Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 12s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Periodontal disease as a Risk Indicator of Cardiovascular Disease: A Clinicobiochemical study

Dr.Abhishek Gautam[1], Dr.Saurabh Shekhar*[2], Dr.Shiprita Kumari[3], Dr.Kumari Upasana[4], Dr Sakshi Raina[5]

[1]M.D.S. Periodontology, Tutor,Department of Periodontics,Government Dental College and Hospital,Rahui, Nalanda, Bihar,India.

Email ID: dr.abhi90@gmail.com

[2]M.D.S., Oral Pathology, India

[3]B.D.S, India

[4]Head, Department of Periodontics,Government Dental College and Hospital, Rahui, Nalanda, Bihar,India.

[5]M.D.S. Orthodontics, Dental Officer, ECHS Polyclinic, Danapur Cantt, Bihar,India.

***Corresponding Author**

Dr. Saurabh Shekhar

M.D.S., Oral Pathology, India

Cite this paper as: Dr.Abhishek Gautam, Dr.Saurabh Shekhar, Dr.Shiprita Kumari, Dr.Kumari Upasana, Dr Sakshi Raina, (2025) Periodontal disease as a Risk Indicator of Cardiovascular Disease: A Clinicobiochemical study. *Journal of Neonatal Surgery*, 14 (12s), 947-954.

ABSTRACT

Purpose: To determine whether patients with chronic periodontitis have higher plasma concentrations of risk markers for atherosclerosis.

Materials and Methods: A randomized, controlled study was conducted on 17 patients suffering from chronic generalized periodontitis and 17 healthy individuals. Each patient underwent comprehensive periodontal examination and haematological evaluation, recorded at baseline.

Results: The groups were matched age, sex and physical built wise and no significant difference in the BMI of two groups ($p=0.354$) could be seen. Lipid profile in study group (4.4±0.5) was significantly higher statistically ($p<0.001$) as compared to the control group (3.3±0.5). Mean C-reactive protein (CRP), was found to be 4.7±2.3 mg/lt in the study group and 2.4±1.6 mg/lt in the control group, showing a significant difference statistically ($p=0.002$).

Conclusion: Periodontal status was the only differentiating factor between the two group and the cardiovascular risk factors (like CRP) were more pronounced in the severe periodontitis group, indicating that the risk of cardiovascular disease is significantly higher in these patients.

Keywords: *Cardiovascular disease, atherogenesis, periodontal disease, lipid profile, C - reactive protein..*

1. INTRODUCTION

During the last two decades, there has been an increasing interest in the impact of periodontal health on atherosclerosis and subsequent cardiovascular disease (CVD). Patients with periodontal disease share many of the same risk factors as patients with cardiovascular disease including age, gender (predominantly male), lower socioeconomic status, stress and smoking.[1]

Atherothrombosis is understood to be a disorder of inflammation and innate immunity, as well as a disorder of lipid accumulation. The process of cellular adhesion, monocyte and macrophage attachment, and transmigration of immune cells across the endothelium are crucial steps in early atherogenesis.[2]During the inflammatory process cytokines and other pro-inflammatory mediators (*e.g.*, C-reactive protein [CRP], tumor necrosis factor [TNF-α]) initiate a cascade of biochemical reactions and cause endothelial damage and facilitate cholesterol plaque attachment.[3]

There is ample clinical evidence demonstrating that many biomarkers of inflammation are elevated years in advance of the firstmyocardial infarction (MI) or thrombotic stroke and these biomarkers are highly predictive of recurrent MI, recurrent stroke, and cardiovascular death.[2]

Yet, due to the multi-factorial nature of dental infection and CVD, confirming a causal association is difficult, and the

published results are conflicting.[4-6]

Increased biomarkers of inflammation like C - Reactive protein has emerged as a potent and independent predictor of thrombotic disorder.[7] Although CRP levels correlated with the greatest risk for cardiovascular events, however elevation of other biomarkers like lipoprotein, homocysteine, total cholesterol, low density lipoprotein (LDL) cholesterol and the ratio of total cholesterol/HDL cholesterol are also significantly associated with the disease.[8]

Periodontal disease is capable of predisposing to vascular disease due to the abundance of gram-negative species which release the detectable levels of proinflammatory cytokines (*e.g.*, C-reactive protein [CRP], tumor necrosis factor [TNF] in crevicular fluid).[9]

Proinflammtory cytokines inhibit lipoprotein lipase causing lipemia, upregulating adhesion molecule expression on endothelial cells and stimulate mitogenesis and fibrinogenesis.[10]

The present study aims at determining whether patients with severe periodontitis have higher plasma concentrations of established risk markers for atherosclerosis, such as lipoproteins and CRP as compared with the control group.

