The association between periodontal disease parameters and severity of atherosclerosis

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ABSTRACT

Background: Atherosclerosis is the most common cause for heart attack and stroke. In the last decade, several epidemiological studies have found an association between periodontal infection and atherosclerosis. The aim of this research was to determine the possible association between chronic periodontal disease and severity of atherosclerosis.

Materials and Methods: Eighty-two subjects that were referred to Chamran Heart Hospital in Isfahan for angiography were involved in this study. Fifty-nine subjects had coronary artery obstruction (CAO) and 23 showed no obstruction after angiography. The severity of CAO was assessed. Periodontal parameters including pocket depth (PD), gingival recession (R), clinical attachment level (CAL), and bleeding on probing (BOP) of all subjects were recorded. The decayed-missing-filled (DMF) index of all subjects was also measured. For statistical analysis, Pearson correlation test, Chi-square, and independent t-test were used.

Results: There were significant positive correlation between variables R, PD, CAL, decayed (D), missing (M), DMF, BOP, and degree of CAO. However, there were no significant differences between filling variable degree of CAO (left anterior descending, left circumflex, and right coronary artery). Independent t-test showed that the mean of variables R, PD, AL, D, M, and DMF in patients with obstructed arteries were significantly higher than subjects without CAO. But there were no significant differences between variable F in two groups.

Conclusion: The results of this cross-section analytical study showed an association between periodontal disease and dental parameters with the severity of CAO measured by angiography. However, this association must not interpret as a cause and effect relationship.

Key Words: Angiography, association, coronary artery obstruction, periodontal disease

INTRODUCTION

In the last two decades, the strong association between periodontitis and many systemic conditions such as premature delivery, low weight birth infants, metabolic syndrome, and particularly cardiovascular diseases such as myocardial infarction (MI) and coronary heart diseases (CHDs) have been shown.

In this regard, epidemiologic data and meta-analyses have suggested an association between periodontal disease and cardiovascular diseases. Among

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cardiovascular diseases, atherosclerosis is the most common cause for heart attack and stroke. Most commonly, people develop atherosclerosis as a result of diabetes, genetic risk factors, high blood pressure, a high-fat diet, obesity, high blood cholesterol levels, and smoking. However, a sizable number of patients suffering from atherosclerosis do not harbor the classical risk factors. Ongoing infections like periodontal disease have been suggested to play a role in this process. Bartova et al.[12] found an association between periodontal infection and atherosclerosis in particular. Obviously, the ultimate and dangerous result of atherosclerosis would be the partial or complete obstruction of coronary arteries. Hyvärinen et al.[13] considered periodontal infection as a potential risk factor for cardiovascular disease and, in particular, atherosclerosis.

The number and degree of coronary artery obstruction (CAO) are usually measured by angiography. In angiography, the commonly number of obstructed branches namely left anterior descending (LAD), left circumflex (LCX), and right coronary artery (RCA) and their degree of obstructions are measured. Although, the association between periodontal disease and cardiovascular diseases are well established, but scarce information exists regarding the relation between periodontal disease with extend and severity of CAO. The aim of this research was to determine the possible association between periodontal (clinical attachment level [CAL], pocket depth [PD], bleeding on probing [BOP], R) and dental (D, M, F, decayed-missing-filled [DMF]) parameters with degree and number of obstructed coronary arteries measured by angiography.

**MATERIALS AND METHODS**

One hundred and fifty subjects that were referred to Chamran Heart Hospital in Isfahan for angiography were involved in this study. Patients with any systemic disease like diabetes, smokers, body mass index of more than 25 and patient with refractory periodontitis were excluded. Finally, 82 patients aged mean 55.08 (44–69) were recruited for the study. Out of 82 subjects, 58 had chronic periodontitis (28% mild, 57% moderate, and 15% severe). After performing angiography, 59 subjects had some degree of CAO, but 29 had no CAO. The details of dental and periodontal variables and percentage of CAOs are also given.

