ASSESSMENT OF SERUM LIPID PROFILE AND ITS ASSOCIATION WITH CHRONIC PERIODONTITIS - A CLINICO-BIOCHEMICAL STUDY

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ABSTRACT

Aim: Aim of the present study was to evaluate the association of serum lipid profile in patients with chronic periodontitis.

Materials and Methods: The study sample constituted 60 patients (males & females) in the age range of 30 to 50 years. The enrolled patients were allocated in one of the following two groups having 30 participants each: Group A: Individuals without periodontitis, Group B: Individuals with chronic periodontitis. Various periodontal factors such as plaque index, gingival index and pocket depth on probing and clinical attachment levels were documented. Employing a sterilized syringe, 2 ml of IV blood was taken from every patient’s antecubital vein and released into a vacutainer, following which it was subjected to laboratory analysis of complete lipid profile including the total cholesterol (TC), triglyceride (TGL), high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL), very low density lipoprotein levels (VLDL).

Results: Group A (without periodontitis) demonstrated a mean plaque index score of 0.96 ±0.10 and a gingival index of 0.84±0.28. Group B (with chronic periodontitis) had a mean plaque index score was 1.76±0.86, gingival index of 1.82±0.52. The pocket depth on probing was 5.24±0.22 with a clinical attachment level of 4.80±0.74. In Group B with chronic periodontal disease, the serum lipid profile was noted to be higher. In this group the serum lipid levels were TC (224.33±13.64), TGL (118.64±10.56), HDL (40.08±10.12), LDL (156.67±10.42), VLDL (28.82±5.12). In Group A without periodontitis, the serum lipid levels were TC (152.24±8.52), TGL (83.40±6.30), HDL (46.39±8.44), LDL (71.27±9.20), VLDL (13.45±7.31). The difference between the two groups was statistically significant.

Conclusion: This study concludes that high serum lipid profile levels were noted in the group with chronic periodontitis in contrast to the healthy individuals thereby posing as risk for hyperlipidemia within the confines of the inherent limitations of this research.

Keywords: chronic periodontitis, hyperlipidemia, inflammation, serum lipid profile.

I. INTRODUCTION:

The interplay of bacterial plaque biofilm and immune-inflammatory host reaction with ensuing imbalance of the homeostasis of osseous and connective tissues results in periodontal pathosis. It was conventionally believed that periodontitis is a disease affecting the oral cavity with localized damage to the periodontium. On the contrary, existing evidence implies that periodontitis can affect and alter systemic health of an individual.¹
Current research proposes that oral disease may have intense consequences on the systemic health with resultant changes in the blood chemistry as reflected in the elevation of mediators of inflammation, serum lipids as well as proteins. These are linked to endocrinopathies, cardiovascular ailments and can adversely affect pregnancy outcomes. Gram negative bacteria that constitute the microbial biofilm on the surface of teeth are indicated in causation of such effects. Bacterial toxins and lipid-polysaccharide complexes attain entrance into the gingival tissues that begin and continue the process of inflammation. Regardless of the coexistence of coronary disorders, periodontitis is connected with enhanced levels of C-reactive protein and fibrinogen. An organism responds to periodontal infection with generation of various enzymes and markers of inflammation that may be assessed in serum as well as saliva.

Disturbed serum lipid profiles with elevation of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein-cholesterol (LDLC) in blood and reduced high-density lipoprotein cholesterol (HDL-C) level are the chief markers that robustly link to the pathogenesis of cardiovascular diseases. Lipids interact with macrophage cell membranes that impedes receptors on their membrane and other enzymes. This changes the macrophage gene expression of vital polypeptide growth factors and pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin (IL)-1β, that are correlated to periodontitis.

Prior postulation of chronic destructive periodontal disease being a localized inflammation causing disease is now rendered outdated. Presently, it is accepted as a widespread condition with a vast influence on an array of medical disorders with noteworthy systemic connotation. Thus the current research was performed to evaluate the association of serum lipid profile in patients with chronic periodontitis.

II. MATERIALS AND METHODS:

The current clinical research was performed in the department of periodontics, Kalinga institute of dental sciences, Bhubaneswar, India. The study sample constituted 60 patients (males & females) in the age range of 30 to 50 years. These individuals were the attending patients in the outpatient department of periodontics who were anticipating periodontal therapy. The study participants were explained about the aim of the ensuing investigations and their informed consent to the study protocol was obtained.

The inclusion criteria were healthy individuals in the age range of 30-50 years without evidence of any systemic disease. The presence of a minimum of sixteen teeth in these individuals was essential. The exclusion criteria was patients with history of smoking, an acknowledged systemic ailment such as diabetes mellitus/hypertension/others, obesity with a body mass index (BMI) > 30, pregnancy/lactation, individuals with fewer than 16 teeth, history of systemic antibiotics over the last three months and those with aggressive periodontal disease.

