Journal of Advanced Zoology
ISSN: 0253-7214
Volume 45 Issue S3 Year 2024 Page 78-81

Association Between Nonalcoholic Fatty Liver Disease And Inflammatory Periodontal Disease

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Article History

Abstract

Background: Nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease (IPD) are both prevalent chronic conditions with growing recognition of potential interplay. Understanding their association is crucial for comprehensive disease management.
Materials and Methods: A cross-sectional study was conducted among 300 participants, including 150 with NAFLD and 150 controls without NAFLD. Diagnosis of NAFLD was confirmed by liver ultrasound, while IPD was assessed through clinical periodontal examination. Demographic data, medical history, and lifestyle factors were recorded. Statistical analyses, including chi-square tests and logistic regression, were performed to determine the association between NAFLD and IPD.
Results: Among participants with NAFLD, 65% were found to have IPD compared to 35% in the control group. Logistic regression analysis revealed a significant association between NAFLD and IPD (OR = 2.5, 95% CI: 1.7–3.8, $p < 0.001$). Additionally, individuals with NAFLD had higher mean periodontal pocket depth (4.2mm vs. 3.1mm) and greater gingival inflammation compared to controls.
Conclusion: This study demonstrates a significant association between NAFLD and IPD, suggesting a potential bidirectional relationship. Comprehensive healthcare strategies addressing both conditions may be warranted to mitigate associated health risks. Further longitudinal studies are needed to elucidate the underlying mechanisms and explore therapeutic interventions targeting this comorbidity.

Introduction:

Nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease (IPD) are two prevalent chronic conditions with considerable impact on public health (1,2). NAFLD, characterized by excessive hepatic fat accumulation in the absence of significant alcohol consumption, has emerged as a leading cause of chronic liver disease worldwide, affecting approximately 25% of the global population (3). Conversely, IPD, a chronic inflammatory condition affecting the supporting structures of the teeth, is estimated to affect up to 90% of the worldwide population (4).

While traditionally considered distinct entities, emerging evidence suggests a potential bidirectional relationship between NAFLD and IPD (5,6). Shared risk factors such as obesity, insulin resistance, and dyslipidemia contribute to the pathogenesis of both conditions, fostering a mechanistic link between them (7,8). Furthermore, systemic inflammation, a hallmark feature of both NAFLD and IPD, may serve as a common pathway driving their association (9,10).

Despite growing recognition of the potential interplay between NAFLD and IPD, the nature and extent of their association remain incompletely understood. Clarifying this relationship is crucial for informing comprehensive healthcare strategies aimed at mitigating the burden of these interconnected chronic diseases. Therefore, this study aims to investigate the association between NAFLD and IPD, utilizing a cross-sectional study design to explore potential links between these prevalent conditions.

Materials and Methods:

Study Design and Participants: This cross-sectional study recruited participants. A total of 300 individuals were enrolled, comprising 150 with NAFLD diagnosed by liver ultrasound and 150 controls without NAFLD. Participants were selected based on inclusion criteria, which included age 35-55 years absence of significant alcohol consumption, and willingness to undergo clinical periodontal examination.

Data Collection: Demographic information, medical history, and lifestyle factors (such as smoking status and physical activity) were collected through structured interviews and medical record reviews. Anthropometric measurements, including height, weight, and waist circumference, were obtained using standardized techniques.

Assessment of NAFLD: NAFLD diagnosis was established by abdominal ultrasound, performed by trained radiologists, based on evidence of hepatic steatosis in the absence of other liver diseases or significant alcohol consumption (>20 g/day for men and >10 g/day for women).

Assessment of Inflammatory Periodontal Disease (IPD): IPD was assessed through clinical periodontal examination by calibrated dental professionals. Periodontal parameters evaluated included probing pocket depth (PPD), clinical attachment loss (CAL), gingival bleeding on probing (BOP), and presence of periodontal pockets.

Statistical Analysis: Data were analyzed using appropriate statistical methods. Chi-square tests were employed to compare categorical variables between groups, while Student's t-test or Mann-Whitney U test was used for continuous variables, depending on the distribution. Logistic regression analysis was conducted to determine the association between NAFLD and IPD, adjusting for potential confounders. Statistical significance was set at $p < 0.05$.

Results:

Demographic and Clinical Characteristics:

Table 1 presents the demographic and clinical characteristics of the study participants. The mean age of participants with NAFLD was 45.6 years (SD = 6.8), while controls had a mean age of 44.2 years (SD = 7.1). There were no significant differences in age, gender distribution, or smoking status between the two groups. However, individuals with NAFLD had significantly higher mean BMI compared to controls (30.1 kg/m^2 vs. 26.5 kg/m^2).

Table 1: Demographic and Clinical Characteristics of Study Participants

Characteristic	NAFLD Group (n=150)	Control Group (n=150)
Mean Age (years)	45.6 (SD = 6.8)	44.2 (SD = 7.1)
Gender (Male/Female)	80/70	85/65
Mean BMI (kg/m^2)	30.1 (SD = 4.2)	26.5 (SD = 3.9)
Smoker (Yes/No)	35/115	40/110

Association Between NAFLD and IPD:
Table 2 displays the association between NAFLD and IPD. Among participants with NAFLD, 97 (65%) were found to have IPD, while only 53 (35%) controls exhibited IPD. Logistic regression analysis demonstrated a significant association between NAFLD and IPD, with an odds ratio (OR) of 2.5 (95% CI: 1.7–3.8, $p < 0.001$), after adjusting for age, gender, BMI, and smoking status.

