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Association between Chronic Periodontitis and Serum Lipid Levels.

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ABSTRACT

Background: Hyperlipidemia is a known risk factor for cardiovascular diseases. A common biologic mechanism between systemic diseases, such as cardiovascular diseases, and periodontal diseases has been suggested. The aim of this study is to examine the association between blood lipid profile and periodontitis. Aim: To study the correlation between serum lipid profile and periodontitis. **Methods:** The levels of serum lipid profile in 60 subjects, 30 with chronic generalized periodontitis based on clinical attachment loss (CAL) constituting the test group and 30 without periodontitis constituting the control group, were measured and compared with each other. Both these groups were free from other systemic illnesses. Statistical Analysis: The mean CAL was positively correlated with serum low-density lipoprotein (LDL) cholesterol ($P < 0.01$). **Results:** The mean serum LDL cholesterol (126.62) and total cholesterol (173.32) in periodontitis patients were found to be significantly higher as compared to that of the controls. The mean CAL (5.32 mm) was positively correlated with serum LDL cholesterol. The frequency of persons with pathologic values of LDL cholesterol and total cholesterol was significantly higher in periodontitis patients compared with that of the controls. **Conclusion:** These results showed that high serum LDL cholesterol and total cholesterol may be associated with periodontitis in otherwise healthy people. However, it is unclear whether periodontitis causes an increase in the levels of serum LDL.

Keywords: Cardiovascular disease (CVD), cholesterol, low-density lipoprotein (LDL), periodontitis.

INTRODUCTION

Periodontitis is a chronic multifactorial disease which is characterized by the destruction of periodontium and loss of alveolar bone. Various modifiable and non-modifiable risk factors are associated with periodontal disease. Smoking, diabetes mellitus, microorganism, obesity, drug-induced disorders and stress are the modifiable risk factors while non-modifiable risk factors include age, gender, host response, pregnancy, genetics etc.[1] CVD is one of the foremost modifiable risk factor of periodontitis. Both periodontal disease and CVD share many common risk factors such as smoking, diabetes mellitus, age, ethnicity, sex, socioeconomic status, stress and obesity, which would possibly result in confounding any association between them.[1]

A link between periodontitis and CVD has been implicated in the past.[2] Meta-analysis of the relationship between periodontal disease and cardiovascular events has shown that subjects with periodontal diseases have higher odds of developing CVD.[3]

A possible mechanism by which periodontitis may affect cardiovascular health is chronic oral inflammation that may lead to increased blood cholesterol levels .4 Oral bacterial infections may result in the release of proinflammatory mediators such as Interleukin-1β (IL-1β), IL-6 and Tumour Necrosis Factor alpha (TNF –α) which may get released into the systemic circulation and this may further alter fat metabolism and promote atherosclerosis by directing the activation of monocytes and alteration in the lipoproteins to a more atherogenic profile.[4]

Various systemic inflammatory markers such as C-Reactive Protein (CRP), TNF-α, IL-1, IL-6, and IL-8 have been established as an inflammatory biomarkers for periodontitis and CVD.[5]

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MATERIALS & METHODS

In this cross-sectional study, a total of 75 subjects were recruited from Department of Periodontology, Govt Dental College and Hospital, Jammu, Jammu & Kashmir, India, for the present study. Out of these, subjects were excluded as they were

diagnosed with diabetes , hypertension and Smoking . Finally, 60 participants were selected based on the inclusion and exclusion criteria and were divided into two groups. The power of the study was calculated which came out to be 90%. The groups consisted of Group I- systemically healthy subjects with generalized moderate or severe periodontitis CP, Group II- systemically healthy and periodontally healthy volunteers who served as controls. The written informed consent was obtained from all participants enrolled in the current investigation.

The inclusion criteria included the subjects with (i) age ≥ 35 years and (ii) presence of at least 24 teeth (iii) CAL ≥ 5 mm in more than 30% of sites in Group A (iv) healthy and intact periodontium in Group B. The exclusion criteria included (i) presence of any systemic disease or conditions that could affect periodontal tissues. e.g. diabetes mellitus, CVD, liver diseases, metabolic syndrome or other endocrine diseases (ii) history of periodontal treatment in past six months (iii) smoking and alcoholism (iv) anomalies of blood and immune system such as anaemia and leukaemia (v) systemic medication or antibiotic treatment for the previous six months. (vi) pregnant females.

