E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/16.02.416

Research Article

Correlation of Ultrasonographic Fatty Liver Grading with Liver Function Tests in Patients with Non-Alcoholic Fatty Liver Disease: A Cross-Sectional Study

Dr Krupalsingh N Sisodiya
Professor and HOD, Department of Radiodiagnosis and Imaging, JMF's ACPM Medical College & Hospital, Morane (P.L.) Sakri Road, Dhule - 424001, Maharashtra, INDIA. Email: sisodiyakrupal@gmail.com

Dr Rahul G Bachhav
Associate Professor, Department of Radiodiagnosis and Imaging, JMF's ACPM Medical College & Hospital, Morane (P.L.) Sakri Road, Dhule - 424001, Maharashtra, INDIA. Email: drrahulrajebachhav@gmail.com

Dr Ashish J Agrawal
Associate Professor, Department of Radiodiagnosis and Imaging, JMF's ACPM Medical College & Hospital, Morane (P.L.) Sakri Road, Dhule - 424001, Maharashtra, INDIA. Email: ashish.radiology@gmail.com

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a common metabolic liver disorder ranging from simple steatosis to steatohepatitis, fibrosis and cirrhosis. Ultrasonography is widely used for detecting and grading hepatic steatosis, while liver function tests provide biochemical evidence of hepatocellular injury, cholestasis and synthetic dysfunction. The relationship between ultrasonographic severity and biochemical abnormalities requires further evaluation. **Aim:** To determine the correlation between ultrasonographic fatty liver grading and liver function-test parameters among patients with NAFLD. **Materials and Methods:** This hospital-based, observational cross-sectional study included 120 adults with ultrasonographically diagnosed NAFLD. Hepatic steatosis was classified as grade I, II or III according to hepatic echogenicity, hepatorenal contrast, posterior acoustic attenuation and visualisation of intrahepatic vessels and the diaphragm. Serum bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total protein, albumin and globulin were assessed. Parameters across the three grades were compared using analysis of variance or Welch ANOVA. Spearman's rank correlation was used to determine associations between ultrasonographic grade and liver function-test parameters. A p value below 0.05 was considered statistically significant. **Results:** The mean age was 46.37±11.82 years, and 68 (56.7%) patients were male. Grade I, II and III fatty liver were identified in 53 (44.2%), 41 (34.2%) and 26 (21.7%) patients, respectively. Elevated ALT was present in 42.5%, GGT in 36.7%, AST in 32.5% and ALP in 23.3%. Mean AST increased from 29.46±9.38 U/L in grade I to 58.31±22.47 U/L in grade III, while ALT increased from 34.18±12.46 U/L to 76.42±28.36 U/L (both $p<0.001$). Significant grade-related increases were also observed in

bilirubin, ALP and GGT. Albumin declined from 4.31±0.39 g/dL in grade I to 3.79±0.53 g/dL in grade III ($p<0.001$). Fatty liver grade showed significant positive correlations with ALT ($\rho=0.56$), GGT ($\rho=0.51$), AST ($\rho=0.48$), direct bilirubin ($\rho=0.43$) and ALP ($\rho=0.42$), with all $p<0.001$. Albumin showed a significant negative correlation with grade ($\rho=-0.31$, $p<0.001$). **Conclusion:** Increasing ultrasonographic fatty liver grade was significantly associated with higher ALT, AST, GGT, ALP and bilirubin and lower serum albumin. Combining ultrasonographic grading with liver function tests may facilitate severity assessment and identification of patients requiring fibrosis-specific evaluation. However, these methods cannot replace elastography, validated fibrosis scores or histopathological assessment when advanced liver disease is suspected.

KEYWORDS: Non-alcoholic fatty liver disease; Ultrasonographic grading; Liver function tests

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD), currently included within the broader nomenclature of metabolic dysfunction-associated steatotic liver disease, is characterised by excessive accumulation of fat in hepatocytes in individuals without significant alcohol consumption or another identifiable cause of hepatic steatosis. It represents a spectrum extending from simple steatosis to steatohepatitis, progressive fibrosis, cirrhosis and hepatocellular carcinoma. NAFLD has become one of the most common causes of chronic liver disease worldwide, paralleling the increasing prevalence of obesity, type 2 diabetes mellitus, dyslipidaemia and metabolic syndrome. Its global prevalence has been estimated at approximately 24%, and its growing burden is particularly important in South-East Asian countries, including India [1,2].

Most patients with NAFLD remain asymptomatic or present with nonspecific symptoms such as fatigue and right upper-quadrant discomfort. Consequently, the disease is frequently detected incidentally during abdominal ultrasonography or through abnormal liver function tests. Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), bilirubin, albumin and total protein are commonly measured while evaluating suspected hepatic disease. Nevertheless, normal aminotransferase levels do not exclude NAFLD, steatohepatitis or clinically significant fibrosis. Conversely, elevated liver enzymes are not specific to NAFLD and must be interpreted after excluding alternative hepatic disorders [3].

Ultrasonography is widely used as the initial imaging investigation for suspected fatty liver because it is non-invasive, inexpensive, readily available and free from ionising radiation. The principal sonographic features include increased hepatic echogenicity, hepatorenal contrast, posterior beam attenuation, reduced visualisation of intrahepatic vessels and poor delineation of the diaphragm. Based on these findings, hepatic steatosis can be classified as grade I, II or III. Although conventional ultrasonography has limitations in detecting mild steatosis and cannot reliably differentiate simple steatosis from steatohepatitis or accurately stage fibrosis, it remains the most practical modality for routine evaluation and population-level assessment [3-5].

AIM

To determine the correlation between ultrasonographic fatty liver grading and liver function-test parameters in patients with non-alcoholic fatty liver disease.

OBJECTIVES

1. To classify the severity of fatty liver as grade I, II or III using ultrasonographic findings.
2. To assess liver function-test parameters among patients with different ultrasonographic grades of NAFLD.
3. To determine the direction and strength of correlation between ultrasonographic fatty liver grade and individual liver function-test parameters.

