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The Correlation between the Triglyceride-Glucose Index and the Severity of Non-Alcoholic Fatty Liver Disease in Obese Patients

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Abstract

Objective: Non-alcoholic fatty liver disease (NAFLD) has become the most common chronic liver disease worldwide, particularly among individuals with obesity. This study aimed to investigate the association between the triglyceride-glucose (TyG) index and NAFLD severity in obese patients. **Methods:** This cross-sectional study included 93 obese adults from Puren Hospital Affiliated to Wuhan University of Science and Technology between January 2022 and December 2023. NAFLD severity was assessed using abdominal ultrasonography and categorized as non-severe (n = 70) or severe (n = 23). The TyG index was calculated as ln [fasting triglyceride (mg/dL) × fasting plasma glucose (mg/dL)/2]. Spearman correlation and logistic regression models, and receiver operating characteristic (ROC) analysis were used to examine associations between the TyG index and NAFLD severity. **Results:** Patients with severe NAFLD had significantly higher TyG indices compared to those with non-severe disease (9.40 vs. 8.83, P = 0.003). The proportion of patients with severe NAFLD was significantly higher in the high TyG group (68.8% vs. 15.6%, P < 0.001). Spearman correlation revealed that the TyG index was positively associated with NAFLD severity (P = 0.003). In multivariate logistic regression, the TyG index was independently associated with severe NAFLD after adjusting for confounders. A one-unit increase in TyG was associated with a fourfold increase in the odds of severe NAFLD (adjusted OR = 4.092, 95% CI: 1.825 - 9.175, P = 0.001), and participants with high TyG index had 18.114 times the odds of severe NAFLD (95% CI: 4.452 - 73.708, P < 0.001) compared to the reference group. ROC curve analysis showed that the TyG index had an area under the curve (AUC) of 0.704 (95% CI: 0.576 - 0.833, P = 0.003) for discriminating severe NAFLD, indicating a moderate discriminatory ability.

Conclusion: The TyG index is significantly and independently associated with the severity of NAFLD in obese patients. As a simple and non-invasive marker, it may serve as a useful tool for early screening and risk stratification of severe NAFLD in clinical practice.

Keywords

Triglyceride-Glucose Index, Non-Alcoholic Fatty Liver Disease, Obesity, Insulin Resistance, Liver Ultrasonography

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is a chronic liver disease characterized by hepatic steatosis in the absence of significant alcohol consumption, viral hepatitis, drug-induced liver injury, or other specific causative factors [1]. With the continuous rise in global obesity rates, the prevalence of NAFLD has increased year by year and has become one of the most common chronic liver diseases worldwide. Statistics show that the prevalence of NAFLD in the general population is approximately 25%, while in obese individuals, the rate exceeds 70% [2]-[4]. NAFLD can progress to non-alcoholic steatohepatitis (NASH), liver fibrosis, cirrhosis, and even hepatocellular carcinoma [5]. Additionally, it is closely associated with metabolic disorders such as cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), and chronic kidney disease (CKD), significantly impacting patients' quality of life and life expectancy [6]-[8].

Currently, liver biopsy remains the gold standard for the diagnosis and assessment of NAFLD severity [9]. However, its invasive nature, low patient acceptance, high cost, and sampling variability limit its widespread clinical use. Therefore, identifying a simple, cost-effective, reproducible, and non-invasive biomarker for identifying the presence and severity of NAFLD is of great clinical importance. In recent years, increasing attention has been paid to the relationship between metabolic indicators and NAFLD. The triglyceride-glucose index (TyG index), which is calculated based on fasting triglyceride (TG) and fasting plasma glucose (FPG) levels, is a simple marker that has been widely used to evaluate insulin resistance (IR) [10]. Previous studies have demonstrated that the TyG index shows good sensitivity and specificity in predicting T2DM, CVD, and metabolic syndrome [11]-[13]. The pathogenesis of NAFLD is complex, with IR considered one of its central pathophysiological mechanisms [14]. IR is particularly common in obese patients and contributes to hepatic fat accumulation, inflammatory responses, and the progression of fibrosis [15]. Therefore, as a surrogate marker of IR, the TyG index may be closely associated with the development and progression of NAFLD. Some preliminary studies have confirmed a significant association between the TyG index and the risk of NAFLD [16] [17]. However, whether the TyG index can serve as a reliable tool for assessing the severity of NAFLD—especially

among obese individuals—has not yet been systematically investigated.

