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ORIGINAL RESEARCH

Prediction of Hypertension Risk in Patients with Fatty Liver Disease Using the Triglyceride-Glucose-Body Mass Index

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Purpose: Fatty liver disease (FLD) and hypertension (HTN) exhibit a bidirectional relationship: FLD elevates HTN risk, while HTN accelerates FLD progression to fibrosis. Research on this link is limited, with insulin resistance (IR) proposed as a key mechanism. The triglyceride-glucose-body mass index (TyG-BMI), an emerging IR marker, is poorly characterized regarding its association with FLD and HTN. This study assessed TyG-BMI's predictive value for HTN risk in FLD patients and its clinical significance.

Patients and Methods: A retrospective cohort of 6,257 FLD patients confirmed by ultrasonography from the Health Examination Center of Chengde Medical University was analyzed. Participants were categorized into FLD-HTN (n=2,804) and normotensive FLD (FLD-0, n=3,453) groups based on blood pressure measurements. Multivariable logistic regression models adjusted for confounders assessed TyG-BMI's independent association with HTN risk. Receiver operating characteristic (ROC) curve analysis, with DeLong's test, compared the discriminative ability of TyG-BMI against conventional indices (BMI and TyG index). Additionally, quartile-based stratification (Q1-Q4) further explored dose-response relationships.

Results: Multivariable-adjusted models showed a 1.6% increase in HTN risk for each unit increase in TyG-BMI (OR=1.016, 95% CI: 1.014–1.018, $P < 0.001$). A significant positive correlation was found between TyG-BMI and both systolic (r=0.264) and diastolic blood pressure (r=0.263, both $P < 0.001$). ROC curve analysis demonstrated that TyG-BMI (AUC=0.624) outperformed BMI (AUC=0.593) and the TyG index (AUC=0.603) (DeLong's test, $P <0.01$) in discriminating HTN risk. Notably, individuals in the highest TyG-BMI quartile (Q4) had a 3.38-fold higher risk of HTN compared to those in the lowest quartile (Q1) (OR=3.380, 95% CI: 2.842–4.020).

Conclusion: TyG-BMI is a significant predictor of HTN risk in FLD patients, offering a clinically useful tool for targeted prevention strategies.

Keywords: fatty liver disease, hypertension, insulin resistance, triglyceride-glucose-body mass index

Introduction

Hypertension (HTN), a major modifiable risk factor for all-cause morbidity and mortality worldwide, is strongly associated with functional or organic damage to vital organs (heart, brain, and kidneys) and an elevated risk of cardiovascular diseases (CVD).[1,2] Consequently, effective prevention of HTN is paramount to reducing the global disease burden and extending human lifespan.[3]

Fatty liver disease (FLD) has emerged as a significant public health concern in recent years, primarily due to the escalating prevalence of diabetes and obesity. FLD is closely associated with a spectrum of metabolic disorders, including but not limited to centripetal obesity, dyslipidemia, hyperglycemia, and hepatic dysfunction.[4]

A landmark meta-analysis encompassing 11 observational cohort studies (n=390,348 hypertension-free adults across diverse populations) revealed that FLD confers a 66% increased risk of incident HTN (HR=1.66, 95% CI: 1.38–2.01)

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over a median 5.7-year follow-up.[5] Emerging evidence further establishes FLD not only as a pivotal risk factor and accelerator of HTN progression but also as a mediator in obesity-related hypertensive pathogenesis.[6] Given the adverse prognostic implications of HTN and FLD comorbidity, proactive screening for HTN in FLD patients is clinically imperative to predict CVD morbidity and mortality in this population.

Insulin resistance (IR) constitutes a central pathophysiological mechanism in FLD development,[7] driving excessive production of lipotoxic and proinflammatory mediators that induce hepatic steatosis and injury.[8] This IR-driven cascade may represent the principal link between FLD and hypertension. However, conventional IR biomarkers such as the homeostasis model assessment of insulin resistance (HOMA-IR)[9] are often influenced by the accuracy of insulin measurements and exhibit poor reproducibility.[10,11] Therefore, a non-insulin numerical index, such as the triglyceride-glucose-body mass index (TyG-BMI), may serve as a more convenient, practical, and cost-effective alternative for assessing IR.

