Section: Radiology

Original Research Article

CORRELATION OF ULTRASONOGRAPHIC FATTY LIVER GRADING WITH LIVER FUNCTION TESTS AND METABOLIC PARAMETERS: A CROSS-SECTIONAL STUDY

N. Aarthi Reddy[1], Divya Raju Alluri[2], Harshavardhan Reddy G.[3]

[1]*Associate Professor, Department of Radiology, SVS Medical college, Yenugonda, Mahabubnagar, Telangana 509002, India.*
[2]*Associate Professor, Department of Radiology, SVS Medical college, Yenugonda, Mahabubnagar, Telangana 509002, India.*
[3]*Assistant professor, Department of Radiology, SVS Medical college, Yenugonda, Mahabubnagar, Telangana 509002, India.*

Received *: 02/08/2026*
Received in revised form *: 01/09/2026*
Accepted *: 12/09/2026*

Corresponding Author:
Dr. N Aarthi Reddy,
Associate Professor, Department of Radiology, SVS Medical college, Yenugonda, Mahabubnagar, Telangana 509002, India.
Email: draarthi.reddy@gmail.com

DOI: 10.70034/ijmedph.2026.4.262

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 1724-1733

ABSTRACT

Background: Fatty liver disease is closely associated with obesity, insulin resistance, diabetes mellitus and dyslipidaemia. Ultrasonography is commonly used for its detection and grading, although the relationship between ultrasonographic severity and biochemical or metabolic abnormalities requires further evaluation.
Aim: To assess the correation of ultrasonographic fatty liver grading with liver function tests and metabolic parameters among adult patients.
Materials and Methods: This hospital-based cross-sectional study included 200 adults with ultrasonographically detected fatty liver. Demographic and clinical information was recorded, and anthropometric measurements and blood pressure were assessed. Participants underwent liver function tests, fasting blood glucose, glycated haemoglobin and lipid-profile estimation. Fatty liver was ultrasonographically categorized as Grade I, Grade II or Grade III. Differences across grades were analyzed using one-way analysis of variance and the chi-square test. Spearman's correlation analysis and multivariable ordinal logistic regression were used to identify factors associated with increasing fatty liver grade. A p value below 0.05 was considered statistically significant.
Results: The mean age of participants was 49.6±10.8 years, and 58.5% were male. Grade I fatty liver was present in 46.5%, Grade II in 35.5% and Grade III in 18.0%. The prevalence of obesity increased from 33.3% in Grade I to 83.3% in Grade III, diabetes mellitus from 29.0% to 72.2%, dyslipidaemia from 45.2% to 86.1%, and metabolic syndrome from 30.1% to 83.3% (all $p<0.001$). BMI, waist circumference, blood pressure, bilirubin, AST, ALT, alkaline phosphatase, GGT, fasting glucose, HbA1c, total cholesterol, triglycerides and LDL cholesterol increased significantly with increasing fatty liver grade, whereas serum albumin and HDL cholesterol decreased significantly (all $p<0.001$). The strongest positive correlations were observed with waist circumference ($\rho=0.62$), ALT ($\rho=0.59$), BMI ($\rho=0.58$), GGT ($\rho=0.57$), HbA1c ($\rho=0.56$) and triglycerides ($\rho=0.55$); all $p<0.001$. On multivariable analysis, BMI (aOR=1.18; 95% CI: 1.08-1.29), waist circumference per 5-cm increase (aOR=1.31; 95% CI: 1.12-1.54), diabetes mellitus (aOR=1.87; 95% CI: 1.04-3.38), ALT, GGT, HbA1c and triglycerides remained independently associated with increasing fatty liver grade. Higher HDL cholesterol was inversely associated with severity (aOR=0.79; 95% CI: 0.66-0.94).
Conclusion: Higher ultrasonographic fatty liver grades were associated with greater central adiposity, impaired glycaemic control, elevated liver enzymes and an adverse lipid profile. Combining ultrasonographic grading with hepatic and metabolic assessment may help identify patients requiring intensive risk-factor modification and further evaluation for advanced liver disease.
Keywords: Fatty liver; Ultrasonography; Metabolic parameters.

INTRODUCTION

Fatty liver disease is characterized by excessive accumulation of triglycerides within hepatocytes and has emerged as one of the most common causes of chronic liver disease worldwide. The condition is now commonly described as metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease, when hepatic steatosis occurs in association with cardiometabolic risk factors and without another predominant cause of liver injury.[1] Its prevalence has increased substantially because of the rising burden of obesity, type 2 diabetes mellitus, dyslipidaemia, hypertension and sedentary lifestyles. A recent meta-analysis estimated that fatty liver disease affects approximately one-third of the global adult population, making it an important clinical and public-health concern.[2]

The clinical spectrum of fatty liver disease ranges from simple hepatic steatosis to steatohepatitis, progressive fibrosis, cirrhosis and hepatocellular carcinoma. Most patients remain asymptomatic during the early stages and are frequently diagnosed incidentally during abdominal ultrasonography or evaluation of abnormal liver function tests. Although alanine aminotransferase and aspartate aminotransferase may be elevated, normal liver enzyme levels do not exclude clinically significant steatosis or fibrosis.[3] Therefore, assessment based solely on liver function tests may underestimate the presence and severity of the disease.

Ultrasonography is widely used as the initial imaging modality for detecting fatty liver because it is non-invasive, inexpensive, readily available and free from ionizing radiation. Fatty infiltration is identified by increased hepatic echogenicity, hepatorenal contrast, posterior beam attenuation and reduced visualization of intrahepatic vessels and the diaphragm. Based on these features, hepatic steatosis can be categorized as Grade 0, Grade I, Grade II or Grade III. However, conventional ultrasonography has limited sensitivity for mild steatosis and its grading may be affected by operator dependence and coexisting fibrosis.[4]

Fatty liver is closely associated with central obesity, insulin resistance, hyperglycaemia, elevated triglycerides and reduced high-density lipoprotein cholesterol. Increasing ultrasonographic severity may therefore be accompanied by progressive abnormalities in liver enzymes and metabolic parameters. Identifying these relationships could help clinicians recognize patients at greater cardiometabolic and hepatic risk and prioritize them for further evaluation, lifestyle modification and follow-up.[5] The present study was consequently undertaken to determine the correlation of ultrasonographic fatty liver grading with liver function tests, anthropometric measurements, glycaemic indices and lipid parameters.

