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PROSPECTIVE ASSESSMENT OF METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE AND CHANGES IN LIVER FUNCTION PARAMETERS AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS

Richa Sharma[1], Anugu Snigdha Reddy[2]

[1]Professor & Head, Department of General Medicine, Sims, Hapur, Uttar Pradesh, India.
[2]Junior Resident 1, Department of General Medicine, Sims, Hapur, Uttar Pradesh, India.

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Corresponding Author:
Dr. Anugu Snigdha Reddy,
Junior Resident , Department of General Medicine, Sims, Hapur, Uttar Pradesh, India.
Email: snigdhareddyanugu@gmail.com

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ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a common hepatic manifestation of metabolic dysfunction among patients with type 2 diabetes mellitus (T2DM). It is associated with insulin resistance, obesity, dyslipidaemia, and progressive liver injury. Early identification of MASLD and assessment of liver function abnormalities are essential for preventing hepatic complications. **Aim:** To assess the prevalence and severity of MASLD, associated metabolic risk factors, and changes in liver function parameters among patients with T2DM.

Materials and Methods: The present hospital-based, prospective observational analytical study included 115 patients with established T2DM at a tertiary care hospital. Demographic characteristics, anthropometric measurements, glycaemic parameters, lipid profiles, and liver function tests were evaluated. MASLD was diagnosed using abdominal ultrasonography, while fibrosis risk was assessed using the Fibrosis-4 (FIB-4) index. Changes in biochemical parameters were evaluated during follow-up. Statistical analysis was specified using IBM SPSS Statistics version 28.0, with $p<0.05$ considered statistically significant.

Results: MASLD was identified in 78 (67.83%) patients, with mild, moderate, and severe steatosis observed in 29.57%, 25.22%, and 13.04% of the total population, respectively. MASLD patients demonstrated significantly higher BMI (28.39 ± 2.97 vs. 25.47 ± 2.77 kg/m²), HbA1c (8.54 ± 0.98% vs. 7.83 ± 1.07%), and ALT levels (51.19 ± 11.17 vs. 26.84 ± 9.74 U/L). High FIB-4 risk was observed in 5.22% of patients. Follow-up assessment demonstrated significant reductions in ALT, AST, GGT, HbA1c, and BMI ($p<0.001$). Obesity, poor glycaemic control, hypertension, and dyslipidaemia were independently associated with MASLD.

Conclusion: MASLD was highly prevalent among patients with T2DM and was significantly associated with metabolic abnormalities and altered liver function parameters. Early screening, fibrosis risk assessment, and comprehensive metabolic management may improve clinical monitoring and outcomes.

