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Original Research Article

Association of Obesity and Glycaemic Control with Metabolic Dysfunction-Associated Steatotic Liver Disease in Patients with Type 2 Diabetes Mellitus

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Conflict of interest: Nil

Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is highly prevalent among patients with type 2 diabetes mellitus (T2DM) and is associated with an increased risk of progressive liver fibrosis. This study evaluated the association of MASLD with obesity and glycaemic control in patients with T2DM.

Methods: This hospital-based cross-sectional study included 100 adults with T2DM and ultrasonographic evidence of MASLD. Clinical, anthropometric, and biochemical data were collected. Hepatic steatosis was assessed by ultrasonography, while liver fibrosis was evaluated using shear wave elastography, the MASLD Fibrosis Score, Fibrosis-4 (FIB-4) index, and the Body Mass Index, AST/ALT Ratio, and Diabetes (BARD) score.

Results: The mean age was predominantly within 18–40 and 51–60 years, with females comprising 59.0% of participants. The mean BMI and HbA1c were 32.2 ± 2.87 kg/m² and 8.12 ± 1.65%, respectively. Mild steatosis (57.0%) was the most common ultrasonographic finding, while significant (44.0%) and advanced fibrosis (38.0%) predominated on elastography. Most participants were classified as high risk by the MASLD Fibrosis Score (60.0%) and BARD score (97.0%). No significant association was observed between steatosis severity and BMI (p=0.086), HbA1c (p=0.931), or liver stiffness (p=0.767).

Conclusion: MASLD was common among patients with T2DM and was frequently accompanied by obesity, poor glycaemic control, and significant liver fibrosis. Routine non-invasive fibrosis assessment may facilitate early identification of high-risk individuals and improve clinical management.

Keywords: Metabolic dysfunction-associated steatotic liver disease; Type 2 diabetes mellitus; Obesity; HbA1c; Shear wave elastography; Liver fibrosis.

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Introduction

Metabolic dysfunction associated steatotic liver disease (MASLD) is currently the most common chronic liver disease worldwide and has become a major public health concern because of the increasing prevalence of obesity and type 2 diabetes mellitus (T2DM). It is defined by the presence of hepatic steatosis in individuals with metabolic dysfunction after excluding significant alcohol intake and other causes of liver disease.[1] MASLD encompasses a spectrum ranging from simple steatosis to metabolic dysfunction associated steatohepatitis (MASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma.[2] The disease is closely linked to insulin resistance, obesity, dyslipidaemia, and chronic hyperglycaemia, making it an important hepatic manifestation of metabolic syndrome.[3] The understanding of fatty liver disease has evolved

considerably over the past few decades. Earlier terms such as non-alcoholic steatohepatitis (NASH) and non-alcoholic fatty liver disease (NAFLD) were gradually replaced after recognition that metabolic dysfunction is the primary driver of the disease.[4] In 2023, an international consensus introduced the term MASLD to better reflect its metabolic basis and provide a unified disease definition for both clinical practice and research.[5] Globally, MASLD affects nearly one quarter of the adult population, while its prevalence increases to 55–70% among patients with T2DM.[6] Diabetic individuals are more likely to develop progressive liver disease, including steatohepatitis and advanced fibrosis, and are at greater risk of cardiovascular disease, which remains the leading cause of mortality in this population.[7] The burden of MASLD has increased rapidly across the Asia Pacific region because of urbanisation, sedentary lifestyles, and dietary changes. South Asians are particularly susceptible, developing metabolic complications at lower body mass index (BMI) values owing to increased visceral adiposity and central obesity.[8]

India has one of the world's largest populations with T2DM, making MASLD an emerging healthcare challenge.[9] Previous Indian studies have reported a high prevalence of hepatic steatosis among diabetic patients, with obesity, central adiposity, and poor glycaemic control identified as major risk factors.[10] The relationship between MASLD and T2DM is bidirectional, as each condition promotes the development and progression of the other through insulin resistance and metabolic dysfunction.[11] Glycated haemoglobin (HbA1c), a marker of long term glycaemic control, has been associated with hepatic steatosis, inflammation, and fibrosis, while obesity, particularly abdominal obesity, plays a central role in disease progression.[12]

Although liver biopsy remains the reference standard for diagnosis, non-invasive techniques such as ultrasonography, shear wave elastography, and fibrosis scoring systems are increasingly used for the assessment of hepatic steatosis and fibrosis.[13] However, data evaluating the association of MASLD with obesity and HbA1c using these modalities in Indian patients with T2DM remain limited.[14]

Therefore, this study was undertaken to determine the prevalence of MASLD among patients with T2DM and to evaluate its association with obesity and HbA1c levels, thereby contributing evidence for early detection and risk stratification in this high risk population.