Recent investigations have highlighted the TyG-BMI index as a promising alternative. Liu et al reported a robust independent association between TyG-BMI and HTN susceptibility, with findings remaining consistent after comprehensive covariate adjustment.[12] Substantiating this, an Iranian population-based study demonstrated that elevated TyG-BMI levels correlated with increased HTN odds, outperforming both BMI and TyG index alone in discriminating hypertensive status.[13]

Nevertheless, critical knowledge gaps persist regarding the TyG-BMI-hypertension relationship specifically in FLD populations and the diagnostic accuracy of TyG-BMI for HTN prediction in this context. To address these pivotal questions, this study aims to investigate the association between TyG-BMI and HTN incidence and to evaluate the diagnostic utility of TyG-BMI for HTN detection in FLD patients.

Patients and Methods

Study Population

This cross-sectional study initially enrolled 10186 patients with ultrasound-confirmed FLD from the Health Examination Center of Chengde Medical University Affiliated Hospital between September 2023 and September 2024. After excluding participants with incomplete data on key variables (eg, blood pressure, TyG-BMI components), 6,257 eligible individuals were stratified into two groups: 2,804 cases in the HTN group (FLD-HTN) and 3,453 controls in the normotensive group (FLD-0). The study protocol was approved by the Institutional Ethics Committee of Chengde Medical University Affiliated Hospital (No. CYFYLL2020147) in accordance with the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective nature of data collection.

Inclusion Criteria

1. Adherence to the diagnostic criteria for FLD recommended by the Asia-Pacific Working Group[14]. The diagnosis of FLD was confirmed based on the presence of ≥ 2 of the following sonographic abnormalities on abdominal ultrasound: increased echogenicity of the liver in the near field and decreased echogenicity in the far field; increased echogenicity of the liver parenchyma compared with the kidney parenchyma; and impaired visualization of intrahepatic vascularization and biliary tract structures.
2. Compliance with the diagnostic criteria for HTN.[15] The European Society of Cardiology (ESC)/European Society of Hypertension (ESH) Blood Pressure Guidelines recommend a standard classification for the definition of HTN. The diagnostic criteria were systolic BP (SBP) ≥ 140 mmHg and/or diastolic BP (DBP) ≥ 90 mmHg.

Study Methods

Physical Examination

Height, weight, and BP were measured by a trained internist using standardized equipment. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m²). After the participant had rested for at least 15 minutes in a seated position, BP was measured three times on the non-dominant arm using an automated sphygmomanometer (OMRON HEM-7312), with at least 1-minute intervals between measurements. The mean of the three readings was recorded for analysis.

Laboratory Investigations

All participants fasted for 10 hours prior to venipuncture. Five milliliters of venous blood were collected from an antecubital vein. Serum samples were separated by centrifugation and analyzed on a Beckman AU5800 automated analyzer. Biochemical parameters, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), total cholesterol (TC), triglycerides (TG), fasting blood glucose (FBG), and total protein (TP), were measured spectrophotometrically. Blood urea nitrogen (BUN) was measured enzymatically, while serum creatinine (Scr) and uric acid (UA) were determined via ion-selective electrode methodology.

The TyG index was calculated using the following equation:

$$\mathrm{Ln}\left(\mathrm{TG}\,[\mathrm{mg/dL}]\ \times\ \mathrm{FBG}\,[\mathrm{mg/dL}]/2\right).$$ [16]

TyG-BMI was calculated using the following equation:

$$\mathrm{Ln}\left(\mathrm{fasting\ TG}\,(\mathrm{mg/dl})\ \times\ \mathrm{FBG}\,(\mathrm{mg/dl})/2\right)\ \times\ \mathrm{BMI}.$$ [17]

Statistical Methods

Data were analyzed using SPSS 26.0 and R 4.2.1 with two-tailed α=0.05. Continuous variables with normal distribution are presented as mean ± SD, while skewed variables as median (Q1-Q3). Categorical variables were expressed as n (%) and compared using chi-square tests.

Group comparisons:Applied Kruskal–Wallis *H*-tests (non-parametric) or one-way ANOVA (parametric) with post-hoc Bonferroni corrections for multiple comparisons across TyG-BMI quartiles. Regression analyses: Conducted stepwise multivariate linear regression after verifying absence of multicollinearity.Performed univariate logistic regression to identify HTN-associated factors.Developed adjusted multivariate logistic models incorporating covariates: age, sex, ALT, AST, TC, etc. Diagnostic performance: Evaluated TyG-BMI's predictive capacity using receiver operating characteristic (ROC) curves, with area under the curve (AUC) comparisons via DeLong's test.

