**Evaluation of Alteration in Serum Lipid Profile in Patients with Chronic Periodontitis**

**ABSTRACT**

The current study aimed to investigate the alteration in serum lipid profile in patients with chronic periodontitis, and to correlate the serum levels of total cholesterol (TC), triglyceride (TGL), high density lipoprotein (HDL), low density lipoprotein (LDL) between periodontitis patients and healthy individuals. Total of 40 subjects in the age of 35-60 years were selected and divided into two groups equally based on their periodontal disease status into Test group (20 subjects with chronic periodontitis) and Control group (20 subjects with healthy periodontium). 5 ml of venous blood samples were taken for measurement of serum lipid profile. The study conducted on both the groups showed a highly statistical significance for total cholesterol (p<0.026) and LDL (p=0.001). Thus, showing a positive correlation between periodontitis and hyperlipidemia which plays an important role in the pathogenesis of adverse conditions such as atherosclerosis and cardiovascular complications.

**Keywords:** Periodontitis, hyperlipidemia, cardiovascular disease, HDL, LDL, TC, TGL.

**INTRODUCTION**

Chronic periodontitis is the most frequent form of the disease characterized by an inflammation of the tissues that support the teeth, which results in destruction of the periodontal ligament and loss of the adjacent bone that support the teeth. This disease develops when dental plaques release bacteria responsible for the release of toxic substances and enzymes from sub-alveolar bacteria groups. Severe form of the disease is present in 10% to 15% cases and even in 30% of the population. Recent studies indicate this oral disease may have profound effects on systemic health, changing the blood chemistry with a rise in inflammatory mediators, proteins, and lipids in the serum, and their association with cardiovascular disorders, endocrine disorders, and an adverse relationship in the outcome of pregnancy. It is caused by Gram negative bacteria present on the tooth surface as microbial biofilms. Lipo polysaccharides and other microbial substances gain access to the gingival tissues, initiate and perpetuate inflammation. Periodontitis is associated with the increase in the level of C-reactive protein and fibrinogen, irrespective of coronary diseases.

The response of an organism to the periodontal infection includes production of several enzymes and inflammatory markers which can be analyzed both in serum and saliva. Considering the extent of microbial plaques associated with this disease, its chronic nature and local and systemic immunological responses of the host, it is reasonable to assume that periodontal infections affect the overall health of a patient and could be involved in the development of systemic diseases such as hyperlipoproteinemia and hypertriglyceridemia.

Acute systemic or local chronic infections seem to induce changes in the plasmatic concentration of cytokines and hormones, which determine changes in the lipid metabolism. Therefore, recent studies illustrate the existence of a relation between periodontal disorders and hyperlipidaemia, which power the probable effect of periodontal disease as an underlying factor for hyperlipidaemia. Hyperlipidaemia is a state with an abnormal lipid profile, which is characterized by elevated blood concentrations of triglycerides, elevated levels of total cholesterol and low-density lipoprotein (LDL) and decreased levels of high-density lipoprotein-cholesterol (HDL). It has been suggested that hyperlipidaemia could be associated with periodontitis. Further, these individuals have a perturbed lipid profile (increased serum cholesterol) not explained just by their lifestyle, but perhaps causally related to chronic episodes of bacteremia and endotoxin dissemination.

Hyperlipidemia is considered to be one of the major risk factors for cardiovascular disease (CVD). Hyperlipidemia can lead to development of atherosclerosis, cardiac ischemic disorders, myocardial infarctions, and strokes and in case of high triglyceride levels, it can also result in pancreatitis. Of late, a number of common infectious inflammatory conditions, including periodontitis, have been hypothesized to promote atherogenesis and increase the risk for cardiovascular and cerebrovascular events by continuous release of chronic inflammatory markers to systemic circulation. Various risk factors such as age, smoking habit, diet, obesity, lack of physical exercise, and modern living lifestyle have been confirmed to be the predisposing factors for hyperlipidemia. There is abundant literature delving into the influence of periodontal diseases on hyperlipidaemia. Treatment of hyperlipidaemia consists of administering drugs responsible for decreasing serum lipids in the blood stream. The interrelationship between periodontitis and hyperlipidemia provides an example of a systemic disease.
predisposing to oral infection, and once the oral infection establishes, it exacerbates the systemic disease. Certain systemic disorders and conditions alter host tissues and physiology which may impair host barrier integrity and host defense to periodontal infection resulting in more destructive disease. Several studies have indicated that subjects with periodontal disease may have a higher risk for cardiovascular diseases when compared to subjects with a healthy periodontium. Total cholesterol and low-density lipoprotein (LDL) in patients affected by these diseases were shown to be higher than those in the control group in one study.

