Clinical, Biochemical, and Ultrasonographic Evaluation of Hepatic Dysfunction in Type 2 Diabetes Mellitus

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Abstract

Background: Type 2 diabetes mellitus (T2DM) and hepatic steatosis frequently coexist; metabolic dysfunction-associated fatty liver disease (MAFLD), the renamed successor to non-alcoholic fatty liver disease (NAFLD), is now the most common hepatic complication of T2DM. Indian data integrating clinical, biochemical, and ultrasonographic hepatic findings in T2DM remain limited.

Objectives: To determine the frequency of deranged liver enzymes and ultrasonographic MAFLD in T2DM, and to correlate these with glycaemic control and lipid profile.

Methods: A hospital-based, cross-sectional study of 100 T2DM patients (aged over 30 years) at a tertiary centre in Bengaluru, India, over 18 months assessed anthropometric, glycaemic, lipid, liver enzyme, renal, and ultrasonographic parameters. MAFLD was diagnosed per 2020 consensus criteria; fibrosis risk was estimated using the BARD score. One-way ANOVA, chi-square, and Pearson correlation were used.

Results: Mean age peaked at 50–59 years (32%); sex distribution was near-equal (male 52%). Obesity was present in 83%; poor glycaemic control (HbA1c ≥7%) in 69%. Dyslipidaemia was common (LDL elevated in 88%, HDL low in 79%). AST was elevated in 58% and ALT in 64%. Ultrasonographic MAFLD was present in 41%. HbA1c was higher in MAFLD than non-MAFLD participants (9.21±2.11% vs 7.27±0.98%; $p<0.001$), and correlated with ALT, uric acid, and triglycerides (all $p<0.05$). High BARD fibrosis risk was found in 85%.

Conclusions: Hepatic dysfunction is highly prevalent in T2DM and closely linked to poor glycaemic control, obesity, and dyslipidaemia, supporting routine liver screening in diabetes care.

Keywords: glycated haemoglobin; hepatic ultrasonography; liver function tests; metabolic-associated fatty liver disease; type 2 diabetes mellitus

Introduction

Diabetes mellitus comprises a group of metabolic disorders characterised by chronic hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. The global burden of diabetes has risen sharply over the past four decades, with prevalence nearly doubling between 1980 and 2014 [1,2]. In India, the diabetic population was projected to rise from 31.7 million in 2000 to 79.4 million by 2030, and India continues to contribute one of the largest absolute burdens of type 2 diabetes mellitus (T2DM) globally [3,4].

Non-alcoholic fatty liver disease (NAFLD), first described in 1986, has emerged as the most common chronic liver disease worldwide, encompassing a spectrum from simple steatosis to steatohepatitis, fibrosis, and cirrhosis [5]. An international consensus panel proposed renaming this condition metabolic dysfunction-associated fatty liver disease (MAFLD), defined by hepatic steatosis with T2DM, obesity, or other features of metabolic syndrome [6,7]. Global MAFLD/NAFLD prevalence is approximately 25% in the general population, rising to 55–70% among individuals with T2DM [8,9], reflecting a bidirectional relationship [10].

The liver is central to glucose and lipid homeostasis and is particularly vulnerable to insulin resistance and chronic hyperglycaemia [9,10]. Hyperuricaemia and dyslipidaemia have been implicated as additional

drivers of hepatic steatosis [11–13], and coexistence of MAFLD with T2DM is linked to higher cardiovascular, renal, and liver-related morbidity [14,15].

Several studies from India and neighbouring South Asian countries have reported a high burden of deranged liver function tests and ultrasonographic fatty liver among patients with T2DM [16–27]. The present study was undertaken to evaluate liver function test abnormalities, assess ultrasonographic hepatic steatosis, and examine their associations with MAFLD status and glycaemic control in a single Indian T2DM cohort.