Serological differences in subjects with periodontitis might provide insight into the reported epidemiological association between periodontitis and cardiovascular disease.

2. MATERIAL AND METHODS

The study was designed as randomized, controlled, full mouth clinical study.

Patient Sample

17 patients suffering from chronic generalized periodontitis visiting the outpatient department of Periodontics,Government Dental College and Hospital, Rahui,Nalanda,were selected for the study. An informed consent was taken from all the patients.

The control group consisted of 17 healthy individuals. Each group consisted of 7 males and 10 females.

INCLUSION CRITERIA

- Age group 35-60 years
- Patients suffering from chronic severe generalized periodontitis (> 30 % sites affected)
- Clinical attachment loss ≥ 6mm
- Horizontal bone loss of 1/3rd or more of the root length with bleeding on probing/ angular bony defects/ furcation involvement of multi-rooted teeth
- Patients with no contraindication to periodontal therapy

EXCLUSION CRITERIA

- Patients with known history of cardiovascular disease
- Patients suffering from any systemic illness
- Pregnant and lactating women
- Uncooperative patients

Procedure-

The patients underwent comprehensive periodontal examination, hematological, lipid and inflammatory markers were analyzed and anthropometric measurements taken.

CLINICAL EXAMINATION FOR PERIODONTAL DISEASE

- Patients underwent comprehensive periodontal examination using a UNC-15 periodontal probe
- Radiographs like OPG/IOPA were taken to assess the extent of bone loss

Clinical parameters investigated in the study-

1. Pocket depth was using periodontal probe
2. Clinical attachment loss
3. Plaque index (Loe 1964)

Haematological, lipid and inflammatory indices analysis

1. 5ml venous blood was collected and analysed.

2. Triglycerides were determined using GPO-POD method and total cholesterol using CHOD-PAD method. The lipid profile was calculated as the ratio between total cholesterol and HDL (TC/HDL).
3. hsC-Reactive protein levels were determined using turbidimetric immunoassay .
4. Differential leucocyte counts were done to assess monocyte counts.

Anthropometric measurements

1. Blood pressure was measured using a sphygmomanometer.
2. Body mass index was calculated in kg/m^2 using the formula weight/ height

DATA ANALYSIS

The data collected was tabulated and statistical analysis was carried out using 'student t test' and 'p' values were calculated.

3. RESULTS

There were 7 males (41.2%) and 10 females (58.8%) in the study group (Table I), the control group was matched accordingly and there was no difference in the gender wise proportion of the two groups. The male to female ratio of the study was 0.7:1. (Figure I)

Age wise the groups were matched. Mean age of control group was 42.4±6.7 years whereas that of study group was 42.2±7.2 years (Table II), thus showing no statistically significant difference (p=0.941) in the two groups.However, in study groups the mean number of teeth (26.3±2.5) was significantly lower (p=0.005) as compared to control group (28.6±2.0). (FigureII)

There were a mean of 10.8±3.7 pathological sites with ≥4 mm of pocket depth in study group as compared to 1.3±1.5 in control group, thus showing a statistically significant (p<0.001) difference between the two groups.

As regards mean depth of pathological was concerned, it was 2.1±2.3 mm in control group and 4.3±1.5 mm in study group, thus statistically showing significantly deeper pathological pockets in study group as compared to control group (p=0.001). (Figure III)

Evaluation of hematological and cardiovascular risk markers in two groups revealed (Table III) – significantly higher SBP and DBP in study group as compared to control group (p<0.05) (figure IV). Mean total cholesterol level in study group was 168.5±22.9 mg/dl as compared to 140.1±15.8 (p<0.001). Mean Serum HDL levels in study group (38.0±4.6 mg/dl) were significantly lower (p=0.006) as statistically compared to that in control group (43.5±6.2 mg/dl). Lipid profile (TC/HDL) in study group (4.4±0.5) was significantly higher statistically (p<0.001) as compared to that in control group (3.3±0.5) (figure V). As the groups were matched age, sex and physical built type, no significant difference in the BMI of two groups (p=0.354) could be seen.

