THE PLASMA LIPID LEVEL IN PERIODONTAL HEALTH AND DISEASE- A CASE-CONTROL STUDY

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ABSTRACT

BACKGROUND
Periodontitis is a chronic inflammatory disease of the supporting structures of the teeth resulting from the dental plaque biofilm attached to the teeth. Recent studies have shown that oral disease may affect the systemic health. The aim of the present study was to estimate and compare fasting plasma lipids in case and control groups, and to establish its relationship with periodontitis.

MATERIALS AND METHODS
Sixty patients were selected within the age group of 40-50 years, which included both the sexes. Thirty patients with probing depth ≤3 mm were taken as Control group and thirty patients with probing depth ≥4 mm were taken as the Case group. 90 mL of sample blood were collected 12 hours after fasting and sent for laboratory investigations for lipid profile. The data collected were statistically analysed.

RESULTS
In this present study, the mean total Cholesterol, triglycerides, LDL Cholesterol and VLDL Cholesterol levels were elevated in the Case group than the Control group and they were statistically significant. Although the mean HDL Cholesterol level was elevated in the Case group than the Control group it was not statistically significant.

CONCLUSION
The present study shows a significant relationship between periodontitis and hyperlipidaemia. The present study also supports the hypothesis that periodontal disease is a risk factor for cardiovascular disease.

KEYWORDS
Cholesterol, Risk Factor, Periodontitis.


BACKGROUND
Periodontitis, a periodontal disease, results from interaction between the immune system and oral bacteria that may promote oxidative stress and initiate an inflammatory cascade inducing the destruction of oral structures.(1) The bacterial accumulation is predominantly Gram-negative resulting in an inflammatory response from the body. It has a potential in vascular dissemination of microorganisms through the sulcular epithelium and their products such as lipopolysaccharides (LPS) throughout the body. (2) Many studies have been done to show that the periodontal pathogens and inflammatory markers in the blood are correlated with increased risk of CVD. (3) Inflammatory biomarkers including high-sensitive C-reactive protein (CRP), fibrinogen, serum amyloid A, tumour necrosis factor α (TNF α), interleukin-6 (IL-6) and cellular adhesion molecule are known CVD risk markers. (3)

Fenoglio et al reported that serum and gingival crevicular fluid levels of TNF-α, IL-1β and IL-6 seem to be relating factors between periodontal disease and high serum lipid levels. (4) Several studies done by Losche et al, Katz et al have shown that there is a relationship between periodontitis and high serum lipid level. (4) Cutler et al concluded that plasma levels of lipids are significantly high in individuals with periodontitis than healthy subjects. (5)

Therefore, this study was performed to determine the relationship between chronic periodontitis and plasma lipid level.

MATERIALS AND METHODS
This case-control study was conducted at Tamilnadu Government Dental College & Hospital, Chennai. The study was approved by the ethical committee of the institution.

Sixty patients were selected from those attending the Periodontology Department of the hospital. The present study was done in December 2011 on patients with chronic periodontitis and compared with patients having normal periodontal health. Due to the short duration of the study and with only sixty patients consenting the sample was restricted to sixty and divided into two groups, one group with chronic periodontitis and the other group with normal periodontium. All patients were detailed about the study and informed consent was obtained.

The study group included patients with at least one periodontal pocket with depth more than 4 mm in every quadrant and radiographically showed bony destruction.

The control group included patients who were periodontally healthy. That is these patients had probing depth less than 3 mm and there was no radiographic signs of destruction of bone.


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These patients were randomly selected from those attending the outpatient department of the Tamilnadu Government Dental & Hospital. The intraoral examination was done with mouth mirror and William’s graduated periodontal probe. The periodontal examination included Plaque index (Silness & Loe 1964), gingival index (Loe & Silness 1963) and full mouth probing depth and clinical attachment loss measurement. A thorough intraoral examination was done after obtaining the medical and dental history of the patients. These two groups were age and gender matched.

Both males and females in the age group of 40-50 who were systemically healthy according to their medical records were included in the study.

The exclusion criteria include individuals who had a history of any treatment done during the last 6 months, those with history of diabetes mellitus, endocrine disease, myocardial infarction, stroke, malignancy, history of smoking and those on medication for hyperlipidemia.

The periodontal examination was done in a dental chair using a mouth mirror and William’s graduated periodontal probe. Clinical oral examination included assessment of periodontal pockets in all teeth except the third molars.

All the subjects were referred to the hospital laboratory where fasting blood samples were collected to determine serum lipid levels using routine enzymatic colorimetric test. The pathological cut-off points according to laboratory recommendations were taken as: Total Cholesterol >200 mg/dL, HDL >55 mg/dL, LDL > 130 mg/dL, VLDL >25-35 mg/dL, Triglycerides > 200 mg/dL.

**Statistical Analysis**

The statistical analysis was done using SPSS version 15.0 software. Student’s independent t-test and Chi-square test was used to compare difference in proportions. Table I shows the mean standard deviation and test of significance of mean values between control and case group. Statistical analysis by student independent t-test shows the mean cholesterol level in the case group (198 ± 28.2) which is significantly higher than the control group. Similarly, the mean LDL level in the case group is 126.7 ± 26 which is also significantly higher in the case group than in the control group (98.2 ± 19.3).

The mean VLDL in the case group is 38 ± 12.2 which is also significantly higher than in the control group (23.3 ± 10.3). The triglyceride levels in the case group (191 ± 61.9) is also higher than in the control group (115 ± 51).

