Evaluation of Serum Adiponectin and Resistin Biomarkers in Patients with Non-Alcoholic Fatty Liver Disease

Dr. Rajesh Chahar, Dr. Sandeep Tripathi, Dr. Vinita Ailani, Dr. Sunita Saini

1. Ph.D. Scholar, Department of Medical Biochemistry, National Institute of Medical Sciences & Research, Jaipur, Rajasthan, Email id: rajesh9460@gmail.com
2. Professor, Department of Medical Biochemistry, National Institute of Medical Sciences & Research, Jaipur, Rajasthan, Email ID: sandeeptripathiphd@gmail.com
3. Professor, Department of Physiology, National Institute of Medical Sciences & Research, Jaipur, Rajasthan, Email id: ailanivinita@gmail.com
4. Associate Professor, Department of Pathology, Shri Kalyan Govt. Medical College, Sikar, Rajasthan, Email id: drvisusaini8615@gmail.com

Corresponding Author
Dr. Rajesh Chahar
Ph.D. scholar
Department of Medical Biochemistry,
National Institute of Medical Sciences & Research, Jaipur, Rajasthan,
Email id: rajesh9460@gmail.com
Contact No.9468604396

Abstract

Background:
Non-Alcoholic Fatty Liver Disease (NAFLD) is a major cause of chronic liver disease worldwide and is closely associated with obesity, insulin resistance, and metabolic syndrome. Adipokines, including adiponectin and resistin, have been implicated in the pathogenesis and progression of NAFLD and may serve as potential biomarkers of disease activity.

Objective:
To evaluate serum adiponectin and resistin levels as potential biomarkers and to determine their association with liver enzyme concentrations in patients with Non-Alcoholic Fatty Liver Disease.

Methods:
This cross-sectional observational study was conducted among 50 adult patients (aged 18–65 years) with ultrasonography-confirmed NAFLD. Serum adiponectin and resistin levels were measured by enzyme-linked immunosorbent assay (ELISA). Liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT), were estimated by kit methods. Correlations between adipokines and liver enzymes were assessed using Pearson's correlation coefficient. Statistical analysis was performed using SPSS software, and a p-value <0.05 was considered statistically significant.

Results:
The mean age of the study participants was 44.7 ± 10.9 years, and the mean BMI was 28.9 ± 3.8 kg/m². Mean serum adiponectin and resistin levels were 5.4 ± 2.1 µg/mL and 9.8 ± 3.2 ng/mL, respectively. Mean ALT, AST, and GGT levels were 63.2 ± 18.5 U/L, 47.1 ± 14.8 U/L, and 38.6 ± 18.4 U/L, respectively. Serum adiponectin showed significant negative correlations with ALT ($r = -0.45$, $p = 0.001$), AST ($r = -0.39$, $p = 0.005$), and GGT ($r = -0.48$, $p < 0.001$). In contrast, resistin demonstrated significant positive correlations with ALT ($r = 0.51$, $p < 0.001$), AST ($r = 0.43$, $p = 0.002$), and GGT ($r = 0.49$, $p < 0.001$).

Conclusion:
Altered adipokine profiles characterized by decreased adiponectin and increased resistin levels are significantly associated with elevated liver enzyme concentrations in patients with NAFLD. These findings suggest that adiponectin and resistin may serve as promising biomarkers for assessing and monitoring hepatic injury in NAFLD.

Keywords: Non-Alcoholic Fatty Liver Disease, Adiponectin, Resistin, Adipokines, Biomarkers, Liver Enzymes.
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Introduction

Non-Alcoholic Fatty Liver Disease (NAFLD) is the most prevalent chronic liver disease worldwide, affecting approximately 25–30% of the adult population and posing a significant public health burden. It is characterized by excessive accumulation

of triglycerides within hepatocytes in the absence of significant alcohol consumption. It encompasses a spectrum of liver disorders ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma (1,2). The pathogenesis of NAFLD is complex and multifactorial, involving insulin resistance, oxidative stress, mitochondrial dysfunction, lipid accumulation, and chronic inflammation. The currently accepted "multiple-hit" hypothesis suggests that metabolic, genetic, environmental, and inflammatory factors act simultaneously to promote hepatic steatosis and disease progression (3,4). NAFLD is strongly associated with obesity, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome, highlighting the importance of adipose tissue dysfunction in its development (5).

Adipose tissue is increasingly recognized as an active endocrine organ that secretes numerous bioactive peptides known as adipokines. These adipokines regulate glucose homeostasis, lipid metabolism, insulin sensitivity, and inflammatory responses. Dysregulation of adipokine secretion has been implicated in the pathogenesis of several metabolic disorders, including NAFLD (6,7).

