Section: General Medicine

Original Research Article

PREVALENCE AND PREDICTORS OF METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS: AN OBSERVATIONAL STUDY

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Received *: 15/09/2026*
Received in revised form *: 14/10/2026*
Accepted *: 28/10/2026*

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DOI: 10.70034/ijmedph.2026.4.273

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

Int J Med Pub Health
2026; 16 (4); 1798-1804

ABSTRACT

Background: Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is an increasingly recognized hepatic complication among patients with Type 2 Diabetes Mellitus (T2DM). Its association with obesity, insulin resistance, poor glycaemic control, and dyslipidaemia highlights the importance of early identification and appropriate metabolic assessment. The aim is to determine the prevalence of MASLD and evaluate its clinical, anthropometric, and biochemical predictors among patients with T2DM attending a tertiary care hospital.

Materials and Methods: This hospital-based, observational, cross-sectional analytical study included 92 patients with T2DM. Detailed demographic, clinical, anthropometric, and biochemical assessments were performed. MASLD was diagnosed using abdominal ultrasonography and established diagnostic criteria. Patients were categorized into MASLD and non-MASLD groups. The Fibrosis-4 (FIB-4) index was calculated to assess fibrosis risk. Statistical analysis was performed using SPSS version 28.0, including appropriate comparative tests and multivariable binary logistic regression. A p-value <0.05 was considered statistically significant.

Results: MASLD was identified in 62 patients (67.39%). Among affected patients, mild, moderate, and severe steatosis were observed in 46.77%, 37.10%, and 16.13%, respectively. Patients with MASLD had significantly higher BMI (p=0.007), waist circumference (p=0.026), HbA1c (p=0.001), triglycerides (p=0.002), ALT (p<0.001), and AST (p<0.001). Multivariable analysis identified BMI ≥25 kg/m² (aOR=2.94; p=0.048) and HbA1c ≥8% (aOR=4.78; p=0.004) as significant independent predictors of MASLD.

Conclusion: MASLD was frequently observed among patients with T2DM. Elevated BMI and poor glycaemic control emerged as significant independent predictors. Routine hepatic assessment and effective metabolic management may facilitate early identification and appropriate risk stratification.

Keywords: Metabolic Dysfunction-Associated Steatotic Liver Disease; Type 2 Diabetes Mellitus; Glycated Haemoglobin; Obesity; Hepatic Steatosis.

INTRODUCTION

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) has emerged as an important chronic liver disorder closely linked to the increasing burden of Type 2 Diabetes Mellitus (T2DM), obesity, and other cardiometabolic abnormalities. It is characterized by excessive accumulation of fat within hepatocytes in the presence of metabolic dysfunction and warrants attention in diabetes care. Liver disease may remain unrecognized when diabetes care focuses mainly on cardiovascular, renal, neurological, and ophthalmic complications. Including liver assessment in diabetes care supports timely detection and appropriate risk stratification. This is especially relevant for patients undergoing regular metabolic