All the 60 subjects screened on the basis of probing depth (PD) and clinical attachment loss (CAL). The Simplified Oral Hygiene Index (OHI-S) was recorded to find out the mean oral hygiene status of the selected subjects. 6 On the basis of history and clinical findings, the subjects were divided into two groups, Group A with and Group B without periodontitis. The subjects with 5 mm and more of CAL at more than 30% of the sites were grouped under periodontitis cases (i.e., Group A). At the first visit, detailed demographic and medical history was recorded and oral examinations were carried out on all the subjects by an examiner. The PD, OHI-S, and CAL were recorded, along with blood pressure of the patients to rule out those with hypertension; so, they were not part of this study. Then, the patients were sent for hematological investigation for lipid profile to the Department of Biochemistry, on the subsequent day with approximately 14 h fasting (empty stomach). The lipid profile included serum cholesterol, triglyceride (TG), high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), and LDL estimation.

The standard clinical periodontal parameters (PD, bleeding on probing, plaque, and CAL) were measured using a mouth mirror, an explorer, and William's graduated periodontal probe.

probe. Measurement of fasting serum lipid profile was done in the Department of Biochemistry, Govt Dental College & Hospital Jammu using the routine enzymatic method. In order to identify the subjects with pathological values, serum LDL cholesterol greater than 160 was used as the cutoff point as per the laboratory's recommendation. The Range of Serum LDL adopted in this hospital was levels less than 100 as optimal whereas a level of 100-129 was named as near-optimal, 130-159 was border line, any value above 160 was considered high and a value after 180 was very high.

Statistical analysis

The demographic data (i.e., age, sex, etc.), the values from clinical examinations, and biochemical analysis were tabulated and consulted for statistical analysis basically to find out the correlation between the level of lipid profile value and the clinical parameters.

On statistical analysis, the means of the different variables of clinical and biochemical values were tested using the Student's t-test and the association between the variables was examined using Pearson correlation coefficient. The level of significance was set up at 5% ($P < 0.05$). The software used for this analysis was Statistical Package For the Social Sciences (SPSS) version 11.0.

Out of 60 individuals selected for the study, 30 were males and 60 were females. It was noticed that there was a slight increase in the cholesterol level in males when compared to females. The mean age of the participants in this study group was 38 ± 1.2 years. There was a direct positive correlation between the increase in age and cholesterol levels.

Grouping of the subjects was based on their mean clinical attachment level, mean PD, and mean oral hygiene index. The patients with periodontitis (Group A) had a mean CAL of 5.28 mm, PD of 4.9 mm, and mean oral hygiene index 2.98, respectively, [Table 1]. It has been observed that the patients of Group A (with periodontitis) had a greater number of elderly individuals when compared to those of Group B (without periodontitis), with a mean age of 39.14 years.

It has also been observed that PD and CAL were significantly different between Group A and Group B but the OHI-S was not statistically significant when compared [Figure 1]. [Table 1] depicts a direct positive association of total cholesterol and LDL with age of the patients with periodontitis (Group A) when compared to those without periodontitis (Group B).

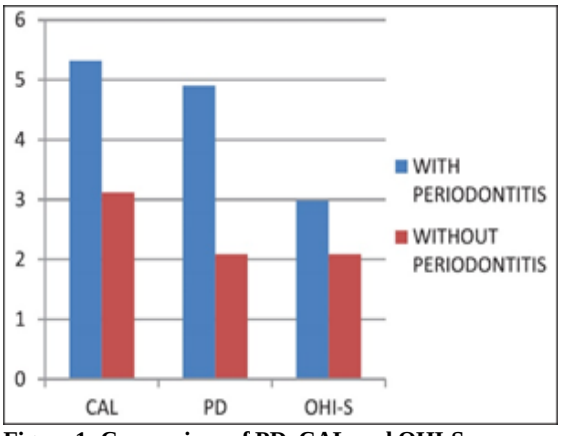

Figure 1: Comparison of PD, CAL and OHI-S.

Table 1: Comparison of PD, CAL and OHI-S.

Variables	Group	*n*	Mean	Mean LDL level	*P* value
Age (years)	Periodontitis	30	39.4	6.10	0.184
	Without periodontitis	30	34.4	4.22	0.97
CAL (mm)	Periodontitis	30	5.32	173.2	0.006
	Without periodontitis	30	3.12	134.3	1.088
PD	Periodontitis	30	4.98	173.2	0.002
	Without periodontitis	30	2.09	134.3	2.43
OHI-S	Periodontitis	30	2.13	173.2	1.32
	Without periodontitis	30	2.21	134.3	1.33

BMI: The average BMI calculated during the initial screening was 24.9 less for all the subjects that was within the normal range as per the obesity criteria of WHO (2000). The formula that was used to calculate the BMI was BMI = weight/height 2 (kg/m 2).

