KARBALA JOURNAL OF MEDICINE

Research Article

Correlation of Liver Enzymes with Ultrasound Grading in Patients with Non-alcoholic Fatty Liver

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Article information:
Received: 25-05-2025
Accepted: 30-06-2025
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https://doi.org/10.70863/karbalajm.v18i1.3855

Abstract

Background: Nonalcoholic fatty liver disease (NAFLD) is a leading cause of liver disease worldwide and is projected to become the foremost cause of end-stage liver disease, affecting both adults and children. This study evaluated the association between ultrasound-based NAFLD grading and changes in liver enzymes: alanine aminotransferase (ALT), aspartate aminotransferase (AST), total serum bilirubin (TSB), triglycerides (TG), and cholesterol (CH).

Methods: A case-control study was conducted between December 2024 and March 2025, involving 176 participants. The study population was divided into two groups: 100 patients diagnosed with NAFLD and 76 healthy individuals serving as controls. Diagnosis and grading of NAFLD (mild, moderate, and severe) were based on ultrasonography and biochemistry analysis.

Results: Biochemical analysis revealed significant differences between NAFLD patients and healthy controls, as well as across the different NAFLD grades. ALT, AST, TSB, TG, and CH levels were significantly elevated in NAFLD patients compared to healthy controls ($p = 0.001$ for all parameters). A progressive increase in these biochemical markers was observed as disease severity advanced. Among the NAFLD grades, grade 2 was the most commonly observed. Post-hoc Least Significant Difference (LSD) tests confirmed significant differences in these parameters between all NAFLD grades ($p = 0.05$), indicating worsening hepatic and metabolic dysfunction throughout disease progression.

Conclusions: Serum levels of ALT, AST, TSB, TG and CH showed a positive correlation with NAFLD severity, reflecting the underlying pathophysiological progression of the disease. These biochemical markers may serve as valuable adjuncts to non-invasive diagnostic tools such as ultrasound, which is commonly used for initial detection and grading of hepatic steatosis. When used alongside advanced modalities like transient elastography (FibroScan), they can enhance disease staging accuracy and support more targeted clinical management strategies.

Keywords: NAFLD, AST, ALT, bilirubin, triglyceride, cholesterol

Introduction

Nonalcoholic fatty liver disease (NAFLD) is one of the most important causes of liver disease worldwide and will probably emerge as the leading cause of end-stage liver disease in the coming decades, with the disease affecting both adults and children [1]. It can be subdivided into nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH). Five to twenty percent of cases progress from NAFL to NASH [2]. Patients with NASH have a higher risk of progressing to liver cirrhosis compared to those with NAFL without inflammation or liver cell injury, who generally have a stable form of the disease. NAFLD is characterized by the accumulation of fats in the liver. Relatively benign NAFLD often progresses to fibrosis, cirrhosis, and liver malignancies. Although NAFLD precedes fibrosis, continuous lipid overload keeps fueling fibrosis, and the process of disease progression remains unhindered. Patients with NASH are at a significantly higher risk of developing hepatocellular carcinoma (HCC). NASH is currently the third most common cause of HCC [3]. In contrast, HCC is rarely reported in patients with isolated NAFLD [4-5].

NAFLD has emerged as a major challenge because of it prevalence, difficulties in diagnosis, complex

pathogenesis, and lack of approved therapies [6-7]. As the burden of hepatitis C abates over the next decade, non-alcoholic fatty liver disease will become the major form of chronic liver disease in adults and children and could become the leading indication for liver transplantation [8]. NAFLD is the leading cause of diffuse liver disease, with a global prevalence of 25.24% and progresses to fibrosis and NASH. Diagnosing NAFLD requires demonstration of increased liver fat, and ultrasound imaging is widely used for screening. Specific blood tests to diagnose NAFLD and NASH are not yet available, and alanine aminotransaminase (ALT) has been used as a marker in population-based studies [9]. The ALT is generally found in serum and bodily tissues, particularly the liver. Since it is released into the serum as a result of tissue damage, acute damage to hepatic cells may result in a rise in the blood concentration of ALT [10]. In clinical practice, biochemical markers such as ALT, aspartate aminotransferase (AST), total serum bilirubin (TSB), triglycerides (TG), and cholesterol (CH) are frequently assessed in patients with suspected NAFLD. These markers may reflect liver injury and metabolic dysfunction, but their utility in staging NAFLD remains under investigation.

This study aims to evaluate how serum levels of ALT, AST, TSB, TG, and CH vary across ultrasound-defined grades of NAFLD, and to determine whether these markers can reliably reflect disease severity.