Materials and Methods

This hospital-based cross-sectional study was conducted in the Department of General Medicine, SGT Hospital, over a period of 18 months after obtaining approval from the Institutional Ethics Committee. Adult patients with type 2 diabetes mellitus (T2DM) attending the outpatient and inpatient departments were screened for eligibility. A total of 100 consecutive eligible participants were enrolled using a convenient sampling technique.

Patients aged more than 18 years with T2DM and ultrasonographic evidence of hepatic steatosis were included in the study irrespective of the duration of diabetes. Patients younger than 18 years or older than 85 years, those with a history of alcohol consumption, viral hepatitis (hepatitis A, B, C, D, or E), jaundice, alcoholic liver disease, pregnancy, or use of known hepatotoxic medications such as methotrexate, carbamazepine, erythromycin, sodium valproate, or isoniazid were excluded.

Written informed consent was obtained from all participants after explaining the study objectives in their local language. Demographic details, medical history, and clinical information were recorded using a predesigned case record proforma. Height and weight were measured using standardized techniques, and body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared (kg/m^2). BMI was categorized according to the World Health Organization Asian criteria.

Venous blood samples (5 mL) were collected under standard aseptic precautions for laboratory investigations. Glycated haemoglobin (HbA1c) was measured from whole blood using the immunoturbidometric method. Routine biochemical investigations, including liver function tests and platelet count, were performed using standard laboratory protocols. Diabetes mellitus was diagnosed according to the World Health Organization diagnostic criteria.[15]

All participants underwent abdominal ultrasonography to evaluate hepatic steatosis. Liver steatosis was graded based on hepatic echogenicity, liver-kidney contrast, visualization of the diaphragm, portal vein walls, and posterior hepatic parenchyma. Steatosis was classified as absent, mild, moderate, or severe according to established ultrasonographic criteria.

Liver fibrosis was further assessed using two-dimensional shear wave elastography (SWE) performed with a GE LOGIQ P10 XDclear ultrasound system equipped with a C1-6D convex array transducer. Examinations were carried out through a right intercostal approach with patients in the supine position, the right arm elevated above

the head, and during breath holding. Measurements were obtained from segments VII or VIII of the right hepatic lobe. Liver stiffness values were interpreted according to the METAVIR fibrosis staging system, categorizing fibrosis as F0, F1, F2, F3, or F4 based on established kilopascal cut-off values. The risk of advanced liver fibrosis was also evaluated using validated non-invasive fibrosis indices, including the MASLD Fibrosis Score, Fibrosis-4 (FIB-4) index, and the Body Mass Index, AST-to-ALT Ratio, and Diabetes (BARD) score. These scores were calculated using standard published equations and categorized into low-, intermediate-, or high-risk groups according to recommended cut-off values.[16], [17], [18]

The primary variables assessed were age, BMI, HbA1c level, ultrasonographic grade of hepatic steatosis, liver stiffness measured by shear wave elastography, and fibrosis risk determined by the MASLD Fibrosis Score, FIB-4 index, and BARD score.

Data were entered into Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS) software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Associations between categorical variables were evaluated using Pearson's chi-square test or Fisher's exact test, as appropriate. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 100 patients with type 2 diabetes mellitus (T2DM) and metabolic dysfunction-associated steatotic liver disease (MASLD) were included in the study. Participants were predominantly aged 18–40 years and 51–60 years (27.0% each), followed by 41–50 years (25.0%). Females constituted 59.0% of the study population. The mean duration of T2DM was 6.13 ± 2.70 years, with most patients (62.0%) having diabetes for 5–10 years. Hypertension was present in only 3.0% of participants. The mean body mass index (BMI) was 32.2 ± 2.87 kg/m², and the majority (78.0%) were classified as Obese II according to the Asian BMI criteria. The mean HbA1c level was 8.12 ± 1.65%, indicating suboptimal glycaemic control. The mean serum total cholesterol, triglycerides, HDL, LDL, and VLDL levels were 144.83 ± 39.65 mg/dL, 152.36 ± 60.61 mg/dL, 48.13 ± 13.99 mg/dL, 77.44 ± 20.78 mg/dL, and 30.44 ± 12.16 mg/dL, respectively. (Table 1) Ultrasonography demonstrated that mild hepatic steatosis (Grade 1) was the most common finding, observed in 57.0% of participants, followed by moderate steatosis (40.0%), while severe steatosis was identified in only 2.0% of patients. Hepatomegaly was present in 40.0% of participants, whereas cholelithiasis, splenomegaly, renal abnormalities, gallbladder abnormalities, and ascites were relatively uncommon. Shear wave elastography revealed significant fibrosis (F2) in 44.0% of participants, followed by advanced fibrosis (F3) in 38.0%. Mild fibrosis (F1), no fibrosis (F0), and cirrhosis (F4) were observed in 14.0%, 2.0%, and 2.0% of participants, respectively. The overall mean liver stiffness was 8.84 ± 1.27 kPa. (Table 2)