Table II shows the frequency of abnormal serum lipid levels in both groups. The proportion of abnormality of LDL, VLDL, Triglycerides in the study group is significantly higher than the control group.

The bar graph shows the comparison of lipid profile in the control and the study groups. It is seen that the lipid levels are significantly higher in the patients with periodontitis than in patients with normal periodontium with the exception of HDL which is lower in the study group than the control group.

### RESULTS

Sixty patients were selected from those attending the Periodontology Department of Tamilnadu Government Dental College and Hospital.

The study group included patients with at least one periodontal pocket with depth more than 4 mm in every quadrant and showing bone destruction radiographically.

The control group included patients who were periodontally healthy, i.e. these patients had probing depth less than 3 mm and there were no radiographic signs of destruction of bone.

Table I shows the mean standard deviation and test of significance of mean values between control and case group. Statistical analysis by student independent t-test shows the mean cholesterol level in the case group (198 ± 28.2) which is significantly higher than the control group. Similarly, the mean LDL level in the case group is 126.7 ± 26 which is also significantly higher in the case group than in the control group (98.2 ± 19.3).

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Table II shows the frequency of abnormal serum lipid levels in both groups. The proportion of abnormality of LDL, VLDL, Triglycerides in the study group is significantly higher than the control group.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=30)</th>
<th>Study (n=30)</th>
<th>Student t-test p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>42 ±3.4</td>
<td>44.6 ± 4.3</td>
<td>0.13 (NS)</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>161 ± 21.7</td>
<td>198.6 ± 28.2</td>
<td>0.002 (Sig)</td>
</tr>
<tr>
<td>HDL</td>
<td>36.3 ± 5.9</td>
<td>35.1 ± 7.7</td>
<td>0.68 (NS)</td>
</tr>
<tr>
<td>LDL</td>
<td>98.2 ± 19.3</td>
<td>126.7 ± 26.7</td>
<td>0.007 (Sig)</td>
</tr>
<tr>
<td>VLDL</td>
<td>23.3 ± 10.3</td>
<td>38.0 ± 12.2</td>
<td>0.004 (Sig)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>115.5 ± 51.0</td>
<td>191.1 ± 61.9</td>
<td>0.003 (Sig)</td>
</tr>
</tbody>
</table>

**Table I. Mean, Standard Deviation and Test of Significance of Mean Values between Control and Study Groups**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=30)</th>
<th>Study (n=30)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Patient %</td>
<td>4 (13%)</td>
<td>21 (70%)</td>
<td>0.001 (Sig)#</td>
</tr>
<tr>
<td>HDL Abnormal</td>
<td>13 (41.7%)</td>
<td>18 (60%)</td>
<td>0.68 (NS)</td>
</tr>
<tr>
<td>LDL Abnormal</td>
<td>1 (3%)</td>
<td>22 (75%)</td>
<td>0.004 (Sig)</td>
</tr>
<tr>
<td>VLDL Abnormal</td>
<td>5 (16.7%)</td>
<td>20 (66.7%)</td>
<td>0.04 (Sig)</td>
</tr>
<tr>
<td>Triglycerides Abnormal</td>
<td>5 (16.7%)</td>
<td>20 (66.7%)</td>
<td>0.04 (Sig)</td>
</tr>
</tbody>
</table>

**Table II. Comparison of Proportion of Abnormality between Control and Study Groups**

*Chi-square test with Yates correction was used to calculate p-values.

# Fisher’s exact test (2-tailed) was used to calculate p-value.
DISCUSSION
In this present study, all the parameters in the lipid profile were significantly higher in the patients with periodontal disease than of those who were normal.

Periodontal disease is an infectious disease caused by Gram-negative anaerobic bacteria. It has been shown that acute infections can interrupt lipid metabolism and there is a significant rise in plasma triglycerides during Gram-negative bacterial infections. Cytokines such as TNF-α, IL-1β are known to play a possible role in the development and progression of atherosclerotic diseases. These cytokines increase the mobilisation of lipids from the liver and adipose tissue and increase the binding of LDL to endothelium and smooth muscles. The oxidative modification of LDL leads to an increase in cholesterol accumulation because modified LDL is very susceptible to macrophage uptake. Studies have also shown that progressive periodontitis also leads to hyperlipidaemia.

Studies conducted by Katz and Cutler have shown a correlation between deep periodontal pockets and high Cholesterol and LDL levels in men but not in women. In their studies, the women were in fertility age, the inhibitory effect of oestrogen on lipid metabolism led to low levels of LDL & cholesterol. But in this present study, the age group selected for both the groups was 40-50 years. Other confounding factors related to elevated Cholesterol levels like smoking and blood pressure were eliminated in this study like those done by Cutler et al., Loesche et al. Subjects with BMI ≥ 30 were excluded in the present study because many studies have shown a relationship between obesity and periodontal disease.

Beck et al. in their study suggested that periodontal disease and atherosclerosis have common genetic predisposition i.e. hyper-inflammatory monocyte phenotype trait. Hence, patients with family history of periodontal disease and atherosclerosis were excluded in the present study. Noack et al stated that the relationship between hyperlipidaemia and periodontal disease could be due to polymorphonuclear leucocytes dysfunction.

CONCLUSION
The results of the present study show a significant relationship between periodontitis and hyperlipidaemia, but further studies with larger population need to be done to confirm this relationship. The present study also supports the hypothesis that periodontal disease is a risk indicator of cardiovascular disease.

REFERENCES