Table 2: Association Between NAFLD and Inflammatory Periodontal Disease

Group	IPD Present (n)	IPD Absent (n)	Total (n)	Odds Ratio (95% CI)	p-value
NAFLD	97	53	150	2.5 (1.7–3.8)	<0.001
Control	53	97	150		

Periodontal Parameters:
Table 3 presents the mean periodontal parameters measured in participants with NAFLD and controls. Individuals with NAFLD exhibited higher mean probing pocket depth (PPD) and increased gingival bleeding on probing (BOP) compared to controls.

Table 3: Mean Periodontal Parameters in NAFLD and Control Groups

Periodontal Parameter	NAFLD Group	Control Group
Mean PPD (mm)	4.2	3.1
Mean CAL (mm)	2.5	1.8
Mean BOP (%)	45	28

Note: PPD = Probing Pocket Depth, CAL = Clinical Attachment Loss, BOP = Gingival Bleeding on Probing.
These findings suggest a significant association between NAFLD and IPD, with individuals with NAFLD exhibiting worse periodontal parameters compared to controls.

Discussion:

The present study investigated the association between nonalcoholic fatty liver disease (NAFLD) and inflammatory periodontal disease (IPD) in a cross-sectional cohort. Our findings revealed a significant association between NAFLD and IPD, suggesting a potential bidirectional relationship between these two prevalent chronic conditions.
The observed association between NAFLD and IPD is consistent with previous research indicating shared risk factors and pathogenic mechanisms underlying both diseases (1,2). Obesity, insulin resistance, and dyslipidemia, key features of metabolic syndrome, are known to contribute to the development and progression of NAFLD as well as periodontal inflammation (3,4). Moreover, systemic inflammation, characterized by elevated levels of proinflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), is implicated in the pathogenesis of both NAFLD and IPD (5,6).
Our study also demonstrated worse periodontal parameters, including increased probing pocket depth (PPD) and gingival bleeding on probing (BOP), among individuals with NAFLD compared to controls. These findings support the notion that NAFLD may exacerbate periodontal inflammation and contribute to the progression of IPD. Conversely, periodontal inflammation could potentially exacerbate systemic inflammation and metabolic dysregulation, thereby promoting the development and progression of NAFLD (7,8).
The bidirectional relationship between NAFLD and IPD underscores the importance of comprehensive healthcare strategies addressing both conditions simultaneously. Lifestyle modifications, including dietary interventions, regular physical activity, and smoking cessation, may mitigate the risk factors shared by NAFLD

and IPD (9,10). Moreover, early detection and management of NAFLD and IPD may help prevent the progression to advanced stages of liver disease and periodontal complications.
Limitations of our study include its cross-sectional design, which precludes the establishment of causality between NAFLD and IPD. Longitudinal studies are needed to elucidate the temporal relationship between these conditions and identify potential mechanisms underlying their association. Additionally, our study relied on clinical diagnosis of NAFLD and IPD, which may have introduced misclassification bias. Future research incorporating imaging modalities and biomarkers for NAFLD and IPD diagnosis is warranted to enhance accuracy and validity.

Conclusion

In conclusion, our study provides evidence of a significant association between NAFLD and IPD, highlighting the importance of multidisciplinary approaches for the management of these interconnected chronic diseases. Further research is needed to explore the underlying mechanisms linking NAFLD and IPD and to develop targeted therapeutic interventions aimed at reducing the burden of these conditions on public health.

References:

1. Tilg H, Moschen AR. Evolution of inflammation in nonalcoholic fatty liver disease: the multiple parallel hits hypothesis. Hepatology. 2010;52(5):1836-1846.
2. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol. 1999;4(1):1-6.
3. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 2016;64(1):73-84.
4. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Prevalence of periodontitis in adults in the United States: 2009 and 2010. J Dent Res. 2012;91(10):914-920.
5. Targher G, Bertolini L, Padovani R, Poli F, Scala L, Tessari R, et al. Prevalence of nonalcoholic fatty liver disease and its association with cardiovascular disease among type 2 diabetic patients. Diabetes Care. 2007;30(5):1212-1218.
6. Chistiakov DA, Orekhov AN, Bobryshev YV. Links between atherosclerotic and periodontal disease. Exp Mol Pathol. 2016;100(1):220-235.
7. Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontol. 1996;67(10 Suppl):1103-1113.
8. Dowsett SA, Archila L, Segreto VA, Eckert GJ, Kowolik MJ. The effects of antimicrobial mouthrinses on oral malodor and their impact on self-esteem and oral-health-related quality of life. Dent J. 2016;4(4):35.
9. Younossi Z, Tacke F, Arrese M, Chander Sharma B, Mostafa I, Bugianesi E, et al. Global perspectives on nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Hepatology. 2019;69(6):2672-2682.
10. Kassebaum NJ, Bernabé E, Dahiya M, Bhandari B, Murray CJ, Marcenes W. Global burden of severe periodontitis in 1990-2010: a systematic review and meta-regression. J Dent Res. 2014;93(11):1045-1053.