Materials and Methods

Study design and patients

A case-control study was conducted at Imam Al-Hussein Medical City Hospital and the Karbala Centre for Digestive and Liver Diseases and Surgery in Karbala City in Iraq, focusing on patients presenting with fatty liver disease from December 2024 to March 2025. This study included 176 samples, including 100 patients with NAFLD. Among the NAFLD patients, 61 were males and 39 were females, with a mean age of 52 ± 6 years. The second group consisted of 76 healthy individuals who served as the control group. This group included 42 males and 34 females, with a mean age of 42 ± 7. All participants in the control group had normal abdominal ultrasound findings and no history of liver disease, metabolic syndrome, or significant alcohol intake.

Biochemical tests

Serum levels of ALT, AST, TSB, TG, and CH were measured using an automated chemistry analyzer (ABBOTT ARCHITECT c4000, Abbott Laboratories, USA). Normal reference ranges in Table 1.

Table 1: Normal reference ranges of biochemical tests

Parameter	Normal range
Alanine Aminotransferase (ALT)	7–56 U/L
Aspartate Aminotransferase (AST)	10–40 U/L
Total Serum Bilirubin	0.3–1.2 mg/dL
Triglycerides	<150 mg/dL
Total Cholesterol	<200 mg/dL

Ultrasound Grading Systems

Ultrasound grading of NAFLD was performed using abdominal ultrasonography, a non-invasive imaging method widely used to detect and evaluate hepatic steatosis. The liver was assessed in comparison to the right kidney, using transverse and longitudinal views obtained via a right upper quadrant scan.

NAFLD was classified into four grades based on echogenicity and structural visualization [11]:

Grade 0: normal liver echogenicity, grade 1 or mild steatosis: slight, diffuse increase in liver echogenicity with normal visualization of the diaphragm and intrahepatic vessels.

Grade 2 or moderate steatosis: moderate increase in echogenicity with slightly impaired visualization of the diaphragm and intrahepatic vessels.

Grade 3 or severe steatosis: marked increase in echogenicity, poor penetration of the posterior liver segment, and poor or no visualization of the diaphragm and intrahepatic vessels.

This grading is based on four main criteria: Liver brightness compared to the kidney, visualization of the diaphragm, intrahepatic vessel clarity, and deep attenuation of the ultrasound signal, which limits the penetration of sound waves and obscures deeper liver structure,s is also a key feature considered in the grading of hepatic steatosis.

Inclusion Criteria

Patients included in this study were those with nonalcoholic fatty liver disease (NAFLD) through ultrasound (US) and clinical assessment by physicians. The study enrolled both male and female participants, with an age restriction of 18 years or older. Additionally, healthy individuals without any prior history of NAFLD were selected as controls, matching the patient group in terms of age and sex to ensure comparability between the two groups.

Exclusion Criteria

Individuals with alcoholic fatty liver disease, inflammatory bowel disease, or autoimmune

diseases, immunocompromised status, malignancy, and current signs or symptoms of infection.

Ethical approval

All research participants were informed and allowed to give their consent before sample collection. This study adhered to ethical standards, with approval from the Institutional Review Board (IRB) of Karbala Health Directorate (Ethical No. 4031 in 2024), and all participants provided informed consent. The authors certify that they have obtained all appropriate patient consent forms. In the forms, the patients have given their consent for their clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity.

Statistical analysis

The statistical analysis was carried out using the Statistical Package for Social Sciences (SPSS, IBM, version 26.0). The data distribution's normality was assessed using the Shapiro-Wilk and Kolmogorov-Smirnov tests. Mean ± standard deviation (SD) is a representation of continuous data having a normal distribution. One-way analysis of variance (ANOVA) was used to compare biochemical parameters among NAFLD grades and between NAFLD patients and healthy controls. Post-hoc comparisons were performed using the Least Significant Difference (LSD) test to determine pairwise differences between groups. A p-value of < 0.05 was considered statistically significant.

Results

The biochemical analysis revealed significant differences between the NAFLD patients and the healthy control group. Mean serum AST levels were markedly elevated in NAFLD patients compared to controls (p=0.001). Similarly, ALT levels were significantly higher in NAFLD subjects than in controls (p= 0.001). TSB was also elevated among NAFLD patients in contrast to the control group (p= 0.001). Lipid profile abnormalities were evident, with TG levels nearly doubling in NAFLD patients relative to controls (p=0.000). CH concentrations were also significantly increased in the NAFLD group compared to the control group (p=0.000) as detailed in Table 2.