review who may otherwise have no indication for targeted liver investigations.[1] The term MASLD was introduced to replace Non-Alcoholic Fatty Liver Disease (NAFLD), reflecting a shift toward diagnostic criteria based on positive evidence of cardiometabolic dysfunction rather than terminology defined primarily by exclusion. MASLD requires evidence of hepatic steatosis together with at least one cardiometabolic risk factor, of which T2DM is an established example. It also distinguishes MASLD from steatosis attributable to harmful alcohol intake or alternative aetiologies. This framework emphasizes metabolic dysfunction as a defining characteristic of liver disease and facilitates communication among different clinical specialties.[2] The association between T2DM and hepatic steatosis is biologically complex and bidirectional. Insulin resistance increases adipose tissue lipolysis and the delivery of free fatty acids to the liver, while hyperinsulinaemia and disturbed glucose metabolism enhance hepatic lipogenesis. Mitochondrial dysfunction, oxidative stress, altered adipokine secretion, and low-grade systemic inflammation further contribute to hepatocellular injury. Conversely, accumulated liver fat may aggravate hepatic insulin resistance, interfere with glucose regulation, and perpetuate metabolic disturbances. These processes explain the frequent coexistence of diabetes and liver steatosis.[3] Several clinical and biochemical characteristics may influence the likelihood of MASLD among patients with T2DM. Generalized obesity, central adiposity, poor long-term glycaemic control, hypertriglyceridaemia, low high-density lipoprotein cholesterol, hypertension, and physical inactivity are potentially relevant factors. Body mass index and waist circumference provide accessible estimates of adiposity, whereas glycated haemoglobin reflects chronic glycaemic exposure. Lipid profiles and liver function tests help characterize associated abnormalities. Steatosis may also occur without overt obesity, limiting assessment based solely on body weight. This is particularly relevant where central adiposity and metabolic abnormalities coexist despite relatively modest body mass index values.[4] MASLD encompasses a broad clinical spectrum extending from uncomplicated hepatic steatosis to Metabolic Dysfunction-Associated Steatohepatitis (MASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma. Its clinical significance is not restricted to hepatic outcomes, as associated cardiometabolic disease may complicate the overall management of T2DM. Many patients remain asymptomatic, and the absence of markedly elevated liver enzymes does not reliably exclude steatosis or clinically important fibrosis. Recognition requires clinical assessment, laboratory tests, and non-invasive liver evaluation. This supports timely referral and individualized management.[5] Abdominal ultrasonography is a widely available, non-invasive method for identifying hepatic steatosis in hospital practice. Increased liver echogenicity, hepatorenal contrast, posterior attenuation, and reduced visualization of intrahepatic vessels support the diagnosis, while sonographic appearance can provide an approximate grading of fatty infiltration. However, conventional ultrasonography has limited sensitivity for mild steatosis and cannot reliably distinguish uncomplicated fat accumulation from steatohepatitis or establish the degree of fibrosis. Elastography and magnetic resonance methods offer additional information but may be unavailable. Pragmatic diagnostic pathways remain essential.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, observational, cross-sectional analytical study to determine the prevalence and predictors of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) among patients diagnosed with Type 2 Diabetes Mellitus (T2DM). The study aimed to evaluate the association of MASLD with demographic characteristics, anthropometric measurements, glycaemic control, lipid abnormalities, liver function parameters, metabolic risk factors, and diabetes-related complications. Additionally, the study assessed independent clinical and biochemical predictors associated with the presence of MASLD. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients attending the outpatient department or admitted to medical wards with an established diagnosis of T2DM were evaluated for eligibility. Institutional A total of 92 patients diagnosed with T2DM were included in the study using a consecutive sampling technique. Eligible patients fulfilling the inclusion criteria were recruited and subjected to detailed clinical, biochemical, and ultrasonographic evaluations. Based on abdominal ultrasonography and established diagnostic criteria, patients were classified into two groups: Group A comprised patients with MASLD, while Group B included patients without MASLD. The prevalence of MASLD was calculated as the proportion of affected patients among the total study population.

Inclusion Criteria

Patients aged 18 years and above with an established diagnosis of T2DM, irrespective of diabetes duration, were included. Both male and female patients receiving oral antidiabetic medications, insulin therapy, or combination treatment were eligible. Patients willing to undergo clinical examination, laboratory investigations, and abdominal ultrasonography were enrolled after providing informed consent.

Exclusion Criteria

Patients with significant alcohol consumption exceeding 140 g/week in women or 210 g/week in men were excluded to avoid alcohol-associated or mixed-etiology steatotic liver disease. Patients with chronic hepatitis B or C, autoimmune hepatitis, Wilson disease, haemochromatosis, known drug-

induced hepatic steatosis, or other established causes of chronic liver disease were excluded. Pregnant women, patients with severe systemic illness, and individuals with incomplete essential clinical or investigation records were also excluded.

Methodology: Detailed demographic and clinical information was recorded using a structured proforma. Parameters included age, sex, residential background, duration of diabetes, family history of diabetes, smoking status, alcohol consumption, dietary habits, physical activity, and current antidiabetic treatment. Associated comorbidities, including hypertension, dyslipidaemia, cardiovascular disease, and chronic kidney disease, were documented. Diabetes-related complications, including peripheral neuropathy, nephropathy, and retinopathy, were assessed from clinical findings and available medical records.