Results

Clinical and Biochemical Characteristics of FLD-0 and FLD-HTN Groups

The study cohort comprised 6,257 FLD patients, including 2,804 hypertensive (FLD-HTN) and 3,453 normotensive (FLD-0) individuals. As detailed in Table 1, FLD-HTN participants exhibited significantly higher age prevalence ($P < 0.001$), male predominance ($P < 0.001$), and elevated BMI ($P < 0.001$) compared to FLD-0 controls. Metabolically, the FLD-HTN group demonstrated marked elevations in glycemic parameters (FBG, TyG index, TyG-BMI), lipid profiles (TC, TG, LDL-C, HDL-C), hepatic enzymes (ALT, AST, GGT), and renal biomarkers (SCr, BUN, UA) (all $P < 0.01$).

Table 1 Clinical and Biochemical Characteristics of the Study Population

Variable	FLD −0	FLD -HTN	*P value*
Participants(n)	3453	2804	
Sex (n %)			< 0.001
Male	2177 (63%)	1927 (68.7%)	
Female	1276 (37%)	877 (31.3%)	
Age(year)	41 (34, 54)	50 (39, 59)	< 0.001
BMI (kg/m2)	26.2 (24.3, 28.4)	27.2 (25.2, 29.7)	< 0.001
SBP (mmHg)	121 (114, 128)	143 (136, 152)	< 0.001
DBP (mmHg)	79 (73, 84)	95 (91, 100)	< 0.001
FBG (mmol/L)	5.09 (4.76, 5.52)	5.37 (4.93, 6.08)	< 0.001
TC (mmol/L)	5.06 (4.41, 5.75)	5.24 (4.56, 5.93)	< 0.001
TG (mmol/L)	1.65 (1.2, 2.37)	1.92 (1.36, 2.77)	< 0.001
HDL-C (mmol/L)	1.21 (1.07, 1.38)	1.25 (1.09, 1.43)	< 0.001
LDL-C (mmol/L)	3.15 (2.68, 3.62)	3.24 (2.77, 3.74)	< 0.001

(*Continued*)

Table 1 (Continued).

Variable	FLD −0	FLD -HTN	*P value*
ALT (U/L)	24.4 (17.2, 35.7)	26.1 (18.4, 40.3)	< 0.001
AST (U/L)	22.3 (18.8, 27.7)	24 (20, 30.3)	< 0.001
GGT (U/L)	30.9 (21.6, 47.8)	37.3 (24.6, 61.3)	< 0.001
TP (g/L)	74.1 (71.6, 76.6)	74.5 (71.9, 77.2)	< 0.001
SCr (μmol/L)	68.1 (57.1, 77.5)	68.8 (58.8, 78.1)	0.007
BUN (mmol/L)	4.79 (4.1, 5.61)	4.92 (4.17, 5.76)	< 0.001
UA((μmol/L)	381.1 (315.7, 445)	384.5 (325.45, 450.85)	0.002
TyG	8.83 (8.5, 9.22)	9.04 (8.68, 9.48)	< 0.001
TyG-BMI	232.54 (213.18, 256.7)	248.21 (225.34, 274.05)	< 0.001

Notes: Data are presented as the mean value ± SD or median (interquartile range). $P < 0.05$ was considered statistically significant.

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine amino-transferase; AST, aspartate aminotransferase; GGT, gamma glutamyl transferase;TP, total protein; SCr, Serum creatinine; BUN, blood urea nitrogen; UA, Uric acid; TyG, TyG triglyceride-glucose index; TyG-BMI,Triglyceride Glucose-Body Mass Index.

Correlation Between TyG-BMI and Blood Pressure in FLD Patients

Scatterplot analysis (Figure 1) revealed progressive increases in both systolic blood pressure (SBP) and diastolic blood pressure (DBP) with rising TyG-BMI values. Spearman correlation analysis confirmed positive associations between TyG-BMI and SBP (r=0.264, $P < 0.001$) as well as DBP (r=0.263, $P < 0.001$).

Multivariate Analysis of HTN Risk Factors in FLD

Univariate logistic regression (Table 2) identified male sex, advanced age, elevated BMI, and dysregulated glucolipid metabolism as significant HTN risk predictors (all *P<0.05)*. Multivariate models (Table 3) maintained these associations: Model I (adjusted for age/sex): TyG-BMI retained significant predictive value (OR=1.24, 95% CI:1.18–1.31).

Model II (full metabolic adjustment): TyG-BMI remained independently associated with HTN risk (OR=1.17, 95% CI:1.10–1.25).

