Original Article

# Evaluation of Non-Alcoholic Fatty Liver Disease Using Ultrasonography and Its Correlation with Metabolic Parameters

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## Abstract

**Background:** Non-alcoholic fatty liver disease, or NAFLD (also known as fatty liver disease or NAFLD), is the most prevalent chronic liver disease in the world and is closely linked with obesity, insulin resistance, dyslipidemia and other factors of metabolic syndrome. Early diagnosis of NAFLD is crucial to avoid cirrhosis and development of hepatocellular carcinoma. In many clinical situations, the imaging modality ultrasonography is widely available, non-invasive and low cost options for detection and grading of hepatic steatosis. The current study was conducted to assess NAFLD by ultrasonography and check its correlation with metabolic parameters. **Material and Methods:** A cross sectional observational study was done at hospital using 340 subjects above 18 years of age who were undergoing abdominal ultrasonography. Demographic, anthropometric, biochemical and ultrasonographic data was taken. The diagnosis of NAFLD and grade of severity were made based on the accepted ultrasonography criteria. The metabolic parameters evaluated were body mass index (BMI), fasting blood glucose (FBG), lipid profile, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels. **Results:** Among the 340 participants, 198 individuals were diagnosed with NAFLD, yielding an overall prevalence of 58.2%. Grade I steatosis was the most common ultrasonographic finding (47.5%), followed by Grade II (34.3%) and Grade III (18.2%). Participants with NAFLD had significantly higher BMI (29.8 ± 4.2 vs. 24.1 ± 3.6 kg/m²), fasting blood glucose (118.4 ± 29.6 vs. 93.7 ± 18.2 mg/dL), total cholesterol (213.5 ± 39.8 vs. 181.2 ± 30.5 mg/dL), triglycerides (191.8 ± 61.4 vs. 124.7 ± 43.1 mg/dL), LDL cholesterol (136.4 ± 32.1 vs. 112.8 ± 27.5 mg/dL), ALT (54.6 ± 20.4 vs. 29.5 ± 11.2 U/L), and AST levels (42.1 ± 16.8 vs. 25.8 ± 9.5 U/L) compared with participants without NAFLD (p<0.001 for all). **Conclusion:** NAFLD was highly prevalent in the study population and demonstrated strong associations with obesity, impaired glucose metabolism, dyslipidemia, and elevated liver enzymes. The severity of hepatic steatosis increased in parallel with worsening metabolic abnormalities.

**Keywords:** Non-alcoholic fatty liver disease; Ultrasonography; Metabolic syndrome; Body mass index; Dyslipidemia; Hepatic steatosis.

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## Introduction

The non-alcoholic fatty liver disease (NAFLD) is now the leading chronic liver disease of the world, as it is highly prevalent and associated with metabolic disorders, and is therefore a significant public health problem.[1] NAFLD is a spectrum which includes hepatic abnormalities, from simple steatosis up to more severe non-alcoholic steatohepatitis (NASH), advanced liver fibrosis, cirrhosis and hepatocellular carcinoma.[2] In those who drink little or no alcohol and after exclusion of other causes of hepatic steatosis, is characterized by excessive accumulation of triglycerides in hepatocytes.[3]

The prevalence of NAFLD has been significantly rising in the recent decades, coinciding with the epidemics of obesity, type 2 diabetes mellitus, dyslipidaemia and metabolic syndrome.[4] It is estimated that one-quarter of the world's population is afflicted with NAFLD, and this occurs as one of the major causes of liver-related morbidity and mortality.[4] NAFLD is now a growing problem in India due to the high prevalence reported (9–32% of population groups) as a result of rapid urbanization, sedentary lifestyle and dietary changes, as well as the increasing rate of obesity in the country. In addition to being a liver disease, it is now known that the liver is the hepatic component of metabolic syndrome, which is also a risk factor for the development of the disease.[5,6]

They are many links in the chain that lead to the pathogenesis of NAFLD. Hepatic fat accumulation has been regarded as a cardinal process in the progression of these diseases through insulin resistance.[6] The influx of free fatty acids into the liver, elevation of de novo lipogenesis, mitochondrial dysfunction, oxidative stress, production of inflammatory cytokines and changes in the gut microbiome can all play a role in hepatic steatosis and liver injury.[7] The modern “multiple-hit” concept suggests that various metabolic, genetic, environmental and inflammatory processes interact and combine to trigger and sustain liver injury in vulnerable people.[8]

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NAFLD is closely linked with metabolic abnormalities. Central Obesity is one of the most important risk factors of hepatic steatosis.[9] Likewise, the presence of type 2 diabetes mellitus has been proven to be significantly associated with the development of NAFLD, and speed up the progression to advanced fibrosis and cirrhosis.[10] It is common for those with affected to have dyslipidemia (high triglycerides, low high-density lipoprotein cholesterol, and elevated low-density lipoprotein cholesterol).[11] In addition, there are consistent associations between hypertension, insulin resistance, metabolic syndrome, presence and severity of NAFLD.[6,12] Therefore, it is important to evaluate metabolic parameters of NAFLD patients for risk stratification and management.