This study aims to explore the relationship between the TyG and the severity of NAFLD in obese patients. It will analyze the correlation between the TyG index and imaging-based liver grading, in order to evaluate its potential clinical value in risk stratification and early screening of NAFLD among obese individuals. Through this research, we hope to provide new theoretical support for non-invasive assessment of NAFLD and offer insights for early intervention and personalized treatment strategies.

2. Methods

2.1. Study Population

This was a cross-sectional study that consecutively enrolled obese adult patients from the Department of Gastroenterology at Puren Hospital Affiliated to Wuhan University of Science and Technology between January 2022 and December 2023. A total of 145 obese patients were initially screened for eligibility. Inclusion criteria were as follows: 1) age ≥ 18 years; 2) diagnosis of obesity, defined as a body mass index (BMI) ≥ 28.0 kg/m^2 [18]; and 3) complete records of liver ultrasonography and relevant laboratory data. Exclusion criteria included: 1) a history of significant alcohol consumption (more than 30 g/day for men or 20 g/day for women); 2) presence of other known chronic liver diseases, such as viral hepatitis, drug-induced liver injury, autoimmune liver disease, alcoholic fatty liver disease, or cirrhosis; 3) severe systemic comorbidities, including malignancies or end-stage heart or renal failure; and 4) incomplete data or missing key variables (e.g., fasting TG and FPG). Of the 145 patients screened, 52 were excluded (n = 28 due to incomplete laboratory data; n = 15 due to significant alcohol consumption history; n = 9 due to other chronic liver diseases). A total of 93 eligible obese patients were ultimately included in the analysis. Complete case analysis was performed, as no imputation methods were applied for missing data. A flowchart illustrating the participant selection process is provided in Supplementary Figure S1.

2.2. Data Collection and Definitions

Demographic characteristics (age, sex), clinical history (smoking status, comorbidities, medication use), and anthropometric measurements (height, weight, systolic blood pressure [SBP] and diastolic blood pressure [DBP]) were collected using standardized questionnaires and electronic medical records. BMI was calculated as weight (kg) divided by the square of height (m^2). Blood pressure was measured twice after 5 minutes of rest using a calibrated sphygmomanometer, and the average was recorded. Venous blood samples were drawn after an overnight fast of at least 8 hours and analyzed in the hospital's central laboratory using standardized automated equipment. Laboratory indicators included FPG, TG, cholesterol profiles (total cholesterol [TC], high-density lipoprotein cholesterol [HDL-C], low-density lipoprotein cholesterol [LDL-C], apolipoprotein A1 [ApoA1], apolipoprotein B [ApoB], lipoprotein(a)), liver enzymes (alanine aminotransfer-

ase [ALT], aspartate aminotransferase [AST], gamma-glutamyl transferase [GGT], alkaline phosphatase [ALP], total bilirubin (TB), albumin), kidney function markers (blood urea nitrogen [BUN], creatinine, uric acid), electrolytes (potassium, sodium, calcium, chloride), glycated hemoglobin A1c (HbA1c), fibrinogen, D-dimer, and complete blood count (white blood cell count [WBC], hemoglobin, platelet count). Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation [19].

Comorbidities were defined according to established clinical criteria. Diabetes was defined as a previous diagnosis, current use of antidiabetic medications, or laboratory results meeting the American Diabetes Association (ADA) criteria (FPG ≥ 7.0 mmol/L and/or HbA1c ≥ 6.5%) [20]. Hypertension was defined as prior diagnosis, current antihypertensive medication use, or SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg [21]. Dyslipidemia was defined as TG ≥ 1.7 mmol/L, LDL-C ≥ 3.4 mmol/L, TC ≥ 5.2 mmol/L, HDL-C < 1.0 mmol/L for men or <1.3 mmol/L for women, or current use of lipid-lowering therapy [22]. Hyperuricemia was defined as a serum uric acid level > 420 μmol/L in men or >360 μmol/L in women [23]. Current smoking was defined as smoking at least one cigarette per day in the past 30 days. All comorbidity diagnoses were verified through a combination of clinical history, medication records, and laboratory data.

