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Original Research Article

Ultrasonographic Assessment of Hepatic Steatosis and Its Association with Metabolic Risk Factors

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Abstract:

Background: Hepatic steatosis was frequently associated with obesity, diabetes mellitus, dyslipidaemia and other metabolic abnormalities. This study assessed the frequency of ultrasonographically detected hepatic steatosis and evaluated its association with metabolic risk factors.

Material and Methods: A hospital-based analytical cross-sectional study was conducted among 220 adults aged 18–70 years. Participants underwent clinical, anthropometric and biochemical assessment followed by abdominal ultrasonography. Hepatic steatosis was graded as Grade 0 to Grade 3 according to sonographic features. Associations between steatosis and metabolic risk factors were assessed using appropriate statistical tests and multivariable logistic regression.

Results: Hepatic steatosis was detected in 126 (57.3%) participants, including Grade 1 in 65 (29.5%), Grade 2 in 45 (20.5%) and Grade 3 in 16 (7.3%). Compared with participants without steatosis, those with steatosis had significantly higher BMI, waist circumference, fasting glucose, HbA1c, triglycerides, LDL-cholesterol, ALT and AST levels and lower HDL-cholesterol (all $P \leq 0.006$). BMI ≥23 kg/m², central obesity and diabetes mellitus were more frequent among participants with steatosis (83.3%, 78.6% and 44.4%, respectively) than those without steatosis (59.6%, 46.8% and 17.0%, respectively). Increasing metabolic risk-factor burden was significantly associated with steatosis ($P<0.001$). BMI ≥23 kg/m², central obesity, diabetes mellitus, hypertriglyceridaemia and low HDL-cholesterol remained independently associated with steatosis.

Conclusion: Ultrasonographically detected hepatic steatosis was common and was significantly associated with adverse metabolic characteristics. Early identification and management of modifiable metabolic risk factors may help reduce the associated hepatic and metabolic burden.

Keywords: Hepatic steatosis; Ultrasonography; Metabolic risk factors; Obesity; Diabetes mellitus; Dyslipidaemia.

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Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a major chronic liver disorder and is closely linked to the increasing burden of obesity, insulin resistance, type 2 diabetes mellitus and other cardiometabolic abnormalities. The recent transition from the term non-alcoholic fatty liver disease to MASLD emphasized the metabolic basis of hepatic steatosis and incorporated cardiometabolic risk factors into the disease definition. MASLD was defined by the presence of hepatic steatosis together with at least one cardiometabolic risk factor in the absence of another predominant cause of steatosis [1,2].

The burden of MASLD has also become particularly relevant in India. A large multicentre Indian study involving 13,750 adults reported a MASLD prevalence of 68.2%, with substantial variation across geographic regions [3]. The high prevalence observed in Indian populations has been attributed to the coexistence of increasing obesity and diabetes with distinctive metabolic susceptibility. Recent evidence from Asian Indian individuals with type 2 diabetes has further demonstrated a substantial burden of hepatic steatosis, emphasizing the close relationship between hepatic fat accumulation and metabolic abnormalities [4].

Hepatic steatosis is not merely a hepatic manifestation of metabolic dysfunction but is associated with an increased risk of extrahepatic complications, particularly cardiovascular disease. Obesity, dyslipidaemia, hypertension and type 2 diabetes represent important determinants of MASLD and frequently coexist in affected individuals [5]. The identification of hepatic steatosis in individuals with these risk factors may therefore provide an opportunity for early recognition of a broader cardiometabolic risk profile.

Imaging has an important role in the non-invasive detection of hepatic steatosis. Conventional B-mode ultrasonography remains widely used because of its accessibility, relatively low cost and absence of ionizing radiation. Increased hepatic echogenicity, reduced visualization of intrahepatic structures and posterior acoustic attenuation are commonly used sonographic indicators of steatosis. Although newer quantitative ultrasound techniques have improved the ability to detect and quantify hepatic fat, conventional ultrasonography continues to have practical relevance in routine clinical settings [6].

Despite the recognized relationship between hepatic steatosis and metabolic risk factors, the magnitude and pattern of these associations may vary across populations. Therefore, the present study was conducted to assess the prevalence and ultrasonographic severity of hepatic steatosis and to determine its association with selected metabolic risk factors, including obesity, central adiposity, diabetes mellitus, hypertension and dyslipidaemia, among adults attending a tertiary care hospital.