Liver biopsy is considered the gold standard for diagnosis and grading of NAFLD, but it is an invasive procedure with complications, variable sampling, and high cost that for these reasons is not routinely used in clinical practice.[13] Hence, the use of non-invasive imaging techniques has been increasingly significant in the diagnosis and evaluation of liver steatosis. Ultrasonography is the most commonly used imaging method for the detection of fatty liver as it is safe, readily available, inexpensive, and does not emit any ionizing radiation.[14] The sonographic criteria of fatty liver are the increased liver echogenicity, the blurring of the vessels, the deep attenuation of ultrasound penetration and the weak visualization of the diaphragm.[15] Ultrasonography has high sensitivity and specificity for moderate to severe hepatic steatosis and commonly used in epidemiological studies and as baseline data in routine clinical screening.[16]

Hepatic steatosis grade from ultrasonography can be useful to obtain information about the severity of the fatty infiltration and can be correlated with metabolic derangements.[15] The ultrasonographic grade of fatty liver has been correlated significantly with body mass index (BMI), waist circumference, fasting blood glucose, serum triglycerides, total cholesterol and liver enzyme levels in several studies.[17,18] It is clinically relevant to recognise these relationships, as patients with worse grades of steatosis could be more susceptible to cardiovascular disease, diabetes mellitus and chronic liver damage.[19]

Early diagnosis and evaluation are essential to prevent the progression of the disease and complications as NAFLD is becoming more common and is closely linked with metabolic risk factors. Ultrasonography is a useful and practical tool for the diagnosis of fatty liver during a routine clinical examination, and measurement of metabolic parameters is useful to determine the severity of the disease and determine the management approach. Thus, elucidation of association between ultrasonographic parameters and metabolic indices could help to understand the burden, progression and clinical implications of NAFLD.

## Materials and Methods

**Study Design and Setting:** This hospital-based cross-sectional observational study was conducted in the Department of Radiodiagnosis in collaboration with the Department of General Medicine at a tertiary care teaching hospital. The study was carried out from November 2024 to December 2025 after obtaining approval from the Institutional Ethics Committee. The study adhered to the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

**Study Population:** A total of 340 adult participants who underwent abdominal ultrasonography during the study period were included. Consecutive sampling was employed to recruit eligible participants attending outpatient and inpatient services. Individuals aged 18 years and above who consented to participate and underwent ultrasonographic evaluation of the liver were considered eligible for inclusion.

**Inclusion Criteria**

1. Adults aged ≥18 years.
2. Individuals undergoing abdominal ultrasonography for routine health evaluation or clinical indications.
3. Participants willing to provide informed written consent.

**Exclusion Criteria**

1. History of significant alcohol consumption (>20 g/day in women and >30 g/day in men).
2. Known chronic liver diseases including viral hepatitis, autoimmune hepatitis, Wilson's disease, hemochromatosis, and drug-induced liver disease.
3. Patients with hepatic malignancy or metastatic liver disease.
4. Pregnant women.
5. Individuals with incomplete clinical, biochemical, or ultrasonographic data.

**Clinical and Anthropometric Assessment:** Demographic characteristics including age and sex were recorded using a structured data collection form. Anthropometric measurements were obtained by trained personnel following standardized procedures. Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale, while height was measured to the nearest 0.1 cm using a stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m²).

Blood pressure was measured using a standardized sphygmomanometer after at least 5 minutes of rest in a seated position. Waist circumference was measured midway between the lower rib margin and the iliac crest.

**Biochemical Investigations:** After an overnight fast of 8–12 hours, venous blood samples were collected from all participants. The following metabolic parameters were analyzed using standard laboratory methods:

- Fasting blood glucose (FBG)
- Total cholesterol (TC)
- Triglycerides (TG)
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C)
- Serum alanine aminotransferase (ALT)
- Serum aspartate aminotransferase (AST)

All biochemical analyses were performed in the central clinical laboratory using automated analyzers with regular quality control procedures.

