

Original Research Article

Serum C- reactive protein level and lipid profile in periodontitis and coronary artery disease in people of Bhuj region

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ABSTRACT

Background: Many epidemiological studies have investigated the relationship between periodontal disease and coronary artery disease but their result is heterogeneous. This review article is designed to update the association between periodontal disease and cardiovascular events. Hence, the aim of the present study was to assess whether there is a link between periodontal and coronary artery disease (CAD).

Methods: This study includes 90 subjects, which were divided into 3 groups: 30 subjects -healthy individuals, 30 subjects- having Periodontitis, 30 subjects- suffering from coronary artery diseases.

Results: The mean OHI-S scores for group 1, 2 and 3 were 0.95 ± 0.60 , 2.91 ± 0.90 , 1.54 ± 0.74 respectively. CRP level group 2 and 3 were significantly higher than group 1 ($p = 0.002$ and 0.00 respectively) whereas difference between group 2 and 3 was not significantly significant. Lipid profiles, cholesterol, low density lipoprotein (LDL), triglycerides higher in group 2 as compare to 1 and 3. High density lipoprotein (HDL) levels between groups were not significantly different.

Conclusions: Based on results and other reports Periodontitis may be a risk factor for CAD and possibly there can be an association between periodontal diseases.

Keywords: C-reactive protein, Coronary artery disease, Lipid metabolism, Periodontitis

INTRODUCTION

Inflammation referring to a protective tissue response to injury has been implicated in the pathogenesis of many human diseases. It plays central role in complex multifactorial chronic inflammatory diseases including periodontitis and CAD.¹ Periodontitis is a chronic infectious/inflammatory disease of multifactorial etiology. Though it is initiated by dental plaque associated microorganism, the inflammatory process is sustained by the host. Bacteria that have shown to contribute to periodontitis include *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponoma denticloa*,

Fusobacterium nucleatum, *Prevotella intermedia*, *Eikenella corrodens*, *Streptococcus intermedius*.

Atherosclerosis is inflammatory plays a continuous role in development destabilization and rupture of atheroma.² Ischemic heart disease (IHD) refers to a group of closely related syndromes that is caused by an imbalance between myocardial oxygen demand and blood supply.³ The probable link between oral and systemic disease dates back to 1900 when the concept of 'oral sepsis' was put forward by a British physician William Hunter subsequently in 1912. It was superseded by 'focal infection' by Frank Billings.⁴

Two major mechanisms of focal infection were proposed: An actual metastasis of organisms from a focus and spread of toxins or toxic products from a remote focus to other tissues by blood stream. Though this theory was disregarded due to various reason.⁴ Over the last two decades the whole concept of focal infection has resurfaced with the work done by Matilla et al in 1989 who found a highly significant association between poor oral health and acute myocardial infarction.⁵

Of various oral health conditions researchers have investigated the relationship between Periodontitis and atherosclerotic cardiovascular diseases and have thrown light on the underlying biologic possibility that exists between them. Reason for Periodontitis being implicated could be attributed to the fact that there is a continuous bacteremia that poses a constant microbial challenge for a prolonged period.⁵ Prevalence of periodontitis is reported to be between 20-50% in population worldwide. Previous studies have determined the role of inflammation and its biomarkers in CAD. In this context, chronic Periodontitis is one of the most prevalent infection. This has been observed, for instance in the increase in CRP level which is a prognostic marker of cardiovascular risk.⁶

However, these studies were not conclusive due to lack of homogeneity in chronic periodontitis assessment and the presence of other variables such as smoking. Therefore, the association with chronic periodontitis and CAD remain inconclusive. This study has not been carried out in Bhuj region of Gujarat so we thought it appropriate to do this study in Bhuj region of Rajasthan, with the aim and objective to assess possible links between periodontitis and CAD by investigating CRP level, lipid metabolism in periodontitis and CAD patients.

METHODS

This study includes 90 subjects, which were divided into group of 30 subjects. Group 1: healthy control, Group 2: patients diagnosed with Periodontitis, Group 3: patients suffering from CAD. Subjects who had taken antibiotic or NSAID within 2 months of sample collection and patients who were pregnant, smokers and those who are contraindicated for probing or any other systemic disease were excluded.