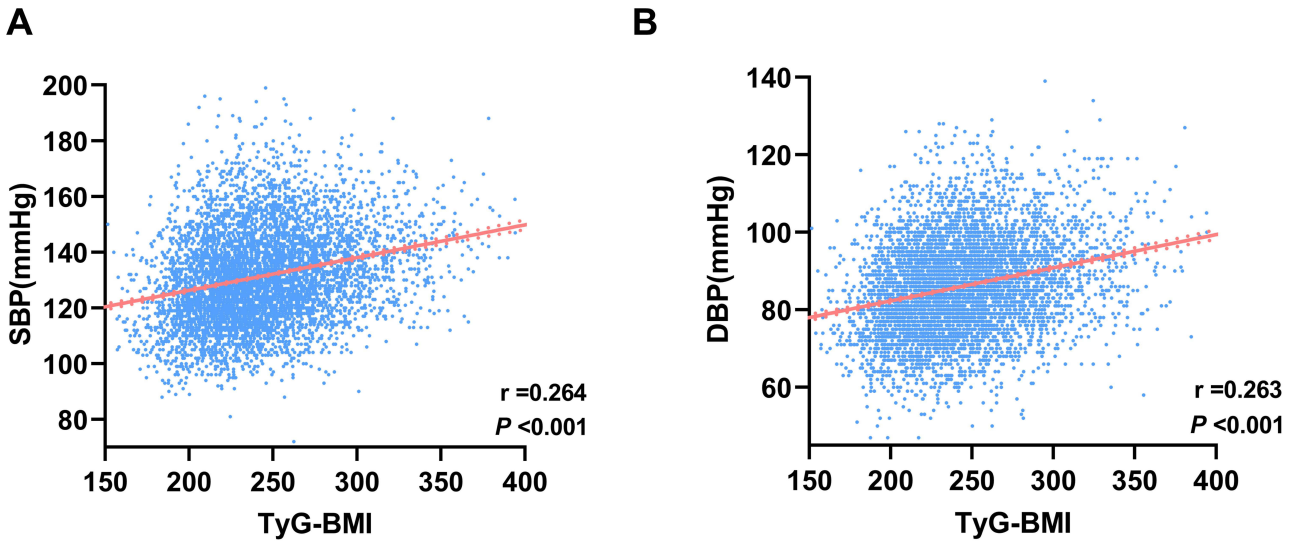

Figure 1 Scatter Plots Illustrating the Association between TyG-BMI Index and SBP and DBP Status. (**A**) SBP, (**B**) DBP. Each point represents an individual patient. The correlations between SBP and DBP with the TyG-BMI index were evaluated using the Spearman correlation test. A *P* value < 0.05 indicates a statistically significant difference.

Table 2 Univariate Logistic Regression Analysis for HTN

Characteristics	OR (95% CI) Univariate Analysis	*P value* Univariate Analysis
Sex (n %)		
Male	Reference	
Female	0.776 (0.699–0.863)	< 0.001
Age(year)	1.034 (1.030–1.038)	< 0.001
BMI (kg/m2)	1.103 (1.086–1.120)	< 0.001
FBG (mmol/L)	1.226 (1.182–1.271)	< 0.001
TC (mmol/L)	1.162 (1.109–1.218)	< 0.001
TG (mmol/L)	1.121 (1.087–1.157)	< 0.001
HDL-C (mmol/L)	1.618 (1.335–1.960)	< 0.001
LDL-C (mmol/L)	1.180 (1.103–1.262)	< 0.001
ALT (U/L)	1.005 (1.003–1.007)	< 0.001
AST (U/L)	1.015 (1.011–1.019)	< 0.001
GGT (U/L)	1.007 (1.006–1.008)	< 0.001
TP (g/L)	1.035 (1.022–1.049)	< 0.001
SCr (μmol/L)	1.004 (1.001–1.006)	0.018
BUN (mmol/L)	1.076 (1.035–1.118)	< 0.001
UA ((μmol/L)	1.001 (1.000–1.001)	< 0.001
TyG-BMI	1.013 (1.011–1.014)	< 0.001
TyG	1.747 (1.609–1.896)	< 0.001

Table 3 Multifactor Logistic Regression Analysis for HTN

	Unjust-Model		Model I		Model II	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
BMI	1.103 (1.086–1.120)	< 0.001	1.144 (1.125–1.163)	< 0.001	1.142 (1.122–1.162)	< 0.001
TyG	1.747 (1.609–1.896)	< 0.001	1.666 (1.531–1.813)	< 0.001	1.741 (1.544–1.964)	< 0.001
TyG-BMI	1.013 (1.011–1.014)	< 0.001	1.015 (1.014–1.017)	< 0.001	1.016 (1.014–1.018)	< 0.001

Notes: Model I was adjusted for age, sex. Model II was further adjusted for age, sex,TC,HDL-C,LDL-C,ALT,GGT,TP,BUN,UA.