The specific objective of the ongoing study was to assess if chronic periodontitis is associated with hyperlipidemia and to compare the levels of serum cholesterol in patients with periodontitis and those who are periodontally healthy.

MATERIALS AND METHODS

A case control study on serum lipid profile was carried out in 20 subjects with chronic periodontitis aged 30-65 years diagnosed based on the existence of calculus and plaques, presence of at least one periodontal pocket with a depth equal to or more than 4 mm in each quadrant and evidence of alveolar bone destruction, correlating with plaques and calculus, determined on panoramic radiograph was compared with 20 control subjects with good general health and with no history of systemic diseases. The exclusion criteria for the study were dental treatment during the past 6 months, diabetes, smoking, alcoholics, pregnant, lactating and postmenopausal women, aggressive periodontitis, cardiac diseases, rheumatoid arthritis, obesity, patients taking any drug for hypercholesterolemia and any other systemic disease, which can alter the course of periodontal disease or serum lipid levels.

Serum lipid levels measurement

Informed consent was obtained from each participant in the study. 5 ml of venous blood samples was collected in plain bulb from patients with periodontitis and normal healthy individuals. Blood samples were centrifuged at 3000g for 10 minutes. Serum collected was estimated for lipid profile. Serum total cholesterol, triglyceride and HDL-cholesterol were measured by an enzymatic kit method. LDL-C and VLDL was calculated according to Friedwald’s formulae: VLDLC=Triglyceride/5 and LDL-C= Total cholesterol-(VLDLC + HDL-C).

Statistical analysis

Statistical analysis was done by using students ‘t’ test and the data were expressed as mean ± standard deviation. Probability values of <0.05 were considered to be statistically significant. The comparison of control group was carried out with the study group with respect to mean value and standard deviation. This biochemical analysis is to correlate between the level of lipid profile and clinical parameter.

RESULTS

The present study was conducted in 40 subjects, including 20 cases of periodontitis and 20 control subjects with general good health levels of total cholesterol, triglyceride, HDL and LDL in blood serum were measured in both groups and assuming a normal distribution curve, average level for two groups were calculated using t-test. The prevalence of abnormal serum lipid levels in both the group is tabulated in Table 1.

Patients and Healthy Individual

![Figure 1: Comparison of serum lipid profile among chronic periodontitis](image)

### Table 1: Correlation between serum lipid profile in case and control group.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>Standard deviation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CHOLESTROL</strong></td>
<td>Periodontitis</td>
<td>20</td>
<td>182.75</td>
<td>167.15</td>
<td>26.179</td>
</tr>
<tr>
<td></td>
<td>Without periodontitis</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TGL</strong></td>
<td>Periodontitis</td>
<td>20</td>
<td>189.35</td>
<td>104.50</td>
<td>37.399</td>
</tr>
<tr>
<td></td>
<td>Without periodontitis</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HDL</strong></td>
<td>Periodontitis</td>
<td>20</td>
<td>36.65</td>
<td>43.95</td>
<td>7.8889</td>
</tr>
<tr>
<td></td>
<td>Without periodontitis</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LDL</strong></td>
<td>Periodontitis</td>
<td>20</td>
<td>106.30</td>
<td>87.25</td>
<td>21.303</td>
</tr>
<tr>
<td></td>
<td>Without periodontitis</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>VLDL</strong></td>
<td>Periodontitis</td>
<td>20</td>
<td>40.75</td>
<td>20.85</td>
<td>15.193</td>
</tr>
<tr>
<td></td>
<td>Without periodontitis</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Mean total cholesterol of case and control groups (182.75±26.179 mg/dl and 167.15±24.132 mg/dl, respectively) showed significant difference. (T=1.269, p=0.026). Triglyceride level of case and control groups (189.35±37.399 mg/dl and 104.5±33.376 mg/dl, respectively) did not show any significant difference. (T=0.38, p=0.53). Comparing mean HDL between the two groups (36.65±7.889 mg/dl and 43.95±10.665 mg/dl, respectively) showed no significant difference. (T=0.79, p=0.47).

