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Original Research

Evaluating Salivary Lipid Profile in Patients with and without Periodontal Disease – A Cross Sectional Study

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ABSTRACT:
Introduction: Periodontitis is an inflammatory condition triggered by an imbalance in the periodontal bacterial community, leading to damage of the tooth-supporting structures. Over recent decades, numerous studies have highlighted a significant link between periodontitis and coronary heart disease. Lipids gets adhered in the intima layer, where it is oxidized, and leads to the beginning of coronary heart disease. Saliva, being an ultrafiltrate of plasma, serves as a valuable non-invasive medium for diagnostic purposes. The present study aimed to assess salivary lipid profile parameters such as Triglycerides (TGL), High-Density Lipoproteins (HDL), Low-Density Lipoproteins (LDL), Very Low-Density Lipoproteins (VLDL), and Total Cholesterol in individuals with and without periodontal disease. **Methods**: This study included 30 subjects. Unstimulated salivary samples were collected into sterile containers from 15 patients with chronic periodontitis and 15 individuals with healthy periodontium. Lipid profile levels were estimated using calorimetric method. **Results:** Patients with periodontal disease exhibited increased salivary levels of LDL, Total Cholesterol and decreased HDL levels by supporting the hypothesis that salivary lipid levels will be increased in patients with chronic periodontitis which may act as a precursor for atherosclerosis. **Conclusion**: Patients with chronic periodontitis have increased LDL and total cholesterol levels in saliva which could be a risk factor for developing atherosclerosis.
Keywords: Periodontitis, Saliva, Lipid, Coronary Heart Disease, Arthrosclerosis.

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INTRODUCTION

Dysbiotic microflora of periodontal pathogens are the prime causative agents of periodontitis, an inflammatory, chronic, multifactorial disease that results in the degeneration of the tissues that support teeth. [1] It is characterized by the development of pathological periodontal pockets together with the obliteration of both the alveolar bone and the periodontal ligament fibers. [2]

Salivary lipids may contribute to the initial mineralization process of dental plaque and calculus. The oral cavity serves as a valuable reflection of both systemic and oral health. Saliva, an ultrafiltrate of plasma, contains secreted lipids and offers several advantages over serum, including simple, non-invasive collection, easy storage, and suitability for repeated sampling. It is also one of the most cost-effective approaches for large-scale population screening. [4 -7]

Various studies in the past few decades have shown a high correlation between periodontitis and coronary heart disease, including a number of disorders such as arthrosclerosis and myocardial infarction. [8] The present study was aimed at evaluating the salivary lipid profile in patients with and without periodontal disease.

MATERIALS AND METHODS

The participants for the study were selected from the Outpatient section, Department of Oral Medicine and Radiology. The institutional Review Board approved the study protocol and the clearance reference number is (RDCH/PRL/IRB/D- 3908/2023). The study was clearly explained to the patient and informed consent was obtained. The cross-sectional study included 30 subjects. 15 patients with periodontal disease and 15 subjects with healthy periodontium.

Inclusion and Exclusion criteria

Patients with minimum of 20 permanent teeth, Gingival Index between 1-2 (Loe and Silness gingival index), probing depth > 5mm were included as case group and participants with Gingival index of less than 0.5 were taken as control group.

Patients suffering from conditions requiring antibiotics prophylaxis prior to dental procedure, diabetes, Use of immunosuppressive and anti-inflammatory drugs throughout the last three months prior to the study, pregnant and lactating mothers were excluded from the study.

Sample collection

Saliva samples were obtained in the morning between 8:00 AM and 11:00 AM to minimize diurnal variations. Participants were instructed to avoid toothbrushing for at least 45 minutes prior to collection and to refrain from undergoing any dental procedures within the preceding 24 hours. After being seated comfortably, each participant rinsed their mouth with water to eliminate food particles. They were then asked to lean forward and expectorate saliva into a sterile container until 5 ml of whole, unstimulated saliva was collected. The samples were centrifuged at 3000 rpm for 10 minutes to remove cellular debris and reduce turbidity. The resulting supernatant was transferred into a fresh test tube for further laboratory analysis.