Assessment using non-invasive fibrosis scores demonstrated that the mean MASLD Fibrosis Score was 0.57 ± 1.42, with 60.0% of participants categorized as high risk (F3–F4), while 28.0% had indeterminate scores and 10.0% were classified as low risk (F0–F2). The mean FIB-4 index was 1.91 ± 1.23, with 50.0% of participants in the intermediate-risk category, 32.0% in the low-risk category, and 18.0% in the high-risk category. The mean BARD score was 3.38 ± 0.95, and the majority of participants (97.0%) were classified as high risk for advanced fibrosis. (Table 3)

The association between BMI category and ultrasonographic steatosis severity was evaluated using the Asian BMI classification. Most participants with both mild and moderate steatosis belonged to the Obese II category.

Although moderate steatosis was more frequent among participants with higher BMI, the association between BMI category and steatosis severity was not statistically significant ($\chi^2 = 6.59$, $p = 0.086$). (Table 4) Comparison of glycaemic parameters across ultrasonographic steatosis grades showed no statistically significant differences. Mean HbA1c values were comparable across steatosis grades ($p = 0.931$). Similarly, fasting blood glucose ($p = 0.742$) and postprandial blood glucose ($p = 0.864$) did not differ significantly according to steatosis severity. (Table 5)

The relationship between ultrasonographic steatosis severity and liver fibrosis assessed by shear wave elastography demonstrated that significant fibrosis (F2) and advanced fibrosis (F3) were the predominant fibrosis stages among patients with mild and moderate steatosis. Mean liver stiffness ranged from 8.76 ± 1.47 kPa in patients with moderate steatosis to 9.88 kPa in the single participant without steatosis. However, no statistically significant difference in liver stiffness was observed across steatosis grades ($p = 0.767$), indicating that ultrasonographic steatosis severity was not significantly associated with liver stiffness in the present study. (Table 6)

Table 1: Baseline Demographic, Clinical, Anthropometric, and Biochemical Characteristics of the Study Population (N = 100)

Variable		**n (%)/Mean ± SD**
Age group	18–40 years	27 (27.0)
	41–50 years	25 (25.0)
	51–60 years	27 (27.0)
	61–70 years	14 (14.0)
	>70 years	7 (7.0)
Sex	Male	41 (41.0)
	Female	59 (59.0)
Duration of T2DM	<5 years	33 (33.0)
	5–10 years	62 (62.0)
	>10 years	5 (5.0)
Mean duration (years)		6.13 ± 2.70
Hypertension	Present	3 (3.0)
	Absent	97 (97.0)
Anthropometric parameters	Height (cm)	155.8 ± 4.80
	Weight (kg)	78.0 ± 5.03
	Body mass index (kg/m²)	32.2 ± 2.87
BMI category (Asian criteria)	Obese I (25.0–29.9 kg/m²)	22 (22.0)
	Obese II (≥30 kg/m²)	78 (78.0)
Glycaemic profile	HbA1c (%)	8.12 ± 1.65
Lipid profile (mg/dL)	Total cholesterol	144.83 ± 39.65
	Triglycerides	152.36 ± 60.61
	HDL cholesterol	48.13 ± 13.99
	LDL cholesterol	77.44 ± 20.78
	VLDL cholesterol	30.44 ± 12.16

Table 2: Ultrasonographic Findings and Shear Wave Elastography Assessment of the Study Population (N = 100)

