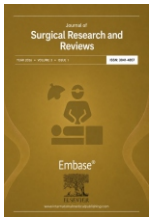
JSRR
Journal of Surgical Research & Reviews
www.internationalmedicalpublishing.com



Research Article

Section: Medicine

Prevalence of Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) in Young Type 2 Diabetes Mellitus Patients & Its Association with Insulin Resistance

Khwaja Saifullah Zafar[1*], Syed Hasan Amir[1], Hamid Ashraf[1] & Anup Sharma[1]

[1]Department of Medicine, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, India

HIGHLIGHTS

- High MASLD prevalence
- Insulin resistance linked to MASLD
- Metabolic risks increased MASLD
- Fibrosis was common
- HOMA-IR predicted MASLD

Key Words:
Metabolic dysfunction-associated steatotic liver disease,
Type 2 Diabetes Mellitus
Insulin resistance
HOMA-IR
FibroScan
Hepatic fibrosis

ABSTRACT

Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease worldwide. Young-onset Type 2 Diabetes Mellitus (T2DM) increases the risk of MASLD due to insulin resistance, obesity, and adverse metabolic profiles, predisposing individuals to early hepatic steatosis and fibrosis. **Aim & Objective:** This study aimed to determine the prevalence of MASLD among young adults with T2DM and to evaluate its association with insulin resistance and other metabolic risk factors. **Materials & Methods:** A hospital-based cross-sectional study included 92 adults aged 18–40 years with T2DM. Clinical, anthropometric, biochemical, and glycaemic parameters were assessed. Insulin resistance was evaluated using HOMA-IR, while hepatic steatosis and fibrosis were assessed by FibroScan (CAP and LSM). Statistical analyses included group comparisons, correlations, logistic regression, and ROC analysis. **Results:** MASLD was identified in 51 participants, yielding an overall prevalence of **55.4%**. Insulin resistance (HOMA-IR ≥1.7) was present in **73.9%** of the study population and was significantly more frequent among participants with MASLD than those without the disease (64.7% vs. 20.0%, p<0.001). Individuals with MASLD demonstrated significantly higher HOMA-IR values, fasting insulin levels, HbA1c, fasting and postprandial blood glucose, body mass index, waist circumference, triglycerides, LDL cholesterol, and liver enzyme levels. FibroScan assessment revealed evidence of advanced fibrosis in **26.1%** of participants. HOMA-IR showed good diagnostic performance for predicting MASLD (AUC = 0.842), with an optimal cut-off value of **3.15**. **Conclusion:** MASLD is common in young adults with T2D M and is strongly linked to insulin resistance, obesity, poor glycaemic control, dyslipidaemia, and fibrosis, highlighting the need for routine screening and early metabolic interventions.

*** Corresponding Author:** Khwaja Saifullah Zafar, e-mail: khwaja.md@amu.ac.in
Article History: Received 20 July 2026; Received in Revised form 05 September 2026; Accepted 12 September 2026
How To Cite: Khwaja Saifullah Zafar, Syed Hasan Amir, Hamid Ashraf & Anup Sharma. Prevalence of Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) in Young Type 2 Diabetes Mellitus Patients & Its Association with Insulin Resistance. *JSRR : Journal of Surgical Research & Reviews.* 2026;3(1):1-11.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) has become a major global public health challenge, with an alarming rise in its incidence among adolescents and young adults over the past two decades. Earlier onset of T2DM is associated with prolonged exposure to hyperglycaemia and metabolic abnormalities, resulting in accelerated development of both microvascular and macrovascular complications. Rapid urbanization, sedentary lifestyles, unhealthy dietary habits, and the increasing prevalence of obesity have collectively contributed to this epidemiological shift. Among these factors, central obesity and insulin resistance are considered the principal drivers of early-onset metabolic disease and its associated complications [1,2].

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly referred to as non-alcoholic fatty liver disease (NAFLD), is now recognized as the hepatic manifestation of systemic metabolic dysfunction. The recent change in nomenclature reflects a better understanding of the disease process by emphasizing its close relationship with obesity, insulin resistance, dyslipidaemia, and Type 2 Diabetes Mellitus rather than defining the condition by the absence of alcohol consumption. MASLD encompasses a broad clinical spectrum ranging from simple hepatic steatosis to metabolic dysfunction-associated steatohepatitis (MASH), progressive fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma.

The burden of MASLD has increased in parallel with the global epidemic of obesity and diabetes, making it one of the leading causes of chronic liver disease worldwide. Recent epidemiological studies suggest that nearly two-thirds of individuals with T2DM have evidence of hepatic steatosis, while a substantial proportion develop clinically significant fibrosis. Importantly, liver-related morbidity is no longer confined to older adults. Increasing rates of obesity and metabolic syndrome have led to a growing prevalence of MASLD among younger individuals, exposing them to decades of ongoing hepatic injury and substantially increasing their lifetime risk of advanced fibrosis, cirrhosis, cardiovascular disease, and liver-related mortality [3].

Insulin resistance is the central pathophysiological mechanism linking T2DM and MASLD. Reduced insulin sensitivity promotes excessive lipolysis in adipose tissue, leading to increased delivery of free fatty acids to the liver. This results in hepatic triglyceride accumulation, enhanced de novo lipogenesis, oxidative stress, mitochondrial dysfunction, and chronic low-grade inflammation. In turn, hepatic steatosis further aggravates systemic insulin resistance by disrupting insulin signalling pathways and altering the secretion of inflammatory cytokines and adipokines. This bidirectional relationship creates a self-perpetuating cycle that accelerates both metabolic dysfunction and progression of liver disease [4].

Young-onset T2DM represents a particularly aggressive phenotype characterized by severe insulin resistance, central obesity, poor glycaemic control, dyslipidaemia, and an earlier decline in pancreatic β-cell function. Consequently, affected individuals experience complications at a younger age and after a shorter duration of diabetes than those diagnosed later in life. Given that the same metabolic disturbances contribute to hepatic fat accumulation, young adults with T2DM constitute a population at especially high risk for developing MASLD and progressive liver fibrosis [5,6].

