

Original Article

Prevalence, clinical profile and risk factors of metabolic dysfunction–associated steatotic liver disease (MASLD) in newly diagnosed type 2 diabetes mellitus

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Received: 3 March 2026 / Accepted: 25 June 2026

Abstract

There is a high incidence of association metabolic dysfunction–associated steatotic liver disease (MASLD) among patients with type 2 diabetes mellitus (T2DM) which if not detected and treated at the earliest can result into serious complications. We aim to study the prevalence, clinical profile and risk factors of MASLD in newly diagnosed type 2 diabetes patients. This was a cross-sectional study involving 272 individuals with new onset T2DM diagnosed in previous 6 months who met the inclusion criteria. Patients were stratified according to the presence/absence of MASLD. MASLD presence and severity were defined by ultrasonographic detection of steatosis in the absence of other liver diseases. The anthropometric, clinical and laboratory data were collected retrospectively. Parameters as predictors of MASLD were identified using linear regression models. The prevalence of MASLD by Ultrasound examination was 64%, with higher prevalence in females (71.4%) than males (57.5%). The patients with MASLD demonstrated significantly higher mean BMI, waist-hip ratio, AST, ALT, GGT, SUA, FPG, FINS and triglycerides but lower HDL. Pearson correlation showed significantly positive associations of MASLD severity with BMI ($p < 0.001$), WHR ($p < 0.001$), TG ($p = 0.013$), AST ($p = 0.009$), ALT ($p = 0.004$) and TG ($p = 0.012$). Linear regression analysis confirmed independent positive associations of MASLD with BMI ($p = 0.032$), WHR ($p = 0.025$), ALT ($p = 0.032$), HDL ($p = 0.019$) and TG ($p = 0.012$). The significant prevalence of MASLD in newly diagnosed T2DM patients in this study underscores the importance of incorporating routine screening in diabetic care for early detection and management of hepatic impairment, aiming to reduce the burden of liver-related complications in diabetic populations.

Keywords: metabolic dysfunction–associated steatotic liver disease (MASLD), type 2 diabetes mellitus (T2DM), and obesity

Introduction

Diabetes mellitus is a modern epidemic and MASLD is the most common liver disorder worldwide [1]. There is high incidence of association MASLD in patients with T2DM which if not detected and treated at the earliest can leads to further complications, such as

MASH, liver cirrhosis and hepatocellular carcinoma, being increasingly recognized [2–5].

The prevalence of MASLD has escalated in recent years due to shifts in dietary patterns and socioeconomic status, making it the most prevalent chronic liver disease and a significant public health concern globally [6, 7]. The global pooled prevalence of MASLD



among adults with T2DM is approximately 65.33% and is enormous considering the huge number of patients with T2DM worldwide. Regionally, prevalence is highest in Eastern Europe (80.62%), followed by the Middle East (71.24%), and lowest in Africa (53.10%). Among patients with liver biopsy data, the pooled prevalence of MASH/NASH, significant fibrosis, and advanced fibrosis is 66.44%, 40.78%, and 15.49%, respectively [8]. The reported prevalence of MASLD among individuals with T2DM varies widely in India, ranging from 45.0% to 72.0% across different regional studies [9, 10].

Although the precise pathophysiological mechanisms underlying MASLD is not fully elucidated, current evidence suggests a multifactorial process involving steatosis, metabolic dysfunction, oxidative stress (OS), and immune-mediated inflammatory pathways [11, 12]. Insulin resistance along with hyperinsulinemia, a compensatory response to insulin resistance, plays a central role in the development and progression of MASLD and T2DM. The adipose tissue expansion under the influence of genetic factors, sedentary life style and high caloric intake triggers adipose tissue IR and inflammation [13, 14]. Combined with increased reactive oxygen species production and subsequent oxidative stress, this creates a pro-inflammatory hepatic environment that leads to liver injury and fibrosis [11, 15, 16]. Additionally, adipose IR contributes to systemic IR, impairing insulin action in peripheral tissues such as muscle, thereby hindering glucose uptake and regulation contributing to impaired glycemia and metabolic dysregulation [17].