Parameter	**Category**	**n (%)**	**SWE (kPa), Mean ± SD**
Hepatic steatosis (USG)	Absent	1 (1.0)	—
	Mild (Grade 1)	57 (57.0)	—
	Moderate (Grade 2)	40 (40.0)	—
	Severe (Grade 3)	2 (2.0)	—
METAVIR fibrosis stage (SWE)	F0 (No fibrosis)	2 (2.0)	3.72 ± 0.37
	F1 (Mild fibrosis)	14 (14.0)	7.55 ± 1.06
	F2 (Significant fibrosis)	44 (44.0)	8.71 ± 0.47
	F3 (Advanced fibrosis)	38 (38.0)	9.52 ± 0.11
	F4 (Cirrhosis)	2 (2.0)	13.21 ± 1.21
Overall liver stiffness			**8.84 ± 1.27**
Additional ultrasonographic findings	Hepatomegaly	40 (40.0)	—
	Cholelithiasis	8 (8.0)	—
	Splenomegaly	8 (8.0)	—
	Renal abnormalities	20 (20.0)	—
	Gallbladder abnormality	1 (1.0)	—
	Ascites	4 (4.0)	—

Table 3: Distribution of Non-invasive Fibrosis Scores Among Study Participants with Type 2 Diabetes Mellitus and MASLD (N = 100)

Fibrosis Assessment Tool	**Summary Statistic**	**Risk Category**	**n (%)**
MASLD Fibrosis Score	Mean ± SD: **0.57 ± 1.42** Median (Range): **0.87 (−3.72 to 3.88)**	F0–F1	2 (2.0)
		F0–F2	10 (10.0)
		Indeterminate	28 (28.0)
		F3–F4	60 (60.0)
FIB-4 Index	Mean ± SD: **1.91 ± 1.23** Median (Range): **1.73 (0.32 to 7.97)**	Low risk	32 (32.0)
		Intermediate risk	50 (50.0)
		High risk	18 (18.0)

BARD Score	Mean ± SD: **3.38 ± 0.95** Median (Range): **4.00 (1–4)**	Low risk (1 point)	3 (3.0)
		High risk (2–4 points)	97 (97.0)

Table 4: Association between Body Mass Index Category and Ultrasonographic Steatosis Severity in Patients with Type 2 Diabetes Mellitus and MASLD (N = 100)

Steatosis Grade	Obese I (25.0–29.9 kg/m²) n (%)	Obese II (≥30 kg/m²) n (%)	Total n (%)	P Value
Absent	0 (0.0)	1 (1.0)	1 (1.0)	0.086
Mild (Grade 1)	17 (17.0)	40 (40.0)	57 (57.0)	
Moderate (Grade 2)	4 (4.0)	36 (36.0)	40 (40.0)	
Severe (Grade 3)	1 (1.0)	1 (1.0)	2 (2.0)	
Total	**22 (22.0)**	**78 (78.0)**	**100 (100.0)**	
Chi-square test; p<0.05 Significant				

Table 5: Comparison of Glycaemic Parameters According to Ultrasonographic Steatosis Severity in Patients with Type 2 Diabetes Mellitus and MASLD (N = 100)

Parameter	Absent (n = 1)	Mild (Grade 1) (n = 57)	Moderate (Grade 2) (n = 40)	Severe (Grade 3) (n = 2)	p-value
HbA1c (%)	9.00	8.10 ± 1.47	8.11 ± 1.91	8.45 ± 2.19	0.931
Fasting blood glucose (mg/dL)	167.0	147.25 ± 23.84	151.76 ± 24.97	143.00 ± 15.56	0.742
Postprandial blood glucose (mg/dL)	287.0	255.60 ± 39.37	253.80 ± 40.79	240.00 ± 7.07	0.864
one-way ANOVA; p<0.05 Significant					

Table 6: Association between Ultrasonographic Steatosis Severity and Liver Fibrosis Assessed by Shear Wave Elastography in Patients with Type 2 Diabetes Mellitus and MASLD (N = 99)

Steatosis Grade	F0 n (%)	F1 n (%)	F2 n (%)	F3 n (%)	F4 n (%)	Liver Stiffness (kPa) Mean ± SD	p-value
Absent	0 (0.0)	0 (0.0)	0 (0.0)	1 (100.0)	0 (0.0)	9.88	**0.767**
Mild (Grade 1)	0 (0.0)	8 (14.0)	30 (52.6)	18 (31.6)	1 (1.8)	8.86 ± 1.15	
Moderate (Grade 2)	2 (5.1)	6 (15.4)	14 (35.9)	16 (41.0)	1 (2.6)	8.76 ± 1.47	
Severe (Grade 3)	0 (0.0)	0 (0.0)	0 (0.0)	2 (100.0)	0 (0.0)	9.34 ± 0.28	
Overall	**2 (2.0)**	**14 (14.1)**	**44 (44.4)**	**37 (37.4)**	**2 (2.0)**	**8.84 ± 1.27**	
F0: No fibrosis; **F1:** Mild fibrosis; **F2:** Significant fibrosis; **F3:** Advanced fibrosis; **F4:** Cirrhosis. One-way ANOVA comparing mean liver stiffness across ultrasonographic steatosis grades.							