Predictive Performance of Diagnostic Indices

ROC analysis (Table 4 and Figure 2) demonstrated superior discriminative capacity of TyG-BMI (AUC=0.624, 95% CI:0.610–0.638) compared to TyG (AUC=0.603) and BMI (AUC=0.593) ($P < 0.01$ by DeLong's test). Notably, TyG and BMI showed comparable predictive utility (P=0.321 for AUC difference).

TyG-BMI Stratification Analysis

Quartile stratification of TyG-BMI (Q1: ≤217.90, n=1,565; Q2:217.90–239.28, n=1,564; Q3:239.28–264.29, n=1,564; Q4:>264.29, n=1,564) revealed dose-dependent metabolic deterioration (Table 5). Q4 exhibited peak values in FBG, TC, TG, LDL-C, hepatic enzymes, and UA (all $P < 0.001$), alongside minimal HDL-C levels ($P < 0.001$).

Table 4 Areas Under the Receiver Operating Characteristic Curves for Each Evaluated Parameters in Identifying Hypertension

	AUC	95% CI low	95% CI up	Best Threshold	Specificity	Sensitivity
BMI*	0.593	0.579	0.607	27.450	0.577	0.478
TyG*	0.603	0.589	0.617	8.904	0.577	0.602
TyG-BMI	0.624	0.61	0.638	244.470	0.596	0.545

Notes: *$P < 0.01$, DeLong test was used to compare the AUC of TyG-BMI and TyG index/BMI.
Abbreviations: BMI, body mass index; TyG index, triglyceride and glucose index; TyG-BMI, triglyceride glucose-body mass index; AUC, the area under the curve.

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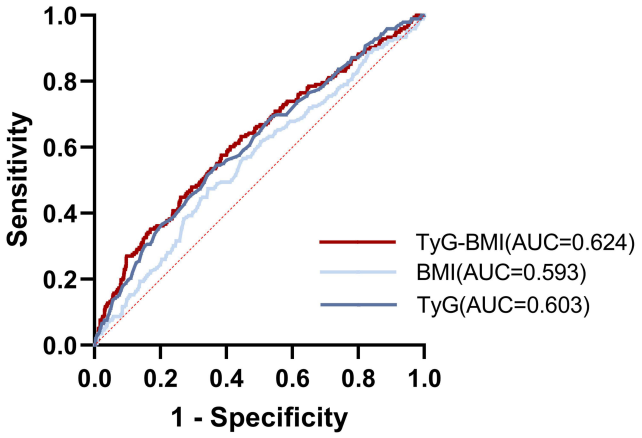

Figure 2 ROC Curves of TyG, BMI, and TyG-BMI Indexes for Predicting FLD-HTN. This figure displays the ROC curves for the TyG, BMI, and TyG-BMI indices in predicting FLD-HTN. The comparison of the areas under the curve (AUC) was performed, and a *P* value < 0.05 indicates a statistically significant difference in AUC.

Blood Pressure Patterns Across TyG-BMI Quartiles

Stratified analysis (Figure 3A and B) showed stepwise increases in both SBP and DBP across ascending TyG-BMI quartiles ($P < 0.001$). HTN prevalence escalated from 32.7% in Q1 to 58.1% in Q4 ($P < 0.001$) (Figure 3C). Multivariable-adjusted logistic regression (Table 6) confirmed gradient HTN risks: Q4 demonstrated 3.21-fold higher odds (95% CI:2.78–3.71) versus Q1 after full adjustment.

Table 5 Demographic and Clinical Characteristics of Participants by TyG-BMI Index

