e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(7); 130-135

Original Research Article

Metabolic Footprints of Fatty Liver in Type 2 Diabetes: Clinical and Biochemical Predictors from a Prospective Indian Hospital-Based Study

Kunam Nikitha[1], Lunavath Anusha[2], Sekhar Reddy M.V.[3]

[1]Associate Professor, Department of General Medicine, GSL Medical College, Rajahmundry, Andhra Pradesh, India

[2]Assistant Professor, Department of General Medicine, Prathima Relief Institution of Medical College (PRIMS), Warangal, Telangana, India

[3]Assistant Professor, Department of Endocrinology, GSL Medical College, Rajahmundry, Andhra Pradesh, India

Received: 03-05-2026 / Revised: 04-06-2026 / Accepted: 05-07-2026

Corresponding Author: Dr. Sekhar Reddy MV

Conflict of interest: Nil

Abstract:

Background: Non-alcoholic fatty liver disease (NAFLD) is commonly associated with type 2 diabetes mellitus (T2DM) and may progress silently to steatohepatitis and fibrosis. Early identification of clinical and biochemical predictors is useful for risk stratification.

Aim: To evaluate the clinical and biochemical predictors of NAFLD among patients with T2DM.

Methods: This prospective observational study was conducted at GSL Medical College, Rajahmundry, from January to May 2026. A total of 124 patients with T2DM were included. Clinical details, anthropometric measurements, glycaemic parameters, lipid profile and liver enzymes were recorded. Abdominal ultrasonography was used to diagnose NAFLD. Patients with and without NAFLD were compared, and predictors were analysed statistically.

Results: NAFLD was detected in 78 patients (62.9%), while 46 patients (37.1%) had no fatty liver. Patients with NAFLD had significantly higher BMI, waist circumference, systolic blood pressure, fasting blood glucose, postprandial blood glucose, HbA1c, triglycerides, LDL cholesterol, ALT and AST levels. Multivariate analysis identified BMI ≥25 kg/m², high-risk waist circumference, HbA1c ≥8%, triglycerides ≥150 mg/dL and ALT elevation as independent predictors.

Conclusion: NAFLD was highly prevalent among T2DM patients. Obesity markers, poor glycaemic control, hypertriglyceridaemia and ALT elevation were useful predictors for identifying high-risk diabetic patients.

Keywords: Type 2 diabetes mellitus; Non-alcoholic fatty liver disease; HbA1c; Triglycerides; Waist circumference.

DOI: 10.25258/ijcpr.18.7.21

Introduction

Non-alcoholic fatty liver disease (NAFLD), now increasingly described under metabolic dysfunction-associated steatotic liver disease terminology, is a major hepatic manifestation of insulin resistance and is highly relevant in patients with type 2 diabetes mellitus (T2DM). In India, the growing burden of obesity, central adiposity and T2DM has made NAFLD an important clinical problem, because many patients remain asymptomatic until steatohepatitis or fibrosis develops [1]. Indian data show that NAFLD among patients with T2DM is strongly associated with metabolic risk clustering, including higher body mass index, waist-height ratio, dyslipidaemia, poor glycaemic status and liver enzyme abnormalities [2]. Recent Indian studies also emphasize that fibrosis risk in diabetic fatty liver can be identified using simple biochemical and non-invasive parameters such as AST, ALT, platelet count, bilirubin, FIB-4, APRI and NAFLD fibrosis score, which are feasible in routine clinical practice [3]. Early recognition of such predictors is important for risk stratification, timely lifestyle intervention and referral for advanced liver assessment. The aim of this study was to evaluate the clinical and biochemical predictors of NAFLD among patients with T2DM.

Methods

This prospective observational study was conducted in the department of General Medicine in collaboration with the Department of Biochemistry and Radiodiagnosis, GSL Medical College, Rajahmundry, Andhra Pradesh, from January 2026 to May 2026. Adult patients diagnosed with T2DM

and attending the outpatient department or admitted to the hospital during the study period were considered for enrolment. Patients were included if they were aged ≥18 years, had a confirmed diagnosis of T2DM based on previous medical records, antidiabetic treatment history, or standard diagnostic criteria, and were willing to provide informed consent. Patients with significant alcohol intake, known chronic liver disease, viral hepatitis, autoimmune liver disease, drug-induced liver injury, pregnancy, malignancy, chronic renal failure, or those receiving hepatotoxic drugs were excluded. Ethical clearance was obtained from the Institutional Ethics Committee before initiation of the study, and written informed consent was obtained from all participants.