MATERIALS AND METHODOLOGY

Source of Data

Data were obtained from patients referred to the Department of Radiodiagnosis and Imaging for abdominal ultrasonography at JMF's ACPM Medical College & Hospital, Morane (P.L.), Sakri Road, Dhule, Maharashtra. Patients who demonstrated ultrasonographic evidence of fatty liver and fulfilled the eligibility criteria were enrolled. Relevant clinical information and laboratory findings were collected from patient interviews, clinical records and reports available in the hospital information system.

Study Design

This was a hospital-based, observational, analytical cross-sectional study.

Study Location

The study was conducted in the Department of Radiodiagnosis and Imaging in collaboration with the relevant clinical departments and central clinical laboratory of JMF's ACPM Medical College & Hospital, Morane (P.L.), Sakri Road, Dhule-424001, Maharashtra, India.

Study Duration

The study was conducted over 18 months, including participant enrolment, ultrasonographic examination, laboratory evaluation, data verification and statistical analysis.

Sample Size

A total of 120 patients with ultrasonographically diagnosed NAFLD who fulfilled the eligibility criteria were included in the study. Participants were enrolled consecutively until the required sample size was achieved.

Sampling Technique

A consecutive sampling technique was used. Every eligible patient diagnosed with fatty liver during the study period was invited to participate until 120 patients had been enrolled.

Inclusion Criteria

1. Patients aged 18 years or older.
2. Patients showing ultrasonographic evidence of fatty liver.
3. Patients with no history of significant alcohol consumption.
4. Patients whose complete liver function-test reports were available within a clinically relevant period of the ultrasonographic examination.
5. Patients who provided written informed consent to participate.

Exclusion Criteria

1. Patients with significant alcohol consumption, defined as more than 20 g/day in women or more than 30 g/day in men.
2. Patients with hepatitis B, hepatitis C or another documented viral hepatitis.
3. Patients with autoimmune hepatitis, Wilson disease, haemochromatosis or other recognised chronic liver disorders.
4. Patients with known cirrhosis, hepatic malignancy or metastatic liver disease.
5. Patients receiving medications known to produce hepatic steatosis or significant hepatotoxicity, such as amiodarone, methotrexate, tamoxifen, corticosteroids or valproate.
6. Pregnant women.

7. Patients with biliary obstruction or acute hepatobiliary infection.
8. Patients with congestive cardiac failure or another condition causing hepatic congestion.
9. Patients with poor sonographic visualisation that prevented reliable grading of hepatic steatosis.
10. Patients with incomplete clinical or biochemical data.

Procedure and Methodology

Eligible patients were informed about the purpose and procedure of the study, and written informed consent was obtained. Demographic and clinical information, including age, sex, weight, height, body mass index, symptoms, history of diabetes mellitus, hypertension, dyslipidaemia, medication use and alcohol consumption, was recorded using a predesigned case-record form.

All participants underwent abdominal ultrasonography after fasting for approximately six to eight hours. Examinations were performed using a standard ultrasound system equipped with a 2-5 MHz curvilinear transducer. Patients were examined in supine and left lateral decubitus positions. Longitudinal, transverse, subcostal and intercostal scans were obtained where necessary.

The liver was evaluated for its size, surface, parenchymal echogenicity, echotexture, posterior acoustic attenuation, visualisation of intrahepatic vessel walls and visibility of the diaphragm. Hepatic echogenicity was compared with the echogenicity of the right renal cortex whenever the right kidney was suitable for comparison. Associated abnormalities involving the gallbladder, biliary tract, portal vein, spleen and pancreas were also noted.

Fatty liver was categorised as follows:

- **Grade I (mild):** A slight, diffuse increase in hepatic echogenicity with normal visualisation of the diaphragm and intrahepatic vessel borders.
- **Grade II (moderate):** A moderate diffuse increase in hepatic echogenicity, with mildly impaired visualisation of intrahepatic vessels and the diaphragm.
- **Grade III (severe):** A marked increase in hepatic echogenicity with posterior beam attenuation and poor or absent visualisation of intrahepatic vessels, the posterior portion of the right hepatic lobe and the diaphragm.

To reduce observer-related variation, examinations were performed or reviewed by radiologists experienced in abdominal ultrasonography. The radiologists evaluated the images using predefined grading criteria. Wherever findings were uncertain, the images were jointly reviewed and the final grade was assigned by consensus.

Liver function-test measurements included serum total bilirubin, direct bilirubin, indirect bilirubin, AST, ALT, ALP, GGT, total protein, albumin and globulin. The AST/ALT ratio and albumin/globulin ratio were calculated from the corresponding biochemical measurements. The ultrasonographic grade was subsequently correlated with each liver function-test parameter.

Sample Processing

Approximately 5 mL of venous blood was collected from each participant under aseptic precautions, preferably after overnight fasting. The blood was transferred into a sterile plain or serum-separator tube and was allowed to clot at room temperature. The sample was then centrifuged at approximately 3,000 revolutions per minute for 10 minutes to separate the serum.

The serum was inspected for haemolysis and processed in the central clinical laboratory using an automated biochemistry analyser. Serum AST and ALT were measured using standard kinetic methods; ALP and GGT were measured using standard enzymatic

methods; bilirubin was estimated using a diazo-based method; and total protein and albumin were measured using biuret and bromocresol green methods, respectively. Globulin was calculated by subtracting albumin from total protein. Haemolysed, contaminated or otherwise unsuitable specimens were rejected, and repeat samples were obtained wherever feasible. Internal quality-control procedures and routine calibration of the analyser were undertaken according to laboratory protocols.

Data Collection

Data were collected using a structured case-record form containing the following sections:

1. Demographic characteristics.
2. Anthropometric measurements.
3. Clinical symptoms and relevant medical history.
4. Alcohol-consumption and medication history.
5. Metabolic risk factors and comorbidities.
6. Ultrasonographic findings and fatty liver grade.
7. Individual liver function-test parameters.
8. Calculated biochemical ratios and final study classification.

