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Short Communication

SERUM LIPID LEVELS IN PERIODONTITIS PATIENTS: A CASE-CONTROL STUDY

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ABSTRACT

Objective: We aimed to evaluate serum lipid levels in periodontitis patients and compared with healthy subjects.

Methods: A case-control study was conducted among 30 participants in the age group of 35-60 y, including 15 subjects in case group with community periodontal index (CPI) score of ≥ 2 and 15 subjects in the control group with a CPI score of ≤ 1 based on their periodontal status. Fasting blood samples were taken for measurement of lipid profiles, including total cholesterol, triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) using an enzymatic method. Odds ratio and independent t-test at *p=0.05 significance level were calculated.

Results: Periodontitis patients were 4.1 times at risk to have high triglyceride, 1.3 times at risk to have high total cholesterol and 0.3 times at risk to have higher HDL. Independent t-test showed higher total cholesterol/HDL ratio (*p=0.033) and lowered HDL levels (*p=0.027) in periodontitis patients.

Conclusion: Periodontitis group has significantly higher total cholesterol/high-density lipoprotein ratio and lower high-density lipoprotein levels when compared to the healthy group.

Keywords: Periodontitis, Serum lipid levels, Cardiovascular disease, Total cholesterol, High-density lipoprotein, Low-density lipoprotein, Very lowdensity lipoprotein, Triglycerides

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Oral health is a well-known indicator of general health. The impact of oral infections like dental caries and periodontitis on systemic illness has been continuously observed over recent researches. Periodontitis is a common debilitating inflammatory disease leading to the destruction of periodontal ligaments, adjacent alveolar bone and finally loss of teeth [1, 2]. The microorganisms responsible for periodontitis releases endotoxin into the bloodstream and causes systemic or local inflammatory reactions, thereby initiating a cytokine cascade with increased levels of proinflammatory cytokines [3, 4]. In turn, these cytokines promote enhanced lipogenesis, increased lipolysis and reduced lipid clearance, thereby increasing serum lipid levels, which was proved to be one of the risk factors for coronary artery disease [4-6]. Recent literature has illustrated a positive correlation between periodontal disorders and hyperlipidemia. Sumit Tiwari et al. (2016) [2] and Deepu Mathew Panickal et al. (2016) [3] demonstrated significantly higher levels of serum lipid levels in subjects with chronic periodontitis when compared with healthy controls [7-9]. However, on the other hand, Machado et al., Hamissi et al. did not confirm any relationship between hyperlipidemia and periodontitis [11, 12]. Therefore, we have aimed this study to assess serum lipid levels in periodontitis patients and compared with healthy subjects in order to confirm the possible causal relationship between periodontal diseases and cardiovascular diseases.

A case-control study was designed and conducted among thirty patients reported to the medical outpatient department of the Sathyabama General Hospital. The study population included patients with a minimum of 20 teeth, age group of above 35-60 y without systemic illness and community periodontal index (CPI) score \geq 2. Patient with systemic conditions like diabetes, hyperlipidemia, cardiovascular disorders, conditions related to elevated levels, respectively, patients with a history of dental treatment in the past six months, including oral prophylaxis, patients with aggressive periodontitis and patients who have undergone any surgical therapy in the past six months were excluded.

The study protocol had been reviewed and approved by the institutional human ethical committee (IHEC) of the Sathyabama

Institution. A written informed consent was obtained from participants who fulfilled the eligibility criteria and willing to participate in the study. Prior to the commencement of the study, inter-examiner calibration was carried out to ensure uniform interpretations and consistent examination. The kappa value was found to be 0.92.

Thirty subjects were randomly selected from the medical outpatient department by the examiner and the periodontitis patients were screened by the community periodontal index of treatment needs (CPITN) using a community periodontal probe with code 2 and above [10]. On the basis of history and clinical findings, 15 case group subjects with community periodontal index (CPI) score ≥ 2 and 15 control group subjects with a CPI score ≤ 1 were selected.

Fasting blood samples were collected to estimate the lipid profile, including total cholesterol, triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) using enzymatic methods [5].

The recorded data were compiled and entered into Microsoft Excel 2010 spreadsheet and then exported to the data editor of statistical package for social sciences (SPSS) software to version 16 for statistical analysis. Descriptive statistics were carried out to check frequency distribution. The independent t-test was performed to compare means of serum lipid levels between periodontitis patients and healthy persons. The odds ratio was done to check the strength of association between serum lipid levels and periodontitis. To compare the mean of variables, independent t-test was set at *p<0.05 significance level.

Table 1 illustrated serum total cholesterol level was 53.33% higher in the diseased group when compared to 46.66% in the healthy group, VLDL was 66.66% higher in the diseased group compared to 13.33% in the control group and TG was 60% higher in the case group when compared with healthy individuals.