MASLD, a hepatic manifestation of metabolic syndrome, is strongly associated and highly prevalent in patients with T2DM, obesity, and cardiovascular disease and is linked to adverse metabolic and cardiovascular outcomes, including increased mortality [18]. As MASLD is often asymptomatic, it frequently remains undiagnosed; therefore, early detection in newly diagnosed T2DM patients is essential for timely management and targeted interventions. Therefore, the aim of this study was to contribute new insights into MASLD prevalence and enhancing the understanding of underlying risk factors for the incidence and progression of MASLD in T2DM patients.

Material and methods

Study design and patients

This cross-sectional study enrolled 272 consecutive adults' patients diagnosed within the preceding six

months who presented to the outpatient department of SKIMS, a tertiary care endocrinology centre between June 2020 and July 2023.

Inclusion and exclusion criteria

Patients aged 18 years or above diagnosed with T2DM within the preceding six months were included in the study. Individuals with a Type 1 diabetes mellitus, history of alcoholism, pregnancy, known liver disease, including viral, drug and autoimmune hepatitis, kidney disease, endocrinopathies, genetic disorders, or the use of medications known to cause fatty liver were excluded from the study.

Laboratory, anthropometric and clinical data collection

The baseline information of patients, which includes age, sex, symptoms, duration of disease, clinical manifestations, laboratory values, and demographic details, were recorded as per protocol. Physical examination findings included general physical examination for signs of insulin resistance, anthropometric measures like weight, height, waist circumference, waist hip ratio (WHR), systolic and diastolic blood pressures and ankle reflex were also recorded. The BMI was calculated as weight (in kg) divided by height in m² and WHR was calculated as the ratio of the waist to the hip circumference (in cm).

Venous blood samples were obtained for the estimation of SUA, creatinine, triglycerides (TGs), cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), glycosylated hemoglobin (HbA1c), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and glutamyl transpeptidase (GGT). HOMA-IR was used as a measure of Insulin resistance ($\text{HOMA-IR} = \text{FINS (mIU/L)} \times \text{FPG (mmol/L)} / 22.5$). The serum lipids, Uric acid and transaminase were estimated by High end analyzer Beckman Coulter AU 680.

MASLD diagnosis was based on imaging evidence of hepatic steatosis plus presence of at least one cardiometabolic risk factor [19, 20]. The hepatic steatosis diagnosis was based on the characteristic echo patterns of B mode liver ultrasonography performed by a single experienced senior Radiologist, with a convex 3.5 to 5.0 MHz (P5 GE), that cannot be explained by other liver diseases [21]. Fatty liver grading by US was done based on the standard criteria accepted by American, Gastroenterological Association [22]. Grade I: Increased

hepatic echogenicity with visible periportal and diaphragmatic echogenicity. Grade II: Increased hepatic echogenicity with imperceptible periportal echogenicity, without obscuration of the diaphragm. Grade III: Increased hepatic echogenicity with imperceptible periportal echogenicity and obscuration of the diaphragm.

Statistical analysis

Data was expressed as mean±standard deviation (SD), or as median with interquartile range (IQR), or percentage, whichever was appropriate for the subject's characteristic description. Group differences were compared using the Pearson chi-square or Fisher's exact test for categorical variables, and the Student t test or the Mann-Whitney U test for continuous variables. To measure the strength of the linear relationship between normally distributed variables, the Pearson correlation coefficient was used. Additionally, multiple linear regression analyses using the stepwise method were performed to assess the independent variables affecting the dependent variable MASLD. A P-value of less than 0.05 was considered statistically significant for all statistical tests. All statistical analyses were performed with IBM SPSS Statistics 23.0 (IBM Corporation, NY) using Mac.

Results

A total of 272 individuals (146 males and 126 females) with new onset type 2 diabetes mellitus diagnosed within previous 6 months who met the inclusion criteria were analyzed to assess the prevalence and predictors of MASLD. There were 174 (64%) cases including 84 males and 90 females, which had some grade of steatosis and were categorised into the MASLD group (n=174) and non-MASLD group (n=98).