Mean CRP, which has recently emerged as a new marker of cardiovascular risk, was found to be 4.7±2.3 mg/lt in the study group as compared to 2.4±1.6 mg/lt in control group, thus showing a significant difference statistically (p=0.002). (Figure VI)

Figure I

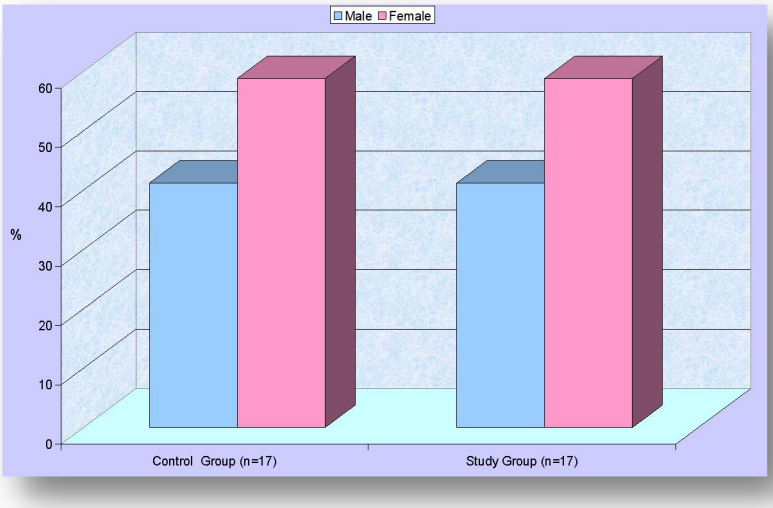


Table I: Sexwise Distribution in Two Groups

S.No.	Gender	Control Group (n=17)		Study Group (n=17)	
		No.	%	No.	%
1.	Male	7	41.2	7	41.2
2.	Female	10	58.8	10	58.8

Figure II

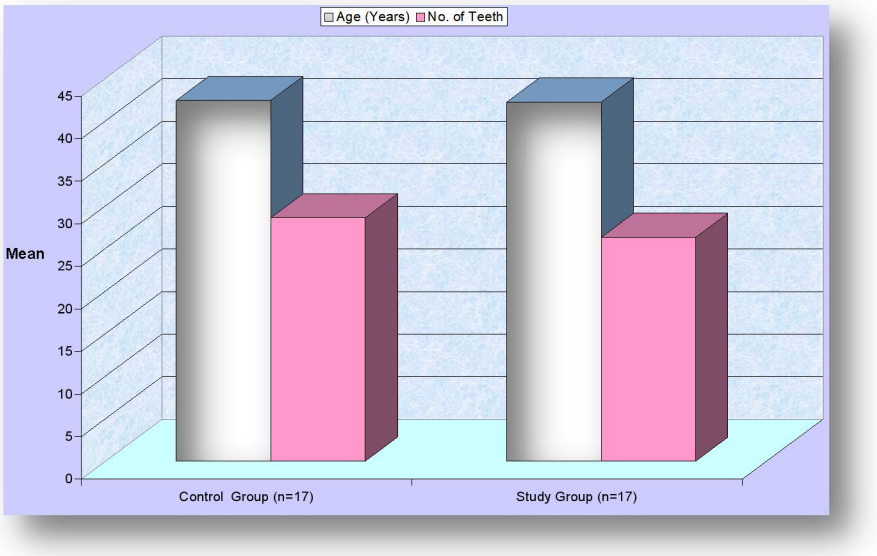


Table II: Age and Periodontal Characteristics of the two groups

S.No.	Characteristic	Control Group (n=17)		Study Group (n=17)		Statistical Significance	
		Mean	SD	Mean	SD	"t"	"p"
1.	Age	42.4	6.7	42.2	7.2	0.074	0.941
2.	No. of Teeth	28.6	2.0	26.3	2.5	2.999	0.005
3.	No. of Sites with ≥4 mm of Pocket depth	1.3	1.5	10.8	3.7	9.789	<0.001
4.	Depth of Pathological pockets (mm)	2.1	2.3	4.3	1.5	3.532	0.001

