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Interconnected health: The oral cavity's role in heart disease: systemic review

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ABSTRACT Cardiovascular disease is the most common cause of morbidity and mortality globally. Studies show that oral health, namely periodontal disease, is significantly linked to the risk of developing cardiovascular disease (CVD). This paper presents an overview of the biologic and epidemiologic factors linking heart disease and the oral cavity with an emphasis on systemic inflammation, bacterial translocation, and endothelial dysfunction. The paper will explore the role of oral health in CVD prevention and treatment.

KEYWORDS heart disease, periodontal disease, oral health, systemic inflammation, atherosclerosis

1. INTRODUCTION

Cardiovascular diseases (CVDs) represent a wide spectrum of disorders of the heart and blood vessels, including coronary artery diseases (CAD), heart failure, and strokes. The advances in clinical care notwithstanding, cardiovascular diseases continue to be the leading contributor to morbidity and mortality globally. In contrast, oral health disorders—especially periodontitis, a chronic inflammatory disease affecting the supporting structures of the teeth—are becoming increasingly common and a significant public health concern. Current evidence acknowledges a bidirectional relationship between oral health and cardiovascular health where periodontal disease not only reflects the status of systemic health but also contributes to the pathogenesis of cardiovascular health. The transmission of this relationship is primarily through systemic inflammation, microbial dissemination, and immune-modulated endothelial dysfunction [1]–[3].

While the links established in previous studies [4]–[6] offered a foundation for evidence, recent findings including Mendelian randomization analyses, have further confirmed the causal relationship between periodontitis and risk of cardiovascular disease. Understanding these interactions is critical to advancing models for integrated care that connect dental and cardiovascular medicine to establish comprehensive health approaches and early prevention interventions.

2. ORAL CAVITY AND PERIODONTAL DISEASE

The oral cavity contains a variety of naturally occurring microbial flora which, under certain circumstances including poor oral hygiene, can lead to periodontal disease. Periodontitis is a chronic inflammatory process resulting in the destruction of the supporting structures of the teeth, including inflammation of the gum, loss of alveolar bone and ultimately tooth loss in the more severe cases. Chronic periodontitis has been identified as one potential factor in numerous systemic diseases, including CVD [8]–[10]. The oral cavity is a complex ecosystem that contains many diverse microbial communities, many of which are harmless and benign in the presence of normal oral hygiene. Poor oral hygiene, combined with a genetic predisposition and/or a range of environmental factors, can lead to a state of dysbiosis - an imbalance of the oral microbial flora which can contribute to periodontal disease. Periodontitis is often misconstrued as solely an infectious disease; in fact, it is in fact a chronic inflammatory disease that progresses with destructive effects on the periodontal ligament and alveolar bone so that untreated periodontitis can lead to tooth loss.

Periodontitis is recognizing beyond a localized disease in the oral cavity but an inflammatory systemic stressor that can affect organ systems that distant from each other. Chronic periodontitis instigates a prolonged immune response and the systemic spread of pathogenic bacteria—namely Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola (the "red complex")— [2]. These pathogens can infiltrate gingival tissues, and potentially enter the blood circulation, acting as the additional source of chronic inflammation.

Recent works have evaluated the frameworks in which periodontal disease properties may be worsening systemic diseases such as atherosclerosis and insulin resistance [1], [3]. Many studies have shown that persons suffering with severe periodontitis have higher inflammatory markers, including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α,), which are also implicated in cardiovascular disease pathophysiology [11].

Epidemiological studies have consistently depicted that persons with periodontitis have greater risk of developing cardiovascular complications such as myocardial infarction and stroke [12]. These data suggest the need for interdisciplinary care models that incorporate oral health surveys during cardiovascular risk assessment approaches.

3. PATHOPHYSIOLOGICAL MECHANISMS LINKING PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE

An increasing number of studies support the link between periodontal disease and cardiovascular disease (CVD) development and progression by several important biological pathways. Each pathway demonstrates the capability of chronic oral inflammation to exert systemic influences conducive to atherogenesis, endothelial dysfunction, and vascular inflammation. Though there are several proposed pathways, the four most commonly cited are:

3.1 Systemic inflammation

Periodontal pathogens cause a chronic, low-grade systemic inflammatory response that results in increased levels of inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). These inflammatory mediators are associated with endothelial activation, atherosclerotic plaque development, and cardiovascular events such as myocardial infarction and stroke [3], [11].

3.2 Bacterial translocation and endotoxemia

Periodontitis can also promote bacterial translocation through ulcerated gingival tissues that permit pathogens, including Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, and Treponema denticola, to enter the blood circulation. Disseminated pathogens or their products (e.g., endotoxins, lipopolysaccharides) may:

- Infect endothelial cells
- Activate toll-like receptors (TLRs)
- Initiate pro-atherogenic immune responses

Microbial DNA from periodontal pathogens has been detected in atherosclerotic plaques, suggesting that there is a direct contribution of microbes to cardiovascular lesion development [1], [2], [12].