Materials and Methods

Study Design and Setting: A hospital-based analytical cross-sectional study was conducted at a tertiary care teaching hospital. The study assessed the frequency and ultrasonographic grade of hepatic steatosis and evaluated its association with metabolic risk factors.

Study Population: The study included adult participants aged 18–70 years who attended the outpatient departments or health-screening services of the study hospital and underwent abdominal ultrasonography during the study period.

Sample Size: The sample size was calculated using the following formula for estimation of a single proportion: $n = Z^2pq / d^2$ where n represented the required sample size, Z represented the standard normal deviate at a 95% confidence level (1.96), p represented the anticipated prevalence of hepatic steatosis, q represented $1 - p$, and d represented the allowable absolute precision.

A prevalence of hepatic steatosis of 43.6% was considered based on previously published Indian data [7]. Using an expected prevalence of 43.6%, a 95% confidence level, and an absolute precision of 7%, the minimum calculated sample size was approximately 193 participants. After considering a 10% allowance for incomplete data or non-evaluable ultrasonographic examinations, the required sample size was approximately 213 participants. Therefore, a total of 220 participants were included in the study.

Sampling Technique: Participants who fulfilled the eligibility criteria were recruited using consecutive sampling until the required sample size of 220 participants was achieved.

Inclusion Criteria: Participants were included if they fulfilled the following criteria:

1. They were aged 18–70 years.
2. They underwent abdominal ultrasonography at the study centre.
3. They provided written informed consent.
4. They were able to provide relevant clinical, anthropometric and biochemical information.

Exclusion Criteria: Participants were excluded if they had:

1. Known chronic liver disease other than suspected metabolic dysfunction-associated steatotic liver disease.
2. Known cirrhosis or hepatocellular carcinoma.
3. Chronic hepatitis B or hepatitis C infection.
4. Significant alcohol consumption suggestive of alcohol-related liver disease.
5. Known autoimmune, cholestatic or hereditary liver disease.
6. Acute hepatitis or acute systemic illness at the time of assessment.
7. A history of medications associated with significant hepatic steatosis.
8. Pregnancy.
9. Previous liver transplantation or major hepatic surgery.
10. Inadequate visualization of the liver during ultrasonography because of excessive bowel gas or other technical limitations.
11. Inability or unwillingness to provide informed consent.

Clinical and Demographic Assessment: A structured data collection form was used to record the demographic and clinical characteristics of the participants. Information regarding age, sex, height, weight, body mass index (BMI), waist circumference, hip circumference, waist-to-hip ratio, blood pressure, diabetes mellitus, hypertension, dyslipidaemia, medication use, alcohol consumption, smoking status and relevant medical history was recorded.

Body weight was measured using a calibrated weighing scale with the participants wearing light clothing and without footwear. Height was

measured using a stadiometer. BMI was calculated by dividing body weight in kilograms by the square of height in metres.

Waist circumference was measured midway between the lowest rib and the iliac crest at the end of normal expiration. Hip circumference was measured at the level of the maximum circumference of the buttocks. The waist-to-hip ratio was calculated from these measurements.

Blood pressure was measured using a calibrated sphygmomanometer after the participant had rested in a seated position for at least 5 minutes. Two blood pressure measurements were obtained at an interval of approximately 2 minutes, and the mean of the two readings was recorded.

Biochemical Investigations: Venous blood samples were collected after an overnight fasting period of 8–12 hours. The following biochemical investigations were performed for all participants: fasting plasma glucose, glycated haemoglobin (HbA1c), total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase, and total bilirubin.

The presence of diabetes mellitus, prediabetes and dyslipidaemia was determined using the documented clinical history, current treatment and biochemical findings.

Ultrasonographic Examination: All participants underwent abdominal ultrasonography using a commercially available ultrasound system equipped with a 3.5–5 MHz curvilinear transducer. The examination was performed after a fasting period of approximately 6–8 hours.

The liver was examined in multiple longitudinal and transverse planes with the participant in the supine position and, when required, in the left lateral decubitus position. Hepatic echogenicity was assessed in comparison with the right renal cortex. The visualization of intrahepatic vessels and the diaphragm was evaluated. Posterior acoustic attenuation, liver size, focal fatty sparing and focal areas of steatosis were also assessed.

