ASSESSMENT OF ASSOCIATION BETWEEN NON-ALCOHOLIC FATTY LIVER DISEASE AND INFLAMMATORY PERIODONTAL DISEASE

Padam Singh1, Dr. Pooja Mittal2, Dr. Himanshi Agarwal3, Dr. Priyamwada sethi4, Dr. Rohan Vashisht5, Dr. Harsukhman Kaur6

1Professor, Department of Periodontics, Desh Bhagat Dental College, Mandi gobindghar, Punjab, India;
2,5Assistant Professor, Department of Periodontics, Desh Bhagat Dental College and Hospital, Mandi gobindgarh, Punjab, India;
3Assistant Professor, Department of Periodontics, Institute of Dental Sciences, Bareilly, U.P., India;
4Assistant professor, Department of Periodontics, Career dental college, Lucknow, U.P., India;
6Assistant Professor, Department of Periodontics, BJS Dental College, Hospital and Research Institute, Ludhiana, Punjab, India

ABSTRACT

Background: Chronic periodontitis is an infectious disease induced by oral bacteria that can lead to the destruction of soft tissues surrounding the teeth, bones, and ligaments. The present study was conducted to assess association between non-alcoholic fatty liver disease and inflammatory periodontal disease.

Materials & Methods: 60 patients with non-alcoholic fatty liver disease were included. Patients were divided into 2 groups. Group I comprised of patients of NAFLD and group II had 60 healthy subjects. Periodontal status such as bleeding on probing (BOP), probing depth (PD), clinical attachment loss (CAL) was recorded. All patients were subjected to an ultrasound examination of abdomen and transient elastography (fibroscan) for the assessment of hepatic steatosis and fibrosis.

Results: Group I had 45 males and 15 females and group II had 40 males and 20 females. The mean AST (U/l) in group I was 72.1 and in group II was 23.6, ALT (U/l) was 70.5 in group I and 25.1 in group II, TG level (mg/dl) was 190.2 in group I and 165.2 in group II, HDL (mg/dl) was 41.3 in group I and 39.4 in group II, mean bleeding on probing was 2.6 in group I and 1.2 in group II, CAL (mm) was 4.5 mm in group I and 2.1 mm in group II and probing depth was 3.2 mm in group I and 2.4 mm in group II. 42% in group I and 15% in group II had periodontitis. The difference was significant (P< 0.05).

Conclusion: Patients with non-alcoholic fatty liver disease showed a higher prevalence of periodontal disease as compared to healthy subjects.

Key words: Healthy, Non-alcoholic fatty liver disease, Periodontal disease.

I. INTRODUCTION

Periodontal disease, especially chronic periodontitis, is an infectious disease induced by oral bacteria that can lead to the destruction of soft tissues surrounding the teeth, bones, and ligaments. Bacteria in plaques are closely involved with the onset of periodontal disease, and the mucosal epithelium is inflamed by exotoxins produced by the bacteria. The shedding of lipopolysaccharide from periodontal flora stimulates the endothelial cells, monocytes, and macrophages to initiate a proinflammatory response resulting in long-standing sustained increase in many cytokines enhancing inflammation, i.e., interleukins 1β (IL-1β), 6, and tumor necrosis factor (TNF)-α. A huge body of contemporary epidemiological studies have reported periodontal disease as a risk factor for various overall health conditions, including cardiovascular disease, type 2 diabetes, adverse pregnancy outcomes, and rheumatoid arthritis.

Periodontal disease results in not only tooth loss but also the aggravation of numerous types of systemic diseases, including type 2 diabetes, cardiovascular diseases, preterm low birth weight, and non-alcoholic fatty liver disease.
(NAFLD). Thus, monitoring and management of periodontitis is important because it is present in almost half the adult population.4

NAFLD is a clinical entity characterized by the presence of hepatic steatosis affecting at least 5% of hepatocytes in individuals who consume little or no alcohol and who do not have a secondary cause of hepatic steatosis. NAFLD includes heterogeneous spectra ranging from simple steatosis to NASH and liver cirrhosis.5 Liver fibrosis is an independent risk factor affecting the prognosis of patients with NASH and a systematic review and meta-analysis concluded that liver fibrosis was the most important liver histological finding for all-cause and liver disease-related mortality in NAFLD. Porphyromonas gingivalis is most prevalent pathogen was detected in significantly higher frequency in periodontal disease in NAFLD patients than in the non-NAFLD participants.6 The present study was conducted to assess association between non-alcoholic fatty liver disease and inflammatory periodontal disease.

II. MATERIALS & METHODS

The present study was conducted among 60 patients with non-alcoholic fatty liver disease. All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. Patients were divided into 2 groups. Group I comprised of patients of NAFLD and group II had 60 healthy subjects. A careful oral examination was done. Periodontal status such as bleeding on probing (BOP), probing depth (PD), clinical attachment loss (CAL) were recorded. Body mass index (BMI) and waist-hip ratio for central obesity and abdominal examination for any organomegaly were recorded. After routine hematological, biochemical (including fasting plasma glucose, lipid profile), all patients were subjected to an ultrasound examination of abdomen and transient elastography (fibroscan) for the assessment of hepatic steatosis and fibrosis. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

III. RESULTS

Table I Distribution of patients

<table>
<thead>
<tr>
<th>Groups</th>
<th>Group I</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Status</td>
<td>NAFLD</td>
<td>Healthy</td>
</tr>
<tr>
<td>M:F</td>
<td>45:15</td>
<td>40:20</td>
</tr>
</tbody>
</table>

Table I shows that group I had 45 males and 15 females and group II had 40 males and 20 females.

