



Cross-sectional study of periodontal status in healthy persons and those with coronary artery disease

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Abstract

Introductions: Infections and among them extravascular infection may cause atherosclerosis and provoke their complication. Some evidence suggests that dental infections and periodontal disease are involved in pathogenesis of coronary artery disease.

Materials and Methods: Patients were chosen from private cardiology clinic. In this double blind controlled cross-sectional study, patients were in two groups. Those who had CAD* by given criteria, and those in non-CAD group. Periodontal examinations were performed by periodontologist in a blind manner.

Results: Of 250 subjects screened and examined by cardiologist, 39 agreed and presented for dental examinations, of whom 10 were edentulous, and 29 were dentate. Of dentate subjects 11 had CAD and 18 were free of CAD. For all periodontal parameters, greater values were recorded for CAD group, indicating a more severe periodontal disease among CAD subjects as compared to non-CAD group. CAD subjects showed a significantly greater level of dental plaque accumulation as compared to non-CAD group ($P=0.004$). Percentage of edentulous subjects was greater among CAD subjects than non-CAD subjects.

Conclusions: All periodontal parameters were not significantly greater, among CAD patients than non-CAD control subjects, suggesting no association between poor periodontal status and coronary artery disease. Further studies are needed.

Keywords: CAD, periodontal disease

*coronary artery disease

Introduction

Cardiovascular disease is the number one killer worldwide. Recently; interest has increased in the possibility that infection may cause atherosclerosis (1).

Potential mechanisms whereby chronic infections may play a role in atherogenesis are myriad. Some of these mechanisms are: direct vessel wall colonization that may damage the vessel, enhancing the preexisting chronic inflammatory response of the body

to standard risk factors such as hyperlipidemia, and influencing pre-existing plaque by enhancing T-cell activation or other inflammatory responses that may participate in the destabilization of the intimal cap (2).

Extravascular infection might also influence the development of atheromatous lesions and provoke their complication (1). In some patients immune or autoimmune reactions may contribute to atherogenesis (3).

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Recent evidence suggests that dental infections and particular periodontal disease are involved in the pathogenesis of coronary artery disease (4).

Although Malthaner SC didn't found significant association between periodontal disease and chronic CAD (5), many investigators revealed periodontitis was significantly higher in CAD patients in comparison with general population and that it was with increased risk of CAD (6,7).

Based on the analysis of the data of 80 patients (50 CAD, 30 non CAD), there was statistically significant association between bleeding on probing (BOP) and gingival index (GI) and CAD in patients with AMI¹; and facial / lingual clinical attachment level (CAL) in patients with unstable angina (8).

In a cohort study conducted by Beck J, Garcia, levels of bone loss and cumulative incidence of total CHD² and fatal CHD indicated a biologic gradient between severity of exposure and occurrence of disease (9).

Montebugnoli et al observed oral status in 18 males at baseline, after 4 months as a control and 3 months after an intensive protocol of scaling and root planning. Preliminary results from their study suggested an association between poor oral status and CHD, and provided evidence that the improvement of periodontal status may influence the systemic inflammatory and haemostatic situation (10).

Materials and Methods

This is a double blind controlled cross-sectional study. The patients were chosen from private cardiology clinic. First, patients were examined by cardiologist.

Electrocardiography was done, echocardiography and ETT were done if necessary. Data from myocardial perfusion SPECT and

coronary angiography were noted. Patients were in two groups; those who had CAD and those without CAD.

Those in CAD group (study group) had at least one of the following criteria: 1) History of unstable angina and significant ST-T changes in ECG with positive ETT or myocardial perfusion SPECT suggesting reversible cold spots. 2) Apparent history of MI with pathologic Q waves in ECG and rising blood levels of myocardial enzymes 3).

Fixed significant atherosclerotic stenotic lesion in at least one coronary artery (LMCA, LAD, LCX, RCA) in coronary angiography. Those in non CAD group (control group) were patients without symptoms of angina or equivalent angina, with normal ECG and negative ETT without criteria of CAD group mentioned above. Patients in both groups didn't have any congenital or rheumatic heart anomaly requiring IE³ prophylaxis before periodontal examination.

Also in order to encourage patients for periodontal examination their teeth were scaled free of charge. Of 250 patients examined by cardiologist, 44 patients were chosen of whom 5 patients refused periodontal exam and were excluded from study. Periodontal exams were performed by periodontologist.