**Ultrasonographic Evaluation:** Abdominal ultrasonography was performed using a high-resolution ultrasound system equipped with a 3.5–5.0 MHz convex transducer. All examinations were conducted by experienced radiologists who were blinded to the biochemical findings.

The diagnosis of NAFLD was established based on characteristic sonographic features including:

1. Diffuse increase in hepatic echogenicity relative to the renal cortex.
2. Increased posterior beam attenuation.
3. Blurring of intrahepatic vascular margins.
4. Poor visualization of the diaphragm.

Hepatic steatosis was graded according to established ultrasonographic criteria:

**Grade I (Mild Fatty Liver):** Slight diffuse increase in hepatic echogenicity with normal visualization of the diaphragm and intrahepatic vessel borders.

**Grade II (Moderate Fatty Liver):** Moderate increase in hepatic echogenicity with mildly impaired visualization of intrahepatic vessels and diaphragm.

**Grade III (Severe Fatty Liver):** Marked increase in hepatic echogenicity with poor or absent visualization of intrahepatic vessels, posterior liver segments, and diaphragm.

Participants were categorized into NAFLD-positive and NAFLD-negative groups based on ultrasonographic findings.

**Outcome Measures:** The primary outcome was the prevalence and ultrasonographic grading of NAFLD. Secondary outcomes included the correlation between ultrasonographic grades of fatty liver and metabolic parameters such as BMI, fasting blood glucose, lipid profile, and liver enzyme levels.

**Statistical Analysis:** Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 26. (IBM Corp., Armonk, NY, USA).

Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test.

Comparisons between groups were performed using the independent-samples t-test or Mann–Whitney U test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables. One-way analysis of variance (ANOVA) with post hoc analysis was used to compare metabolic parameters across different grades of fatty liver.

Correlation between ultrasonographic grades of NAFLD and metabolic parameters was evaluated using Pearson's or Spearman's correlation coefficients, as appropriate. Multivariable logistic regression analysis was performed to identify independent predictors of NAFLD after adjusting for potential confounders.

A two-tailed p-value <0.05 was considered statistically significant.

## RESULTS

A total of 340 participants were included in the study. The mean age of the study population was 46.8 ± 12.4 years (range: 18–78 years). Of the participants, 188 (55.3%) were males and 152 (44.7%) were females. Ultrasonography identified NAFLD in 198 participants, yielding an overall prevalence of 58.2%.

**Table 1: Demographic Characteristics of the Study Population (N = 340)**

| Variable | Frequency (%) |
|---|---|
| Male | 188 (55.3) |
| Female | 152 (44.7) |
| Age 18–30 years | 52 (15.3) |
| Age 31–45 years | 106 (31.2) |
| Age 46–60 years | 122 (35.9) |
| Age >60 years | 60 (17.6) |
| NAFLD Present | 198 (58.2) |
| NAFLD Absent | 142 (41.8) |

[Table 1] presents the demographic profile of the 340 study participants. The majority of participants were males (55.3%), while females accounted for 44.7% of the study population. The largest proportion of participants belonged to the 46–60 years age group (35.9%), followed by the 31–45 years age group (31.2%). Ultrasonographic examination revealed that 198 participants (58.2%) had evidence of non-alcoholic fatty liver disease (NAFLD), whereas 142 participants (41.8%) showed no sonographic evidence of hepatic steatosis. These findings indicate a relatively high burden of NAFLD among the study population.

**Table 2: Distribution of Ultrasonographic Grades of NAFLD (n = 198)**

| Grade | Frequency | Percentage (%) |
|---|---|---|
| Grade I | 94 | 47.5 |
| Grade II | 68 | 34.3 |
| Grade III | 36 | 18.2 |

[Table 2] illustrates the severity of hepatic steatosis among the 198 participants diagnosed with NAFLD. Grade I (mild) fatty liver was the most prevalent category, accounting for 47.5% of NAFLD cases. Grade II (moderate) steatosis was observed in 34.3% of affected individuals, while Grade III (severe) steatosis was identified in 18.2% of participants. The predominance of Grade I disease suggests that most cases were detected during the early stage of fatty liver infiltration, highlighting the potential utility of ultrasonography for early diagnosis and intervention.