Despite growing recognition of MASLD as an important complication of diabetes, routine screening remains inconsistent, particularly among younger adults who are often perceived to have a lower risk of advanced liver disease. Because MASLD is frequently asymptomatic during its early stages, delayed diagnosis may allow progression to irreversible fibrosis before clinical detection. Early identification of hepatic steatosis and fibrosis provides an opportunity for timely lifestyle modification, optimization of glycaemic control, weight reduction, and

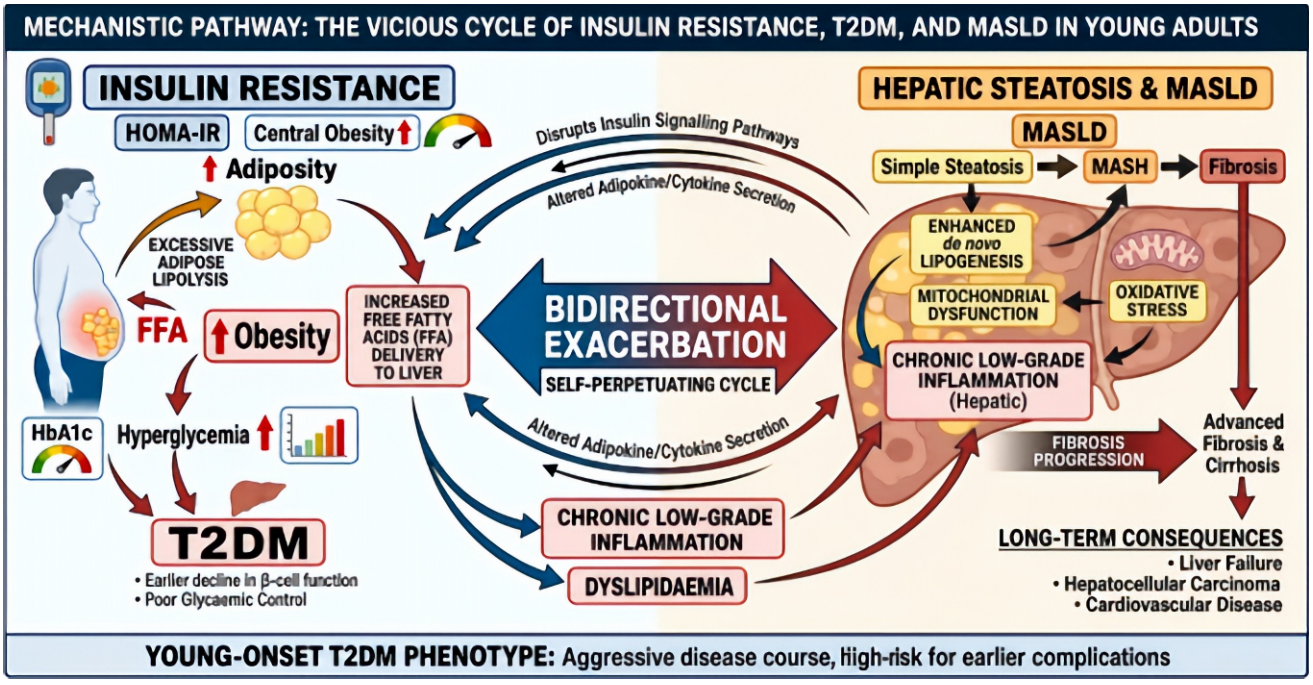


Figure 1: Vicious cycle linking insulin resistance, T2DM, MASLD, inflammation, and fibrosis in young adults.

implementation of therapies aimed at reducing long-term hepatic and cardiovascular complications [7-10]. **Figure 1.** Mechanistic pathway illustrating the vicious cycle linking insulin resistance, T2DM, and MASLD in young adults, highlighting adipose lipolysis, free fatty acid flux, hepatic steatosis, inflammation, oxidative stress, and progressive fibrosis.

Although several studies have examined MASLD in patients with T2DM, evidence focusing specifically on young adults remains limited, particularly in the Indian population. Data regarding the prevalence of MASLD, the burden of insulin resistance, and the extent of liver fibrosis in this age group are still scarce. Understanding these associations is essential for identifying high-risk individuals and developing effective screening strategies [11,12].

Therefore, the present study was undertaken to determine the prevalence of MASLD among young adults with Type 2 Diabetes Mellitus and to evaluate its association with insulin resistance. In addition, the study explored the relationship between MASLD and anthropometric, biochemical, glycaemic, and fibrosis-related parameters to better characterize the metabolic profile of this high-risk population.

MATERIAL & METHODS

This single-center, hospital-based, cross-sectional study was conducted at the Department of General Medicine, JNMCH, Aligarh, from February 2024 to February 2026. Ethical approval has been obtained from the Ethical Approval Committee of JNMCH, Aligarh.

Study Population

The study included **92 consecutive young adults** diagnosed with **Type 2 Diabetes Mellitus (T2DM)** who attended the outpatient clinics or were admitted to the Department of General Medicine during the study period.

Inclusion Criteria

Participants were eligible if they:

- Were **18-40 years** of age;
- Had a confirmed diagnosis of **Type 2 Diabetes Mellitus** according to the American Diabetes Association (ADA) criteria;
- Provided written informed consent to participate.

Exclusion Criteria

Patients were excluded if they:

- Were older than 40 years;
- Had liver disease due to causes other than metabolic dysfunction, including significant alcohol intake, chronic hepatitis B or C infection, autoimmune liver disease, wilson disease, haemochromatosis, or other chronic liver disorders;
- Had acute hepatitis;
- Were pregnant or lactating;
- Had pancreatic disease or pancreatic insufficiency;
- Had forms of diabetes other than T2DM;
- Were receiving potentially hepatotoxic medications;
- Declined participation in the study.

Clinical Evaluation

A detailed medical history was obtained for each participant, including demographic characteristics, duration of diabetes, current treatment, lifestyle factors, and diabetes-related complications. A comprehensive physical examination was performed, including measurement of **height, weight, body mass index (BMI), waist circumference, and blood pressure** using standardized techniques.

Laboratory Investigations

Following an overnight fast, venous blood samples were collected for biochemical analysis. The following investigations were performed:

- Complete blood count
- Liver function tests (AST, ALT, bilirubin, serum proteins, albumin)
- Fasting blood glucose
- Postprandial blood glucose
- Glycated haemoglobin (HbA1c)
- Fasting serum insulin
- Lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C, VLDL-C)
- Viral serology (HBV, HCV, and HIV)

Insulin resistance was estimated using the **Homeostatic Model Assessment for Insulin Resistance (HOMA-IR)** according to the standard formula:

HOMA-IR = [Fasting Plasma Glucose (mg/dL) × Fasting Insulin (μIU/mL)] ÷ 405

Participants were categorized as having normal insulin sensitivity, borderline insulin resistance, or insulin resistance using predefined HOMA-IR thresholds.

Assessment of Hepatic Steatosis and Fibrosis

All participants underwent transient elastography using **FibroScan®** to evaluate hepatic steatosis and fibrosis.