Anthropometric and Physical Examination: All participants underwent general physical examination and anthropometric assessment. Height, weight, body mass index (BMI), waist circumference, systolic blood pressure, and diastolic blood pressure were recorded. BMI was calculated as weight in kilograms divided by height in metres squared. Overweight and obesity were classified using Asian-specific BMI criteria. Central obesity was assessed using waist circumference cut-offs of ≥90 cm for men and ≥80 cm for women. Hypertension was identified from documented diagnosis, antihypertensive treatment, or elevated blood pressure measurements.

Biochemical Investigations: Venous blood samples were collected for fasting blood glucose, postprandial blood glucose, glycated haemoglobin (HbA1c), lipid profile, liver function tests, renal function tests, and complete blood count. The lipid profile included total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Liver function parameters included alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase, serum bilirubin, albumin, and total protein. Serum creatinine, estimated glomerular filtration rate, platelet count, and urinary albumin-to-creatinine ratio were also assessed.

Ultrasonographic Evaluation and Diagnosis of MASLD: Abdominal ultrasonography was performed to identify hepatic steatosis based on increased hepatic echogenicity, hepatorenal contrast, posterior beam attenuation, and reduced visualisation of intrahepatic vessels. Hepatic steatosis was categorised as mild (Grade I), moderate (Grade II), or severe (Grade III). MASLD was diagnosed when ultrasonographic hepatic steatosis was present alongside T2DM, a recognised cardiometabolic risk factor, after evaluation for significant alcohol consumption and alternative causes of hepatic steatosis. Diagnostic classification followed contemporary MASLD recommendations.

Assessment of Liver Fibrosis: The Fibrosis-4 (FIB-4) index was calculated using age, AST, ALT, and platelet count to estimate the probability of advanced hepatic fibrosis. FIB-4 values were categorised using established risk thresholds, with age-adjusted interpretation in older patients. The association between FIB-4 scores, metabolic parameters, and MASLD was evaluated.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS), version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. The independent samples t-test or Mann–Whitney U test was applied for continuous variables, while the Chi-square test or Fisher's exact test was used for categorical variables. Multivariable binary logistic regression was performed using a limited number of clinically relevant variables to identify independent predictors of MASLD. Adjusted odds ratios with 95% confidence intervals were calculated. A p-value of less than 0.05 was considered statistically significant.

RESULTS

[Table 1] Prevalence and Ultrasonographic Grading of MASLD Among Patients with Type 2 Diabetes Mellitus (N=92)

[Table 1] demonstrates the prevalence and ultrasonographic severity of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) among 92 patients with Type 2 Diabetes Mellitus. MASLD was identified in 62 patients (67.39%), whereas 30 patients (32.61%) showed no evidence of hepatic steatosis. Among the 62 patients diagnosed with MASLD, mild hepatic steatosis (Grade I) was the most common finding, observed in 29 patients (46.77%). Moderate hepatic steatosis (Grade II) was detected in 23 patients (37.10%), while severe hepatic steatosis (Grade III) was observed in 10 patients (16.13%). Overall, 33 patients (53.23%) with MASLD had moderate-to-severe hepatic steatosis. These findings indicate a high prevalence of MASLD among patients with T2DM, with mild and moderate grades accounting for the majority of cases.

[Table 2] Comparison of Demographic and Clinical Characteristics Between Patients With and Without MASLD (N=92)

[Table 2] compares the demographic and clinical characteristics of patients with and without MASLD. The mean age was comparable between patients with MASLD (54.77 ± 9.35 years) and those without MASLD (54.90 ± 10.18 years), with no statistically significant difference (p=0.952). Male patients constituted 53.23% of the MASLD group and 56.67% of the non-MASLD group (p=0.756), while diabetes duration was also comparable between the groups (8.63 ± 3.99 versus 8.27 ± 4.22 years; p=0.697). However, mean body mass index was significantly higher among patients with MASLD (26.11 ± 3.56 kg/m²) compared with those without MASLD (23.96

± 3.38 kg/m²; p=0.007). Similarly, mean waist circumference was significantly greater in the MASLD group (90.76 ± 11.44 cm versus 85.23 ± 10.67 cm; p=0.026). Hypertension (46.77% versus 36.67%; p=0.359) and family history of diabetes (48.39% versus 46.67%; p=0.877) did not differ significantly between the groups, indicating that obesity-related anthropometric parameters were more strongly associated with MASLD in this comparison.