**Table 3: Comparison of Metabolic Parameters Between NAFLD and Non-NAFLD Groups**

| Variable | NAFLD (n=198) Mean ± SD | Non-NAFLD (n=142) Mean ± SD | p-value |
|---|---|---|---|
| BMI (kg/m$^2$) | 29.8 ± 4.2 | 24.1 ± 3.6 | <0.001 |
| Fasting Blood Glucose (mg/dL) | 118.4 ± 29.6 | 93.7 ± 18.2 | <0.001 |
| Total Cholesterol (mg/dL) | 213.5 ± 39.8 | 181.2 ± 30.5 | <0.001 |
| Triglycerides (mg/dL) | 191.8 ± 61.4 | 124.7 ± 43.1 | <0.001 |
| HDL-C (mg/dL) | 39.8 ± 7.6 | 47.5 ± 8.4 | <0.001 |
| LDL-C (mg/dL) | 136.4 ± 32.1 | 112.8 ± 27.5 | <0.001 |
| ALT (U/L) | 54.6 ± 20.4 | 29.5 ± 11.2 | <0.001 |
| AST (U/L) | 42.1 ± 16.8 | 25.8 ± 9.5 | <0.001 |

[Table 3] compares anthropometric and biochemical parameters between participants with and without NAFLD. Individuals diagnosed with NAFLD demonstrated significantly higher body mass index (29.8 ± 4.2 kg/m$^2$ versus 24.1 ± 3.6 kg/m$^2$), fasting blood glucose levels (118.4 ± 29.6 mg/dL versus 93.7 ± 18.2 mg/dL), total cholesterol levels (213.5 ± 39.8 mg/dL versus 181.2 ± 30.5 mg/dL), and triglyceride concentrations (191.8 ± 61.4 mg/dL versus 124.7 ± 43.1 mg/dL) compared with participants without NAFLD ($p<0.001$ for all comparisons).

**Table 4: Metabolic Parameters According to Ultrasonographic Grade of Fatty Liver**

| Variable | Grade I (n=94) | Grade II (n=68) | Grade III (n=36) | p-value |
|---|---|---|---|---|
| BMI (kg/m$^2$) | 27.6 ± 3.5 | 30.4 ± 3.9 | 33.2 ± 4.1 | <0.001 |
| FBG (mg/dL) | 108.3 ± 22.1 | 119.7 ± 27.8 | 136.5 ± 34.6 | <0.001 |
| Triglycerides (mg/dL) | 162.4 ± 42.6 | 198.8 ± 55.4 | 244.6 ± 70.2 | <0.001 |
| ALT (U/L) | 45.3 ± 15.1 | 57.6 ± 18.4 | 73.8 ± 24.2 | <0.001 |

[Table 4] evaluates the relationship between the severity of hepatic steatosis and selected metabolic parameters. A progressive increase in BMI was observed from Grade I to Grade III NAFLD, indicating that greater adiposity was associated with more severe liver fat accumulation. Similarly, fasting blood glucose levels increased significantly across the three grades, suggesting worsening glucose metabolism with increasing steatosis severity.

**Table 5: Correlation of NAFLD Grade with Metabolic Parameters**

| Variable | Correlation Coefficient (r) | p-value |
|---|---|---|
| BMI | 0.612 | <0.001 |
| Fasting Blood Glucose | 0.485 | <0.001 |
| Total Cholesterol | 0.421 | <0.001 |
| Triglycerides | 0.644 | <0.001 |
| ALT | 0.587 | <0.001 |
| AST | 0.529 | <0.001 |

[Table 5] presents the correlation analysis between ultrasonographic grades of NAFLD and various metabolic parameters. The strongest positive correlation was observed between NAFLD grade and serum triglyceride levels ($r = 0.644$), followed closely by BMI ($r = 0.612$). Significant positive correlations were also identified for ALT ($r = 0.587$), AST ($r = 0.529$), fasting blood glucose ($r = 0.485$), and total cholesterol ($r = 0.421$).

**Table 6: Independent Predictors of NAFLD**

| Variable | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| BMI ≥ 25 kg/m$^2$ | 3.82 | 2.11–6.94 | <0.001 |
| Diabetes Mellitus | 2.67 | 1.48–4.82 | 0.001 |
| Triglycerides ≥150 mg/dL | 3.29 | 1.92–5.64 | <0.001 |
| Elevated ALT | 2.14 | 1.23–3.72 | 0.007 |

[Table 6] summarizes the results of multivariable logistic regression analysis performed to identify factors independently associated with NAFLD. After adjustment for potential confounding variables, participants with a BMI ≥25 kg/m$^2$ had approximately 3.8 times greater odds of developing NAFLD compared to those with normal BMI (Adjusted OR = 3.82; 95% CI: 2.11–6.94; $p<0.001$).

