www.icrj.ir

Original Article

Relationship Between Periodontal Disease and Acute Myocardial Infarction

M Zamirian[1], S Raoofi[2], H Khosropanah[2], R Javanmardi[2]

[1]Department of cardiology, cardiovascular research center; [2]Faculty of Dentistry, Shiraz University of Medical Sciences, Shiraz, Iran.

Background: Conventional risk factors for coronary artery disease and myocardial infarction do not explain all of the clinical and epidemiological features of the disease. Periodontal disease is a common bacterial and destructive disorder of oral tissues. Many studies demonstrate close association between chronic periodontitis and development of generalized inflammation, vascular endothelial injury, and atherosclesis.
Periodontal disease has been convincingly emerging as an important independent risk factor for ischemic heart disease. A case - control study was carried out to assess the prevalence of periodontitis in patients with Acute myocardial Infarction (AMI) and evaluate the possible relationship between AMI and chronic periodontitis.
Patients and Methods: A number of 160 patients, aged 35 to 70 years old, enrolled in the study. Eighty patients (43 men, 37 women) were examined four days after hospitalization due to AMI. Control group consisted of 80 persons (38 men, 42 women) with normal coronary angiography. The following periodontal parameters were examined: Plaque index (PI), gingiral index (GI), bleeding on probing (BOP), probing depth (PD), clinical attachment loss (CAL) and number of sites with CAL.
Results: The case, compared to control showed significantly worse results for some periodontal variables studied: The mean of PD and PD > 3 mm, CAL, and number of sites with CAL, had worse results compared to control despite similar oral hygiene and frequency of brushing. The confounding factors for the present study were found to be hypertension and diabetes.
Conclusion: The association between periodontitis and acute myocardial infarction was significant after adjusting for conventional risk factors for AMI.

Keywords: Coronary Artery Disease, Periodontal Disease, Acute Myocardial Infarction.

Introduction

Epidemiologic and pathologic studies suggest that only half to two thirds of cardiovascular risk is explained by the classic risk factors.[1-3] Periodontal disease is a chronic gram Negative oral infection that has been associated with an increased risk for cardiovascular events.[4-6] The chronic inflammatory process of periodontitis and the host response provide the basis for the hypothetical association between coronary Artery disease (CAD) and periodontal disease. Some individuals may respond to a given microbial or lipopolysacharide (LPS) challenge with and exaggerated intense inflammatory response, as reflected in high levels of pro inflammatory mediators such as prostaglandin E_2(PG E_2), interleukin – 1 B (IL – 1B), and tumor necrosis factor -α (TNF - α).[7]

Correspondence:
Mahmood Zamirian
Cardiovascular Research Center, Faghihi Hospital, Zand Ave.
Shiraz, Iran
Tel & Fax: +98-711-2343529
E.mail: drzamirian@yahoo.com

A number of studies have reported that periodontal disease is associated with cardiovascular outcomes such as myocardial infarction, coronary artery disease (CAD) and stroke. Two studies reported that CAD is the condition most commonly found in patients with periodontitis. [8,9]

Joshipura et al. found a significant association between teeth loss due to periodontal disease and CAD in men.[10] Paunio et al. also reported an association between missing teeth and ischemic heart disease.[11] In addition, edentulous individuals have been shown to have rates of cardiovascular disease equal to or exceeding the rates in individuals with periodontal disease.[12,13]

The purpose of present case – control study was to assess the periodontal condition of patients with AMI and verify the possible association between AMI and certain clinical periodontal parameters.

Patients and Methods:

The study protocol was approved by the ethical committee of Shiraz University of Medical Sciences. All participating subjects provided informed consent. A total of 160 patients aged 35-70 years were enrolled in the study. Cases consisted of 80 patients during their index admission with AMI. Control group consisted of 80 persons with no angiographic evidence of CAD.

AMI was diagnosed on the basis of a history of chest pain lasting ≥ 30 minutes with ischemic electrocardiographic abnormalities and serial serum creatine kinase – myocardial band (CK-MB) and serum troponin I (TnI) level.