Aim

To assess the correlation of ultrasonographic fatty liver grading with liver function tests and metabolic parameters among adult patients.

Objectives

1. To determine the ultrasonographic grades of fatty liver among the study participants.
2. To assess liver function tests, anthropometric indices, glycaemic parameters and lipid profiles among patients with different grades of fatty liver.
3. To determine the correlation and association of ultrasonographic fatty liver grade with liver function tests and metabolic parameters.

MATERIALS AND METHODS

Source of Data

The study participants were recruited from adult patients attending the outpatient departments and inpatient services of the Departments of General Medicine and Gastroenterology. Patients referred to the Department of Radiodiagnosis for abdominal ultrasonography and diagnosed with fatty liver were evaluated for eligibility. Relevant demographic, clinical, anthropometric, biochemical and ultrasonographic information was obtained from the participants, clinical examination, laboratory reports and hospital records.

Study Design

The study was a hospital-based observational cross-sectional study.

Study Location

The study was conducted jointly in the Departments of General Medicine, Gastroenterology, Radiodiagnosis and Biochemistry.

Study Duration

The study was carried out over 12 months, including participant recruitment, investigations, data collection, statistical analysis and report preparation.

Sample Size

A total of 200 eligible participants with ultrasonographically detected fatty liver were included in the study. The sample size was estimated using the single-proportion formula:

$$n = \frac{Z_{1-\alpha/2}^2 \times p(1-p)}{d^2}$$

where $Z_{1-\alpha/2} = 1.96$at a 95% confidence level, $p$represented the anticipated prevalence of fatty liver and $d$represented the allowable absolute error. After allowing for incomplete investigations and records, the final sample size was rounded to 200 participants. Consecutive eligible patients were enrolled until the required sample size was achieved.

Inclusion Criteria

Participants were included when they fulfilled the following criteria:

1. Adults aged 18 years or older.
2. Patients attending the selected outpatient or inpatient departments during the study period.

3. Presence of fatty liver on abdominal ultrasonography.
4. Availability of liver function tests, fasting blood glucose and lipid profile measurements.
5. Participants who provided written informed consent.

Exclusion Criteria

The following patients were excluded:

1. Patients with significant alcohol consumption, defined as more than 20 g/day among women or more than 30 g/day among men.
2. Patients with hepatitis B, hepatitis C or another known chronic viral hepatitis.
3. Patients with autoimmune hepatitis, Wilson disease, haemochromatosis or other established chronic liver diseases.
4. Patients with decompensated cirrhosis, hepatic malignancy or biliary obstruction.
5. Patients receiving drugs known to cause hepatic steatosis, such as corticosteroids, amiodarone, tamoxifen, methotrexate or valproate.
6. Pregnant or lactating women.
7. Patients with acute febrile illness, acute hepatitis or severe systemic disease at the time of evaluation.
8. Patients with incomplete clinical, ultrasonographic or biochemical information.
9. Patients who declined to provide informed consent.

Procedure and Methodology

Approval was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from each participant. Confidentiality of participants' information was maintained throughout the study.

A detailed clinical history was recorded, including age, sex, presenting complaints, duration of illness, dietary habits, physical activity, alcohol consumption, smoking, diabetes mellitus, hypertension, dyslipidaemia, medication use and family history of metabolic disease. A general and systemic examination was performed.

Body weight was measured to the nearest 0.1 kg using a calibrated weighing scale, with the participant wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm using a stadiometer. Body mass index was calculated as:

$$\text{BMI} = \frac{\text{Weight in kg}}{\text{Height in metres}^2}$$

Waist circumference was measured midway between the lowest rib and iliac crest at the end of normal expiration. Blood pressure was measured in the sitting position after at least five minutes of rest using an appropriately sized cuff. Two readings were obtained, and their average was recorded.

Abdominal ultrasonography was performed after fasting for six to eight hours using a standard ultrasound machine equipped with a 3-5 MHz curvilinear transducer. Whenever feasible, examinations were performed by the same experienced radiologist, who was unaware of the biochemical findings. Hepatic echogenicity was compared with the renal cortex, and visualization of the diaphragm, portal vein walls and intrahepatic vessels was assessed.

Ultrasonographic fatty liver was graded as follows:

- **Grade 0:** Normal hepatic echogenicity with clear visualization of the diaphragm and intrahepatic vessels.
- **Grade I (mild):** Slight, diffuse increase in hepatic echogenicity with normal visualization of the diaphragm and portal vein borders.
- **Grade II (moderate):** Moderate increase in echogenicity with mildly impaired visualization of the intrahepatic vessels and diaphragm.
- **Grade III (severe):** Marked increase in echogenicity with poor or absent visualization of the posterior liver, diaphragm and intrahepatic vessels.

The ultrasonographic grade was subsequently compared with liver function tests and metabolic parameters.

Sample Processing

After an overnight fast of eight to twelve hours, approximately 8-10 mL of venous blood was collected under aseptic precautions. Blood for serum-based investigations was transferred into a plain or serum-separator tube, allowed to clot and centrifuged at approximately 3,000 revolutions per minute for 10 minutes. The separated serum was analyzed using a calibrated automated biochemistry analyzer.

Liver function testing included total bilirubin, direct bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, gamma-glutamyl transferase, total protein, serum albumin and globulin. The metabolic assessment included fasting blood glucose, glycated haemoglobin, total cholesterol, triglycerides, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. Additional blood collected in an ethylenediaminetetraacetic acid tube was used for glycated haemoglobin estimation. Samples that were hemolyzed, insufficient or improperly labelled were not analyzed, and repeat samples were obtained wherever appropriate.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 28.0. Continuous variables were assessed for normality using the Shapiro-Wilk test and graphical methods. Normally distributed variables were summarized as mean and standard deviation with 95% confidence intervals, whereas skewed variables were presented as median and interquartile range. Categorical variables were expressed as frequency and percentage.

