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A Comparison of Blood Glucose Levels and Dyslipidemia in Patients with Chronic Periodontitis and Healthy Controls

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Abstract

***Background**: Numerous research studies have indicated a potential connection between periodontal disease and an increased susceptibility to cardiovascular conditions. This study aimed to assess fasting plasma lipid profiles and blood glucose levels in patients with chronic periodontitis. **Methods**: 25 participants diagnosed with chronic periodontitis were divided into mild, moderate, and severe groups based on clinical attachment loss (CAL). Additionally, 25 healthy controls matched for age and gender were included. Venous blood samples were collected after an overnight fast and analyzed for serum triglycerides, total cholesterol, LDL, HDL, and blood glucose in both study and control groups. Comprehensive periodontal measurements including probing pocket depth and CAL were taken at six sites per tooth using William's periodontal probe. **Results:** Blood glucose and triglyceride levels were notably higher in individuals with periodontal disease (P<0.05) compared to controls. When aggregating the mild, moderate, and severe periodontitis patients into a single group and comparing them with the control group, statistically significant differences were observed for Blood glucose and triglycerides. Additionally, blood glucose levels were significantly elevated in patients compared to the control group. **Conclusion:** In the present study, it was found that there is a deranged lipid profile in patients with periodontal diseases. Severity and poor control of periodontal disease might likely affect the level of lipids and glucose in blood which may increase the risk for CVD.*

***Keywords**: Chronic Periodontitis, Lipid Profile, Dyslipidemia, Cardiovascular Disease*

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Introduction

Periodontal diseases rank among the most prevalent and extensively distributed conditions globally, primarily contributing to tooth-related health issues and even tooth loss. [1] These diseases stem from a chronic immuno-inflammatory response triggered by bacteria and their byproducts, ultimately leading to the destruction of periodontal tissues. The severity and frequency of periodontal disease tend to be higher in individuals with prolonged disease duration and those experiencing systemic complications. [2] However, it can also manifest in patients with well-managed systemic conditions. There is a growing body of evidence suggesting that periodontal infections could potentially impact systemic health, not only in individuals who are medically compromised or immunodeficient, but also in otherwise healthy individuals. [3-6] Infections have been shown to influence lipid metabolism in a manner that could promote the development of atherosclerosis. Atherosclerosis initiates due to the accumulation of lipids in specific areas. Both periodontitis and atherosclerosis exhibit intricate causative factors, including genetic and gender predispositions, and likely share numerous risk factors. [7] Diabetes mellitus, arising from dysfunctions in insulin-dependent

glucose and lipid metabolism, manifests with classic symptoms such as increased thirst, excessive urination, and heightened appetite, often accompanied by persistent fatigue and weight loss. Complications associated with diabetes encompass retinopathy, nephropathy, neuropathy, and cardiovascular disease. Notably, periodontitis is now recognized as the sixth complication of diabetes. [8] Periodontitis is caused by a small group of gram-negative bacteria that form biofilms on root surfaces. Substances like Lipopolysaccharide (LPS) and other microbial components enter gingival tissues, triggering and sustaining immune-inflammatory responses. This process leads to the release of high levels of proinflammatory cytokines, which further stimulate the production of matrix metalloproteinases responsible for damaging gingival connective tissues, cementum, and the periodontal ligament. [9] Additionally, prostaglandins, which mediate alveolar bone degradation, are produced. Periodontitis may heighten vulnerability to systemic diseases through various mechanisms. LPS and proinflammatory cytokines from inflamed periodontal tissues can enter the bloodstream in significant quantities, contributing to increased serum lipid levels and insulin resistance. Numerous studies propose that chronic low-level systemic exposure to LPS can induce widespread alterations in lipid and glucose metabolism. [10] Therefore, long-standing, or chronic periodontitis-induced insulin resistance and hyperlipidemia could serve as precursors to active diabetes and cardiovascular diseases due to the sustained elevation of proinflammatory cytokines. Furthermore, common risk factors such as smoking, male gender, race/ethnicity, stress, genetics, and aging are shared between periodontitis and certain systemic diseases like cardiovascular disease. [11] Substantial evidence suggests that chronic periodontitis, particularly severe cases in early life, substantially heightens the risk of developing systemic diseases. This study aims to illustrate how untreated periodontitis impacts overall health by disrupting lipid and glucose metabolism.