The completed forms were reviewed for completeness and consistency. Each participant was assigned a unique study identification number. Personally identifiable information was kept separately from the analytical database to maintain confidentiality.

Statistical Methods

Data were entered into Microsoft Excel and analysed using an appropriate statistical software package. Continuous variables were summarised as mean and standard deviation when approximately normally distributed and as median and interquartile range when skewed. Categorical variables were expressed as frequency and percentage.

The distribution of continuous variables was assessed using graphical methods and the Shapiro-Wilk test. Liver function-test parameters across ultrasonographic grades I, II and III were compared using one-way analysis of variance followed by an appropriate post-hoc test for normally distributed variables. The Kruskal-Wallis test, followed by adjusted pairwise comparisons, was used for non-normally distributed variables.

Because ultrasonographic fatty liver grade was an ordinal variable, its correlation with individual liver function-test parameters was assessed using Spearman's rank correlation coefficient (ρ). Pearson's correlation coefficient was used only where both variables satisfied its assumptions. Associations between categorical variables were evaluated using the chi-square test or Fisher's exact test, as appropriate.

Where relevant, multivariable ordinal logistic regression was performed to evaluate liver function-test parameters independently associated with increasing ultrasonographic fatty liver grade after adjustment for age, sex, body mass index, diabetes mellitus and dyslipidaemia. Adjusted odds ratios were reported with 95% confidence intervals. Multicollinearity and the proportional-odds assumption were assessed before interpreting the model. All statistical tests were two-tailed, and $p<0.05$ was considered statistically significant.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from every participant before enrolment. Confidentiality was maintained throughout the study, and participation did not interfere with the routine diagnostic evaluation or treatment of the patients

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OBSERVATION AND RESULTS

Table 1. Overall clinical, ultrasonographic and biochemical profile of patients with NAFLD (N=120)

Study parameter	Overall, n (%) or Mean (SD)	Effect estimate (95% CI)	Test of significance	P value
Age, years	46.37 (11.82)	Mean: 46.37 (44.23-48.51)	One-sample t=42.97	<0.001*
Male sex	68 (56.7%)	Proportion: 56.7% (47.7%-65.2%)	Binomial z=1.46	0.144
Female sex	52 (43.3%)	Proportion: 43.3% (34.8%-52.3%)		
BMI, kg/m²	27.84 (4.16)	Mean: 27.84 (27.09-28.59)	One-sample t=7.48†	<0.001*
Obesity, BMI ≥30 kg/m²	34 (28.3%)	Proportion: 28.3% (21.0%-37.0%)	Binomial z=4.38‡	<0.001*
Type 2 diabetes mellitus	43 (35.8%)	Proportion: 35.8% (27.8%-44.7%)	Binomial z=1.30§	0.193
Hypertension	37 (30.8%)	Proportion: 30.8% (23.3%-39.6%)	Binomial z=0.20§	0.841
Dyslipidaemia	49 (40.8%)	Proportion: 40.8% (32.4%-49.8%)	Binomial z=2.53§	0.011*
Grade I fatty liver	53 (44.2%)	Proportion: 44.2% (35.6%-53.1%)	χ^2=9.15¶	0.010*
Grade II fatty liver	41 (34.2%)	Proportion: 34.2% (26.3%-43.0%)		
Grade III fatty liver	26 (21.7%)	Proportion: 21.7% (15.2%-30.0%)		
Elevated AST	39 (32.5%)	Proportion: 32.5% (24.8%-41.3%)	Cochran-Armitage Z=4.39#	<0.001*
Elevated ALT	51 (42.5%)	Proportion: 42.5% (33.9%-51.5%)	Cochran-Armitage Z=5.11#	<0.001*
Elevated ALP	28 (23.3%)	Proportion: 23.3% (16.7%-31.7%)	Cochran-Armitage Z=3.09#	0.002*
Elevated GGT	44 (36.7%)	Proportion: 36.7% (28.6%-45.6%)	Cochran-Armitage Z=4.67#	<0.001*
Hyperbilirubinaemia	17 (14.2%)	Proportion: 14.2% (9.0%-21.5%)	Cochran-Armitage Z=2.53#	0.011*
Hypoalbuminaemia	13 (10.8%)	Proportion: 10.8% (6.4%-17.6%)	Cochran-Armitage Z=2.71#	0.007*

Fatty liver grade versus ALT		Spearman ρ=0.56 (0.42-0.67)	t=7.31	<0.001*
Fatty liver grade versus AST		Spearman ρ=0.48 (0.33-0.61)	t=5.94	<0.001*
Fatty liver grade versus albumin		Spearman ρ=-0.31 (-0.46 to -0.14)	t=-3.54	<0.001*

†Compared with the upper limit of normal BMI of 24.9 kg/m².
‡Compared with an expected prevalence of 15%.
§Compared with an expected prevalence of 30%.
¶Goodness-of-fit test against equal grade distribution. #Trend across grades I-III.
*Statistically significant.

The study included 120 patients with a mean age of 46.37±11.82 years (95% CI: 44.23-48.51). Males constituted 56.7% and females 43.3% of the study population, although the difference in sex distribution was not statistically significant (p=0.144). The mean BMI was 27.84±4.16 kg/m², which was significantly higher than the upper limit of normal BMI (t=7.48, p<0.001). Obesity was present in 28.3% of patients, significantly exceeding the expected prevalence of 15% (p<0.001). Type 2 diabetes mellitus, hypertension and dyslipidaemia were observed in 35.8%, 30.8% and 40.8% of patients, respectively. Dyslipidaemia was significantly more prevalent than the reference proportion of 30% (p=0.011), whereas diabetes and hypertension were not.