Figure III

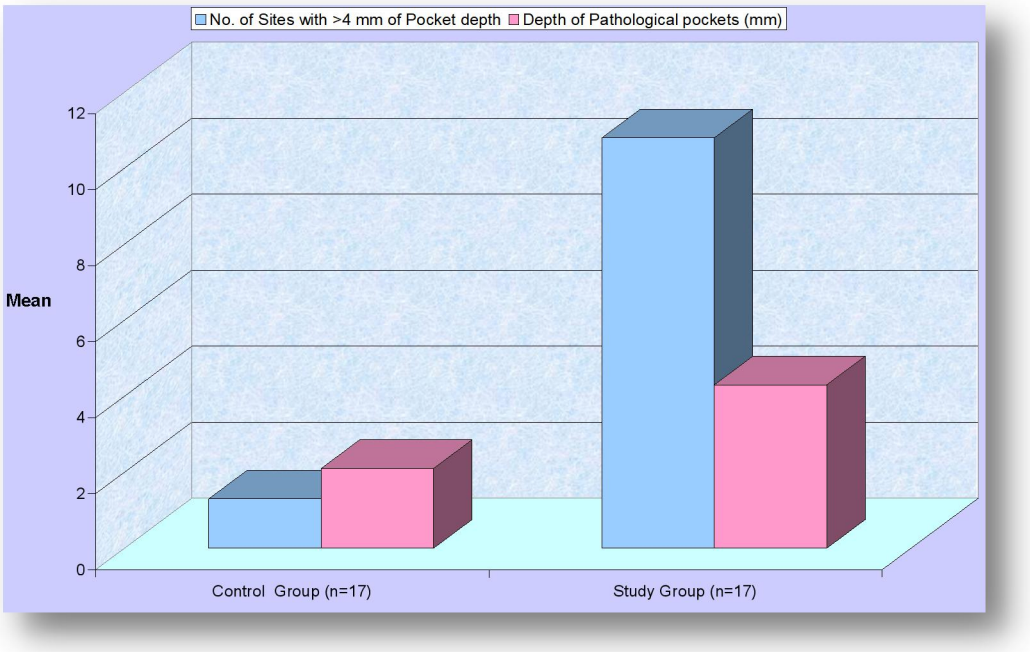


Table III: Hematological and Cardiovascular Risk Markers in Two Groups

S.No.	Marker	Control Group (n=17)		Study Group (n=17)		Statistical Significance	
		Mean	SD	Mean	SD	"t"	"p"
1.	SBP (mm of Hg)	128.8	10.5	140.8	13.8	-2.832	0.008
2.	DBP (mm of Hg)	83.6	6.0	89.1	6.7	-2.484	0.018
3.	Total Cholesterol (mg/dl)	140.1	15.8	168.5	22.9	-4.221	<0.001
4.	HDL (mg/dl)	43.5	6.2	38.0	4.6	2.943	0.006
5.	Lipid Profile (TC/HDL)	3.3	0.5	4.4	0.5	-6.757	<0.001
6.	BMI (kg/m^2)	23.0	3.1	24.1	3.6	-0.941	0.354
7.	CRP (mg/lt)	2.8	1.6	4.7	2.3	-3.352	0.002