Procedure

The collected samples were analyzed for salivary lipids calorimetrically using Beer's Lamberts law. Various reagents are used for the analysis such as End point assay - GPO PAP assay kit for Triglyceride, Enzymatic end point assay – CHOD PAP assay kit for total cholesterol, Accelerator selective detergent assay for High density lipoprotein. VLDL and LDL were calculated through the below mentioned formula. VLDL calculated using the formula triglyceride/5 and LDL was calculated through the formula VLDL – High density lipoprotein(mg/dl).

After the biochemical analysis, the values were read with optical density using calorimeter.

Data collected were subject to statistical analysis through SPSS Version 22 Software, IBM Statistics, USA. Inter group analysis was done using t–Test. Overall inter group comparison done using ANOVA. Based on Post–hoc analysis using Bonferroni test after adjusting for multiple comparisons

RESULTS

This cross-sectional study aimed to assess salivary lipid profile levels in individuals diagnosed with generalized chronic periodontitis. Clinical parameters were recorded at baseline, and saliva samples were collected on the same day. The samples were analyzed using a colorimetric method to determine total cholesterol, triglycerides, HDL, LDL, and VLDL concentrations. The results were compiled, and statistical analysis was performed using SPSS (Statistical Package for the Social Sciences), Version 22, IBM, USA.

[Table 1] shows comparison of the mean values of salivary total cholesterol, triglycerides, HDL, LDL and VLDL levels of control group with values of study group. The difference in salivary total cholesterol between control group (4.09 ± 2.51) and study group (38.67 ± 13.72) was found to be statistically significant with p-value of <.001*. The difference in salivary triglycerides levels between the control group (10.63 ± 4.35) and study group (14.12 ± 14.82) was found to be statistically non-significant with p-value of 0.389. The mean difference in salivary HDL between control group (31.66 ± 10.54) and study group (17.88 ± 7.67) was found to be statistically significant with p-value of <.001*. The mean difference in salivary LDL between control group (1.83 ± 0.88) and study group (17.96 ± 13.74) was found to be statistically significant with p-value of <.001*. The mean difference in salivary VLDL between control group (2.12 ± 0.87) and test group (2.82 ± 2.96) was found to be statistically non-significant with p-value of 0.389.

[Figure 1] depicts the difference among lipid profiles between case and control group. It illustrates that Total cholesterol and low density lipoprotein was found to be increased in case compared to control group. Likewise, high density lipoprotein was increased among control group.

[Table 2] shows over all inter-group comparison of the mean values of total cholesterol, triglycerides, HDL, LDL and VLDL levels between the control group with study group based on ANOVA. The result values were found to be statistically significant stating that there is noteworthy difference in salivary lipid profile of patients with periodontitis except triglycerides and VLDL.

[Figure 2] compares the overall lipid profile level among case and control group. The graph renders that total cholesterol was high among case group and High density lipoprotein were increased among control group.

Table 1: Salivary lipid levels in control and case group

	Groups	N	Mean	Standard Deviation	p-value
Total Cholesterol	Control	15	4.09	2.51	< 0.001*
	Case	15	38.67	13.72	
Triglycerides	Control	15	10.63	4.35	0.389
	Case	15	14.12	14.82	
High Density Lipoproteins	Control	15	31.66	10.54	< 0.001*
	Case	15	17.88	7.67	
Low Density Lipoproteins	Control	15	1.83	0.88	< 0.001*
	Case	15	17.96	13.74	
Very Low Density Lipoproteins	Control	15	2.12	0.87	0.389
	Case	15	2.82	2.96	
p-value based on Independent-*t*-Test * = Statistically Significant (p < 0.05)					

Table 2: Overall Inter group comparison of salivary lipid levels between control and case group

		N	Mean	Standard Deviation	p-value
Control	Total Cholesterol	15	4.09	2.51	< 0.001*
	Triglycerides	15	10.63	4.35	
	High Density Lipoproteins	15	31.66	10.54	
	Low Density Lipoproteins	15	1.83	.88	
	Very Low Density Lipoproteins	15	2.12	.87	
Case	Total Cholesterol	15	38.67	13.72	< 0.001*
	Triglycerides	15	14.12	14.82	
	High Density Lipoproteins	15	17.88	7.67	
	Low Density Lipoproteins	15	17.96	13.74	
	Very Low Density Lipoproteins	15	2.82	2.96	
p-value based on Analysis of Variance (ANOVA) * = Statistically Significant (p < 0.05)					