[Table 3] Comparison of Glycaemic, Lipid, and Liver Function Parameters Between Patients With and Without MASLD (N=92)

[Table 3] presents the comparison of glycaemic status, lipid profile, liver function parameters, and fibrosis indices between patients with and without MASLD. Mean fasting blood glucose was higher in the MASLD group (207.70 ± 26.67 mg/dL) than in the non-MASLD group (199.22 ± 24.40 mg/dL), although the difference was not statistically significant (p=0.135). However, mean HbA1c was significantly elevated among MASLD patients (8.30 ± 1.13% versus 7.50 ± 1.04%; p=0.001), suggesting an association between poorer long-term glycaemic control and hepatic steatosis. Serum triglyceride levels were also significantly higher in patients with MASLD (158.93 ± 42.28 mg/dL versus 127.92 ± 44.30 mg/dL; p=0.002), whereas HDL and LDL cholesterol levels showed no significant differences (p=0.913 and p=0.561, respectively). Liver enzyme levels were significantly elevated in the MASLD group, with mean ALT values of 34.63 ± 9.78 U/L versus 20.75 ± 9.31 U/L and AST values of 26.54 ± 7.39 U/L versus 20.14 ± 6.03 U/L (both p<0.001). The mean FIB-4 index was comparable between the groups (1.12 ± 0.46 versus 1.09 ± 0.61; p=0.810), indicating no statistically significant difference in this non-invasive fibrosis index.

[Table 4] Association of Metabolic Risk Factors, Fibrosis Risk, and Diabetes-Related Complications with MASLD (N=92)

[Table 4] illustrates the association of metabolic abnormalities, fibrosis risk, and diabetes-related complications with MASLD. A BMI ≥25 kg/m² was significantly more frequent among patients with MASLD (64.52%) compared with those without MASLD (30.00%; p=0.002), while central obesity was observed in 67.74% and 43.33% of patients, respectively (p=0.025). Poor glycaemic control, defined as HbA1c ≥8%, was significantly more common in the MASLD group (59.68%) than in the non-MASLD group (23.33%; p=0.001). Similarly, hypertriglyceridaemia (≥150 mg/dL) was present in 58.06% of MASLD patients compared with 30.00% of non-MASLD patients (p=0.012). The proportion of patients with FIB-4 values above the age-adjusted low-risk threshold was comparable between the groups (25.81% versus 23.33%; p=0.797). Diabetic nephropathy (30.65% versus 40.00%; p=0.374) and diabetic retinopathy (30.65% versus 23.33%; p=0.465) also showed no statistically significant associations with MASLD, suggesting that obesity, poor glycaemic control, and hypertriglyceridaemia were the principal metabolic factors associated with hepatic steatosis in the presented data.

[Table 5] Multivariable Binary Logistic Regression Analysis of Predictors of MASLD Among Patients with Type 2 Diabetes Mellitus (N=92)

[Table 5] presents the multivariable binary logistic regression analysis performed to identify independent predictors of MASLD among patients with T2DM. After adjustment for the other variables included in the model, BMI ≥25 kg/m² was significantly associated with MASLD, with an adjusted odds ratio (aOR) of 2.94 (95% CI: 1.01–8.55; p=0.048), indicating approximately threefold higher odds of MASLD among overweight or obese patients. Poor glycaemic control (HbA1c ≥8%) emerged as the strongest statistically significant predictor, with an aOR of 4.78 (95% CI: 1.67–13.68; p=0.004). Hypertriglyceridaemia (≥150 mg/dL) demonstrated increased adjusted odds of MASLD (aOR=2.39; 95% CI: 0.82–6.95), although the association did not reach statistical significance (p=0.109). Hypertension showed no meaningful independent association with MASLD (aOR=1.04; 95% CI: 0.37–2.94; p=0.937).