2.3. Measurement and Classification of TyG Index

The TyG index was calculated using the following formula: TyG index = ln [fasting TG (mg/dL) × FPG (mg/dL)/2] [10]. For calculation, TG and FPG values in mmol/L were converted to mg/dL using the conversion factors: TG × 88.57 and FPG × 18. The TyG index was treated both as a continuous variable and a categorical variable. Based on the optimal cutoff value of 9.84, participants were divided into two groups: a low TyG group (n = 77) and a high TyG group (n = 16). This cutoff value was determined using receiver operating characteristic (ROC) curve analysis, which provided the best balance between sensitivity and specificity for discriminating NAFLD severity.

2.4. Assessment and Classification of NAFLD

NAFLD was assessed using abdominal ultrasonography performed by two experienced radiologists who were blinded to clinical and laboratory data. Hepatic steatosis was graded based on established sonographic criteria, including liver-to-kidney contrast, clarity of intrahepatic vessels, and posterior beam attenuation [24]. Steatosis severity was classified into four categories: none (normal liver echotexture), mild (slight diffuse increase in echogenicity with clear vessel visualization), moderate (moderate increase in echogenicity with partial obscuration of vessels), and severe (marked echogenicity with poor visualization of intrahepatic architecture). For statistical analysis, patients were grouped into a non-severe NAFLD group (n = 70), including those with no, mild, or moderate steatosis, and a severe NAFLD group (n = 23), defined as those with severe hepatic steatosis

only. This dichotomization was clinically motivated by the fact that severe hepatic steatosis represents a distinct stage with substantially higher risk of progression to non-alcoholic steatohepatitis (NASH) and advanced fibrosis compared to mild or moderate steatosis [25]. Furthermore, the limited sample size of individual severity categories precluded meaningful ordinal comparisons.

2.5. Statistical Analysis

All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed data were expressed as mean ± standard deviation and compared between groups using the independent samples t-test. Non-normally distributed variables were presented as median (interquartile range) and analyzed using the Mann-Whitney U test. Categorical variables were expressed as counts (percentages) and compared using the chi-square test or Fisher's exact test, as appropriate. Spearman correlation analysis was conducted to evaluate the associations between the TyG index, NAFLD severity, and other clinical variables. Univariate logistic regression analysis was used to identify potential factors associated with the severity of NAFLD. Variables with $P < 0.05$ in the univariate analysis were further included in multivariate logistic regression models to assess the independent association of the TyG index with NAFLD severity. Two multivariate models were constructed: Model 1 was adjusted for diabetes and hypertension only, and Model 2 was adjusted for diabetes, hypertension, HbA1c, BMI, ALT, AST, ALP, serum chloride, and HDL-C. Multicollinearity among covariates in Model 2 was assessed using the variance inflation factor (VIF). All VIF values were below 5 (range: 1.12 - 3.84), indicating no concerning level of multicollinearity. Notably, the correlation between the TyG index and HbA1c was moderate (Spearman's $\rho = 0.460$, $P < 0.001$), supporting their simultaneous inclusion. To further address potential concerns regarding overadjustment, an alternative parsimonious model (Model 3) was constructed, adjusting only for BMI, hypertension, and ALT. The discriminatory ability of the TyG index for severe NAFLD was evaluated using ROC curve analysis. The optimal cutoff value for the TyG index was determined using Youden's index, which maximizes the sum of sensitivity and specificity. The area under the curve (AUC) and 95% confidence interval (CI) were calculated. A two-sided P value < 0.05 was considered statistically significant.