The ultrasonographic examination was performed by an experienced radiologist who was blinded to the biochemical results as far as practicable.

Ultrasonographic Grading of Hepatic Steatosis: Hepatic steatosis was graded according to the following ultrasonographic criteria:

Grade 0 – No steatosis: The liver showed normal echogenicity, with normal visualization of intrahepatic vessels and the diaphragm.

Grade 1 – Mild steatosis: The liver showed a mild diffuse increase in echogenicity compared with the renal cortex, while the portal veins and diaphragm remained clearly visualized.

Grade 2 – Moderate steatosis: The liver showed a moderate increase in echogenicity with reduced visualization of intrahepatic vessels and the diaphragm.

Grade 3 – Severe steatosis: The liver showed marked increased echogenicity with prominent posterior acoustic attenuation and poor or absent visualization of intrahepatic vessels and the diaphragm.

Participants with Grade 1, Grade 2 or Grade 3 steatosis were considered to have ultrasonographically detected hepatic steatosis.

Assessment of Metabolic Risk Factors: The metabolic risk factors assessed in the study included obesity, central adiposity, diabetes mellitus or dysglycaemia, hypertension and dyslipidaemia.

For the purpose of analysis, overweight or obesity was assessed using BMI. Central obesity was defined as a waist circumference of ≥90 cm in men or ≥80 cm in women. Hypertriglyceridaemia was defined as a triglyceride concentration of ≥150 mg/dL. Reduced HDL-cholesterol was defined as <40 mg/dL in men or <50 mg/dL in women. Elevated blood pressure was defined as a blood pressure of ≥130/85 mmHg or current treatment for hypertension.

Study Outcomes: The primary outcome was the presence of ultrasonographically detected hepatic steatosis.

The secondary outcomes included the distribution of hepatic steatosis according to ultrasonographic grade and its association with BMI, waist circumference, diabetes or dysglycaemia, hypertension, lipid abnormalities and liver enzyme abnormalities. The relationship between the severity of hepatic steatosis and the number of metabolic risk factors was also assessed.

Statistical Analysis: The collected data were entered into a computerized database and analysed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation for normally distributed variables and as median with interquartile range for non-normally distributed variables. Categorical variables were expressed as frequencies and percentages.

Participants were categorized into two groups according to the presence or absence of ultrasonographically detected hepatic steatosis. Continuous variables between the two groups were compared using the independent-samples t test or Mann–Whitney U test, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test.

Differences among the three grades of hepatic steatosis were assessed using one-way analysis of variance or the Kruskal–Wallis test, depending on the distribution of the data.

Variables that showed a clinically relevant or statistically significant association with hepatic steatosis during univariable analysis were included in multivariable logistic regression analysis to identify independent predictors of hepatic steatosis. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. A two-sided P value of <0.05 was considered statistically significant.

Results

A total of 220 participants were included in the study. The mean age of the study population was 47.6 ± 11.2 years, and 132 (60.0%) participants were male and 88 (40.0%) were female. The mean BMI was 26.1 ± 4.2 kg/m², while the mean waist circumference was 92.4 ± 11.1 cm. Overall, 161 (73.2%) participants had a BMI ≥23 kg/m² and 143 (65.0%) had central obesity. Hypertension, diabetes mellitus and dyslipidaemia were present in 91 (41.4%), 72 (32.7%) and 88 (40.0%) participants, respectively (Table 1).

Ultrasonography demonstrated hepatic steatosis in 126 (57.3%) participants, whereas 94 (42.7%) had no evidence of steatosis. Among participants with steatosis, Grade 1, Grade 2 and Grade 3 changes were observed in 65 (29.5%), 45 (20.5%) and 16 (7.3%) participants, respectively. Increased hepatic echogenicity was the most frequent sonographic finding. Reduced visualization of intrahepatic vessels was observed in 61 (27.7%) participants, while reduced visualization of the diaphragm and posterior acoustic attenuation were observed in 48 (21.8%) and 42 (19.1%) participants, respectively (Table 2).