Differences in normally distributed continuous parameters across ultrasonographic fatty liver grades were assessed using one-way analysis of variance, followed by an appropriate post-hoc test. The Kruskal-Wallis test was used for non-normally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact

test, as appropriate. A chi-square test for trend was applied to ordered categorical variables where relevant.

Because ultrasonographic fatty liver grade was an ordinal variable, its correlation with liver function tests, BMI, waist circumference, blood glucose, HbA1c and lipid parameters was evaluated primarily using Spearman's rank correlation coefficient. Pearson's correlation coefficient was used only when both variables fulfilled the assumptions of parametric analysis. Multivariable ordinal logistic regression was performed, where appropriate, to identify metabolic and biochemical factors independently associated with increasing fatty liver grade. Adjusted odds ratios were reported with 95% confidence intervals. All tests were two-tailed, and a p value below 0.05 was considered statistically significant.

Data Collection

Data were collected prospectively using a predesigned, pretested case-record form. The form included the following sections:

1. Sociodemographic information.
2. Presenting complaints and relevant medical history.
3. Alcohol, smoking, dietary and physical-activity history.
4. Anthropometric measurements and blood pressure.
5. Presence of diabetes, hypertension, obesity and dyslipidaemia.
6. Ultrasonographic findings and fatty liver grade.
7. Liver function test results.
8. Fasting blood glucose and glycated haemoglobin.
9. Serum lipid profile.
10. Final clinical and metabolic assessment.

Each participant was assigned a unique identification number. Completed forms were checked for accuracy and completeness before data entry. Data were stored securely and were accessible only to the investigators.

RESULTS

Table 1: Overall demographic, clinical, biochemical and ultrasonographic profile of participants (N=200)

Study parameter	n (%) or Mean (SD)	95% CI	Test statistic	p value
Age, years	49.6 (10.8)	48.09-51.11		
Age group			χ^2=18.01	<0.001
18-39 years	42 (21.0)	15.9-27.2		
40-59 years	91 (45.5)	38.7-52.4		
≥60 years	67 (33.5)	27.3-40.3		
Sex			χ^2=5.78	0.016
Male	117 (58.5)	51.6-65.1		
Female	83 (41.5)	34.9-48.4		
Body mass index, kg/m²	28.7 (4.3)	28.10-29.30		
Waist circumference, cm	96.8 (10.7)	95.31-98.29		
Systolic blood pressure, mmHg	131.6 (15.4)	129.45-133.75		
Diastolic blood pressure, mmHg	82.9 (9.1)	81.63-84.17		
Obesity	103 (51.5)	44.6-58.3	χ^2=0.18	0.671
Diabetes mellitus	89 (44.5)	37.8-51.4	χ^2=2.42	0.120
Hypertension	96 (48.0)	41.2-54.9	χ^2=0.32	0.572
Dyslipidaemia	122 (61.0)	54.1-67.5	χ^2=9.68	0.002
Metabolic syndrome	97 (48.5)	41.7-55.4	χ^2=0.18	0.671
Fasting blood glucose, mg/dL	119.8 (32.6)	115.26-124.34		
HbA1c, %	6.7 (1.4)	6.51-6.89		
Total cholesterol, mg/dL	194.7 (41.8)	188.88-200.52		
Triglycerides, mg/dL	171.6 (66.4)	162.35-180.85		
HDL cholesterol, mg/dL	41.2 (8.7)	39.99-42.41		
LDL cholesterol, mg/dL	119.3 (35.7)	114.33-124.27		
Total bilirubin, mg/dL	0.84 (0.31)	0.80-0.88		
AST, U/L	38.6 (17.4)	36.18-41.02		
ALT, U/L	47.9 (23.8)	44.59-51.21		
Alkaline phosphatase, U/L	108.7 (34.6)	103.88-113.52		
Gamma-glutamyl transferase, U/L	52.8 (31.7)	48.39-57.21		
Serum albumin, g/dL	4.18 (0.43)	4.12-4.24		
Ultrasonographic fatty liver grade			χ^2=24.79	<0.001
Grade I	93 (46.5)	39.7-53.4		
Grade II	71 (35.5)	29.2-42.4		
Grade III	36 (18.0)	13.3-23.9		

Table 1 presents the demographic, clinical, biochemical and ultrasonographic characteristics of 200 participants. Their mean age was 49.6±10.8 years (95% CI: 48.09-51.11), with the largest proportion aged 40-59 years (45.5%), followed by ≥60 years (33.5%) and 18-39 years (21.0%). The age-group distribution was statistically significant (χ^2=18.01, p<0.001). Males constituted 58.5% of participants compared with 41.5% females, indicating significant male predominance (χ^2=5.78, p=0.016). The mean BMI was 28.7±4.3 kg/m², and the mean waist circumference was 96.8±10.7 cm. Obesity was present in 51.5%, diabetes mellitus in 44.5%, hypertension in 48.0%, dyslipidaemia in

61.0% and metabolic syndrome in 48.5% of participants. Dyslipidaemia occurred significantly more frequently than its absence (χ^2=9.68, p=0.002), whereas the corresponding distributions for obesity, diabetes, hypertension and metabolic syndrome were not statistically significant. The mean fasting blood glucose and HbA1c were 119.8±32.6 mg/dL and 6.7±1.4%, respectively. The mean total cholesterol was 194.7±41.8 mg/dL, triglycerides 171.6±66.4 mg/dL, HDL cholesterol 41.2±8.7 mg/dL and LDL cholesterol 119.3±35.7 mg/dL. Regarding liver function, the mean AST was 38.6±17.4 U/L, ALT 47.9±23.8 U/L, alkaline phosphatase 108.7±34.6 U/L, GGT 52.8±31.7 U/L and serum albumin 4.18±0.43 g/dL. Grade I fatty liver was the most frequent ultrasonographic grade, affecting 93 (46.5%) participants, followed by Grade II in 71 (35.5%) and Grade III in 36 (18.0%). The distribution of fatty liver grades was statistically significant (χ^2=24.79, p<0.001).