3. Results

3.1. Baseline Characteristics Stratified by NAFLD Severity

As shown in Table 1, patients in the severe NAFLD group demonstrated significantly different clinical and biochemical characteristics compared to those in the non-severe group. The prevalence of diabetes (43.5% vs. 15.7%, $P = 0.006$) and hypertension (69.6% vs. 44.3%, $P = 0.035$) was markedly higher in the severe group. Moreover, the BMI was significantly elevated in patients with severe

NAFLD (41.91 vs. 35.58 kg/m^2, P = 0.003). In terms of liver function, the severe group showed significantly higher levels of ALT (55.60 vs. 32.95 U/L, P = 0.012), AST (36.50 vs. 22.30 U/L, P = 0.007), and ALP (93.29 vs. 81.15 U/L, P = 0.015). In addition, FPG (6.02 vs. 5.32 mmol/L, P = 0.012), HbA1c (6.36% vs. 5.72%, P = 0.013), and serum chloride (104.50 vs. 106.30 mmol/L, P = 0.012) were all significantly altered in the severe group. The level of TG was also higher (2.39 vs. 1.72 mmol/L, P = 0.013), and the TyG index was significantly elevated (9.40 vs. 8.83, P = 0.003). Other variables showed no statistically significant differences between the two groups (P > 0.05).

Table 1. Baseline characteristics stratified by NAFLD severity.

Variables	Total population	Non-severe NAFLD group	Severe NAFLD group	P value
N	93	70	23	
Age, years	32.65 ± 7.90	33.26 ± 7.93	30.78 ± 7.67	0.194
Gender, n (%)				0.086
Male	20 (21.5)	12 (17.1)	8 (34.8)	
Female	73 (78.5)	58 (82.9)	15 (65.2)	
Smoking, n (%)	7 (7.5)	5 (7.1)	2 (8.7)	1.000
Diabetes, n (%)	21 (22.6)	11 (15.7)	10 (43.5)	0.006
Hypertension, n (%)	47 (50.5)	31 (44.3)	16 (69.6)	0.035
Dyslipidemia, n (%)	55 (59.1)	38 (54.3)	17 (73.9)	0.097
Hyperuricemia, n (%)	55 (59.1)	42 (60.0)	13 (56.5)	0.768
Antidiabetic medications, n (%)	2 (2.2)	2 (2.9)	0 (0.0)	1.000
Antihypertensive medications, n (%)	4 (4.3)	3 (4.3)	1 (4.3)	1.000
BMI, kg/m^2	36.21 (33.07, 43.27)	35.58 (32.09, 41.33)	41.91 (36.05, 46.48)	0.003
SBP, mmHg	134.34 ± 16.09	132.57 ± 16.35	139.74 ± 14.28	0.064
DBP, mmHg	87.00 (78.00, 96.50)	86.50 (78.00, 95.25)	90.00 (80.00, 98.00)	0.310
WBC, ×10^9/L	8.36 (6.91, 9.60)	8.35 (6.89, 9.22)	8.38 (6.86, 9.88)	0.530
Hemoglobin, g/L	135.00 (127.00, 142.50)	133.00 (127.00, 141.25)	138.00 (129.00, 148.00)	0.116
Platelet count, ×10^9/L	280.72 ± 72.71	282.46 ± 76.91	275.43 ± 59.31	0.690
ALT, U/L	35.80 (24.25, 68.95)	32.95 (24.15, 57.80)	55.60 (35.80, 96.10)	0.012
AST, U/L	24.10 (19.15, 37.90)	22.30 (18.20, 33.23)	36.50 (22.80, 62.50)	0.007
Total bilirubin, μmol/L	7.46 (5.85,10.39)	7.38 (6.06, 9.80)	7.53 (5.27, 11.89)	0.834
ALP, U/L	84.15 ± 20.99	81.15 ± 18.97	93.29 ± 24.44	0.015
GGT, U/L	33.20 (20.40, 56.95)	31.70 (19.78, 53.73)	37.40 (23.50, 61.20)	0.211
Albumin, g/L	44.30 (42.80, 46.00)	44.60 (42.78, 46.40)	43.40 (42.80, 44.90)	0.102
Uric acid, μmol/L	383.00 (327.95, 484.85)	382.35 (320.73, 476.33)	395.90 (334.70, 497.00)	0.262
BUN, mmol/L	4.55 (3.76, 5.49)	4.53 (3.74, 5.26)	4.56 (3.76, 6.08)	0.493
Creatinine, μmol/L	56.30 (51.60, 64.10)	56.35 (51.95, 64.63)	56.00 (51.60, 60.40)	0.499