Grade I fatty liver was the most common ultrasonographic category, affecting 53 (44.2%) patients, followed by grade II in 41 (34.2%) and grade III in 26 (21.7%). The distribution of fatty liver grades differed significantly from an equal distribution (χ^2=9.15, p=0.010). Elevated ALT was the most frequent biochemical abnormality, occurring in 42.5% of patients, followed by elevated GGT in 36.7%, AST in 32.5% and ALP in 23.3%. Hyperbilirubinaemia and hypoalbuminaemia were present in 14.2% and 10.8%, respectively. Each of these biochemical abnormalities demonstrated a significant increasing trend across grades I-III (all p<0.05). Fatty liver grade had significant positive correlations with ALT (ρ=0.56, p<0.001) and AST (ρ=0.48, p<0.001) and a significant negative correlation with albumin (ρ=-0.31, p<0.001).

Table 2. Classification of fatty liver severity according to ultrasonographic findings (N=120)

Ultrasonographic finding	Grade I (n=53)	Grade II (n=41)	Grade III (n=26)	Overall prevalence (95% CI)	Test of significance	P value
Increased hepatic echogenicity	53 (100.0%)	41 (100.0%)	26 (100.0%)	100.0% (96.9%-100.0%)	Descriptive criterion	
Increased hepatorenal contrast	47 (88.7%)	39 (95.1%)	26 (100.0%)	112/120; 93.3% (87.4%-96.6%)	Fisher's exact=4.28	0.107
Mildly impaired visualisation of intrahepatic vessels	9 (17.0%)	33 (80.5%)	7 (26.9%)	49/120; 40.8% (32.4%-49.8%)	χ^2=42.26	<0.001*

Poorly visualised intrahepatic vessels	1 (1.9%)	8 (19.5%)	24 (92.3%)	33/120; 27.5% (20.3%-36.1%)	Fisher's exact=79.64	<0.001*
Posterior acoustic attenuation	7 (13.2%)	23 (56.1%)	24 (92.3%)	54/120; 45.0% (36.4%-53.9%)	χ^2=49.08	<0.001*
Reduced visualisation of diaphragm	3 (5.7%)	19 (46.3%)	23 (88.5%)	45/120; 37.5% (29.3%-46.4%)	χ^2=55.21	<0.001*
Poor visualisation of posterior right hepatic lobe	1 (1.9%)	7 (17.1%)	22 (84.6%)	30/120; 25.0% (18.1%-33.4%)	Fisher's exact=70.73	<0.001*
Hepatomegaly	27 (50.9%)	29 (70.7%)	21 (80.8%)	77/120; 64.2% (55.3%-72.2%)	χ^2=8.41	0.015*
Focal fatty sparing	8 (15.1%)	11 (26.8%)	7 (26.9%)	26/120; 21.7% (15.2%-30.0%)	χ^2=2.34	0.311
Gallbladder calculus	6 (11.3%)	9 (22.0%)	4 (15.4%)	19/120; 15.8% (10.4%-23.4%)	Fisher's exact=2.01	0.374
Portal vein diameter, mm, Mean (SD)	10.18 (1.06)	10.67 (1.12)	11.34 (1.28)	Mean difference†: 1.16 (0.62-1.70)	ANOVA F=10.23	<0.001*
Liver span, cm, Mean (SD)	14.62 (1.37)	15.41 (1.48)	16.38 (1.63)	Mean difference†: 1.76 (1.06-2.46)	ANOVA F=13.37	<0.001*

†Mean difference between grade III and grade I.
*Statistically significant.

Increased hepatic echogenicity was observed in all 120 patients because it was the principal diagnostic criterion for fatty liver. Increased hepatorenal contrast was present in 93.3% of patients, rising from 88.7% in grade I to 100% in grade III; however, the difference across grades was not statistically significant (p=0.107). Mild impairment in the visualisation of intrahepatic vessels was most frequently observed in grade II disease (80.5%), compared with 17.0% in grade I and 26.9% in grade III (χ^2=42.26, p<0.001). By contrast, poor visualisation of intrahepatic vessels increased markedly from 1.9% in grade I and 19.5% in grade II to 92.3% in grade III disease (p<0.001).

Posterior acoustic attenuation increased progressively from 13.2% in grade I to 56.1% in grade II and 92.3% in grade III (χ^2=49.08, p<0.001). Similarly, reduced visualisation of the diaphragm increased from 5.7% to 46.3% and 88.5%, respectively (χ^2=55.21, p<0.001). Poor visualisation of the posterior right hepatic lobe was found in 1.9% of grade I, 17.1% of grade II and 84.6% of grade III cases (p<0.001). Hepatomegaly was present in 64.2% overall and became more frequent with increasing severity, from 50.9% in grade I to 80.8% in grade III (χ^2=8.41, p=0.015). Focal fatty sparing and gallbladder calculus were not significantly associated with fatty liver grade (p=0.311 and p=0.374, respectively).

The mean portal vein diameter increased from 10.18±1.06 mm in grade I to 11.34±1.28 mm in grade III, producing a grade III-grade I mean difference of 1.16 mm (95% CI: 0.62-1.70; p<0.001). The mean liver span also increased significantly from 14.62±1.37 cm to 16.38±1.63 cm, with a mean difference of 1.76 cm (95% CI: 1.06-2.46; p<0.001).

Table 3. Comparison of liver function-test parameters across ultrasonographic grades of NAFLD (N=120)

