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# Periodontal Disease as a Risk Factor for Acute Myocardial Infarction

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## Abstract

The aim of the present study was to estimate the possible association between periodontal disease and acute myocardial infarction (AMI). A case-control study was conducted in Feira de Santana, Bahia, Brazil. The sample consisted of 290 individuals (216 controls and 74 cases). The cases were patients hospitalized with a primary diagnosis of AMI, while the controls were people accompanying the cases and other hospitalized patients, without any previous history of AMI. A complete clinical periodontal examination was performed. The clinical and laboratory diagnosis of AMI was made using electrocardiograms, CK-MB enzyme assays and troponin assays. Crude and confounder-adjusted association measurements (odds ratios-OR) were obtained at a significance level of 5%. The results presented a statistically significant principal association measurement of $OR_{crude}$ = 2.52 (95% CI [1.38 - 4.70]). After adjustment for age, sex, schooling level, smoking habit, hypertension and diabetes, this measurement became $OR_{adjusted}$ = 1.51 (95% CI [0.73 - 3.14]). The results did not show any statistically significant association between the exposure and the outcome. However, the epidemiological findings showed that periodontitis occurred more frequently among individuals with AMI.

***Keywords:*** *Periodontitis; Cardiovascular Disease; Epidemiology; Periodontal Medicine*

## Introduction

Periodontal disease has been intensely investigated as a risk factor for certain systemic complications such as premature and/or low birth weight [1], diabetes [2], pulmonary disease [3] and coronary heart disease (CDV) [4]. Because of the persistence of high rates of CDV and its major impact on public health, many of these researches have studied the possible contribution of periodontal disease in its occurrence.

The biological plausibility for this association relates to the impact of systemic inflammatory products released against periodontal infection, such as cytokines (interleukin-1 and necrosis factor tumoral) and prostaglandin, that may promote endothelial lesions and thus trigger, exacerbate or accelerate the atherosclerotic degeneration of vessels [5]. In addition to the presence of these pathogens, several other correlated factors are involved in periodontal disease, such as age, hygiene habits, schooling level, access to healthcare services and smoking [6].

A major CDV is acute myocardial infarction(AMI) that is a disease resulting from atherosclerotic processes that promote prolonged ischemia in the cardiac muscles [7]. Many risk factors for AMI are known, such is hipercholesterolemia, smoking, arterial hypertension, diabetes, genetic factors, sedentarism, diet, emotional stress, socioeconomic factors, cultural factors, leisure activities and housing are examples of these factors [8,9]. However, they do not fully explain its high frequency and morbidity-mortality rates, which have motivated the investigation of other related factors such as the periodontal disease.

Hence, given the complexity of the factors associated with these two diseases and contradictory results, controversy continues to surround the existing investigations on this topic. Some studies have confirmed that such an association exists [10-14], while others have concluded that it does not exist [15-17].

## Aim

The aim of the present study was to investigate the possible association between periodontal disease and AMI.

## Material and Methods

This case-control study was conducted in two public health institutions in the city of Feira de Santana, Bahia, Brazil: Clériston Andrade General Hospital (HGCA) and the Cardiological Institute of Northeastern Bahia (ICNB) at Hospital Dom Pedro de Alcântara, between May and December 2009.

### Sample

The Case Group was composed of individuals who had a confirmed diagnosis of a first occurrence of AMI. On the other hand, the Control Group was formed by individuals who were accompanying the cases diagnosed as AMI or who were accompanying other patients admitted to the clinical medicine and surgical clinics of both hospitals. All patients were given information about the study and agreed to participate in the study after signing a free and informed consent form.

We calculated a minimum sample of 69 cases and 207 controls, with a power of 80% and confidence interval of 95%. It was based on a study previously conducted in one of the hospitals involved in this study that estimated the frequency of periodontitis of 95% among patients with CVD and 81% in those with no history of CVD [18]. The sample was determined thorough the Epi-Info software*, maintaining the proportions of three controls for each case.

Out of 458 individuals initially evaluated, 168 were excluded considering the following criteria: had undergone periodontal treatment within the last three months preceding the investigation; had presented fewer than four teeth in total; had presented other cardiopathies or a previous history of AMI; history of percutaneous coronary revascularization within the last six months; history of surgery within the last two months; or inability to communicate verbally. The final sample consisted of 290 individuals, of whom 216 were controls and 74 were cases.

