Original Article

Chronic Periodontitis as a Potential Risk Factor for Coronary Heart Disease

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ABSTRACT

Introduction: Cardiovascular and periodontal diseases are common inflammatory conditions in human population. Periodontal disease is associated with elevations of several markers of chronic inflammation and because of evidences implicating chronic inflammation in the etiology of coronary artery disease (CAD), an etiologic relationship between periodontal disease and CAD has been hypothesized. This study was conducted to find out whether chronic periodontitis is a potential risk factor for coronary heart disease.

Methodology: The present study was conducted in the Department of Cardiology, at a tertiary care hospital. Fifty patients who had heart disease as confirmed by coronary angiogram and met inclusion criteria were selected as cases and other fifty with no coronary heart disease served as controls. Periodontal disease data were collected by using Community Periodontal Index (CPI) and Loss of Attachment (LOA) Index.

Results: A significant difference was found between cases and controls for mean sextant values of CPI score 0, CPI score 1, and CPI score 2. A significant difference was found for mean sextant values between cases and controls for LOA score 0, 1, 2, 3, and 4. A statistically significant difference was found between individuals from case and control group with respect to highest LOA score.

Conclusion: Periodontal disease level as measured by CPI and LOA index was higher among cases with coronary artery disease when compared to controls. Chronic periodontitis can serve as a potential risk factor for coronary heart disease.

Keywords: Cardiovascular disease, Coronary artery disease, Periodontitis.

INTRODUCTION

Coronary artery disease (CAD) remains the principal cause of death in most countries, despite significant preventive and therapeutic advances. Hypertension, hyperlipidemia, diabetes mellitus, positive family history, and habit of smoking are the major risk factors for CAD. CAD is one of the leading causes of premature death in adults.[1,2] Coronary atherosclerosis is the most frequent cause of CAD and plaque disruption with superimposed thrombus is the main mechanism of myocardial infarction.

CAD and periodontitis are common inflammatory conditions among human population. Chronic infections have been implicated as an increased risk factor for coronary atherosclerosis. A key role for inflammation has been established suggesting that inflammatory processes underlie all phases of coronary atherosclerosis, from the initial formation of plaques to their progression and rupture, which lead to clinical events such as unstable angina, acute myocardial infarction, and sudden death.[2]

In periodontitis, there is a possibility of bacteremia due to the proximity of the infectious agents with the connective tissue and its vascular components. In moderate and advanced cases, the endotoxins (for example, lipopoly-saccharides of the microbial wall) can stimulate the accumulation of plaque contributing to the formation of thrombi and atheroma plaque.[3] Lipopolysaccharides that pass to the blood, along the side of inflammatory mediators such as tumor necrosis factor (TNF) or interleukin 1β can induce secretion of acute-phase proteins, such as

C-reactive protein (CRP) in the liver. These proteins can form deposits in the damaged blood vessels, with the consequent activation of phagocytes and release of nitrous oxide, contributing to the formation of atheromas.[4]

Periodontitis is associated with elevations of several markers of chronic inflammation and there is evidence implicating chronic inflammation in the etiology of CAD. Various studies[5-10] revealed a strong relationship between periodontitis and CAD, however few studies[11-13] showed no clear association between periodontitis and CAD. So, there is a need to assess whether periodontitis is a true risk factor for CAD or not. Hence, the present study was conducted to find out whether periodontitis is a risk factor for coronary artery disease.

METHODS

Prior to start of the study, the study protocol was submitted to the Institutional Ethics Committee and ethical clearance to conduct the study was obtained. Participation was voluntary, and a written informed consent was obtained from all participants.

A case control study was conducted over a period of two months in 2015-16. Inclusion criteria for cases referred to those individuals who had coronary artery disease confirmed by cardiologist using coronary angiogram. Those patients who had other co-morbidities like diabetes, liver disease, and chronic renal failure were excluded. Those who underwent periodontal treatment, current smokers, were on antibiotic administration, and edentulous patients were also excluded from the study. Finally fifty patients who had coronary heart disease confirmed by angiogram and consented to participate in the study were selected as cases. The control group (50 participants) was randomly selected among the relatives of the patients who had no history of any systemic diseases and were non-smokers at the time of examination. The controls were matched for age (mean age was 52.9±7.3 years) and sex with the case group. The data were collected by a principal investigator who was accompanied by a trained recorder to record all the details. The investigator was calibrated for intra examiner reliability, with a kappa value of 0.76. The recorder was explained about the scores and criteria used in the assessment form and trained for recording the scores on the same subjects.