Table 1 shows the general, periodontal, dental and cardiac characteristic of study population: The mean age of subjects was 55.08 (44–69). Out of 82 subjects, 58 had chronic periodontitis (28% mild, 57% moderate, and 15% severe). After performing angiography, 59 subjects had some degree of CAO, but 29 had no CAO. The details of dental and periodontal variables and percentage of CAOs are also given.

**RESULTS**

Table 1 shows the general, periodontal, dental and cardiac characteristic of study population:

The mean age of subjects was 55.08 (44–69). Out of 82 subjects, 58 had chronic periodontitis (28% mild, 57% moderate, and 15% severe). After performing angiography, 59 subjects had some degree of CAO, but 29 had no CAO. The details of dental and periodontal variables and percentage of CAOs are also given.

Table 2 shows the frequency distribution and percentage obstruction of LAD, LCX, and RCA in all subjects.

Table 3 shows the correlation coefficient of R, PD, AL, D, M, F, DMF, and BOP with the percentage of LAD, LCX, and RCA obstruction. The Pearson correlation coefficient showed that there was a significant positive correlation between variables R, PD, AL, D, M, DMF, BOP, and the degree of coronary artery (LAD, LCX, RCA) and degree of obstruction. Periodontal parameters including PD, R, CAL, and BOP of all subjects were recorded. The DMF index of all subjects was also measured. For PD measurement, standard Williams’s periodontal probe was applied using the blind method in 6 sites of every tooth. Patients were grouped as periodontitis if they had 3 pockets with the depth of more than 3 mm. O’Leary microbial plaque index was used for recording the superficial plaques (mesial, distal, lingual, and buccal). At the examination time, a dental disclosing tablet was given to the patients. Having patients chewed the tablet and washed of the mouth, dyed surfaces were examined using the probe tip to find the soft accumulations in the dentogingival junction. For BOP, the Ainamo and Bay index (1975) based on which the probe sulcus twisted was used. Any bleeding after 10 s was considered as the BOP and recorded in the examination chart. Gingival recession was recorded in mm and CAL was calculated as the sum of PD and R at each site.
**Table 1: Characteristic of study population including details of the dental and periodontal findings with individual age and cardiac parameters**

| Subjects with and without CAO and status of periodontal and dental conditions | Numbers and Means |
|---|---|
| Number of subject | 82 |
| CAO | 59 |
| No CAO | 23 |
| Status of periodontal condition | 58 |
| Chronic periodontitis | 21 |
| Generalized gingivitis | 9 |
| Healthy gingiva | 2.62±1.62 |
| CAL (mm) | 2.84±1 (number: 59 - obstructed artery) |
| PPD (mm) | 0.52±0.67 (number: 23 - nonobstructed artery) |
| BOP | 45.2%±12.5 |
| R (mm) | 1.2±0.64 (number: 59 - obstructed artery) |
| D | 0.39±0.5 (number: 23 - nonobstructed artery) |
| M | 4.93±3.23 |
| F | 8.6±4.75 |
| DMF | 3.89±3.09 |
| LAD obstruction percentage | 54.53±45.45 |
| LCX obstruction percentage | 43.79±44.85 |
| RCA obstruction percentage | 41.35±44.88 |
| Obstructed arteries (number) | 1.47±1.13 |
| Number of patients with obstructed arteries | 59 |
| Number of patients without obstructed arteries | 23 |
| DMF: Decayed-missing-filled; RCA: Right coronary artery; LCX: Left circumflex; LAD: Left anterior descending; BOP: Bleeding on probing; PPD: Probing pocket depth; CAL: Clinical attachment level; CAO: Coronary artery obstruction; D: Decayed; M: Missing; F: Filled; R: Recession |