Participants with hepatic steatosis had a significantly higher mean age than those without steatosis (49.2 ± 10.8 vs. 45.5 ± 11.4 years, P = 0.013). The mean BMI, waist circumference and waist-to-hip ratio were also significantly higher in participants with steatosis than in those without steatosis. Similarly, systolic and diastolic blood pressure values were significantly higher among participants with hepatic steatosis (Table 4).

Metabolic abnormalities were significantly more frequent among participants with hepatic steatosis. A BMI ≥23 kg/m² was present in 105 (83.3%) participants with steatosis compared with 56 (59.6%) participants without steatosis (P <0.001). Central obesity was observed in 99 (78.6%) participants with steatosis compared with 44 (46.8%) without steatosis (P <0.001). Diabetes mellitus was present in 56 (44.4%) participants with steatosis compared with 16 (17.0%) participants without steatosis (P <0.001). Hypertension and dyslipidaemia were also significantly more frequent in the steatosis group (Table 3).

The biochemical profile also differed significantly between the two groups. Participants with hepatic steatosis had higher mean fasting glucose, HbA1c, total cholesterol, triglycerides and LDL-cholesterol levels than participants without steatosis. The mean triglyceride concentration was 178.6 ± 71.4 mg/dL in the steatosis group compared with 137.5 ± 54.2 mg/dL in the non-steatosis group (P <0.001). HDL-cholesterol was significantly lower among participants with hepatic steatosis (42.8 ± 8.7 vs. 47.1 ± 9.2 mg/dL, P <0.001). Mean ALT and AST levels were also significantly higher in participants with steatosis (Table 4).

An increasing frequency of metabolic risk factors was observed with increasing ultrasonographic severity of hepatic steatosis. The prevalence of BMI ≥23 kg/m² increased from 59.6% among participants without steatosis to 87.5% among those with Grade 3 steatosis (P <0.001). Central obesity increased from 46.8% in Grade 0 to 81.3% in Grade 3 steatosis. Diabetes mellitus was present in 17.0% of participants without steatosis and increased to 68.8% among those with Grade 3 steatosis. Similar increases were observed for hypertension, dyslipidaemia and hypertriglyceridaemia (Table 5).

The presence of hepatic steatosis was also associated with an increasing number of metabolic risk factors. Steatosis was identified in 20.8% of participants without any metabolic risk factor, compared with 33.3% of those with one risk factor, 56.0% of those with two risk factors and 100.0% of those with three or more risk factors. This association showed a statistically significant trend (P <0.001) (Table 6).

Multivariable logistic regression analysis identified several factors that remained independently associated with hepatic steatosis. BMI ≥23 kg/m² was associated with increased odds of hepatic steatosis (adjusted OR [aOR] 2.47, 95% CI: 1.30–4.69; P = 0.006). Central obesity was also independently associated with hepatic steatosis (aOR 2.83, 95% CI: 1.54–5.21; P = 0.001). Diabetes mellitus showed a significant association (aOR 2.76, 95% CI: 1.46–5.22; P = 0.002), as did hypertriglyceridaemia (aOR 2.14, 95% CI: 1.17–3.91; P = 0.014) and low HDL-cholesterol (aOR 1.89, 95% CI: 1.01–3.55; P = 0.047). Age, sex and hypertension did not remain statistically significant independent predictors in the multivariable model (Table 7).

Table 1: Baseline demographic and clinical characteristics of the study participants (N=220)

Variable	Overall (N=220)
Age (years), mean ± SD	47.6 ± 11.2
Age ≥50 years, n (%)	104 (47.3)
Male sex, n (%)	132 (60.0)
Female sex, n (%)	88 (40.0)
BMI (kg/m²), mean ± SD	26.1 ± 4.2
BMI ≥23 kg/m², n (%)	161 (73.2)
Waist circumference (cm), mean ± SD	92.4 ± 11.1
Central obesity, n (%)	143 (65.0)
Waist-to-hip ratio, mean ± SD	0.91 ± 0.08
Systolic BP (mmHg), mean ± SD	132.8 ± 16.4
Diastolic BP (mmHg), mean ± SD	82.1 ± 9.6
Hypertension, n (%)	91 (41.4)
Diabetes mellitus, n (%)	72 (32.7)
Dyslipidaemia, n (%)	88 (40.0)
Current smokers, n (%)	49 (22.3)
Alcohol consumption, n (%)	38 (17.3)