Table 2: Distribution of ultrasonographic fatty liver grades and associated ultrasound findings (N=200)

Ultrasonographic finding	Grade I (n=93), n (%)	Grade II (n=71), n (%)	Grade III (n=36), n (%)	Total, n (%)	95% CI of total (%)	Test statistic	p value
Fatty liver grade	93 (46.5)	71 (35.5)	36 (18.0)	200 (100.0)		χ^2=24.79†	<0.001
Increased hepatic echogenicity	93 (100.0)	71 (100.0)	36 (100.0)	200 (100.0)	98.1-100.0		
Increased hepatorenal contrast	67 (72.0)	66 (93.0)	36 (100.0)	169 (84.5)	78.8-88.9	χ^2=24.69	<0.001
Posterior beam attenuation	14 (15.1)	57 (80.3)	36 (100.0)	107 (53.5)	46.6-60.3	χ^2=96.82	<0.001
Impaired visualization of vessels	8 (8.6)	39 (54.9)	29 (80.6)	76 (38.0)	31.6-44.9	χ^2=66.18	<0.001
Impaired visualization of diaphragm	6 (6.5)	31 (43.7)	31 (86.1)	68 (34.0)	27.8-40.8	χ^2=77.34	<0.001
Hepatomegaly	21 (22.6)	38 (53.5)	28 (77.8)	87 (43.5)	36.8-50.4	χ^2=36.47	<0.001

†Chi-square goodness-of-fit test comparing the observed grade distribution against equal expected proportions. Other comparisons used the Pearson chi-square test across fatty liver grades.

Table 2 demonstrates a progressive increase in abnormal ultrasonographic findings with increasing fatty liver grade. Increased hepatic echogenicity was observed in all 200 participants. Increased hepatorenal contrast was present in 72.0% of Grade I, 93.0% of Grade II and all Grade III participants, showing a significant association with increasing grade (χ^2=24.69, p<0.001). Posterior beam attenuation increased markedly from 15.1% in Grade I to 80.3% in Grade II and 100.0% in Grade III (χ^2=96.82, p<0.001). Similarly, impaired visualization of intrahepatic vessels increased from 8.6% in Grade I to 54.9% in Grade II and 80.6% in Grade III (χ^2=66.18, p<0.001). Impaired visualization of the diaphragm was identified in only 6.5% of Grade I participants but increased to 43.7% in Grade II and 86.1% in Grade III (χ^2=77.34, p<0.001). Hepatomegaly also became progressively more frequent, increasing from 22.6% in Grade I to 53.5% in Grade II and 77.8% in Grade III (χ^2=36.47, p<0.001).

Table 3: Comparison of liver function tests, anthropometric indices, glycaemic parameters and lipid profiles according to fatty liver grade (N=200)

Study parameter	Grade I (n=93), Mean (SD); 95% CI	Grade II (n=71), Mean (SD); 95% CI	Grade III (n=36), Mean (SD); 95% CI	Test statistic	p value
Age, years	46.2 (9.8); 44.18-48.22	50.7 (10.1); 48.31-53.09	56.1 (9.6); 52.85-59.35	F=13.44	<0.001
BMI, kg/m²	26.4 (3.2); 25.74-27.06	29.3 (3.6); 28.45-30.15	33.4 (4.1); 32.01-34.79	F=55.63	<0.001
Waist circumference, cm	90.8 (8.4); 89.07-92.53	98.7 (8.9); 96.59-100.81	108.5 (9.7); 105.22-111.78	F=59.87	<0.001
Systolic BP, mmHg	125.7 (13.6); 122.90-128.50	133.6 (14.1); 130.26-136.94	142.8 (16.2); 137.32-148.28	F=21.03	<0.001
Diastolic BP, mmHg	79.6 (8.1); 77.93-81.27	84.2 (8.7); 82.14-86.26	88.9 (9.4); 85.72-92.08	F=17.65	<0.001
Total bilirubin, mg/dL	0.72 (0.24); 0.67-0.77	0.86 (0.29); 0.79-0.93	1.12 (0.38); 0.99-1.25	F=26.18	<0.001
AST, U/L	29.7 (10.8); 27.48-31.92	40.9 (14.7); 37.42-44.38	57.1 (22.3); 49.56-64.64	F=46.76	<0.001
ALT, U/L	34.6 (15.7); 31.37-37.83	51.8 (19.6); 47.16-56.44	74.1 (28.4); 64.49-83.71	F=53.98	<0.001
Alkaline phosphatase, U/L	94.3 (27.8); 88.57-100.03	111.6 (31.4); 104.16-119.04	139.8 (39.2); 126.54-153.06	F=28.09	<0.001
GGT, U/L	35.4 (19.7); 31.34-39.46	56.2 (27.8); 49.61-62.79	88.7 (39.6); 75.30-102.10	F=50.71	<0.001
Serum albumin, g/dL	4.29 (0.36); 4.22-4.36	4.16 (0.41); 4.06-4.26	3.92 (0.49); 3.75-4.09	F=11.34	<0.001
Fasting glucose, mg/dL	102.6 (21.8); 98.11-107.09	124.9 (29.7); 117.86-131.94	154.1 (39.3); 140.80-167.40	F=39.89	<0.001
HbA1c, %	5.9 (0.8); 5.74-6.06	6.9 (1.2); 6.62-7.18	8.3 (1.6); 7.76-8.84	F=52.74	<0.001
Total cholesterol, mg/dL	176.3 (34.7); 169.15-183.45	202.8 (38.9); 193.58-212.02	226.4 (45.6); 210.97-241.83	F=27.16	<0.001

Triglycerides, mg/dL	137.8 (43.9); 128.76-146.84	183.7 (57.6); 170.05-197.35	236.9 (78.2); 210.44-263.36	F=43.52	<0.001
HDL cholesterol, mg/dL	45.7 (8.3); 43.99-47.41	39.3 (7.1); 37.62-40.98	33.4 (6.8); 31.10-35.70	F=38.97	<0.001
LDL cholesterol, mg/dL	103.2 (29.6); 97.10-109.30	125.8 (32.7); 118.05-133.55	149.6 (38.4); 136.61-162.59	F=31.63	<0.001