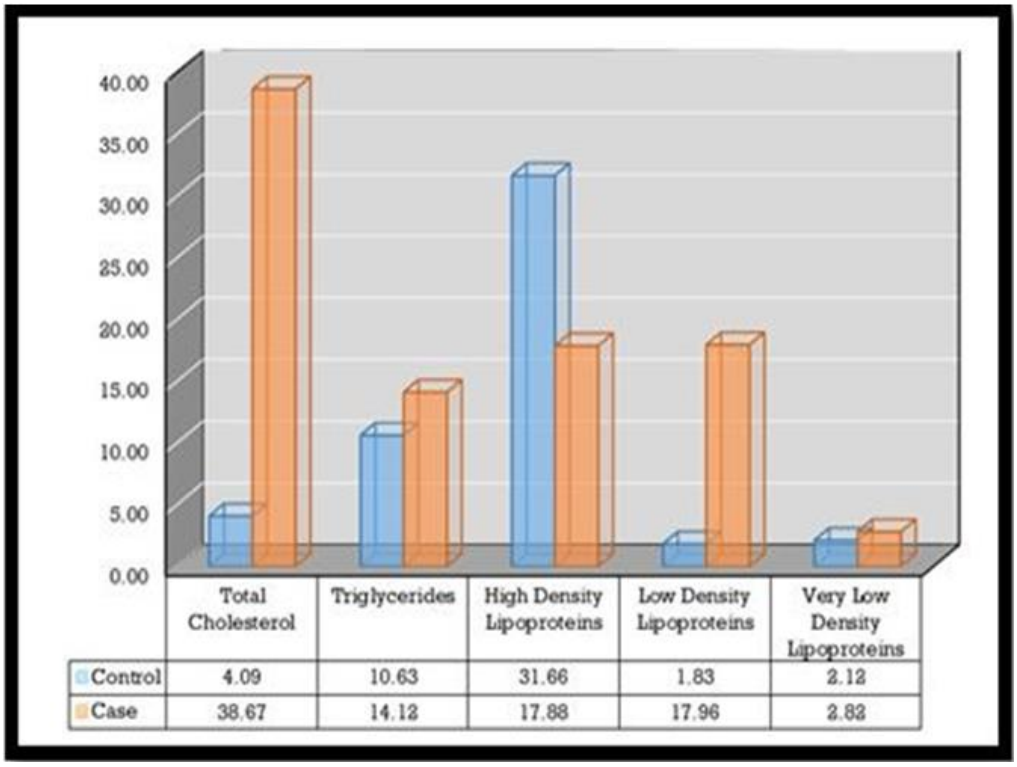

FIGURE 1: Depicts the difference among lipid profile between case and control group

