be aware of the association between these two diseases. Patients with severe periodontitis should be advised to see a physician to check for signs of ACVD. Those patients should be informed that PD is associated with increased risk of cardiovascular complications and therefore their periodontal condition requires treatment [11]. Furthermore, subjects with ACVD have to adhere to proper oral hygiene measures and regular check-ups with a dental practitioner.

Although there is lack in evidence of a direct cause-effect relationship between PD and ACVD, evidence from published studies have confirmed the reduction in the systemic burden of inflammation following periodontal therapy. Thus, cardiologists should notify patients with atherosclerosis about the importance of good oral and dental health. Patients should be advised of the need to have regular home and professional dental care. Furthermore, the physician can recommend referral to a dentist or periodontist for oral and periodontal examination, assessment and treatment when necessary [12]. Cooperation between the dentist and the cardiologist is of paramount importance for patients on anticoagulant/antiplatelet medication prior to any oral or periodontal surgeries to avoid any complications such as excessive bleeding and ischemic events.

Conclusion

The infection hypothesis linking oral infections, particularly periodontal disease, and cardiovascular disease represents a fascinating area of research. While further investigation is needed to establish causality and clarify the underlying mechanisms, the accumulating evidence suggests that maintaining optimal oral health may contribute to reducing the risk of cardiovascular disease. This highlights the importance of regular dental care, good oral hygiene practices, and the potential integration of oral health assessments in cardiovascular risk assessment and management protocols.

Evidence from the studies detailed in this review supports the notion that there is a link between PD and ACVD. These two diseases share several systemic inflammatory mechanisms including increases in levels of inflammatory mediators, lipids, and hemostatic and thrombotic factors. Furthermore, they share several risk factors such as smoking and genetics. However, the extent of the impact of PD on the initiation and progression of ACVD is not clear yet and needs to be further examined. Microbiological studies have shown that periodontal pathogens can cause bacteremia and invasion of distant tissues. Evidence from epidemiological studies shows that the odds ratio of atherosclerotic disease is greater in patients with PD in comparison to non-PD individuals. Interventional studies could not examine the effect of periodontal therapy on primary prevention of ACVD such as ischemic heart disease and cardiovascular death due to methodological, financial and, most importantly, ethical considerations. Therefore, surrogate markers of cardiovascular events have been examined rigorously and periodontal therapy has shown significant influence on these markers in the short term.

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