Categorical metabolic parameters according to fatty liver grade

Study parameter	Grade I (n=93), n (%)	Grade II (n=71), n (%)	Grade III (n=36), n (%)	Total, n (%)	Test statistic	p value
Obesity	31 (33.3)	42 (59.2)	30 (83.3)	103 (51.5)	χ^2=28.56	<0.001
Diabetes mellitus	27 (29.0)	36 (50.7)	26 (72.2)	89 (44.5)	χ^2=21.32	<0.001
Hypertension	30 (32.3)	40 (56.3)	26 (72.2)	96 (48.0)	χ^2=19.67	<0.001
Dyslipidaemia	42 (45.2)	49 (69.0)	31 (86.1)	122 (61.0)	χ^2=21.27	<0.001
Metabolic syndrome	28 (30.1)	39 (54.9)	30 (83.3)	97 (48.5)	χ^2=31.26	<0.001

Tests used: One-way ANOVA for continuous variables and Pearson chi-square test for categorical variables.

Table 3 shows that anthropometric measurements and blood pressure increased significantly with increasing fatty liver grade. Mean age increased from 46.2±9.8 years in Grade I to 50.7±10.1 years in Grade II and 56.1±9.6 years in Grade III (F=13.44, p<0.001). Mean BMI increased from 26.4±3.2 kg/m² in Grade I to 33.4±4.1 kg/m² in Grade III (F=55.63, p<0.001), while waist circumference increased from 90.8±8.4 cm to 108.5±9.7 cm (F=59.87, p<0.001). Mean systolic and diastolic blood pressures also increased significantly across the three grades (F=21.03 and F=17.65, respectively; both p<0.001).

Liver function parameters showed progressive abnormalities with increasing fatty liver severity. Mean AST increased from 29.7±10.8 U/L in Grade I to 57.1±22.3 U/L in Grade III (F=46.76, p<0.001), while ALT increased from 34.6±15.7 U/L to 74.1±28.4 U/L (F=53.98, p<0.001). Similar progressive increases were observed in total bilirubin, alkaline phosphatase and GGT, with all comparisons being statistically significant (p<0.001). In contrast, serum albumin decreased from 4.29±0.36 g/dL in Grade I to 3.92±0.49 g/dL in Grade III (F=11.34, p<0.001).

Glycaemic and lipid parameters also worsened significantly with increasing fatty liver grade. Mean fasting blood glucose increased from 102.6±21.8 mg/dL in Grade I to 154.1±39.3 mg/dL in Grade III (F=39.89, p<0.001), and mean HbA1c increased from 5.9±0.8% to 8.3±1.6% (F=52.74, p<0.001). Mean total cholesterol, triglycerides and LDL cholesterol increased progressively across the grades, whereas mean HDL cholesterol decreased from 45.7±8.3 mg/dL in Grade I to 33.4±6.8 mg/dL in Grade III (F=38.97, p<0.001). The prevalence of obesity increased from 33.3% in Grade I to 83.3% in Grade III (χ^2=28.56, p<0.001). Corresponding increases were found for diabetes mellitus (29.0% to 72.2%; χ^2=21.32), hypertension (32.3% to 72.2%; χ^2=19.67), dyslipidaemia (45.2% to 86.1%; χ^2=21.27) and metabolic syndrome (30.1% to 83.3%; χ^2=31.26); all associations were statistically significant at p<0.001.

Table 4: Correlation and multivariable association of liver function tests and metabolic parameters with ultrasonographic fatty liver grade (N=200)

A. Spearman correlation with fatty liver grade

Study parameter	Spearman's ρ	95% CI for ρ	p value
Age	0.31	0.18-0.43	<0.001
BMI	0.58	0.48-0.67	<0.001
Waist circumference	0.62	0.52-0.70	<0.001
Systolic blood pressure	0.37	0.24-0.48	<0.001
Diastolic blood pressure	0.32	0.19-0.44	<0.001
Total bilirubin	0.39	0.27-0.50	<0.001
AST	0.54	0.44-0.64	<0.001
ALT	0.59	0.49-0.67	<0.001
Alkaline phosphatase	0.42	0.30-0.52	<0.001
GGT	0.57	0.47-0.66	<0.001
Serum albumin	−0.29	−0.41 to −0.16	<0.001
Fasting blood glucose	0.51	0.40-0.61	<0.001
HbA1c	0.56	0.46-0.65	<0.001
Total cholesterol	0.41	0.29-0.52	<0.001
Triglycerides	0.55	0.45-0.64	<0.001
HDL cholesterol	−0.49	−0.59 to −0.38	<0.001
LDL cholesterol	0.44	0.32-0.54	<0.001

B. Ordinal logistic regression for factors associated with increasing fatty liver grade

Predictor	Adjusted odds ratio	95% CI	Wald χ^2	p value
Age, per 10-year increase	1.29	1.03-1.62	4.91	0.027
BMI, per 1 kg/m² increase	1.18	1.08-1.29	13.46	<0.001

Waist circumference, per 5-cm increase	1.31	1.12-1.54	11.18	0.001
Diabetes mellitus	1.87	1.04-3.38	4.34	0.037
Hypertension	1.46	0.81-2.63	1.61	0.205
ALT, per 10-U/L increase	1.24	1.09-1.42	10.17	0.001
GGT, per 10-U/L increase	1.17	1.05-1.30	8.18	0.004
HbA1c, per 1% increase	1.38	1.10-1.73	7.72	0.005
Triglycerides, per 25-mg/dL increase	1.21	1.08-1.36	10.32	0.001
HDL cholesterol, per 5-mg/dL increase	0.79	0.66-0.94	6.92	0.009
LDL cholesterol, per 10-mg/dL increase	1.08	0.99-1.18	3.04	0.081

Spearman correlation analysis demonstrated significant relationships between ultrasonographic fatty liver grade and all the evaluated anthropometric, biochemical and metabolic parameters. The strongest positive correlation was observed with waist circumference (ρ=0.62; 95% CI: 0.52-0.70; $p<0.001$), followed by ALT (ρ=0.59), BMI (ρ=0.58), GGT (ρ=0.57), HbA1c (ρ=0.56), triglycerides (ρ=0.55), AST (ρ=0.54) and fasting blood glucose (ρ=0.51); all $p<0.001$. Moderate positive correlations were also found with LDL cholesterol, alkaline phosphatase, total cholesterol, total bilirubin and blood pressure. Conversely, HDL cholesterol showed a moderate inverse correlation with fatty liver grade (ρ=−0.49; 95% CI: −0.59 to −0.38; $p<0.001$), while serum albumin showed a weaker but significant inverse correlation (ρ=−0.29; 95% CI: −0.41 to −0.16; $p<0.001$). Thus, higher fatty liver grades were accompanied by greater adiposity, poorer glycaemic control, higher liver enzyme levels and a more adverse lipid profile.

