

Research Article

Association between Glycemic Control and Hepatic Steatosis Severity in Type 2 Diabetic Patients

Shaista Mujeeb¹, Mumtaz Ali Chhutto², Mujeeb-ur-Rehman Abro³, Kamran Ali Memon⁴, Saad Ali Ansari⁵, Barkat Ali Dahar⁶

¹Lecturer, Department of Biochemistry, Shaheed Mohtarma Benazir Bhutto Medical University, Larkana, Pakistan.

²Associate Professor, Department of Medicine Unit-2, Chandka Medical College Hospital, Shaheed Mohtarma Benazir Bhutto Medical University, Larkana, Pakistan.

³Assistant Professor, Department of Diabetes & Endocrinology, Shaheed Mohtarma Benazir Bhutto Medical University, Larkana, Pakistan.

⁴Senior Registrar, Department of Gastroenterology, Hepatology & Nutrition, Gambat Institute of Medical Sciences, Gambat, Pakistan.

⁵Medical Officer, Chandka Medical College Hospital, Larkana, Pakistan.

⁶Assistant Professor, Department of Physiology, Khairpur Medical College, Khairpur Mir's, Pakistan.

Corresponding author: Dr. Kamran Ali Memon, ⁴Senior Registrar, Department of Gastroenterology, Hepatology & Nutrition, Gambat Institute of Medical Sciences, Gambat, Pakistan.

Email: drkamranali26@gmail.com

Received: 22.02.2026, Revised: 24.05.2026, Accepted: 24.05.2026

ABSTRACT

Background: Hepatic steatosis is common among patients with type 2 diabetes mellitus and may worsen with poor glycemic control and metabolic dysfunction.

Objective: To assess the association between glycemic control and hepatic steatosis severity in patients with type 2 diabetes mellitus.

Methodology: This cross-sectional analytical study was conducted at Department of Medicine Unit-2, Chandka Medical College Hospital, Shaheed Mohtarma Benazir Bhutto Medical University, Larkana from 1st May 2025 to 31st October 2025, including 240 patients with type 2 diabetes mellitus. Baseline demographic and clinical variables included age, gender, duration of diabetes, body mass index, blood pressure, smoking status, comorbidities including hypertension and dyslipidemia, and antidiabetic treatment history.

Results: Patients with moderate/severe steatosis were older, had longer diabetes duration, and higher body mass index compared with those having no/mild steatosis. HbA1c increased progressively from 6.8±0.9% in patients without steatosis to 10.1±1.6% in severe steatosis (p<0.001). Fasting blood glucose, alanine aminotransferase, aspartate aminotransferase, and triglycerides also increased significantly with steatosis severity. Hemoglobin A1c showed the strongest positive correlation with steatosis grade (r=0.68; p<0.001). Poor glycemic control was the strongest predictor of moderate-to-severe hepatic steatosis (aOR 4.26; 95% CI: 2.08-8.71; p<0.001).

Conclusion: Poor glycemic control is significantly associated with increased hepatic steatosis severity in type 2 diabetic patients.

Keywords: Type 2 Diabetes Mellitus, Hepatic Steatosis, Fatty Liver, Glycemic Control.

INTRODUCTION

Chronic hyperglycemia due to insulin resistance and progressive beta-cell dysfunction is the hallmark of type 2 diabetes mellitus (T2DM) which is one of the major public health threats globally.¹ This has increased in all parts of the world because of a lack of activity, unhealthy eating patterns, obesity and ageing of the population.² In addition to the microvascular and macrovascular complications, a close relationship between type 2 diabetes and metabolic liver disease (hepatic steatosis) is now recognized.³ Hepatic steatosis, which is

also called non-alcoholic fatty liver disease (NAFLD) or metabolic dysfunction-associated steatotic liver disease (MASLD), is defined by an abnormal amount of fat in the liver cells with no obvious secondary causes such as chronic alcohol use.⁴ It is known as the hepatic expression of metabolic syndrome and is closely related with obesity, insulin resistance, dyslipidaemia, hypertension and type 2 diabetes mellitus.⁵ Hepatic steatosis is more common in diabetic patients than in the general population: 40–70% of diabetics have hepatic steatosis.⁶