Table 2: Ultrasonographic findings and grading of hepatic steatosis

Ultrasonographic finding	n (%)
Hepatic steatosis	126 (57.3)
No steatosis (Grade 0)	94 (42.7)
Mild steatosis (Grade 1)	65 (29.5)
Moderate steatosis (Grade 2)	45 (20.5)
Severe steatosis (Grade 3)	16 (7.3)
Increased hepatic echogenicity	126 (57.3)
Reduced visualization of intrahepatic vessels	61 (27.7)
Reduced visualization of diaphragm	48 (21.8)
Posterior acoustic attenuation	42 (19.1)
Focal fatty sparing	18 (8.2)
Hepatomegaly	37 (16.8)

Table 3: Comparison of metabolic risk factors according to presence of hepatic steatosis

Metabolic risk factor	Steatosis present (n=126)	No steatosis (n=94)	P value
Age ≥50 years, n (%)	70 (55.6)	34 (36.2)	0.006
BMI ≥23 kg/m², n (%)	105 (83.3)	56 (59.6)	<0.001
Central obesity, n (%)	99 (78.6)	44 (46.8)	<0.001
Hypertension, n (%)	62 (49.2)	29 (30.9)	0.007
Diabetes mellitus, n (%)	56 (44.4)	16 (17.0)	<0.001
Dyslipidaemia, n (%)	63 (50.0)	25 (26.6)	0.001
Hypertriglyceridaemia, n (%)	54 (42.9)	21 (22.3)	0.002
Low HDL-cholesterol, n (%)	49 (38.9)	20 (21.3)	0.006
Elevated blood pressure, n (%)	69 (54.8)	39 (41.5)	0.049

Table 4: Comparison of anthropometric and biochemical parameters between participants with and without hepatic steatosis

Parameter	Steatosis present (n=126)	No steatosis (n=94)	P value
Age (years)	49.2 ± 10.8	45.5 ± 11.4	0.013
BMI (kg/m²)	27.4 ± 4.1	24.4 ± 3.7	<0.001
Waist circumference (cm)	97.1 ± 9.8	86.0 ± 9.5	<0.001
Waist-to-hip ratio	0.93 ± 0.07	0.88 ± 0.07	<0.001
Systolic BP (mmHg)	135.6 ± 16.2	129.1 ± 15.8	0.003
Diastolic BP (mmHg)	83.7 ± 9.7	79.9 ± 8.9	0.003
Fasting glucose (mg/dL)	116.8 ± 35.4	98.7 ± 24.1	<0.001
HbA1c (%)	6.3 ± 1.2	5.6 ± 0.8	<0.001
Total cholesterol (mg/dL)	207.4 ± 42.1	190.6 ± 36.8	0.002
Triglycerides (mg/dL)	178.6 ± 71.4	137.5 ± 54.2	<0.001

HDL-cholesterol (mg/dL)	42.8 ± 8.7	47.1 ± 9.2	<0.001
LDL-cholesterol (mg/dL)	128.5 ± 32.6	117.3 ± 29.4	0.006
ALT (U/L)	38.6 ± 19.7	27.4 ± 12.5	<0.001
AST (U/L)	31.8 ± 12.6	25.7 ± 9.4	<0.001

Table 5: Distribution of metabolic risk factors according to the grade of hepatic steatosis

Risk factor	Grade 0 (n=94)	Grade 1 (n=65)	Grade 2 (n=45)	Grade 3 (n=16)	P value
BMI ≥23 kg/m², n (%)	56 (59.6)	52 (80.0)	39 (86.7)	14 (87.5)	<0.001
Central obesity, n (%)	44 (46.8)	48 (73.8)	38 (84.4)	13 (81.3)	<0.001
Hypertension, n (%)	29 (30.9)	26 (40.0)	26 (57.8)	10 (62.5)	0.006
Diabetes mellitus, n (%)	16 (17.0)	20 (30.8)	25 (55.6)	11 (68.8)	<0.001
Dyslipidaemia, n (%)	25 (26.6)	26 (40.0)	27 (60.0)	10 (62.5)	<0.001
Hypertriglyceridaemia, n (%)	21 (22.3)	22 (33.8)	22 (48.9)	10 (62.5)	<0.001
Low HDL-cholesterol, n (%)	20 (21.3)	22 (33.8)	20 (44.4)	7 (43.8)	0.012