Characteristics	Q1	Q2	Q3	Q4	*P value*
Participants(n)	1565	1564	1564	1564	
Hypertension, n (%)					< 0.001
No	1053 (67.3%)	946 (60.5%)	799 (51.1%)	655 (41.9%)	
Yes	512 (32.7%)	618 (39.5%)	765 (48.9%)	909 (58.1%)	
Sex, n (%)					< 0.001
Male	819 (52.3%)	995 (63.6%)	1097 (70.1%)	1193 (76.3%)	
Female	746 (47.7%)	569 (36.4%)	467 (29.9%)	371 (23.7%)	
Age(year)	48 (37, 58)	48 (37, 58)	46 (35, 56.25)	41 (33, 52)	< 0.001
BMI (kg/m2)	23.7 (22.7, 24.8)	25.8 (24.9, 26.7)	27.7 (26.6, 28.7)	30.7 (29.1, 32.8)	< 0.001
FBG (mmol/L)	4.99 (4.7, 5.38)	5.15 (4.82, 5.62)	5.29 (4.88, 5.87)	5.44 (4.96, 6.32)	< 0.001
TC (mmol/L)	1.25 (0.95, 1.63)	1.67 (1.26, 2.19)	1.99 (1.48, 2.73)	2.55 (1.77, 3.74)	< 0.001
TG (mmol/L)	5 (4.33, 5.67)	5.08 (4.44, 5.76)	5.22 (4.5, 5.89)	5.31 (4.6, 5.95)	< 0.001
HDL-C (mmol/L)	1.3 (1.15, 1.48)	1.25 (1.1, 1.42)	1.22 (1.07, 1.37)	1.15 (1.01, 1.31)	< 0.001
LDL-C (mmol/L)	3.08 (2.62, 3.57)	3.19 (2.72, 3.64)	3.25 (2.76, 3.73)	3.25 (2.78, 3.75)	< 0.001
ALT (U/L)	19.8 (14.9, 27.1)	23.7 (17.2, 33.6)	27.4 (19.6, 40.7)	32.85 (22.2, 50.9)	< 0.001
AST (U/L)	21.4 (18.2, 25.2)	22.4 (18.9, 27.3)	23.9 (20, 30)	25.5 (20.5, 34)	< 0.001
GGT (U/L)	24.1 (18, 35.9)	31 (22, 47.1)	37.6 (26.2, 57.8)	45.4 (30.4, 71.8)	< 0.001
TP (g/L)	73.7 (71.2, 76.4)	74.2 (71.5, 76.8)	74.3 (72, 76.9)	74.7 (72.2, 77.3)	< 0.001
SCr (μmol/L)	64.4 (55.1, 74.3)	68.5 (57.6, 77.9)	70.15 (60.1, 78.6)	69.8 (59.8, 79.3)	< 0.001
BUN (mmol/L)	4.84 (4.12, 5.69)	4.875 (4.09, 5.67)	4.9 (4.21, 5.69)	4.8 (4.1, 5.62)	0.243
UA((μmol/L)	343.6 (291.8, 400.4)	375.1 (316.7, 437.7)	395.5 (332.0, 457.1)	421 (356.4, 488.3)	< 0.001