This study was approved by the Ethics Committee of Feira de Santana State University, Bahia, Brazil (protocol no. 025 /2004).

## Data Gathering Procedures

All subjects who participated in the study answered a questionnaire in order to obtain data relating to sociodemographic, biological and lifestyle factors.

All clinical measurements performed were obtained by a single examiner(NSAF), trained by as experience specialist professional (ISGF). The probing depth(PD) procedures and recession/hyperplasia were performed and recorded at six sites for each tooth. The probing depth(PD) and recession/hyperplasia procedures were performed and recorded at six sites for each tooth: mesiovestibular, mesiolingual, distovestibular, distolingual, mid-vestibular and mid-lingual. In addition, bleeding on probing was determined as the rate observed at the abovementioned six sites, i.e. according to whether bleeding occurred within 10 seconds after removing the probe from the pocket or sulcus. All clinical measurements were made using a Williams probe† and the reproducibility and concordance of the clinical measurements were calculated by means of the intra-examiner kappa index for PD (0.6017) and recession/hyperplasia (0.6863) and inter-examiner kappa index for PD (0.6080) and recession/hyperplasia (0.6671).

**AMI was diagnosed** by the cardiologist at the hospital(JMN), in accordance with the clinical symptoms presented by the patients at the time of admission to the healthcare institute, i.e. with or without new ischemic electrocardiographic abnormalities, and in accordance with laboratory blood tests to assay the enzyme levels of cardiac necrosis markers, i.e. curves for the MB fraction of creatine kinase(CK-MB) and for troponin. Thus, the diagnosis of AMI was confirmed by characteristic gradual increase in troponin T or I, or faster increase and decrease in CK-MB, associated with at least one of the following criteria: anginous or presumably anginous pain, or the ischemic equivalent (intense sweating, feelings of imminent death or syncope); development of pathological Q waves on the electrocardiogram; or electrocardiographic abnormalities indicative of elevated ischemia or depression of the ST segment.

* Epi-Info, version 6.04, Centers for Disease Control and Prevention, Atlanta, GA
† Hu-Friedy, Chicago, USA

Individuals who had at least 30% of their teeth presenting at least one site with clinical attachment level ≥ 5 mm were considered as having chronic periodontitis, according to criteria established by Hass., *et al* [19].

## Data analysis procedures

Firstly, bivariate analysis was performed in order to describe the sample according to the distribution of the covariables of interest, in relation to the dependent variable(AMI). The chi-square statistical test was used and the odds ratio association measurement was estimated, along with its respective 95% confidence interval.

Stratified analysis was then applied in order to evaluate any association between periodontitis and AMI. This approach made it possible to investigate covariables that might be effect modifiers and/or confounding variables. In these stratified analyses, the odds ratios of the specific strata were compared by means of the Breslow-Day test, at a significance level of 0.20.

Next, multivariate analyses were performed through application of logistic regression. Covariables that might be effect modifiers were again investigated, by introducing product terms into the complete model, applying the likelihood ratio test with significance level of 10%. The confounding effect of the covariables was also investigated employing of the "backward" strategy. Covariables were considered to be confounders if there was a difference of at least 10% between the crude and adjusted association measurements (OR).

The final regression model was established from the preceding analyses and/or in accordance with the previously established theoretical and empirical bases. The diagnosis of the final model was assessed according to the goodness of fit of the model, through the Hosmer-Lemeshow test, and according to the discriminatory capacity of the model, through the area under the ROC curve. The data analysis was performed using the statistical software program STATA 10.0.

## Results

In this sample males predominated among the cases and females among the controls. The mean age was 51.02 years and the median was 49 years. The mean number of teeth present among the sample was 16.53 teeth and the median was 17.00 teeth.

The distribution of the sociodemographic characteristics and those relating to general and oral health conditions and lifestyle among the participants (Tables 1 and 2) showed that the case and control groups were comparable for most of the covariables evaluated, except for the hospital where the participant was evaluated, age, sex, individual and family income, presence of self-reported hypertension, and smoking habit ($p \leq 0.05$).