Hepatic steatosis was assessed using the **Controlled Attenuation Parameter (CAP)**, whereas liver fibrosis was evaluated using **Liver Stiffness Measurement (LSM)** expressed in kilopascals (kPa).

Fibrosis stages were classified according to liver stiffness values:

- **F0–F1 (No or mild fibrosis):** <7.0 kPa
- **F2 (Significant fibrosis):** 7.0–8.9 kPa
- **F3 (Advanced fibrosis):** 9.0–11.9 kPa
- **F4 (Cirrhosis):** ≥12.0 kPa

Additional non-invasive fibrosis indices, including the **Fibrosis-4 Index (FIB-4)**, **AST-to-Platelet Ratio Index (APRI)**, and **MASLD Fibrosis Score**, were also calculated to complement FibroScan findings.

Outcome Measures

Primary Outcome

- To determine the prevalence of **Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD)** among young adults with Type 2 Diabetes Mellitus.

Secondary Outcomes

- To evaluate the association between MASLD and insulin resistance.
- To examine the relationship between MASLD and anthropometric variables.
- To assess the association of MASLD with glycaemic status, lipid profile, liver enzymes, and non-invasive fibrosis markers.
- To evaluate the diagnostic performance of HOMA-IR for identifying MASLD.

Statistical Analysis

Statistical analyses were performed using **IBM SPSS Statistics version 26**, **MedCalc version 20**, and **GraphPad Prism version 9**.

Data quality was assessed before analysis by checking for missing values, outliers, and normality of distribution using the **Shapiro–Wilk test**.

Continuous variables are presented as **mean ± standard deviation (SD)** or **median (interquartile range)** according to data distribution, whereas categorical variables are expressed as **frequency and percentage**. Comparisons between groups were performed using the **independent Student's t-test** or the **Mann–Whitney U test**, as appropriate. Categorical variables were analysed using the **Chi-square test** or **Fisher's exact test**.

Correlations between metabolic variables were evaluated using **Spearman's rank correlation coefficient**.

To identify factors independently associated with MASLD, **multivariable logistic regression analysis** was performed after adjustment for clinically relevant covariates.

Receiver operating characteristic (**ROC**) curve analysis was used to evaluate the predictive performance of HOMA-IR and fasting insulin for identifying MASLD, and the optimal cut-off value was determined using the **Youden Index**. A **two-tailed p-value <0.05** was considered statistically significant.

RESULTS

The study included 92 young patients with Type 2 Diabetes Mellitus, with a nearly equal gender distribution comprising 45 males (48.9%) and 47 females (51.1%). The mean age of the participants was 29.42 ± 6.53 years (range 18–40 years), with the majority belonging to the 26–30 years age group (33.7%), followed by the 18–25 years group (26.1%). Gender distribution across age groups was relatively balanced. MASLD was identified in 51 patients, resulting in a prevalence of 55.4%, while 41 patients (44.6%) had no evidence of MASLD. The prevalence of MASLD was comparable between males (57.8%) and females (53.2%), with no statistically significant gender-based difference (p = 0.658). Hematological parameters, including hemoglobin, total leukocyte count, and platelet count, were generally within normal physiological ranges. Liver function assessment demonstrated mild elevations of AST (41.8 ± 18.6 U/L) and ALT (49.6 ± 24.3 U/L), suggestive of mild hepatocellular involvement, whereas bilirubin levels and serum protein profiles remained within normal limits, indicating preserved hepatic synthetic and excretory function. All participants tested negative for HBV, HCV, and HIV. Glycaemic evaluation revealed suboptimal diabetic control, with mean HbA1c of 8.12 ± 1.46%, fasting glucose of 156.8 ± 42.5 mg/dL, postprandial glucose of 228.4 ± 58.7 mg/dL, and fasting insulin of 18.6 ± 8.9 µIU/mL, reflecting significant insulin resistance and metabolic dysfunction. Lipid analysis demonstrated a characteristic diabetic dyslipidaemic pattern with elevated total cholesterol (198.6 ± 42.4 mg/dL), LDL cholesterol (118.7 ± 34.8 mg/dL), triglycerides (186.4 ± 78.5 mg/dL), and VLDL cholesterol (37.3 ± 15.7 mg/dL), along with relatively low HDL cholesterol levels (41.2 ± 8.6 mg/dL). Anthropometric assessment showed a mean BMI of 27.48 ± 3.77 kg/m² and increased waist circumference in both sexes, indicating a predominance of overweight and central obesity. According to Asian BMI classification criteria, 42.4% of participants were classified as Obese Class I, 22.8% as Obese Class II, and 19.6% as overweight, while only 15.2% had normal BMI. Overall, the findings highlight a high burden of MASLD, obesity, poor glycaemic control, insulin resistance, and atherogenic dyslipidaemia among young adults with Type 2 Diabetes Mellitus.

FibroScan assessment showed a mean CAP score of 292.6 ± 42.8 dB/m and mean liver stiffness of 8.42 ± 3.18 kPa, indicating significant hepatic steatosis with predominantly mild-to-moderate fibrosis. Based on FibroScan, 41.3% had no/mild fibrosis (F0–F1), 32.6% had significant fibrosis (F2), & 26.1% had advanced fibrosis (F3), with no cases of cirrhosis (F4), suggesting that most participants had early-to-moderate stages of liver disease **(Table 1)**.

Assessment of insulin resistance using HOMA-IR demonstrated a high burden of metabolic dysfunction among the study participants. The mean HOMA-IR value was 3.84 ± 2.16, with a median of 3.32 and a range of 0.72–11.84, indicating considerable variability in insulin sensitivity within the cohort. Based on HOMA -IR classification, 68 participants (73.9%) had insulin resistance (HOMA-IR ≥1.7), while 14 participants (15.2%) were categorized as having borderline or intermediate insulin resistance (HOMA-IR 1.0–1.69). Only 10 participants (10.9%) demonstrated normal insulin sensitivity with HOMA-IR values below 1.0. These findings highlight the high prevalence of insulin resistance among young patients with Type 2 Diabetes Mellitus and support its important role in the development and progression of MASLD and related metabolic complications.

Adeate glycaemic control (HbA1c <7%) was more common among MASLD-negative participants (41.5%) than MASLD-positive participants (21.6%), while poor glycaemic control (HbA1c ≥7%) was more prevalent in those with MASLD (78.4% vs. 58.5%). This significant association (p = 0.041) suggests that higher HbA1c levels are linked to an increased prevalence of MASLD **(Table 2)**.