The periodontal status was assessed by using Community Periodontal Index (CPI) and Loss of Attachment (LOA) Index (Table 1) as per WHO methodology 1997.[14] Socio-demographic details were obtained by personal interview.

Statistical analysis

The collected data were organized and tabulated. Excel sheets were prepared using Microsoft Office. Then the data were subjected to various statistical tests using SPSS 17.0.0 software. Student t-test was used to compare mean CPI and LOA scores between cases and controls. Chi-

Table 1: Community Periodontal Index (CPI) and Loss of Attachment (LOA) Index

Community Periodontal Index (CPI)[14]	
Code 0	Healthy
Code 1	Bleeding observed, directly or by using a mouth mirror, after probing
Code 2	Calculus detected during probing, but all of the black band on the probe visible
Code 3	Pocket 4-5 mm in depth (gingival margin situated on black band on the probe)
Code 4	Pocket 6 mm or more (black band of the probe not visible)
Code X	Excluded sextant (less than two teeth present)
Loss of Attachment (LOA)[14]	
Code 0	LOA 0-3 mm (CEJ not visible and CPI score 0-3) If the CEJ is not visible and the CPI score is 4 or if the CEJ is visible
Code 1	LOA 4-5 mm (CEJ within black band)
Code 2	LOA 6-8 mm (CEJ between upper limit of black band and 8.5 mm ring)
Code 3	LOA 9-11 mm (CEJ between 8.5 and 11.5 mm rings)
Code 4	LOA 12 mm or more (CEJ beyond 11.5 mm ring)
Code X	Excluded sextant (less than two teeth present)

square test was used to compare number of participants with highest CPI and LOA scores between cases and controls. Level of significance was kept as $p \leq 0.05$.

RESULTS

Table 2 shows the distribution of cases and controls according to sex. Distribution of subjects with mean sextant values according to CPI values is shown in table 3. Among the cases, mean sextant value for CPI score 0 was 0.68±1.28, for CPI score 1 was 2.0±1.69, 2.04±1.86 for CPI score 2, 2.04±1.86 for CPI 3, and 0.10±0.30 for CPI score 4. We found a significant difference between cases and controls for mean sextant values of CPI score 0, CPI score 1, and CPI score 2. No significant difference was found between mean sextant values for CPI score 3, CPI score 4, and CPI score X between cases and controls.

Table 4 shows distribution of subjects with mean sextant values according to LOA values. A significant difference was found between cases and controls for LOA score 0, 1, 2, 3, and 4. Distribution of study participants according to highest CPI score and highest LOA score is shown in tables 5 and 6, respectively. A statistically significant difference was found between individuals from case and control group in respect with highest LOA score.

Table 2: Demographic distribution of study participants

Group of individuals	Males	Females	Total
Cases	37	13	50
Controls	36	14	50
Total	73	27	100

Table 3: Distribution of participants according to mean sextant values for CPI index or scores

	CPI 0	CPI 1	CPI 2	CPI 3	CPI 4	CPI X
Case (N= 50)	0.68±1.28	2.0±1.69	2.04±1.86	0.34±0.84	0.10±0.30	0.82±1.98
Control (N = 50)	2.48±2.19	0.30±0.76	1.68±1.42	0.3±0.82	0.06±0.24	1.12±1.95
p value	0.00 (S)	0.00 (S)	0.01 (S)	0.66 (NS)	0.14 (NS)	0.48 (NS)

S; Significant p value < 0.05 NS; Not Significant

Table 4: Distribution of participants according to mean sextant values for LOA index or scores

Group	LOA 0	LOA 1	LOA 2	LOA 3	LOA 4	LOA X
Case (N= 50)	0.41±1.01	1.61±1.85	2.41±1.91	0.47±0.96	0.27±0.63	0.84±2.00
Control (N = 50)	3.68±2.34	0.72±0.88	0.40±0.80	0.08±0.44	0.01±0.20	1.12±1.95
p value	0.00 (S)	0.00 (S)	0.00 (S)	0.00 (S)	0.00 (S)	0.53 (NS)

S; Significant p value < 0.05 NS; Not Significant

Table 5: Distribution of participants according to highest CPI index

	CPI 0	CPI 1	CPI 2	CPI 3	CPI 4	CPI X	Total
Case (N=50)	0	7	24	8	5	6	50
Control (N=50)	5	2	25	11	3	4	50
Total	5	9	49	19	8	10	100