**Table 2: Frequency distribution and percentage of obstruction of left anterior descending, left circumflex and right coronary artery in subjects**

| Percentage of obstruction | LAD | LCX | RCA |
|---|---|---|---|
| n | Percentage | n | Percentage | n | Percentage |
| 0-50 | 35 | 42.7 | 43 | 52.4 | 48 | 58.5 |
| 51-70 | 3 | 3.7 | 3 | 3.7 | 1 | 1.2 |
| 71-90 | 17 | 20.7 | 17 | 20.7 | 11 | 13.4 |
| 91-100 | 27 | 32.9 | 19 | 23.2 | 22 | 26.9 |
| Total | 82 | 100 | 82 | 100 | 82 | 100 |

LAD: Left anterior descending; LCX: Left circumflex; RCA: Right coronary artery

**Table 3: Correlation coefficient of recession, pocket depth, clinical attachment level, decayed, missing, filled, decayed-missing-filled, bleeding on probing with percentage of left anterior descending, left circumflex and right coronary artery obstruction**

| Variables | LDA | LCX | RCA |
|---|---|---|---|
| r | P | r | P | r | P |
| R | 0.523 | <0.001 | 0.489 | <0.001 | 0.433 | <0.001 |
| PD | 0.533 | <0.001 | 0.589 | <0.001 | 0.627 | <0.001 |
| CAL | 0.607 | <0.001 | 0.632 | <0.001 | 0.635 | <0.001 |
| D | 0.500 | <0.001 | 0.515 | <0.001 | 0.495 | <0.001 |
| M | 0.468 | <0.001 | 0.674 | <0.001 | 0.669 | <0.001 |
| F | 0.027 | 0.405 | -0.063 | 0.286 | -0.026 | 0.286 |
| DMF | 0.543 | <0.001 | 0.646 | <0.001 | 0.650 | <0.001 |
| BOP | 0.574 | <0.001 | 0.638 | <0.001 | 0.575 | <0.001 |

D: Decayed; M: Missing; F: Filled; R: Recession; BOP: Bleeding on probing; PD: Pocket depth; CAL: Clinical attachment level; LCX: Left circumflex; RCA: Right coronary artery; DMF: Decayed-missing-filled

**Table 4: The mean of variables recession, clinical attachment level, decayed, missing, filled, decayed-missing-filled in patients with and without obstructed arteries**

| Variables | Nonobstructed arteries (number of subjects: 23) | Obstructed arteries (number of subjects: 59) | P |
|---|---|---|---|
| Mean | SD | Mean | SD |
| R | 0.39 | 0.5 | 1.2 | 0.64 | <0.001 |
| PD | 0.52 | 0.67 | 2.08 | 1 | <0.001 |
| CAL | 0.91 | 0.9 | 3.29 | 1.33 | <0.001 |
| D | 1.83 | 1.47 | 6.15 | 2.91 | <0.001 |
| M | 3.87 | 2.05 | 10.46 | 4.19 | <0.001 |
| F | 3.17 | 2.76 | 4.17 | 3.19 | 0.192 |
| DMF | 8.87 | 3.4 | 20.78 | 5.32 | <0.001 |

D: Decayed; M: Missing; F: Filled; R: Recession; DMF: Decayed-missing-filled; CAL: Clinical attachment level; PPD: Probing pocket depth; SD: Standard deviation

RCA) obstruction. However, there were no significant differences between variable F and CAO (LAD, LCX, and RCA).

Table 4 shows the mean of variables R, AL, D, M, F, and DMF in patients with and without obstructed arteries. Independent t-test showed that the mean of variables R, PD, AL, D, M, and DMF in patients with obstructed arteries were significantly higher than subjects without CAO. However, there was no significant difference between variable F in two groups.

**DISCUSSION**

The present study was a cross-sectional investigation of periodontal and dental parameters including PD, R, BOP, DMF, M, D, and F in patients with CAO (undergone for angiography). An association between these parameters (except filling variable) and number and degree of CAO were assessed. The results showed a strong association between parameters of periodontal disease with number and degree of CAO. These results are similar to other studies which
have proven periodontal infection as a potential risk factor for cardiovascular disease and, in particular, atherosclerosis.[6,13]

However, a cross-sectional study from Sweden by Frisk et al.[14] reported no such relation between dental infections and cardiovascular disease; however, their study population was all female and looked at only endodontic variables and CHD and did not look at periodontal variables.