Participants with MASLD had a higher prevalence of diabetes-related complications, with significantly greater rates of diabetic neuropathy (47.1% vs. 19.5%, p=0.006), retinopathy (33.3% vs. 14.6%, p=0.041), nephropathy (29.4% vs. 9.8%, p=0.022), and hypertension (66.7% vs. 36.6%, p=0.004) compared to those without MASLD. Although cardiovascular and cerebrovascular complications were also more frequent in the MASLD-positive group, these associations were not statistically significant, indicating an overall increased burden of diabetic complications among patients with MASLD **(Table 3)**.

Dyslipidaemia was significantly more common among participants with MASLD, affecting 84.3% compared to 48.8% of those without MASLD. The strong association between dyslipidaemia and MASLD (p<0.001) highlights the close relationship between lipid abnormalities and metabolic liver disease in young patients with Type 2 Diabetes Mellitus **(Table 4)**.

The study population had a mean duration of Type 2 Diabetes Mellitus (T2DM) of 5.84 ± 3.12 years (median 5 years; range 1-15 years), indicating variable disease chronicity among young adults. Patients with metabolic dysfunction-associated steatotic liver disease (MASLD) had a significantly longer duration of diabetes than those without MASLD (7.1 ± 3.2 vs. 4.2 ± 2.1 years, $p < 0.001$), suggesting that prolonged exposure to hyperglycaemia and metabolic stress contributes to the development of hepatic steatosis. A strong association was observed between insulin resistance and MASLD, with the prevalence of MASLD increasing progressively across HOMA-IR categories. MASLD was present in only 20.0% of participants with normal insulin sensitivity, compared with 64.7% of those with insulin resistance (HOMA-IR ≥1.7), demonstrating a highly significant relationship between insulin resistance and hepatic steatosis ($p < 0.001$). Among insulin-resistant individuals, MASLD was more common in males than females (69.7% vs. 60.0%, $p = 0.018$), indicating a significant gender-wise association. Participants with MASLD exhibited markedly higher body mass index (29.1 ± 3.6 vs. 25.5 ± 2.8 kg/m²), waist circumference (103.4 ± 9.8 vs. 94.6 ± 8.7 cm), fasting blood glucose (168.2 ± 44.6 vs. 142.7 ± 35.8 mg/dL), postprandial glucose (241.8 ± 61.2 vs. 211.6 ± 49.8 mg/dL), HbA1c (8.6 ± 1.4% vs. 7.5 ± 1.2%), fasting insulin (22.4 ± 8.2 vs. 13.8 ± 5.6 µIU/mL), and HOMA-IR (4.8 ± 2.1 vs. 2.6 ± 1.3) compared with MASLD-negative individuals (all $p < 0.05$). They also demonstrated a more atherogenic lipid profile, characterized by elevated total cholesterol, triglycerides, and LDL cholesterol, along with lower HDL cholesterol levels. Furthermore, MASLD-positive participants had significantly higher AST and ALT levels, indicating greater hepatocellular injury, and showed substantially elevated CAP scores (324.8 ± 28.6 vs. 252.5 ± 24.2 dB/m), FIB-4 scores, APRI scores, and MASLD Fibrosis Scores, reflecting increased hepatic fat accumulation and fibrosis risk. Similarly, insulin-resistant participants had significantly higher BMI, waist circumference, fasting and postprandial glucose levels, HbA1c, fasting insulin, total cholesterol, triglycerides, LDL cholesterol, AST, and ALT, while HDL cholesterol was significantly lower compared with participants having normal or borderline insulin sensitivity (all $p < 0.001$). These findings collectively demonstrate that insulin resistance is closely linked with obesity, central adiposity, poor glycaemic control, dyslipidaemia, hepatic injury, and fibrosis-related changes, highlighting its central role in the pathogenesis and progression of MASLD among young patients with T2DM.

Correlation analysis showed strong interrelationships between obesity, insulin resistance, glycaemic status, dyslipidaemia, and hepatic steatosis, with BMI and waist circumference positively associated with HOMA-IR, CAP score, and metabolic parameters. Liver fat (CAP score) and fibrosis markers (LSM, FIB-4, APRI, NFS) correlated significantly with glycaemic indices and triglycerides, while HDL cholesterol showed inverse associations with insulin resistance and lipid levels, indicating that metabolic dysfunction and central obesity are key drivers of MASLD and liver fibrosis progression in Type 2 Diabetes Mellitus **(Table 5)**.

Multivariable logistic regression analysis, which included BMI, waist circumference, HbA1c, triglycerides, HOMA-IR, ALT, and duration of diabetes, showed that although several variables were associated with MASLD on univariate analysis, none remained statistically significant independent predictors after adjustment. HOMA-IR showed increased odds of MASLD (aOR 1.83), suggesting a contributory role of insulin resistance, while ALT demonstrated a borderline association (aOR 1.21; $p = 0.082$), and waist circumference and diabetes duration also showed positive but non-significant trends. The overall model was statistically robust (likelihood ratio $p < 0.001$, pseudo $R^2 = 0.888$), and the Hosmer–Lemeshow test confirmed good calibration ($\chi^2 = 6.18$, $p = 0.627$), indicating acceptable model fit despite the absence of single independent predictors, thereby suggesting that MASLD in young T2DM is driven by an interacting cluster of metabolic abnormalities rather than isolated factors. ROC analysis showed that HOMA-IR (AUC 0.842; cut-off 3.15; sensitivity 80.4%; specificity 75.6%) and fasting insulin (AUC 0.816; cut-off 17.8 µIU/mL; sensitivity 78.4%; specificity 70.7%) both had good diagnostic performance for MASLD, with no statistically significant difference between them on DeLong testing ($p = 0.254$), indicating comparable predictive ability. Group comparisons further demonstrated that MASLD-positive participants had significantly worse anthropometric, glycaemic, lipid, hepatic, and fibrosis-related parameters than MASLD-negative individuals, with very large effect sizes for CAP score, fasting insulin, and HOMA-IR, and large effect sizes for BMI, waist circumference, triglycerides, ALT, AST, and liver stiffness, confirming strong metabolic and hepatic clustering in MASLD. Overall, effect size analysis highlighted that insulin resistance and hepatic steatosis measures showed the greatest differences, reinforcing MASLD as a manifestation of a severe insulin-resistant metabolic phenotype in young patients with Type 2 Diabetes Mellitus.

