Research Article

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Relationship among Chronic Periodontitis and Serum Lipid Levels with Its Risk to Atherosclerosis

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ABSTRACT

Background: Regarding the high prevalence of hyperlpidaemia, which is one of the major risk factors of cardiovascular disease, and uncertain reports about the relation between periodontal disease and serum lipid profile, this study was conducted to assess this relation. The historical cohort study was conducted on 45 cases including 30 persons with chronic periodontitis as case group, and 15 healthy subjects as control group.

Both groups had the same age and weight ranges, sex and diet, without any periodontal treatment history in the past six months, underlying systemic disease such as diabetes, anti-hyperlipidemic drugs or active tobacco smoking history. Low density lipoprotein (LDL), High density lipoprotein (HDL), Triglyceride (TG) and total cholesterol (CHOL) were measured by direct enzymatic assay.

TG level was 116.713+/-30.485 mg/dl in control group and 166.197+/-69.332 in case group (P = <0.001). In control group, LDL was 90.347+/-21.565 and in case group, 105.813+/-19.197, which presents a significantly higher level (P=<0.01) in case group. Other serum level indices also showed any highly significant difference between the two groups. Our study along with other studies till date has provided evidence that periodontal disease has a causal link to atherosclerosis. Further research must be conducted to definitively establish the role of periodontal disease in the etiology of atherosclerosis.

Keywords: LDL, HDL, TG, CHOL.

INTRODUCTION

Periodontal disease (PD), or periodontitis, is defined as a bacterially induced disease of the tooth-supporting (periodontal) tissues. It is characterized by inflammation and bone loss; therefore, understanding how they are linked would help to address the most efficacious therapeutic approach. It is caused by Gram negative bacteria present on the tooth surface as microbial biofilms. Microbial biofilms are formed on the gingival tissue, which further leads to an inflammatory reaction, finally leading to destruction of periodontal ligament and alveolar bone.

The retort of a bacterium to the periodontal infection includes production of several enzymes and inflammation markers which can be analyzed both in serum and saliva[1]. Recent study indicates that hyperlipidemia may be associated with periodontal disease in healthy individuals; yet whether periodontitis causes an increase in levels of plasma lipids or whether hyperlipidemia is a risk factor for periodontal infection and cardiovascular disease.[2]

AIMS & OBJECTIVES

1. To assess the blood parameters like Total leukocyte count, Differential leukocyte count

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and thrombocytes in chronic periodontitis patients and controls.

2. To assess the lipid profile in chronic periodontitis patients and controls.

3. To evaluate the correlation of blood parameters and lipid profile with chronic periodontitis and controls.

MATERIALS & METHODS

The study procedure was undertaken according to the ethical guidelines of 1975 declaration of Helsinki and a written informed consent was obtained from the subjects. Patients were inducted from OPD of department of Periodontics. A Total 50 subjects aged between 25 to 60 years were included in the study. Out of which 30 patients were those suffering from chronic Periodontics and 20 were healthy controls.

Exclusion criteria:

Patients with Diabetes, cardiac heart disease, hypertension, those with anti hyperlipidemia drug consumption and pregnant women were excluded from the study. [1]

Inclusion criteria:

Both groups had the same age and weight ranges, sex and diet. Patients included were: 1) A minimum of teeth to be present, 2) At least 12 to be posteriors (excluding third molar),[1] 3)At least one pocket of 6mm or above depth with bone destruction to be present, and 4) Periodontal diagnosis involved measuring of pocket depth and gingival recession with the help of graduated periodontal probe and assessment of Russell's periodontal index. Venous blood was extracted and kept in two test tubes. One empty and one contained EDTA (Ethyl diamino tetra acetic acid).

Blood parameter estimation:

For blood parameters, that is total white blood cell count, Differential leukocyte count, thrombocytes and sample with EDTA was used. EDTA Samples were run in Sysmex XT 1800i fully automatic analyzer.

Lipid Profile Estimation:

For lipid profile, blood was allowed to clot, centrifuged at 3000 rpm for 20 min., serum was separated and stored at -4 degree C and used. Serum was analyzed using COBAS INTEGRA 400 plus fully auto analyzer to estimate Triglyceride (TGL), High density lipoprotein (HDL), Low density lipoprotein (LDL) & Total Cholesterol.