| Characteristics | Controls (N = 216) | Cases (N =74) | Total (N = 290) | OR [95% CI] |
|---|---|---|---|---|
| | N % | N % | N % | |
| **Place of residence** | | | | |
| Urban | 166 76.8% | 59 79.7% | 225 77.6% | 0.84 [0.40- 1.66] |
| Rural | 50 23.2% | 15 20.3% | 65 22.4% | |
| **Hospital** | | | | |
| HGCA | 208 96.3% | 48 64.9% | 256 88.3% | 14 [5.68 – 37.82] [a] |
| ICNB | 8 3.7% | 26 35.1% | 34 11.7% | |
| **Age** | | | | |
| ≤ 49 years | 133 61.6% | 19 25.7% | 152 52.4% | 4.63 [2.49 – 8.8] [a] |
| > 49 years | 83 38.4% | 55 74.3% | 138 47.6% | |
| **Sex** | | | | |
| Female | 190 88.0% | 24 32.4% | 214 73.8% | 15.2 [7.69 – 30.2] [a] |
| Male | 26 12.0% | 50 67.6% | 76 26.2% | |
| **Piped water supply** | | | | |
| Yes | 184 85.2% | 64 86.5% | 248 85.6% | 0.89 [0.37 - 2.0] |
| No | 32 14.8% | 10 13.5% | 42 14.5% | |
| **Conjugal situation** | | | | |
| With companion | 147 68.1% | 50 67.6% | 197 67.9% | 1.02 [0.55 - 1.85] |
| Without companion | 69 31.9% | 24 32.4% | 93 32.1% | |
| **Income** | | | | |
| > 1 min. salary | 43 19.9% | 33 44.6% | 76 26.2% | 0,30 [0.16 - 0.56] [a] |
| ≤ 1 min. salary | 173 80.1 % | 41 55.4% | 214 73.8% | |
| **Family income** [b] | | | | |
| > 1.16 min. salary | 93 43.1% | 52 70.3% | 145 50.0% | 0.31 [0.17 -0.51] [a] |
| ≤ 1.16 min. salary | 123 56.9% | 22 29.7% | 145 50.0% | |
| **Household density** | | | | |
| ≤ 3 dwellers | 93 43.1% | 47 63.5% | 140 48.3% | 0.43 [0.24 - 0.77] [a] |
| > 3 dwellers | 123 56.9% | 27 36.5% | 150 51.7% | |
| **Number of children** [c] | | | | |
| ≤ 3 children | 116 53.9% | 34 45.9% | 150 51.9% | 1.37 [ 0.78 - 2.42] |
| > 3 children | 99 46.1% | 40 54.1% | 139 48.1% | |
| **Schooling level** | | | | |
| ≥ 4 years of study | 151 69.9% | 49 69.2% | 200 69.0% | 1.2 [0.64 - 2.14] |
| < 4 years of study | 65 30.1% | 25 33.8% | 90 31.0% | |

***Table 1:*** *Sociodemographic characteristics (n and %) of the case group (with acute myocardial infarction) and control group (without any history of acute myocardial infarction). Feira de Santana, Bahia, Brazil, 2009 (N = 290).*

[a] *Statistically significant: $p \leq 0.05$*

[b] *Minimum monthly salary at the time of data collection: R$ 465,00 (US$ 201.04)*