Liver function-test parameter	Grade I (n=53), Mean (SD)	Grade II (n=41), Mean (SD)	Grade III (n=26), Mean (SD)	Grade III-Grade I difference (95% CI)	Test of significance	P value
Total bilirubin, mg/dL	0.71 (0.23)	0.86 (0.31)	1.14 (0.48)	0.43 (0.24-0.62)	Welch ANOVA F=10.86	<0.001*
Direct bilirubin, mg/dL	0.22 (0.09)	0.29 (0.13)	0.43 (0.21)	0.21 (0.13-0.29)	Welch ANOVA F=14.31	<0.001*
Indirect bilirubin, mg/dL	0.49 (0.18)	0.57 (0.22)	0.71 (0.31)	0.22 (0.11-0.33)	ANOVA F=8.68	<0.001*
AST, U/L	29.46 (9.38)	41.72 (14.63)	58.31 (22.47)	28.85 (20.41-37.29)	Welch ANOVA F=25.42	<0.001*
ALT, U/L	34.18 (12.46)	53.67 (19.82)	76.42 (28.36)	42.24 (31.88-52.60)	Welch ANOVA F=34.76	<0.001*
AST/ALT ratio	0.89 (0.24)	0.81 (0.22)	0.79 (0.27)	-0.10 (-0.22 to 0.02)	ANOVA F=1.93	0.150
ALP, U/L	91.72 (24.18)	112.84 (31.63)	139.27 (42.46)	47.55 (31.60-63.50)	Welch ANOVA F=18.19	<0.001*
GGT, U/L	31.64 (13.27)	48.91 (21.38)	72.53 (32.81)	40.89 (29.63-52.15)	Welch ANOVA F=23.68	<0.001*
Total protein, g/dL	7.29 (0.53)	7.18 (0.59)	6.91 (0.68)	-0.38 (-0.67 to -0.09)	ANOVA F=3.87	0.024*
Albumin, g/dL	4.31 (0.39)	4.08 (0.46)	3.79 (0.53)	-0.52 (-0.74 to -0.30)	ANOVA F=11.87	<0.001*

Globulin, g/dL	2.98 (0.48)	3.10 (0.54)	3.12 (0.61)	0.14 (-0.11 to 0.39)	ANOVA F=0.87	0.422
Albumin/globulin ratio	1.48 (0.29)	1.34 (0.31)	1.24 (0.34)	-0.24 (-0.39 to -0.09)	ANOVA F=5.64	0.005*

*Statistically significant at p<0.05.

Serum bilirubin levels increased significantly with ultrasonographic severity. Mean total bilirubin rose from 0.71±0.23 mg/dL in grade I to 0.86±0.31 mg/dL in grade II and 1.14±0.48 mg/dL in grade III (Welch ANOVA F=10.86, p<0.001). Grade III patients had mean total bilirubin levels 0.43 mg/dL higher than grade I patients (95% CI: 0.24-0.62). Direct and indirect bilirubin followed similar patterns, with grade III-grade I differences of 0.21 mg/dL and 0.22 mg/dL, respectively (both p<0.001).

A pronounced progressive increase was observed in serum transaminase levels. Mean AST increased from 29.46±9.38 U/L in grade I to 41.72±14.63 U/L in grade II and 58.31±22.47 U/L in grade III (F=25.42, p<0.001). Mean ALT increased from 34.18±12.46 U/L to 53.67±19.82 U/L and 76.42±28.36 U/L, respectively (F=34.76, p<0.001). Compared with grade I, grade III patients had AST and ALT levels higher by 28.85 U/L (95% CI: 20.41-37.29) and 42.24 U/L (95% CI: 31.88-52.60), respectively. In contrast, the AST/ALT ratio did not differ significantly among the grades (p=0.150).

Mean ALP increased from 91.72±24.18 U/L in grade I to 139.27±42.46 U/L in grade III, representing a mean difference of 47.55 U/L (95% CI: 31.60-63.50; p<0.001). Mean GGT similarly increased from 31.64±13.27 U/L to 72.53±32.81 U/L, with a mean difference of 40.89 U/L (95% CI: 29.63-52.15; p<0.001). Conversely, mean total protein declined from 7.29±0.53 g/dL in grade I to 6.91±0.68 g/dL in grade III (p=0.024), while serum albumin decreased from 4.31±0.39 g/dL to 3.79±0.53 g/dL (p<0.001). The albumin/globulin ratio also declined significantly across the three grades (p=0.005), whereas globulin levels showed no significant difference (p=0.422). Overall, grade III disease was associated with higher bilirubin, AST, ALT,

Table 4. Direction and strength of correlation between ultrasonographic fatty liver grade and liver function-test parameters (N=120)

Liver function-test parameter	Spearman's ρ	Direction and strength	95% CI for ρ	Test statistic, t	P value
Total bilirubin	0.39	Moderate positive	0.22-0.53	4.60	<0.001*
Direct bilirubin	0.43	Moderate positive	0.27-0.57	5.18	<0.001*
Indirect bilirubin	0.31	Weak positive	0.14-0.46	3.54	<0.001*
AST	0.48	Moderate positive	0.33-0.61	5.94	<0.001*
ALT	0.56	Moderate positive	0.42-0.67	7.31	<0.001*
AST/ALT ratio	-0.14	Very weak negative	-0.31 to 0.04	-1.54	0.126
ALP	0.42	Moderate positive	0.26-0.56	5.02	<0.001*

GGT	0.51	Moderate positive	0.36-0.63	6.45	<0.001*
Total protein	-0.21	Weak negative	-0.38 to -0.03	-2.34	0.021*
Albumin	-0.31	Weak negative	-0.46 to -0.14	-3.54	<0.001*
Globulin	0.11	Very weak positive	-0.07 to 0.29	1.20	0.232
Albumin/globulin ratio	-0.28	Weak negative	-0.44 to -0.11	-3.17	0.002*

*Statistically significant at $p<0.05$.

Ultrasonographic fatty liver grade demonstrated statistically significant positive correlations with most biochemical indicators of hepatocellular and cholestatic dysfunction. ALT showed the strongest positive correlation with fatty liver grade ($\rho=0.56$; 95% CI: 0.42-0.67; $p<0.001$), followed by GGT ($\rho=0.51$; 95% CI: 0.36-0.63; $p<0.001$) and AST ($\rho=0.48$; 95% CI: 0.33-0.61; $p<0.001$). Moderate positive correlations were also observed for direct bilirubin ($\rho=0.43$, $p<0.001$) and ALP ($\rho=0.42$, $p<0.001$). Total bilirubin showed a borderline weak-to-moderate positive correlation ($\rho=0.39$; 95% CI: 0.22-0.53; $p<0.001$), while indirect bilirubin demonstrated a weak positive correlation ($\rho=0.31$; $p<0.001$).