Chi square p value > 0.05 = 0.10 NS; Not significant

Table 6: Distribution of participants according to highest LOA score

	LOA 0	LOA 1	LOA 2	LOA 3	LOA 4	LOA X	Total
Case (N=50)	0	2	23	11	8	6	50
Control (N=50)	17	13	14	2	0	4	50
Total	17	15	37	13	8	10	100

Chi square p value <0.5 = 0.00 Degree of freedom = 5

DISCUSSION

Cardiovascular diseases rank among the leading causes of death and thereby have an important clinical and epidemiological role. No concrete evidence has been found from studies relating periodontal disease to cardiovascular disease. Systematic reviews and meta-analysis have shown that periodontal disease may only slightly increase the risk of cardiovascular disease. In contrast, some studies have found a significant relationship between periodontal status (based on clinical measures of probing pocket depth/CAL) and acute myocardial infarction.[15]

Our results are similar to the results of the study conducted by Fadel et al[16], in which they found more severe periodontal disease in patients with CAD. Similarly, Geerts et al[17] found that periodontitis was significantly more frequent in CAD patients than in controls (CAD patients: 91%; controls: 66%). There are several biological mechanisms by which periodontal disease might be etiologically associated with CAD. First, studies have suggested that periodontal disease represents a chronic infection resulting in a chronic inflammatory state. This hypothesis is supported by many studies showing fibrinogen, CRP, serum amyloid A and Von Willebrand factor elevations in association with periodontal disease. Notably, periodontal treatment studies have shown improvements in measures of systemic inflammation such as CRP and serum IL-6 with treatment.[18-20]

The other biological consideration is intermittent bacteremia associated with periodontal disease and its possible role either in the chronic inflammatory state or more directly on endothelial tissue surfaces.[21] In addition, data from the prospective ARIC study have shown that carotid artery intima-media wall thickness was associated with severe periodontal disease; others have shown this as well.[22] While not statistically significant, data from a subsample of the ARIC study showed a relationship between periodontal disease and coronary artery calcification by CT (relative risk 1.51; 95% CI 0.544.23) after 24 years of follow-up. Further, some studies have shown increased platelet activation in vivo in association with periodontal disease, which could contribute to plaque instability and thrombosis.[23]

Periodontal disease has also been implicated as a risk factor for stroke as well as carotid atherosclerosis. Several studies evaluating this outcome were conducted among cohorts included in this review and showed relative risks for stroke in association with periodontal disease in the range of 1.2 to 3.0. In addition, a relationship was shown among individuals with peripheral vascular disease and periodontal disease, as well as tooth loss. Furthermore, data from angiography studies have shown a relationship between the extent of atherosclerosis and the degree of periodontal disease.[24] These data support the role of periodontal disease in generalized atherosclerosis and support our finding of an association between periodontal disease and CHD.

On the contrary, Bazile et al[25] found no significant association between probing depth, and CAD. In accordance to our results, studies conducted by Mattila et al[26], Persson et al[27], Pearson et al[28] have shown a strong positive association after adjustment for many or all Framingham risk factors between CAD and various measures of periodontal disease. Complex mechanisms have been proposed to establish correlation between coronary artery disease and periodontal diseases.[29-31]

Longitudinal studies with standardized measures of periodontal disease and careful follow-up can be used to find evidence between CAD and periodontal disease. The ideal longitudinal study should start in childhood and should consider socioeconomic status since this CAD risk factor could confound the identified relationship. From a public health perspective, if further studies consistently

identify periodontal disease as a risk factor for CAD and treatment studies show benefit, the implications are significant since periodontal disease is mostly avoidable and treatable when not prevented. In addition, good preventive dental care has multiple other benefits, particularly on quality of life. Furthermore, identifying individuals at higher risk for CAD than predicted by traditional risk factors could facilitate treatment of risk factors known to decrease CAD events in high-risk individuals, such as those with hyperlipidemia.

This was a case-control study, and, as such, only associations can be demonstrated and causation or direction of the association cannot be found. To definitively establish an etiological link between periodontal disease and CAD, well-planned randomized controlled trials with follow-up are required. However, there are important ethical considerations as well as feasibility issues related to a randomized trial of an intervention known to be of benefit for reasons other than the question under study. The best planned intervention trial would be one that would start in early childhood rather than adult life.

CONCLUSION

Periodontal disease level as measured by CPI and LOA index was higher among individuals with coronary artery disease as than compared to controls.

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