The diagnosis of ST elevation myocardial infarction (STEMI) was made if there was ST segment elevation of 2mm or more in precordial leads or 1 mm or more in limb leads and non ST elevation myocardial infarction (NSTEMI) was diagnosed if there was ST depression and / or T wave inversion combined with typical rise of serial CK – MB and TnI levels according to local laboratory standards.

Both patients with (STEMI) and (NSTEMI) were included in the study and undergone a through oral examination after 4 days of hospitalization when all were free of anticoagulation therapy.

Subjects who used immunosuppressant and antidepressant drugs, chemotherapy, antibiotics in previous 2 months, those with renal failure, with less than 8 teeth, with periodontal treatment within the preceding two months and finally patients with concomitant valvular heart disease as well as those requiring bacterial endocarditis prophylaxis were excluded.

Every patient underwent a complete clinical examination. Method for measurement of body mass index (BMI), waist to hip ratio, and blood pressure have been described previously.[14,15] Hypertension was defined as having a systolic blood pressure≥140 mmHg. and/or diastolic blood pressure≥90 mmHg. or receiving blood pressure reducing medications. Plasma high density lipoprotein (HDL) and low density lipoprotein (LDL) cholesterol, triglyceride, and fasting serum glucose (all in mg/dl) were checked using enzymatic calorimetric determination method.

Diagnosis of hyperlipidemia was made according to National cholesterol education program adult treatment panel III (NCEP – ATP III) and also included persons consuming anti lipid medications. Participants were categorized as

Table 1. The age of control and cases

	N	Minimum	Maximum	Mean	Standard Deviation	P value
Age in controls	80	35.0	70.0	51.9	9.4	0.031
Age in cases	80	39.0	70.0	54.0	8.7	

smoker if they were current smoker and non smoker if they were not.

Periodontal Examination

The periodontal measurements were performed at the bed side by one of the researcher, using mirrors, william's probes with Michigan markings (HU–Friedy[2M]), and portable lamps (Duracell).

All teeth excluding third molars were examined for periodontal PD and CAL at six sites per tooth, using mm-graded periodontal probes.

All measurements were rounded to the lowest whole mm. Teeth and/or sites in which the cementoenamel junction could not be identified due to severe crown destruction or gross calculus deposits were excluded.[16]

The presence or absence of gingival bleeding was assessed approximately 30 seconds after pocket depth probing.

The plaque and gingival indices were determined by using the plaque index and gingival index for each tooth as described by loe.[17]

The degree of periodontitis was defined as previously reported[18] by the percentage of sites with loss of attachment > 3 mm as following: 0% = absent, 1-32% = mild, 33-66% = moderate, 67 – 100% =severe.

Although a blind observer method would have been preferable for clinical examinations, the fact that the acute myocardial infarction and angiographically normal patients were in different departments made this scenario impractical.

Statistical Analysis:

The chi–square and student t-test were used to compare the results between case and control group. To identify the risk factors as confounding factors a multiple logistic regression model was constructed. A P-value of less than 0.05 was considered significant. Analysis were performed with SPSS (windows version 13.0 software package).

Results

Mean age of control and cases were 51.9 and 54 years respectively (Table 1).

Table 2. Degree of periodontitis in persons with and without AMI

		N	Percentage	P-Value
Absent-mild	Control	23	28.8	0.016
	Case	9	11.3	
Moderate	Control	45	56.3	
	Case	52	65.0	
Severe	Control	12	15	
	Case	19	23.8	