On multivariable ordinal logistic regression, BMI remained independently associated with increasing fatty liver grade, with an 18% increase in the odds of a higher grade for every 1 kg/m² increase in BMI (aOR=1.18; 95% CI: 1.08-1.29; $p<0.001$). A 5-cm increase in waist circumference was associated with 31% higher odds of greater fatty liver severity (aOR=1.31; 95% CI: 1.12-1.54; p=0.001). Diabetes mellitus was also an independent factor (aOR=1.87; 95% CI: 1.04-3.38; p=0.037). Significant biochemical predictors included ALT (aOR=1.24 per 10-U/L increase; p=0.001), GGT (aOR=1.17 per 10-U/L increase; p=0.004), HbA1c (aOR=1.38 per 1% increase; p=0.005) and triglycerides (aOR=1.21 per 25-mg/dL increase; p=0.001). Higher HDL cholesterol was independently protective against increasing severity (aOR=0.79 per 5-mg/dL increase; 95% CI: 0.66-0.94; p=0.009). Hypertension and LDL cholesterol did not remain independently significant after adjustment.

DISCUSSION

The present cross-sectional study evaluated the relationship between ultrasonographic fatty liver grade and hepatic, anthropometric, glycaemic and lipid parameters among 200 adults. The findings demonstrated a clear metabolic gradient: increasing ultrasonographic severity was accompanied by greater central and generalized obesity, higher blood pressure, worsening glycaemic control, an adverse lipid profile and progressive abnormalities in liver enzymes. Waist circumference, ALT, BMI, GGT, HbA1c and triglycerides showed the strongest positive correlations with fatty liver grade, while HDL cholesterol and serum albumin demonstrated inverse correlations.

Demographic, clinical and metabolic profile

The mean age of the participants was 49.6±10.8 years, and 45.5% were aged 40-59 years. Males constituted 58.5% of the study population. These findings indicate that ultrasonographically detected fatty liver was predominantly encountered among middle-aged adults and was relatively more common among men. Younossi et al. (2016),[1] in a systematic review and meta-analysis, similarly reported that fatty liver disease was commonly identified among middle-aged adults and was strongly associated with obesity and other components of metabolic syndrome. Sex-related differences in visceral adiposity, insulin sensitivity, sex hormones and lifestyle exposures may partially explain the observed male predominance. Lonardo et al. (2019),[2] also observed that fatty liver was generally more frequent and severe among men and postmenopausal women, although the effect of sex varied according to age and metabolic status.

The mean BMI was 28.7±4.3 kg/m², and the mean waist circumference was 96.8±10.7 cm, suggesting a considerable burden of generalized and central adiposity. Obesity was present in 51.5%, diabetes mellitus in 44.5%, hypertension in 48.0%, dyslipidaemia in 61.0% and metabolic syndrome in 48.5% of participants. The predominance of metabolic abnormalities in the present study is consistent with the contemporary concept of metabolic dysfunction-associated steatotic liver disease. Mitra et al. (2020),[3] reported that the increasing burden of fatty liver disease in India was closely related to urbanization, sedentary behaviour, central obesity, diabetes and dyslipidaemia. They also highlighted that Asian individuals may develop fatty liver at a comparatively lower BMI because of greater visceral adiposity and insulin resistance.

The high prevalence of diabetes and dyslipidaemia in the present study was clinically important. Godoy-Matos et al. (2020),[4] described fatty liver disease as part of a continuous metabolic spectrum extending from obesity and insulin resistance to metabolic syndrome and type 2 diabetes. Insulin resistance increases adipose-tissue lipolysis and hepatic delivery of free fatty acids, while hyperinsulinaemia promotes de novo lipogenesis. These mechanisms facilitate hepatic triglyceride accumulation and may account for the coexistence of obesity,

hyperglycaemia and dyslipidaemia observed in the present participants.

Grade I fatty liver was the most frequent ultrasonographic grade, occurring in 46.5% of participants, followed by Grade II in 35.5% and Grade III in 18.0%. The predominance of mild-to-moderate disease is consistent with the frequently asymptomatic nature of hepatic steatosis and its incidental detection during abdominal imaging. Younossi et al. (2023),[5] reported that fatty liver disease now affects a substantial proportion of the global adult population, although only a smaller subgroup progresses to steatohepatitis, advanced fibrosis or cirrhosis.

Ultrasonographic findings

Increased hepatic echogenicity was observed in all participants because it constituted the fundamental ultrasonographic feature of fatty infiltration. Other findings demonstrated a significant progressive pattern. Increased hepatorenal contrast rose from 72.0% in Grade I to 100.0% in Grade III. Posterior beam attenuation increased from 15.1% to 100.0%, impaired visualization of intrahepatic vessels from 8.6% to 80.6%, impaired visualization of the diaphragm from 6.5% to 86.1%, and hepatomegaly from 22.6% to 77.8%. All these findings were significantly associated with increasing fatty liver grade ($p<0.001$).

These observations correspond to the conventional ultrasonographic criteria used to grade hepatic steatosis. With greater hepatic fat accumulation, the difference in echogenicity between the liver and renal cortex increases, ultrasound-beam penetration becomes progressively reduced, and visualization of deeper hepatic structures becomes impaired. Ferraioli et al. (2021),[6] similarly described hepatic hyperechogenicity, hepatorenal contrast, posterior attenuation and reduced visualization of vascular structures as the principal sonographic features of steatosis.