Materials and Methods

Study Design and Setting

This was a hospital-based, descriptive, cross-sectional observational study conducted in the Department of General Medicine at Rajarajeswari Medical College and Hospital (RRMCH), Bengaluru, India, among inpatients and outpatients with T2DM, over 18 months. Ethics approval: RRMCH-IEC/76/2024, dated March 12, 2024. Written informed consent was obtained from all participants.

Participants

Patients aged more than 30 years with a pre-existing diagnosis of T2DM (WHO criteria) and BMI >18.5 kg/m² were eligible. Exclusion criteria: type 1 or gestational diabetes, BMI <18.5 kg/m², age <30 years, thyroid disorders, chronic liver disease, alcohol use disorder, hepatitis, chronic kidney disease, pre-existing dyslipidaemia, statin therapy, critical illness, or non-consent.

Sample Size

Sample size was calculated for proportion estimation at 95% confidence interval (minimum n=75). A total of 100 consecutive eligible participants were enrolled.

Data Collection

Each participant underwent comprehensive evaluation: detailed history, physical examination, and anthropometric measurement (height, weight, BMI classified per South-East Asian BMI classification). Laboratory investigations included fasting and postprandial blood glucose, HbA1c, fasting lipid profile, liver function tests (AST, ALT), and renal function tests (urea, creatinine, uric acid). Abdominal ultrasonography graded hepatic steatosis as normal, Grade 1, or Grade 2. 12-lead ECG and 2D echocardiography assessed cardiovascular status.

Definitions

MAFLD was diagnosed in participants with ultrasonographic hepatic steatosis in the presence of T2DM, per the 2020 international consensus criteria [6]. Cut-offs: HbA1c ≥7% (poor glycaemic control); AST and ALT >35 U/L (enzyme derangement); triglycerides ≥150 mg/dL, LDL ≥70 mg/dL (elevated); HDL <40 mg/dL (low); urea >45 mg/dL, creatinine >1.2 mg/dL, uric acid >6.5 mg/dL (elevated). BARD score: BMI ≥28 kg/m² (1 pt) + AST/ALT ratio ≥0.8 (2 pts) + T2DM (1 pt); score 2–4 = high risk.

Statistical Analysis

Continuous variables: mean ± SD. Categorical variables: frequency (n) and percentage (%). One-way ANOVA (F-statistic with degrees of freedom) compared mean HbA1c across MAFLD status and

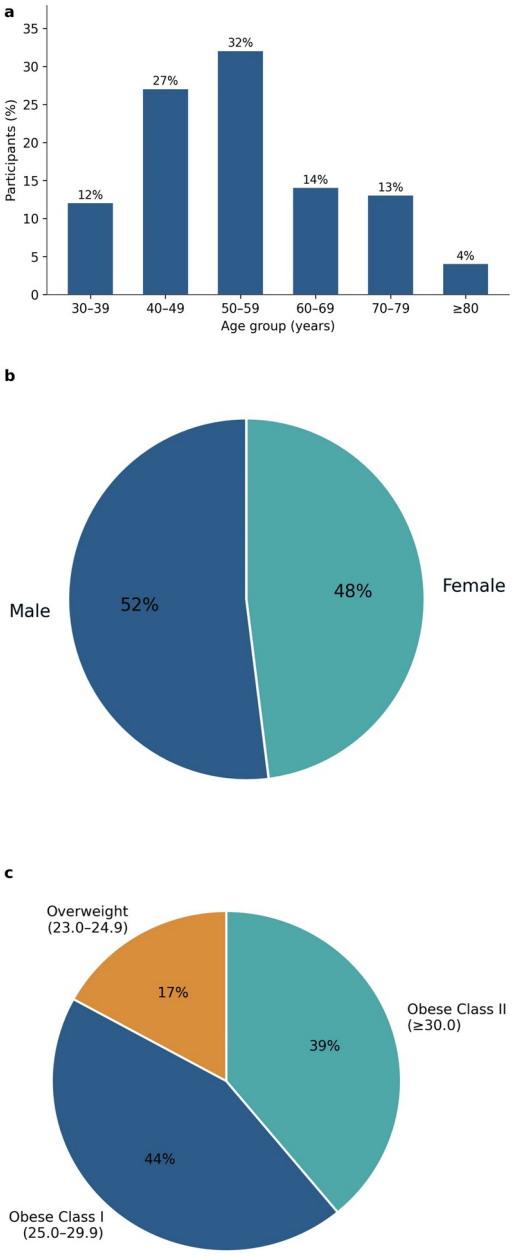
ultrasonographic grades. Chi-square test (χ^2, degrees of freedom reported) assessed associations between categorical variables. Pearson's correlation coefficient (r) examined relationships between HbA1c and biochemical parameters. $p<0.05$ (two-tailed) was considered statistically significant. IBM SPSS Statistics v22.0 (IBM Corp., Armonk, NY, USA) was used.