Keywords: Metabolic Dysfunction-Associated Steatotic Liver Disease; Type 2 Diabetes Mellitus; Liver Function Tests; Hepatic Steatosis; Fibrosis-4 Index.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is an increasingly important manifestation of chronic liver disease and is closely linked to disturbances in glucose and lipid metabolism. The terminology emphasizes the contribution of cardiometabolic abnormalities rather than defining the disorder primarily through the exclusion of alcohol consumption. MASLD encompasses excess hepatic fat in individuals with at least one cardiometabolic risk factor, after appropriate consideration of other causes of steatosis. Its clinical spectrum extends from uncomplicated steatosis to metabolic dysfunction-associated steatohepatitis (MASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma. Recognition of this spectrum is essential because patients may remain asymptomatic while hepatic injury evolves.[1] Type 2 diabetes mellitus (T2DM) and MASLD share several pathogenic mechanisms, including insulin resistance, visceral adiposity, chronic low-grade inflammation, and altered lipid handling. In insulin-resistant states, increased release of free fatty acids from adipose tissue and enhanced hepatic lipid synthesis favour triglyceride accumulation within hepatocytes. Hyperglycaemia, oxidative stress, and lipotoxicity may subsequently promote hepatocellular injury and fibrogenesis. Conversely, hepatic steatosis may aggravate systemic insulin resistance, complicating diabetes management. These disturbances may be amplified by sedentary behaviour and dietary excess. This reciprocal relationship makes liver health an important aspect of comprehensive diabetic care alongside established cardiovascular, renal, neurological, and ophthalmic assessments.[2] The burden of MASLD is particularly relevant in tertiary care settings, where patients with diabetes frequently present with obesity, hypertension, dyslipidaemia, or other metabolic disorders. Clinical manifestations may be nonspecific, ranging from fatigue and vague abdominal discomfort to an entirely silent course. A systematic approach is therefore needed to identify hepatic steatosis in patients undergoing routine diabetes evaluation. Anthropometric measurements, dietary and lifestyle histories, blood pressure, glycaemic indices, and lipid profiles provide useful clinical context. Abdominal ultrasonography remains an accessible tool for detecting steatosis, although its sensitivity is limited when hepatic fat accumulation is mild and it cannot reliably identify steatohepatitis.[3] Determining the severity of liver involvement requires attention not only to hepatic fat but also to the possibility of fibrosis. Progressive fibrosis is central to the long-term clinical consequences of steatotic liver disease, and substantial liver injury may develop before obvious symptoms occur. Consequently, classification based on ultrasonographic steatosis alone provides an incomplete evaluation. Serum-based indices and elastography can support additional risk stratification, helping identify individuals who require closer assessment or specialist referral. Such evaluation is especially relevant in people with T2DM because coexisting metabolic abnormalities may increase vulnerability to progressive liver disease. A normal physical examination cannot be relied upon to exclude fibrosis.[4] Routine liver function parameters are readily available but require careful interpretation. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) reflect hepatocellular injury, while gamma-glutamyl transferase (GGT) and alkaline phosphatase (ALP) provide additional information about the hepatic biochemical profile. Bilirubin and albumin contribute to assessment of hepatic function, although mild alterations are not specific for MASLD. Normal aminotransferase concentrations do not exclude clinically significant fibrosis. The Fibrosis-4 (FIB-4) index, calculated from age, transaminases, and platelet count, offers an inexpensive initial assessment, with transient elastography providing further evaluation where indicated. Interpretation should account for patient age, concurrent illness, medication exposure, and reference ranges.[5] Prospective monitoring offers an opportunity to describe how hepatic biochemical parameters change alongside glycaemic control, weight, waist circumference, and lipid concentrations. Changes in prescribed glucose-lowering medicines, lifestyle practices, and associated metabolic conditions may accompany fluctuations in liver enzymes, but biochemical improvement should not automatically be equated with histological recovery. Documenting serial measurements is valuable for understanding temporal associations and for identifying patients requiring further evaluation. Patterns of change are more informative when baseline abnormalities are documented consistently. It also supports a more integrated approach to diabetes management in which hepatic and metabolic outcomes are considered together rather than as independent problems.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, prospective observational analytical study to assess the prevalence and severity of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and evaluate changes in liver function parameters among patients diagnosed with Type 2 Diabetes Mellitus (T2DM). The study also aimed to determine associations between hepatic steatosis, glycaemic control, anthropometric measurements, lipid abnormalities, metabolic risk factors, and liver fibrosis risk. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients with T2DM attending outpatient clinics or admitted to medical wards were screened for eligibility. A total of 115 patients with established T2DM were included using a consecutive sampling technique. Eligible patients were enrolled

after evaluation of clinical history, biochemical investigations, and ultrasonographic findings. Participants were categorized into MASLD-positive and MASLD-negative groups according to established diagnostic criteria.

Inclusion Criteria

Patients aged 18 years and above with an established diagnosis of T2DM, irrespective of sex, were eligible. Patients receiving oral antidiabetic drugs, insulin therapy, or combination treatment were considered for inclusion. Participants willing to undergo clinical assessment, biochemical investigations, abdominal ultrasonography, and scheduled follow-up evaluations were included.

Exclusion Criteria

Patients with chronic hepatitis B or C infection, autoimmune hepatitis, Wilson disease, haemochromatosis, drug-induced liver injury, or other established causes of hepatic steatosis were excluded. Alcohol consumption of 140 g/week or more in women or 210 g/week or more in men was an exclusion criterion to avoid including MetALD cases. Patients with decompensated liver disease, acute hepatitis, severe acute illness, malignancy affecting the liver, or pregnancy were also excluded.

Methodology

Detailed clinical histories were recorded using a structured proforma. Demographic and clinical parameters included age, sex, duration of diabetes, family history, smoking status, alcohol consumption, dietary habits, physical activity, hypertension, dyslipidaemia, and current medications. Anthropometric measurements included height, weight, body mass index (BMI), waist circumference, waist-to-hip ratio, and blood pressure. Obesity, central adiposity, metabolic abnormalities, and associated comorbidities were evaluated as potential factors associated with MASLD.