[Figure 1] illustrates the prevalence of non-alcoholic fatty liver disease (NAFLD) among the 340 study participants based on ultrasonographic examination. Out of the total study population, 198 participants (58.2%) were diagnosed with NAFLD, whereas 142 participants (41.8%) showed no ultrasonographic evidence of fatty liver. The findings indicate that more than half of the study population was affected by NAFLD, highlighting a substantial burden of hepatic steatosis in the studied cohort. The high prevalence observed may reflect the increasing impact of metabolic risk factors such as obesity, dyslipidemia, insulin resistance, and diabetes mellitus within the population. These results underscore the importance of routine ultrasonographic screening and early identification of individuals at risk for metabolic liver disease.

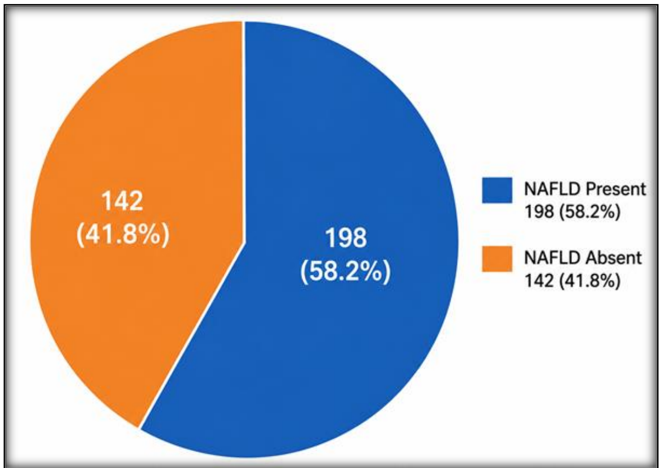
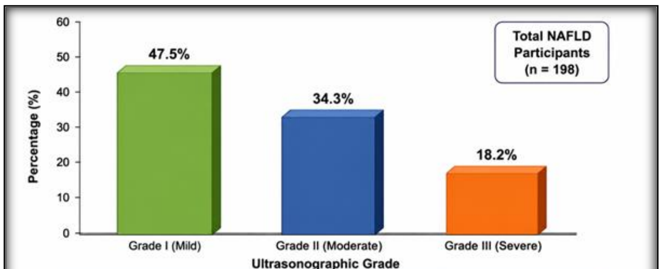



**Figure 1: Prevalence of NAFLD among study participants as detected by ultrasonography**




| Ultrasonographic Grade | Number of Participants (n) | Percentage (%) |
|---|---|---|
| Grade I (Mild) | 94 | 47.5 |
| Grade II (Moderate) | 68 | 34.3 |
| Grade III (Severe) | 36 | 18.2 |
| Total | 198 | 100.0 |

**Figure 2: Distribution of ultrasonographic grades of non-alcoholic fatty liver disease among affected participants**

[Figure 2] illustrates the distribution of ultrasonographic grades of non-alcoholic fatty liver disease (NAFLD) among the 198 participants diagnosed with hepatic steatosis. Grade I (mild) fatty liver was the most frequently observed category, accounting for 94 participants (47.5%), followed by Grade II (moderate) fatty liver in 68 participants (34.3%). Grade III (severe) fatty liver represented the smallest proportion, affecting 36 participants (18.2%).

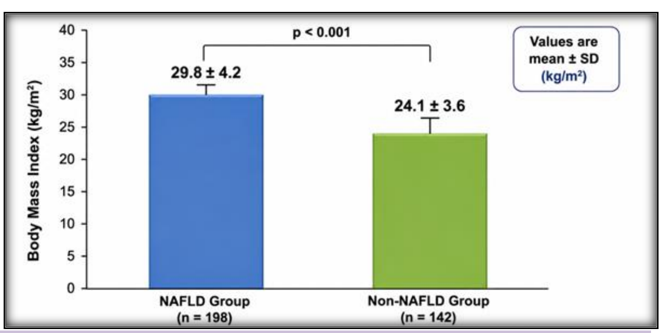



**Figure 3: Comparison of mean body mass index between NAFLD and non-NAFLD groups**

[Figure 3] compares the mean body mass index (BMI) between participants with non-alcoholic fatty liver disease (NAFLD) and those without NAFLD. The mean BMI was significantly higher among participants diagnosed with NAFLD (29.8 ± 4.2 kg/m²) compared with participants without NAFLD (24.1 ± 3.6 kg/m²). Statistical analysis demonstrated a highly significant difference between the two groups ($p < 0.001$).