The American Heart Association supports the hypothesis that there is an association between periodontitis and atherosclerotic vascular disease, independent of their confounders.[15]

Few studies have also shown the association between extent and severity of periodontitis with extent and severity of cardiovascular disease (CVD). Kodovazenitis et al.[16] in a case–control study showed a consistent association between periodontitis and acute MI (AMI). The strength of the association increased concomitantly with the robustness of the criteria used to define periodontitis.

Holmlund and Lind[17] could show in their investigation that the number of missing teeth was inversely correlated with the number of carotid arteries with atherosclerotic plaques.

Marfil-Álvarez et al.[5] in a cross-sectional and analytical study reported the first research data demonstrating that the extent and severity of periodontitis is positively associated with AMI size (in patients who underwent diagnostic coronary angiography) as measured by serum troponin I and myoglobin levels.

Also to the best of our knowledge, this is the only study looked at association of periodontal infection and extend and severity of CAO measured by coronary angiography.

There are following possible explanations for this association:

• The role of periodontal pathogens: Hyvärinen et al.,[13] reported significantly higher salivary levels of Aggregatibacter actinomycetemcomitans in patients with stable coronary artery disease (CAD) compare to subjects showing no pathological findings by coronary angiography. High salivary levels of A. actinomycetemcomitans and systemic exposure to the bacterium were associated with increased risk for CAD. In this regard, biological evidence suggests a potential causal role for periodontal bacteria that could be involved directly in atherogenesis[18,19] or indirectly by increasing circulating cytokines and inflammatory mediators. Periodontal bacteria could migrate from the gingival/dental sites to the vascular wall via the bloodstream and act directly via their virulence factors, such as gingipains, proteinases, fimbriae, and/or lipopolysaccharides. In addition, the capacity of periodontal bacteria to induce leukocyte recruitment may contribute to this biological process.[20] Numerous clinical studies have shown the presence of DNA from periodontal pathogens in atherosclerotic plaques[21] and others have recovered viable, Porphyromonas gingivalis and A. actinomycetemcomitans from plaque samples[22‑24]

• In CHD patients with periodontitis, BOP is strongly associated with some strong systemic inflammatory mediators like C-reactive protein (CRP) levels; this association possibly reflects the potential significance of the local periodontal inflammatory burden for systemic inflammation.[25]

• Endothelium damage caused by the formation of lipid stripes may lead to bacteria penetrating into blood circulation after oral cavity procedures for patients with aggressive and chronic periodontitis[13]

• The possibility of a potential role for periodontal microorganisms, especially T. forsythia, in neutrophil activation within hemorrhagic atherosclerotic carotid plaques has also been shown.[20] The results of this and many other studies indicate a strong association between periodontal infection and CHD. On the other hand, interventional studies demonstrated beneficial effects of scaling and root planning on systemic levels of CRP, fibrinogen, and white blood cells.[26] Therefore, a proper preventive and therapeutic program for periodontal diseases would be essential for these patients. Reichert et al.,[27] showed that use of floss/interdental brushes was associated with lower risk for new cardiovascular events among patients with CHD. Saffi et al.,[28] also showed that periodontal therapy lowered the level of CRP and other proinflammatory biomarkers in patients with stable CAD.

CONCLUSION

There was a clear association between periodontal disease parameter like PD, R, BOP, and obstruction of coronary arteries measured by Angiography. Therefore,
a proper treatment and good periodontal maintenance program could have a positive effect on the stability, severity, and extent of CHD. A well-controlled study which possibly compares the progression of CHD in periodontitis patients with and without a therapeutic periodontal program is suggested.

However, this was an analytical study with cross-section design, and its results could only establish an association between periodontal and oral parameters with CAO but this must not interpret as a causative or merely cause and effect relationship.

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Conflicts of interest
The authors of this manuscript declare that they have no conflicts of interest, real or perceived, financial or non-financial in this article.

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