Negative correlations were observed for parameters reflecting hepatic protein synthesis. Total protein demonstrated a weak negative correlation with fatty liver grade ($\rho=-0.21$; 95% CI: -0.38 to -0.03; $p=0.021$), while albumin showed a significant weak negative correlation ($\rho=-0.31$; 95% CI: -0.46 to -0.14; $p<0.001$). The albumin/globulin ratio also declined as the ultrasonographic grade increased ($\rho=-0.28$; 95% CI: -0.44 to -0.11; $p=0.002$). The AST/ALT ratio showed a very weak negative correlation that was not statistically significant ($\rho=-0.14$, $p=0.126$), and globulin demonstrated a very weak, nonsignificant positive correlation ($\rho=0.11$, $p=0.232$).

DISCUSSION

The present cross-sectional study evaluated the relationship between ultrasonographic fatty liver grading and liver function-test parameters among 120 patients with non-alcoholic fatty liver disease (NAFLD). The principal findings were that patients commonly had metabolic risk factors; ultrasonographic abnormalities became progressively more prominent from grade I to grade III disease; serum bilirubin, AST, ALT, ALP and GGT increased significantly with increasing ultrasonographic grade; and serum total protein, albumin and the albumin/globulin ratio decreased. ALT demonstrated the strongest correlation with ultrasonographic grade, followed by GGT and AST. These findings indicate that worsening sonographic steatosis was accompanied by greater biochemical evidence of hepatocellular injury and, to a lesser degree, impaired hepatic synthetic function.

Clinical, metabolic and biochemical profile

The mean age of the participants was 46.37±11.82 years, indicating that ultrasonographically detectable NAFLD predominantly affected middle-aged adults. There was a modest male predominance, with males comprising 56.7% of the sample, although the sex distribution was not statistically significant. The global meta-analysis conducted by Riazi et al. (2022)[1] similarly showed that NAFLD was more prevalent in men than women and increased with age. Younossi et al. (2023)[2] estimated the overall global prevalence of NAFLD at

approximately 30% and reported that the burden had increased substantially over recent decades. The middle-aged profile in the present study is therefore consistent with the age at which prolonged exposure to obesity, insulin resistance, diabetes and dyslipidaemia becomes clinically apparent.

The mean BMI was 27.84±4.16 kg/m², and 28.3% of the patients were obese. This confirms the close relationship between excess adiposity and hepatic steatosis. Nevertheless, the finding that fewer than one-third of patients met the BMI ≥30 kg/m² criterion indicates that NAFLD should not be regarded exclusively as a disease of overt obesity. Elhence et al. (2022)[3], in a systematic review and meta-analysis of Indian studies, estimated the pooled prevalence of NAFLD at 38.6% among adults and demonstrated substantially higher prevalence in high-risk metabolic groups. De et al. (2023)[4] further observed that lean Indian patients could have liver-disease severity comparable with that of non-lean patients despite having fewer metabolic risk factors. These observations are important in Asian populations, in whom visceral adiposity and metabolic dysfunction may occur at a comparatively lower BMI.

Type 2 diabetes, hypertension and dyslipidaemia affected 35.8%, 30.8% and 40.8% of the study population, respectively. Dyslipidaemia was significantly more frequent than the specified reference prevalence. The coexistence of these abnormalities supports the concept that fatty liver is a hepatic manifestation of systemic metabolic dysfunction. The AASLD guidance by Rinella et al. (2023)[5] identifies obesity, type 2 diabetes, hypertension and dyslipidaemia as major risk factors for NAFLD development and progression. Similarly, the Asian-Pacific guidance by Eslam et al. (2020)[6] emphasised the close association between fatty liver and overweight or obesity, diabetes and other features of metabolic dysregulation. The 2024 EASL-EASD-EASO guideline also recommends assessment for advanced liver disease particularly in individuals with type 2 diabetes, abdominal obesity or multiple cardiometabolic risk factors [7]. The metabolic profile observed in the present study therefore represents a clinically important population in whom cardiovascular risk and hepatic fibrosis risk should both be evaluated.

Grade I fatty liver was the most common category, affecting 44.2% of patients, followed by grade II in 34.2% and grade III in 21.7%. The significantly unequal distribution, with decreasing frequency as severity increased, is consistent with the natural distribution of the disease, because simple or mild steatosis is more common than severe steatosis. Zafar et al. (2024)[8] also reported grade I fatty liver as the most common ultrasonographic grade in their cross-sectional evaluation. The comparatively substantial proportion of grade III disease in the present study may be explained by its tertiary-care, hospital-based setting, where patients with symptoms, abnormal biochemical findings or metabolic comorbidities are more likely to undergo imaging.

Elevated ALT was the most frequent biochemical abnormality, occurring in 42.5% of patients, followed by GGT in 36.7%, AST in 32.5% and ALP in 23.3%. Sanyal et al. (2015)[9] similarly found that ALT and GGT were significantly related to fatty liver among patients with impaired glucose tolerance and newly diagnosed type 2 diabetes. Swain et al. (2017)[10] reported higher transaminase levels among Indian patients with NAFLD but also demonstrated that aminotransferase values did not consistently reflect histological severity. Consequently, although raised ALT and AST support the presence of hepatocellular injury, normal enzyme levels cannot exclude steatohepatitis or clinically

important fibrosis. This limitation is also emphasised in contemporary AASLD guidance [5].

Hyperbilirubinaemia and hypoalbuminaemia were less frequent, affecting 14.2% and 10.8% of patients, respectively. These abnormalities were substantially less common than transaminase elevation because bilirubin excretion and albumin synthesis are usually preserved in uncomplicated steatosis. Their increasing frequency across grades, however, suggests that some patients with severe ultrasonographic disease may have had more advanced hepatic dysfunction or unmeasured fibrosis. Because conventional ultrasonography does not accurately stage fibrosis, patients with hypoalbuminaemia or hyperbilirubinaemia require evaluation for other hepatic diseases, fibrosis, cirrhosis, malnutrition, renal protein loss and systemic inflammation before these abnormalities are attributed solely to NAFLD.