## Discussion

The present study evaluated the prevalence of non-alcoholic fatty liver disease (NAFLD) using ultrasonography and investigated its association with various metabolic parameters in a cohort of 340 participants. The findings demonstrated a high prevalence of NAFLD (58.2%), with Grade I steatosis being the most common ultrasonographic presentation. Furthermore, significant associations were observed between NAFLD and body mass index (BMI), fasting blood glucose, lipid abnormalities, and liver enzyme levels. These findings reinforce the growing evidence that NAFLD is closely linked to metabolic dysfunction and represents an important component of the metabolic syndrome.

In the present study, NAFLD was detected in 58.2% of participants. This prevalence is higher than the global pooled prevalence of approximately 25% reported by Younossi et al,[1] but is consistent with the increasing burden of NAFLD observed in populations characterized by obesity, diabetes, and sedentary lifestyles. Similar high prevalence rates have been reported in studies involving hospital-based and high-risk populations. Estes et al. projected a substantial increase in NAFLD prevalence worldwide owing to the parallel rise in obesity and type 2 diabetes mellitus, emphasizing the need for early detection and intervention.[20] Likewise, Le et al. reported increasing prevalence trends in Asian populations, reflecting rapid lifestyle and dietary transitions.[21]

The predominance of Grade I fatty liver (47.5%) observed in our study suggests that a considerable proportion of affected individuals were identified during the early stages of hepatic steatosis. Similar observations have been reported by Ballestri et al., who noted that mild steatosis is frequently detected in asymptomatic individuals undergoing routine abdominal ultrasonography.[22] Early-stage disease detection is clinically important because lifestyle modifications and metabolic risk-factor control may halt or reverse disease progression before the development of advanced fibrosis or cirrhosis.

A major finding of the present study was the significantly higher BMI among participants with NAFLD compared with those without NAFLD. Obesity is widely recognized as one of the strongest risk factors for hepatic steatosis. The mean BMI of the NAFLD group (29.8 ± 4.2 kg/m²) was significantly greater than that of the non-NAFLD group (24.1 ± 3.6 kg/m²), and BMI demonstrated a strong positive correlation with steatosis severity. These findings are consistent with the observations of Rinella, who highlighted obesity-induced insulin resistance and adipose tissue dysfunction as major contributors to hepatic lipid accumulation.[23] Similarly, Stefan et al. reported that increasing adiposity is associated with both the occurrence and progression of NAFLD through mechanisms involving chronic low-grade inflammation and altered lipid metabolism.[24] The logistic regression analysis further identified BMI ≥25 kg/m² as an independent predictor of NAFLD, emphasizing the critical role of excess body weight in disease development.

The present study also demonstrated significantly elevated fasting blood glucose levels among participants with NAFLD, and glucose levels increased progressively with higher ultrasonographic grades. These findings support the established relationship between insulin resistance and hepatic steatosis. Insulin resistance promotes increased lipolysis, enhanced free This leads to increased fatty acid flux to the liver and increased de novo lipogenesis, and thus triglyceride deposition in the hepatocytes. Insulin resistance is the major pathogenic mechanism underlying NAFLD and type 2 diabetes mellitus and metabolic syndrome, said Cusi.[25] In addition, Mantovani et al. found that the likelihood of acquiring diabetes and cardiovascular diseases among patients with NAFLD was significantly higher than that of healthy controls.[26] These observations are corroborated by our result which identified diabetes mellitus as an independent predictor of NAFLD.

Another major characteristic among those with NAFLD was dyslipidemia. The group with NAFLD had significantly higher triglycerides, total cholesterol, LDL and lower HDL than the other group. Furthermore, the triglyceride level had the highest correlation with ultrasonographic severity. The results were consistent with previous studies that showed that NAFLD is accompanied by lipid metabolism abnormalities. Eslam et al. highlighted that hypertriglyceridemia has a direct role in the hepatic fat accumulation or steatohepatitis.[27] Likewise, Byrne and Targher found that hypertriglyceridemia is a robust predictor of cardiovascular risk and hepatic steatosis in NAFLD patients.[28] The lipid abnormalities are also strongly linked to NAFLD in our regression model, which further supports the pathogenic role of these abnormalities.

