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Original Research

Assessment of association between nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease

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ABSTRACT:
Background: 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. NAFLD has been associated with infections of gastrointestinal tract, such as small intestinal bacterial overgrowth. The present study assessed association between nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease. **Materials & Methods:** 56 patients of NAFLD of both genders were divided into 2 groups. Group I had NAFLD and group II had healthy control. Parameters such as family history of diabetes, HTN, coronary artery disease, and stroke were recorded. Body mass index (BMI), hematological, biochemical and ultrasound examination of abdomen was performed. CAL, PD and BOP was also recorded. **Results:** Group I had 36 males and 20 females and group II had 28 males and 28 females. The mean AST level was 72.4 in group I and 24.8 in group II and ALT level was 79.3 in group I and 25.7 in group II. The difference was significant (P< 0.05). The mean calculus index simplified in group I was 31.3 and in group II was 50.2, debris index simplified in group I was 32.6 and in group II was 51.4, probing depth was 36.2 in group I and 46.1 in group II, bleeding on probing was 52.4 in group I and 31.8 in group II, clinical attachment loss was 53.9 in group I and 32.9 in group II and OHI-S was 30.2 in group I and 51.6 in group II. The difference was significant (P< 0.05). **Conclusion:** Patients with NAFLD showed a higher prevalence of periodontal disease as compared to healthy individuals.
Key words: nonalcoholic fatty liver disease, periodontal disease, oral hygeine index

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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.[1] Bacteria in plaques are closely involved with the onset of periodontal disease, and the mucosal epithelium is inflamed by exotoxins produced by the bacteria.[2] 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 nonalcoholic fatty liver disease (NAFLD). Thus, monitoring and management of periodontitis is important because it is present in almost half the adult population. There are three hypotheses for how periodontitis affects systemic diseases.[3]

NAFLD has been associated with infections of gastrointestinal tract, such as small intestinal bacterial overgrowth. Oral bacteria have also been implicated in the induction of endotoxemia and subsequent hepatic inflammatory responses directly or indirectly through imparting alterations in gut flora.[4] More recently, Porphyromonasgingivalis, most prevalent pathogen was detected in significantly higher frequency in periodontal disease in NAFLD patients than in the non-NAFLD participants (46.7% vs. 21.7%, odds ratio [OR]: 3.16). Moreover, NASH patients showed a higher detection rate of P. gingivalis than that in the non-NAFLD participants.[5]The present study assessed association between nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease.

MATERIALS & METHODS

The present study comprised of 56 patients of NAFLD of both genders. All were informed regarding the study and their written consent was obtained. Ethical clearance was obtained before starting the study.

Demographic data such as name, age, gender etc. was recorded. Patients were divided into 2 groups. Group I had NAFLD and group II had healthy control. Parameters such as family history of diabetes, HTN, coronary artery disease, and stroke were recorded. Body mass index (BMI) and waist-hip ratio for central obesity and abdominal examination for any organomegaly were recorded. Assessment of hematological, biochemical and ultrasound examination of abdomen was performed. CAL, PD and BOP was also recorded. Results of the study was compiled and compared in both groups. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Status	NAFLD	Healthy control
M:F	36:20	28:28

Table I shows that group I had 36 males and 20 females and group II had 28 males and 28 females.

Table II Comparison of parameters

Parameters	Group I	Group II	P value
AST	72.4	24.8	0.01
ALT	79.3	25.7	0.01

Table II shows that mean AST level was 72.4 in group I and 24.8 in group II and ALT level was 79.3 in group I and 25.7 in group II. The difference was significant (P< 0.05).

Table III Oral clinical parameters in both groups

Parameters	Group I	Group II	P value
CIS	31.3	50.2	0.02
DIS	32.6	51.4	0.03
PD	36.2	46.1	0.05
BOP	52.4	31.8	0.04
CAL	53.9	32.9	0.02
OHI- S	30.2	51.6	0.01

Table III, shows that mean calculus index simplified in group I was 31.3 and in group II was 50.2, debris index simplified in group I was 32.6 and in group II was 51.4, probing depth was 36.2 in group I and 46.1 in group II, bleeding on probing was 52.4 in group I and 31.8 in group II, clinical attachment loss was 53.9 in group I and 32.9 in group II and OHI- S was 30.2 in group I and 51.6 in group II. The difference was significant (P< 0.05).

DISCUSSION

Increased inflammatory mediator levels were decreased after successful periodontal treatment. Secondly, in patients with periodontitis, bacteremia may originate from periodontal pockets, as patients with generalized chronic periodontitis showed ulcers in inflamed periodontal pockets, and the total area of the ulcers was estimated to be as large as the size of the palm.[6] Alterations in the oral microbiome due to periodontal disease may affect the gut microbiome.[7] Salivary levels of Aggregatibacteractinomycetemcomitans, Porphyromonasgingivalis, and Prevotellaintermedia were determined by bacterial culture and related to clinical periodontal status in subjects with varying degrees of periodontitis.[8] 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.[9]The present study assessed association between nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease.

We found that group I had 36 males and 20 females and group II had 28 males and 28 females. The mean AST level was 72.4 in group I and 24.8 in group II and ALT level was 79.3 in group I and 25.7 in group II. Dusejaet al[10] evaluated the possible association between nonalcoholic 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 non-significant. 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. Patients with hepatic disease showed a higher prevalence of periodontal disease, worse oral hygiene and periodontal health status compared to healthy individuals.

We found that mean calculus index simplified in group I was 31.3 and in group II was 50.2, debris index simplified in group I was 32.6 and in group II was 51.4, probing depth was 36.2 in group I and 46.1 in group II, bleeding on probing was 52.4 in group I and 31.8 in group II, clinical attachment loss was 53.9 in group I and 32.9 in group II and OHI- S was 30.2 in group I and 51.6 in group II.[11]Many cross-sectional and prospective epidemiological studies have indicated that periodontal disease is a risk factor for NAFLD. An in vivo animal model revealed that infection with periodontopathic bacteria accelerates the progression of NAFLD accompanied by enhanced steatosis. Moreover, the detection of periodontopathic bacteria in the liver may demonstrate that the bacteria have a direct impact on NAFLD. Furthermore, Porphyromonasgingivalis lipopolysaccharide induces inflammation and accumulation of intracellular lipids in hepatocytes. Th17 may be a key molecule for explaining the relationship between periodontal disease and NAFLD.[12]

CONCLUSION

Authors found that patients with NAFLD showed a higher prevalence of periodontal disease as compared to healthy individuals.

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