[c] *Information on one case was lost*

| Characteristics | Controls (N = 216) | Cases (N = 74) | Total (N = 290) | OR [95% CI] |
|---|---|---|---|---|
| | N % | N % | N % | |
| **Last dentist consultation** | | | | |
| Less than 1 year ago | 181 83.2% | 69 93.2% | 250 86.2% | 0.37 [0.11-1.0] |
| More than 1 year ago | 35 16.2% | 5 6.8 % | 40 13.8% | |
| **Blood pressure measured regularly** | | | | |
| Yes | 166 76.9% | 58 78.4% | 224 77.2% | 0.91 [0.45 -1.79] |
| No | 50 23.1% | 16 21.6% | 66 22.8% | |
| **Ever visited a dentist?** | | | | |
| Yes | 214 99.1 % | 72 97.3% | 286 98.6 % | 2.97 [0.21- 41] |
| No | 2 0.9 % | 2 2.7 % | 4 1.4% | |
| **Reason for visiting dentist**[a] | | | | |
| Prevention | 142 65.7% | 42 56.8 % | 184 63.5 % | 1.46 [0.81- 2.59] |
| Oral problems | 74 34.3% | 31 41.9% | 105 36.2% | |
| **Dental flossing** | | | | |
| Yes | 77 35.6% | 30 40.5% | 107 36.9% | 0.81 [0.45 -1.45] |
| No | 139 64.4% | 44 59.5% | 183 63.1% | |
| **Previous gingival treatment** | | | | |
| Yes | 23 10.6% | 7 9.5% | 30 10.3% | 1.14 [0.44 - 3.29] |
| No | 193 89.4% | 67 90.5% | 260 89.7% | |
| **Systemic infection** | | | | |
| No | 212 98.15% | 69 93.2% | 281 96.9% | 3.84 [0.79 - 19] |
| Yes | 4 1.85% | 5 6.8% | 9 3.1% | |
| **Hypertension** | | | | |
| No | 138 63.9% | 33 44.6% | 171 59.0% | 2.18 [1.84- 3.89][b] |
| Yes | 78 36.1% | 41 55.4% | 119 41.0% | |
| **Kidney disease** | | | | |
| No | 211 97.7% | 273 98.7% | 284 97.9 % | 0.57 [0.02- 5.29] |
| Yes | 5 2.3% | 1 1.3% | 6 2.1% | |
| **High LDL cholesterol** | | | | |
| No | 175 81.0% | 53 71.6% | 228 78.6% | 1.69 [ 0.86-3.22] |
| Yes | 41 19.0% | 21 28.4% | 62 21.4% | |
| **Diabetes** | | | | |
| No | 204 94.4% | 66 89.2% | 270 93.1% | 2.0 [0.69- 5.7] |
| Yes | 12 5.6% | 8 10.8% | 20 6.9% | |
| **Systemic disease in family** | | | | |
| No | 160 74.1% | 54 73.0% | 214 73.8% | 1.0 [0.54 - 1.98] |
| Yes | 56 25.9% | 20 27.0% | 76 26.2% | |
| **Previous heart disease** | | | | |
| No | 207 95.8% | 71 95.9% | 278 95.9 | 0.97 [0.16- 4.0] |
| Yes | 9 4.2% | 3 4.1% | 12 4.1% | |
| **Heart disease in family** | | | | |
| No | 132 61.1% | 50 67.6% | 182 62.8% | 0.75 [0.41 - 1.3] |
| Yes | 84 38.9% | 24 32.4% | 108 37.2% | |
| **Smoking habit** | | | | |
| Nonsmoker | 124 57.4% | 27 36.5% | 151 52.1% | 2.34 [1.2- 4.2][b] |
| Smoker or former smoker | 92 42.6% | 47 63.5 % | 139 47.9% | |
| **Physical activity** | | | | |
| Yes | 50 23.1% | 16 21.6% | 66 22.8% | 1 [0.55- 2.21 ] |
| No | 16 76.9% | 58 78.4% | 224 77.2% | |
| **Alcohol beverage consumption** | | | | |
| No | 152 70.34% | 50 67.6% | 202 69.7% | 1.14 [ 0.61-2.0] |
| Yes | 64 29.6 % | 24 23.4 % | 88 30.3% | |
| **Ever been an alcohol consumer?**[c] | | | | |
| No | 104 48.1% | 27 36.5 % | 131 45.2 % | 1.61 [0.9 - 2.9] |
| Yes | 48 22.2% | 23 31.1% | 71 24.5% | |

***Table 2:*** *General health conditions and lifestyle factors among the case group (with acute myocardial infarction) and control group (without any history of acute myocardial infarction). Feira de Santana, Bahia, Brazil, 2009 (N = 290).*

[a]*Information on one case was lost*

[b]*Statistically significant: p ≤ 0.05*

[c]*64 individuals in the control group and 24 individuals in the case group were not included because they had been alcohol consumers*

As for the oral clinical variables, statistically significant differences were detected for periodontitis, clinical attachment level (CAL) =1 or 2 mm, clinical attachment level ≥5 mm and visible plaque index ($p \leq 0.05$) between the two groups. Periodontitis was diagnosed in 50.0% of the control group and 71.6% of the cases (Table 3).