Results

Of the 100 participants enrolled, demographic, anthropometric, and comorbidity characteristics are summarised in Table 1 and Figure 1.

Table 1. Demographic and Anthropometric Characteristics of Study Participants (N = 100)

Characteristic	Value
Age group, years	
30–39	12 (12.0%)
40–49	27 (27.0%)
50–59	32 (32.0%)
60–69	14 (14.0%)
70–79	13 (13.0%)
≥80	4 (4.0%)
Sex	
Male	52 (52.0%)
Female	48 (48.0%)
BMI category (South-East Asian classification)	
Overweight (23.0–24.9 kg/m²)	17 (17.0%)
Obese Class I (25.0–29.9 kg/m²)	44 (44.0%)
Obese Class II (≥30.0 kg/m²)	39 (39.0%)
Comorbidity pattern	
T2DM alone	57 (57.0%)
T2DM + hypertension	41 (41.0%)
T2DM + hypertension + CVA	2 (2.0%)
Anthropometry (mean ± SD)	
Height, cm	164.12 ± 6.33
Weight, kg	77.85 ± 10.58
BMI, kg/m²	28.88 ± 3.40


a
Participants (%)
0
5
10
15
20
25
30
35
12%
27%
32%
14%
13%
4%
30–39
40–49
50–59
60–69
70–79
≥80
Age group (years)
b
Male
52%
48%
Female
c
Overweight
(23.0–24.9)
17%
Obese Class II
(≥30.0)
39%
44%
Obese Class I
(25.0–29.9)

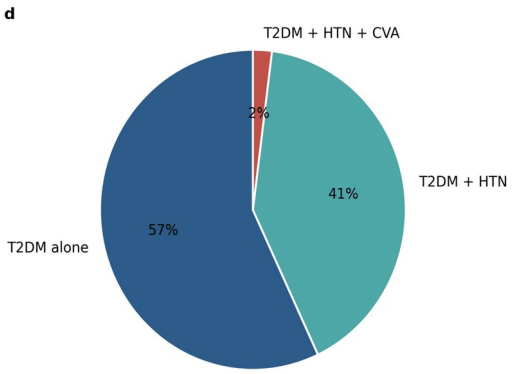


Figure 1. Baseline characteristics of study participants (N=100). (a) Age distribution; (b) Sex distribution; (c) BMI category distribution (South-East Asian classification); (d) Comorbidity pattern. Corresponding counts (n) and percentages are provided in Table 1 and the text.

The largest proportion of participants (n=32, 32%) were aged 50–59 years, followed by n=27 (27%) aged 40–49 years (Figure 1a). Sex distribution was near-equal: n=52 (52%) male and n=48 (48%) female (Figure 1b). Obesity (Class I or II) was present in n=83 (83%): Class I n=44 (44%) and Class II n=39 (39%); none were underweight or of normal BMI (Figure 1c). T2DM alone was recorded in n=57 (57%), T2DM with hypertension in n=41 (41%), and T2DM with hypertension and cerebrovascular accident in n=2 (2%) (Figure 1d).