Table II Assessment of parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group I</th>
<th>Group II</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>72.1</td>
<td>23.6</td>
<td>0.01</td>
</tr>
<tr>
<td>ALT</td>
<td>70.5</td>
<td>25.1</td>
<td>0.02</td>
</tr>
<tr>
<td>TG</td>
<td>190.2</td>
<td>165.2</td>
<td>0.03</td>
</tr>
<tr>
<td>HDL</td>
<td>41.3</td>
<td>39.4</td>
<td>0.12</td>
</tr>
<tr>
<td>Mean BOP</td>
<td>2.6</td>
<td>1.2</td>
<td>0.05</td>
</tr>
<tr>
<td>CAL (mm)</td>
<td>4.5</td>
<td>2.1</td>
<td>0.02</td>
</tr>
<tr>
<td>Probing depth (mm)</td>
<td>3.2</td>
<td>2.4</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Table II, graph I shows that mean AST (U/l) in group I was 72.1 and in group II was 23.6, ALT (U/l) was 70.5 in group I and 25.1 in group II. TG level (mg/dl) was 190.2 in group I and 165.2 in group II, HDL (mg/dl) was 41.3 in group I and 39.4 in group II, mean bleeding on probing was 2.6 in group I and 1.2 in group II, CAL (mm) was 4.5 mm in group I and 2.1 mm in group II and probing depth was 3.2 mm in group I and 2.4 mm in group II. The difference was significant (P< 0.05).
Table III Prevalence of periodontitis in both groups

<table>
<thead>
<tr>
<th>Periodontitis</th>
<th>Group I</th>
<th>Group II</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>42%</td>
<td>15%</td>
<td>0.05</td>
</tr>
<tr>
<td>No</td>
<td>58%</td>
<td>85%</td>
<td></td>
</tr>
</tbody>
</table>

Table III, graph I shows that 42% in group I and 15% in group had periodontitis. The difference was significant (P< 0.05).

IV. DISCUSSION

Salivary levels of Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and Prevotella intermedia were determined by bacterial culture and related to clinical periodontal status in subjects with varying degrees of periodontitis. The gastrointestinal tract begins with the mouth and proceeds to the intestines; ingested bacteria travel through the tract; thus, affect gut microbiota composition. Dysbiosis of the gut microbiota can lead to
several diseases, including diabetes, rheumatoid arthritis, and inflammatory bowel disease. With the rising incidence and prevalence, an enhanced understanding of the diverse aspects of NAFLD is appearing and is alarming in context of its clinical implications. Approximately 10%–30% have the potentially advancing type of NAFLD, NASH (NASH/hepatocellular injury and inflammation). Further, 25%–40% of NASH patients may progress to liver fibrosis over time, resulting in cirrhosis in 20%–30%. Such cirrhotic patients are highly susceptible to develop hepatocellular carcinoma (2.6%/year). NASH is considered synonymous to the metabolic syndrome (MS) affecting the liver, i.e., as an associate morbidity to diabetes mellitus (DM) type 2, hyperlipidemia, and hypertension (HTN).

The present study was conducted to assess association between non-alcoholic fatty liver disease and inflammatory periodontal disease.

In present study, group I had 45 males and 15 females and group II had 40 males and 20 females. Duseja et al. evaluated the possible association between non-alcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease among north Indian population. A total of 40 cases, i.e., patients with NAFLD and 40 healthy volunteers were included over a period of 8 months and their periodontal status was compared. The status of their hepatic health was ascertained by anthropometric, imaging, and biochemical evaluation including ultrasound examination of abdomen and transient elastography. The study revealed that only 11.9% and 20% of participants had periodontitis, in healthy controls and hepatic disease patients, respectively. A statistically significant difference was observed in clinical parameters of periodontal status, except for malocclusion. Comparative analysis of tumor necrosis factor-α (TNF-α), interleukin-6, C-reactive protein, and cytokeratin-18 revealed differences in mean scores, though statistically nonsignificant. Only aspartate transaminase, number of missing teeth, and bleeding on probing (BOP) were observed with higher odds ratios for hepatic disease patients. Spearman correlation analysis revealed significant positive correlations between TNF-α and BOP, for cases.

We found that mean AST (U/l) in group I was 72.1 and in group II was 23.6, ALT (U/l) was 70.5 in group I and 25.1 in group II. TG level (mg/dl) was 190.2 in group I and 165.2 in group II, HDL (mg/dl) was 41.3 in group I and 39.4 in group II, mean bleeding on probing was 2.6 in group I and 1.2 in group II, CAL (mm) was 4.5 mm in group I and 2.1 mm in group II and probing depth was 3.2 mm in group I and 2.4 mm in group II. 42% in group I and 15% in group II had periodontitis. A recent large sample investigation reported an association of periodontitis with hepatic steatosis and significant liver fibrosis (NHANES III cohort). Similar findings have been revealed regarding significant liver fibrosis in a prospective study of largely biopsy-proven NAFLD. In fact, authors confirmed the association with steatosis and demonstrated a gradient of periodontitis with worsening liver injury. This investigation did not take in account of any biochemical markers such as hepatic enzymes or inflammatory serum markers, but similar clinical periodontal findings could not be replicated in our investigation. Authors found that patients with non-alcoholic fatty liver disease showed a higher prevalence of periodontal disease as compared to healthy subjects.

V. CONCLUSION

Authors found that patients with non-alcoholic fatty liver disease showed a higher prevalence of periodontal disease as compared to healthy subjects.

REFERENCES