Shapiro-Wilk testing showed that most metabolic and hepatic variables, including glycaemic indices, insulin resistance markers, lipids, liver enzymes, CAP score, and liver stiffness measurement,

were not normally distributed, while BMI, HDL, and LDL showed approximate normality, supporting the use of non-parametric tests in analysis. Clinically, the study highlights insulin resistance and central obesity as key drivers of MASLD in young T2DM patients, with strong associations with hepatic steatosis, dyslipidaemia, and fibrosis, while FibroScan findings indicate predominantly early-stage disease, underscoring the importance of early screening and intervention to prevent progression and related complications **(Table 6)**.

Table 1: FibroScan and Fibrosis Profile of Study Participants (n = 92)

Parameter	Value
Controlled Attenuation Parameter (CAP), dB/m	292.6 ± 42.8
Liver Stiffness Measurement (LSM), kPa	8.42 ± 3.18
Fibrosis Risk Assessment	Majority of participants had low -to-intermediate fibrosis risk based on FIB -4, APRI, and MASLD Fibrosis Score; a subset demonstrated elevated fibrosis indices suggestive of progressive liver disease.
Fibrosis Category (FibroScan-based)	LSM (kPa)
No/Mild Fibrosis (F0 –F1)	< 7.0
Significant Fibrosis (F2)	7.0–8.9
Advanced Fibrosis (F3)	9.0–11.9
Cirrhosis (F4)	≥ 12.0

Table 2: HbA1c analysis

HbA1c Category	MASLD Present n (%)	95% CI	MASLD Absent n (%)	95% CI	Total	p-value
HbA1c <7%	11 (21.6%)	11.3–35.3%	17 (41.5%)	26.3–57.9%	28	0.041
HbA1c ≥7%	40 (78.4%)	64.7–88.7%	24 (58.5%)	42.1–73.7%	64	
Total	**51**		**41**		**92**	

Table 3: Distribution of Diabetes-related Complications According to MASLD Status

Complication	MASLD Present (n=51)	95% CI	MASLD Absent (n=41)	95% CI	Total n (%)	p-value
Diabetic Neuropathy	24 (47.1%)	33.1–61.5%	8 (19.5%)	9.3–34.6%	32 (34.8)	0.006
Diabetic Retinopathy	17 (33.3%)	20.8–47.9%	6 (14.6%)	5.6–29.2%	23 (25.0)	0.041
Diabetic Nephropathy	15 (29.4%)	17.5–43.8%	4 (9.8%)	2.7–23.1%	19 (20.7)	0.022
Cardiovascular Complications	13 (25.5%)	14.3–39.6%	6 (14.6%)	5.6–29.2%	19 (20.7)	0.201
Cerebrovascular Accidents	6 (11.8%)	4.4–23.9%	2 (4.9%)	0.6–16.5%	8 (8.7)	0.243
Hypertension	34 (66.7%)	52.1–79.2%	15 (36.6%)	22.1–53.1%	49 (53.3)	0.004

Table 4: Prevalence of dyslipidaemia

Dyslipidaemia Status	MASLD Present n (%)	95% CI	MASLD Absent n (%)	95% CI	p-value
Present	43 (84.3)	71.4-93.0%	20 (48.8)	33.3-64.5%	
Absent	8 (15.7)	7.0-28.6%	21 (51.2)	35.5-66.7%	<0.001
Total	**51**		**41**		

Table 5: Correlation between Anthropometric, Glycaemic, Lipid, and Fibrosis-related Variables among Study Participants

Variable 1	Variable 2	r	p-value
BMI	Waist Circumference, HOMA -IR, CAP Score	0.72, 0.58, 0.49	<0.001,<0.001, 0.002
Waist Circumference	Triglycerides, CAP Score, HbA1c	0.46, 0.61, 0.39	0.004, <0.001, 0.011
HOMA-IR	HbA1c, Fasting Insulin, CAP Score, ALT	0.64, 0.81, 0.57, 0.42	<0.001, <0.001, <0.001, 0.006
HbA1c	Triglycerides, ALT, CAP Score	0.41, 0.38, 0.44	0.008, 0.012, 0.004
Triglycerides	CAP Score, FIB-4 Score	0.48, 0.31	0.002, 0.018
CAP Score	Liver Stiffness Measurement, APRI Score, NFS	0.66, 0.43, 0.51	<0.001, 0.005, 0.001
Liver Stiffness Measurement	FIB-4 Score, APRI Score, NFS	0.62, 0.58, 0.69	<0.001, <0.001, <0.001
HDL Cholesterol	HOMA-IR, Triglycerides	−0.36, −0.52	0.019, 0.001

Table 6: Normality Testing Using Shapiro–Wilk Test

Variable	W Statistic	p-value	Interpretation
BMI (kg/m²)	0.972	0.061	Normally distributed
Waist Circumference (cm)	0.968	0.038	Not normally distributed
Fasting Blood Sugar (mg/dL)	0.931	<0.001	Not normally distributed
Postprandial Blood Sugar (mg/dL)	0.924	<0.001	Not normally distributed
HbA1c (%)	0.956	0.006	Not normally distributed
Fasting Insulin (µIU/mL)	0.887	<0.001	Not normally distributed
HOMA-IR	0.861	<0.001	Not normally distributed
Total Cholesterol (mg/dL)	0.964	0.021	Not normally distributed
Triglycerides (mg/dL)	0.842	<0.001	Not normally distributed
HDL Cholesterol (mg/dL)	0.978	0.124	Normally distributed
LDL Cholesterol (mg/dL)	0.971	0.054	Normally distributed
AST (U/L)	0.902	<0.001	Not normally distributed
ALT (U/L)	0.879	<0.001	Not normally distributed
CAP Score (dB/m)	0.962	0.017	Not normally distributed
Liver Stiffness Measurement (kPa)	0.846	<0.001	Not normally distributed

DISCUSSION

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as one of the most prevalent chronic liver diseases worldwide and is increasingly recognized as an important hepatic manifestation of metabolic syndrome. The coexistence of MASLD and Type 2 Diabetes Mellitus (T2DM) reflects a shared pathophysiological basis centred on insulin resistance, obesity, chronic inflammation, and dysregulated lipid metabolism. Young adults with T2DM are particularly susceptible because they are exposed to these metabolic abnormalities at an earlier age, predisposing them to prolonged hepatic injury and an increased lifetime risk of advanced fibrosis, cirrhosis, and cardiovascular disease. The present study evaluated the prevalence of MASLD among young Indian adults with T2DM and explored its relationship with insulin resistance and other metabolic risk factors [13,14].