Biochemical Investigations

Venous blood samples were collected under standardized conditions. Glycaemic parameters included fasting blood glucose, postprandial blood glucose, and glycated haemoglobin (HbA1c). Liver biochemical parameters included alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total and direct bilirubin, serum albumin, total protein, and albumin-to-globulin ratio. Prothrombin time and international normalized ratio were assessed where indicated. Lipid profile measurements included total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol. Serum creatinine, estimated glomerular filtration rate, and urine albumin-to-creatinine ratio were also documented.

Assessment and Diagnosis of MASLD

All participants underwent abdominal ultrasonography performed by an experienced radiologist. Hepatic steatosis was assessed using increased liver echogenicity, hepatorenal contrast, posterior beam attenuation, and reduced visualization of intrahepatic vessels. Steatosis was classified as mild, moderate, or severe according to standard sonographic criteria. MASLD was diagnosed when hepatic steatosis was identified in a patient with T2DM after exclusion of competing causes. Participants were classified into MASLD-positive and MASLD-negative groups for comparative analysis.

Assessment of Liver Fibrosis Risk

Liver fibrosis risk was assessed using the Fibrosis-4 (FIB-4) index, calculated from age, AST, ALT, and platelet count. Participants were categorized into low-, intermediate-, and high-risk groups using established age-appropriate thresholds. Transient elastography findings were documented when available to provide additional information regarding liver stiffness and fibrosis severity. Associations between fibrosis risk, diabetes-related parameters, metabolic abnormalities, and hepatic biochemical markers were evaluated.

Follow-up Assessment and Outcome Measures

Participants underwent repeat clinical and biochemical evaluations during scheduled follow-up visits. Changes in ALT, AST, ALP, GGT, bilirubin, albumin, HbA1c, fasting glucose, lipid profile, BMI, and waist circumference were documented. Changes in antidiabetic medications, lipid-lowering treatment, and lifestyle measures were recorded. The primary outcomes were MASLD prevalence and changes in liver biochemical parameters. Secondary outcomes included associations of MASLD severity and fibrosis risk with glycaemic control, obesity, dyslipidaemia, and other metabolic characteristics.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics 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. The independent-samples t-test or Mann–Whitney U test was used for between-group comparisons. Chi-square or Fisher's exact test was applied to categorical variables. Paired t-tests or Wilcoxon signed-rank tests assessed changes in biochemical parameters. Pearson's or Spearman's correlation coefficients evaluated associations between metabolic and hepatic parameters. Multivariable logistic regression was used to identify independent factors associated with MASLD. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1. Comparison of Baseline Demographic and Clinical Characteristics Between MASLD and Non-MASLD Patients (N=115)

Table 1 demonstrates that patients with MASLD had a significantly higher mean age (55.21 ± 9.74 years) compared with non-MASLD patients (50.86 ± 8.91 years; p=0.020). Male patients constituted 57.69% of

the MASLD group and 43.24% of the non-MASLD group, although this difference was statistically insignificant (p=0.147). The mean duration of diabetes was significantly longer among MASLD patients (10.15 ± 3.87 years) than non-MASLD patients (8.22 ± 4.87 years; p=0.038). Hypertension (65.38% vs. 35.14%; p=0.002) and dyslipidaemia (61.54% vs. 29.73%; p=0.001) were significantly more prevalent among patients with MASLD. Similarly, BMI ≥25 kg/m² was observed in 88.46% of MASLD patients compared with 54.05% of non-MASLD patients (p<0.001). Poor glycaemic control, represented by HbA1c ≥8%, was significantly more common in the MASLD group (74.36% vs. 45.95%; p=0.003), suggesting an association between metabolic abnormalities and hepatic steatosis.