Table 3. Different periodontal variables in case and control groups

Variable	Group	Mean	S.D	P-Value
No. of sites where PD was measured	Control	128.7	36.3	0.31
	Case	117.3	29.2	
Sum of measure PD	Control	400.4	119.3	0.77
	Case	395.2	108.7	
Sites with PD >3mm	Control	9.2	14.0	<0.001
	Case	20.4	18.4	
Sum of PD>3 mm	Control	49.8	77.4	<0.001
	Case	107.6	100.9	
No. of sites with CAL	Control	20.4	19.3	<0.001
	Case	36.4	22.1	
Sum of CAL	Control	91.9	101.2	<0.001
	Case	164.7	114.2	
BOP	Control	62.9	33.3	0.19
	Case	56.4	29.2	
GI	Control	1.64	0.36	0.68
	Case	1.62	0.38	
PI	Control	1.80	0.53	0.54
	Case	1.85	0.59	

Statistically significant differences were found between acute infarction cases and the control group in regard to the number of sites with PD > 3mm, clinical attachment loss and sum of PD and CAL (P=0.016) (Table 2).

The cases showed worse results for periodontal variables studied (gingival recession, PD > 3mm and CAL) versus control. However there were no significant difference in BOP and PI and GI between case and control group (Table 3).

Discussion:

The present study demonstrated that subjects with AMI had significantly higher proportion of sites with gingival inflammation.

In our study, we compared moderate and severe degree of periodontitis with absent to mild periodontitis.

Advanced periodontitis probably implies a sufficiently long evolution of the disease to become a risk factor for coronary artery disease. Severe periodontitis has been reported to be associated with a greater thickness of the muscle layer of the coronary artery supporting the role played by the pathogenesis of periodontitis in the formation of atheroma and subsequent acute myocardial infarction.

Advanced periodontitits is characterized by the production of acute episodes of bacteremia

that alongside with other factors, can trigger ischemic events.[19,20] Some authors have supported the systemic administration of antibiotics in patients with coronary artery disease in order to reduce the risk of these bacterial induced ischemic episodes.[21]

Destefan, et al. reported a 25% increase in risk for future coronary heart disease in association with PD after adjustment for traditional risk factors.[22]

Similarly, Beck, et al. reported an odd ratio of 1.5 for any future coronary events and odds for an ischemic stroke among individual with less than 24 teeth compared with subjects with more than 24 teeth during 12 years follow up.[23,24]

Destruction of the alveolar bone and supporting structures surrounding the teeth with the formation of significant pockets (>3 mm) is a prolonged process involving a chronically local gram negative infection and a profound local inflammatory response. Gradual break down of the oral systemic barrier with ulceration of the pocket epithelium allows entry of locally produced inflammatory mediators and gram negative bacterial components into the systemic circulation.[25,26]

The results of the present study suggest that with increasing severity of periodontitis there is a substantial risk for AMI. Our findings suggest that dentists and specialists in the treatment of periodontal disease have to identify patients at risk for AMI.

Acknowledgements

This work was financially supported by Vice Chancellor for Research of Shiraz University of Medical Sciences. The authors declare that they have no Conflicts of Interest.