Adiponectin is an anti-inflammatory and insulin-sensitizing adipokine that exerts protective effects on the liver by enhancing fatty acid oxidation and reducing hepatic inflammation. Several studies have demonstrated decreased circulating adiponectin levels in patients with NAFLD, and lower concentrations have been associated with increased disease severity (8–10). In contrast, resistin is a pro-inflammatory adipokine that promotes insulin resistance and inflammatory cytokine production. Elevated serum resistin levels have been reported in patients with NAFLD and have been linked to hepatic inflammation and fibrosis (11,12).

Although liver biopsy remains the gold standard for the diagnosis and staging of NAFLD, its invasive nature, sampling variability, and risk of complications limit its routine use. Therefore, the identification of reliable non-invasive biomarkers for early detection and monitoring of NAFLD is of considerable clinical importance (13,14).

In view of the emerging role of adipokines in metabolic and hepatic disorders, the present study was undertaken to evaluate serum adiponectin and resistin levels and to investigate their relationship with liver enzyme concentrations in patients with NAFLD. The findings may provide insight into the utility of these adipokines as potential non-invasive biomarkers for the assessment and monitoring of hepatic injury in NAFLD.

Materials and Methods

Study Design and Setting

This cross-sectional observational study was conducted in the Department of Medical Biochemistry, NIMS University, Jaipur, in collaboration with the Department of General Medicine at S. K. Medical College, Sikar, Rajasthan. The study was carried out after obtaining approval from the Institutional Ethics Committee, and informed written consent was obtained from all participants before enrollment.

Study Population

A total of 50 adult patients aged between 18 and 65 years diagnosed with Non-Alcoholic Fatty Liver Disease (NAFLD) were included in the study. The diagnosis of NAFLD was established based on clinical evaluation, laboratory investigations, and ultrasonographic evidence of hepatic steatosis. Patients aged 18–65 years and an ultrasonography-confirmed diagnosis of NAFLD were included in this study. History of significant alcohol consumption, Viral hepatitis (Hepatitis B or Hepatitis C), Autoimmune liver diseases, Drug-induced liver disease, Pregnancy, Patients with malignancy, chronic kidney disease, or severe systemic illness were excluded from the study

Data Collection

Demographic and clinical data, including age, gender, body weight, height, body mass index (BMI), blood pressure, smoking status, alcohol consumption, and relevant medical history, were recorded using a pre-designed case record form.

Body Mass Index (BMI) was calculated using the formula:

BMI (kg/m²) = Weight (kg) / Height² (m²)

Sample Collection and Analysis:

After an overnight fasting period of 8–12 hours, approximately 5 mL of venous blood was collected under aseptic conditions from each participant. Blood samples were allowed to clot and centrifuged at 3000 rpm for 10 minutes to separate serum. The serum samples were aliquoted and stored at −80°C until analysis. Serum liver enzyme concentrations were estimated using standard enzymatic methods on a fully automated clinical chemistry analyzer: Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), and Gamma-Glutamyl Transferase (GGT). Serum adiponectin and resistin concentrations were measured using commercially available Enzyme-Linked Immunosorbent Assay (ELISA) kits according to the manufacturer's instructions.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The association between serum

adipokines and liver enzymes was assessed using Pearson's correlation coefficient (r). A p-value <0.05 was considered statistically significant.

Results: A total of 50 patients with ultrasonography-confirmed NAFLD were included in the study. Demographic characteristics, anthropometric measurements, serum adipokine concentrations, and liver enzyme levels were analyzed. The associations between adipokines and liver enzymes were further evaluated using Pearson's correlation analysis. Table 1 demonstrates that the mean age of the study participants was 44.7 ± 10.9 years. Male participants constituted 60% of the study population, while females accounted for 40%. The mean BMI was 28.9 ± 3.8 kg/m², indicating that most participants were overweight or obese, which is a recognized risk factor for NAFLD

Table 1. Demographic and Anthropometric Characteristics of NAFLD Patients (n = 50)

Parameter	**Mean**± SD
Age (years)	44.7 ± 10.9
Gender (Male), n (%)	30 (60.0)
Gender (Female), n (%)	20 (40.0)
BMI (kg/m²)	28.9 ± 3.8

In Table 2 observed that Serum adiponectin levels were relatively low, whereas resistin concentrations were elevated among NAFLD patients. These findings suggest alterations in adipokine secretion, which may contribute to insulin resistance, inflammation, and hepatic injury associated with NAFLD.