Table 2. Prevalence, Ultrasonographic Severity of MASLD, and Distribution of FIB-4 Fibrosis Risk Among Study Participants (N=115)

Table 2 illustrates that MASLD was identified in 78 (67.83%) of the 115 patients with type 2 diabetes mellitus, while 37 (32.17%) showed no ultrasonographic evidence of hepatic steatosis. Mild hepatic steatosis was observed in 34 (29.57%) patients, moderate steatosis in 29 (25.22%), and severe steatosis in 15 (13.04%). Regarding fibrosis risk, 55 (47.83%) participants were classified into the low-risk category, 54 (46.96%) into the intermediate-risk category, and 6 (5.22%) into the high-risk category according to FIB-4 assessment. Among patients with severe steatosis, 60.00% demonstrated intermediate fibrosis risk and 20.00% demonstrated high fibrosis risk, compared with 35.14% and 5.41%, respectively, among patients without steatosis. The proportion of patients classified as low fibrosis risk decreased from 59.46% among those without steatosis to 20.00% among those with severe steatosis.

Table 3. Comparison of Glycaemic, Anthropometric, and Metabolic Parameters Between MASLD and Non-MASLD Patients (N=115)

Table 3 shows significant differences in anthropometric, glycaemic, and metabolic parameters between MASLD and non-MASLD patients. The mean BMI was significantly higher in the MASLD group (28.39 ± 2.97 kg/m²) compared with the non-MASLD group (25.47 ± 2.77 kg/m²; p<0.001), while mean waist circumference was also significantly increased (98.79 ± 9.17 cm vs. 89.85 ± 8.32 cm; p<0.001). Patients with MASLD demonstrated significantly higher HbA1c (8.54 ± 0.98% vs. 7.83 ± 1.07%; p=0.001) and fasting blood glucose levels (178.46 ± 29.36 vs. 152.12 ± 28.27 mg/dL; p<0.001), indicating comparatively poorer glycaemic control. Mean triglyceride levels (198.07 ± 43.54 vs. 150.55 ± 39.33 mg/dL; p<0.001) and LDL cholesterol levels (120.28 ± 23.89 vs. 109.46 ± 23.86 mg/dL; p=0.026) were significantly elevated among MASLD patients. Conversely, HDL cholesterol was significantly lower in the MASLD group (42.19 ± 5.76 vs. 47.94 ± 6.46 mg/dL; p<0.001). The mean eGFR was also significantly lower among MASLD patients (83.24 ± 14.36 vs. 90.76 ± 16.08 mL/min/1.73 m²; p=0.018), suggesting an association between hepatic steatosis and adverse metabolic and renal function profiles.

Table 4. Comparison of Baseline Liver Function Parameters Between MASLD and Non-MASLD Patients (N=115)

Table 4 demonstrates significant differences in baseline liver function parameters between patients with and without MASLD. Mean ALT levels were markedly elevated in MASLD patients (51.19 ± 11.17 U/L) compared with non-MASLD patients (26.84 ± 9.74 U/L; p<0.001), while AST levels were also significantly higher (37.67 ± 9.48 vs. 26.50 ± 8.14 U/L; p<0.001). Similarly, ALP (112.28 ± 19.42 vs. 102.76 ± 19.42 U/L; p=0.017) and GGT (54.35 ± 16.11 vs. 34.92 ± 15.87 U/L; p<0.001) were significantly increased among MASLD patients. Total bilirubin levels were comparatively higher in the MASLD group (0.89 ± 0.19 vs. 0.74 ± 0.18 mg/dL; p<0.001). In contrast, serum albumin concentrations were significantly lower among MASLD patients (4.15 ± 0.23 vs. 4.32 ± 0.21 g/dL; p<0.001). Platelet counts were also significantly reduced in the MASLD group (203.96 ± 38.77 vs. 230.10 ± 36.83 ×10³/µL; p=0.001), indicating that MASLD was associated with an altered hepatic biochemical profile, although these findings alone cannot establish advanced liver disease.

Table 5. Changes in Liver Function and Metabolic Parameters During Follow-up Among Patients with MASLD (n=78)

Table 5 illustrates significant improvements in liver function and metabolic parameters during follow-up among the 78 patients diagnosed with MASLD. Mean ALT levels decreased from 51.19 ± 11.17 U/L at baseline to 43.75 ± 12.97 U/L during follow-up, representing a mean reduction of 7.44 U/L (p<0.001), while AST decreased from 37.67 ± 9.48 to 32.73 ± 10.80 U/L (p<0.001). Similarly, GGT decreased by 7.53 U/L and ALP by 5.90 U/L, with both changes statistically significant (p<0.001). Total bilirubin showed a small but statistically significant reduction from 0.89 ± 0.19 to 0.86 ± 0.21 mg/dL (p=0.001), whereas serum albumin increased from 4.15 ± 0.23 to 4.21 ± 0.24 g/dL (p<0.001). Improvements in metabolic control were also observed, with HbA1c decreasing from 8.54 ± 0.98% to 8.05 ± 1.05% and BMI declining from 28.39 ± 2.97 to 27.78 ± 3.09 kg/m² (both p<0.001).