| Characteristics | Controls (N = 216) | Cases (N = 74) | Total (N = 290) | OR [95% CI] |
|---|---|---|---|---|
| | N % | N % | N % | |
| **Periodontitis** | | | | |
| Without periodontitis | 108 50.0% | 21 28.4% | 129 44.5% | |
| With periodontitis | 108 50.0% | 53 71.6% | 161 55.5% | 2.52 [1.38 – 4.70][a] |
| **Number of teeth** | | | | |
| ≥ 17 teeth | 117 54.2% | 38 51.4% | 155 53.4% | |
| <17 teeth | 99 45.3% | 36 48.6% | 135 46.6% | 1.11 [0.63 - 1.96] |
| **Number of teeth with Probing Depth ≥ 4 mm** | | | | |
| < 4 teeth | 107 55.4% | 38 51.4% | 145 54.5% | |
| ≥ 4 teeth | 85 44.3% | 36 48.6% | 121 45.5% | 1.19 [0.67 – 2.11] |
| **Visible Plaque Index** | | | | |
| < 57% plaque | 99 46.1% | 45 60.8% | 144 49.8% | |
| ≥ 57% plaque | 116 53.9% | 29 39.2% | 145 50.2% | 0.55 [0.30 – 0.97] [a] |
| **Bleeding on Probing** | | | | |
| ≥ 25% | 53 24.8% | 12 16.2% | 65 22.6% | |
| < 25% | 161 75.2% | 62 83.8% | 223 77.4% | 0.58 [0.26 - 1.21] |
| **Gingivitis**[b] | | | | |
| Without gingivitis | 100 92.6% | 21 100.0% | 121 93.8% | |
| With gingivitis | 8 7.4% | 0 0.0% | 8 22.2% | 0 [0 - 2.39] |
| **Number of teeth with CAL = 1 or 2** | | | | |
| ≤ 2 teeth CAL 1 + 2 | 148 68.5% | 61 82.4% | 209 72.1% | |
| > 2 teeth CAL 1 + 2 | 68 31.5% | 13 17.6% | 81 27.9% | 0.46 [0.21 -0.92] [a] |
| **Number of teeth with CAL = 3 or 4** | | | | |
| ≤ 7 teeth CAL 3 + 4 | 111 51.54% | 40 54.1% | 151 52.1% | |
| > 7 teeth NIC 3 + 4 | 105 48.6% | 34 45.9% | 139 47.9% | 0.89 [0.51 – 1.57] |
| **Number of teeth with CAL ≥ 5** | | | | |
| ≤ 5 teeth CAL ≥ 5 | 133 61.6% | 29 39.2% | 162 55.9% | |
| > 5 teeth CAL ≥ 5 | 83 38.4% | 45 60.8% | 128 44.1% | 2.48 [1.39 -4.44] [a] |

***Table 3:*** *Distribution of oral clinical characteristics between the case group (with acute myocardial infarction) and control group (without any history of acute myocardial infarction). Feira de Santana, Bahia, Brazil, 2010 (N = 290).*

*[a]Statistically significant: $p \leq 0.05$*

*[b]N = 129, corresponding to the full sample minus 161 individuals with periodontitis.*

*CAL: Clinical Attachment Level.*

Regarding the main association, an $OR_{crude}$ of 2.52(95%CI: [1.38 - 4.70]), i.e. individuals with periodontitis had a 2.52 times greater chance of developing AMI than did those without periodontitis. After the bivariate analysis, age, sex, diabetes, smoking habit, income and schooling level were selected for testing for interactions. In the stratified analysis, none of these covariables was identified as an effect modifier.

After analyzing the data for interactions and confounding, modeling was performed to seek a final logistic regression model that would satisfactorily explain the association under examination. This last analysis showed that sex, age and smoking habit were confounding factors. Although the covariables schooling level, hypertension and diabetes did not show statistically significant changes in the odds ratio, they were kept in the model because of their epidemiological relevance to this topic.

In addition, the same models incorporating covariables that were indicative of confounding or epidemiologically important were tested and evaluated for goodness of fit using the Akaike information criterion. Lastly, the final model chosen in this study on the association between periodontitis and AMI, with adjustments for age, sex, schooling level, smoking habit, diabetes and hypertension, was tested using the Hosmer-Lemeshow criterion ($p = 0.79$). The final adjusted association measurement between periodontitis and AMI was $OR_{adjusted}$=1.51 (95%CI: [0.73 - 3.14).

## Discussion

The present findings did not indicate a statistically significant association between periodontitis and AMI, even though periodontitis had occurred more frequently among individuals with AMI. Similar results were found in previous studies [16,17] although most of the studies on this association indicate a positive association between periodontitis and AMI [12,14,20,21]. Thus, there is still controversy regarding the results from these studies.