Glycaemic and lipid parameters are shown in Table 2 and Figure 2.

Table 2. Glycaemic and Lipid Profile of Study Participants (N = 100)

Parameter	Mean ± SD	Cut-off	n/N Deranged	% Deranged
FBS, mg/dL	164.13 ± 59.49	—	—	—
PPBS, mg/dL	243.97 ± 66.05	—	—	—
HbA1c, %	8.06 ± 1.81	≥ 7	69/100	69.0%
Triglycerides, mg/dL	169.47 ± 66.98	≥ 150	54/100	54.0%
LDL, mg/dL	105.02 ± 35.79	≥ 70	88/100	88.0%
HDL, mg/dL	35.36 ± 7.52	< 40	79/100	79.0%

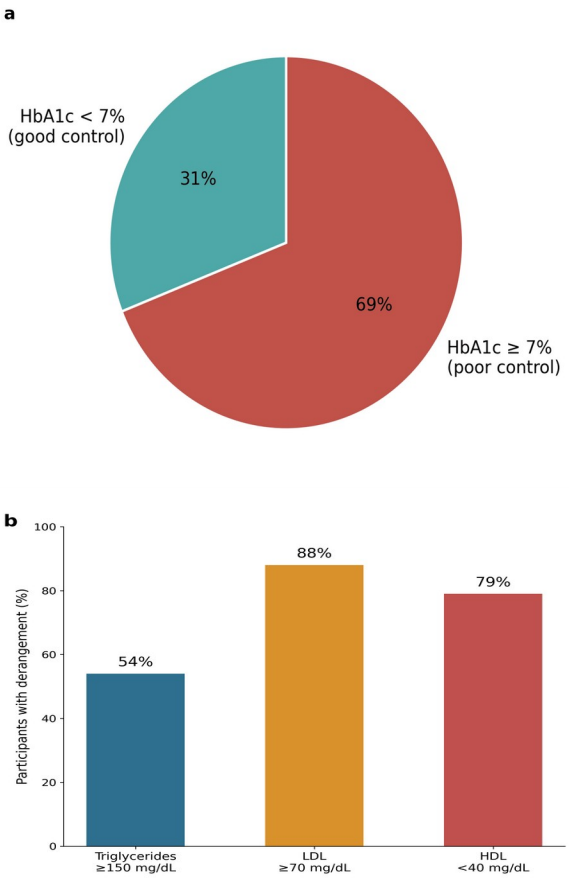


Figure 2. Glycaemic and lipid parameters. (a) Glycaemic control: HbA1c <7% n=31 (31%); HbA1c ≥7% n=69 (69%). (b) Lipid derangement: triglycerides ≥150 mg/dL n=54 (54%); LDL ≥70 mg/dL n=88 (88%); HDL <40 mg/dL n=79 (79%).

Liver enzyme, renal function, and uric acid parameters are shown in Table 3 and Figure 3.

Table 3. Liver Enzyme, Renal, and Uric Acid Parameters (N = 100)

Parameter	Mean ± SD	Cut-off	n/N Elevated	% Elevated
AST, U/L	38.67 ± 13.09	> 35	58/100	58.0%
ALT, U/L	45.45 ± 15.11	> 35	64/100	64.0%
Urea, mg/dL	30.86 ± 8.77	> 45	0/100	0.0%
Creatinine, mg/dL	0.99 ± 0.16	> 1.2	11/100	11.0%
Uric acid, mg/dL	6.47 ± 5.41	> 6.5	31/100	31.0%