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eGFR, mL/min/1.73 m^2	128.66 ± 30.75	126.80 ± 30.92	134.34 ± 30.17	0.310
FPG, mmol/L	5.49 (4.85, 6.11)	5.32 (4.83, 5.99)	6.02 (5.09, 8.70)	0.012
HbA1c, %	5.72 (5.41, 6.28)	5.72 (5.39, 6.08)	6.36 (5.60, 7.90)	0.013
Serum potassium, mmol/L	3.85 ± 0.24	3.85 ± 0.25	3.86 ± 0.20	0.855
Serum sodium, mmol/L	140.82 ± 1.93	140.87 ± 1.70	140.64 ± 2.55	0.692
Serum calcium, mmol/L	2.35 ± 0.11	2.34 ± 0.11	2.36 ± 0.10	0.446
Serum chloride, mmol/L	106.00 (103.60, 107.40)	106.30 (104.50, 107.73)	104.50 (102.40, 106.10)	0.012
Triglycerides, mmol/L	1.82 (1.29, 2.90)	1.72 (1.23, 2.65)	2.39 (1.53, 4.78)	0.013
Total cholesterol, mmol/L	5.00 ± 1.05	4.95 ± 0.98	5.16 ± 1.27	0.408
LDL-C, mmol/L	3.07 ± 0.80	3.01 ± 0.77	3.23 ± 0.88	0.250
HDL-C, mmol/L	1.13 (0.92, 1.29)	1.16 (0.93, 1.31)	1.04 (0.88, 1.19)	0.050
ApoA1, g/L	1.28 (1.12, 1.44)	1.29 (1.12, 1.44)	1.24 (1.11, 1.44)	0.820
ApoB, g/L	0.96 ± 0.20	0.95 ± 0.19	1.01 ± 0.24	0.192
Lipoprotein(a), mg/L	115.90 (66.35, 209.75)	115.00 (64.60, 206.80)	132.10 (68.10, 250.70)	0.702
Fibrinogen, mg/L	2.90 (2.55, 3.37)	2.90 (2.60, 3.38)	2.93 (2.45, 3.36)	0.972
D-dimer, mg/L	0.24 (0.15, 0.42)	0.24 (0.15, 0.42)	0.25 (0.14, 0.42)	0.873
TyG index	8.92 (8.59, 9.52)	8.83 (8.55, 9.43)	9.40 (8.80, 10.34)	0.003

NAFLD, non-alcoholic fatty liver disease; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; TyG, triglyceride-glucose index.

3.2. Baseline Characteristics Stratified by the Optimal TyG Cutoff Value

As shown in Table 2, patients in the high TyG group (TyG > 9.84) had significantly worse metabolic and liver-related profiles compared to those in the low TyG group. The prevalence of diabetes (62.5% vs. 14.3%, P < 0.001) and dyslipidemia (100.0% vs. 50.6%, P < 0.001) was markedly higher in the high TyG group. In addition, this group exhibited significantly elevated liver enzymes, including ALT (68.95 vs. 32.90 U/L, P < 0.001), AST (38.85 vs. 22.80 U/L, P < 0.001), ALP (98.08 vs. 81.25 U/L, P = 0.003), and GGT (60.00 vs. 29.60 U/L, P < 0.001). Furthermore, participants in the high TyG group had significantly higher FPG (7.04 vs. 5.30 mmol/L, P < 0.001) and HbA1c (7.85% vs. 5.70%, P < 0.001). Differences were also observed in the serum chloride level (103.40 vs. 106.30 mmol/L, P = 0.001), TG (5.01 vs. 1.63 mmol/L, P < 0.001), TC (5.55 vs. 4.89 mmol/L, P = 0.022), HDL-C (0.96 vs. 1.16 mmol/L, P = 0.006), and ApoB (1.09 vs. 0.93 g/L, P = 0.006). Notably, the proportion of patients with severe NAFLD was significantly higher in the high TyG group (68.8% vs. 15.6%, P < 0.001). Other variables showed no statistically significant differences between the two groups (P > 0.05).