A detailed clinical evaluation was performed for each participant using a predesigned study proforma. Demographic details such as age, gender, residence and occupation were recorded. Clinical history included duration of diabetes mellitus, treatment details, dietary habits, physical activity, family history of diabetes, hypertension, dyslipidaemia and other comorbidities. Anthropometric measurements were recorded using standard techniques. Height was measured in centimetres, weight in kilograms, and body mass index was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured at the midpoint between the lower margin of the last rib and the iliac crest. Blood pressure was measured in the sitting position after adequate rest using a calibrated sphygmomanometer. Clinical examination was carried out to identify features such as central obesity, hepatomegaly, acanthosis nigricans and other relevant systemic findings. Based on abdominal ultrasonography, participants were categorized into those with non-alcoholic fatty liver disease and those without fatty liver.

Venous blood samples were collected under aseptic precautions after overnight fasting. Biochemical parameters included fasting blood glucose, postprandial blood glucose, glycated haemoglobin, serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, serum aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, total bilirubin, serum albumin, urea and creatinine. Ultrasonography of the abdomen was performed by an experienced radiologist using standard criteria for hepatic steatosis, including increased hepatic echogenicity, hepatorenal contrast, vascular blurring and poor visualization of the diaphragm. Data were entered into Microsoft Excel and analysed using SPSS software version 22. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. Clinical and biochemical variables were compared between patients with and without NAFLD using Student's t-test or Mann–Whitney U test for continuous variables and chi-square test or Fisher's exact test for categorical variables. Logistic regression analysis was applied to identify independent predictors of NAFLD among patients with T2DM. A p value <0.05 was considered statistically significant.

Results

A total of 124 patients with T2DM were included. Of these, 78 (62.9%) had ultrasonographic evidence of NAFLD, while 46 members (37.1%) did not have fatty liver (Table 1). The mean age of the study population was 53.42 ± 9.86 years, and NAFLD was more frequent in the 51–60 years age group (Table 1). Male predominance was observed in both groups, but gender distribution was not statistically significant (Table 1). Patients with NAFLD had significantly higher mean BMI, waist circumference and systolic blood pressure compared with those without NAFLD (Table 2). Duration of diabetes >5 years was also more common among patients with NAFLD and showed a statistically significant association (Table 1). Biochemical analysis showed that patients with NAFLD had significantly higher fasting blood glucose, postprandial blood glucose and HbA1c levels, suggesting poorer glycaemic control (Table 3). Lipid profile abnormalities were also more marked in the NAFLD group, particularly elevated triglycerides, LDL cholesterol and reduced HDL cholesterol (Table 3). Liver enzyme levels were significantly higher among patients with NAFLD, especially ALT and AST, although most values were within the mild elevation range (Table 3). On multivariate logistic regression, BMI ≥25 kg/m², waist circumference above the risk cut-off, HbA1c ≥8%, triglycerides ≥150 mg/dL and ALT elevation were identified as significant independent predictors of NAFLD among patients with type 2 diabetes mellitus (Table 4). These findings indicate that clinical obesity markers, poor glycaemic control, dyslipidaemia and liver enzyme abnormalities were important predictors of fatty liver in diabetic patients (Tables 2–4).

Table 1: Baseline clinical characteristics of the study members					
Variable	**NAFLD**			**Test value**	**p value**
	Present (n=78)	**Absent (n=46)**	**Total (n=124)**		
Mean age, years	54.18 ± 9.42	52.13 ± 10.51	53.42 ± 9.86	t=1.13	0.26
Age ≤50 years	28 (35.9%)	21 (45.7%)	49 (39.5%)	χ^2=1.17	0.279
Age >50 years	50 (64.1%)	25 (54.3%)	75 (60.5%)		
Male	45 (57.7%)	25 (54.3%)	70 (56.5%)	χ^2=0.13	0.716
Female	33 (42.3%)	21 (45.7%)	54 (43.5%)		
Duration of diabetes ≤5 years	29 (37.2%)	27 (58.7%)	56 (45.2%)	χ^2=5.45	0.02
Duration of diabetes >5 years	49 (62.8%)	19 (41.3%)	68 (54.8%)		
Hypertension present	42 (53.8%)	16 (34.8%)	58 (46.8%)	χ^2=4.20	0.04

Table 2: Anthropometric and clinical predictors among the study members				
Variable	**NAFLD**		**Test value**	**p value**
	Present	**Absent**		
BMI, kg/m²	27.62 ± 3.84	24.91 ± 3.12	t=4.05	<0.001
BMI ≥25 kg/m²	56 (71.8%)	18 (39.1%)	χ^2=12.89	<0.001
Waist circumference, cm	96.48 ± 8.72	89.36 ± 7.94	t=4.55	<0.001
High-risk waist circumference	61 (78.2%)	22 (47.8%)	χ^2=11.98	0.001
Systolic BP, mmHg	136.42 ± 14.28	129.13 ± 12.84	t=2.85	0.005
Diastolic BP, mmHg	84.26 ± 8.92	81.74 ± 7.86	t=1.58	0.116