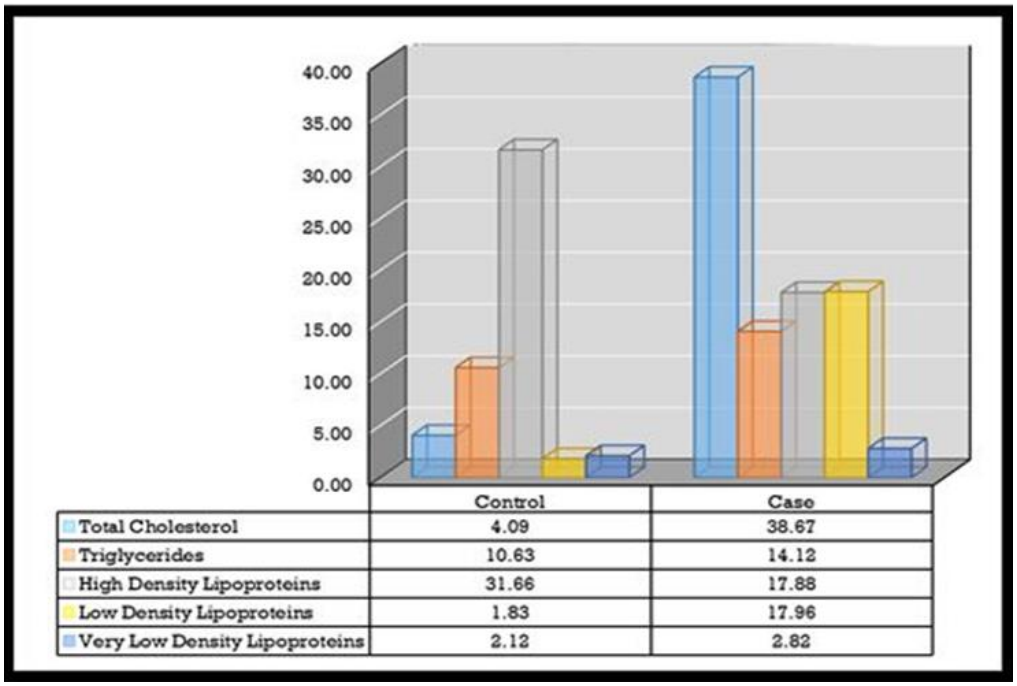

FIGURE 2: Overall comparison of lipid profile among case and control group

DISCUSSION

The etiology of periodontitis involves inflammatory and immune-mediated mechanisms, wherein virulent periodontal pathogens disrupt the host response.[9] These pathogens elicit a persistent bacterial challenge, triggering the release of pro-inflammatory cytokines such as interleukins (IL-1, IL-6, IL-12) and tumor necrosis factor-alpha (TNF-α). This stimulates acute-phase reactants, accompanied by vascular changes. In atherosclerosis initiation, low-density lipoprotein (LDL) accumulates within the intimal layer and undergoes oxidation. Consequently, endothelial cells in the vicinity exhibit increased expression of adhesion molecules, including ICAM-1, VCAM-1, and selectins, promoting the diapedesis of circulating monocytes and lymphocytes into the inflamed intima, where they adhere to these molecules. [10]

Periodontal infection may influence the initiation or progression of atherosclerosis and coronary heart disease via a variety of mechanisms. Damage to vascular endothelium can occur because of the presence of intravascular microorganisms and their products. [11]

Following adhesion to the endothelium, monocytes traverse the endothelial layer and migrate into the arterial intima, where they engulf oxidized low-density lipoprotein (LDL) and transform into lipid-laden foam cells a hallmark of atheromatous plaque.[12]

Progressive plaque development and intimal thickening narrow the vascular lumen, significantly reducing blood flow. Rupture of an atheromatous plaque exposes arterial collagen and tissue factor from monocytes and macrophages, initiating platelet activation and the coagulation cascade. Subsequent platelet and fibrin aggregation form a thrombus, which may obstruct the vessel and precipitate ischemic events such as angina or myocardial infarction. [13]

In a recent study, the total serum cholesterol levels, low-density lipoproteins (LDL), triglycerides, very-low density lipoproteins (VLDL), oxidized LDL and phospholipase A2 are elevated in periodontitis. [14] Whereas, High-density lipoprotein (HDL) levels were reduced in patients with periodontitis compared with healthy individuals and the levels were reversed after periodontal therapy. [15]

There has been an increased LDL value and decreased HDL concentration in patients with periodontitis compared to individuals with healthy periodontium. [3] Another research in the literature has demonstrated a correlation of LDL levels with clinical signs of inflammation and periodontal tissue destruction in a population. [16]

The results of our present study revealed that in periodontal patients there was a significant increase in Total cholesterol, low density lipoprotein level and in healthy patients there was a significant increase in High density lipoprotein levels by supporting the hypothesis that salivary lipid levels will be increased in patients with chronic periodontitis which may act as a precursor for atherosclerosis.

CONCLUSION

This study has showed that there is a relationship between salivary lipid profile and chronic periodontitis which could be a risk factor for atherosclerosis and coronary heart disease.

Salivary diagnostics holds a tremendous promise in future for diagnosis. It could evolve as an imminent approach to evaluate and investigate various health conditions as a chair side non-invasive diagnostic

tool. In future studies large sample size could be used and therapeutic outcome could be evaluated.

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