The prevalence of MASLD by ultrasonographic criteria was 64%, with higher prevalence in females (71.4%) than males (57.5%). Grade I fatty liver was observed in 58.3% males and 41.2% females and Grade II in 41.7% males and 58.8% females.

The comparison of the demographic, clinical, and biochemical laboratory parameters between the groups is shown in Table 1. The patients with MASLD demonstrated significantly elevated mean BMI (26.96±3.91 vs. 26.32±3.09; $p<0.001$), waist-hip ratio (0.99±0.056 vs. 0.96±0.052; $p<0.001$), AST (41.18±21.00 vs. 31.59±19.02; $p=0.007$), ALT (59.48±35.17 vs. 42.40±28.95; $p=0.004$), SUA (4.59±0.96 vs. 4.09±1.09; $p=0.009$), FPG (252.23±162.3 vs. 242.25±119.69; $p=0.002$), FINS (14.09±1.02 vs. 11.13±4.59; $p=0.001$), TG (220±105 vs. 192±105; $p=0.001$), and lower HDL-C (32±7.8 vs. 39±9.3;

Table 1: Comparative analysis of demographic, clinical, and biochemical variables of individuals with and without MASLD.

Variables	Non MASLD	MASLD	P-value
Age (years)	41.27±10.75	43.53±8.52	0.483
Duration of DM (years)	6.29±6.1	5.72±5.4	0.578
BMI (kg/m ²)	26.32±3.09	26.96±3.91	0.000*
WHR	0.96±0.052	0.99±0.056	0.000*
AST (U/L)	31.59±19.02	41.18±21.00	0.007*
ALT (U/L)	42.40±28.95	59.48±35.17	0.004*
Albumin (g/L)	4.33±0.39	4.42±0.35	0.207
ALP (U/L)	129±42.67	119±39.37	0.181
Uric acid (mg/dl)	4.09±1.09	4.59±0.96	0.009*
Fasting PG (mg/dl)	242.25±119.69	252.23±162.3	0.002*
PP PG (mg/dl)	289.45±89.2	284.64±63.7	0.231
HbA1C (%)	11.12±2.83	10.19±2.31	0.123
FINS (mU/l)	11.13±4.59	14.09±1.02	0.001*
HOMA-IR	7.0±4.84	7.83±6.16	0.321
Cholesterol (mg/dl)	183±54.01	195±43.21	0.152

Table 1: Continued.

Variables	Non MASLD	MASLD	P-value
Triglyceride (mg/dl)	192±105	220±105	0.001*
HDL-C (mg/dl)	39±9.3	32±7.8	0.023*
LDL-C (mg/dl)	131±34	141±30	0.113

Note: Data are presented as mean±standard deviation. BMI – body mass index; WHR – waist-to-hip ratio; ALT – alanine aminotransferase; AST – aspartate aminotransferase; SUA – serum uric acid; FPG – fasting plasma glucose; 2hPG, plasma glucose 2 h after a 75 g oral glucose load; HbA1c – glycated haemoglobin; FINS – fasting insulin; HOMA-IR – homeostasis model assessment of insulin resistance; LDL-C – low-density lipoprotein cholesterol; NAFLD, non-alcoholic fatty liver disease. * – $p < 0.05$ was considered as statistically significant difference.

$p=0.023$). Higher body mass index (BMI), Waist hip ratio (WHR) and Serum uric acid were significantly associated with higher grade of MASLD on US (mean 25.2 in grade 1 versus 26.2 in grade 2; $p < 0.001$), (mean 0.97 in grade 1 versus 1.01 in grade 2; < 0.001) and (4.6 in grade 1 and 6.6 in grade 2; $p < 0.001$) respectively (Table 2).

Pearson analysis revealed that SUA was significantly and positively correlated with BMI ($p < 0.001$), WHR ($p < 0.001$), AST ($p = 0.009$), ALT ($p = 0.004$), SUA ($p = 0.007$) and GGT ($p = 0.004$) (Table 3).