The fact that the adjusted association measurement in this study was not statistically significant, can be attributed to some residual confounding, such as the majority of the controls were female, whereas a greater proportion of the case group consisted of men with a diagnosis of AMI. Thus, the present findings need to be evaluated with caution, as a possible selection bias may have induced such outcomes.

The divergence in findings among different studies is probably due to issues related to the methods used in these studies, such as the criteria for the diagnoses of the exposure (periodontitis) and the outcome (AMI). The exposure measurement used in the present study classified individuals according to the severity of their chronic periodontitis, based on the knowledge that there is a gradational relationship between the severity of this exposure and the outcome investigated, i.e. a dose-response relationship [22]. This meant that the criterion that was used only selected individuals with the greatest severity. i.e. clinical attachment loss ≥ 5 mm. Moreover, this condition was strengthened through also taking into consideration the generalized extent of the periodontal disease. i.e. all the individuals diagnosed as presenting periodontitis had a clinical attachment loss ≥ 5 mm for more than 30% of their teeth.

There is no single clinical criterion in the literature that can be considered as gold standard for diagnosing periodontitis, leading to great variability between studies and imprecision of exposure measurements, thus making it difficult to compare findings. Implementing such large studies becomes a complex matter since unfavorable conditions for oral examination are involved, such as hospital beds and admission to intensive care units, among others. Hence, the more subjective aspects of the oral clinical examinations may not be taken into account for confirming the diagnosis of periodontal infection, such as bleeding on probing. Thus, the issues highlighted here were not taken into consideration in some studies, since only the probing depth [13], or only the bleeding on probing index were used as the clinical criterion [23].

Another important point in the present study was the extremely careful measurements performed to determine the outcome. The occurrence of AMI was established by means of clinical and laboratory diagnoses. The symptoms suggestive of ischemia and/or myocardial necrosis were confirmed by means of laboratory blood tests to evaluate the levels of the enzyme creatine kinase (CK) and its isoenzyme MB (thus constituting the CK-MB marker) and the troponin levels. These tests are traditionally used in order to obtain a precise diagnosis of this cardiological condition, in addition to the electrocardiographic evaluation. Many studies have used only information extracted from hospital patient charts [15,18,24] others used results from only one of the diagnostic tools mentioned above [25], which might have lead to inadequate and incomplete, generating classification bias.

It should also be emphasized that, in an attempt to diminish the variability when obtaining clinical parameters both at oral and at systemic level, additional care was taken, such as certification and standardization of the examiners. The periodontal clinical measurements on the 290 participants were obtained by a single dentist (NSAF), trained by an experienced peridontist (ISGF). In all participants, the cardiological diagnosis was performed by a single cardiology specialist (JMN).

Our results showed that case and control groups were homogenous for the great majority of the characteristics evaluated, thus enabling intergroup comparability. The sample was carefully selected in an attempt to avoid selection bias, particularly in view of possible distortions caused by hospital controls, consequent to the classic bias of Berkson [26]. Considering that all participants were treated in the two hospitals selected for the present study, it is possible that they shared unmeasured characteristics. This fact might have minimized the occurrence of confounding factors.

Another important precaution taken in this study was to include in the final analysis model classic variables of confusion, such as age, sex, smoking habit, schooling level, hypertension and diabetes. These variables and others that presented statistically significant differences between the comparison groups, or that were classic confounders or modifiers, were evaluated as potential confounders or effect modifiers such as age and smoking habit, among others.

Lastly, to strengthen the issue of temporality between periodontitis and AMI, all participants were evaluated for the presence of periodontitis within seven days of the AMI diagnosis. This criterion was adopted based on the knowledge that periodontal disease begins at a subclinical level as soon as dental biofilm occurs and deposition of periodontal pathogens increases if the biofilm is not removed. Individuals who are more susceptible to periodontal disease may show clinical signs of reversible inflammation at the gingival level, within seven days of dental biofilm deposition.

In summary, research on a possible association between periodontal disease and acute myocardial infarction (AMI) is on the rise. Mainly because these two conditions constitute major public health problems, given the expressive prevalence of periodontitis and the high mortality rate from cardiovascular diseases, especially associated with AMI. The present study along with other investigations, has sought to minimize the gaps in the literature dealing with this theme. However, further studies are needed in order to determine the role of periodontitis in the occurrence of cardiovascular outcomes, with appropriate study designs and samples capable of detecting the deleterious effects of these two diseases.

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**Volume 10 Issue 2 May 2017**