Research Article

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Association between Chronic Periodontitis and Serum Lipid Levels with its Risk to Atherosclerosis

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ABSTRACT

Aim: 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 the relation.

Materials and Methods: The 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 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 levels (CHOL) were measured by direct enzymatic assay.

Results: 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 highly significant difference between the two groups.

Conclusion: The 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: HDL Cholesterol, LDL Cholesterol, Triglycerides.

INTRODUCTION

Periodontitis is a bacterially induced chronic inflammatory disease, which destroys the connective tissue and bone that supports teeth. 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 lead to an inflammatory reaction, finally leading to destruction of periodontal ligament and alveolar bone.

The response of an organism to the periodontal infection includes production of several enzymes and inflammatory markers which can be analyzed both in serum and saliva[1]. Recent studies

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have illustrated a correlation between high serum lipid levels and periodontitis by proving the effect of periodontal diseases as an underlying factor for hyperlipidaemia, finally leading to atherosclerosis[2].

AIMS & OBJECTIVES

1. To evaluate the blood parameters like Total leukocyte count, Differential leukocyte count and thrombocytes in chronic periodontitis patients and controls.
2. To evaluate the lipid profile in chronic periodontitis patients and controls.
3. To evaluate the relation of blood parameters and lipid profile of patients with chronic periodontitis and controls.

MATERIALS & METHODS

The study protocol was taken according to the ethical guidelines of 1975 declaration of Helsinki and a written informed consent was taken from the subjects. Patients were selected from OPD of Department of Periodontics.

Total 45 subjects aged between 25 to 60 years were recruited for the study. Out of them 30 patients were suffering from chronic Periodontics and 15 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 and diet.

1. A minimum of four teeth to be present in each quadrant.
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.
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 drawn and stored in two test tubes out of which one was 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 count sample with EDTA was used. EDTA Samples were run in Sysmex XT 1800i (Fig 1) fully automatic analyzer.

Fig 1: 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 (Fig 2) plus fully auto analyzer to estimate triglyceride (TGL), High density lipoprotein (HDL), Low density lipoprotein (LDL) & Total Cholesterol levels.

Fig 2: Cobas Integra 400 plus.

Table 1: Statistical analysis of blood parameters and lipid profile in chronic periodontitis and Healthy controls.

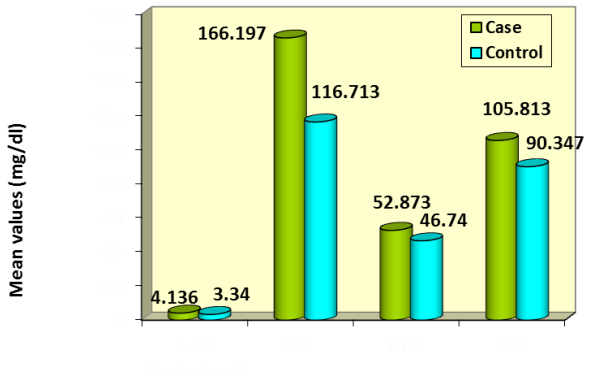
	Case		Control		P value
Total Cholesterol	4.136	1.040	3.34	0.625	<0.001 (HS)
TGL	166.197	69.332	116.713	30.485	<0.001 (HS)
HDL	52.873	10.508	46.740	8.254	<0.05 (S)
LDL	105.813	19.197	90.347	21.565	<0.01 (S)
Thrombocytes	471.900	25.547	296.200	78.479	<0.001 (HS)
Neutrophil	76.867	7.895	63.467	6.255	<0.001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

RESULTS

When compared case with controls, total cholesterol levels were highly significant (Table 1). Same was in the case with TGL ($p<0.001$), Thrombocytes ($p<0.001$), Neutrophils ($p<0.001$), and WBC count ($p<0.001$). HDL and LDL (Graph 1) were significant with p value being 0.05 and 0.01 respectively.

DISCUSSION

Microbial infection when introduced to the oral cavity cause the activation of leukocytes, then leading to soft tissue destruction. Microbial infection also leads to release of cytokines and matrix metalloproteinases which then cause destruction of connective tissue and bone[3]. This leads to vasodilation which causes an increase in rate of blood flow which is followed by stasis of blood stream. 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]. 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, one side they cause an alteration in the fat metabolism which leads to chronic hypertriglyceridemia and 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 bacteraemia causes an enhancement in number of antibodies against P.gingivalis, which causes subclinical endotoxaemia.



Graph 1: Graphical comparison of TGL, LDL & HDL between Case & Control Patients.

This in turn induces change in lipid metabolism through increased hepatic lipoprotein production. This finally causes hypertriglyceridemia and binding of lipoprotein to endotoxins that enhances their secretion[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 in this study blood parameters and lipid profile were checked thus providing good information on not only assessment of status of disease but also can predict future risk of atherosclerosis in chronic periodontitis patients.

This 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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