Table 1: Prevalence and Ultrasonographic Grading of MASLD Among Patients with Type 2 Diabetes Mellitus (N=92)

Parameter	Number of patients	Percentage (%)
MASLD status (N=92)		
MASLD present	62	67.39
MASLD absent	30	32.61
Ultrasonographic grading among MASLD patients (n=62)		
Grade I (Mild steatosis)	29	46.77
Grade II (Moderate steatosis)	23	37.10
Grade III (Severe steatosis)	10	16.13
Total MASLD patients	62	100.00

Table 2: Comparison of Demographic and Clinical Characteristics Between Patients With and Without MASLD (N=92)

Parameter	MASLD present (n=62)	MASLD absent (n=30)	p-value
Age (years), mean ± SD	54.77 ± 9.35	54.90 ± 10.18	0.952
Male sex	33 (53.23%)	17 (56.67%)	0.756
Duration of diabetes (years), mean ± SD	8.63 ± 3.99	8.27 ± 4.22	0.697
BMI (kg/m²), mean ± SD	26.11 ± 3.56	23.96 ± 3.38	0.007
Waist circumference (cm), mean ± SD	90.76 ± 11.44	85.23 ± 10.67	0.026
Hypertension	29 (46.77%)	11 (36.67%)	0.359
Family history of diabetes	30 (48.39%)	14 (46.67%)	0.877

Table 3: Comparison of Glycaemic, Lipid, and Liver Function Parameters Between Patients With and Without MASLD (N=92)

Biochemical parameter	MASLD present (n=62)	MASLD absent (n=30)	p-value
Fasting blood glucose (mg/dL)	207.70 ± 26.67	199.22 ± 24.40	0.135
HbA1c (%)	8.30 ± 1.13	7.50 ± 1.04	0.001
Triglycerides (mg/dL)	158.93 ± 42.28	127.92 ± 44.30	0.002
HDL cholesterol (mg/dL)	46.63 ± 7.65	46.82 ± 7.69	0.913
LDL cholesterol (mg/dL)	103.66 ± 20.93	100.29 ± 27.95	0.561
ALT (U/L)	34.63 ± 9.78	20.75 ± 9.31	<0.001
AST (U/L)	26.54 ± 7.39	20.14 ± 6.03	<0.001
FIB-4 index	1.12 ± 0.46	1.09 ± 0.61	0.810

Table 4: Association of Metabolic Risk Factors, Fibrosis Risk, and Diabetes-Related Complications with MASLD (N=92)

Parameter	MASLD present (n=62)	MASLD absent (n=30)	p-value
BMI ≥25 kg/m²	40 (64.52%)	9 (30.00%)	0.002
Central obesity	42 (67.74%)	13 (43.33%)	0.025
Poor glycaemic control (HbA1c ≥8%)	37 (59.68%)	7 (23.33%)	0.001
Hypertriglyceridaemia (≥150 mg/dL)	36 (58.06%)	9 (30.00%)	0.012
FIB-4 above age-adjusted low-risk threshold	16 (25.81%)	7 (23.33%)	0.797
Diabetic nephropathy	19 (30.65%)	12 (40.00%)	0.374
Diabetic retinopathy	19 (30.65%)	7 (23.33%)	0.465

Table 5: Multivariable Binary Logistic Regression Analysis of Predictors of MASLD Among Patients with Type 2 Diabetes Mellitus (N=92)

Predictor variable	Adjusted odds ratio (aOR)	95% Confidence interval	p-value
BMI ≥25 kg/m²	2.94	1.01–8.55	0.048
HbA1c ≥8%	4.78	1.67–13.68	0.004
Triglycerides ≥150 mg/dL	2.39	0.82–6.95	0.109
Hypertension	1.04	0.37–2.94	0.937