In the present study, MASLD was detected in **55.4%** of participants, indicating that more than one-half of young adults with T2DM already had evidence of hepatic steatosis. This finding is clinically important because it demonstrates that liver involvement begins early in the course of diabetes and is not restricted to older individuals or those with longstanding disease. The observed prevalence is consistent with recent reports by **Shil et al. (2025)** and **Hu et al. (2024)**, who documented a similarly high burden of fatty liver disease among adolescents and young adults with diabetes. Although differences in study populations, diagnostic criteria, and imaging modalities may account for minor variations in prevalence across studies, the available evidence consistently supports the strong association between T2DM and MASLD.

The participants in our study demonstrated poor overall metabolic health, characterised by elevated HbA1c, fasting and postprandial glucose levels, dyslipidaemia, obesity, and increased waist circumference. These findings are in keeping with the concept that MASLD represents the hepatic manifestation of systemic metabolic dysfunction rather than an isolated liver disorder. Chronic hyperglycaemia, increased adiposity, and insulin resistance promote excessive hepatic lipid accumulation through enhanced lipolysis, increased free fatty acid flux to the liver, and stimulation of de novo lipogenesis. These mechanisms contribute not only to hepatic steatosis but also to oxidative stress, mitochondrial dysfunction, and persistent low-grade inflammation, thereby accelerating progression to fibrosis.

Insulin resistance emerged as the most important metabolic abnormality associated with MASLD in this study. Nearly three-quarters of the participants exhibited insulin resistance, and patients with MASLD had significantly higher fasting insulin concentrations and HOMA-IR values than those without MASLD. Furthermore, the prevalence of MASLD increased progressively with increasing HOMA-IR, highlighting the central role of insulin resistance in disease pathogenesis. These findings are consistent with the observations of **Tang et al. (2025)** and **Beyazal Polat et al. (2025)**, who reported that HOMA-IR is strongly associated with hepatic steatosis and steatohepatitis in individuals with metabolic dysfunction. The present findings reinforce the concept that insulin resistance is not merely associated with MASLD but represents a fundamental mechanism linking diabetes and liver disease [15-18]

Anthropometric indices also demonstrated a strong relationship with MASLD. Patients with hepatic steatosis had significantly higher body mass index and waist circumference than those without MASLD, indicating that both generalized and central obesity contribute substantially to disease development. Visceral adiposity is metabolically active and promotes secretion of pro-inflammatory cytokines, increased free fatty acid release, and reduced adiponectin levels, all of which impair insulin sensitivity and facilitate hepatic fat accumulation. Similar associations between obesity, abdominal adiposity, and MASLD have been consistently reported in previous epidemiological studies, emphasizing the importance of weight management in preventing disease progression.

Dyslipidaemia was another prominent finding in the present study. Participants with MASLD demonstrated significantly higher triglyceride and LDL cholesterol levels together with lower HDL cholesterol concentrations, reflecting the characteristic atherogenic lipid profile associated with insulin-resistant states. These lipid abnormalities further contribute to hepatic fat deposition & increase cardiovascular risk. Since cardiovascular disease remains the leading cause of mortality in patients with MASLD, early

recognition and treatment of dyslipidaemia should form an integral component of comprehensive disease management. An important observation in the present study was the association between MASLD and poor glycaemic control. Individuals with MASLD had significantly higher fasting glucose, postprandial glucose, and HbA1c levels than those without hepatic steatosis. Poor glycaemic control enhances hepatic glucose production, stimulates lipogenesis, and perpetuates insulin resistance, thereby promoting progression of liver disease. Conversely, hepatic steatosis further worsens systemic insulin resistance, creating a self-reinforcing cycle that accelerates metabolic deterioration. These findings highlight the importance of achieving optimal glycaemic control to reduce both hepatic and cardiovascular complications [19,20].

FibroScan assessment demonstrated that although most participants had mild-to-moderate fibrosis, more than one-quarter already had advanced fibrosis despite their relatively young age. This observation underscores the aggressive nature of metabolic liver disease in young adults with T2DM. Early identification of fibrosis is clinically important because fibrosis stage is the strongest predictor of liver-related morbidity and mortality. The use of transient elastography together with non-invasive fibrosis scores provides an effective strategy for identifying high-risk individuals who require closer surveillance and intensive management [21-23].

The present study also demonstrated a higher prevalence of diabetic microvascular complications, including neuropathy, nephropathy, retinopathy, and hypertension, among participants with MASLD. These findings support the concept that MASLD reflects generalized metabolic dysfunction rather than isolated hepatic disease. Chronic inflammation, endothelial dysfunction, oxidative stress, and insulin resistance represent common mechanisms underlying both hepatic injury and diabetic vascular complications. Consequently, detection of MASLD should prompt clinicians to evaluate patients for other diabetes-related complications and to intensify management of cardiovascular risk factors.

Correlation analysis further demonstrated significant positive relationships between obesity, insulin resistance, glycaemic indices, hepatic steatosis, and fibrosis markers. Body mass index, waist circumference, HOMA-IR, triglycerides, and HbA1c all showed significant correlations with CAP score and liver stiffness measurements, supporting the close interaction between adiposity, metabolic dysfunction, and progressive liver injury. The inverse association between HDL cholesterol and insulin resistance further highlights the interdependence of lipid abnormalities and metabolic liver disease [24,25].

Although several metabolic variables were significantly associated with MASLD in univariate analyses, multivariable logistic regression did not identify any single independent predictor after adjustment for potential confounding factors. This finding should not be interpreted as evidence against the importance of insulin resistance or obesity; rather, it reflects the complex and interrelated nature of metabolic risk factors. MASLD develops through the cumulative effects of obesity, dysglycaemia, dyslipidaemia, insulin resistance, and chronic inflammation, making it difficult for any single variable to account for disease risk independently. Nevertheless, HOMA-IR demonstrated excellent diagnostic performance with an area under the ROC curve of **0.842**, suggesting that it may serve as a practical and inexpensive screening tool for identifying young diabetic patients at increased risk of MASLD.

The findings of the present study have important clinical implications. Considering the high prevalence of MASLD and the substantial burden of insulin resistance observed in this relatively young population, routine screening for hepatic steatosis should be considered in all young adults with T2DM, particularly those with obesity, central adiposity, poor glycaemic control, or dyslipidaemia. Early lifestyle modification, weight reduction, optimization of glycaemic control, and the use of antidiabetic agents with proven hepatic benefits may reduce disease progression and improve long-term metabolic outcomes [26].