The pathophysiological association of glycemic control and hepatic steatosis is bidirectional and complicated. Continuous hyperglycemia leads to de-novo-lipogenesis, oxidative stress, mitochondrial dysfunction and activation of inflammatory cytokines which all play a role in hepatic fat accumulation and disease progression.⁷ On the other hand, the hepatic steatosis increases insulin resistance and further compromises glycemic control, causing a vicious cycle. In contrast, insulin resistance becomes worse in the presence of hepatic steatosis, which further hampers glycemic control.⁸ Therefore, hepatic steatosis may coexist with poor glycemic control and it may be worsened by poor glycemic control and may be a risk factor for its progression to steatohepatitis, fibrosis, cirrhosis and hepatocellular carcinoma.⁹ Long-term glycemic control is widely considered to be a direct measure of glycated hemoglobin (HbA1c) and is often used to evaluate the metabolic risk among diabetics.¹⁰ Increased prevalence and severity of hepatic steatosis have been suggested to be related to higher HbA1c levels in several studies.¹¹ The results of different studies have been mixed, but some show a strong relationship between obesity and insulin resistance, whereas others indicate that these correlations are not as great as the one between glycemic status and insulin resistance.¹²

Liver ultrasonography, controlled attenuation parameter measurements, computed tomography (CT) and magnetic resonance imaging (MRI) are valid methods for assessing severity of hepatic steatosis, with liver biopsy being used in selected cases.¹³ Due to the accessibility, low cost and non-invasive nature of ultrasound, and its applications in routine clinical practice, it is one of the most widely used diagnostic methods.¹⁴ Clinically, the identification of liver steatosis in patients with diabetes is important as it can be related to increased cardiometabolic and liver risks.¹⁵ Knowing the relationship between the degree of glycemic control and the degree of hepatic steatosis is relevant to the care of patients. Better glycaemic control could have the potential to decrease the amount of fat in the liver, and slow the progression of the disease, thereby reducing long-term complications.^{16,17} However few local data are available about this association in patients suffering from type 2 diabetes mellitus.

MATERIALS AND METHODS

This was a cross-sectional analytical study conducted at Department of Medicine Unit-2, Chandka Medical College Hospital, Shaheed Mohtarma Benazir Bhutto Medical University, Larkana from 1st May 2025 to 31st October 2025, including 240 patients with diagnosed type 2 diabetes mellitus, to assess the association between glycemic control and hepatic steatosis severity. Patients older than 18 years with the diagnosis of type 2 diabetes mellitus were enrolled. Eligible patients were those with available HbA1C results at the time of study evaluation and with abdominal ultrasonography for detecting hepatic steatosis. Patients with complete clinical, biochemical, and imaging records and those who consented to participate after adequate information was given were included. People with high levels of alcohol consumption, chronic viral hepatitis, or known chronic liver disease caused by non-metabolic factors were excluded. The patients with autoimmune hepatitis, Wilson disease, hemochromatosis, or drug-induced fatty liver disease, pregnant women, patients taking hepatotoxic drugs, and those with incomplete laboratory or imaging data were also excluded. Demographic and clinical data such as age, gender, diabetes duration, BMI, blood pressure, smoking history, drug history, and comorbidities (hypertension and dyslipidaemia) were included as baseline variables. Laboratory tests, which were performed, included fasting blood glucose, HbA1c, lipid profile, serum triglycerides, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and fasting insulin (where available). HbA1C was classified as good control (HbA1C<7%), moderate control (HbA1C 7-8.9%), and poor control (HbA1C ≥9%). Hepatic steatosis was subjectively graded as mild (Grade I), moderate (Grade II), and severe (Grade III) by abdominal ultrasonography following standard radiological criteria based on hepatic echogenicity, visualization of vascular structures, and posterior beam attenuation. A correlation was done between glycemic control and the severity of hepatic steatosis in clinical practice.

The data was analyzed through SPSS-6.0. Chi-square test was performed to evaluate the association among glycemic control categories and hepatic steatosis grades. The continuous variables were analysed using independent t-tests or one-way ANOVA as appropriate. Moderate-to-severe hepatic steatosis was analyzed for independent predictors using multivariable logistic regression analysis. A p-

value ≤ 0.05 was considered statistically significant.

RESULTS

Patients with moderate/severe steatosis were older (55.3 ± 11.1 vs. 49.8 ± 9.7 years; $p < 0.001$), had longer diabetes duration (10.2 ± 5.1 vs. 6.4 ± 3.8 years; $p < 0.001$), and higher BMI (31.4 ± 4.7 vs. 26.8 ± 3.9 kg/m²; $p < 0.001$). Hypertension, dyslipidemia, and family history of diabetes were also significantly more common in the moderate/severe group, showing a stronger metabolic risk burden (Table 1).