Table 6: Association between the number of metabolic risk factors and hepatic steatosis

Number of metabolic risk factors	Steatosis present, n (%)	No steatosis, n (%)	Total
0	5 (20.8)	19 (79.2)	24
1	21 (33.3)	42 (66.7)	63
2	42 (56.0)	33 (44.0)	75
≥3	58 (100.0)	0 (0.0)	58

Trend P <0.001

Table 7: Multivariable logistic regression analysis of factors independently associated with hepatic steatosis

Variable	Adjusted OR	95% CI	P value
Age ≥50 years	1.62	0.91–2.88	0.101
Male sex	1.38	0.78–2.45	0.271
BMI ≥23 kg/m²	2.47	1.30–4.69	0.006
Central obesity	2.83	1.54–5.21	0.001
Hypertension	1.71	0.96–3.04	0.069
Diabetes mellitus	2.76	1.46–5.22	0.002
Hypertriglyceridaemia	2.14	1.17–3.91	0.014
Low HDL-cholesterol	1.89	1.01–3.55	0.047

Discussion

The present study evaluated the occurrence of ultrasonographically detected hepatic steatosis and its relationship with metabolic risk factors among 220 adults. Hepatic steatosis was identified in 57.3% of participants, with mild, moderate and severe steatosis observed in 29.5%, 20.5% and 7.3%, respectively. The relatively high frequency of steatosis in the study population was consistent with the increasing recognition of metabolic dysfunction-associated steatotic liver disease (MASLD) as a common metabolic disorder. Current clinical guidance recommends particular attention to individuals with obesity, type 2 diabetes, abnormal liver enzymes or radiological evidence of hepatic steatosis because these groups have a greater likelihood of clinically relevant liver disease [8].

In the present study, participants with hepatic steatosis had significantly greater BMI, waist circumference and waist-to-hip ratio than those without steatosis. BMI ≥23 kg/m² and central obesity were present in 83.3% and 78.6% of participants with steatosis, compared with 59.6% and 46.8%, respectively, among those without steatosis. These findings supported the close relationship between excess adiposity, particularly abdominal adiposity, and hepatic fat accumulation. Recent population-based evidence has demonstrated that cardiometabolic risk factors contribute substantially to the development and progression of MASLD, with the metabolic phenotype influencing subsequent hepatic outcomes [9].

Diabetes mellitus showed one of the strongest associations with hepatic steatosis in the present study. Diabetes was present in 44.4% of participants with steatosis compared with 17.0% of those without steatosis, and it remained independently associated with steatosis after multivariable adjustment (aOR 2.76, 95% CI 1.46–5.22). This finding was clinically important because insulin resistance and abnormal glucose metabolism are central components of the metabolic environment associated with hepatic fat accumulation. A recent Indian multicentre study involving biopsy-confirmed MASLD reported that

type 2 diabetes was independently associated with advanced fibrosis, with diabetic participants having almost twice the odds of advanced fibrosis compared with those without diabetes [10]. Thus, the association observed in the present study may have important implications beyond the presence of steatosis itself.

The present study also demonstrated significant associations between hepatic steatosis and lipid abnormalities. Participants with steatosis had higher triglyceride and LDL-cholesterol concentrations and lower HDL-cholesterol concentrations than participants without steatosis. Hypertriglyceridaemia and low HDL-cholesterol remained independently associated with hepatic steatosis in the multivariable model. These findings were consistent with recent evidence showing that glucose and lipid metabolic abnormalities frequently cluster with obesity and elevated blood pressure in MASLD. Individuals exhibiting more severe cardiometabolic dysfunction have also been reported to have greater risks of advanced fibrosis and adverse long-term outcomes [9].

A progressive relationship was observed between the number of metabolic risk factors and the presence of hepatic steatosis. Steatosis was present in 20.8% of participants without metabolic risk factors, increasing to 33.3% among those with one risk factor, 56.0% among those with two risk factors and 100% among those with three or more risk factors. This pattern suggested that hepatic fat accumulation was more closely related to the overall metabolic burden than to an isolated abnormality. Similar findings have been reported in longitudinal research, in which increasing numbers of cardiometabolic risk factors among individuals with MASLD were associated with progressively greater cardiovascular risk [11].