CONCLUSION

The present study demonstrates that **Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)** is highly prevalent among young adults with **Type 2 Diabetes Mellitus**, affecting more than half of the study population. The findings highlight a strong association between MASLD and insulin resistance, obesity, central adiposity, poor glycaemic control, dyslipidaemia, and early hepatic fibrosis, emphasizing the close interplay between metabolic dysfunction and liver disease.

Among the various metabolic parameters evaluated, **HOMA-IR** showed good diagnostic performance for identifying individuals at increased risk of MASLD, underscoring the pivotal role of insulin resistance in disease pathogenesis. The presence of advanced fibrosis in a considerable proportion of these relatively young patients further indicates that clinically significant liver injury may develop early in the course of diabetes, even before overt hepatic symptoms become apparent.

These findings support the incorporation of **routine screening for MASLD** in young adults with T2DM, particularly in those with obesity, poor glycaemic control, or other features of metabolic syndrome. Early identification through non-invasive tools such as **FibroScan** and metabolic risk assessment provides an opportunity for timely intervention, including intensive lifestyle modification, weight reduction, optimization of glycaemic control, and management of dyslipidaemia, with the aim of preventing progression to advanced fibrosis, cirrhosis, and liver-related complications. Although this study provides valuable insights into the burden of MASLD in young Indian adults with T2DM, larger multicentre prospective studies are required to validate these findings, evaluate long-term clinical outcomes, and identify additional genetic and molecular determinants of disease progression. Future research should also assess the impact of emerging therapeutic strategies targeting insulin resistance and metabolic dysfunction on the prevention and treatment of MASLD in this high-risk population.

In conclusion, MASLD should be regarded as an integral component of the metabolic disease spectrum rather than an isolated hepatic disorder. Early recognition and comprehensive management of metabolic risk factors are essential to reduce both liver-related and cardiometabolic complications & to improve long-term health outcomes in young adults with Type 2 Diabetes Mellitus.

LIMITATIONS & FUTURE PERSPECTIVES

Several limitations should be acknowledged. First, this was a single-centre cross-sectional study, limiting the generalizability of the findings and precluding causal inference. Second, liver biopsy, the reference standard for diagnosing steatohepatitis and fibrosis, was not performed because of its invasive nature. Third, the relatively modest sample size may have reduced the statistical power to detect independent predictors in multivariable analysis. Finally, inflammatory biomarkers, adipokines, and genetic factors such as **PNPLA3** and **TM6SF2** polymorphisms were not evaluated, although these may influence susceptibility to MASLD. Overall, the present study demonstrates that MASLD is highly prevalent among young adults with T2DM and is closely associated with insulin resistance, obesity, poor glycaemic control, dyslipidaemia, and early hepatic fibrosis. These findings support routine metabolic risk assessment and early liver screening in this high-risk population to facilitate timely intervention and reduce future liver-related and cardiovascular complications. Future studies should incorporate multi-centre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic & management approaches. Such efforts will improve overall patient care & help minimize complications.

Strengths of the Study

The present study has several strengths. It specifically focused on young adults with T2DM, a population that remains underrepresented in the existing literature. Hepatic steatosis and fibrosis were evaluated using FibroScan, a validated non-invasive modality with greater diagnostic accuracy than conventional ultrasonography. In addition, detailed assessment of insulin resistance, anthropometric variables, glycaemic parameters, lipid profile, and fibrosis indices allowed comprehensive evaluation of the metabolic determinants of MASLD.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical healthcare, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

MASLD: Metabolic Dysfunction-Associated Steatotic Liver Disease
T2DM: Type 2 Diabetes Mellitus
HOMA-IR: Homeostatic Model Assessment of Insulin Resistance
HbA1c: Glycated Hemoglobin
CAP: Controlled Attenuation Parameter
LSM: Liver Stiffness Measurement
LDL: Low-Density Lipoprotein
ROC: Receiver Operating Characteristic
AUC: Area Under the Curve
T2DM: Type 2 Diabetes Mellitus

AUTHOR INFORMATION

Dr. Khwaja Saifullah Zafar: Professor
Dr. Syed Hasan Amir: Associate Professor
Dr. Hamid Ashraf: Professor
Dr. Anup Sharma: Third Year, Junior Resident

AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

ACKNOWLEDGEMENT

The authors sincerely acknowledge the seniors of the Department of Medicine, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, India. We are grateful to our institute for providing the necessary resources to carry out this work. We also extend our heartfelt thanks to our colleagues and technical staff for their valuable assistance during the study.

CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

FUNDING

None

ETHICAL APPROVAL & CONSENT TO PARTICIPATE

All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

PUBLISHER'S NOTE

All statements made in this article are the sole responsibility of the authors and do not necessarily reflect the views of the publisher, editors, or reviewers. The journal maintains a neutral stance regarding jurisdictional claims in institutional affiliations presented in published work.

ARCHIVING INFORMATION

- zenodo
- Self-archiving on Google and Amazon Web Services (AWS) cloud servers, as well as on three dedicated in-house servers.
- PKP Preservation Network