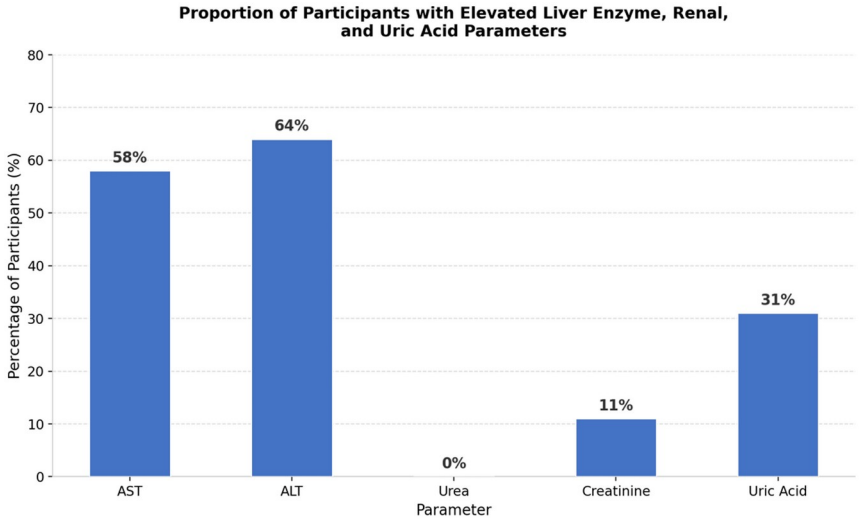


Figure 3. Proportion of participants with elevated liver enzyme, renal, and uric acid parameters. Values shown as n/100 (%): AST n=58 (58%); ALT n=64 (64%); urea n=0 (0%); creatinine n=11 (11%); uric acid n=31 (31%).

Liver enzymes were elevated in the majority: AST >35 U/L in n=58 (58%; mean 38.67±13.09 U/L) and ALT >35 U/L in n=64 (64%; mean 45.45±15.11 U/L). Renal parameters were largely preserved: normal urea in all participants (n=0 elevated, 0%; mean 30.86±8.77 mg/dL); elevated creatinine in n=11 (11%; mean 0.99±0.16 mg/dL); elevated uric acid in n=31 (31%; mean 6.47±5.41 mg/dL).

Table 4. Ultrasonographic Liver Findings, MAFLD Distribution, and BARD Fibrosis Risk (N = 100)

USG / Risk Category	n	%
Ultrasonographic liver finding		
Normal liver	59	59.0%
Grade 1 steatosis	34	34.0%
Grade 2 steatosis	7	7.0%
MAFLD status		
Present	41	41.0%
Absent	59	59.0%
BARD fibrosis risk category		
High risk (score 2–4)	85	85.0%
Low risk (score 0–1)	15	15.0%
Mean BARD score (± SD)	2.88 ± 1.08	