The progressive increase in these findings supports the internal consistency of the grading system used in the present study. However, conventional B-mode ultrasonography remains dependent on operator experience, equipment settings and patient habitus. It is less sensitive for mild steatosis and cannot reliably distinguish simple steatosis from steatohepatitis or determine the stage of fibrosis. The EASL-EASD-EASO guidelines (2016),[7] therefore recommended interpreting ultrasonography together with metabolic risk factors, liver biochemistry and validated non-invasive fibrosis scores rather than using ultrasonography as an isolated measure of disease severity.

Anthropometric measurements and blood pressure

A progressive and statistically significant increase in BMI and waist circumference was observed across the ultrasonographic grades. Mean BMI increased from 26.4±3.2 kg/m² in Grade I to 33.4±4.1 kg/m² in Grade III, while waist circumference increased from 90.8±8.4 cm to 108.5±9.7 cm. Correspondingly, the prevalence of obesity increased from 33.3% in Grade I to 83.3% in Grade III. These findings demonstrated a strong relationship between the severity of adiposity and the ultrasonographic severity of fatty liver.

Central obesity is particularly important because visceral adipose tissue releases free fatty acids and pro-inflammatory adipokines directly into the portal circulation. This process promotes hepatic insulin resistance, oxidative stress and triglyceride accumulation. Chalasani et al. (2018),[8] identified obesity, particularly visceral adiposity, as one of the strongest clinical risk factors for the development and progression of fatty liver disease. The stronger correlation observed with waist circumference ($\rho=0.62$) than with BMI ($\rho=0.58$) in the present study suggests that the distribution of body fat may be more closely related to hepatic steatosis than body weight alone.

Systolic and diastolic blood pressures also increased significantly across the three grades. Hypertension was present in 32.3% of Grade I, 56.3% of Grade II and 72.2% of Grade III participants. Although hypertension was associated with grade on unadjusted analysis, it did not remain statistically significant in the multivariable model (aOR=1.46; $p=0.205$). This attenuation may have occurred because hypertension shares common pathways with obesity, insulin resistance and dyslipidaemia. Its effect may consequently have been partly explained by the stronger anthropometric and biochemical predictors included in the adjusted model.

Liver function tests

AST, ALT, alkaline phosphatase, GGT and total bilirubin showed significant progressive increases with increasing fatty liver grade. Mean ALT increased from 34.6±15.7 U/L in Grade I to 74.1±28.4 U/L in Grade III, while AST increased from 29.7±10.8 U/L to 57.1±22.3 U/L. GGT showed a similar increase from 35.4±19.7 U/L to 88.7±39.6 U/L. These findings suggest that increasing ultrasonographic steatosis was accompanied by greater hepatocellular and metabolic stress.

ALT had a moderately strong positive correlation with fatty liver grade ($\rho=0.59$), followed by GGT ($\rho=0.57$) and AST ($\rho=0.54$). After adjustment, every 10-U/L increase in ALT was associated with 24% higher odds of being in a more severe ultrasonographic grade, while every 10-U/L increase in GGT was associated with 17% higher odds. Rinella et al. (2023),[9] noted that aminotransferase elevation is commonly associated with metabolic fatty liver and may reflect hepatocellular injury; however, normal aminotransferase values do not exclude steatohepatitis or advanced fibrosis. Therefore, the observed correlations support the usefulness of these enzymes in risk assessment but do not establish them as independent measures of histological severity.

Serum albumin decreased from 4.29±0.36 g/dL in Grade I to 3.92±0.49 g/dL in Grade III and showed a significant inverse correlation with fatty liver grade

(ρ=−0.29). Although the mean albumin level remained relatively preserved, this downward trend may indicate increasing hepatic dysfunction, systemic inflammation, nutritional differences or associated comorbidity among participants with more severe ultrasonographic disease. Nevertheless, because albumin may be affected by renal disease, inflammation and nutritional status, the observed relationship should be interpreted cautiously.

Glycaemic parameters

Mean fasting blood glucose increased from 102.6±21.8 mg/dL in Grade I to 154.1±39.3 mg/dL in Grade III, while mean HbA1c increased from 5.9±0.8% to 8.3±1.6%. The prevalence of diabetes increased progressively from 29.0% in Grade I to 72.2% in Grade III. Fasting glucose and HbA1c were positively correlated with fatty liver grade, with correlation coefficients of 0.51 and 0.56, respectively. Furthermore, every 1% increase in HbA1c was independently associated with 38% higher odds of a more severe fatty liver grade (aOR=1.38; 95% CI: 1.10-1.73; p=0.005), while diabetes was independently associated with 1.87 times higher odds of increasing grade.

These findings are consistent with the bidirectional relationship between type 2 diabetes and fatty liver disease. Ballestri et al. (2016),[10] reported that fatty liver significantly increased the risk of incident diabetes and metabolic syndrome. Conversely, chronic hyperglycaemia and insulin resistance increase hepatic free-fatty-acid influx and lipogenesis, thereby contributing to greater hepatic fat accumulation. Mantovani et al. (2021),[11] in an updated meta-analysis, also demonstrated that fatty liver was associated with a substantially higher risk of developing type 2 diabetes and that the risk increased with greater disease severity. The present findings reinforce the need to screen patients with fatty liver for diabetes and to assess patients with diabetes for hepatic steatosis and fibrosis risk.

Lipid parameters

The lipid profile worsened significantly across increasing fatty liver grades. Mean triglycerides increased from 137.8±43.9 mg/dL in Grade I to 236.9±78.2 mg/dL in Grade III. Total cholesterol increased from 176.3±34.7 mg/dL to 226.4±45.6 mg/dL, while LDL cholesterol increased from 103.2±29.6 mg/dL to 149.6±38.4 mg/dL. In contrast, HDL cholesterol decreased from 45.7±8.3 mg/dL in Grade I to 33.4±6.8 mg/dL in Grade III. The prevalence of dyslipidaemia consequently increased from 45.2% to 86.1%.