Materials and Methods

This cross-sectional study was conducted in the Department of Dentistry, Outpatients visiting the Dental department were included in the study. Written consent was obtained from all the participants of the study after explaining the nature of the study in the vernacular language.

Inclusion Criteria:

1. Patients with periodontal diseases
2. Aged 20 and above
3. Voluntarily willing to participate in the study

Exclusion criteria

1. Patients with significant systemic diseases
2. History of periodontal treatments
3. Pregnant and lactating females
4. Not willing to participate in the study

Based on the inclusion and exclusion criteria n=25 cases with various periodontal diseases and n=25 age and sex-matched controls without any periodontal diseases were included in the study. After selecting the subjects, written informed consent. The study procedure was explained before obtaining consent. A comprehensive medical and dental history of the participants was collected. The intra-oral examination was performed using a mouth mirror and William's Periodontal Probe. Periodontal evaluation involves measuring Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL).

Probing Pocket Depth (PPD): PPD was measured from the gingival margin to the pocket base using William's Periodontal Probe. The probe was inserted into the gingival sulcus around the tooth. Six measurements were taken per tooth – three on the buccal and three on the lingual side (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, Distolingual). Probing depth per tooth was the sum of measurements per tooth divided by 6. Similarly, the mean probing depth was the sum of tooth scores divided by the total number of teeth examined. Clinical Attachment Level (CAL): CAL was measured from the cementoenamel Junction (CEJ) to the pocket base using William's Periodontal Probe. If the gingival margin aligned with the anatomic crown, the attachment level was calculated by subtracting the distance from the gingival margin to CEJ from the probing pocket depth. When the gingival margin coincided with the CEJ, attachment loss was the same as the

probing pocket depth. If the gingival margin was below the CEJ, attachment loss was greater than probing pocket depth, and the distance between CEJ and the gingival margin was added to PPD. Six measurements were taken per tooth – three on the buccal and three on the lingual side (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, Distolingual). The clinical attachment less per tooth was the sum of measurements per tooth divided by 6. Similarly, the mean attachment loss was the sum of tooth scores divided by the total number of teeth examined. Blood samples were collected for investigation in the laboratory for serum lipid profile and blood glucose estimation by a semi-automatic **Erba Chem 7 analyzer.**

Statistical analysis: All the available data was uploaded to an MS Excel spreadsheet and analyzed by SPSS version 21 in Windows format. All the categorical variables were represented as mean, standard deviations, and percentages, and categorical variables were represented by p values. The student's 't' test was used to determine the level of significance.

Results

The study comprised a total of 50 participants. Among these, 25 individuals (without periodontal disease) exhibiting a CAL (Clinical Attachment Level) of 0 mm were selected as the control group and had a healthy periodontium. The study group was divided into three categories: The remaining 25 participants were mild (CAL = 1-2mm), moderate (CAL = 2-3mm), and severe (CAL ≥ 5mm), forming the study group. The primary clinical parameter utilized for comparison and analysis between these two groups was CAL, a measure of attachment loss. Additionally, the study incorporated blood parameters such as glucose and lipid profile levels. The investigation focused on assessing the disparities in these blood parameters between the control and study groups to determine whether periodontitis could be considered a risk factor for cardiovascular disease (CVD). Out of the n=25 cases of the study group, 80% were males and 20% were females. The male to female ratio was 4:1. Similarly, in the control group 80% males were included and 20% females were included. The age range of the patients in the study group was from 25 – 55 years the mean age of the study cohort was 42.5 ± 5.5 years. The age range of the control group was 20 – 40 years the mean age was 35.5 ± 3.5 years. The difference in the age between the study group and controls was calculated by paired t-test and the p-value was 0.125 hence differences were insignificant.