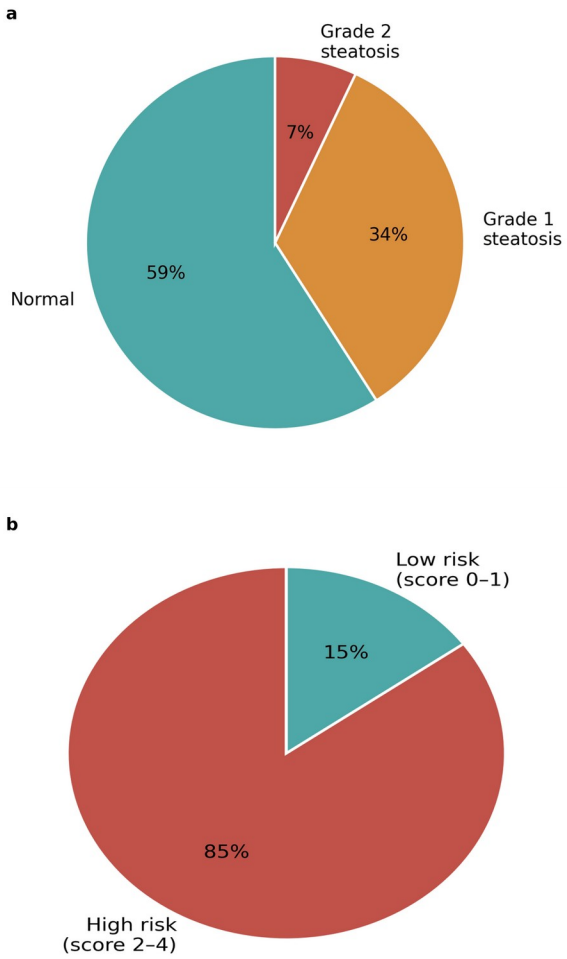


Figure 4. Ultrasonographic and fibrosis risk findings. (a) Distribution of ultrasonographic fatty liver grade: Normal n=59 (59%), Grade 1 steatosis n=34 (34%), Grade 2 steatosis n=7 (7%). (b) BARD fibrosis risk category: High risk (score 2–4) n=85 (85%); Low risk (score 0–1) n=15 (15%).

Abdominal ultrasonography demonstrated a normal liver in n=59 (59%), Grade 1 hepatic steatosis in n=34 (34%), and Grade 2 steatosis in n=7 (7%); overall, n=41 (41%) met diagnostic criteria for MAFLD. The association between ultrasonographic fatty liver grade and comorbidity pattern did not reach statistical significance (chi-square test: χ^2=7.445, df=4, p=0.114). Using the BARD score, n=85 (85%) were classified as high risk for advanced hepatic fibrosis (mean score 2.88±1.08).

Table 5. HbA1c and BMI According to Ultrasonographic Fatty Liver Grade (One-Way ANOVA)

USG Grade	n	Mean HbA1c (%) ± SD	Mean BMI (kg/m²) ± SD	p-value
Normal	59	7.27 ± 0.98	28.50 ± 3.55	
Grade 1	34	8.99 ± 1.87	29.14 ± 3.29	
Grade 2	7	10.26 ± 3.01	30.77 ± 1.73	
Overall (one-way ANOVA)	100	—	—	< 0.001

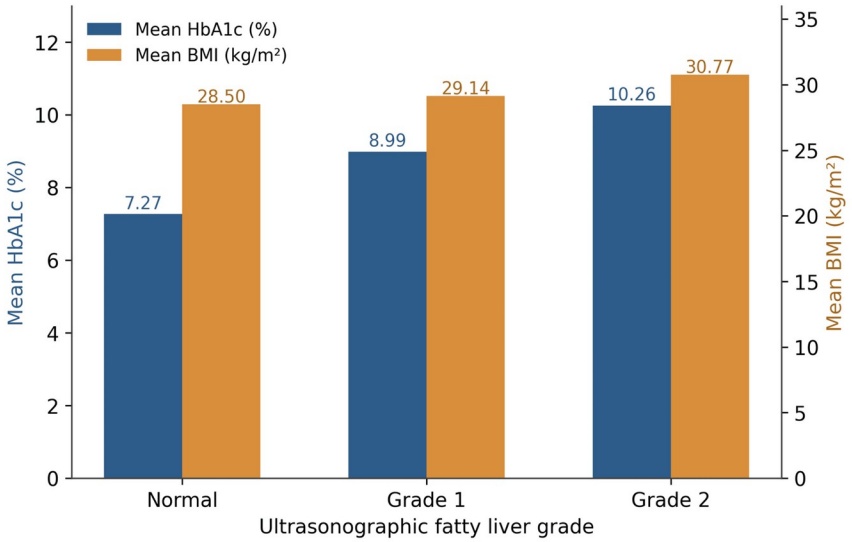


Figure 5. Mean HbA1c (%) and mean BMI (kg/m²) by ultrasonographic fatty liver grade. Group sizes: Normal n=59, Grade 1 n=34, Grade 2 n=7. One-way ANOVA p<0.001 for both parameters.