ALT and AST were significantly higher in the subjects with NAFLD when compared to the subjects without NAFLD based on the liver enzyme analysis. Further, enzyme levels were found to be high in a progressive manner in Grade I to III steatosis. Increased levels of aminotransferases are usually associated with hepatocellular injury and inflammation. It is interesting to note, however, that patients with normal liver enzyme levels may still have NAFLD. Sanyal pointed out that while liver enzymes are associated with disease activity in certain patients, histological severity could exist even in those with a normal biochemical profile of the liver.[29] However, in the present study, we found a strong correlation between high ALT and NAFLD, which is in line with previous studies that deemed ALT as a useful marker of metabolic liver injury.[30]

The correlation analysis showed that BMI, fasting blood glucose, triglycerides, ALT and AST levels had significantly positive correlation with ultrasonographic grade. As the liver fat becomes worse, the metabolic abnormalities worsen, according to these findings. This is confirmed by the studies of Yki-Järvinen who described NAFLD as a manifestation of and a cause of overall metabolic dysfunction.[31] Hypertriglyceridemia, hyperlipidemy, and inflammation are activated by higher amounts of visceral fat in the liver, which further causes insulin resistance, disease progression and a vicious circle of cardiometabolic complications.

Multivariable logistic regression analysis revealed that obesity, diabetes mellitus, hypertriglyceridemia and elevated ALT level were independent predictors of NAFLD. These results agree with evidence in recent years, which backs up the notion that NAFLD is not merely a liver-centered disease, but rather a metabolic one. International consensus statements have now drawn greater attention to the metabolic underpinnings of fatty liver disease, and the need to perform a thorough assessment of cardiac risk in those with fatty liver disease.[32]

This study is descriptive, and has several strengths: the relative size of the sample and systematic evaluation of both ultrasonographic parameters and metabolic parameters. Some restrictions should be noted, though. Due to the cross-sectional design, it is not possible to draw causal conclusions about the time course of metabolic anomalies and NAFLD. Further, ultrasonography is often performed, is cheap, but has low sensitivity for detecting mild steatosis, and is less accurate for determining the severity of fibrosis than liver biopsy or advanced imaging techniques. Moreover, the study was performed at one center, and the results might not be generalizable.

Despite these restrictions, the results indicate that NAFLD is a significant public health issue and it is strongly linked with obesity, diabetes, dyslipidemia and liver enzyme abnormalities. Passive screening for the identification of subjects at risk, along with metabolic assessment, may help to act promptly to prevent and/or slow the onset of hepatic and cardiovascular complications.

## Conclusion

This study showed that non-alcoholic fatty liver disease (NAFLD) is highly prevalent, as ultrasonographic signs of hepatic steatosis were seen more than half of the cases. A significant percentage of patients had early disease stages (Grade I, mild steatosis). However, the fact that a significant proportion of study participants had moderate and severe steatosis underscores the severity of advanced fatty liver disease in the population.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### Conflicts of interest

There are no conflicts of interest.

## 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. 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.
3. Angulo P. Nonalcoholic fatty liver disease. N Engl J Med. 2002;346(16):1221-1231.
4. Younossi ZM, Blissett D, Blissett R, Henry L, Stepanova M, Younossi Y, et al. The economic and clinical burden of nonalcoholic fatty liver disease in the United States and Europe. Hepatology. 2016;64(5):1577-1586.
5. Das K, Das K, Mukherjee PS, Ghosh A, Ghosh S, Mridha AR, et al. Nonobese population in a developing country has a high prevalence