Examinations were done in a blind manner. Cardiologist didn't have any information about periodontal status, and periodontologist was unaware of results of cardiac exams of the patients. Risk factors of atherosclerosis, age and educational level were noted, and all records were encoded. Of total 39 subjects 27 were men and 12 were women. Four age groups were arranged: 4th, 5th and 6th decades of age and those older than 60 years. Of total 39 subjects 1 was in decade 4, 6 were in decade 5, 17 in decade 6 of age and 15 were older than 60 years.

¹- Acute myocardial infarction

²- Coronary heart disease

³- Infective endocarditis

Statistical analysis

Periodontal indices were pooled within each patient for each parameter, so that a single figure was produced for each subject for that periodontal parameter. CAD status of each subject was recorded as either positive or negative.

Analysis of variance with generalized linear model was used to test the relation of CAD and each of the periodontal parameters, with plaque level, age and educational level comprising the independent co-variables. Because of the presence of too many covariates, no interaction term was tested. Chi-square test was used to express the relationship between edentulousness and coronary artery disease status.

Results

Of 250 subjects who were studied and examined by the cardiologist investigator, 39

were edentulous, and 29 were dentate. Of the 29 dentate subjects 11 had CAD and 18 were free of CAD.

Table 1 shows the mean and standard deviation of periodontal parameters among CAD and non-CAD subjects. The difference between the two groups was not significant for any of the periodontal parameters, however, for all periodontal parameters, greater values were recorded for CAD group, indicating a more severe periodontal disease among CAD subjects as compared to non-CAD subjects.

Age proved to be a significant factor for the percentage of pockets deeper than 4mm ($P=0.024$) and percentage of bleeding on probing ($P=0.043$). Plaque level had significant effect on the percentage of pockets deeper than 4mm ($P=0.039$). Other co-variate effects were not significant.

Table 1: Mean \pm standard deviation of probing depth (PD), attachment level (AL), percentage of sites with probing depth > 4 (%PD >4), sites with attachment loss >4 mm (%AL >4), gingival index (GI), percentage of sites with bleeding on probing (%BOP) and the number of missing teeth among test and control patients.

	N	PD	AL	%PD >4 mm	%AL >4 mm	%BOP	Missing teeth	GI
CAD	11	3.22 \pm 0.69	4.95 \pm 2.10	29.82 \pm 29.06	60.3 \pm 35.9	0.85 \pm 0.29	11.46 \pm 8.44	1.57 \pm 0.42
Non-CAD	18	2.75 \pm 0.55	3.23 \pm 1.29	13.17 \pm 14.02	25.29 \pm 30.10	0.72 \pm 0.31	6.83 \pm 6.02	1.40 \pm 0.39
CAD status P.value		0.291	0.147	0.088	0.12	0.799	0.732	0.868
Education level P.value		0.599	0.677	0.215	0.739	0.385	0.051	0.450
Plaque index		0.076	0.147	0.039*	0.53	0.217	0.192	0.180
age P.value		0.539	0.544	0.024*	0.773	0.043*	0.102	0.970

* Statistically significant

When the age and education levels were coded among subjects, CAD group had a no significant greater value for age than non-CAD group (4.18 ± 0.750 versus 3.44 ± 1.46 , $P=0.086$).

Furthermore, lower value for education level was obtained for CAD as compared to non-CAD, however, the difference was not significant (1.27 ± 1.79 versus 1.89 ± 2.17 , $P=0.42$). CAD subjects showed a significantly greater level of dental plaque accumulation as compared to non-CAD group (2.48 ± 0.45 for CAD and 1.68 ± 0.92 for non-CAD, $P=0.004$).

Table 2 demonstrates the relationship between edentulousness and CAD. Chi-square test showed that the percentage of edentulous subjects was greater among CAD subjects (42.1%) than non-CAD subjects (10%) (Chi-square=5.27, $P<0.05$).

Table 2: Contingency table of association between edentulousness and CAD status

	Dentate	Edentate	Total
CAD	11(57.9%)	8(42.1%)	19
Non-CAD	18(90%)	2(10%)	20
total	29	10	39

Chi-square = 5.267, $P<0.05$ (d. f=1) (significant difference)

Pearson correlation coefficients between individual factors such as age, education, plaque level, hypertension, overweight status, and hypercholesterolemia in one hand and periodontal factors in another hand was measured (Table 3). Most coefficient values (r) were low, indicating a weak but direct relationship between individual risk factors of CAD and periodontal disease.