The results demonstrate significant biochemical and metabolic variations across different grades of NAFLD, highlighting progressive hepatic injury and dyslipidemia with advancing disease severity. Patients with NAFLD, categorized as grade 1 (46 patients), grade 2 (38 patients), and grade 3 (16 patients) based on abdominal ultrasound results. Liver enzymes AST and ALT showed a marked stepwise elevation from grade 1 to grade 3 (AST: 16.41±6.98 to 29.63±9.69 U/L, p =0.000; ALT: 20.44±8.75 to 38.69±11.52 U/L, p=0.000), indicating worsening hepatocellular damage. TSB levels nearly doubled between grade 1 and grade 3 (0.49±0.19 to 0.90±0.29 mg/dL, p=0.000), suggesting impaired hepatic excretory function in advanced disease. Lipid parameters revealed significant grade-dependent increases, with TG rising from 174.70±75.30 mg/dL in grade 1 to 221.06±86.19 mg/dL in grade 3 (p =0.014), and CH levels escalating from grade 1; 189.54±57.84 mg/dL to grade 3; 266.69±81.21 mg/dL (p=0.002) (Table 3). Post-hoc LSD tests confirmed significant differences between all grade comparisons for these parameters (p= 0.04).

These findings collectively demonstrate that NAFLD progression is characterized by the progressive hepatocyte injury evidenced by rising AST/ALT, worsening metabolic dysfunction as reflected in lipid profile derangements, and declining hepatic synthetic/excretory capacity as indicated by bilirubin elevation.

Discussion

This study demonstrated a significant and progressive increase in liver enzymes (AST and ALT), TSB, TG, and CH levels across increasing ultrasound-based grades of nonalcoholic fatty liver disease (NAFLD). These findings are in alignment with the pathophysiological understanding of NAFLD as a disease characterized by hepatocyte injury, systemic metabolic dysfunction, and impaired liver function. The elevation of AST and ALT enzymes from grade 1 to grade 3 is indicative of worsening hepatocellular damage [12]. ALT, being more specific to liver tissue, serves as a sensitive marker for hepatic inflammation and necrosis in NAFLD patients [12]. Elevated AST levels, though less specific, reinforce this finding when interpreted in conjunction with ALT [13]. The progressive rise in total serum bilirubin levels suggests a decline in hepatic excretory function, which often accompanies the transition from steatosis to steatohepatitis and ultimately fibrosis [14-15].

Table 2: Comparison of biochemical parameters between NAFLD patients and healthy controls

Parameter	Control Group Mean ± SD	NAFLD Patients Mean ± SD	P-value
AST (U/L)	10.21 ± 3.40	22.92 ± 10.50	0.000
ALT (U/L)	13.65 ± 3.70	29.88 ± 17.45	0.000
TSB (mg/dL)	0.51 ± 0.18	0.63 ± 0.29	0.0014
TG (mg/dL)	90.41 ± 15.50	199.65 ± 76.60	0.000
CH (mg/dL)	111.08 ± 22.51	218.93 ± 70.23	0.000

AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, TSB: Total Serum Bilirubin, TG: Triglycerides, CH: Cholesterol, SD: Standard Deviation

Table 3: Biochemical and lipid profile at every NAFLD grade

Parameter	Grade 1 Mean ± SD	Grade 2 Mean ± SD	Grade 3 Mean ± SD	P-value
AST (U/L)	16.41 ± 6.98	27.97 ± 9.97	29.63 ± 9.69	0.000
ALT (U/L)	20.44 ± 8.75	37.61 ± 21.50	38.69 ± 11.52	0.000
TSB (mg/dL)	0.49 ± 0.19	0.68 ± 0.31	0.90 ± 0.29	0.000
TG (mg/dL)	174.70 ± 75.30	220.84 ± 66.03	221.06 ± 86.19	0.01
Cho (mg/dL)	189.54 ± 57.84	234.39 ± 64.42	266.69 ± 81.21	0.002

AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, TSB: Total Serum Bilirubin, TG: Triglycerides, CH: Cholesterol, SD: Standard Deviation

Serum triglycerides and cholesterol showed significant elevation with NAFLD severity, which is consistent with the literature describing the metabolic underpinnings of NAFLD. Dyslipidemia is a hallmark of metabolic syndrome and contributes to hepatic fat accumulation via increased delivery of free fatty acids to the liver and de novo lipogenesis [16-17]. The liver plays a central role in lipid metabolism, and disruption of these processes is reflected in increased circulating lipid levels [18]. Several studies have previously established similar associations. Jwarchan *et al.* **(2020)** reported a positive correlation between sonographic grading of fatty liver and serum ALT and cholesterol levels, supporting the current findings [19]. Similarly, Andlib *et al.* (2019) noted a consistent elevation in liver enzymes and lipid profiles with increasing fatty infiltration observed on ultrasound [20]. The present results extend these observations by providing robust statistical significance across all biochemical parameters. In comparison to the findings by Ahmed et al. (2022), who investigated TGF-β1 signaling in NAFLD progression, our results emphasize the clinical biochemical correlates rather than molecular pathways [18]. Neuschwander-Tetri (2017) also emphasized the systemic nature of NAFLD, highlighting its association with insulin resistance, inflammation, and oxidative stress, which likely explain the biochemical patterns observed in our patients [21]. Furthermore, the use of non-invasive biomarkers as proxies for liver injury is gaining clinical traction [22]. Several reports advocate for the use of ALT and AST as part of routine NAFLD screening and monitoring protocols, especially in resource-limited settings where liver biopsy or advanced imaging may not be feasible [23-24]. While liver biopsy remains the gold standard, ultrasound-based grading, complemented by biochemical markers, provides a pragmatic approach to risk stratification and monitoring. This is particularly relevant given the high global prevalence of NAFLD, estimated to be over 25% [25]. With no approved pharmacotherapy for NAFLD, early identification and lifestyle interventions remain the cornerstone of management [26].