Mean HbA1c increased from $6.8 \pm 0.9\%$ in patients without steatosis to $10.1 \pm 1.6\%$ in severe steatosis ($p < 0.001$). Fasting blood glucose, ALT, AST, and triglycerides also increased across grades, while HDL decreased from 46.1 ± 7.8 to 33.9 ± 6.4 mg/dL, indicating worsening metabolic and hepatic dysfunction with steatosis severity (Table 2, Fig. 1).

A significant association was observed between glycemic control and severity of hepatic steatosis ($p < 0.001$). Among patients with good glycemic control (HbA1c $< 7\%$), the majority had no steatosis 24 (57.1%) or mild steatosis 26 (33.3%), while only 7 (8.5%) had moderate and 1 (2.6%) had severe steatosis. In

the moderate control group (HbA1c 7–8.9%), mild steatosis was most common 38 (48.7%), followed by moderate steatosis 34 (41.5%), no steatosis 15 (35.7%), and severe steatosis 9 (23.7%). In contrast, patients with poor glycemic control (HbA1c $\geq 9\%$) showed a clear shift toward advanced steatosis, with 41 (50.0%) having moderate steatosis and 28 (73.7%) having severe steatosis, while only 3 (7.1%) had no steatosis (Table 3).

Correlation analysis showed a strong positive relationship between HbA1c and steatosis grade ($r = 0.68$; $p < 0.001$). Fasting blood glucose ($r = 0.59$), BMI ($r = 0.54$), triglycerides ($r = 0.49$), diabetes duration ($r = 0.46$), and ALT ($r = 0.43$) also showed significant positive correlations, suggesting that worsening glycemic and metabolic status is closely linked with hepatic steatosis severity (Table 4).

Multivariate analysis identified poor glycemic control as the strongest predictor of moderate-to-severe hepatic steatosis (aOR 4.26; 95% CI: 2.08–8.71; $p < 0.001$). Other significant predictors included BMI ≥ 30 kg/m² (aOR 3.51), diabetes duration > 10 years (aOR 2.73), hypertriglyceridemia (aOR 2.58), and elevated ALT (aOR 2.11) [Table 5].

Table 1: Baseline Demographic, Clinical, and Metabolic Characteristics of Type 2 Diabetic Patients Stratified By Hepatic Steatosis Severity (N = 240)

Variable	Mild/No Steatosis (n=120)	Moderate/Severe Steatosis (n=120)	p-value
Age (years)	49.8±9.7	55.3±11.1	<0.001
Gender			
Male	47 (39.2%)	57 (47.5%)	0.19
Female	73 (60.8%)	63 (52.5%)	
Duration of diabetes (years)	6.4±3.8	10.2±5.1	<0.001
BMI (kg/m ²)	26.8±3.9	31.4 ± 4.7	<0.001
Hypertension	58 (48.3%)	84 (70.0%)	0.001
Dyslipidemia	49 (40.8%)	79 (65.8%)	<0.001
Smoking	14 (11.7%)	24 (20.0%)	0.07
Family history of diabetes	68 (56.7%)	83 (69.2%)	0.04

Table 2: Comparative Glycemic and Biochemical Parameters According to Hepatic Steatosis Grade (N = 240)

Variable	No Steatosis (n=42)	Mild (n=78)	Moderate (n=82)	Severe (n=38)	p-value
Fasting Blood Glucose (mg/dL)	132.6±28.4	154.8±36.2	182.4±42.7	214.5±51.6	<0.001
HbA1c (%)	6.8±0.9	7.6±1.1	8.7±1.3	10.1±1.6	<0.001
ALT (U/L)	31.4±10.7	41.6±14.2	54.9±19.8	68.2±24.3	<0.001
AST (U/L)	28.9±9.3	36.8±12.1	45.7±15.9	57.4±18.7	<0.001
Triglycerides (mg/dL)	141.3±34.6	176.8±41.9	214.7±52.8	248.9±61.2	<0.001
HDL (mg/dL)	46.1±7.8	41.8±8.1	37.5±7.3	33.9±6.4	<0.001

Table 3: Distribution of Glycemic Control Categories across Hepatic Steatosis Severity (n = 240)