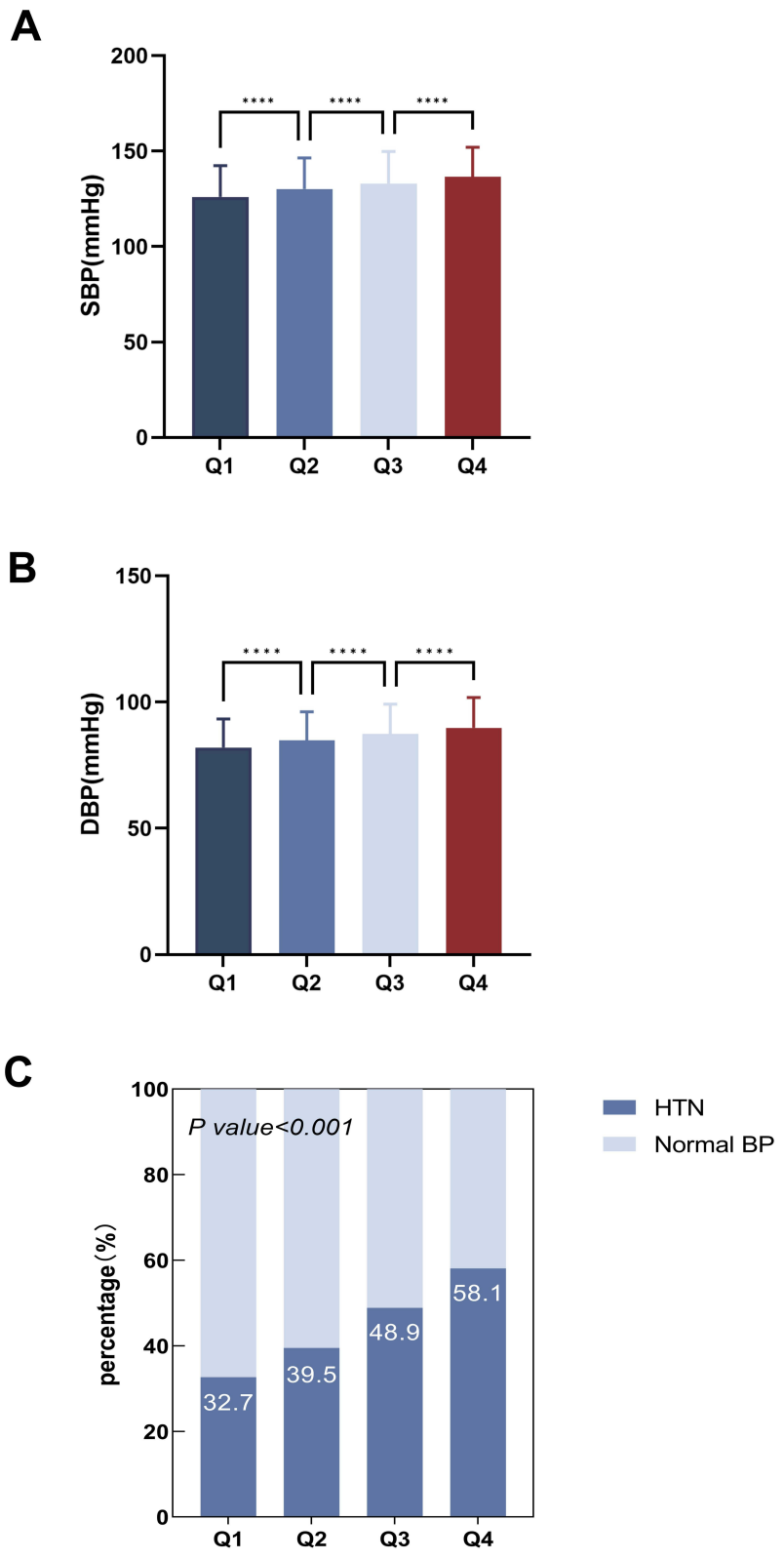

Figure 3 Blood Pressure Distribution According to TyG-BMI Index Quartiles. (**A**) Mean and standard error of SBP, (**B**) Mean and standard error of DBP, (**C**) Percentage distribution of blood pressure categories.****$P < 0.0001$.

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Table 6 Multivariate Logistic Regression Analysis of HTN Risk Across TyG-BMI Quartiles

	Unjust-Model		Model I		Model II	
	OR (95% CI)	***P***	**OR (95% CI)**	***P***	**OR (95% CI)**	***P***
Q1	Ref.		Ref.		Ref.	
Q2	1.344 (1.161–1.555)	< 0.001	1.335 (1.146–1.555)	< 0.001	1.315 (1.125–1.537)	< 0.001
Q3	1.969 (1.704–2.276)	< 0.001	2.054 (1.764–2.391)	< 0.001	1.954 (1.665–2.293)	< 0.001
Q4	2.854 (2.467–3.302)	< 0.001	3.556 (3.042–4.158)	< 0.001	3.380 (2.842–4.020)	< 0.001

Notes: Model I was adjusted for age and sex Model IIwas further adjusted for for TC, HDL-C, LDL-C, ALT, AST, GGT, TP, SCr, UA.

Discussion

FLD has emerged as the most prevalent chronic liver disorder worldwide in recent decades, affecting approximately 25% of the adult population. Previous studies have demonstrated that both the presence and severity of FLD are associated with elevated BP, prehypertension, and established HTN.[18,19] This study is the first to investigate the association between the TyG-BMI index and HTN specifically in FLD patients, while validating its clinical utility as a predictive biomarker in this population. Our results demonstrate that TyG-BMI not only serves as an independent risk factor for incident HTN in FLD patients (OR=3.362, 95% CI: 2.827–3.999), but also exhibits superior predictive performance (AUC=0.624) compared to conventional indices including BMI (AUC=0.593) and the TyG index (AUC=0.603). By focusing specifically on patients with FLD, these findings highlight the potential utility of the TyG-BMI index in this specific population and offer valuable insights for early intervention strategies targeting this metabolic disorder. Huang et al[20] demonstrated a significant association between TyG-BMI and HTN risk (OR=1.31, 95% CI: 1.25–1.37) in 2,016 Japanese non-diabetic participants comparing normotensive and HTN individuals. Similarly, Peng et al[21] established significant positive correlations of TyG-BMI with both high-normal blood pressure and HTN among 15,464 non-diabetic subjects. Notably, TyG-BMI exhibited superior predictive value over BMI and TyG index in identifying these conditions, consistent with our observations.