Table 6. Multivariable Logistic Regression Analysis of Factors Associated with MASLD Among Patients with Type 2 Diabetes Mellitus (N=115)

Table 6 presents the multivariable logistic regression analysis identifying factors independently associated with MASLD among patients with type 2 diabetes mellitus. Increasing BMI was significantly associated with MASLD, with each 1 kg/m² increase corresponding to 1.50 times higher adjusted odds of MASLD (aOR=1.50; 95% CI: 1.22–1.84; p<0.001).

Poor glycaemic control was another significant factor, with each 1% increase in HbA1c associated with 2.36 times higher adjusted odds of MASLD (95% CI: 1.30–4.29; p=0.005). Hypertension demonstrated a significant independent association, with affected patients having 4.90 times higher adjusted odds of MASLD compared with those without hypertension (95% CI: 1.58–15.15; p=0.006). Dyslipidaemia showed the strongest adjusted association among the variables included in the model (aOR=6.37; 95% CI: 2.07–19.62; p=0.001). Although longer diabetes duration was associated with slightly increased odds of MASLD (aOR=1.09; 95% CI: 0.96–1.25), the association was not statistically significant (p=0.183).

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between MASLD and Non-MASLD Patients (N=115)

Parameters	MASLD (n=78)	Non-MASLD (n=37)	p-value
Age (years), mean ± SD	55.21 ± 9.74	50.86 ± 8.91	0.020
Male sex	45 (57.69%)	16 (43.24%)	0.147
Duration of diabetes (years), mean ± SD	10.15 ± 3.87	8.22 ± 4.87	0.038
Hypertension	51 (65.38%)	13 (35.14%)	0.002
Dyslipidaemia	48 (61.54%)	11 (29.73%)	0.001
BMI ≥25 kg/m²	69 (88.46%)	20 (54.05%)	<0.001
HbA1c ≥8%	58 (74.36%)	17 (45.95%)	0.003

Table 2: Prevalence, Ultrasonographic Severity of MASLD, and Distribution of FIB-4 Fibrosis Risk Among Study Participants (N=115)

Ultrasonographic findings	Total patients n (%)	Low FIB-4 risk n (%)	Intermediate FIB-4 risk n (%)	High FIB-4 risk n (%)
No hepatic steatosis	37 (32.17%)	22 (59.46%)	13 (35.14%)	2 (5.41%)
Mild steatosis	34 (29.57%)	17 (50.00%)	17 (50.00%)	0 (0.00%)
Moderate steatosis	29 (25.22%)	13 (44.83%)	15 (51.72%)	1 (3.45%)
Severe steatosis	15 (13.04%)	3 (20.00%)	9 (60.00%)	3 (20.00%)
Total	115 (100.00%)	55 (47.83%)	54 (46.96%)	6 (5.22%)

Table 3: Comparison of Glycaemic, Anthropometric, and Metabolic Parameters Between MASLD and Non-MASLD Patients (N=115)

Parameters	MASLD (n=78), mean ± SD	Non-MASLD (n=37), mean ± SD	p-value
BMI (kg/m²)	28.39 ± 2.97	25.47 ± 2.77	<0.001
Waist circumference (cm)	98.79 ± 9.17	89.85 ± 8.32	<0.001
HbA1c (%)	8.54 ± 0.98	7.83 ± 1.07	0.001
Fasting blood glucose (mg/dL)	178.46 ± 29.36	152.12 ± 28.27	<0.001
Triglycerides (mg/dL)	198.07 ± 43.54	150.55 ± 39.33	<0.001
LDL cholesterol (mg/dL)	120.28 ± 23.89	109.46 ± 23.86	0.026
HDL cholesterol (mg/dL)	42.19 ± 5.76	47.94 ± 6.46	<0.001
eGFR (mL/min/1.73 m²)	83.24 ± 14.36	90.76 ± 16.08	0.018

Table 4: Comparison of Baseline Liver Function Parameters Between MASLD and Non-MASLD Patients (N=115)