Table 3: Pearson correlation coefficient between periodontal parameters and individual factors

	Mean missing	Mean PD	N (PD>4mm)	%AL>4mm	Mean GI	Mean POB
age code	0.281	0.026	-0.214	0.083	0.033	0.412
Education	-0.406	-0.090	-0.056	-0.254	-0.269	-0.107
Mean P1	0.464	0.442	0.429	0.547	0.381	0.343
Hypertension	0.344	0.289	0.242	0.276	0.117	0.450
Weight>90kg	-0.070	-0.036	0.048	0.263	0.027	0.144
hypercholesterolemia	0.191	0.093	-0.066	0.054	0.157	0.168

Discussion

The results of this study demonstrated that all periodontal parameters were grater, although not significantly, among CAD patients than non-CAD control subjects. This hold true after adjustment for confounding factors such as age, education level and plaque level.

Two different theories could be put forward to justify these findings. First, CAD may be independent of or even promoter for periodontal disease. CAD patients most probably have had a less healthy life-style. Likewise these individuals might have had a lower standard of oral health and hygiene,

because of their general health believes.

Furthermore, CAD patients once involved with CAD may become engaged with their more important heart disease rather than their oral health. Such people may spend less time for things such as oral hygiene. This theory itself is consistent with the observation of larger degree of periodontal disease among subjects with CAD.

The second scenario is that periodontal disease precedes CAD. According to this scenario, poor oral hygiene may favor plaque accumulation and gingival inflammation.

This turns monocytes and macrophages in a hyperactive phenotype with subsequent extensive release of proinflammatory cytokines and prostaglandins. Continued periodontal destruction with edema and subtle bleeding of the gingiva may give rise to an asymptomatic bacteremia and influx of lipopolysaccharides in the circulation. Mastragelopoulos et al. demonstrated that periodontal bacteria could be recovered from atheromatous human specimens at high prevalence (11).

The production of heat shock proteins and platelet aggregation proteins may then become stimulated. All these biochemical material favour the development and progression of thromboembolic and atherogenic events. In this way, periodontal disease may be considered a risk factor for coronary heart disease. These suggested mechanisms have recently been reviewed by De Nardin E (12).

It was found from our study that edentulous subjects were more likely to be in the CAD rather than non-CAD group. It could be assumed that edentulousness, the eventual sequela of periodontal disease, in fact, represents a past history of periodontal destruction or dental infection. Such patient, theoretically, has experienced all biological events such as atherogenesis, etc. resulting from bacteremia from such infections over long periods of his / her life time.

Moreover, edentulous patients are forced to have soft foods, comprising mainly, of high calorie/lipid and low protein constituents. Such diet regimen may lead to rise in low density lipoproteins and subsequent increased monocytic function and sensitization due to injection of lipid particles by blood monocytes.

These monocytes as mentioned earlier, are risk factor for atherosclerosis and cardiovascular disease. Our results indicated poor correlation between periodontitis and important classic risk factors of CAD such as hypercholesterolemia, hypertension and

overweight.

An alternative design for studies looking at the inter-relationship between CAD and periodontal disease is a multi-variate statistical model with CAD as a response variable and periodontal disease and all other classic risk factors of CAD such as hypertension, etc. as independent variables.

Jimenez-Beato analyzed clinical and physiopathological factors which significantly supported association between periodontal pathology and cardiovascular disease.

He concluded that further studies were needed to have the proof that said association represents a determinate risk factor for the suffering of cardiovascular diseases (13). The hypothesis of association between CAD and periodontal disease has emerged nearly one decade ago. Since then many investigators have tested this hypothesis.

Although most studies show a trend in favor of this hypothesis, it still need more extensive evidence to unequivocally apply this hypothesis to health care policy.

The data may still be regarded as insufficient (14). This association, if true, has great medical-dental implications (4). Further studies using large populations are warranted.

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References

- 1- Libby P. The vascular biology of atherosclerosis; Braunwald's heart disease. A textbook of cardiovascular medicine. 7th edition. Philadelphia: Elsevier Saunders; 2005; p.921-937.
- 2- Muhlestein JB, Anderson JL. Chronic infection and coronary artery disease. *Cardiol-clin* 2003; 21(3): 333-62.