The enrolled patients were allocated in one of the following two groups having 30 participants each:

Group A: Individuals without periodontitis
Group B: Individuals with chronic periodontitis

III. COLLECTION OF CLINICAL DATA:

Each of the study participants was seated in an upright and comfortable posture in the dental chair. Following this all individuals were subjected to a full-mouth periodontal assessment. A standardized examiner carried out probing of six regions (distobuccal, buccal, mesiobuccal, distolingual, lingual and mesiolingual) for each tooth (with the exclusion of the third molars), employing the Williams periodontal probe. Various periodontal factors such as plaque index, gingival index and pocket depth on probing were documented. The periodontal pocket was recorded as the distance from the free gingival margin to the bottom of pocket at the closest millimeter. The clinical attachment point was calculated as the distance between the CEJ to the bottom of pocket at the closest millimeter.

Assessment of Lipid Profile:

Blood was drawn after an eight-hour fasting, prior to conduct of the dental procedures. Employing a sterilized syringe, 2 ml of IV blood was taken from every patient’s antecubital vein and released into a vacutainer, following which it was subjected to lab analysis of complete lipid profile including the total cholesterol (TC),
triglyceride (TGL), high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL), very low-density lipoprotein levels (VLDL).

**Statistical analysis:**

Students’ ‘t’ test was used for the statistical analysis with the data being expressed as mean and standard deviation. Statistical significance was deemed at probability values of <0.05. Comparative assessment of the study and control group was done in relation to mean value and standard deviation. The biochemistry was correlated with lipid profiles and factors judged by clinical evaluation.

IV. RESULTS:

Table 1 depicts the descriptive analysis of factors related to the periodontium on clinical examination for both the study groups. Group A (without periodontitis) demonstrated a mean plaque index score of 0.96±0.10 and a gingival index of 0.84±0.28. Group B (with chronic periodontitis) had a mean plaque index score of 1.76±0.86, gingival index of 1.82±0.52. The pocket depth on probing was 5.24±0.22 with a clinical attachment level of 4.80±0.74.

Table 1: Evaluation of the clinical periodontal parameters for two groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>Plaque index (Mean±SD)</th>
<th>Gingival index (Mean±SD)</th>
<th>Probing pocket depth (Mean±SD)</th>
<th>Clinical attachment level (Mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A: without periodontitis</td>
<td>0.96±0.10</td>
<td>0.84±0.28</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Group B: with chronic periodontitis</td>
<td>1.76±0.86</td>
<td>1.82±0.52</td>
<td>5.24±0.22</td>
<td>4.80±0.74</td>
</tr>
</tbody>
</table>

Table 2 demonstrates serum lipid profile comparative assessment amid both the groups. In Group B with chronic periodontal disease, the serum lipid profile was noted to be higher. In this group the serum lipid levels were as follows: TC (224.33±13.64), TGL (118.64±10.56), HDL (40.08±10.12), LDL (156.67±10.42), VLDL (28.82±5.12). In Group A without periodontitis, the serum lipid levels were TC (152.24±8.52), TGL (83.40±6.30), HDL (46.39±8.44), LDL (71.27±9.20), VLDL (13.45±7.31). The difference between the two groups was statistically significant.

Table 2: Comparison of Serum lipid profile between the groups

<table>
<thead>
<tr>
<th>Lipid Profile</th>
<th>Group A (Mean±SD)</th>
<th>Group B (Mean±SD)</th>
<th>t value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mg/dl)</td>
<td>152.24±8.52</td>
<td>224.33±13.64</td>
<td>8.56</td>
<td>0.001</td>
</tr>
<tr>
<td>TGL (mg/dl)</td>
<td>83.40±6.30</td>
<td>118.64±10.56</td>
<td>9.88</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>46.39±8.44</td>
<td>40.08±10.12</td>
<td>11.74</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>71.27±9.20</td>
<td>156.67±10.42</td>
<td>9.06</td>
<td>0.001</td>
</tr>
<tr>
<td>VLDL (mg/dl)</td>
<td>13.45±7.31</td>
<td>28.82±5.12</td>
<td>7.27</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table 3 delineates the relationship amid the factors assessed clinically and the serum lipid profile in Group B. Serum level of TC exhibited a positive correlation that was of significance with (PI, PPD and CAL), while GI demonstrated a positive non-significant relationship. The serum levels of TGL exhibited a positive non-significant relation with (GI, PPD, CAL) as well as a negative correlation with (PI) that was not significant. HDL depicted positive correlation that was not significant with (PI, PPD) and negative relation that was not significant with (GI, CAL), LDL exhibited positive association that was not significant with (PI, CAL) and of significance with (GI) while having a negative correlation with (PPD) that wasn’t significant and VLDL delineated positive yet non-significant correlation with (PPD) and negative relationship with (PI, GI, CAL) that was not significant.
Table 3: Correlation between serum lipid profile and clinical parameters with chronic periodontitis group