Liver function parameters	MASLD (n=78), mean ± SD	Non-MASLD (n=37), mean ± SD	p-value
ALT (U/L)	51.19 ± 11.17	26.84 ± 9.74	<0.001
AST (U/L)	37.67 ± 9.48	26.50 ± 8.14	<0.001
ALP (U/L)	112.28 ± 19.42	102.76 ± 19.42	0.017
GGT (U/L)	54.35 ± 16.11	34.92 ± 15.87	<0.001
Total bilirubin (mg/dL)	0.89 ± 0.19	0.74 ± 0.18	<0.001
Serum albumin (g/dL)	4.15 ± 0.23	4.32 ± 0.21	<0.001
Platelet count (×10³/µL)	203.96 ± 38.77	230.10 ± 36.83	0.001

Table 5: Changes in Liver Function and Metabolic Parameters During Follow-up Among Patients with MASLD (n=78)

Parameters	Baseline, mean ± SD	Follow-up, mean ± SD	Mean change	p-value
ALT (U/L)	51.19 ± 11.17	43.75 ± 12.97	−7.44	<0.001
AST (U/L)	37.67 ± 9.48	32.73 ± 10.80	−4.93	<0.001
GGT (U/L)	54.35 ± 16.11	46.82 ± 17.65	−7.53	<0.001
ALP (U/L)	112.28 ± 19.42	106.38 ± 22.34	−5.90	<0.001
Total bilirubin (mg/dL)	0.89 ± 0.19	0.86 ± 0.21	−0.03	0.001
Serum albumin (g/dL)	4.15 ± 0.23	4.21 ± 0.24	+0.06	<0.001
HbA1c (%)	8.54 ± 0.98	8.05 ± 1.05	−0.49	<0.001
BMI (kg/m²)	28.39 ± 2.97	27.78 ± 3.09	−0.61	<0.001

Table 6: Multivariable Logistic Regression Analysis of Factors Associated with MASLD Among Patients with Type 2 Diabetes Mellitus (N=115)

Variables	Adjusted odds ratio (aOR)	95% confidence interval	p-value
BMI (per 1 kg/m² increase)	1.50	1.22–1.84	<0.001
HbA1c (per 1% increase)	2.36	1.30–4.29	0.005
Duration of diabetes (per year)	1.09	0.96–1.25	0.183
Hypertension (present vs absent)	4.90	1.58–15.15	0.006
Dyslipidaemia (present vs absent)	6.37	2.07–19.62	0.001