Mean LDL levels between case and control groups (106.3±21.303 mg/dl and 87.25±9.819 mg/dl, respectively) showed significantly different. (T=2.51, p=0.001). Mean VLDL levels between case and control groups (40.75±15.193 mg/dl and 20.85±5.204 mg/dl, respectively) showed significant difference. (T=3.92, p=0.05).

Therefore, there is a strong positive statistical association between the existence of periodontal pockets and plasma lipid levels, thereby confirming a positive relationship between periodontitis and hyperlipidemia.

**DISCUSSION**

In the present study, the serum total cholesterol, TGL, LDL, and HDL were assessed in 20 periodontal patients and 20 healthy individuals which showed a significant increase in the level of total cholesterol and LDL. Which is similar to the studies conducted by Losche et al 2000, who showed that serum levels of total cholesterol, TG, and LDL were higher in patients compared with those in the control group.

The HDL-C level, although not statistically significant, is slightly less in the hyperlipidemia group than in the control group. Serum HDL concentration usually decreases in subjects with chronic infection. In the present study, the patients did not have significant decrease in the HDL levels; this could be due to the fact that most of the subjects had mild to moderate hyperlipidemia as expressed by the mean values.

Periodontitis is an infectious disease caused by gram negative anaerobic bacteria. Gram negative periodontal pathogens trigger systemic release of interleukin -1 beta (IL-1 beta) and TNF alpha. This trigger of interleukin and TNF acts in two ways, one side they cause an alteration in the fat metabolism which leads to chronic hypertriglyceridemia and on the other hand production of IL-1 beta causes continued tissue destruction and pathologic wounding of gingiva. Also, it has been found that presence of bacteriaeae causes an enhancement in number of antibodies against P.gingivalis, which causes subclinical endotoxaemia. This in turn, induces change in lipid metabolism through increased hepatic lipoprotein production. This finally causes hypertriglyceridemia and binding of lipoprotein to endotoxins that enhances their secretion. It is known that activated PMNs cause an increase in release of superoxide anions and in high dietary fat PMNs lose their antibacterial function. Noack et al. stated that the relationship between hyperlipidemia and periodontal infection could be due to polymorph nuclear leucocytes (PMN-cells) dysfunction.

There are some theories that periodontitis predispose individuals to systemic disease. Inflammation can operate in all stages of periodontitis from initiation through progression and ultimately the thrombotic complications of atherosclerosis, thus emerging as an integrative cardiovascular factor. Thus, this inflammation may be responsible for the greater risk of cardiovascular disease in patients with periodontitis. This on-going study has enlightened a positive correlation between periodontitis and hyperlipidaemia and therefore, Hyperlipidaemia can lead to cardiovascular diseases, which have high mortality rates. Therefore, it is recommended that patients with heart disease should strongly consider periodontal treatment.

**CONCLUSION**

Based on the present study, the chronic periodontitis patients show elevated levels of total cholesterol and LDL in comparison to healthy subjects who pose an increased risk of hyperlipidemia. This study proves a relationship between periodontal disease and hyperlipidemia. Hyperlipidemia can lead to cardiovascular diseases, with high mortality rates. Thus, it may be concluded that periodontal treatment contributes to control the lipid levels, particularly in patients with cardiovascular disease. Hence, patients with heart disease are highly recommended to consider periodontal treatment, so that the systemic complications due to elevated lipid levels could be prevented.

**REFERENCES**

3. Dr.NitinKhuller, Dr. Dinesh Duhan, Dr.Yashbir Singh Raghav, Dr.Shivli Sharma, Changes In Serum Lipid Levels As A Possible Risk For Cardiovascular Disease In Chronic Periodontitis Patients: A Cross Sectional Study, J periodontol med dinpract, 01,2014, 162-171.


Source of Support: Nil, Conflict of Interest: None.