The study further demonstrated increasing prevalence of metabolic abnormalities with increasing ultrasonographic severity of steatosis. Diabetes mellitus increased from 17.0% in participants without steatosis to 68.8% in those with Grade 3 steatosis, while hypertriglyceridaemia increased from 22.3% to 62.5%. Central obesity and dyslipidaemia showed similar patterns. These observations indicated that increasing sonographic severity of hepatic fat accumulation was accompanied by a progressively adverse metabolic profile. Recent research has similarly shown that distinct cardiometabolic phenotypes characterized by obesity, elevated blood pressure and severe glucose/lipid dysfunction were associated with substantially higher odds of advanced hepatic fibrosis [12].

Liver enzyme concentrations were also significantly higher in participants with hepatic steatosis. Mean ALT and AST levels were higher in the steatosis group than in the non-steatosis group. However, the presence of relatively normal liver enzyme values should not be interpreted as excluding hepatic steatosis or clinically relevant liver disease. Contemporary MASLD recommendations emphasize risk assessment based on metabolic risk factors and non-invasive assessment rather than relying on aminotransferase abnormalities alone [8]. In this context, ultrasonographic detection of steatosis may provide an opportunity to identify individuals who require further metabolic and hepatic evaluation.

Ultrasonography identified hepatic steatosis in more than half of the study population. The technique was particularly useful because it was non-invasive, readily available and practical for routine abdominal assessment. Nevertheless, conventional B-mode ultrasonography primarily provides qualitative or semi-quantitative assessment of hepatic fat and has limitations in detecting mild steatosis and distinguishing steatosis from other parenchymal abnormalities. Therefore, the ultrasonographic grades in the present study should be interpreted as sonographic severity categories rather than direct measurements of hepatic fat content. Where clinically indicated, individuals with steatosis and metabolic risk factors may require further fibrosis assessment using validated non-invasive tools [8].

The clinical relevance of identifying metabolic risk factors among individuals with hepatic steatosis extends beyond liver disease. MASLD has been associated with increased cardiovascular risk, particularly in individuals with greater metabolic dysfunction and hepatic fibrosis. A prospective cohort study reported increased cardiovascular disease risk among individuals with MASLD and elevated fibrosis scores, while the risk increased with the accumulation of cardiometabolic abnormalities [11]. Consequently, detection of hepatic steatosis during routine ultrasonography may represent an opportunity to recognize patients who require comprehensive assessment of cardiovascular and metabolic risk.

The present findings had several practical implications. The strong associations of hepatic steatosis with BMI, central obesity, diabetes, hypertriglyceridaemia and low HDL-cholesterol suggested that individuals undergoing abdominal ultrasonography could potentially benefit from simultaneous metabolic risk assessment. This approach was particularly relevant in settings where ultrasonography was widely available but advanced liver imaging was less accessible. Recent Indian expert recommendations have emphasized integrated management of MASLD through lifestyle modification and appropriate treatment of associated metabolic comorbidities [13,14].

The study had certain limitations. Its cross-sectional design prevented determination of temporal or causal relationships between metabolic risk factors and hepatic steatosis. Conventional ultrasonography was used rather than magnetic resonance-based fat quantification or histological assessment, and therefore mild steatosis could have been underestimated. The study was also conducted at a single tertiary care centre, which could have limited the generalizability of the findings to the wider community. In addition, the study focused primarily on hepatic steatosis and did not include systematic assessment of hepatic fibrosis using transient elastography or liver biopsy. Further prospective, multicentre studies incorporating quantitative steatosis and fibrosis assessment would therefore be useful.

Conclusion

Ultrasonographically detected hepatic steatosis was common among the studied adults and showed a significant association with multiple metabolic risk factors, particularly increased BMI, central obesity, diabetes mellitus, hypertriglyceridaemia and low HDL-cholesterol. The prevalence and severity of steatosis increased with the accumulation of metabolic abnormalities. These findings suggested that abdominal ultrasonography could serve as a useful and accessible tool for identifying individuals at increased metabolic risk and supported the importance of early screening and appropriate management of modifiable metabolic risk factors.

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