DISCUSSION

The present study demonstrated that patients with metabolic dysfunction-associated steatotic liver disease (MASLD) had a significantly higher mean age (55.21 ± 9.74 years) compared with non-MASLD patients (50.86 ± 8.91 years; p=0.020). The mean duration of diabetes was also significantly longer among MASLD patients (10.15 ± 3.87 vs. 8.22 ± 4.87 years; p=0.038). Furthermore, hypertension (65.38% vs. 35.14%) and dyslipidaemia (61.54% vs. 29.73%) were significantly more prevalent in the MASLD group. Similar findings were reported by Targher et al. (2007), who evaluated 2,839 patients with type 2 diabetes mellitus and observed an overall nonalcoholic fatty liver disease (NAFLD) prevalence of 69.50%. The prevalence increased significantly with advancing age, from 65.40% among patients aged 40–59 years to 74.60% among those aged 60 years or older (p<0.001). The age-adjusted prevalence was 71.10% in men and 68.00% in women. These observations support the association between increasing age and hepatic steatosis among diabetic patients, although the present study did not demonstrate a statistically significant sex difference (p=0.147).[7] In the present study, MASLD was identified in 78 of 115 patients, representing an overall prevalence of 67.83%, whereas 32.17% demonstrated no ultrasonographic evidence of hepatic steatosis. Among the entire study population, mild, moderate, and severe steatosis were observed in 29.57%, 25.22%, and 13.04% of patients, respectively. These findings were comparable to those reported by Lomonaco et al. (2021), who investigated 561 patients with type 2 diabetes mellitus and observed hepatic steatosis in 70.00% of participants. Their study also demonstrated liver fibrosis, defined by liver stiffness measurement of at least 7.0 kPa, in 21.00% of patients, while 9.00% had liver stiffness values suggestive of severe fibrosis or cirrhosis. In comparison, 5.22% of patients in the present study were classified into the high FIB-4 risk category.[8] The present study further demonstrated that increasing ultrasonographic severity of hepatic steatosis was associated with a greater proportion of patients belonging to higher FIB-4 risk categories. Among patients with severe steatosis, 60.00% were classified as intermediate risk and 20.00% as high risk, whereas the corresponding proportions among patients without steatosis were 35.14% and 5.41%. Overall, intermediate and high FIB-4 risk categories accounted for 46.96% and 5.22% of the study population, respectively. Kwok et al. (2016) conducted a prospective cohort study involving 1,918 diabetic patients and reported increased controlled attenuation parameter values suggestive of hepatic steatosis in 72.80% of patients (95% CI: 70.70–74.80%). Increased liver stiffness measurements were documented in 17.70% (95% CI: 16.00–19.50%). Their analysis also identified higher BMI, ALT, and other metabolic abnormalities as factors associated with increased liver stiffness. These findings reinforce the importance of fibrosis risk assessment, although FIB-4 categories and elastography-derived liver stiffness measurements are not directly interchangeable.[9] The present study revealed significant differences in anthropometric and glycaemic parameters between MASLD and non-MASLD patients. Mean BMI was significantly higher among MASLD patients (28.39 ± 2.97 vs. 25.47 ± 2.77 kg/m²; p<0.001), while waist circumference was also increased (98.79 ± 9.17 vs. 89.85 ± 8.32 cm; p<0.001). Similarly, HbA1c (8.54 ± 0.98% vs. 7.83 ± 1.07%; p=0.001) and fasting blood glucose (178.46 ± 29.36 vs. 152.12 ± 28.27 mg/dL; p<0.001) were significantly higher in patients with MASLD. Portillo-Sanchez et al. (2015) investigated 103 patients with T2DM and normal plasma aminotransferase concentrations and identified NAFLD in 50.00% using magnetic resonance spectroscopy. Interestingly, NAFLD was also present in 36.00% of non-obese patients. Liver triglyceride content correlated positively with BMI (r=0.29; p=0.003), while higher HbA1c was associated with a greater prevalence of NAFLD and increased hepatic triglyceride accumulation (p=0.010). These findings were consistent with the observed associations between adiposity, poor glycaemic control, and hepatic steatosis in the present study.[10]

Abnormal lipid metabolism constituted another important observation in the present study. Patients with MASLD demonstrated significantly elevated triglyceride concentrations (198.07 ± 43.54 vs. 150.55 ± 39.33 mg/dL; p<0.001) and LDL cholesterol levels (120.28 ± 23.89 vs. 109.46 ± 23.86 mg/dL; p=0.026), whereas HDL cholesterol concentrations were significantly lower (42.19 ± 5.76 vs. 47.94 ± 6.46 mg/dL; p<0.001). Comparable findings were reported by Leite et al. (2009) in a study involving 180 patients with T2DM, in which the prevalence of ultrasonographically detected NAFLD was 69.40%. Patients with NAFLD demonstrated greater obesity, increased waist circumference, and higher triglyceride and ALT concentrations. Their multivariable analysis