Glycemic Control Category	No Steatosis	Mild Steatosis	Moderate Steatosis	Severe Steatosis	Total	p-value
Good Control (HbA1c <7%)	24 (57.1%)	26 (33.3%)	7 (8.5%)	1 (2.6%)	58	<0.001
Moderate Control (HbA1c 7-8.9%)	15 (35.7%)	38 (48.7%)	34 (41.5%)	9 (23.7%)	96	
Poor Control (HbA1c ≥9%)	3 (7.1%)	14 (17.9%)	41 (50.0%)	28 (73.7%)	86	

Table 4: Correlation between Glycemic Parameters and Hepatic Steatosis Severity

Variable	Correlation Coefficient (r)	p-value
HbA1c vs Steatosis Grade	0.68	<0.001
Fasting Blood Glucose vs Steatosis Grade	0.59	<0.001
Duration of Diabetes vs Steatosis Grade	0.46	<0.001
BMI vs Steatosis Grade	0.54	<0.001
Triglycerides vs Steatosis Grade	0.49	<0.001
ALT vs Steatosis Grade	0.43	<0.001

Table 5: Multivariate Logistic Regression Analysis for Predictors of Moderate-To-Severe Hepatic Steatosis

Variable	Adjusted OR	95% CI	p-value
Poor Glycemic Control (HbA1c ≥9%)	4.26	2.08–8.71	<0.001
BMI ≥30 kg/m ²	3.51	1.82–6.77	<0.001
Diabetes Duration >10 years	2.73	1.36–5.46	0.004
Hypertriglyceridemia	2.58	1.29–5.13	0.007
Elevated ALT	2.11	1.05–4.24	0.03

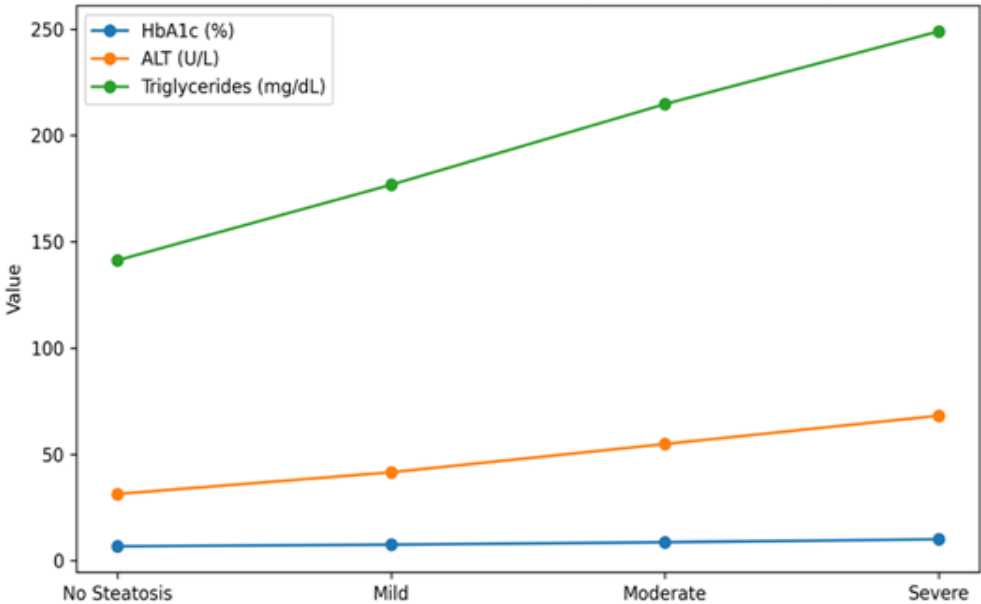


Fig. 1: Comparative Trends In Glycemic and Biochemical Parameters across Different Grades of Hepatic Steatosis in Type 2 Diabetic Patients

DISCUSSION

This study assessed the linkage between glycemic control and the severity of hepatic steatosis among type 2 diabetes mellitus patients, and found a strong correlation

between poor glycemic control and a more severe degree of fatty liver disease. The results confirm the intimate metabolic relationship between chronic hyperglycemia, insulin resistance, dyslipidaemia, and liver fat. Overall,