DISCUSSION

The present hospital-based observational study involving 92 patients with Type 2 Diabetes Mellitus (T2DM) demonstrated a high prevalence of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), which was identified in 62 patients (67.39%), while 30 patients (32.61%) showed no evidence of hepatic steatosis. These findings were comparable with those reported by Sinha et al. (2023), who conducted a cross-sectional study among 132 patients with T2DM in eastern India and observed non-alcoholic fatty liver disease (NAFLD) in 75 patients (approximately 57.00%). The prevalence observed in the present study was approximately 10 percentage points higher than that reported by Sinha et al. This difference may be attributed to variations in demographic characteristics, obesity prevalence, metabolic risk profiles, and diagnostic approaches. Both studies demonstrated a substantial burden of hepatic steatosis among diabetic patients, emphasising the importance of liver assessment in this high-risk population.[7] Regarding ultrasonographic severity, the present study demonstrated that mild hepatic steatosis (Grade I) was the most common finding, affecting 29 patients (46.77%) with MASLD, followed by moderate steatosis (Grade II) in 23 patients (37.10%) and severe steatosis (Grade III) in 10 patients (16.13%). Bhuvanesswar et al. (2025), in an Indian study involving 1,070 individuals with T2DM, reported hepatic steatosis in 75.60% of participants using transient elastography. They observed mild (S1), moderate (S2), and severe (S3) steatosis in 16.00%, 20.10%, and 39.50% of the total study population, respectively. In comparison, severe steatosis accounted for a smaller proportion of affected patients in the present study, whereas mild and moderate steatosis predominated. However, the two distributions cannot be directly compared because the present study used ultrasonographic grades among patients with MASLD, whereas Bhuvanesswar et al. reported controlled attenuation parameter grades among all participants. These findings demonstrate the considerable variation in hepatic steatosis severity among individuals with T2DM.[8] The comparison of demographic and anthropometric characteristics in the present study revealed that age, sex, and duration of diabetes were not significantly associated with MASLD. The mean age was 54.77 ± 9.35 years in patients with MASLD and 54.90 ± 10.18 years in those without MASLD (p=0.952). However, mean BMI was significantly higher in the MASLD group (26.11 ± 3.56 versus 23.96 ± 3.38 kg/m²; p=0.007), along with waist circumference (90.76 ± 11.44 versus 85.23 ± 10.67 cm; p=0.026). Similar findings were reported by Kuchay et al. (2021), who observed NAFLD in 84.20% of Indian patients with T2DM. Their study demonstrated significantly higher BMI among patients with NAFLD compared with those without NAFLD (28.00 versus 25.10 kg/m²; p<0.0001), while obesity defined as BMI ≥25 kg/m² was present in 80.50% and 52.40%, respectively. They also reported no significant association with age or sex, supporting the importance of adiposity rather than demographic characteristics in identifying diabetic patients at increased risk of hepatic steatosis.[9]