Figure IV

Figure IV

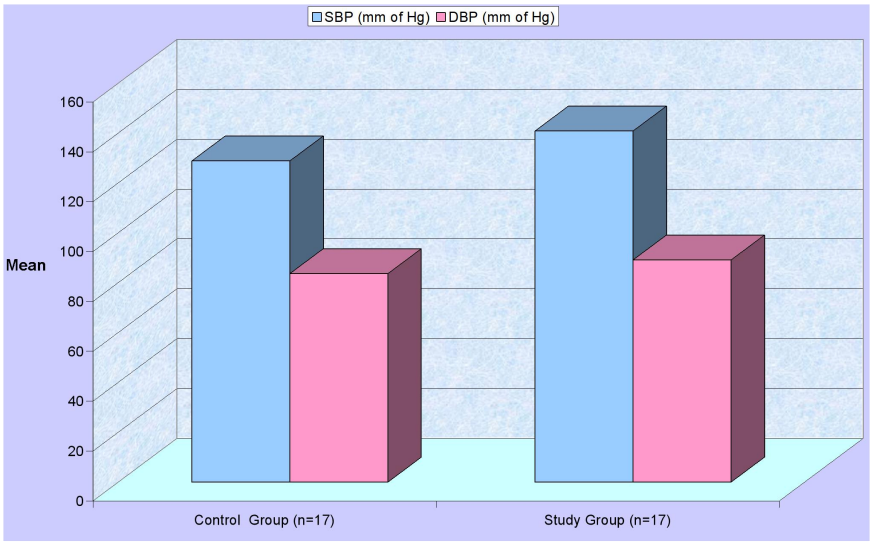


Table IV: Comparison of CRP levels between control and study groups before and after periodontal therapy

S.No.	Marker	Control Group (n=17)	Study Group (n=17)		Statistical Significance	
		(Mean± SD)	Pretreatment (Mean± SD)	Posttreatment (Mean± SD)	"t"	"p"
1.	CRP (mg/lt)	2.8 ± 1.6	4.7±2.3	3.3 ±1.6	-2.852	0.087

Figure V

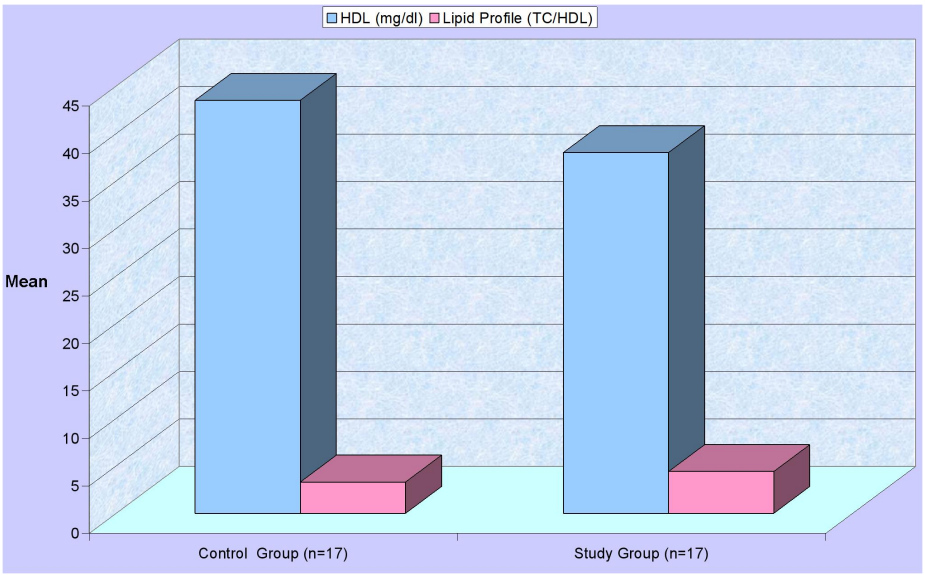
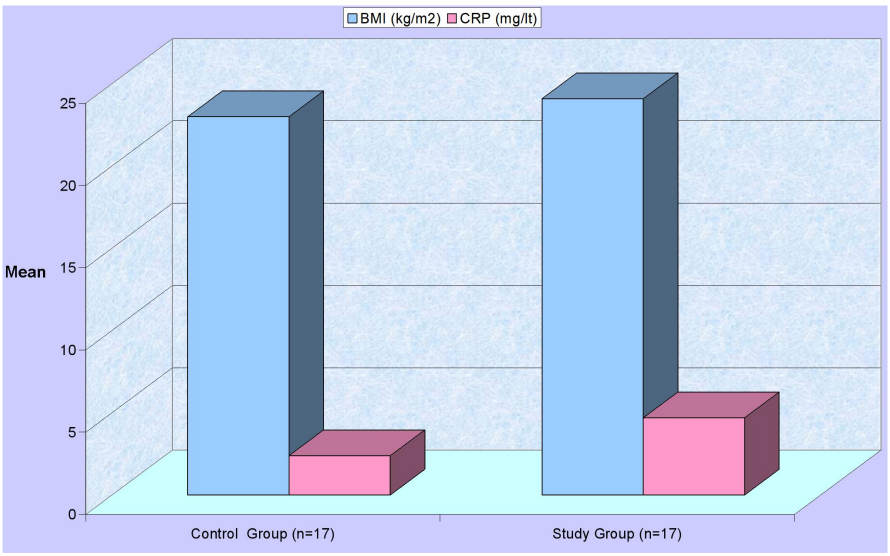