a high prevalence of metabolic risk factors was observed, and half of the patients had moderate to severe hepatic steatosis. Patients with steatosis were significantly older and had longer duration of diabetes and higher BMI, reflecting the time required for the degree of steatosis to be severe, and thus indicating a longer period of metabolic injury. Continuous exposure to insulin resistance and extended hyperglycaemia is likely to result in the speeding up of the accumulation of lipids in the liver and inflammatory processes. Increased age, obesity and diabetes duration were also reported to be strongly associated with advanced hepatic steatosis in diabetic population in previous research.¹⁸ One of the results of this study was the worsening glycemic indices in the progressive stages of hepatic steatosis severity. Higher HbA1c was observed in the patients with severe steatosis ($10.1 \pm 1.6\%$) compared to those without steatosis ($6.8 \pm 0.9\%$). Also, blood glucose level significantly increased with grade of steatosis. This further supports the idea that the effects of poor long-term glycemic control directly lead to liver fat accumulation by increasing de novo lipogenesis and oxidative stress, and decreasing insulin signaling. Another study showed that a high HbA1c level was significantly correlated with worsening FLD in T2DM patients.¹⁹ There were also significant progressive decreases of liver enzyme levels and lipid parameters as steatosis progressed. The levels of ALT and AST gradually increased, and triglyceride levels significantly increased, while HDL levels decreased. These results suggest that progressive hepatocellular damage and metabolic dysfunction occurs in people with severe fatty liver disease. Hypertriglyceridemia is probably due to elevated influx of lipids into the liver and/or a diminished clearance of lipids. A previous study also showed that moderate to severe FLD was associated with high levels of liver enzymes and adverse lipid profile in diabetics.²⁰

When adjusted for glycemic control categories, a stronger association was observed. Poorly controlled diabetic patients tended to have a high proportion of moderate and severe steatosis whereas most patients with good glycemic control had either none or mild steatosis. This trend is indicative of a dose-response relationship between deterioration of glycemic control and hepatic fat content. A prior study also demonstrated that having poor HbA1c control was a significant risk for advanced hepatic steatosis.²¹ The highest correlation with severity of hepatic steatosis was

found with HbA1c ($r=0.68$), followed by fasting glucose, BMI, triglycerides and duration of diabetes. These results indicate that several metabolic parameters are associated with fatty liver severity but the glycemic control is a specific parameter that has a more significant effect on the severity of fatty liver. In another study, HbA1c was also found to be one of the best biochemical markers of the severity of hepatic steatosis in diabetic patients.²² These results have important clinical implications. In recent years, it has been thought that hepatic steatosis was not a trivial incidental, particularly in diabetics, but rather a precursor to steatohepatitis, fibrosis, cirrhosis and cardiovascular disease. Routine glycemic and hepatic assessment of patients may help to identify the high-risk diabetic patient at an early stage, for timely intervention.

Limitations: Being a single-center cross-sectional study, causal relationship between poor glycemic control and hepatic steatosis severity could not be established. Hepatic steatosis was assessed by ultrasonography, which is operator-dependent and less sensitive for detecting mild steatosis compared with advanced imaging or liver biopsy. The sample size was moderate, which may limit subgroup analysis. Lifestyle factors such as diet, physical activity, and medication adherence were not fully assessed. Additionally, long-term follow-up was not performed, so progression to steatohepatitis, fibrosis, or cirrhosis could not be evaluated.

CONCLUSION

Poor glycemic control is strongly associated with increased severity of hepatic steatosis in patients with type 2 diabetes mellitus. Higher HbA1c levels, prolonged duration of diabetes, obesity, hypertriglyceridemia, and elevated liver enzymes were significant predictors of moderate-to-severe hepatic steatosis. The progressive worsening of hepatic steatosis with deteriorating metabolic control highlights the close pathophysiological relationship between diabetes and fatty liver disease.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas. 11th ed. Brussels, Belgium: International Diabetes Federation; 2025.
2. Balkhed W, Bergram M, Iredahl F, Holmberg M, Edin C, Carlh  ll CJ, et al. Evaluating the prevalence and severity of metabolic dysfunction-associated steatotic liver disease in patients with type 2