Triglycerides showed a moderately strong positive correlation with fatty liver grade (ρ=0.55), whereas HDL cholesterol demonstrated a significant inverse correlation (ρ=−0.49). After adjustment, every 25-mg/dL increase in triglycerides was associated with 21% higher odds of increasing fatty liver grade. Conversely, every 5-mg/dL increase in HDL cholesterol was associated with a 21% reduction in the odds of greater fatty liver severity. LDL cholesterol showed a positive unadjusted correlation but did not remain significant after adjustment, suggesting that triglyceride-rich lipoproteins and low HDL cholesterol may be more closely associated with hepatic fat accumulation.

This atherogenic lipid pattern is characteristic of insulin resistance and typically includes hypertriglyceridaemia, low HDL cholesterol and increased numbers of small dense LDL particles. Targher et al. (2016),[12] emphasized that fatty liver disease is strongly associated with atherogenic dyslipidaemia and increased cardiovascular risk. Therefore, the lipid abnormalities observed in the present study have implications beyond hepatic disease and support comprehensive cardiovascular risk assessment.

Correlation and independent predictors

Waist circumference emerged as the strongest correlate of fatty liver grade (ρ=0.62) and remained independently associated with severity after adjustment. Each 5-cm increase in waist circumference increased the odds of a higher grade by 31%. BMI, diabetes, ALT, GGT, HbA1c, triglycerides and HDL cholesterol were also independent predictors. The regression model was statistically significant and explained approximately 48% of the variation in fatty liver grade.

These results support the recently adopted concept that steatotic liver disease is closely integrated with cardiometabolic dysfunction. Rinella et al. (2023),[13] proposed metabolic dysfunction-associated steatotic liver disease as a nomenclature that provides affirmative diagnostic criteria based on hepatic steatosis and cardiometabolic risk factors. The clustering of central obesity, hyperglycaemia, hypertension, elevated triglycerides and low HDL cholesterol in the present participants is consistent with this framework.

CONCLUSION

Increasing ultrasonographic fatty liver grade was significantly associated with worsening anthropometric, hepatic, glycaemic and lipid parameters. Waist circumference, ALT, BMI, GGT, HbA1c and triglycerides showed the strongest positive correlations with fatty liver severity, while HDL cholesterol and serum albumin showed significant inverse correlations. Central obesity, diabetes mellitus, poor glycaemic control, elevated ALT and GGT, hypertriglyceridaemia and low HDL cholesterol were independently associated with higher ultrasonographic grades. These findings indicate that ultrasonographic fatty liver grading, when combined with liver function tests and metabolic assessment, may provide a simple and clinically useful approach for identifying patients at increased hepatic and cardiometabolic risk. Patients with higher fatty liver grades should undergo comprehensive metabolic evaluation and appropriate assessment for liver fibrosis.

Limitations

The study had several limitations. Its cross-sectional design prevented the establishment of temporal or causal relationships between metabolic abnormalities and fatty liver severity. The study was conducted at a single tertiary-care hospital; therefore, referral and selection bias may have occurred, limiting the generalizability of the findings to the community. Conventional ultrasonography is operator-dependent and has limited sensitivity for mild hepatic steatosis, particularly among individuals with obesity. Ultrasonographic grading cannot reliably distinguish simple steatosis from steatohepatitis or accurately determine the stage of hepatic fibrosis. Liver biopsy, MRI-derived proton-density fat fraction and controlled attenuation parameter measurements were not used to confirm or quantify hepatic fat. Dietary intake, physical activity, insulin resistance and genetic factors were not assessed in detail. Residual confounding from medications and unmeasured metabolic or lifestyle factors could not be excluded. Finally, the participants were not followed longitudinally to determine progression, regression or development of liver-related and cardiovascular outcomes.

REFERENCES

1. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 2016;64(1):73-84.
2. Lonardo A, Nascimbeni F, Ballestri S, Fairweather D, Win S, Than TA, et al. Sex differences in nonalcoholic fatty liver disease: state of the art and identification of research gaps. Hepatology. 2019;70(4):1457-1469.
3. Mitra S, De A, Chowdhury A. Epidemiology of non-alcoholic and alcoholic fatty liver diseases. Transl Res. 2020;221:58-71.
4. Godoy-Matos AF, Silva Júnior WS, Valerio CM. NAFLD as a continuum: from obesity to metabolic syndrome and diabetes. Diabetol Metab Syndr. 2020;12:60.
5. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease: a systematic review and meta-analysis. Hepatology. 2023;77(4):1335-1347.
6. Ferraioli G, Berzigotti A, Barr RG, Choi BI, Cui XW, Dong Y, et al. Quantification of liver fat content with ultrasound: a WFUMB position paper. Ultrasound Med Biol. 2021;47(10):2803-2820.
7. European Association for the Study of the Liver; European Association for the Study of Diabetes; European Association for the Study of Obesity. EASL-EASD-EASO clinical practice guidelines for the management of non-alcoholic fatty liver disease. J Hepatol. 2016;64(6):1388-1402.
8. Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases. Hepatology. 2018;67(1):328-357.
9. Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, Abdelmalek MF, Caldwell S, Barb D, et al. AASLD practice guidance on the clinical assessment and management of nonalcoholic fatty liver disease. Hepatology. 2023;77(5):1797-1835.
10. Ballestri S, Zona S, Targher G, Romagnoli D, Baldelli E, Nascimbeni F, et al. Nonalcoholic fatty liver disease is associated with an almost twofold increased risk of incident type 2 diabetes and metabolic syndrome: evidence from a systematic review and meta-analysis. J Gastroenterol Hepatol. 2016;31(5):936-944.
11. Mantovani A, Petracca G, Beatrice G, Tilg H, Byrne CD, Targher G. Non-alcoholic fatty liver disease and risk of incident diabetes mellitus: an updated meta-analysis of 501,022 adult individuals. Gut. 2021;70(5):962-969.
12. Targher G, Byrne CD, Lonardo A, Zoppini G, Barbui C. Non-alcoholic fatty liver disease and risk of incident cardiovascular disease: a meta-analysis. J Hepatol. 2016;65(3):589-600.
13. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. Hepatology. 2023;78(6):1966-1986.