Multiple linear regression analysis showed that MASLD was independently and positively correlated with BMI ($p = 0.032$), WHR ($p = 0.025$), ALT ($p = 0.032$), HDL ($p = 0.024$) and TG ($p = 0.012$) (Table 4).

Discussion

The current study was specifically aimed at evaluating the prevalence, clinical profile and risk factors of MASLD in a population of newly diagnosed T2DM patients. The prevalence of MASLD is high among diabetic subjects due to genetic and environmental influences. The relationship between MASLD and T2DM seems

to be bidirectional; an increase in T2DM prevalence may lead to a rise in MASLD cases, and conversely, an increase in MASLD may also increase the prevalence of T2DM in addition to worsening of the natural course of diabetic complications (both micro and macrovascular) in T2DM patients. This correlation suggests the possibility of a genetic connection, along with obesity and insulin resistance, serving as potential predictors for both T2DM and MASLD.

Our findings demonstrate a substantial burden of MASLD among newly diagnosed T2DM patients with higher rate of prevalence among women than in men. The reported prevalence of MASLD among individuals with T2DM highly variable in India, ranging from 45.0% to 72.0% across different regional studies [9, 10]. Our study demonstrated a prevalence of 64% MASLD among our newly diagnosed T2DM patients, with a higher prevalence observed in females (71.4%) compared to males (57.5%). These results are also in accordance with observations from other studies studying the prevalence of MASLD among T2DM patients [23–25].

The high prevalence in this part of world may reflect a characteristic body composition marked by higher adiposity and lower muscle mass, increasing susceptibility

Table 2: Relationship between serum uric, HOMA IR, BMI, WHR and MASLD.

Parameter median (IQR)	MASLD			P-value
	Absent	Grade 1	Grade 2	
Uric acid (mg/dl)	4 (0.78)	4.6 (0.83)	6.6 (1.2)	0.001
HOMA IR	5.74 (4.61)	6.34 (4.6)	6.78 (4.73)	0.509
BMI (kg/m ²)	24.12 (4.01)	25.2 (3.99)	26.2 (4.4)	<0.001
WHR	0.96 (0.06)	0.97 (0.06)	1.01 (0.07)	<0.001

Note: Median (interquartile range) or n (%). SUA – serum uric acid; HOMA-IR – homeostasis model assessment of insulin resistance; BMI – body mass index; WHR – waist-to-hip ratio; MASLD – Metabolic dysfunction associated steatotic liver disease.

Table 3: Pearson correlation analyses of parameters associated with MASLD.

	r	p
Age (years)	0.116	0.184
DOD (years)	0.051	0.584
BMI (kg/m²)	0.317	0.000*
WHR	0.295	0.000*
SBP (mmHg)	0.166	0.053
DBP (mmHg)	0.154	0.074
AST (U/L)	0.223	0.009*
ALT (U/L)	0.244	0.004*
GGT (U/L)	0.246	0.004*
ALP (U/L)	0.114	0.184
SUA (mg/dl)	0.231	0.007*
TC (mg/dl)	0.124	0.152
TGs (mg/dl)	0.131	0.130
HDL (mg/dl)	-0.236	0.005*
LDL (mg/dl)	0.153	0.075
A1C (%)	0.172	0.156
HOMA-IR	0.072	0.407
FINS (mU/l)	0.147	0.088

Note: DOD – Duration of diabetes; BMI – body mass index; WHR – waist-to-hip ratio; SBP – systolic pressure; DBP – diastolic pressure; ALT – alanine aminotransferase; AST – aspartate aminotransferase; FINS – fasting insulin; TC – total cholesterol; TG – triglycerides; LDL – low density lipoprotein; HDL – high density lipoprotein; FBG – fasting blood glucose; HbA1C – glycosylated haemoglobin; HOMR-IR – Homeostatic model assessment for insulin resistance; FINS – fasting insulin; FBG – fasting blood glucose. * – $p < 0.05$ was considered as statistically significant difference.

to metabolic syndrome, including MASLD and T2DM. Although South Asians may not appear as overtly obese as other ethnic groups, they tend to be more metabolically obese and thus at higher risk for MASLD [26]. Our results underscore the substantial burden of MASLD in patients recently diagnosed with T2DM emphasizes

how critical it is to assess and treat MASLD as soon as possible to mitigate the risk of complication and subsequent morbidity and mortality seen in advanced liver disorders.