3.3 Endothelial dysfunction

Endothelial dysfunction is an early and necessary step in the development of atherosclerosis. Inflammatory mediators from the periodontal tissue are able to decrease the bioavailability of nitric oxide (NO), increasing oxidative stress and leading to a pro-thrombotic state. The end result is changes in:

-à Abnormal vascular tone
-à Monocyte adhesion increased
-à Platelet aggregation increased

Together, endothelial dysfunction facilitates plaque formation and vascular injury [1], [10].

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3.4 Autoimmune & molecular mimicry

A subset of periodontal bacteria present antigens, such as heat-shock proteins, that resemble native host molecules. This molecular mimicry can lead to autoimmune responses and the results manifest as chronic vascular inflammation and damage even after the clearance of the micro-organisms. This phenomenon of molecular mimicry could account for chronic endothelial injury of both periodontal and cardiovascular tissues.

4. EPIDEMIOLOGICAL EVIDENCE

Numerous epidemiological studies have found a link between periodontal disease and cardiovascular events in patients with atherosclerotic diseases, such as myocardial infarction (MI), stroke, and peripheral artery disease (PAD). Early studies, such as Bahekar et al. [4], provided initial evidence of a modest relationship between periodontitis and coronary heart disease. More recent studies have reinforced the relationship between periodontitis and cardiovascular disease, utilizing improved techniques of analysis on much larger numbers of people.

Recent, large scale cohort studies report that individuals with moderate to severe periodontitis have a 25–35% greater risk of CVD compared to out non-periodontitis patients [1], [3]. This relationship remains significant, after controlling for other traditional cardiovascular risk factors, such as smoking, diabetes, and SES (Socioeconomic status).

Additionally, Mendelian randomization studies—which exploit genetic variants as proxies to ameliorate confounding effects—have provided suggestive evidence for a causal relationship between periodontitis and coronary heart disease. One such study demonstrated that genetic liability to periodontitis was associated with risk of coronary events, which bolsters the idea that periodontitis may be a modifiable risk factor for cardiovascular disease.

Often times it is favorable to also have a global context when studying cardiovascular complications linked to periodontitis as lower income and middle income environments see a disproportionate cardiovascular burden associated with periodontitis. Patients have limited access to dental services in lower and middle countries [12]. This creates a case to consider oral health in optimal cardiovascular prevention strategies which have the potential to mitigate risks to all patients, especially those high risk and underserved populations.

Beyond this growing body of evidence, causation has not been proven definitively due mostly to the observational nature of these studies. However, the ability to reproduce findings across variant countries and methods of research provides good basis to consider oral health as a significant contributor to systemic health acquisition, including cardiovascular health.

5. CLINICAL IMPLICATIONS AND PREVENTIVE STRATEGIES

There are indications that improving oral hygiene and managing periodontal disease may positively impact cardiovascular health [7]. Integrative models of care that embed dental evaluations into routine risk assessments for cardiovascular disease could positively impact health outcomes [5]. As such, proper oral hygiene, routine dental and hygienist visits, and professional periodontal treatment (for patients at heightened risk of heart disease) are critical [6].

When combined, mounting evidence suggests that improving oral hygiene practices and managing their periodontal disease will improve measurable cardiovascular health outcomes—especially for individuals at high risk of diseases resulting from atherosclerosis. Clinical research has established that periodontal therapy reduced systemic inflammatory markers (C-reactive protein, CRP and interleukin-6, IL-6) associated with vascular inflammation and destabilizing vascular plaque [7], [11].

There is increasing recognition of including dental evaluations during cardiovascular risk screening, particularly among patients with co-morbidities, including diabetes and hypertension. Multiple organizations have developed interdisciplinary care models to enhance outcomes through collaboration between cardiologists, general medical practitioners, and dental practitioners [3], [12]. Not only does professional periodontal therapy, combined with patients' daily oral hygiene improve local periodontal health, it can enhance endothelial function, improve arterial stiffness, and slow the progression of atherosclerosis in random clinical trials [13].

Importantly, public health policy should recognize and embrace changes reflecting these new understandings. Health education and health promotion strategies to integrate oral health into overall health and well-being may potentially contribute to decreasing the global burden of cardiovascular disease in under-resourced populations that do not have access to dental preventative care [1].

In summary, optimal oral health should be acknowledged as a key element of cardiovascular disease prevention and management programs. This provides added value for oral health examinations, the potential for focused professional periodontal treatment, along with evolving integrated pathways for support in collaboration with engaged medical issues and treatment plans, can be developed when integrating dentist and medical providers.

6. CONCLUSION

The connections between oral health and heart disease emphasize the importance of holistic healthcare. We need more research to elucidate the specific mechanisms of causation, but the evidence supports oral health interventions as an important inclusion in any programming aimed at the prevention and management of cardiovascular disease.

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