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.

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].
Host response to the infection is often accompanied by the release of proinflammatory cytokines, such as interleukin 1 beta (IL-1β), 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-1β, 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-1β, 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 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.

**REFERENCES**

1. Oliveira FJ, Vieira RW, Coelho OR, Petrucci O, Oliveira PPM, Antunes N, et al. Inflamação sistêmica causada pela periodontite crônica em pacientes vítimas de ataque cardíaco isquêmico agudo. Rev Bras Cir Cardiovasc. 2010;25(1):51-8.
2. Beltrán-Aguilar ED, Barker LK, Canto MT, Dye BA, Gooch BF, Griffin SO, et al. Surveillance for dental caries, dental sealants, tooth retention, edentulism, and enamel fluorosis: United States, 1988-1994 and 1999-2002. MMWR Surveill Summ. 2005;54(3):1-43.
3. Socranski S, Haffajee AD. Periodontal microbial ecology. Periodontol 2000. 2005;38:135-87.
4. Loos BG, Hunter J, Varoufaki A. Level of C-reactive protein in periodontitis patients and healthy controls. J Dent Res. 1988;77(special issue):666.
5. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol. 2000;71(10):1528-34.
6. Antunes N, Dragosavc D, Petrucci Junior O, Oliveira PPM, Kossou C, Blotta MHSIL, et al. Ultrafiltração para remover mediadores inflamatórios durante circulação extracorpórea na revascularização do miocárdio. Rev Bras Cir Cardiovasc. 2008;23(2):175-82.
7. Graves DT, Jiang Y, Genco C. Periodontal disease: bacterial virulence factors, host response and impact on systemic health. Curr Opin Infect Dis. 2000;13(3):227-32.
8. Meurman JH, Sanz M, Janket S. Oral health, atherosclerosis, and cardiovascular disease. Crit Rev Oral Biol Med. 2004;15(6):403-13.

9. Page RC. The pathobiology of periodontal disease may affect systemic diseases: inversion of a paradigm. Ann Periodontol. 1998;3(1):108-20.

10. Salvi GE, Carollo-Bittel E, Lang NP. Effects the diabetes mellitus on periodontal and peri-implant conditions: update on associations and risks. J Clin Periodontol. 2008;35(8 Suppl):398-409.

11. Kinane DF, Lowe GD. How periodontal disease may contribute to cardiovascular disease. Periodontol. 2000;23:121-6.

12. Liu R, Moroi M, Yamamoto M, Kubota T, Ono T, Funatsu A, et al. Presence and severity of Chlamydia pneumoniae and Cytomegalovirus infection on coronary plaques are associated with acute coronary syndromes. Int Heart J. 2006;47(4):511-9.

13. Offenbacher S, Beck JD, Moss K, Mendoza L, Paquette DN, Barrow DA, et al. Results from Periodontitis and Vascular Events (PAVE) Study: a pilot multicentered, randomized, controlled trial to study effects of periodontal therapy in a secondary prevention model of cardiovascular disease. J Periodontol. 2009;80(2):190-201.

14. Humphrey LL, Fu R, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. J Gen Intern Med. 2008;23(12):2079-86.