Table 3: Biochemical parameters among the study members				
Parameter	**NAFLD**		**Test value**	**p value**
	Present	**Absent**		
Fasting blood glucose, mg/dL	158.64 ± 38.72	137.26 ± 31.45	t=3.18	0.002
Postprandial blood glucose, mg/dL	236.82 ± 58.41	201.74 ± 49.36	t=3.42	0.001
HbA1c, %	8.42 ± 1.36	7.61 ± 1.18	t=3.36	0.001
Total cholesterol, mg/dL	196.24 ± 42.15	181.36 ± 36.82	t=1.99	0.049
Triglycerides, mg/dL	184.72 ± 56.38	142.63 ± 41.29	t=4.40	<0.001
LDL cholesterol, mg/dL	121.45 ± 32.18	107.28 ± 28.46	t=2.46	0.015
HDL cholesterol, mg/dL	39.26 ± 8.14	44.18 ± 7.96	t=3.28	0.001
ALT, IU/L	48.36 ± 19.42	32.74 ± 13.86	t=4.80	<0.001
AST, IU/L	42.18 ± 16.25	31.86 ± 12.94	t=3.67	<0.001

Table 4: Multivariate logistic regression analysis for predictors of NAFLD			
Predictor variable	**Adjusted odds ratio**	**95% CI**	**p value**
Age >50 years	1.34	0.61–2.94	0.462
Duration of diabetes >5 years	2.12	1.01–4.46	0.047
BMI ≥25 kg/m²	3.48	1.55–7.82	0.003
High-risk waist circumference	3.21	1.38–7.45	0.007
HbA1c ≥8%	2.76	1.25–6.09	0.012
Triglycerides ≥150 mg/dL	3.64	1.62–8.16	0.002
ALT elevation	2.89	1.24–6.71	0.014
Hypertension	1.76	0.81–3.82	0.152

Discussion:

In the present prospective study involving 124 patients with T2DM, ultrasonographic evidence of NAFLD observed in 78 patients, giving a prevalence of 62.9%. This finding confirms that NAFLD is highly common among diabetic patients and supports the view that fatty liver should be considered an important hepatic component of the metabolic syndrome in routine diabetic care. Indian studies have reported a high burden of NAFLD among patients with diabetes, with prevalence estimates commonly exceeding 50%, particularly in hospital-based and high-risk metabolic populations [1, 4]. The pooled Indian evidence also indicates that NAFLD prevalence is substantially higher in diabetic, obese and metabolically unhealthy individuals than in the general population [5]. The present prevalence is comparable to recent Indian reports, including studies where NAFLD or hepatic steatosis among T2DM patients ranged from nearly 57% to more than 70%, depending on the diagnostic modality and population profile [6, 7]. This reinforces the need for active screening, as many patients with diabetic fatty liver remain clinically

silent until fibrosis or advanced liver disease develops [8].

The higher frequency of NAFLD in the 51–60 years age group and in patients with longer duration of diabetes suggests cumulative metabolic exposure as an important contributor to hepatic steatosis. Although age and gender were not independent predictors in the present regression model, duration of diabetes more than five years showed a significant association with NAFLD. This agrees with Indian observations that longer diabetes duration increases the probability of hepatic steatosis and fibrosis risk, probably through prolonged insulin resistance, chronic hyperglycaemia, dyslipidaemia and adipose tissue dysfunction [9]. Mondal et al. observed that increasing age and duration of diabetes were associated with higher FIB-4-based fibrosis risk among diabetic patients attending a primary healthcare NCD clinic in North India [10]. Similarly, studies using elastography in Indian diabetic cohorts have shown that fibrosis risk is not rare and may remain undetected when only routine liver enzymes are used [8, 11]. Therefore, diabetic patients with longer disease duration require more careful hepatic risk assessment, even when symptoms are absent or liver function tests show only mild abnormalities.

