Cardiovascular and periodontal diseases

Doença cardiovascular e doença periodontal

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DOI: 10.5935/1678-9741.20140003

Cardiovascular diseases, especially those associated with atherosclerosis, are still one of the main causes of death worldwide.

There are well-established risk factors for cardiovascular diseases, one of them being elevated levels of serum lipids combined with infections such as odontogenic infections, which consist of dental caries and periodontal disease (gingivitis and periodontitis).

Periodontal and cardiovascular diseases share many risk factors, such as age, educational level, gender, income level, smoking and drinking habits, hypertension, stress, depression, and diabetes. Several studies have shown that patients with periodontitis and acute ischemic syndromes share various characteristics.

It should be noted that severe chronic periodontitis can alter lipid profiles as well as lead to acute coronary events. In addition, the presence of periodontal organisms in coronary arteries has been linked to the development and progression of atherosclerosis. The presence of *Chlamydia pneumoniae* in 35% of the coronary and internal thoracic arteries suggests that this bacterium plays an important role in the progression of atherosclerosis [1].

In the United States, 25% of adults age 60 and older lose all their teeth (edentulism), half of them due to periodontal disease; the other half, to caries [2].

Chronic periodontitis consist of chronic oral infections found on the surface of teeth and in adjacent tissues. Clinically, the onset is marked by gingival inflammation and is followed by formation of a periodontal pocket, which fosters the development and growth of anaerobic Gram-negative bacteria, including *Porphyromonas gingivalis*, *Prevotella intermedia*, *Aggregatibacter actinomycetemcomitans*, and *Tannerella forsythia,* among others [3].

Experimental studies have convincingly demonstrated the release of inflammatory mediators from peripheral monocytes when taken from patients with periodontitis and exposed *in vitro* to bacterial lipopolysaccharides.

The accumulation of bacteria in the periodontal microflora results in the production of lipopolysaccharides, which are released from the external membrane of Gram-negative bacteria.

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Consequently, host response is immediately triggered by recruitment of inflammatory cells, which produce large quantities of proinflammatory cytokines, such as interleukin 6 (IL-6), prostaglandins E2 (PGE2), and matrix metalloproteinases (MMPS), which in turn contribute to the destruction of periodontal tissue. As a result of the high production of these metabolites, in the acute phase, there is a subsequent response from the liver, which produces and synthesizes proteins, one of them being the C-reactive protein found in the blood of patients with chronic periodontal diseases [4-6].

The relationship between odontogenic infections and cardiovascular disease has been described in several studies, including experimental ones, which have shown the release of inflammatory mediators in patients with periodontitis. Thus, diagnosis and treatment of periodontal diseases are important to maintain both oral and systemic health [7].

The past two decades have seen an increasing interest in the impact of oral health on atherosclerosis and, hence, on cardiovascular diseases.

Therefore, it seems that periodontal disease may contribute to the development of cardiovascular disease [8].

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Abbreviations, acronyms & symbols	
IL-1B	Interleukin 1 beta
IL-6	Interleukin 6
INF-Y	Interferon Y
MMPS	Matrix metalloproteinases
PGE2	Prostaglandins E2
TNF-α	Tumor necrosis factor alpha

Host response to the infection is often accompanied by the release of proinflammatory cytokines, such as interleukin 1 beta (IL-1B), IL-6, and tumor necrosis factor alpha (TNF-α), which alter the lipid metabolism and promote hyperlipidemia. In addition, common events in the evolution of the disease are influenced by risk factors or indicators.

Genetic factors, environment, and other acquired habits differ in stage and form from one disease to another. Proinflammatory cytokines, such as IL-1B, TNF-α, and interferon Y (INF-Y), increase and induce the production of PGE2 and MMPS, molecules that promote the destruction of the extracellular matrix of gingival tissue and periodontal ligament as well as the reabsorption of alveolar bone [9]. Products originating from Gram-negative bacteria cell wall (LPS), the leading cause of periodontitis, trigger a host response, with the production and release of proinflammatory cytokines (IL-1B, IL-6, and TNF-α), which in turn induce a host response themselves, elevating the levels of C-reactive protein and fibrinogen [5].

Experimentally, the role played by the *Porphyromonas gingivalis* bacteria in atherosclerotic plaque formation proves that periodontitis causes fat accumulation in the aorta. Thus, chronic periodontitis alter the biochemical profile as well as the white cell count, evidenced by the altered immune response (20% higher). Clinical and laboratory evaluations of systemic diseases performed on healthy patients show a potential connection between periodontal diseases and their lipid and glycemic profiles [10].

Therefore, diagnosis and treatment of periodontal diseases are important not only to maintain good oral health, but also to help mitigate pathological changes such as atherosclerosis and, subsequently, acute myocardial infarction and strokes [7].

Periodontal diseases and their role in the etiology of acute ischemic syndromes have received growing attention in clarifying the possible mechanisms involved in both diseases [11]. Reports found in the literature document the association between acute ischemic syndromes and chronic infections by Gram-negative bacteria such as *Chlamydia pneumoniae* and *Helicobacter pylori* [12].

Hence, periodontal diseases and their possible interactions cannot be overlooked and their association with cardiovascular diseases should be investigated [13].

Periodontal bacterial DNA was observed in 10 out of the 17 samples of coronary arteries, representing approximately 59.9%, in which *Porphyromonas gingivalis* was present in 52.9%, *Aggregatibacter actinomycetemcomitans*, in 35.5%, *Prevotella intermedia,* in 23.5%, and *Tannerella forsythia*, in 11.7%. *Chlamydia pneumoniae* was seen in 35.3% of the coronary and internal thoracic arteries [1].

Thus, the presence of periodontal microorganisms in 10 out of the 17 coronary arteries studied supports the idea that those bacteria may be associated with the development and progression of atherosclerosis, as it has been observed in several epidemiological studies [1,14].

In light of this, we can say that the presence of periodontal microorganisms in the coronary and internal thoracic arteries may be associated with the development and progression of atherosclerosis as well as lesions in cardiac valves.

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