Mean HbA1c was significantly higher in participants with MAFLD (n=41) than without (n=59) (9.21±2.11% vs 7.27±0.98%; one-way ANOVA, F(1,98)=32.6, p<0.001) and increased progressively: Normal 7.27±0.98%, Grade 1 8.99±1.87%, Grade 2 10.26±3.01% (one-way ANOVA, p<0.001). BMI followed a similar pattern (Normal 28.50±3.55, Grade 1 29.14±3.29, Grade 2 30.77±1.73 kg/m²; p<0.001). MAFLD was strongly associated with elevated triglycerides (chi-square test: χ^2=18.3, df=1, p<0.001).

Table 6. Pearson Correlation of HbA1c with Biochemical Parameters (N = 100)

Parameter	Pearson r	p-value	Significance
AST	0.102	0.313	NS
ALT	0.261	0.009	*
Urea	0.072	0.479	NS
Creatinine	0.102	0.314	NS
Uric acid	0.232	0.020	*
Triglycerides	0.303	0.002	*
HDL	0.020	0.847	NS
LDL	0.164	0.102	NS

NS = not significant (p ≥ 0.05). * = statistically significant (p < 0.05). Statistical test: Pearson's correlation coefficient (r).

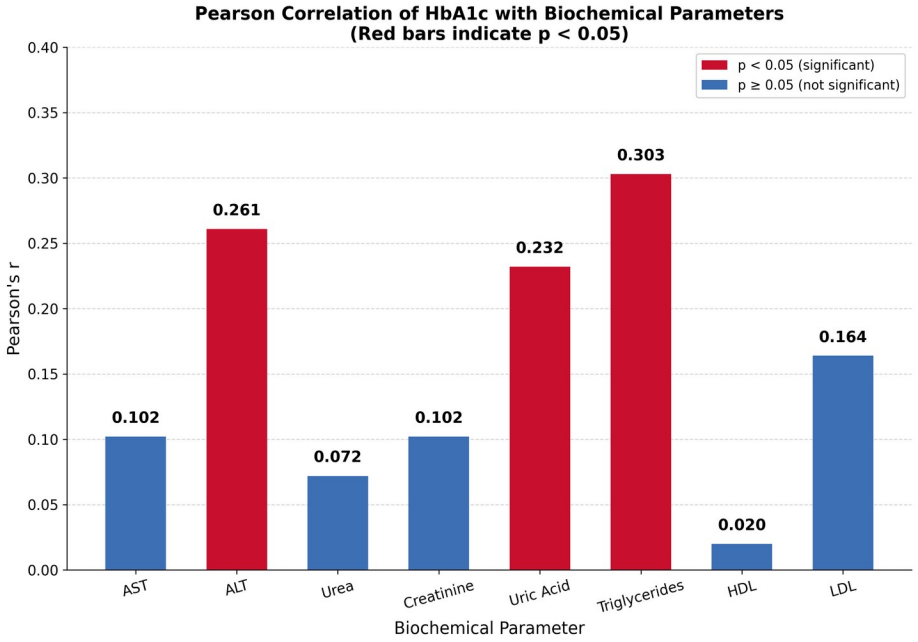


Figure 6. Pearson correlation of HbA1c with biochemical parameters (N=100). Red bars indicate statistically significant correlations (p<0.05): ALT r=0.261 (p=0.009), uric acid r=0.232 (p=0.020), triglycerides r=0.303 (p=0.002).

HbA1c correlated significantly and positively with ALT (Pearson r=0.261, p=0.009), uric acid (r=0.232, p=0.020), and triglycerides (r=0.303, p=0.002), but not with AST (r=0.102, p=0.313), urea (r=0.072, p=0.479), creatinine (r=0.102, p=0.314), HDL (r=0.020, p=0.847), or LDL (r=0.164, p=0.102). Among the n=41 participants with MAFLD, AST was deranged in n=22 (53.7%; mean 39.8±11.9 U/L) and ALT in n=28 (68.3%; mean 47.5±15.1 U/L); at least one enzyme was deranged in n=30 (73.2%) of this subgroup.

