Original Research Article

Effect of Chronic Periodontitis on Serum Lipid Profile: A Randomized, Case Control Clinico-biochemical Study

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Abstract

Background: The localized inflammatory reaction to periodontal pathogenic bacteria or bacterial products is characterized by penetration of the periodontal tissues by inflammatory cells. Recent studies demonstrate the relation between periodontal disease (PD) and hyperlipidemia, which influence the likelihood of PD as an underlying factor for hyperlipidemia.

Aim: The aim of this Randomized, case-control type of study was to elucidate the association between serum lipid parameters in periodontally diseased patients with healthy individuals.

Material and methods: A total number of 44 subjects were segregated into two groups Group A (Control) Group B (Test). Periodontal parameters including Oral hygiene Index-Simplified (OHI-S), Gingival Index (GI), Probing Depth (PD) and Clinical Attachment Level (CAL) were recorded. Lipid profile comprising of Serum cholesterol, Triglycerides, HDL- Cholesterol and LDL was assessed and co related with periodontal parameters.

Results: The results of the present study demonstrated a positive correlation between GI, PD, OHI-S and CAL with serum cholesterol, TG and LDL. The Serum cholesterol, Triglycerides and LDL levels between both the groups showed statistical significance. But the serum HDL levels when compared between both the groups were not statistically significant.

Conclusion: The present study showed a definitive positive correlation between PD and increased lipid levels.

Key words

Periodontal disease, Lipid profile, Lipopolysaccharides.

Introduction

Evidence in the last few years has shed light on the relationship between oral health and systemic health - the potential effects of a wide range of organs and their systems. Examining the regional drifts of the disease will aid to notice the worldwide trends in the load of disease, predominantly cardiovascular disease (CVD) [1]. Chronic inflammation is the basis of the relationship between periodontal disease (PD) and other systemic inflammatory conditions, data exists that individuals with periodontitis have greater threat of presenting endothelial dysfunction, CVD, cerebrovascular accidents and preterm low birth weight infants [2, 3].

The localized inflammatory reaction to periodontal pathogenic bacteria or bacterial products is characterized by penetration of the periodontal tissues by inflammatory cells. The consequences of inflammatory progression of periodontitis systemically leads to production of Pro-inflammatory cytokines such as interleukins-1beta (IL-1β), interleukin 6 (IL-6) C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α) [4]. Tumor necrosis factor (TNF) persuades a rapid risein serum triglyceride, VLDL and cholesterol levels. Even though the mechanism by which TNF increases serum cholesterol levels is unidentified, the upsurge in hepatic cholesterol synthesis may be due to an increase in the activity of 3-hydroxy-3-methyl glutaryl coenzyme A (HMG-CoA) reductase [5].

Hyperlipidemia is a well-known disorder where there is an elevation of the serum levels of total cholesterol and triglycerides (TGL) due to lipid metabolism variation with and rise in the liver lipogenesis and lipolysis in the adipocytes. 85-90% of body lipids are TGL the most abundant lipids, TGL are the glycerol esterified at each of its three hydroxyl groups by a fatty acid, particularly fat from animal source circulates in the plasma complexed to proteins of various densities and plays an important role in the pathogenesis and development of atheroma in the arteries. LDL constitutes both lipid and protein, which transport cholesterol to tissues other than the liver. Whereas HDL comprises same lipid and protein together, which transport cholesterol to the liver for excretion in the bile [6].

Socransky S, et al. documented, pophyromonas gingivalis as the major causative microorganism that causes periodontitis, previous literatures suggests the release of endotoxin from the P.gingivalis, in the form of lipopolysaccharides (LPS) that are involved in creating a host mediated tissue destructive immune response. Recent studies demonstrate the relation between periodontal disease (PD) and hyperlipidemia, which influence the likelihood of PD as an underlying factor for hyperlipidemia [7].

Sharma et al found the association of LDL and cholesterol, and suggested that the rise in LDL and cholesterol is directly related to PI and GI index. Further research explained the association which demonstrates higher level of TGL and lower HDL among the patients suffering from periodontitis than control group significantly [8].

Aim of this Randomized, case-control type of study was to elucidate the association between serum lipid parameters in periodontally diseased patients with healthy individuals.

Materials and methods

A total number of 44 subjects were selected from Department of Periodontics, Sri Sai College of Dental Surgery. The sample size was calculated based on pilot study conducted by our research group. Ethical committee approval was taken from the institute, the nature and intention of the study was explained to the patients and an informed consent was obtained. A detailed case history which included information about the patient's overall medical status and oral status was recorded in a specially prepared proforma.

Control group (Group A) included 22 subjects within the age group 30 – 55 years having minimum of 20 teeth, minimal probing depth (< 3mm), with no mean clinical attachment loss and recession were matched with cases with respect to dietary habits, body mass index (BMI). Body mass index (BMI) was calculated as body weight (kg) divided by height (m^2) [9].