AST and ALT elevation are consistent with previous studies, indicating that liver enzymes are elevated in patients with nonalcoholic fatty liver disease, reflecting hepatic inflammation and injury [27]. The elevation in TSB, though less pronounced, aligns with studies suggesting mild cholestasis or hepatocellular dysfunction in NAFLD [28]. Hypertriglyceridemia and hypercholesterolemia observed in this study further support the well-established link between NAFLD and dyslipidemia, highlighting the metabolic component of the disease [29-30]. This pattern of biochemical alterations emphasizes the importance of routine liver function and lipid profile testing in individuals at risk of NAFLD, and supports the use of non-invasive diagnostic tools in clinical practice [31].

Our findings support a direct and significant association between increasing ultrasound grades of NAFLD and progressive biochemical deterioration. These results highlight the utility of

liver function tests and lipid profiles in assessing disease severity. Incorporating these markers into clinical practice could enhance early diagnosis and enable more personalized management strategies. Ultrasonography, being a non-invasive, widely available, and cost-effective tool, remains a first-line modality for detecting hepatic steatosis. Recent studies confirm that ultrasound grading correlates well with histological severity and metabolic risk factors in NAFLD patients [32-33]. These findings reinforce the value of ultrasound not only for diagnosis but also for risk stratification and follow-up.

This study has several limitations that should be considered when interpreting the results. First, the diagnosis and grading of NAFLD were based solely on abdominal ultrasonography, which, while non-invasive and widely available, is less sensitive in detecting mild steatosis and cannot assess hepatic fibrosis or inflammation with high accuracy. Second, liver biopsy, the gold standard for diagnosing and staging NAFLD, was not performed, limiting the histopathological correlation of biochemical and imaging findings. Third, the cross-sectional design of the study restricts the ability to establish causal relationships or assess longitudinal changes in liver function over time. Additionally, potential confounding factors such as dietary habits, insulin resistance, physical activity, and undiagnosed comorbid conditions were not fully evaluated, which may influence liver enzyme and lipid levels. Finally, the sample size, although adequate for detecting statistical significance, may limit the generalizability of the findings to broader populations.

Conclusions

The findings of this study reveal a clear and statistically significant association between the severity of nonalcoholic fatty liver disease, as graded by abdominal ultrasonography, and alterations in key biochemical parameters, including alanine aminotransferase, aspartate aminotransferase, total serum bilirubin, triglycerides, and cholesterol. A stepwise increase in these markers was observed with progression from mild to severe grades of NAFLD, indicating worsening hepatocellular injury, impaired liver excretory function, and metabolic dysregulation. This biochemical progression aligns with the pathophysiological mechanisms underlying NAFLD, where lipid accumulation, oxidative stress, and inflammation drive hepatic damage and fibrosis.

The use of ALT and AST as indicators of hepatocyte injury, combined with elevated bilirubin suggesting compromised excretory function, provides a valuable, non-invasive biochemical profile correlating with disease severity. Furthermore, the lipid profile abnormalities observed, particularly hypertriglyceridemia and hypercholesterolemia, highlight the metabolic component of NAFLD and reinforce the close relationship between hepatic steatosis and systemic dyslipidemia.

Post-hoc LSD analysis further confirmed the significance of differences between NAFLD grades, supporting the use of these markers not only for diagnosis but also for monitoring disease progression. These results underscore the potential of integrating biochemical testing with imaging modalities to enhance diagnostic accuracy, stratify risk, and guide personalized clinical management. While liver biopsy remains the gold standard for definitive diagnosis, this study supports the evolving role of non-invasive tools in the routine evaluation of NAFLD patients, especially in resource-limited settings. Future studies with larger populations and histological correlation are recommended to validate these findings and explore their utility in clinical decision-making and therapeutic monitoring.

Funding: There is no funding for this research.

Conflict of interest: The authors state that there is no conflict of interest.

Author contributions: Conceptualization's; Z.S.M., Methodology: A.S.A., Formal analysis and investigation: A.S.T., and Z.A.M., Writing: A.M.K., Resource: A.M.K., Supervision: D.M.H.

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