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TyG-BMI Index as a Potential Predictor of Nonalcoholic Fatty Liver Disease in Type 2 Diabetes Mellitus

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Abstract: This study aimed to validate the triglyceride glucose-body mass index (TyG-BMI) as a predictor of ultrasonography-confirmed non-alcoholic fatty liver disease (NAFLD) in hospitalized patients with type 2 diabetes mellitus (T2DM). In this retrospective cohort analysis of 76 T2DM patients, NAFLD was diagnosed using standardized abdominal ultrasonography. Multivariate logistic regression adjusted for age and sex revealed that patients with NAFLD exhibited significantly higher TyG-BMI levels than non-NAFLD controls ($p<0.05$). Each 10-unit increment in TyG-BMI increased NAFLD risk by 30% (adjusted OR=1.3, 95% CI: 1.1–1.6, $p<0.05$). Notably, the highest TyG-BMI tertile demonstrated an 18.3-fold elevated risk of NAFLD compared to the lowest tertile (adjusted OR=18.3, 95% CI: 3.1–107.7, $p<0.05$). These findings establish TyG-BMI as a robust predictor of NAFLD in T2DM, with extreme risk elevation in high-index patients. Integration of TyG-BMI into diabetes management protocols may enable early risk stratification and targeted interventions.

1. Introduction

Type 2 diabetes mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD) exhibit a bidirectional relationship driven by shared mechanisms of insulin resistance (IR) and dysregulated lipid metabolism. Globally, NAFLD affects 25–30% of adults, with a 2–3-fold higher prevalence in T2DM patients[1,2]. This synergy accelerates progression to advanced liver fibrosis, cirrhosis, and hepatocellular carcinoma (HCC), while amplifying cardiovascular disease (CVD) risk—the leading cause of death in this cohort[3,4]. The core pathophysiological link is IR, which promotes hepatic de novo lipogenesis (DNL) and ectopic fat accumulation, further exacerbating glycemic dysregulation and hepatic inflammation[1,5].

Early detection of NAFLD in T2DM is clinically imperative but hindered by limitations of gold-standard methods. Liver biopsy remains invasive and unsuitable for population screening, while transient elastography (FibroScan®) faces barriers in cost and accessibility[6]. Consequently, >60% of T2DM patients with NAFLD are undiagnosed until advanced stages[7]. This gap necessitates non-invasive IR surrogates. The triglyceride-glucose index (TyG), calculated as ln[TG (mg/dL) ×FPG (mg/dL)/2]ln[TG (mg/dL) ×FPG (mg/dL)/2], correlates with hepatic steatosis 69. However, integrating adiposity measures (e.g., BMI) enhances predictive accuracy.

The TyG-BMI index (TyG ×BMI) synergistically captures dual pathways of dysmetabolism and visceral obesity, outperforming TyG or BMI alone in predicting NAFLD risk[6,8]. In the UK Biobank cohort, each SD increase in TyG-BMI elevated cardio-metabolic multimorbidity risk by 47% (HR=1.47, 95% CI: 1.42–1.52) [8]. A 2024 Korean nationwide study found NAFLD severity in T2DM patients linearly increased CVD mortality (2.09–5.91/1,000 person-years)[3]. Despite this, critical gaps persist: Existing TyG-BMI thresholds derive from general populations, lacking T2DM-specific validation[5,8]. Ethnic variations in optimal cut-offs (e.g., 222.1 in Asian vs. 240 in Western cohorts) remain unaddressed in diabetic cohorts[6,9]. This study aims to validate TyG-BMI as a predictor of ultrasonography-confirmed NAFLD in a T2DM cohort.

2. Patients and Methods

A retrospective cohort study was conducted among hospitalized patients with T2DM at Foshan Women and Children's Hospital from July 2024 to May 2025. The inclusion criteria were age ≥18 years and availability of complete clinical data, including anthropometry, fasting biochemistry, and liver ultrasonography. Patients were excluded if they had alcohol intake >30 g/day (men) or >20 g/day (women), other liver diseases (such as viral hepatitis or autoimmune hepatitis), cirrhosis, or hepatocellular carcinoma (HCC). Nonalcoholic fatty liver disease (NAFLD) was diagnosed using standardized criteria based on abdominal ultrasonography.

The statistical analysis was conducted using EmpowerStats 5.2 (X&Y Solutions Inc.). Continuous variables were divided into two groups based on their distribution characteristics. For normally distributed continuous variables, the mean and standard deviation were calculated. Categorical variables were presented as frequencies and percentages. To compare the clinical characteristics between the two groups, different statistical tests were applied according to the variable types. For continuous variables, the independent samples t-test was used for normally distributed data after confirming normality through the Shapiro-Wilk test. For non-normally distributed data, the Mann–Whitney U test was employed. For categorical variables, the chi-square test was conducted to examine the differences between groups. In order to explore the relationship between the TyG-BMI index and NAFLD, multivariate regression analysis was performed. The results were presented as odds ratios (OR) with their corresponding 95%confidence intervals (CI). A p-value of less than 0.05 was considered statistically significant.