Table 1 shows the comparison of the values of parameters recorded in the cases and controls of the study. The cases are the people with the disease, and the controls are the people without the disease. The parameters are blood glucose, total cholesterol, triglycerides, and HDL. The table shows that the cases had significantly higher blood glucose levels than the controls in all three severity groups (mild, moderate, and severe). The cases also had significantly higher triglyceride levels than the controls in the mild and moderate severity groups, but not in the severe severity group. There was no significant difference in total cholesterol or HDL levels between the cases and controls in any of the severity groups. The p-values in the table indicate the statistical significance of the differences between the cases and controls. A p-value of less than 0.05 is considered statistically significant. In conclusion, the table suggests that blood glucose and triglyceride levels are significantly higher in people with periodontal disease than in people without periodontal disease. However, there is no significant difference in total cholesterol or HDL levels between the two groups.

Table 1: Comparison of values of parameters recorded in the cases and controls of the study

Parameter	Study group			Control group	P values
	Mild	Moderate	Severe		
Blood Glucose (mg/dl)	88.57 ± 19.33	93.25 ± 14.68	106.87 ± 15.47	82.33 ± 12.65	0.015*
Total Cholesterol (mg/dl)	164.98 ± 25.54	167.83 ± 23.91	171.35 ± 30.25	145.38 ±15.89	0.189
Triglycerides (mg/dl)	109.82 ± 15.89	131.37 ± 17.68	191.57 ± 26.74	88.64 ± 11.27	0.011*
HDL (mg/dl)	41.35 ± 3.65	42.98 ± 2.87	43.21 ± 2.98	43.21 ± 3.51	0.128
LDL (mg/dl)	102.36 ± 10.98	131.22 ± 19.83	140.25 ± 22.83	97.58 ± 10.35	0.375

* Significant

Table 2 shows the levels of cutoff for five different parameters (glucose, total cholesterol, triglycerides, HDL-C, and LDL-C) in the study group and control group. The cutoff value is the value that separates the two groups. In the glucose parameter, 17 people in the study group

had a glucose level below 100 mg/dL, while 8 people had a glucose level above 100 mg/dL. Similarly, 21 people in the control group had a glucose level below 100 mg/dL, while 4 people had a glucose level above 100 mg/dL. The table shows that the **control group** had a higher percentage of people with levels below the cutoff value for the **total cholesterol** and **HDL-C** parameters. However, the **study group** had a higher percentage of people with levels above the cutoff value for the **glucose, triglycerides, and LDL-C** parameters.

Table 2: Shows the levels of cutoff for five different parameters

Parameter	Levels of cutoff	Study group		Control group	
		Below	Above	Below	Above
Glucose	100 (mg/dl)	8	17	21	4
Total Cholesterol	200 (mg/dl)	14	11	24	1
Triglycerides	150 (mg/dl)	13	12	25	0
HDL-C	40 (mg/dl)	19	6	0	25
LDL-C	130 (mg/dl)	7	18	22	3