- diabetes mellitus in primary care. *J Intern Med* 2025;298(3):173-187.
3. Choi J, Kamath T, Nguyen VH, Przybyszewski E, Song J, Carroll A, et al. GLP-1RA and liver fibrosis progression in MASLD and type 2 diabetes: target trial emulation using propensity score matching. *Liver Int* 2025;45(12):70447.
4. Chen Y, Liang X, Qi Y. Prevalence of metabolic dysfunction-associated steatotic liver disease with clinically significant fibrosis in obese patients with type 2 diabetes mellitus -China, 2017-2024. *China CDC Wkly* 2025; 21:1483-1490.
5. Lindfors A, Strandberg R, Hagström H. Screening for advanced liver fibrosis due to metabolic dysfunction-associated steatotic liver disease alongside retina scanning in people with type 2 diabetes: a cross-sectional study. *Lancet Gastroenterol Hepatol* 2025;10:125-37.
6. Shojaei F, Erfanifar A, Kalbasi S, Nikpour S, Gachkar L. The effect of empagliflozin on non-alcoholic fatty liver disease-related parameters in patients with type 2 diabetes mellitus: a randomized controlled trial. *BMC Endocr Disord* 2025;25:52.
7. Sun Y, Ji H, Sun W, An X, Lian F. Triglyceride glucose index: a promising biomarker for diagnosis and treatment of different diseases. *Eur J Intern Med* 2025;131:3-14.
8. Younossi ZM, Golabi P, Price JK, Owringi S, Gundu-Rao N, Satchi R, et al. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among patients with type 2 diabetes. *Clin Gastroenterol Hepatol* 2024;22(10):1999-2010.
9. Sarkar Das T, Meng X, Abdallah M, Bilal M. An assessment of the feasibility, patient acceptance, and performance of point-of-care transient elastography for metabolic-dysfunction-associated steatotic liver disease: a systematic review and meta-analysis. *Diagnostics* 2024; 14(22):2478.
10. Ravanbakhsh N, Browne D, Weaver C, Gholikhani A, Kavarian P, Mitsinikos T. Comparing imaging modalities in the assessment of fibrosis in metabolic dysfunction-associated steatotic liver disease. *J Pediatr Gastroenterol Nutr* 2024;79:1192-8.
11. Lin H, Lee HW, Yip TC, Tsochatzis E, Petta S, Bugianesi E, et al. Vibration-controlled transient elastography scores to predict liver-related events in steatotic liver disease. *JAMA* 2024;331(15):1287-97.
12. Zhong H, Zhang K, Lin L, Yan Y, Shen L, Chen H, et al. Two-week continuous glucose monitoring-derived metrics and degree of hepatic steatosis: a cross-sectional study among Chinese middle-aged and elderly participants. *Cardiovasc Diabetol* 2024;23(1):322.
13. Wen X, Yang H, Yang M, Tao W, Chen J, Zhao S, et al. Factors that determine glucose variability, defined by the coefficient of variation in continuous glucose monitoring values, in a Chinese population with type 2 diabetes. *Diabetes Obes Metab* 2024; 26(2):611-21.
14. Qadri S, Yki-Järvinen H. Surveillance of the liver in type 2 diabetes: important but unfeasible? *Diabetologia* 2024;67:961-73.
15. Nogueira JP, Cusi K. Role of insulin resistance in the development of nonalcoholic fatty liver disease in people with type 2 diabetes: from bench to patient care. *Diabetes Spectr* 2024; 37:20-28.
16. Otero Sanchez L, Chen Y, Lassailly G, Qi X. Exploring the links between type 2 diabetes and liver-related complications: a comprehensive review. *United European Gastroenterol J* 2024; 12:240-251.
17. Dunn TC, Ajjan RA, Bergenstal RM, Xu Y. Is it time to move beyond TIR to TITR? Real-world data from over 20,000 users of continuous glucose monitoring in patients with type 1 and type 2 diabetes. *Diabetes Technol Ther* 2024; 26:203-10.
18. Zhang Y, Wang F, Tang J, Shen L, He J, Chen Y. Association of triglyceride glucose-related parameters with all-cause mortality and cardiovascular disease in NAFLD patients: NHANES 1999-2018. *Cardiovasc Diabetol* 2024; 23:262.
19. Huang DQ, Nouredin N, Ajmera V, Amangurbanova M, Bettencourt R, Truong E, et al. Type 2 diabetes, hepatic decompensation, and hepatocellular carcinoma in patients with non-alcoholic fatty liver disease: an individual participant-level data meta-analysis. *Lancet Gastroenterol Hepatol* 2023;8(9):829-36.
20. Rinella ME, Lazarus JV, Ratziu V. NAFLD nomenclature consensus group. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *J Hepatol* 2023;79:1542-56.
21. Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB

- National Cross-sectional Study, Phase I and II. *Lancet Diabetes Endocrinol* 2023;11:474-89.
22. Yahaya JJ, Doya IF, Morgan ED, Ngaiza AI, Bintabara D. Poor glycemic control and associated factors among patients with type 2 diabetes mellitus: a cross-sectional study. *Sci Rep* 2023;13(1):9673.