[Table 2] clearly depicts that the lipid profile consists of serum cholesterol, TG, LDL, HDL, and VLDL for all the subjects with and without periodontitis. It was observed that increased cholesterol levels were found in males when compared to females. It was also noticed that the age of the participants were directly proportional to their lipid profile. There was a marked increase in LDL and cholesterol levels in the subjects with periodontitis, indicating direct positive correlation between increased cholesterol and periodontitis [Figure 2]. In contrast to this, no difference was observed in the VLDL, HDL, and TG levels between the groups [Table 2].

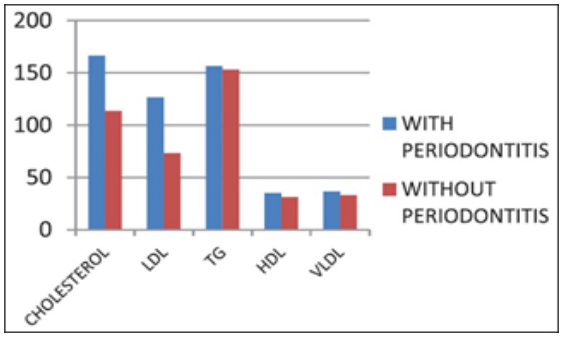

Figure 2: Serum Lipid levels.

Table 2: Serum Lipid levels.

Variables	Group	*n*	Mean	Standard deviation	*P* value
Cholesterol	Periodontitis	30	166.43	6.10	–
	Without periodontitis	30	113.67	4.22	–
LDL	Periodontitis	30	126.62	34.96	<0.001
	Without periodontitis	30	73.41	30.78	–
Total cholesterol	Periodontitis	30	173.32	39.14	<0.001
	Without periodontitis	30	116.87	32.16	–
HDL	Periodontitis	30	35.18	4.87	–
	Without periodontitis	30	31.34	5.23	–
VLDL	Periodontitis	30	36.55	9.14	–
	Without periodontitis	30	33.23	6.45	–

The current thought process of periodontal medicine lays emphasis on the effect of a constant low-grade infection as that of chronic periodontitis on the various systemic disorders involving CVDs, endocrinal disorders, respiratory problems, and adverse pregnancy outcome. This is only possible due to the continuous release of inflammatory markers into systemic circulation.

A cross-sectional, randomized, case-control study was undertaken within a study group of 60 subjects for better understanding the association between periodontal disease and serum lipid levels. This study could reconfirm the existing fact that periodontal disease was commonly prevalent in elderly age groups and also the coexistence of a direct positive relationship between the BMI and hyperlipidemia status. The specification of identification of the periodontitis group was exclusively based only on CAL. The exclusion criteria eliminated the inclusion of obese patients in our ongoing study who were prone to develop hyperlipidemia status, thereby reducing the bias.

RESULTS

The results of the present study demonstrated a positive correlation between PD and clinical attachment level with total cholesterol and LDL [Figure 1]. This shows that with increasing PD and CAL, the values of total cholesterol and LDL increased. This is in accordance with the study conducted by Katz et al. who hypothesized that there is a strong positive statistical association between the existence of periodontal pockets and plasma lipid levels.

DISCUSSION & CONCLUSION

Cardiovascular diseases (CVDs) are common all over the world, and atherosclerosis of coronary arteries is considered to be the leading cause of premature death among men.[7] There is an increasing concern about the blood lipid levels as a risk factor for the development of coronary heart disease. Literature contains various findings about possible relationship between blood serum lipids and periodontal conditions.[8-10] Each of these studies has their own specifications such as definition of periodontal disease, assay for serum lipid levels, study design and number of cases.

Others have shown lack of this relation.[11-13] The statistical analysis of the findings and the evaluation of the obtained results suggest that periodontitis may trigger alterations in lipid levels by possibly eliciting an increased systemic inflammatory response. The data of the present study indicate, correlation between periodontitis and increased values of total cholesterol and LDL.