Discussion

The present hospital-based cross-sectional study evaluated the occurrence of metabolic dysfunction-associated steatotic liver disease (MASLD) in patients with type 2 diabetes mellitus (T2DM) and examined its association with obesity, glycaemic control, and liver fibrosis using ultrasonography, shear wave elastography, and validated non-invasive fibrosis scores. The findings highlight the substantial burden of MASLD among individuals with T2DM and reinforce the close relationship between metabolic dysfunction and hepatic disease.

Most participants belonged to the middle-aged population, with the highest proportions observed in the 18–40 and 51–60 year age groups. Female participants slightly outnumbered males in the present study. Similar demographic patterns have been reported by Ali et al. and Latif and Ahsan, who observed that MASLD is common among middle-aged individuals with T2DM, particularly in populations with a high prevalence of metabolic risk factors.[19], [20] Han et al. also reported that age and female sex were associated with an increased likelihood of fatty liver disease in diabetic patients.[21] These findings suggest that demographic characteristics, together with long-standing metabolic abnormalities, contribute to the development of MASLD. Obesity emerged as a prominent characteristic of the study population. The mean body mass index was 32.2 ± 2.87 kg/m², and most participants were classified as Obese II according to Asian BMI criteria. Although a greater proportion of moderate steatosis was observed among participants with higher BMI, the association between BMI category and steatosis severity did not achieve statistical significance. Similar observations have been reported by Luxmi et al., Afolabi et al., and Han et al., who demonstrated that obesity is an important contributor to hepatic steatosis in patients with T2DM.[21], [22], [23] The absence of statistical significance in the present study may be explained by the predominance of obese individuals within the study population, limiting variability across BMI categories. Poor glycaemic control was

another important finding. The mean HbA1c level was 8.12 ± 1.65%, indicating inadequate long-term glycaemic control in a large proportion of participants. However, HbA1c values did not differ significantly across ultrasonographic grades of steatosis. Comparable findings have been reported by Ali et al., who demonstrated higher HbA1c levels among patients with MASLD, while Han et al. identified poor glycaemic control as an independent metabolic risk factor for fatty liver disease.[19], [21] Although the present study did not demonstrate a statistically significant association between HbA1c and steatosis severity, the consistently elevated HbA1c levels support the close metabolic relationship between diabetes and MASLD.

Assessment of liver fibrosis showed that significant (F2) and advanced (F3) fibrosis constituted the majority of cases on shear wave elastography, whereas cirrhosis was uncommon.

Likewise, non-invasive fibrosis assessment revealed that most participants were classified as high risk by the MASLD Fibrosis Score and BARD score, while half of the cohort fell into the intermediate-risk category according to the FIB-4 index. These findings are consistent with studies by Luo et al. and Öztürk et al., who reported that non-invasive fibrosis assessment provides valuable information for identifying diabetic patients at increased risk of advanced liver fibrosis without the need for liver biopsy.[24], [25]

No statistically significant association was observed between ultrasonographic steatosis severity and liver stiffness measured by shear wave elastography. Similarly, fibrosis risk assessed using the MASLD Fibrosis Score, FIB-4 index, and BARD score did not vary significantly across steatosis grades. These findings suggest that the degree of hepatic steatosis alone may not accurately reflect fibrosis severity, emphasising the importance of incorporating elastography and validated fibrosis scores into the routine evaluation of patients with T2DM and MASLD.

Overall, the present study demonstrates that MASLD is highly prevalent among patients with T2DM and is frequently accompanied by obesity, poor glycaemic control, and a considerable burden of liver fibrosis. These findings support the routine use of non-invasive imaging and fibrosis assessment tools for early identification of high-risk patients, enabling timely intervention to prevent progression to advanced liver disease and its associated complications.

Conclusion

Metabolic dysfunction-associated steatotic liver disease (MASLD) was common among patients with type 2 diabetes mellitus and was frequently associated with obesity and poor glycaemic control. A substantial proportion of participants demonstrated significant or advanced liver fibrosis on shear wave elastography and non-invasive fibrosis assessment, highlighting the need for routine fibrosis evaluation in this high-risk population. Although the severity of hepatic steatosis was not significantly associated with BMI, HbA1c, or fibrosis stage, the overall findings emphasize the importance of early screening using ultrasonography, elastography, and validated fibrosis scores. Timely identification of high-risk patients may facilitate appropriate management and help prevent progression to advanced liver disease.

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