Anthropometric markers were strong predictors of NAFLD in the present study. Patients with NAFLD had significantly higher BMI and waist circumference, and both BMI ≥25 kg/m² and high-risk waist circumference remained independent predictors in multivariate analysis. This finding is biologically plausible because central obesity reflects visceral adiposity, which promotes hepatic free fatty acid flux, insulin resistance, inflammatory cytokine release and de novo lipogenesis. Indian patients are particularly vulnerable because metabolic complications may occur at lower BMI levels compared with Western populations [12]. Mukherjee et al. reported that NAFLD was common among newly diagnosed T2DM patients and was especially associated with obesity markers and high fat intake [13]. Panikar et al. also showed that steatosis and fibrosis stages among Indian NAFLD patients with T2DM were associated with metabolic risk factors [7]. However, Indian literature also emphasizes that lean NAFLD is clinically relevant, and lean Indian patients may still develop significant liver disease severity despite having fewer classical metabolic risk factors [14]. Thus, while obesity remains a major predictor, normal BMI should not completely exclude NAFLD risk in Indian diabetic patients.

Poor glycaemic control was another important finding of the present study. Patients with NAFLD had significantly higher fasting blood glucose, postprandial blood glucose and HbA1c levels than those without fatty liver, and HbA1c ≥8% independently predicted NAFLD. This indicates that hepatic steatosis in diabetic patients is closely linked with inadequate metabolic control. Hyperglycaemia aggravates hepatic lipogenesis and oxidative stress, while hepatic fat accumulation further worsens insulin resistance, creating a bidirectional relationship between NAFLD and T2DM [15]. Pramanik et al. emphasized that NAFLD affects more than half of individuals living with T2DM and that diabetes increases the risk of progressive steatohepatitis and fibrosis [15]. Kumar et al. reported that HbA1c and blood glucose levels correlated significantly with fatty liver grades in patients with T2DM and NAFLD assessed by shear wave elastography [11]. These findings support the present observation that glycaemic control is not only a marker of diabetes status but also an important clinical signal for fatty liver risk. Therefore, liver evaluation should be integrated into diabetic patients with persistently raised HbA1c.

Dyslipidaemia was strongly associated with NAFLD in this study, particularly elevated triglycerides, increased LDL cholesterol and reduced HDL cholesterol. Among these, triglycerides ≥150 mg/dL emerged as an independent predictor of NAFLD. This agrees with the known pathophysiology of diabetic NAFLD, where insulin resistance increases adipose lipolysis, hepatic triglyceride synthesis and very-low-density lipoprotein production. Ray and Agarwala showed that triglycerides, BMI, LDL cholesterol and HbA1c were important metabolic predictors linked to NAFLD severity parameters in Indian subjects [16]. Rajesh et al. also demonstrated improvement in triglycerides, liver enzymes, HbA1c and fibrosis parameters after saroglitazar therapy in South Indian patients with NAFLD and diabetic dyslipidaemia, indirectly supporting the central role of dyslipidaemia in diabetic fatty liver [17]. In the present study, reduced HDL cholesterol among NAFLD patients further reflects an atherogenic lipid profile. This is clinically important because NAFLD in diabetes is not only a liver disease but also a marker of cardiovascular and systemic metabolic risk [18]. Hence, lipid abnormalities should be interpreted as predictors of both hepatic and cardiovascular complications.

Liver enzyme abnormalities, particularly ALT and AST elevation, were significantly higher in patients with NAFLD, and ALT elevation remained an independent predictor in the regression model. However, most enzyme elevations were mild, emphasizing that normal or near-normal aminotransferases cannot exclude NAFLD or fibrosis. INASL guidance recommends that risk assessment should not depend only on liver enzymes, and non-invasive tools such as FIB-4, NAFLD fibrosis score, transient elastography and controlled attenuation parameter may be used for

further stratification where feasible [1]. Recent Indian studies using transient elastography and shear wave elastography have shown a meaningful burden of clinically significant fibrosis among diabetic patients, even in outpatient settings [8, 11]. The present findings therefore support a practical screening approach in which diabetic patients with obesity, high waist circumference, poor glycaemic control, hypertriglyceridaemia and ALT elevation are prioritized for ultrasound and non-invasive fibrosis assessment. The study is limited by single-centre design, ultrasound-based diagnosis and absence of elastography or histology, but it provides useful evidence that simple clinical and biochemical markers can help identify diabetic patients at high risk for NAFLD in routine practice.

Conclusion

In this prospective study, non-alcoholic fatty liver disease was identified in a substantial proportion of patients with type 2 diabetes mellitus. Patients with NAFLD showed higher BMI, increased waist circumference, longer duration of diabetes, poorer glycaemic control, dyslipidaemia and mild liver enzyme elevation compared with those without fatty liver. On multivariate analysis, BMI ≥25 kg/m², high-risk waist circumference, HbA1c ≥8%, triglycerides ≥150 mg/dL and ALT elevation were significant independent predictors. These findings suggest that simple clinical and biochemical parameters can help identify diabetic patients at increased risk of NAFLD and support routine screening in high-risk T2DM patients.

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