Table 2. Serum Adipokine Levels in NAFLD Patients (n = 50)

Adipokine	**Mean ± SD**	**Minimum**	**Maximum**
Adiponectin (µg/mL)	5.4 ± 2.1	2.1	10.8
Resistin (ng/mL)	9.8 ± 3.2	4.5	17.6

Table 3 indicates that the mean ± SD serum ALT, AST, and GGT levels among NAFLD patients were 63.2 ± 18.5 U/L, 47.1 ± 14.8 U/L, and 38.6 ± 18.4 U/L, respectively. The mean serum ALT, AST, and GGT levels were elevated among NAFLD patients. Increased liver enzyme concentrations indicate hepatocellular injury and metabolic dysfunction. The elevated GGT level further supports the presence of hepatic steatosis and oxidative stress, which are commonly observed in patients with NAFLD.

Table 3. Serum Enzyme Levels in NAFLD Patients (n = 50)

Parameter	**Mean ± SD**
ALT (U/L)	63.2 ± 18.5
AST (U/L)	47.1 ± 14.8
GGT (U/L)	38.6 ± 18.4

Table 4 revealed that Pearson's correlation analysis demonstrated significant negative correlations between adiponectin and liver enzymes (ALT, AST, and GGT), indicating that lower adiponectin levels were associated with greater hepatic injury. In contrast, resistin showed significant positive correlations with all liver enzymes, suggesting that higher resistin concentrations are associated with increased liver dysfunction and disease severity in NAFLD patients.

Table 4. Correlation of Serum Adipokines with Liver Enzymes in NAFLD Patients (n = 50)

Adipokine	**ALT (r)**	**p-value**	**AST (r)**	**p-value**	**GGT (r)**	**p-value**
Adiponectin	-0.45	0.001	-0.39	0.005	-0.48	<0.001
Resistin	0.51	<0.001	0.43	0.002	0.49	<0.001

** P<0.001 is significant

Discussion

The present study evaluated the association of serum adiponectin and resistin levels with liver enzyme concentrations in patients with Non-Alcoholic Fatty Liver Disease (NAFLD). The findings demonstrated that reduced adiponectin levels and elevated resistin concentrations were significantly associated with increased ALT, AST, and GGT levels, suggesting a

potential role of these adipokines in hepatic injury and disease progression.

Adiponectin is an adipocyte-derived cytokine with anti-inflammatory, anti-atherogenic, and insulin-sensitizing properties. In the present study, serum adiponectin levels were decreased and showed significant negative correlations with ALT, AST, and GGT concentrations. These findings are consistent with previous studies by Pagano et al. and Hui et al., who reported lower circulating adiponectin levels in patients with NAFLD and NASH compared with healthy controls (15,16). Adiponectin has been shown to inhibit hepatic lipid accumulation, reduce oxidative stress, and suppress inflammatory cytokine production. Therefore, reduced adiponectin concentrations may contribute to hepatocellular injury and progression of fatty liver disease (17). Furthermore, a meta-analysis by Polyzos et al. confirmed that hypoadiponectinemia is significantly associated with the presence and severity of NAFLD (18).

In contrast, serum resistin levels were elevated in the study population and demonstrated significant positive correlations with liver enzymes. Resistin is recognized as a pro-inflammatory adipokine that promotes insulin resistance and stimulates the production of inflammatory mediators such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). Elevated resistin levels observed in the present study support the findings of Yagmur et al. and Senates et al., who reported increased circulating resistin concentrations in patients with NAFLD and chronic liver disease (19,20). Experimental studies have suggested that resistin may directly contribute to hepatic inflammation by activating hepatic stellate cells and promoting fibrogenic pathways (21). The positive correlation between resistin and liver enzymes observed in the current study further supports its potential role as a marker of hepatic injury.

The significant correlations between adipokines and liver enzymes observed in the present study highlight the close interplay between adipose tissue dysfunction and hepatic injury. Elevated ALT, AST, and GGT levels are established biochemical markers of hepatocellular damage, and their association with altered adipokine profiles suggests that these molecules may reflect underlying disease activity. Since liver biopsy remains invasive and unsuitable for routine monitoring, adipokines may serve as useful non-invasive biomarkers for assessing NAFLD severity and progression (22).

The present study has certain limitations. The relatively small sample size and cross-sectional design limit the ability to establish causal relationships. Additionally, a liver biopsy was not performed to correlate adipokine concentrations with histological severity. Nevertheless, the study provides valuable evidence supporting the association between adipokines and hepatic dysfunction in NAFLD patients.

Conclusion: In conclusion, decreased adiponectin and increased resistin levels were significantly associated with elevated liver enzyme concentrations in NAFLD patients. These findings suggest that adipokines may serve as promising non-invasive biomarkers for assessing and monitoring hepatic injury in NAFLD. Further large-scale prospective studies are required to validate their diagnostic and prognostic utility.

Conflict of Interest: None

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Author Contribution: Dr. Rajesh Chahar, Conceptualization, methodology, data analysis, manuscript drafting, and supervision. All authors contributed to data collection, interpretation of results, critical revision of the manuscript, and approved the final version.

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