

## Periodontal Disease as a Potential Risk Factor for Acute Coronary Syndromes

Carlos Vicente Serrano Júnior<sup>1</sup> and Juliana Ascensão de Souza<sup>1,2</sup>

Instituto do Coração do Hospital das Clínicas – FMUSP<sup>1</sup> and Instituto do Coração – InCor-DF - Fundação Zerbini<sup>2</sup> - São Paulo, SP - Brasília, DF, Brazil

Chronic inflammation is recognized as the primary cause of the development and perpetuation of atherosclerotic disease<sup>1</sup>. Conditions associated with the induction and/or maintenance of severe inflammatory activity, such as persistent bacterial infections, have been correlated to coronary artery disease (CAD). A number of studies have evaluated the role of periodontal disease (PD)<sup>2,3</sup>, particularly periodontitis, or chronic bacterial infection of the supporting structures of the teeth. The mechanisms involved in this association are the following: 1) the degree of infection by bacterial pathogens in the dental plaque; 2) persistent exposure to antigens; 3) endotoxin production, and 4) release of inflammatory cytokines present in periodontitis. These mechanisms contribute to the atherogenic process and induction of thromboembolic events, predisposing patients to increased risk of cardiovascular events.

Periodontal disease is highly prevalent throughout the world, especially in developing and/or underdeveloped countries, where oral health is not spread evenly across the population. Moreover, the incidence of atherosclerotic disease has been increasing in these countries. During the last decade, innumerable studies have investigated the correlation between periodontal disease and coronary heart disease.

The Accarini and Godoy study, published in this issue, is the first to associate these two frequent diseases in Brazil. The study, in keeping with the literature, shows a strong correlation between PD and severe obstructive coronary disease, angiographically documented (Heart Cath), in 361 patients admitted with acute coronary syndrome (ACS). The severity of oral disease in the population studied is noteworthy, because approximately 50% of the patients did not have the minimum number of teeth required for an appropriate dental examination. Notwithstanding, 171 patients could be assessed by cardiac catheterization and oral examination. Accarini has shown that patients with ACS and obstructive coronary disease of any degree are 2.5 times more likely to develop active PD, confirming the existent data demonstrating that patients with periodontal disease are at higher risk of experiencing an acute coronary event.

The main difficulty in this type of study is the lack of a universally standardized method to assess periodontal disease. With this in mind, Andriankaja et al<sup>5</sup> demonstrated that the association between PD and myocardial infarction was

consistent, regardless of the different PD classifications and assessment methods.

The pathophysiological mechanism involved in this association has been investigated by several studies. The degree of infection and number of different bacterial species found in the dental plaque seem to be correlated to the presence or not of DAC<sup>6</sup>. Both the intensity and persistence of the inflammatory reaction associated with PD were assessed in CAD patients by measuring serum inflammatory markers, such as C-reactive protein (CRP)<sup>7</sup>, interleukins<sup>4</sup>, and antibodies to known periodontal pathogens colonizing oral mucous membranes<sup>8</sup>. In a recent study, it was demonstrated that, although no statistically significant difference was found in CRP levels among patients with ACS and different periodontal health status, the decrease in PCR levels was slower in patients with advanced PD than in those with better periodontal health<sup>7</sup>. Conversely, Montebugnoli et al<sup>4</sup> have shown that periodontal health improves the inflammatory and haemostatic status, reducing both the levels of CRP, leucocytes, fibrinogen, and other coagulation factors and the oxidative stress, with a significant decrease in oxidized-low density lipoprotein.

The use of antibiotic therapy for CAD prevention is still under intense debate; some studies have reported positive results, while others have not. Currently, it has been hypothesized that periodontal health status plays a role in the response to antibiotic treatment targeting *Chlamydia pneumoniae* to prevent coronary events. In the study by Paju et al<sup>9,10</sup> treatment with clarithromycin decreased recurrence of cardiovascular events in patients without periodontitis, but not in patients with poor periodontal health. These authors suggested that systemic inflammatory effects of periodontal disease may be related to failure of antibiotic therapy in CAD.

Accarini's study is paramount, since it was the first to demonstrate, in the Brazilian population, the potential association between PD and CAD. Although no authoritative data are available for the entire population, PD prevalence is high in Brazil and may be associated with worse prognosis in CAD patients. These data show that CAD management and prevention require a multidisciplinary team, including dental assessment and treatment, in an attempt to reduce periodontal disease and, thereby, the risk of new cardiovascular events.

---

## References

1. Ross R. Atherosclerosis--an inflammatory disease. *N Engl J Med* 1999;340(2):115-26.
2. DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. Dental disease and risk of coronary heart disease and mortality. *BMJ (Clinical research ed)* 1993;306(6879):688-91.
3. Czerniuk MR, Gorska R, Filipiak KJ, Opolski G. Inflammatory response to acute coronary syndrome in patients with coexistent periodontal disease. *J Periodontol* 2004;75(7):1020-6.
4. Montebugnoli L, Servidio D, Miaton RA, et al. Periodontal health improves systemic inflammatory and haemostatic status in subjects with coronary heart disease. *J Clinical Periodontol* 2005;32(2):188-92.
5. Andriankaja OM, Genco RJ, Dorn J, et al. The use of different measurements and definitions of periodontal disease in the study of the association between periodontal disease and risk of myocardial infarction. *J Periodontol* 2006;77(6):1067-73.
6. Spahr A, Klein E, Khuseyinova N, et al. Periodontal infections and coronary heart disease: role of periodontal bacteria and importance of total pathogen burden in the Coronary Event and Periodontal Disease (CORODONT) study. *Arch Intern Med* 2006;166(5):554-9.
7. Czerniuk MR, Gorska R, Filipiak KJ, Opolski G. C-reactive protein in patients with coexistent periodontal disease and acute coronary syndromes. *J Clin Periodontol* 2006;33(6):415-20.
8. Beck JD, Eke P, Heiss G, et al. Periodontal disease and coronary heart disease: a reappraisal of the exposure. *Circulation* 2005;112(1):19-24.
9. Paju S, Pussinen PJ, Sinisalo J, et al. Clarithromycin reduces recurrent cardiovascular events in subjects without periodontitis. *Atherosclerosis* 2006;188(2):412-9.
10. Paju S, Sinisalo J, Pussinen PJ, Valtonen V, Nieminen MS. Is periodontal infection behind the failure of antibiotics to prevent coronary events? *Atherosclerosis* 2006.