References

1 Stamler J. Research related to risk factors. *Circulation* 1979; **60**:1575-87.
2 Pooling project Research Group. Relationship of blood pressure, Serum cholesterol, Smoking habit, relative weight and ECG abnormalities to incidence of major coronary events; final report of the pooling project. *J Chronic Dis.* 1978; **31**: 201-306.
3 Wilson PW, D'Ayostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. *Circulation.* 1998; **97**:1837-47.
4 Beck JD, Pankow J, Tyroler HA, et al. Dental infections and atherosclerosis. *Am Heart J.* 1999; **138**:5528-33.
5 Mattila K, Voltonen V, Nieminen M, et al. Dental infection and the risk of new coronary events: prospective study of patients with documented coronary artery disease. *Clin Infect Dis.* 1995; **20**:588-92.
6 Mattila K, Nieminen M, Valtonen V, et al. Association between dental health and acute myocardial infarction. *Br Med J.* 1989; **298**:779-82.
7 Offenbacher S, Collins JG, Yalta B, Haradon G. Role of prostaglandins in high risk periodontitis patients. In: Genco R, Hamada S, Lehner T, Me Ghee J, Mergengagen S, eds. Molecular Pathogenesis of periodontal Disease. Washington, DC: ASM Press. 1994, p.203-14.
8 Umino M, Nagao M. Systemic disease in elderly dental patients. *Int Dent J*. 1993; **43**:213-8.
9 Nery EB, Meister F, Ellinger RF, et al. Prevalence of medical problems in periodontal patients obtained from three different populations. *J Clin Periodontal*. 1987; **58**:564-8.
10 Joshipura K, Rimm E, Douglass C, et al. Poor oral health and Coronary heart disease. *J Dert Res*. 1996; **75**:1631-6.
11 Paunio K, Impivaara O, Tiekso J, et al. Missing teeth and ischemic heart disease in men aged 45-64 years. *Eur Heart J*. 1993;**14**:54-6.
12 Morrison HI, Ellison LF, Taylor GW. Periodontal disease and risk of fatal coronary heart and cerebrovascular disease. *J Cardivasc Risk.* 1999; **6**:7-11.
13 Wu T, Trevisan M, Genco RJ, et al. Periodontal disease and risk of cerebrovascular disease: The first national health and nutrition examination survey and its follow – rp study. *Arch Intern Med.* 2000; **160**: 2749-55.
14 The ARIC Investigators. ARIC Manual of Operations, No. l l: Sitting Blood Pressure. Version 1.0. Bethesda, MD: The National Heart, Lung and Blood Institute of the National Institutes of Health. 1987; p. 58-9.
15 The ARIC Investigators. ARIC Manual of Operations, No. 2: Cohort Component Procedures. Version 1.0. Chapel Hill, NC: University of North Carolina. 1987; p.86-7.
16 Rutger Persson G Ohlsson 0, Pettersson T, et al. Chronic periodontitis, a significant relationship with acute myocardial infarction. *Eur. Heart J*. 2003; **24**:2108-15.
17 Loe H. The gingival index, the plaque index, and the retention index system. *S.J Periodontal* 1967;**38**(suppl.):610-6.
18 Arbes SJ, Slade GD, Beck JD. Association between extent of periodontal attachment loss and self reported history of heart attack: an analysis of NHANES HI data. *J Dent Res*. 1999; **78**:1777-82.

19 Beck JD, Elter JR, Heiss G, et al. Relationship of periodontal disease to carotid artery intima – media wall thickness. The atherosclerosis risk communities (ARIC) Study. Arterioscler Thrmb Vasc Biol. 2001; **21**:1916-22.
20 Hezberg MC, Meyer MW. Dental plaque, platelets, and cardiovascular diseases. *Ann Periodontol*. 1998; **3**:151-60.
21 Gurfinkel E, Bozovich G, Beck E, et al. Treatment with antibiotic roxi thromycin in patients with acute non – Q – wave coronary syndromes. *Eur Heart J*. 1999; **20**:121-7.
22 Destefano F, anda R, Kahn H, et al. Dental disease and risk of coronary heart disease and mortality. *Br Med J*. 1993; **306**:688-91.
23 Beck JD, Garcia R, Heiss G, et al. Periodontal disease and cardiovascular disease. *J Periodontal.* 1996; **67**:1123-37.
24 Joshipura KJ, Hung Hc, Rimm EB, et al. Periodontal disease, tooth loss, and incidence of ischemic stroke. *Stroke*. 2003; **34**:47-52.
25 Slade GD, Offenbacer S, Be ck JD, et al. Acute – phase inflammatory response to periodontal disease in the US population. *J Dent Res*. 2000; **79**:49-57.
26 Wu T, Trevisan M, Genco RJ, et al. Examination of the relation between periodontal health status and cardiovascular risk factors: serum total and high density lipoprotein cholesterol, C – reactive protein, and plasma Fibrinogen, *Am J Epidemilo.* 2000; **151**:273-82.
27 Fredrilsson MI, Figueredo CMS, Gustafsson A, et al. Effect of periodontitis and smoking on blood leukocytes and acute – phase proteins. *J Periodontal*. 1999; **70**:1355-60.