Participants were informed in detail about the planned study and written informed consents were obtained. An ethical clearance certificate from the institution's ethical committee was obtained prior to the study. Participants were instructed to fast overnight. Armamentarium: mouth mirror, no.5 explorer (shepherd's hook), William's probe- to check pocket depth, CPITN probe.

To assess periodontal status, we have used: OHI-S (oral hygiene index simplified); RUSSELL'S periodontal index; CPITN (community periodontal index of treatment needs). OHI-S was used to assess oral hygiene status. Clinically measurable periodontal disease was principle

indicator of an oral infection- No of teeth examined are only six teeth. Only one surface of the selected tooth is examined.

A 4ml of venous blood sample was obtained from a vein in the cubital fossa using a 5ml disposable syringe and 23gauge needle. 2ml of blood sample without EDTA used to measure CRP and obtain lipid profile. CRP level: Blood sample were centrifuged at 1006.2×g for 5minutes. Serum was separated for CRP levels using a semi autonomic analyzer with CRP detection range >0.6mg/dl.

Characteristics of lipid metabolism: for the lipid profile, blood was allowed to clot, centrifuged at 1006.2×g for 20 minutes, then the serum was separated and stored at -400 °C. Serum cholesterol, triglycerides, high HDL and LDL were analyzed using COBAS integral 400 plus fully automated analyzer. All data analyzed statistically using independent t-test, p value ≤ 0.05 were considered significant.

RESULTS

The mean OHI-S scores for groups 1, 2 and 3 were 0.95±0.60, 2.91±0.90 and 1.54±0.74, respectively. The mean percentage probing pocket depth for group 2 was 63.871±11.512. CRP levels of groups 2 and 3 were significantly higher than group 1 (p= 0.002 and 0.000, respectively), whereas the difference between groups 2 and 3 was not statistically significant (Table 1). Lipid profiles showed that cholesterol, TGL and LDL levels were significantly higher for group 2 compared to both groups 1 and 3. HDL levels between groups were not significantly different (Table 2).

Table 1: Comparisons of serum CRP (mg/dl) levels of groups.

Groups	Mean±SD	p
1	0.8643±0.61342	0.002
2	2.769±0.93213	
1	0.8769±0.1234	0.000
3	3.8648±1.32065	
2	2.789±0.93214	0.153
3	3.6821±1.350974	

DISCUSSION

Periodontal disease has an association with CAD but the influence of periodontal infection/inflammation is unknown. Through this study we found higher serum CRP level in group 2 and group 3 compared to control. The damage generated by periodontitis in the epithelium contributes the passage of bacteria into the blood stream, producing transitory bacteremia. Many periodontal pathogens like streptococcus sanguis porphyromonas gingivalis, Actinomycetemcomitans can reach the circulatory system and participates in disease such rheumatic fever, bacterial endocarditis etc.

Table 2: Comparison of lipid metabolism (mg/dl) among all groups.

Groups	Cholesterol	Triglycerides	HDL	LDL
1	158±26.3	109±28.81	45±7.2	79±21.2
2	207±27.6	44±45.7	46±8.2	102±25.8
	(p =0.001)	(p= 0.017)	(p = 0.64)	(p= 0.041)
1	158±27.3	180±29.4	43±7.6	79±21.2
3	170±29.3	116±30.9	47±7.8	86±18.4
	(p=0.131)	(p=0.419)	(p=0.35)	(p=0.303)
2	209±28.8	144±46.9	50±8.7	102±25.8
3	172±29.5	116±32.4	51±7.6	86±18.4
	(p =0.017)	(p = 0.034)	(p=0.61)	(p=0.025)

ACD associated with periodontitis is not related to iron/vitamin deficiency, it is observed in cases of rheumatoid arthritis, fungal infection and neoplastic illness.⁷⁻⁹ ACD that accompanies periodontitis can be attributed to depressed erythropoiesis by systemically circulating pro- inflammatory cytokines that result from local chronic inflammatory process.

CONCLUSION

Periodontitis constitutes an active lever for systemic subclinical inflammation enhancement and contributes to endothelial and vascular dysfunction. Some evidences suggest that periodontal pathogens could initiate and perpetuate ATH. Based on result and other report periodontitis may be a risk factor for CAD and may be possible that they may share common risk factor. Although etiological association between periodontitis and CAD is not supported by present evidence but future investigation should not be discouraged as these entities are highly prevalent in developed and developing countries.

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Ethical approval: The study was approved by the institutional ethics committee

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