Discussion

There is a hypothesis that periodontal infections may exert an impact on overall health due to several factors, including the substantial presence of disease-associated microorganisms, the persistent nature of the condition, and the local and systemic immune responses in the host. One of the suggested mechanisms proposes that long-term oral inflammation could potentially result in elevated blood cholesterol levels. Nevertheless, research in this area has yielded inconsistent findings. The continuous elevation of pro-inflammatory cytokines in chronic periodontitis has the potential to disrupt normal serum lipid levels, which, in turn, could have adverse consequences on a person's overall systemic health. [12] The periodontium has been identified as a potential reservoir of endotoxins, cytokines, and lipid mediators within the body. Administering low doses of endotoxin to rodents induces swift changes in lipid metabolism, resulting in heightened levels of triglycerides, a type of fat. Infections caused by *P. gingivalis* or other gram-negative periodontal pathogens have the potential to trigger the systemic release of IL-1 beta and TNF-alpha, which can disrupt fat metabolism, leading to persistent hypertriglyceridemia. In animal models, inducing periodontitis in rats using *P. gingivalis* serum has shown an increase in triglyceride levels. [13] This study encompassed both males and females, following a pattern seen in prior research. [14, 15] However, Cutler et al. [14] focused on individuals aged 25 to 60 years, the current study's age range was set at 35 to 70 years for both groups. Similar to earlier investigations, potential confounding factors like smoking, hypertension, and liver disease were eliminated from the study. [16] According to Katz et al., [14] smoking status displays a positive correlation with total cholesterol (TC), low-density lipoprotein (LDL), and triglyceride (TGL) levels, while being inversely linked with high-density lipoprotein (HDL) cholesterol. Smoking-induced vasoconstriction of gingival blood vessels has been suggested to facilitate the invasion of periodontal disease-causing microorganisms. [16] As a result, smokers were excluded from the present study. In this study, participants in the study group were classified into mild, moderate, and severe categories based on their Clinical Attachment Level (CAL). These groups were then compared to the control group in terms of their blood lipid and glucose levels. The objective was to investigate whether there were alterations in lipid profiles and glucose parameters associated with the increasing severity of periodontal disease. To the best of our knowledge, no previous study has explored the relationship between disease severity and lipid profiles or glucose levels in this manner. [17]

Our findings indicated that plasma triglyceride (TGL) levels and plasma glucose levels were significantly higher in individuals with mild, moderate, and severe periodontitis when compared to age- and sex-matched controls ($P < 0.05$). This result aligns with the findings of Matilla et al., [18] who also identified a significant association between triglyceride levels and dental infections. When the combined group of mild, moderate, and severe periodontitis cases was compared to the control group, total cholesterol (TC) levels showed statistical significance. This outcome is consistent with the observations of Cutler et al., [14] who found a significant association between the presence of periodontitis and increased TG levels. There is a suggestion that

proinflammatory cytokines such as IL-1beta and TNF-alpha, which are produced as part of the body's systemic response to periodontal infection, may contribute to insulin resistance and subsequently result in poor glycemic control among individuals with periodontitis. To conclusively determine whether periodontitis indeed triggers insulin resistance and a pre-diabetic condition, future studies will need to assess pre-diabetes rigorously and consistently. The intricate relationship between diabetes mellitus and periodontal disease has been extensively explored in previous research and has sparked considerable debate. However, only a limited number of studies have delved into the interplay between glucose levels in periodontitis patients and vice versa, especially in individuals who do not have diabetes. [19] The physical and chemical properties of cell membranes are largely determined by the type of fatty acids they contain. For example, gamma linoleic acid is a type of unsaturated fatty acid that can make cell membranes more fluid. Changes in the types of fatty acids in the bloodstream or tissues can therefore affect how molecules signal to each other. Lipids can also interact directly with cells, such as myeloid cells. Myeloid cells are a type of immune cell that plays a role in inflammation. When lipids interact with myeloid cells, they can alter the expression of genes for pro-inflammatory cytokines and essential growth factors. Pro-inflammatory cytokines are proteins that promote inflammation, while essential growth factors are proteins that promote cell growth and repair. [20] It is possible that abnormalities in macrophage function, caused by elevations in serum lipids, can alter the secretion of cytokines necessary for normal wound healing. This could lead to chronic inflammation and impaired wound healing. Therefore, it is conceivable that periodontitis, a chronic inflammatory disease of the gums, could be a potential risk factor for coronary artery disease (CAD). This is because periodontitis can lead to elevations in serum lipids and pro-inflammatory cytokine production.

Conclusion

Within the limitations of the present study, it was found that there is a deranged lipid profile in patients with periodontal diseases. Severity and poor control of periodontal disease might likely affect the level of lipids and glucose in blood which may increase the risk for CVD. Therefore, when periodontitis is newly diagnosed in patients with atherosclerotic CVD, periodontists, and physicians managing patients with CVD should closely collaborate to optimize CVD risk reduction and periodontal care.

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***Ethical Clearance*: Obtained**

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