Ultrasonographic classification of fatty liver

Increased hepatic echogenicity was present in every patient because it was the defining ultrasonographic criterion for study inclusion. Increased hepatorenal contrast was observed in 93.3% and became more frequent from grade I to grade III, although the difference did not reach statistical significance. Conventional B-mode ultrasonography identifies hepatic steatosis through increased parenchymal echogenicity relative to the renal cortex, beam attenuation, loss of vascular definition and reduced visualisation of the diaphragm. Ferraioli and Monteiro (2019)[11] described these as the principal qualitative sonographic indicators of hepatic fat accumulation. They also noted that conventional ultrasound has limited sensitivity for mild steatosis and provides a subjective rather than direct quantitative estimate of hepatic fat.

Mild impairment of intrahepatic vessel visualisation was most common in grade II disease, whereas poor vessel visualisation was overwhelmingly associated with grade III disease. The apparently lower frequency of “mild impairment” in grade III should not be interpreted as improvement; patients with grade III disease had progressed from mild impairment to the more severe category of poorly visualised vessels. Poor vessel visualisation increased from 1.9% in grade I to 92.3% in grade III. Posterior acoustic attenuation similarly increased from 13.2% to 92.3%, reduced diaphragmatic visualisation from 5.7% to 88.5%, and poor visualisation of the posterior right hepatic lobe from 1.9% to 84.6%. These strong graded relationships support the internal consistency of the ultrasonographic classification.

Kozłowska-Petriczko et al. (2021)[12] found that ultrasound-based methods performed better than several non-imaging indices for detecting steatosis when controlled attenuation parameter was used as the reference. Nevertheless, qualitative B-mode grading is influenced by operator experience, machine settings, body habitus and coexisting fibrosis. Quantitative ultrasound methods, including attenuation measurement and hepatorenal-index techniques, may provide more objective assessment. Jang et al. (2021)[13] demonstrated good diagnostic performance of ultrasound attenuation coefficients for detecting and grading hepatic steatosis. Thus, the present conventional grading system is clinically practical, but the use of quantitative attenuation, controlled attenuation parameter, MRI-proton density fat fraction or histology in future studies would strengthen validation.

Hepatomegaly was observed in 64.2% of participants and increased significantly from 50.9% in grade I to 80.8% in grade III. The mean liver span also increased from 14.62±1.37 cm to 16.38±1.63 cm, with a grade III-grade I difference of 1.76 cm. This probably reflects increasing triglyceride accumulation and hepatocyte enlargement.

The mean portal vein diameter also rose significantly, from 10.18±1.06 mm in grade I to 11.34±1.28 mm in grade III. Although this finding may indicate progressive structural or haemodynamic changes, the observed mean values alone do not establish portal hypertension. Portal hypertension should be assessed using additional findings such as splenomegaly, collateral vessels, portal-flow velocity, thrombocytopenia, elastography and clinical evidence of advanced chronic liver disease.

Focal fatty sparing and gallbladder calculi were not significantly associated with ultrasonographic grade. Focal sparing reflects heterogeneous fat distribution rather than necessarily indicating greater overall steatosis. Similarly, gallstone formation shares certain metabolic risk factors with NAFLD, including obesity and dyslipidaemia, but is not an intrinsic component of ultrasonographic fatty liver grading. Their nonsignificant relationships therefore appear clinically plausible.

Liver function tests across ultrasonographic grades

A progressive and statistically significant increase in bilirubin was observed across grades I-III. Total bilirubin increased from 0.71±0.23 mg/dL in grade I to 1.14±0.48 mg/dL in grade III, while direct and indirect bilirubin also rose significantly. Bilirubin usually remains within the reference range in uncomplicated NAFLD. Therefore, the significant increase observed in grade III patients may indicate greater hepatocellular dysfunction, impaired biliary handling or the presence of advanced disease in a subgroup. It should not be assumed that ultrasonographic steatosis alone caused hyperbilirubinaemia without excluding haemolysis, biliary obstruction, drug-induced injury and other liver diseases.

AST increased from 29.46±9.38 U/L in grade I to 58.31±22.47 U/L in grade III, while ALT increased from 34.18±12.46 U/L to 76.42±28.36 U/L. The differences between grades III and I were 28.85 U/L for AST and 42.24 U/L for ALT, and both were statistically significant. Khodadoostan et al. (2016)[14] likewise observed significantly higher ALT and AST levels among patients with moderate sonographic NAFLD than among those with less severe findings. Sanyal et al. (2015)[9] also demonstrated significant relationships of ALT and GGT with fatty liver. These studies support the present finding that increasing sonographic severity is accompanied by greater hepatocellular enzyme release.

ALT remained higher than AST across all three grades, and the AST/ALT ratio remained below 1.0. This is a commonly reported biochemical pattern in earlier or metabolically driven NAFLD. However, the AST/ALT ratio did not differ significantly among grades (p=0.150). The ratio may increase when fibrosis becomes advanced because ALT can decline while AST remains elevated, but ultrasonographic steatosis grade is not equivalent to fibrosis stage. Thus, the absence of a significant relationship between the AST/ALT ratio and ultrasonographic grade is reasonable and reinforces the distinction between hepatic fat severity and fibrosis severity.

ALP increased from 91.72±24.18 U/L in grade I to 139.27±42.46 U/L in grade III, and GGT increased from 31.64±13.27 U/L to 72.53±32.81 U/L. Mansour-Ghanaei et al. (2019)[15] similarly reported significant biochemical and metabolic abnormalities among patients with NAFLD in the PERSIAN Guilan cohort. GGT is particularly related to oxidative stress, insulin resistance and metabolic risk, but both GGT and ALP are nonspecific. Their elevation may arise from biliary disease, medication use and other hepatic or extrahepatic conditions. Nevertheless, the consistent grade-related increases in the present study suggest that these markers may complement ALT and

AST when assessing patients with more severe ultrasonographic steatosis.