The higher prevalence of MASLD observed among women in the current study is consistent with results

Table 4: Independent factors affecting MASLD in T2DM patients in multiple linear regression analysis.

Variable	B	S.E	Beta	p
BMI	0.033	0.011	0.308	0.032*
WHR	0.003	0.001	0.377	0.025*
ALT	0.033	0.011	0.309	0.032*
TG	0.003	0.001	0.352	0.026
HDL	-0.003	0.001	-0.325	0.029*

Note: BMI – body mass index; WHR – waist-to-hip ratio; ALT – alanine aminotransferase; TG – triglycerides; HDL – high density lipoprotein. * – $p < 0.05$ was considered as statistically significant difference.

from several earlier studies [27, 28]. Although oestrogen is generally protective against MASLD, hormonal imbalance in overweight and obese individuals characterized by increased androgens and reduced oestrogen may promote visceral adiposity and metabolic dysfunction, thereby contributing to the higher prevalence observed in females [29, 30].

Patients with concomitant T2DM and MASLD exhibited significantly higher measures of both overall and central adiposity, including BMI, body weight, waist circumference, and waist to hip ratio, compared to those with T2DM alone. These findings align with the results from earlier studies that have identified obesity—particularly visceral adiposity—as a key determinant of MASLD development [31].

Moreover, the observed association between increasing BMI and WHR with higher grades of hepatic steatosis on ultrasonography suggests a dose–response relationship, indicating that worsening adiposity contributes not only to the presence but also to the severity of MASLD. These findings support the role of central adiposity in hepatic lipid accumulation and disease progression.

Patients with MASLD in our study exhibited higher levels of obesity, hypertension, hyperglycaemia, insulin resistance, and deranged lipids, all of which are recognised risk factors for the development of MASLD [32, 33]. The results from the study reinforced the well-established clinical association of MASLD with elements of metabolic syndrome including dyslipidaemia, hypertension and obesity.

In the present study BMI and WHR emerged as independent predictor of MASLD, highlighting the contribution of overall adiposity in its development and progression. Furthermore, elevated ALT level also emerged as an independent predictor of MASLD in regression analysis, underscoring the hepatic nature of MASLD. Additionally, higher triglycerides and lower HDL were significant predictors of MASLD among diabetic patients reflecting underlying metabolic unhealthiest associated with MASLD [34].

The limitations of our study should be acknowledged. First, the cross-sectional design precludes causal inferences regarding the associations between the studied variables and MASLD or disease progression. Second, the sensitivity of liver ultrasonography varies depending on the degree of hepatic steatosis, and the absence of advanced diagnostic modalities, such as liver biopsy or advanced imaging techniques, may have limited diagnostic accuracy and clinical applicability. Third, the single-center setting and the ethnically ho-

mogeneous study population may limit the generalizability of our findings to other populations and health-care settings.

Conclusion

MASLD is highly prevalent among patients with T2DM, with obesity emerging as a major metabolic risk factor. Obesity is the major risk factor for MASLD, as BMI and waist circumference not only positively correlate with the presence of MASLD but also with disease severity. Despite the existence of larger-scale studies, this study offers a distinct contribution by focusing on newly diagnosed T2DM patients, an underrepresented yet clinically important population. Thus, any patient diagnosed as T2DM warrants thorough clinical assessment for early detection and management of hepatic impairment, aiming to reduce the burden of liver-related complications in diabetic populations.

Conflict of interest

The authors declare no conflict of interest.

Ethics approval

This study was performed in accordance with the principles outlined in the Declaration of Helsinki and received approval from the hospital's ethics committee (Approval no: SIMS1131/IEC-SKIMS/2019-386, Date: 16/02/2019).

Consent to participate

Written informed consent was obtained from all the participants.

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