Periodontitis is a chronic, inflammatory, destructive disease that affects the supporting tissues of the teeth and is often associated with enhanced concentrations of proatherogenic plasma lipids, i.e. TC and LDL.[8,14] The outcomes of our study support this overall conclusion. But most of the above mentioned studies were unable to infer a positive correlation with all the parameters of lipid profile (i.e. TC, HDL, LDL, VLDL and TG) to arrive at a definitive conclusion on the association between periodontitis and increased lipid profile.

Loesche W et al,[8] determined a significant association between periodontal conditions and the concentration of triglycerides in blood. Krause S et al,[15] stated that hyperlipidemia causes hyperactivity of white blood corpuscles. It was determined that hyperactivity of white blood cells (e.g. increased production of oxygen radicals) may be associated with the development of periodontitis in adults.[16]

Cutler CW et al, in their article, stated that there exists a close relationship between damage to the periodontium, increased concentration of lipids in blood, and the presence of Porphyromonas gingivalis antibodies.

Although the studied sample was small, this study showed that higher triglyceride levels might modulate the production of IL-Ib polymorphonuclear leukocytes, stimulated by P. gingivalis. Morrison, HI et al,[17] in their article, mentioned total blood cholesterol level, C-reactive protein, and fibrinogen as possible intermediate factors that associate periodontitis with increased risk for CVDs. However, these findings do not allow for the determination of causality, i.e. whether periodontal diseases may increase blood lipid concentration, or hyperlipidemia exists as the same risk factor for periodontal diseases and CVDs.6 One possible reason to explain the differences between the results of this study and previous studies, is the difference in how periodontitis is defined.

A possible explanation for the association between hyperlipidemia and periodontal infection was postulated by Noack et al,[18] by assessing neutrophil respiratory burst by whole blood chemiluminescence and they found significant increases in both chemiluminescence and pocket depth on a group of patients with hyperlipidemia. They suggested that the association of hyperlipidemia with periodontitis could be due to the dysfunction of polymorphonuclear leukocytes.

Larger studies and clinical intervention trials are necessary to better define the periodontitis subjects in whom local infection causes significant systemic inflammation, and whether these findings are true or are confounded by other important factors like nutrition, socioeconomic status, or age.

Losche et al., Katz et al., and Uchiumi et al. have found a positive correlation between periodontitis and increased serum lipid profile 7,8 that is parallel to our ongoing study. However, majority of the studies were unable to demonstrate a positive correlation between periodontal disease and all the parameters of lipid profile, i.e., total cholesterol, LDL, HDL, TG, and VLDL.

Recent studies have demonstrated that lipid metabolism may be altered by chronic local and acute systemic infections that are involved in the plasma concentrations of unregulated cytokines and hormones. The main features of this catabolic state are lipid oxidation and elevated free fatty acids, TGs, and LDL cholesterol.[11]

Studies on humans and animals have shown a number of cytokines such as tumor necrosis factor alpha (TNFα) and interleukin-1 beta (IL-1β) are produced in response to systemic gram-negative lipopolysaccharide exposure. It is believed that these cytokines exert effects on lipid metabolism by influencing the production of other cytokines, altering hemodynamics/amino acid utilization of the various tissues involved in lipid metabolism.[18]

Hyperlipidemia is known to cause hyperactivity of white blood cells with increased production of oxygen radicals, which was shown to be frequently associated with progressive periodontitis in adults.[15] However, the validity of evidence-based bidirectional relationship between hyperlipidemia and periodontitis is not yet confirmed and requires further research and evaluation.

The shortcoming of this study is the unicentric, limited population in which the current lifestyle and factors such as diet and physical activities were not taken into consideration that could have altered the outcome of this study. Multiple multicentric studies, including larger sample sizes with associated environmental factors, can be undertaken to combat these limitations and bring out the actual correlation. Interventional studies for reducing the serum lipid profile in cases of periodontitis with hyperlipidemia will help us to emphasize a direct correlation between chronic periodontitis and hyperlipidemia.

The impact of periodontitis, being the most common oral disease burden, on the systemic health must be well understood and necessary steps should be taken to prevent and cure the primary periodontal disease. This ongoing study has enlightened a positive correlation between periodontitis and hyperlipidemia and therefore, strongly recommends the control of periodontal disease by the recommended surgical and nonsurgical interventions, particularly in patients with CVDs. Within the limitation of this study, it may be concluded that the control of the development of CVD may be achieved by the prevention and cure of periodontal diseases.nfirming a positive relationship between periodontitis and hyperlipidemia.

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