Figure VI



4. RESULT

As the two groups were matched demographically and BMI wise, and periodontal status was the only differentiating factor between the two groups, it was seen that the dependent cardiovascular risk factors were more pronounced in the severe periodontitis group, thus showing that among patients of severe periodontitis the risk of cardiovascular irregularities is significantly higher.

5. DISCUSSION

Periodontitis and atherosclerosis have many potential pathogenic mechanisms in common. Both diseases have complex causation, genetic and gender predispositions and potentially share many risk factors like age, smoking, plasma fibrinogen, white cell count, hematocrit, diabetes etc. Since periodontal infections result in low grade bacteremias and endotoxemias in affected patients, systemic effects on vascular physiology appear biologically plausible.

Four specific pathways that explain the link between cardiovascular disease and periodontal infection are-

1. Direct bacterial effects on platelets
2. Autoimmune responses
3. Invasion and/or uptake of bacteria in endothelial cells and macrophages
4. Endocrine like effect of pro-inflammatory mediators

Elevated serum biomarkers like C - reactive protein, lipoprotein, homocysteine, total cholesterol, low density lipoprotein (LDL) and the ratio of total cholesterol/HDL are said to be potent indicators of the underlying disease process.

6. CONCLUSION

However, the effects of periodontal therapy on cardiovascular disease events in patients have yet to be determined; the available data suggests that it can improve surrogate cardiovascular disease outcomes like serum biomarkers and endothelial dysfunction.

Nevertheless, further investigations have to be carried out in future to identify the consistent association of periodontal infections and potential preventive benefits of periodontal interventions.

REFERENCES

[1] Beck J, Offenbacher S., Williams R et al, Periodontitis: a risk factor for coronary heart disease? Ann Periodontol 1998; 3:127-141

[2] Ridker Paul M., Silvertown Josh D, Inflammation, C-Reactive Protein, and Atherothrombosis, J Periodontol 2008; 79:1544-1551

[3] Danesh J, Whincup P, Walker M, et al, Low grade inflammation and coronary heart disease: Prospective study and updated meta analyses, BMJ 2000; 321:199-204

[4] Mattila KJ, Nieminen MS, Valtonen VV et al, Association between dental health and acute myocardial infarction, Br Med J 1989; 298:779-81

[5] DeStafano F, nda RF, Kahn HS et al, Dental disease and a risk of coronary heart disease and mortality, Br

Med J 1993; 306:688-91

[6] Beck J, Garcia R, Heiss G et al. Periodontal disease and cardiovascular disease, J Periodontol 1996; 67:1123-37

[7] Ridker PM, Cushman M, Stampfer MJ, et al, Inflammation, asprin, and the risk of cardiovascular disease in apparently healthy men, N Eng J Med 1997; 336:973-979

[8] Ridker OM, Hennekens CH, Buring JE et al, C-Reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women, N Eng J Med 2000; 342:836-843

[9] Graves DT, Cochran D., The contribution of interleukin-1 and tumor necrosis factor to periodontal tissue destruction, J Periodontol 2003; 74:391-401

[10] Kinane Denis F., Lowe Gordon D.O., How periodontal disease may contribute to cardiovascular disease, Periodontol 2000; 23:121-126

[11] Buhlin K., Gustafsson A, Pockley A.G et al, Risk factors for cardiovascular disease in patients with periodontitis European Heart Journal 2003; 24:2099-2107

..