Discussion

A recent meta-analysis of 33 observational studies (>500,000 individuals) demonstrated that NAFLD more than doubles the risk of incident T2DM, with risk escalating alongside fibrosis severity [14]. The present study extends this bidirectional relationship by characterising the clinical, biochemical, and ultrasonographic hepatic profile of an Indian T2DM cohort.

The age and sex distribution — peak 50–59 years (n=32, 32%), near-equal male-to-female ratio (n=52:48, 52%:48%) — is broadly consistent with other regional cohorts. Thambiah et al. reported an older median age (>60 years in 50.3% of 300 T2DM patients) with similar HbA1c profile (median 8.0%) [16], while Or Rashid et al. described a younger mean age (54 years) and closely matching HbA1c (7.79±1.68%) [17]. The obesity burden in the present cohort (n=83 [83%] Class I/II, mean BMI 28.88±3.40 kg/m²) was higher than Thambiah et al. (median 26.5 kg/m²) [16] and Sinha and Bankura (mean 27.42±5.23 kg/m²) [19].

Liver enzyme derangement was frequent (AST n=58 [58%], ALT n=64 [64%]), higher than Or Rashid et al. (AST n=5 [5%], ALT n=31 [31%]) [17] but comparable to Singh et al. (AST 59.3%, ALT 52.6%) [18]. The positive Pearson correlations between HbA1c and ALT (r=0.261, p=0.009), uric acid (r=0.232, p=0.020), and triglycerides (r=0.303, p=0.002) mirror mechanistic data linking hyperglycaemia-driven insulin resistance, lipotoxicity, and hyperuricaemia to hepatic injury [11–13].

The MAFLD prevalence of n=41 (41%) by ultrasonography falls within the reported regional range: Or Rashid et al. and Sinha and Bankura each reported approximately 57% [17,19]; Muthiah et al. found 69% in a large NHANES-derived cohort [22]; Karande et al. found 28% [25]. The progressive rise in HbA1c and BMI across ultrasonographic grades, together with the strong association between MAFLD and hypertriglyceridaemia (chi-square test: $\chi^2=18.3$, df=1, $p<0.001$), supports a model in which poor glycaemic control and adiposity act synergistically to drive hepatic steatosis [9,10,23].

Notably, n=85 (85%) of participants were classified as high BARD fibrosis risk — a finding that reinforces the case for incorporating ultrasonography, liver enzymes, and validated fibrosis scores into standard diabetes care pathways [24].

Limitations: Single-centre, cross-sectional study with a modest sample (n=100); results may not generalise broadly. Hepatic steatosis was assessed by ultrasonography alone (limited sensitivity for mild steatosis; cannot quantify fibrosis); transient elastography or histological confirmation were not performed. The cross-sectional design precludes causal inference. Lifestyle factors and use of hepatotoxic medications were not systematically captured; longitudinal follow-up was not undertaken.

Conclusions

Hepatic dysfunction — manifested as deranged liver enzymes and ultrasonographic steatosis — is highly prevalent among patients with type 2 diabetes mellitus and closely associated with poor glycaemic control, obesity, dyslipidaemia, and hyperuricaemia. The strong Pearson correlations between HbA1c and hepatic/metabolic parameters (ALT r=0.261, p=0.009; triglycerides r=0.303, p=0.002), together with n=85 (85%) at elevated BARD-predicted fibrosis risk, underscore the need for routine biochemical and ultrasonographic liver screening as part of standard diabetes care. Larger, multi-centre, longitudinal studies incorporating advanced non-invasive fibrosis assessment are warranted.

Declarations

Ethics Approval and Consent to Participate: This study was approved by the Institutional Ethics Committee, Rajarajeswari Medical College and Hospital (approval no. RRMCH-IEC/76/2024, dated March 12, 2024). Written informed consent was obtained from all participants prior to enrolment.

Conflict of Interest: The authors declare no conflict of interest.

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Data Availability: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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