- 3- Libby P. The pathogenesis of atherosclerosis. Harrison's principles of internal medicine. 16th edition. New York: McGraw-Hill; 2005.p. 1425-1430.
- 4- Raya AS, Naeem A, Abou- E KH, EL BS. Coronary artery disease and periodontal disease: is there a link? *Angiology* 2002; 53(2): 141-8.
- 5- Malthaner SC et al. Investigation of the association between angiographically defined coronary artery disease and periodontal disease. *J Periodontol* 2002; 73(10):1169-76.
- 6- Mattila KJ. Dental infections as a risk factor for acute myocardial infarction. *Eur Heart J* 1993; 14: 51-3.
- 7- Bochniak M, Nowicka SJ, Tyrzyk S, Sobiczewski W, Rynkiewicz A. Periodontal and dental state of patients with coronary heart disease. *Przegl lek* 2004; 61(9): 910-3.
- 8- Bazile A, Bissada NF, Nair R, Siegel BP. Periodontal assessment of patients undergoing angioplasty for treatment of coronary artery disease. *J Periodontol* 2002; 73(6): 631-6.
- 9- Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996; 67 (10): 1123-37.
- 10- Montebugnoli L, Sevidio D, Miaton RA, Prati C, Tricoci P et al. Periodontal health improves systemic inflammatory and haemostatic status in subjects with coronary heart disease. *J Clin Periodontol* 2005; 32(2): 188-92.
- 11- Mastragelopoulos N, Haraszthy VI, Zambon JJ, Zafiroopoulos GG. Detection of periodontal pathogenic microorganisms in atheromatous plaque. Preliminary results. *Chirurg* 2002; 73(6): 585-91.
- 12- De Nardin E. The role of inflammatory and immunological mediators in periodontitis and cardiovascular disease. *Ann Periodontol* 2001; 6(1): 30-40.
- 13- Beato JG, Machuca PG. Heart and periodontal diseases: does evidence exist of association? *Med Oral Patol Oral Cir Bucal* 2005; 10(3): 215-20.
- 14- Lavelle C. Is periodontal disease a risk factor for coronary artery disease (CAD)? *J Can Dent Assoc* 2002; 68(3): 176-80.

خلاصه

مطالعه وضعیت پریودنتال در افراد سالم و مبتلایان به بیماری شریان کرونر

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مقدمه: عفونت‌های مختلف و از آن میان، عفونت‌های خارج عروقی ممکن است موجب تصلب شرایین و عوارض آن شوند.

شواهد نشان می‌دهد که عفونت‌های دندانی و بیماری‌های پریودنتال در پاتوژنز بیماری شرایین کرونر دخالت دارند.

روش کار: بیماران از کلینیک خصوصی قلب انتخاب شدند. در این مطالعه دو سوکور بیماران به دو دسته تقسیم شدند. آنها که با معیارهای مورد نظر بیماری شریان کرونر داشتند (گروه مورد مطالعه) و افراد بدون بیماری شریان کرونر (گروه کنترل). معاینه پریودنتال توسط متخصص بیماری‌های لثه بدون اطلاع از دو گروه فوق انجام شد.

نتایج: از ۲۵۰ بیمار که توسط متخصص قلب معاینه شدند، ۳۹ نفر با معاینه دندان و لثه موافقت کردند که از این تعداد ۱۰ نفر بدون دندان بوده و ۲۹ نفر دندان داشتند. از بیماران دارای دندان ۱۱ نفر CAD داشته و ۱۸ نفر بدون CAD بودند. از نظر معیارهای پریودنتال، در گروه CAD ارزش‌های بالاتری ثبت شد که نشان‌دهنده بیماری شدیدتر پریودنتال در گروه مورد مطالعه در مقایسه با گروه کنترل بود. بیماران CAD نسبت به گروه کنترل میزان خیلی بالاتر تجمع پلاک دندانی نشان دادند ($P = ۰,۰۰۴$). درصد افراد بی‌دندان در گروه مطالعه، بیشتر از گروه کنترل بود.

نتیجه‌گیری: همه معیارهای پریودنتال در گروه بیماران CAD نسبت به گروه کنترل تا حدودی بالاتر بود که دال بر همراهی ضعیف بیماری شریان کرونر با وضع بد پریودنتال است. در این زمینه نیاز به مطالعات بیشتر است.

واژه‌های کلیدی: بیماری شریان کرونر، بیماری پریودنتال.