of nonalcoholic fatty liver and significant liver disease. Hepatology. 2010;51(5):1593-1602.
6. Marchesini G, Brizi M, Bianchi G, Tomassetti S, Bugianesi E, Lenzi M, et al. Nonalcoholic fatty liver disease: A feature of the metabolic syndrome. Diabetes. 2001;50(8):1844-1850.
7. Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). Metabolism. 2016;65(8):1038-1048.
8. Tilg H, Moschen AR. Evolution of inflammation in nonalcoholic fatty liver disease: The multiple parallel hits hypothesis. Hepatology. 2010;52(5):1836-1846.
9. Fabbrini E, Sullivan S, Klein S. Obesity and nonalcoholic fatty liver disease: Biochemical, metabolic, and clinical implications. Hepatology. 2010;51(2):679-689.
10. Adams LA, Anstee QM, Tilg H, Targher G. Non-alcoholic fatty liver disease and its relationship with cardiovascular disease and other extrahepatic diseases. Gut. 2017;66(6):1138-1153.
11. Clark JM. The epidemiology of nonalcoholic fatty liver disease in adults. J ClinGastroenterol. 2006;40(Suppl 1):S5-S10.
12. Lonardo A, Ballestri S, Marchesini G, Angulo P, Loria P. Nonalcoholic fatty liver disease: A precursor of the metabolic syndrome. Dig Liver Dis. 2015;47(3):181-190.
13. Bravo AA, Sheth SG, Chopra S. Liver biopsy. N Engl J Med. 2001;344(7):495-500.
14. Hernaez R, Lazo M, Bonekamp S, Kamel I, Brancati FL, Guallar E, et al. Diagnostic accuracy and reliability of ultrasonography for the detection of fatty liver: A meta-analysis. Hepatology. 2011;54(3):1082-1090.
15. Saadeh S, Younossi ZM, Remer EM, Gramlich T, Ong JP, Hurley M, et al. The utility of radiological imaging in nonalcoholic fatty liver disease. Gastroenterology. 2002;123(3):745-750.
16. Hamaguchi M, Kojima T, Itoh Y, Harano Y, Fujii K, Nakajima T, et al. The severity of ultrasonographic findings in nonalcoholic fatty liver disease reflects the metabolic syndrome and visceral fat accumulation. Am J Gastroenterol. 2007;102(12):2708-2715.
17. Bedogni G, Miglioli L, Masutti F, Tiribelli C, Marchesini G, Bellentani S. Prevalence of and risk factors for nonalcoholic fatty liver disease. Hepatology. 2005;42(1):44-52.
18. Targher G, Bertolini L, Padovani R, Rodella S, Tessari R, Zenari L, et al. Relations between carotid artery wall thickness and liver histology in subjects with nonalcoholic fatty liver disease. Diabetes Care. 2006;29(6):1325-1330.
19. Targher G, Day CP, Bonora E. Risk of cardiovascular disease in patients with nonalcoholic fatty liver disease. N Engl J Med. 2010;363(14):1341-1350.
20. Estes C, Anstee QM, Arias-Loste MT, Bantel H, Bellentani S, Caballeria J, et al. Modeling NAFLD disease burden in China, France, Germany, Italy, Japan, Spain, United Kingdom, and United States. J Hepatol. 2018;69(4):896-904.
21. Le MH, Yeo YH, Li X, Li J, Zou B, Wu Y, et al. 2019 Global NAFLD prevalence: A systematic review and meta-analysis. ClinGastroenterolHepatol. 2022;20(12):2809-2817.
22. Ballestri S, Nascimbeni F, Baldelli E, Marrazzo A, Romagnoli D, Lonardo A. NAFLD as a sexual dimorphic disease. Eur J Clin Invest. 2017;47(6):441-452.
23. Rinella ME. Nonalcoholic fatty liver disease: A systematic review. JAMA. 2015;313(22):2263-2273.
24. Stefan N, Häring HU, Cusi K. Non-alcoholic fatty liver disease: Causes, diagnosis, cardiometabolic consequences, and treatment strategies. Lancet Diabetes Endocrinol. 2019;7(4):313-324.
25. Cusi K. Treatment of patients with type 2 diabetes and non-alcoholic fatty liver disease: Current approaches and future directions. Diabetologia. 2016;59(6):1112-1120.
26. Mantovani A, Petracca G, Beatrice G, Tilg H, Byrne CD, Targher G. Non-alcoholic fatty liver disease and increased risk of incident type 2 diabetes mellitus. Gut. 2021;70(5):962-969.
27. Eslam M, Sanyal AJ, George J. MAFLD: A consensus-driven proposed nomenclature for metabolic associated fatty liver disease. Gastroenterology. 2020;158(7):1999-2014.
28. Byrne CD, Targher G. NAFLD: A multisystem disease. J Hepatol. 2015;62(1 Suppl):S47-S64.
29. Sanyal AJ. Past, present and future perspectives in nonalcoholic fatty liver disease. Nat Rev GastroenterolHepatol. 2019;16(6):377-386.
30. Kwo PY, Cohen SM, Lim JK. ACG Clinical Guideline: Evaluation of abnormal liver chemistries. Am J Gastroenterol. 2017;112(1):18-35.
31. Yki-Järvinen H. Non-alcoholic fatty liver disease as a cause and a consequence of metabolic syndrome. Lancet Diabetes Endocrinol. 2014;2(11):901-910.
32. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease. J Hepatol. 2020;73(1):202-209.