identified elevated triglycerides above 2.82 mmol/L (OR approximately 3.7–4.1), obesity (OR=7.10), and increased waist circumference (OR=4.80) as important factors associated with hepatic steatosis. However, unlike the present study, they did not demonstrate a significant association between glycaemic control and steatosis. This difference may reflect variations in patient characteristics, metabolic status, and diagnostic methods.[11] In addition to metabolic abnormalities, the present study identified comparatively lower renal function parameters among patients with MASLD. Mean estimated glomerular filtration rate was significantly lower in the MASLD group (83.24 ± 14.36 mL/min/1.73 m²) compared with the non-MASLD group (90.76 ± 16.08 mL/min/1.73 m²; p=0.018). These observations were supported by the findings of Targher et al. (2008), who conducted a prospective study involving 1,760 patients with type 2 diabetes and normal or near-normal baseline renal function. During a mean follow-up of 6.5 years, 547 participants developed chronic kidney disease. The presence of NAFLD was associated with an increased risk of incident chronic kidney disease (HR=1.69; p<0.001), and this association remained significant after adjustment for conventional risk factors (adjusted HR=1.49; 95% CI: 1.10–2.20; p<0.01). Although the present study demonstrated a significant difference in eGFR between groups, its findings alone cannot establish an increased incidence of chronic kidney disease. Nevertheless, both studies suggest that hepatic steatosis and renal dysfunction may coexist in patients with T2DM.[12] The evaluation of baseline liver function parameters revealed significantly elevated hepatic enzymes among patients with MASLD. Mean ALT was considerably higher in MASLD patients (51.19 ± 11.17 U/L) compared with non-MASLD patients (26.84 ± 9.74 U/L; p<0.001), while AST (37.67 ± 9.48 vs. 26.50 ± 8.14 U/L; p<0.001) and GGT (54.35 ± 16.11 vs. 34.92 ± 15.87 U/L; p<0.001) were also significantly increased. Similar observations were reported by Mandal et al. (2018) in a study involving 210 patients with type 2 diabetes, of whom 117 (55.70%) demonstrated ultrasonographic fatty liver. Their study reported significantly higher ALT levels among diabetic patients with fatty liver compared with those without fatty liver (51.50 ± 27.00 vs. 24.70 ± 7.00 U/L; p<0.001). However, AST concentrations were not significantly different between the groups (26.10 ± 13.00 vs. 22.00 ± 15.00 U/L; p=0.240). This contrasts with the significant AST elevation observed in the present study. These findings suggest that ALT may be particularly useful as a biochemical indicator of hepatic injury, although normal liver enzyme concentrations do not exclude MASLD or advanced fibrosis.[13] The prospective follow-up findings of the present study demonstrated significant improvements in liver function and metabolic parameters among the 78 patients with MASLD. Mean ALT decreased from 51.19 ± 11.17 to 43.75 ± 12.97 U/L, while AST decreased from 37.67 ± 9.48 to 32.73 ± 10.80 U/L (both p<0.001). Mean HbA1c decreased by 0.49 percentage points, and BMI declined by 0.61 kg/m², with both changes statistically significant (p<0.001). Lazo et al. (2010) evaluated 96 adults with T2DM in a randomized lifestyle intervention study, including 46 participants receiving intensive lifestyle intervention and 50 receiving diabetes support and education. After 12 months, the intensive intervention group demonstrated a greater reduction in hepatic steatosis (50.80% vs. 22.80%; p=0.040) and HbA1c (0.70 vs. 0.20 percentage points; p=0.040). BMI decreased by 2.60 kg/m² in the intervention group compared with a slight increase of 0.03 kg/m² in controls. However, unlike the present study, no significant changes in AST or ALT were observed. These findings indicate that improvements in metabolic parameters can accompany reductions in hepatic fat, but the observational design of the present study does not permit attribution of biochemical improvements to a specific intervention.[14] Multivariable logistic regression analysis in the present study demonstrated that elevated BMI, poor glycaemic control, hypertension, and dyslipidaemia were independently associated with MASLD. Each 1 kg/m² increase in BMI was associated with higher adjusted odds of MASLD (aOR=1.50; 95% CI: 1.22–1.84; p<0.001), while each 1% increase in HbA1c demonstrated an aOR of 2.36 (p=0.005). Hypertension (aOR=4.90; p=0.006) and dyslipidaemia (aOR=6.37; p=0.001) also showed significant associations, whereas diabetes duration was not independently significant (p=0.183). Comparable findings were reported by Kuchay et al. (2021) in a prospective Indian study involving 601 patients with T2DM, which identified hepatic steatosis in 84.20% and clinically relevant liver fibrosis in 28.20% of assessed patients. Higher BMI (OR=1.168; p=0.022), elevated ALT (OR=1.031; p=0.001), and lower HDL cholesterol (OR=0.969; p=0.002) were independently associated with hepatic steatosis. Furthermore, hypertension was independently associated with clinically relevant fibrosis (OR=1.729; p=0.018). Although the outcomes and statistical models differed, both studies highlighted the importance of obesity and metabolic abnormalities in identifying diabetic patients at increased risk of liver disease.[15]

CONCLUSION

The present study demonstrated a high prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD) among patients with type 2 diabetes mellitus (67.83%). MASLD was significantly associated with obesity, poor glycaemic control, hypertension, dyslipidaemia, and elevated liver enzymes. Follow-up assessment showed significant improvements in liver function parameters, HbA1c, and BMI. Increasing severity of hepatic steatosis was associated with higher FIB-4 fibrosis risk categories, highlighting the importance of early screening and regular monitoring.

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