Test group (Group B) included 22 subjects within the age group 30 – 55 years definite diagnosis of chronic periodontitis was performed by one expert Periodontist based on the existence of calculus and plaque, at least one pocket with a minimum of 6mm depth, minimum attachment loss of 3 mm and bone destruction appropriate to calculus and plaque in radiograph accordingly in every quadrant.

Any of subjects having history for any systemic infection, cardiac heart disease and history of anti-cholesterol treatment, subjects with pregnancy, deleterious habits like smoking/ alcohol consumption and who had periodontal treatment in the last 6 months were excluded from study.

At the first visit a Periodontist collected a complete medical history, standard clinical periodontal parameters. For each subject, the periodontal disease status was evaluated at 4 sites per tooth (Midbuccal, Distobuccal, Mesiobuccal and lingual, deepest site was recorded) using UNC-15 probe (Hu-Friedy´s, USA). The Pocket depth (PD), Oral Hygiene Index- simplified (OHI-S), Gingival Index (GI) and Clinical attachment level (CAL) were recorded. Then, the patients were sent for haematological investigation for lipid profile on the consequent day with approximately 12 hours fasting. The lipid profile included Low density lipoprotein (LDL), serum cholesterol, triglyceride (TG) and high-density lipoprotein (HDL) estimation.

Statistical Analysis

Following the biochemical analysis of salivary samples, the results were obtained for both the groups and were subjected to statistical analysis. Statistical package for social sciences package (SPSS) version 17 was used for the statistical evaluation. Continuous variables were summarized as mean and standard deviation unpaired ‘t’ test was used for comparison of continuous variables. The level of significance was set up at 5% ($P < 0.05$).

Results

A randomized, case-control study was undertaken with 22 participants in each group to know the association with serum lipid levels and periodontal disease. Mean age of the participants in Group A was 33 ± 2.8 years, out of 22 subjects who participated in the study 10 were female and 12 were male. The mean age of the participants in Group B was 45 ± 5.7 years, out of 22 subjects who participated in the study 09 were female and 13 were male participants.

The results of the present study demonstrated a positive correlation between GI, PD, OHI-S and CAL with serum cholesterol, TG and LDL. The Serum cholesterol, Triglycerides and LDL levels between both the groups showed statistical significance. But the serum HDL levels when compared between both the groups were not statistically significant (**Table - 1, 2**).

Table - 1: Comparison of clinical variables between group A and group B.

Variables	Mean ± SD		P Value
	Group A	Group B	
BMI	23.45 ± 2.1	25.7 ± 1.87	0.047*
GI	1.34 ± 1.7	2.21 ± 0.89	0.039*
PD (mm)	2.45 ± 0.98	6.24 ± 1.4	0.007*
CAL (mm)	0.00	3.40 ± 0.78	0.001*
OHI-S	1.28 ± 0.58	4.97 ± 0.68	0.001*

* P < 0.05 statistically significant

Table - 2: Comparison of serum lipid levels between group A and group B.

Variables	Mean ± SD (mg/dl)		P Value
	Group A	Group B	
Serum Cholesterol	158.9 ± 22.1	170.8 ± 10.87	0.044*
Serum Triglycerides	101.1 ± 28.6	124.6 ± 16.54	0.039*
HDL	47.23 ± 7.67	39.89 ± 5.78	0.078
LDL	109.87 ± 24.3	133.87 ± 14.9	0.011*

* P < 0.05 statistically significant

Discussion

The present study demonstrated higher serum cholesterol, TG and LDL levels in Group B when compared to Group A. The present study is in accordance with many studies that have shown an increase in lipid profile values and PD. The mean serum HDL levels when compared between both the groups were not statistically significant [10-13].

It is established that an increase in pre-inflammatory cytokine in reaction to chronic periodontitis causes a mount in serum lipid levels. Studies have shown a number of cytokines such as TNFα, IL-1β are produced in response to systemic gram-negative LPS exposure [14]. Noack, et al. assessed neutrophil respiratory burst by whole blood chemiluminescence and they found significant increase in both PD and chemiluminescence on hyperlipidemia patients. They suggested that connection of hyperlipidemia with PD could be due to the dysfunction of neutrophils [15]. Conversely, other than the effect of infections from PD, interactions between bacterial LPS, mediators of CVD and modulating effects of dietary lipids on immune system may be important factors responsible for increased serum lipid levels in patients with PD.

On contrary to our study VelantaViciente, et al. [16] found that there was no difference in the serum lipid profile of the periodontal patients and control group. The authors established the varied results could be prejudiced by low age group of the participants, unawareness of dietary factors, mental and physical behavior. In the present study the mean age in Group B was higher than Group A participants, which is satisfactory considering chronic course of disease; Higher age could be a useful factor to set a significant association between chronic Periodontitis and increased lipid levels.

Conclusion

The present study showed a definitive positive correlation between PD and increased lipid levels. Therefore we strongly advocate the control of PD particularly as the age progresses. Within the limitation of this study, it may be concluded that the control of PD could help in limiting hyperlipidemia to certain extent.

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