The biochemical analysis in the present study demonstrated significant associations of MASLD with poor glycaemic control, hypertriglyceridaemia, and elevated liver enzymes. Mean HbA1c was significantly higher among MASLD patients (8.30 ± 1.13% versus 7.50 ± 1.04%; p=0.001), while serum triglycerides were also elevated (158.93 ± 42.28 versus 127.92 ± 44.30 mg/dL; p=0.002). Similarly, ALT levels were significantly higher in patients with MASLD (34.63 ± 9.78 versus 20.75 ± 9.31 U/L; p<0.001). Latif et al. (2024), in a cross-sectional study involving 646 participants with T2DM and/or obesity, reported comparable metabolic abnormalities, with HbA1c values of 7.19 ± 2.29% versus 6.51 ± 1.80%, triglyceride levels of 213.51 ± 168.10 versus 153.60 ± 79.25 mg/dL, and ALT levels of 42.90 ± 27.90 versus 27.39 ± 18.69 U/L in participants with and without MASLD, respectively (all p=0.001). These observations support the association of hepatic steatosis with impaired glucose metabolism, dyslipidaemia, and hepatocellular biochemical abnormalities.[10] Poor glycaemic control emerged as an important metabolic factor associated with MASLD in the present study. An HbA1c level ≥8% was observed in 37 patients (59.68%) with MASLD compared with only 7 patients (23.33%) without MASLD (p=0.001). Additionally, mean fasting blood glucose was higher in patients with MASLD (207.70 ± 26.67 mg/dL) than in those without MASLD (199.22 ± 24.40 mg/dL), although this difference was not statistically significant (p=0.135). Colosimo et al. (2023), in a retrospective longitudinal analysis of 637 patients with T2DM, demonstrated that improvements in glycaemic control were associated with favourable changes in hepatic steatosis measured through the Fatty Liver Index (FLI). They reported a significant association between changes in HbA1c and FLI after adjustment for BMI, age, sex, and glucose-lowering medications (R=0.467; p=0.031). Their findings complement the present observations by providing longitudinal evidence that better glycaemic control is associated with improvement in surrogate measures of hepatic fat accumulation.[11] The assessment of hepatic fibrosis risk in the present study revealed that the mean FIB-4 index was comparable between patients with and without MASLD (1.12 ± 0.46 versus 1.09 ± 0.61; p=0.810). Furthermore, 16 patients (25.81%) with MASLD and 7 patients (23.33%) without MASLD had FIB-4 values above the age-adjusted low-risk threshold, with no statistically significant difference (p=0.797). Datta et al. (2025), in the multicentric MISHTI study involving 472 patients, including 236 patients with T2DM, evaluated FIB-4 against transient elastography for identifying significant hepatic fibrosis. They reported that FIB-4 at a threshold of ≥1.3 demonstrated sensitivity of 85.30% but specificity of only 13.70% in the diabetic group, with a Cohen's kappa of −0.01, indicating poor agreement with elastography. Their study also identified BMI, hypertension, and HbA1c as significant predictors of hepatic fibrosis, with odds ratios of 1.076, 1.824, and 1.279, respectively. Although the present study assessed fibrosis risk rather than confirmed fibrosis, these findings suggest that FIB-4 alone may be insufficient for comprehensive fibrosis assessment among patients with T2DM.[12] The present study also evaluated the relationship between MASLD and diabetes-related microvascular complications. Diabetic nephropathy was observed in 19 patients (30.65%) with MASLD compared with 12 patients (40.00%) without MASLD (p=0.374), whereas diabetic retinopathy was present in 30.65% and 23.33% of patients, respectively (p=0.465). Neither complication demonstrated a statistically significant association with MASLD. In contrast, Deravi et al. (2023), in a prospective cohort study involving 3,123 patients with T2DM, including 1,215 patients with NAFLD and 1,908 without NAFLD, evaluated the incidence of microvascular complications over a median follow-up of five years. Their study demonstrated that NAFLD was significantly associated with incident diabetic nephropathy (OR=1.333; 95% CI: 1.007–1.764) and diabetic neuropathy (OR=1.338; 95% CI: 1.091–1.640). The differences between these findings and the present results may be related to variations in sample size, patient characteristics, and study design, particularly the assessment of prevalent complications in the present cross-sectional study compared with incident complications in the longitudinal study.[13] Multivariable binary logistic regression analysis in the present study identified elevated BMI and poor glycaemic control as statistically significant independent predictors of MASLD. Patients with BMI ≥25 kg/m² had approximately threefold higher adjusted odds of MASLD (aOR=2.94; 95% CI: 1.01–8.55; p=0.048), while patients with HbA1c ≥8% had substantially increased adjusted odds (aOR=4.78; 95% CI: 1.67–13.68; p=0.004). Hypertriglyceridaemia showed increased but statistically non-significant adjusted odds (aOR=2.39; p=0.109), whereas hypertension was not independently associated with MASLD (aOR=1.04; p=0.937). Comparable findings were reported by Afolabi et al. (2018), who investigated 80 Nigerian patients with T2DM and observed an ultrasound-diagnosed NAFLD prevalence of 68.80%. Their regression analysis identified overweight (OR=6.626; p=0.012), obesity (OR=11.508; p=0.009), and poor glycaemic control (OR=3.473; p=0.015) as significant predictors of NAFLD. Although the magnitude of the odds ratios differed, both studies demonstrated that increased body weight and inadequate glycaemic control were important independent factors associated with hepatic steatosis among patients with T2DM.[14]

CONCLUSION

The present study demonstrated a high prevalence of Metabolic Dysfunction-Associated Steatotic Liver

Disease (MASLD) among patients with Type 2 Diabetes Mellitus (67.39%). Increased body mass index, central obesity, poor glycaemic control, and hypertriglyceridaemia were significantly associated with MASLD. Multivariable analysis identified elevated BMI and HbA1c as significant independent predictors of MASLD. These findings highlight the importance of routine hepatic assessment, early identification of metabolic risk factors, and effective glycaemic and weight management to reduce the burden of MASLD among diabetic patients.

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