RESULTS

Table 1: Demographic profile: Age of normal healthy and chronic periodontitis patients.

CASE		CONTROL	
Mean of Age	Std Dev of Age	Mean of Age	Std Dev of Age
42.900	8.992	35.533	7.425

Table 2 - Demographic profile: Gender of normal healthy and chronic periodontitis patients.

Sex	Case	Control
F	9	9
M	21	6
Grand Total	30	15

Table 3: Statistical analysis of blood parameters and lipid profile in chronic periodontitis and Healthy controls.

	Case		Control		P value
Total Cholesterol	4.136	1.040	3.34	0.625	<0.001 (HS)
TGL	166.197	69.332	116.713	30.485	<0.001 (HS)
HDL	52.873	10.508	46.740	8.254	<0.05 (S)
LDL	105.813	19.197	90.347	21.565	<0.01 (S)
Thrombocytes	471.900	25.547	296.200	78.479	<0.001 (HS)
Neutrophil	76.867	7.895	63.467	6.255	<0.001 (HS)
WBC Count	11996.667	1360.650	7113.333	1904.081	<0.001 (HS)

DISCUSSION

Microbial infection when introduced to the oral cavity, cause the activation of leukocytes, then leading to soft tissue destruction. Microbial infection also causes release of cytokines and matrix metalloproteinase, which then causes destruction of connective tissue and bone.[3] The bacterial accumulation causes initiation and progression of periodontitis. This leads to vasodilation which causes an increase in rate of blood flow. Later stasis of blood stream occurs. Central stream of blood flow widens due to exudation of plasma leading to movement of neutrophils closer to vessel wall, finally leading to an increase in number of leukocytes, neutrophils and thrombocytes. [4]

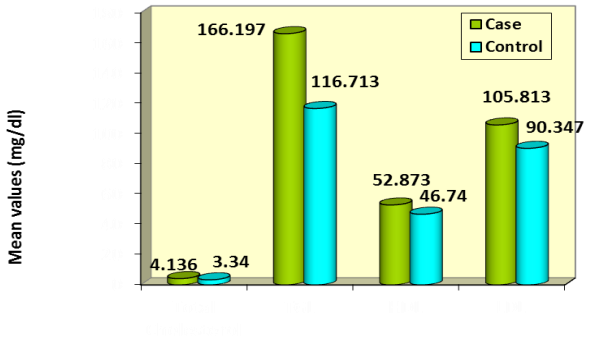

Fig 3: Graphical comparison of TGL, LDL & HDL between Case & Control Patients.

Gram negative periodontal pathogens trigger systemic release of interleukin -1 beta (IL-1 beta) and TNF alpha. This trigger of interleukin and TNF acts in two ways, on one side they cause an alteration in the fat metabolism which leads to chronic hypertriglyceridemia. On the other hand production of IL-1 beta causes continued tissue destruction and pathologic wounding of gingiva.[5]

Also, it has been found that presence of bacteremia causes an enhancement in number of antibodies against P. gingivalis, which causes subclinical endotoxemia. This in turn induces change in lipid metabolism through increased hepatic lipoprotein production. This finally causes Hypertriglyceridemia on one hand and binding of lipoprotein to endotoxins and enhances their secretion on the other hand.[6,7] It is known that activated PMNs cause an increase in release of superoxide anions and in high dietary fat PMNs lose their antibacterial function.[7]

In the present study blood parameters along with the serum total cholesterol, TGL, LDL and HDL, were assessed. A significant increase in the values was seen, which was similar to studies conducted by Losche et. Al 2000, Cutler et.al 1999, Talebi – Ardakani M et.al 2005, which demonstrated higher level of TGL and LDL in periodontitis patients. Thus this study could suggest a significant risk of Atherosclerosis in patients with chronic periodontitis.[8,9,10,11]

CONCLUSION

The conventional periodontal diagnosis methods only provide limited information about the patient's risk of future periodontal breakdown. But blood parameters and lipid profile we studied, give good information on not only assessment of status of disease but also can predict future risk of atherosclerosis in chronic periodontitis patients.

Our study along with other studies till date has provided evidence that periodontal disease has a causal link to atherosclerosis. Further research must be conducted to definitively establish the role of periodontal disease in the etiology of atherosclerosis.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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