The HTN in individuals with FLD demonstrates distinct pathophysiological features. While adverse lifestyle factors and metabolic dysregulation influence HTN risk in both FLD and non-FLD populations, FLD itself acts as a pivotal intermediary linking lifestyle behaviors, metabolic disturbances, and HTN development.[22] Existing evidence indicates that IR constitutes a key mechanistic link between FLD and HTN.[23] Notably, IR, closely associated with metabolic syndrome, is a well-established independent risk factor for HTN. Firstly, compensatory hyperinsulinemia induced by IR may stimulate carotid body chemoreceptors, augmenting sympathetic nervous system activity. This cascade elevates epinephrine and norepinephrine secretion, consequently increasing cardiac output and peripheral vascular resistance.[24,25] Secondly, IR activates the renin-angiotensin-aldosterone system (RAAS), promoting angiotensin II generation. This leads to small artery vasospasm, elevated glomerular hydrostatic pressure, and stimulated aldosterone synthesis with enhanced renal sodium reabsorption, ultimately expanding circulatory volume and elevating BP.[26] Furthermore, under IR conditions, oxidative stress and mitochondrial dysfunction may reduce bioavailable nitric oxide while increasing endothelin production, thereby exacerbating vasoconstriction.[27–29] In populations with FLD, IR, dyslipidemia, and chronic inflammation are generally more prevalent and severe, thereby strengthening their association with HTN.[30] Furthermore, hepatic steatosis and inflammation may directly promote dysregulated BP control via multiple mechanisms, including altered adipokine profiles (eg, leptin and adiponectin), proinflammatory cytokine release, oxidative stress, sympathetic nervous system hyperactivity, and renin-angiotensin-aldosterone system (RAAS) activation.[31] Although these mechanisms also contribute to HTN pathogenesis in individuals without FLD, HTN in this group primarily arises from vascular structural/functional alterations, salt sensitivity, genetic factors, and RAAS activity, rather than direct hepatic lipid accumulation and inflammatory processes.

Crucially, we further identified a significant positive association between TyG-BMI and HTN risk in FLD patients, showing a graded increase in risk with increasing index values. As an integrated biomarker reflecting IR, dysglycemia, dyslipidemia, and obesity, TyG-BMI offers mechanistic insights into the pathogenesis of HTN in FLD. These findings provide critical evidence for the early identification of high-risk patients and the design of targeted interventions. Future studies should elucidate its precise pathophysiological pathways to optimize strategies for HTN prevention. Notably, our subgroup analysis

revealed an inverse relationship between TyG-BMI and age. This phenomenon may be attributed to younger individuals adopting unhealthy lifestyles—including sedentary behaviors and nutritional imbalances—during rapid societal development, consequently accelerating metabolic dysfunction. This observation aligns with the global trend toward earlier-onset metabolic disorders, highlighting the necessity for intensified BP monitoring in young and middle-aged FLD populations.

Although this study is the first to investigate the relationship between TyG-BMI and HTN in FLD patients, several limitations must be acknowledged. First, all data were derived from health examinations of residents in Chengde city, and the age/sex distribution of our cohort may not reflect that of the general population, thus limiting the generalizability of our findings. Second, while statistical analyses can identify associations, they cannot directly elucidate the comprehensive molecular-level disease mechanisms. A complete understanding requires integrating statistical findings with fundamental investigations, such as cellular experiments, animal models, and molecular biology techniques, for multi-level validation. Furthermore, due to covariate limitations within our dataset, we acknowledge the absence of critical variables, including dietary patterns and alcohol consumption data. In summary, while this study provides preliminary evidence supporting the association between TyG-BMI and HTN risk in patients with FLD, future research should incorporate multicenter cohort studies and mechanistic investigations to overcome the aforementioned limitations, thereby advancing a more comprehensive understanding of this association.

Conclusions

This study establishes TyG-BMI as a robust predictor of HTNrisk in FLD patients, exhibiting associations with HTN severity and superior diagnostic performance to traditional indices. These findings provide a risk-stratification framework to guide targeted prevention strategies, particularly for young FLD populations experiencing accelerated metabolic dysregulation.

Dat Sharing Statement

Original data can be obtained via Email to the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Affiliated Hospital of Chengde Medical University (No. CYFYLL2020147) and the requirement for informed consent was waived due to the retrospective nature of the study. This study was performed in adherence to the principles of the Declaration of Helsinki. All data collected was anonymised in accordance with the Personal Information Protection Law of the People's Republic of China. All patient data used is nonidentifiable and confidential.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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