REFERENCE

1. Zhou Y, Chen Y, Tang Y, Zhang S, Zhuang Z, Ni QJD, et al. Rising tide: the growing global burden and inequalities of early-onset type 2 diabetes among youths aged 15–34 years (1990-2021). 2025;17(1):1-14. https://doi.org/10.1186/s13098-025-01673-0
2. Zhang K, Ma Y, Luo Y, Song Y, Xiong G, Ma Y, et al. Metabolic diseases and healthy aging: identifying environmental and behavioral risk factors and promoting public health. 2023;11:1-10. https://doi.org/10.3389/fpubh.2023.1253506
3. Rodriquez IM, O'Sullivan KL. Youth-onset type 2 diabetes: burden of complications and socioeconomic cost. Clin Diabetes Res. 2023;23(5):59-67. https://doi.org/10.1007/s11892-023-01501-7
4. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (MASLD) and nonalcoholic steatohepatitis (MASH): a systematic review. Hepatology. 2023;77(4):1335-1347. https://doi.org/10.1097/hep.0000000000000004
5. Wang J, Du J, Wang M, Jin M, Tang Z, Mao Y. Global, regional, and national burden of MASLD in youths and young adults aged 15-39 years, 1990-2021, its attributable risk factors, and projections to 2035: a systematic analysis of the Global Burden of Disease Study 2021. Front Nutr. 2025;12:1-15. https://doi.org/10.2139/ssrn.4968223
6. Grander C, Grabherr F, Tilg H. Non-alcoholic fatty liver disease: pathophysiological concepts and treatment options. Cardiovasc Res. 2023;119(9):1787-1798. https://doi.org/10.1093/cvr/cvad095
7. Strati M, Moustaki M, Psaltopoulou T, Vryonidou A, Paschou SA. Early onset type 2 diabetes mellitus: an update. Endocrine. 2024;85(3):965-978. https://doi.org/10.1007/s12020-024-03772-w
8. Dharmalingam M, Yamasandhi PG. Nonalcoholic fatty liver disease and type 2 diabetes mellitus. Indian J Endocrinol Metab. 2018;22(3):421-428. https://doi.org/10.4103/ijem.ijem_585_17
9. Marušić M, Paić M, Knobloch M, Liberati Pršo AM. MASLD, insulin resistance, and diabetes mellitus type 2. Can J Gastroenterol Hepatol. 2021;2021:1-9. https://doi.org/10.1155/2021/6613827
10. Finelli C, Tarantino G. What is the role of adiponectin in obesity related non-alcoholic fatty liver disease? World J Gastroenterol. 2013;19(6):802-812. https://doi.org/10.3748/wjg.v19.i6.802
11. Wang M, Wuyun Q, Lian D, Xu G, Zhu G, Zhang N. Global, regional, and national burden of nonalcoholic fatty liver disease (MASLD) among people aged 15 to 49 years from 1990 to 2021, with projections to 2050: a systematic analysis based on the 2021 global burden of disease study. Medicine (Baltimore). 2025;104(37):1-13. https://doi.org/10.1097/md.0000000000044527
12. Ciardullo S, Vergani M, Perseghin G. Nonalcoholic fatty liver disease in patients with type 2 diabetes: screening, diagnosis, and treatment. J Clin Med. 2023;12(17):1-16. https://doi.org/10.3390/jcm12175597
13. Abdelhameed F, Kite C, Lagojda L, Dallaway A, Chatha KK, Chaggar SS, et al. Non-invasive scores and serum biomarkers for fatty liver in the era of metabolic dysfunction-associated steatotic liver disease (MASLD): a comprehensive review from MASLD to MAFLD and MASLD. Curr Obes Rep. 2024;13(3):510-531. https://doi.org/10.1007/s13679-024-00574-z
14. Mu W, Cheng XF, Liu Y, Lv QZ, Liu GL, Zhang JG, et al. Potential nexus of non-alcoholic fatty liver disease and type 2 diabetes mellitus: insulin resistance between hepatic and peripheral tissues. Front Pharmacol. 2019;9:1-10. https://doi.org/10.3389/fphar.2018.01566

15. Tacke F, Horn P, Wong VW-S, Ratziu V, Bugianesi E, Francque S, et al. EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). J Hepatol. 2024;81(3):492-542. https://doi.org/10.1016/j.jhep.2024.04.031
16. Misra S, Ke C, Srinivasan S, Goyal A, Nyriyenda MJ, Florez JC, et al. Current insights and emerging trends in early-onset type 2 diabetes. Lancet Diabetes Endocrinol. 2023;11(10):768-782. https://doi.org/10.1016/s2213-8587(23)00225-5
17. Tanase DM, Gosav EM, Costea CF, Ciocoiu M, Lacatusu CM, Maranduca MA, et al. The intricate relationship between type 2 diabetes mellitus (T2DM), insulin resistance (IR), and non-alcoholic fatty liver disease (MASLD). J Diabetes Res. 2020;2020:1-16. https://doi.org/10.1155/2020/3920196
18. Shil KK, Hasan M, Sultana N, Salam SBA, Mostafa SN, Hasanat MA. High prevalence of fatty liver in Bangladeshi adolescents and young adults with type 2 diabetes: key predictors and screening recommendations. Diabetes Metab Syndr Obes. 2025;18:2019-2027. https://doi.org/10.2147/dmso.s520806
19. Hu C, Cioana M, Saini A, Ragganandan S, Deng J, Nadarajah A, et al. The prevalence of non-alcoholic fatty liver disease in pediatric type 2 diabetes: a systematic review and meta-analysis. Front Adolesc Med. 2024;2:1-18. https://doi.org/10.3389/fradm.2024.1303375
20. Liu R, Liu R, Liu M, Tian Y, Liu J, Wang Y, et al. The inflammatory index and cytokines are associated with non-alcoholic fatty liver disease in type 2 diabetes mellitus. Front Med (Lausanne). 2025;12:1-15. https://doi.org/10.3389/fmed.2025.1659998
21. Khan F, Dsouza S, Khamis AH, Abdul F, Farooqi MH, Sulaiman F, et al. Noninvasive assessment of the severity of liver fibrosis in MASLD patients with long-standing type 2 diabetes. J Gen Intern Med. 2025;40(10):2309-2318. https://doi.org/10.1007/s11606-025-09348-2
22. Tang XX, Wu R, Chen JH, Wang FL, Zhao SL, Lu J, et al. Association between HOMA-IR and metabolic dysfunction-associated steatohepatitis in US adults with MASLD. Metab Open. 2025;1-11.
23. Beyazal Polat H, Beyazal M, Arpa M, Kızılkaya B, Ayaz T, Gündoğdu ÖL, et al. Exploring the prevalence and risk factors of MASLD in patients with newly diagnosed diabetes mellitus: a comprehensive investigation. J Clin Med. 2025;14(10):1-11. https://doi.org/10.3390/jcm14103513
24. Zhao H, Ji B, Wang X, Shi S, Sheng J, Ma X, et al. Association between SPISE and MASLD in patients with type 2 diabetes. Front Med (Lausanne). 2025;12:1-10.
25. Zhao Y, Hu D, Cheng J, Cao H, Wang X, He J, et al. Personalized MASLD and liver fibrosis risk assessment for adults: glycemic status determines optimal choice of non-invasive indices. Diabetol Metab Syndr. 2025;17(1):1-13. https://doi.org/10.1186/s13098-025-02069-w
26. Ying Y, Ji Y, Ju R, Chen J, Chen M. Association between the triglyceride-glucose index and liver fibrosis in adults with metabolism-related fatty liver disease in the United States: a cross-sectional study of NHANES 2017-2020. BMC Gastroenterol. 2025;25(1):1-10. https://doi.org/10.1186/s12876-024-03579-z

Cite this article: Khwaja Saifullah Zafar, Syed Hasan Amir, Hamid Ashraf & Anup Sharma. Prevalence of Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) in Young Type 2 Diabetes Mellitus Patients & Its Association with Insulin Resistance. *JSRR : Journal of Surgical Research & Reviews.* 2026;3(1):1-11.