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>Plaque index (PI)</th>
<th>Gingival index (GI)</th>
<th>Probing pocket depth (PPD)</th>
<th>Clinical attachment level (CAL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>r value 0.434</td>
<td>0.248</td>
<td>0.624</td>
<td>0.714</td>
</tr>
<tr>
<td></td>
<td>P value 0.001</td>
<td>0.579</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>TGL</td>
<td>r value -0.196</td>
<td>0.212</td>
<td>0.311</td>
<td>0.418</td>
</tr>
<tr>
<td></td>
<td>P value 0.072</td>
<td>0.061</td>
<td>0.450</td>
<td>0.311</td>
</tr>
<tr>
<td>HDL</td>
<td>r value 0.219</td>
<td>-0.144</td>
<td>0.358</td>
<td>-0.489</td>
</tr>
<tr>
<td></td>
<td>P value 0.527</td>
<td>0.322</td>
<td>0.560</td>
<td>0.286</td>
</tr>
<tr>
<td>LDL</td>
<td>r value 0.182</td>
<td>0.216</td>
<td>-0.297</td>
<td>0.327</td>
</tr>
<tr>
<td></td>
<td>P value 0.081</td>
<td>0.041</td>
<td>0.070</td>
<td>0.312</td>
</tr>
<tr>
<td>VLDL</td>
<td>r value -0.320</td>
<td>-0.884</td>
<td>0.421</td>
<td>-0.515</td>
</tr>
<tr>
<td></td>
<td>P value 0.543</td>
<td>0.510</td>
<td>0.497</td>
<td>0.426</td>
</tr>
</tbody>
</table>

V. DISCUSSION:

One frequent manifestation of poor oral hygiene that results in progressive degradation of supporting structures of the natural teeth is periodontitis. In this condition, the creation of pro-inflammatory cytokines as well as enzymes responsible for degradation of tissues is begun and perpetuated by oral microbial infection that results in annihilation of periodontium. When left untreated, loss of bone, loosening of teeth, recession of gingiva results.7

Lipids are noteworthy structural as well as bio-regulatory components of human cells along with plasma lipoproteins. The regulatory means of cell formation and the complex metabolics involved in their mechanism of transport in the body determines the plasma levels of the major blood lipids. Gram negative infectious processes associated with periodontal pathosis cause systemic inflammatory changes that are co-related with the formation of hyperlipidaemia, an essential risk factor for initiation and advancement of cardiovascular disease. Hypercholesterolemia and CVD are associated with periodontal pathosis as stated by Katz J et al.8

Mean values of TC, TGL, LDL and VLDL were noted to be greater in the group with chronic periodontal disease while lower mean values were seen in the group with periodontally sound teeth. On the contrary however, HDL levels were greatest in the group with periodontally sound teeth and lowest for the group with chronic periodontitis following elimination of potential confounding factors. An alike finding was reported by Penumarthy S et al.9 who noted that raised levels of lipid parameters in group with periodontal pathosis was basically a result of high levels of pro-inflammatory cytokines that resulted due to chronic periodontal disease.

In this research, lipid profiles were evaluated for the two groups, accounting for recent evidence that suggests periodontal disease as a potential risk parameter for hyperlipidemia. A link amongst periodontitis and hyperlipidemia has been implicated through the research by Katz J et al.8 and Lösche W et al.9 while the research by Machado AC et al.10 does not report any such co-relation.

The TC values were greatest in Group B while they were lower in Group A. The difference amid both groups was of statistical significance. These outcomes are in agreement with research by Henrich J et al.11 and Hamissi Jet al.12 which demonstrated that periodontal pathology was considerably linked to elevated TC levels.

In this study, TGL levels were high in the group with advanced periodontal disease as judged against the group without periodontitis. This was statistically significant and in concordance with prior research performed by Taleghani F et al.13 and Morita M et al.14 have also depicted a rise in TGL levels in patients with chronic periodontitis. The mean VLDL levels were raised in Group B versus Group A and the difference was statistically significant. This is in agreement with the research by Kundu M et al.15 who found VLDL to be significantly greater (p<0.05) in chronic periodontal pathosis group as compared with the control group. Similarly greater mean HDL levels were noted by Morita et al.14, in healthy controls in contrast with the group with pathology, although this was not statistically significant. This is in agreement with our results as we noted greater levels of HDL in Group A when compared to Group B.

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Hyperlipidemia has been acknowledged in causing excessive action of leukocytes with enhanced formation of oxygen radicals that are linked to advancing periodontal disease in adults. Nevertheless, the soundness of evidence-based two directional associations amid hyperlipidemia as well as periodontitis has not yet been ascertained and thus necessitates additional research and assessment.

The limitation of this research is the uni-centric, inadequate populace in which the existing lifestyles as well as parameters like diet and physical actions were not considered which could have lead to alterations in the study outcomes. Numerous multicentric research that include higher sample size along with the linked environmental parameters need to be taken up to get rid of such limiting factors to demonstrate the actual association.

VI. CONCLUSION:

This study concludes that high serum lipid profile levels were noted in the group with chronic periodontitis in contrast to the healthy individuals thereby posing as risk for hyperlipidemia within the confines of the inherent limitations of this research.

REFERENCES: