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ORIGINAL RESEARCH

Evaluation of Inflammatory Biomarkers in Obese Individuals with and without Non-Alcoholic Fatty Liver Disease: A Cross-Link between Gastroenterology and Endocrinology

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Abstract

Background: Non-Alcoholic Fatty Liver Disease (NAFLD) is a common hepatic manifestation of metabolic dysfunction, particularly among obese individuals. Systemic inflammation plays a pivotal role in the progression of NAFLD. Inflammatory biomarkers such as Interleukin-6 (IL-6), C-Reactive Protein (CRP), and Tumor Necrosis Factor-alpha (TNF-α) have been identified as potential indicators of hepatic and metabolic alterations. This study aims to evaluate and compare levels of inflammatory biomarkers in obese individuals with and without NAFLD, highlighting the intersection of gastroenterology and endocrinology.

Materials and Methods:A cross-sectional analytical study was conducted on 100 obese individuals (BMI ≥30 kg/m²) aged 25–60 years. Participants were divided into two groups: Group A (n=50) with ultrasonographically confirmed NAFLD and Group B (n=50) without NAFLD. Fasting blood samples were collected for analysis of IL-6, CRP, and TNF-α levels using ELISA. Clinical parameters including BMI, waist circumference, fasting blood glucose, lipid profile, and liver function tests were also assessed. Statistical analysis was performed using SPSS version 26.0 with $p<0.05$ considered statistically significant.

Results: Mean IL-6 levels were significantly higher in Group A (8.42 ± 2.3 pg/mL) compared to Group B (4.15 ± 1.1 pg/mL; $p<0.001$). CRP levels in Group A averaged 6.88 ± 1.9 mg/L versus 3.14 ± 1.2 mg/L in Group B ($p<0.01$). Similarly, TNF-α levels were elevated in Group A (11.53 ± 2.6 pg/mL) compared to Group B (6.75 ± 1.7 pg/mL; $p<0.001$). A positive correlation was observed between inflammatory biomarkers and liver enzyme levels (ALT and AST) in the NAFLD group.

Conclusion: Obese individuals with NAFLD exhibit significantly elevated inflammatory markers compared to those without hepatic steatosis. These findings support the hypothesis that low-grade chronic inflammation serves as a pathogenic link between metabolic dysfunction and hepatic injury. Early identification and targeted anti-inflammatory interventions may help in halting NAFLD progression and associated endocrine complications.

Keywords: Non-Alcoholic Fatty Liver Disease, Obesity, Inflammatory Biomarkers, Interleukin-6, C-Reactive Protein, Tumor Necrosis Factor-alpha, Metabolic Syndrome, Gastroenterology, Endocrinology.

Introduction

Non-Alcoholic Fatty Liver Disease (NAFLD) represents a spectrum of liver disorders characterized by excessive hepatic fat accumulation in the absence of significant alcohol consumption. It is now recognized as the most common cause of chronic liver disease globally, particularly among obese individuals and those with metabolic syndrome (1). The prevalence of NAFLD continues to rise in parallel with the global obesity epidemic and has become a major public health concern due to its potential progression to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma (2,3). The pathogenesis of NAFLD is multifactorial, involving insulin resistance, dyslipidemia, oxidative stress, and chronic low-grade inflammation (4).

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Among these, systemic inflammation is considered a critical mediator linking metabolic disturbances and liver injury. Pro-inflammatory cytokines such as interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor-alpha (TNF-α) have been implicated in hepatic inflammation and fibrosis (5,6). Elevated levels of these biomarkers are frequently observed in individuals with obesity and metabolic disorders, suggesting their potential role in the pathophysiology and progression of NAFLD (7).

Obesity, especially visceral adiposity, is a well-established risk factor for NAFLD and contributes significantly to the secretion of inflammatory mediators from adipose tissue, which exacerbate hepatic steatosis and insulin resistance (8). This systemic inflammatory response acts as a common ground between endocrinology and gastroenterology, emphasizing the need for a cross-disciplinary approach in the assessment and management of NAFLD in obese populations (9).

The present study aims to evaluate the levels of inflammatory biomarkers in obese individuals with and without NAFLD, exploring the potential of these markers as non-invasive indicators of liver involvement. Understanding the inflammatory profile in these patients may aid in early diagnosis, risk stratification, and targeted therapeutic strategies to mitigate disease progression (10).

Materials and Methods

A total of 100 obese individuals (BMI ≥30 kg/m²) aged between 25 and 60 years were enrolled after obtaining informed written consent. Participants were divided into two groups: Group A included 50 obese individuals with ultrasonographically confirmed NAFLD, and Group B included 50 obese individuals without any sonographic evidence of fatty liver. Exclusion criteria included patients with a history of alcohol consumption >20 g/day, viral hepatitis, autoimmune liver disease, use of hepatotoxic drugs, or other chronic illnesses.

All participants underwent a detailed clinical evaluation, including anthropometric measurements (weight, height, BMI, waist circumference). Fasting blood samples were collected and analyzed for inflammatory biomarkers, namely Interleukin-6 (IL-6), C-Reactive Protein (CRP), and Tumor Necrosis Factor-alpha (TNF-α), using standardized ELISA kits as per manufacturer instructions. Additional laboratory investigations included liver function tests (ALT, AST), lipid profile, and fasting blood glucose.

Abdominal ultrasonography was performed by a trained radiologist using a high-resolution B-mode ultrasound scanner to assess the presence and grade of hepatic steatosis. NAFLD was diagnosed based on increased echogenicity of the liver parenchyma, blurring of vascular margins, and deep attenuation.

Statistical analysis was performed using IBM SPSS software version 26.0. Continuous variables were presented as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. The independent sample t-test was used to compare means between the two groups. Pearson correlation analysis was used to evaluate associations between inflammatory biomarkers and liver enzymes. A p-value <0.05 was considered statistically significant.

Results

A total of 100 obese participants were included in the study, divided equally into Group A (with NAFLD) and Group B (without NAFLD). The mean age of participants in Group A was 44.3 ± 7.8 years, while in Group B it was 42.6 ± 6.9 years. Both groups had a comparable gender distribution, with a slight female predominance in each group.

Inflammatory Biomarkers

The mean levels of inflammatory biomarkers were significantly higher in the NAFLD group compared to the non-NAFLD group. Interleukin-6 (IL-6) levels in Group A averaged 8.42 ± 2.3 pg/mL, while Group B showed a mean of 4.15 ± 1.1 pg/mL (p<0.001). Similarly, C-Reactive Protein (CRP) levels were 6.88 ± 1.9 mg/L in Group A and 3.14 ± 1.2 mg/L in Group B (p<0.01). Tumor Necrosis Factor-alpha (TNF-α) also demonstrated a significant elevation in the NAFLD group (11.53 ± 2.6 pg/mL) compared to the control group (6.75 ± 1.7 pg/mL) (p<0.001) (Table 1).

Liver Enzymes and Correlation

Liver enzyme levels (ALT and AST) were higher in Group A, with ALT at 59.8 ± 12.5 U/L versus 32.6 ± 10.4 U/L in Group B (p<0.01), and AST at 45.2 ± 9.8 U/L versus 28.7 ± 8.3 U/L (p<0.01). A positive correlation was observed between IL-6 and ALT (r = 0.63, p<0.001), as well as TNF-α and AST (r = 0.58, p<0.01) in the NAFLD group (Table 2).

Table 1: Comparison of Inflammatory Biomarkers Between Groups

Biomarker	Group A (NAFLD) Mean ± SD	Group B (Non-NAFLD) Mean ± SD	*p*-value
IL-6 (pg/mL)	8.42 ± 2.3	4.15 ± 1.1	<0.001
CRP (mg/L)	6.88 ± 1.9	3.14 ± 1.2	<0.01
TNF-α (pg/mL)	11.53 ± 2.6	6.75 ± 1.7	<0.001

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Table 2: Liver Enzyme Levels and Correlation with Inflammatory Biomarkers (Group A)

Parameter Pair	Correlation Coefficient (r)	*p*-value
IL-6 vs ALT	0.63	<0.001
TNF-α vs AST	0.58	<0.01
CRP vs ALT	0.49	<0.05

These findings indicate that inflammatory markers are significantly elevated in obese individuals with NAFLD and correlate strongly with liver enzyme levels (Tables 1 and 2).

Discussion

This study aimed to evaluate the levels of inflammatory biomarkers—IL-6, CRP, and TNF-α—in obese individuals with and without NAFLD, highlighting the inflammatory link bridging gastroenterology and endocrinology. The findings demonstrated significantly elevated levels of all three markers in individuals with NAFLD compared to their non-NAFLD obese counterparts. These results reinforce the role of chronic low-grade systemic inflammation in the pathogenesis of NAFLD.

NAFLD has been increasingly recognized as the hepatic manifestation of metabolic syndrome and is closely associated with insulin resistance, obesity, dyslipidemia, and type 2 diabetes mellitus (1,2). Adipose tissue, particularly visceral fat, acts as an active endocrine organ that secretes numerous pro-inflammatory cytokines such as IL-6 and TNF-α, which in turn contribute to hepatic steatosis, hepatocellular injury, and fibrogenesis (3,4). Our observation of elevated IL-6 in the NAFLD group aligns with previous reports indicating that IL-6 promotes hepatic insulin resistance and steatosis by disrupting insulin signaling pathways (5,6).

CRP, an acute-phase protein synthesized in the liver in response to IL-6 stimulation, is widely used as a marker of systemic inflammation. Elevated CRP levels in NAFLD patients observed in our study further corroborate previous evidence that CRP is not only a marker but also a participant in disease progression, potentially promoting endothelial dysfunction and hepatic inflammation (7,8). Studies have demonstrated that higher CRP concentrations are predictive of advanced fibrosis in NAFLD (9,10).

TNF-α, a key inflammatory cytokine produced by macrophages and adipocytes, plays a pivotal role in hepatic fat accumulation and hepatocellular apoptosis (11). Increased TNF-α levels in our study support existing data that associate it with hepatic inflammation and fibrogenesis in NAFLD patients (12). Additionally, TNF-α interferes with adiponectin signaling and promotes insulin resistance, further exacerbating hepatic lipid accumulation (13).

The correlation observed between inflammatory biomarkers and liver enzymes (ALT and AST) suggests that these cytokines are involved in liver injury and may serve as non-invasive markers for hepatic inflammation. This finding is consistent with prior studies reporting that elevated transaminases reflect ongoing hepatocellular damage mediated by inflammatory processes in NAFLD (14,15).

The clinical implications of this study are significant, as they highlight the potential utility of IL-6, CRP, and TNF-α as diagnostic and prognostic indicators in obese individuals at risk for NAFLD. Early detection of elevated inflammatory markers may prompt further hepatic evaluation and targeted lifestyle or pharmacological interventions aimed at reducing systemic inflammation and preventing disease progression.

However, this study has some limitations. The cross-sectional design restricts causal interpretation, and the sample size, while adequate for preliminary evaluation, may not reflect the broader population. Liver biopsy, the gold standard for NAFLD diagnosis and staging, was not employed due to its invasiveness; instead, ultrasonography was used for screening hepatic steatosis, which may have limited sensitivity in detecting mild cases.

Conclusion

Obese individuals with NAFLD exhibit significantly elevated levels of inflammatory biomarkers such as IL-6, CRP, and TNF-α compared to those without hepatic steatosis. These findings highlight the central role of systemic inflammation in the pathogenesis of NAFLD and underscore the potential of these markers as non-invasive indicators for early detection and risk assessment.

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