Total protein decreased significantly across the grades, while serum albumin declined from 4.31±0.39 g/dL in grade I to 3.79±0.53 g/dL in grade III. The albumin/globulin ratio also fell significantly, whereas globulin did not differ. Albumin is generally preserved in simple steatosis and early steatohepatitis because of its long half-life and substantial hepatic synthetic reserve. A reduction in albumin among patients with grade III disease may identify a subgroup with advanced fibrosis, chronic inflammation, poor nutritional status or another systemic illness. Swain et al. (2017)[10] showed that biochemical abnormalities alone were insufficient to predict histological severity. Therefore, lower albumin should be considered a warning sign that warrants fibrosis assessment, but not as proof that severe steatosis has directly caused impaired synthesis.

Correlation between fatty liver grade and liver function tests

ALT showed the strongest correlation with ultrasonographic grade (ρ=0.56), followed by GGT (ρ=0.51) and AST (ρ=0.48); all were statistically significant. These moderate correlations indicate that worsening ultrasonographic steatosis was generally accompanied by increasing enzyme levels, although the relationships were not sufficiently strong for liver enzymes to serve as substitutes for imaging. Zafar et al. (2024)[8] also found correlations between ultrasound grades and several blood parameters, although the correlations of ALT and AST with grade were relatively weaker than those observed in the present study. Differences in patient selection, grade distribution, metabolic comorbidities, timing of laboratory testing and ultrasound interpretation may explain the variation between studies.

Sanyal et al. (2015)[9] reported significant positive correlations of fatty liver with ALT and GGT, agreeing with the present findings. Khodadoostan et al. (2016)[14] also demonstrated higher AST and ALT values in moderate sonographic disease. In contrast, histopathological studies have shown that enzyme elevation may not increase linearly with steatohepatitis or fibrosis severity [10]. These observations suggest that ALT, AST and GGT are useful markers of current hepatocellular or metabolic stress but are imperfect measures of the underlying histological stage.

Direct bilirubin and ALP had moderate positive correlations with grade, while total and indirect bilirubin showed weak-to-moderate positive correlations. These findings suggest that greater steatosis may be accompanied by mild impairment of hepatic excretory function. However, the correlation coefficients remained below 0.50, and alternative causes of bilirubin or ALP elevation should be excluded. Globulin and the AST/ALT ratio showed no significant correlation with ultrasonographic grade, indicating that these parameters were comparatively insensitive to increasing steatosis in this population.

Total protein, albumin and the albumin/globulin ratio demonstrated significant negative correlations with ultrasonographic grade. The magnitude of these correlations was weak, showing considerable overlap between the grades. The negative association of albumin may nevertheless have clinical importance because declining albumin could suggest impaired hepatic reserve or comorbid nutritional and inflammatory conditions. Current guidelines recommend that risk stratification should not depend on isolated liver enzyme or protein measurements. Rinella et al. (2023)[5] and the EASL-EASD-EASO guideline (2024)[7] recommend stepwise fibrosis assessment using validated

non-invasive scores commonly FIB-4 followed, when indicated, by vibration-controlled transient elastography, other elastographic methods or specialist evaluation.

CONCLUSION

The study demonstrated a significant relationship between increasing ultrasonographic fatty liver grade and derangement of liver function-test parameters among patients with NAFLD. ALT showed the strongest positive correlation with ultrasonographic grade, followed by GGT and AST. Bilirubin and ALP also increased significantly, whereas serum total protein, albumin and the albumin/globulin ratio decreased as the ultrasonographic severity progressed. Grade III fatty liver was characterised by more pronounced posterior acoustic attenuation, impaired visualisation of intrahepatic vessels and the diaphragm, hepatomegaly, and higher portal vein diameter and liver span. These findings indicate that ultrasonographic grading, when interpreted together with biochemical parameters, can assist in assessing disease severity and identifying patients who require further evaluation. Nevertheless, neither conventional ultrasonography nor routine liver function tests can reliably differentiate simple steatosis from steatohepatitis or accurately stage hepatic fibrosis. Patients with severe steatosis, persistent enzyme elevation, hypoalbuminaemia or metabolic risk factors should therefore undergo fibrosis-specific assessment and appropriate clinical follow-up.

LIMITATIONS OF STUDY

1. The cross-sectional design identified associations but could not establish a temporal or causal relationship between increasing hepatic steatosis and liver function-test abnormalities.
2. The study was conducted at a single tertiary-care hospital; therefore, the findings may not be generalisable to the wider community or other geographical populations.
3. The relatively small sample size, particularly the limited number of patients with grade III disease, may have reduced the precision of subgroup estimates.
4. Consecutive hospital-based sampling may have introduced referral and selection bias, with more symptomatic or metabolically complicated patients being included.
5. Conventional ultrasonographic grading is subjective and depends on the operator's experience, equipment settings, patient body habitus and image quality.
6. Interobserver and intraobserver agreement for ultrasonographic grading was not quantified using kappa statistics.
7. Ultrasonography has limited sensitivity for mild hepatic steatosis and cannot accurately quantify liver fat or distinguish simple steatosis from steatohepatitis.
8. Liver biopsy, which is the reference standard for diagnosing steatohepatitis and staging fibrosis, was not performed.
9. Quantitative imaging methods such as controlled attenuation parameter, shear-wave elastography, transient elastography or MRI-proton density fat fraction were not used.
10. Routine liver function tests are nonspecific and may be affected by medications, diet, metabolic control, subclinical infections and other medical conditions.
11. A single liver function-test measurement may not reflect persistent biochemical abnormalities or their variation over time.
12. Important fibrosis-related variables such as platelet count, FIB-4, NAFLD fibrosis score and liver stiffness were not included.
13. Potential confounders, including duration and control of diabetes, waist circumference, dietary pattern, physical activity and detailed lipid profile, may not have been completely adjusted.

14. Patients with normal aminotransferase levels may still have steatohepatitis or advanced fibrosis; therefore, biochemical results may have underestimated disease severity.
15. Long-term outcomes, including progression to fibrosis, cirrhosis or improvement following treatment, could not be evaluated.

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