On the other hand, periodontitis patient had lower HDL when compared to 6.66% in healthy individuals. LDL level was 13.33% in both case and control groups.

rs Case	Control	Odds ratio	t-test	p-value
N (%)	N (%)			
sterol 8 (53.33)	7 (46.66)	1.3	-0.7695	0.448
0	1 (6.66)	0.3	2.3277	*0.027
2 (13.33)	2 (13.33)	1	0.8772	0.388
10 (66.66)	2 (13.33)	13	-0.9011	0.375
9 (60)	4 (26.66)	4.1	-0.8181	0.420
sterol/HDL ratio			-2.2440	*0.033
- ;;	rs Case N (%) esterol 8 (53.33) 0 2 (13.33) 10 (66.66) 9 (60) esterol/HDL ratio	Case Control N (%) N (%) *sterol 8 (53.33) 7 (46.66) 0 1 (6.66) 2 (13.33) 10 (66.66) 2 (13.33) 10 (66.66) 9 (60) 4 (26.66)	Case Control Odds ratio N (%) N (%) *sterol 8 (53.33) 7 (46.66) 1.3 0 1 (6.66) 0.3 2 (13.33) 1 10 (66.66) 2 (13.33) 13 9 (60) 4 (26.66) 4.1	rs Case Control Odds ratio F-test N (%) N (%)

Table 1: Prevalence of serum lipid levels in diseased group and control group along with odds ratio and t-test for equality of means

Abbreviations – High-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), triglycerides (TG). 1*Significant difference (p<0.05).

Odds ratio demonstrated that periodontitis patients were 4.1 times at risk to have high triglyceride levels, 13 times at risk to have VLDL, 1.3 times at risk to have high total cholesterol and 0.3 times to have high HDL.

From the independent t-test, the group means are significantly different among periodontitis patient and normal persons. We observed that *p=0.027 for HDL level and *p=0.033 for total cholesterol/HDL ratio concluding periodontitis patients have significantly lower HDL and higher total cholesterol/HDL ratio when compared to normal persons indicating a higher risk for cardiovascular disease.

However, the association between altered lipid profile and periodontitis has been investigated in several studies with inconsistent results and findings. Fewer studies [2, 16] reported decreased HDL concentrations, suggesting a positive association between chronic periodontitis and cardiovascular disease (CVD), also there were studies without a significant relationship between serum lipid levels and chronic periodontitis [11, 12].

The underlying mechanism relating periodontitis to increased serum lipid levels may be lipopolysaccharides (LPS) of dental plaque diffuse into the systemic circulation and they elicit systemic lipopolysaccharides specific antibody response resulting in lipid metabolism disturbance and a hypercoagulable state through elevation of circulating cytokines. Monocytes derived cytokines such as tumour necrosis factor (TNF- α), interleukins (IL-1, 6, 8) have powerful effects on hepatic protein synthesis, tissue catabolism, and lipid metabolism. Both TNF- α and IL-1 alters lipid metabolism by inhibiting synthesis of lipoprotein lipase. Bacterial toxins can also induce changes in cholesterol concentrations; leading to reduced high-density lipoprotein and increased low-density lipoprotein [13].

Decreased HDL level has been considered as a risk factor for coronary heart disease due to anti-inflammatory and anti-atherogenic properties [13, 14]. Total cholesterol/HDL ratio, known as the atherogenic or Castelli index was considered as an important component and an indicator of vascular risk [15], the predictive value of which was greater than the isolated parameters. An increase in total cholesterol concentration was proved as an atherogenic lipid marker, whereas reduced HDL cholesterol concentration was correlated with numerous risk factors, including the components of the metabolic syndrome, and probably involves independent risk [17, 18].

Individuals with high total cholesterol/HDL ratio or LDL/HDL cholesterol ratio have greater cardiovascular risk owing to the imbalance between the cholesterol carried by atherogenic and protective lipoproteins. This may be due to an increase in the atherogenic component contained in the numerator, a decrease in the anti-atherosclerotic trait of the denominator, or both confirming significant associations with acute coronary syndrome [19].

The drawback of this study included more elaborate study design and lesser sample size to emphasize a direct correlation between periodontitis and abnormal serum lipid levels. Within the limitation of this study, we observed a positive link between periodontitis and elevated serum lipid levels among the patients reported at the medical outpatient department in our institution, suggesting prevention and treatment of periodontal disease could eventually hinder the onset and progress of cardiovascular diseases among periodontitis patients.

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AUTHORS CONTRIBUTIONS

Concept and design of the study by Dr MA. Adil Ahmed. Data acquisition by Dr Judyangel D and Rajathi R. Statistical analysis and data interpretation by Dr Judyangel D. Manuscript drafting by Rajathi R, Rafiya S, Pooja Kumari and Pragadeeswari D. Manuscript editing and review by Dr Judyangel D. All authors took part in the conduction of study.

CONFLICT OF INTERESTS

The authors have no conflict of interest to declare.

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