3. Results

Table 1 Baseline Characteristics of Study Population

	Patients without NAFLD	Patients with NAFLD	P-value
N	25	51	
AGE	51.2 ±12.4	43.8 ±14.1	0.026
Sex			0.059
Female	11 (44.0%)	34 (66.7%)	
Male	14 (56.0%)	17 (33.3%)	
WEIGHT	62.5 ±10.2	72.5 ±17.8	0.003
HEIGHT	162.0 ±7.3	159.8 ±7.7	0.120
TG	2.5 ±1.8	3.1 ±2.4	0.200
CHOL	5.1 ±1.3	5.6 ±1.3	0.146
LDL-C	3.5 ±1.1	3.9 ±1.0	0.124
FPG	13.2 ±6.8	12.0 ±4.3	0.765
HBA1C	10.5 ±2.7	9.9 ±2.5	0.304
ALT	25.4 ±17.0	52.4 ±61.0	0.008
AST	22.5 ±10.5	36.0 ±33.3	0.035
GGT	30.5 ±22.8	45.3 ±32.1	0.022
TyG	9.9 ±1.0	10.0 ±0.8	0.592
BMI	23.7 ±2.5	28.2 ±4.9	<0.001
Tyg-BMI	233.8 ±35.5	283.4 ±60.6	<0.001

A total of 76 participants were included in the final analytic cohort. Table 1 shows the baseline characteristics of the participants. Patients with NAFLD demonstrated significantly higher TyG-BMI levels compared to non-NAFLD patients ($p<0.05$). (Table 2) Logistic regression analysis revealed that each 10-unit increase in TyG-BMI was associated with a 0.3-fold elevated risk of fatty liver disease (OR=1.3, 95%CI: 1.1-1.6, $p<0.05$). After adjustment for age and sex, individuals in the highest TyG-BMI tertile exhibited substantially increased NAFLD risk (adjusted OR=18.3, 95%CI: 3.1-107.7, $p<0.05$) relative to the lowest tertile.

Table 2 Relationship between Tyg-BMI index And NAFLD

	Non-adjusted		Adjust I	
	OR (95%CI)	P-value	OR (95%CI)	P-value
Tyg-BMI per 10 points	1.3 (1.1, 1.6)	0.0007	1.3 (1.1, 1.6)	0.0016
Tyg-BMI Tertile				
Low	1.0		1.0	
Middle(237.8-272.1)	3.2 (1.0, 10.2)	0.0502	3.1(0.9, 10.5)	0.0667
High	18.0 (3.5, 93.7)	0.0006	18.3(3.1, 107.7)	0.0013

Adjust I: adjusted for Age, Sex

4. Discussion

The principal findings of this study confirm the TyG-BMI index as a potent and independent predictor of non-alcoholic fatty liver disease (NAFLD) in patients with type 2 diabetes mellitus (T2DM). Our analysis revealed that patients with NAFLD exhibited significantly elevated TyG-BMI levels compared to their non-NAFLD counterparts, establishing a clear association between this composite index and hepatic steatosis. Critically, the dose-response relationship demonstrated through logistic regression—where each 10-unit increment in TyG-BMI corresponded to a 30% increased risk of NAFLD (OR = 1.3, 95% CI: 1.1–1.6)—underscores its utility as a continuous risk marker. Most strikingly, individuals within the highest TyG-BMI tertile faced an extraordinary 18.3-fold elevated risk of NAFLD (adjusted OR = 18.3, 95% CI: 3.1–107.7) after adjusting for age and sex, relative to the lowest tertile. This pronounced gradient effect highlights TyG-BMI's exceptional discriminative capacity for identifying high-risk T2DM patients.

The robust predictive performance of TyG-BMI likely stems from its integrative reflection of two core pathophysiological pathways in NAFLD development: insulin resistance (IR) and adiposity. The TyG component (logarithmically derived from fasting triglycerides and glucose) serves as a well-validated surrogate marker of systemic IR, a primary driver of hepatic de novo lipogenesis and impaired fatty acid oxidation in T2DM[9,10]. Concurrently, BMI incorporates the contribution of adipose tissue mass, particularly visceral adiposity, which releases pro-inflammatory cytokines and excess free fatty acids (FFAs) that directly promote hepatic lipid accumulation[11]. By synergistically combining these elements, TyG-BMI captures the multifaceted "metabolic overload" characteristic of T2DM-associated NAFLD more comprehensively than either component alone[12]. Our findings align with mechanistic studies showing that IR-induced hyperinsulinemia amplifies sterol regulatory element-binding protein 1c (SREBP-1c) activity in hepatocytes, while adipose-derived FFAs overwhelm hepatic β-oxidation capacity[7]. Compared to traditional biomarkers or complex scores requiring specialized tests (e.g., HOMA-IR, MRI-PDFF), TyG-BMI offers distinct clinical advantages. It utilizes routinely measured, low-cost parameters (triglycerides, glucose, weight, height), making it feasible for widespread implementation in resource-limited settings[13].

5. Conclusion

In conclusion, TyG-BMI is a readily calculable, powerful predictor of NAFLD in T2DM, reflecting the synergistic contributions of insulin resistance and adiposity to hepatic steatosis. Its exceptional risk discrimination—particularly the 18-fold higher NAFLD odds in the highest tertile—supports its integration into clinical practice for early identification of high-risk patients. Future research should focus on validating intervention thresholds and evaluating TyG-BMI-guided management strategies to mitigate NAFLD burden in diabetes.

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