Table 2. Baseline characteristics stratified by the optimal TyG cutoff value.

Variables	Low TyG group	High TyG group	P value
N	77	16	
Age, years	32.53 ± 8.11	33.19 ± 7.02	0.765
Gender, n (%)			1.000
Male	17 (22.1)	3 (18.8)	
Female	60 (77.9)	13 (81.3)	
Smoking, n (%)	6 (7.8)	1 (6.30)	1.000
Diabetes, n (%)	11 (14.3)	10 (62.5)	<0.001
Hypertension	37 (48.1)	10 (62.5)	0.293
Dyslipidemia, n (%)	39 (50.6)	16 (100.0)	<0.001
Hyperuricemia, n (%)	46 (59.7)	9 (56.3)	0.796
Antidiabetic medications, n (%)	2 (2.6)	0 (0.0)	1.000
Antihypertensive medications, n (%)	3 (3.9)	1 (6.3)	0.537
BMI, kg/m^2	36.21 (32.65, 43.43)	36.84 (33.88, 41.98)	0.733
SBP, mmHg	133.32 ± 15.63	139.25 ± 17.90	0.182
DBP, mmHg	87.00 (78.00, 95.50)	92.00 (81.25, 98.00)	0.198
WBC, ×10^9/L	8.36 (6.78, 9.23)	8.29 (7.12, 11.91)	0.272
Hemoglobin, g/L	133.00 (127.00, 141.50)	139.50 (131.25, 145.75)	0.082
Platelet count, ×10^9/L	282.51 ± 73.59	272.13 ± 69.98	0.606
ALT, U/L	32.90 (23.15, 57.90)	68.95 (41.55, 148.08)	<0.001
AST, U/L	22.80 (18.10, 32.40)	38.85 (33.93, 64.15)	<0.001
Total bilirubin, μmol/L	7.33 (5.87, 10.39)	7.60 (4.98, 10.92)	0.835
ALP, U/L	81.25 ± 19.41	98.08 ± 23.29	0.003
GGT, U/L	29.60 (19.75, 49.30)	60.00 (36.95, 100.75)	<0.001
Albumin, g/L	44.40 (42.35, 46.30)	44.15 (43.25, 45.28)	0.783
Uric acid, μmol/L	382.00 (323.20, 480.40)	393.85 (335.50, 496.95)	0.541
BUN, mmol/L	4.50 (3.70, 5.23)	4.94 (4.11, 6.37)	0.063
Creatinine, μmol/L	56.80 (53.05, 64.40)	53.30 (48.43, 59.10)	0.066
eGFR, mL/min/1.73 m^2	125.86 ± 29.73	142.14 ± 32.94	0.053
FPG, mmol/L	5.30 (4.80, 5.98)	7.04 (5.85, 11.79)	<0.001
HbA1c, %	5.70 (5.40, 6.10)	7.85 (5.74, 8.62)	<0.001
Serum potassium, mmol/L	3.84 ± 0.25	3.90 ± 0.18	0.342
Serum sodium, mmol/L	141.01 ± 1.86	139.88 ± 2.06	0.032
Serum calcium, mmol/L	2.34 ± 0.11	2.39 ± 0.08	0.106
Serum chloride, mmol/L	106.30 (104.50, 107.75)	103.40 (101.70, 105.60)	0.001
Triglycerides, mmol/L	1.63 (1.24, 2.44)	5.01 (3.90, 7.11)	<0.001
Total cholesterol, mmol/L	4.89 ± 0.96	5.55 ± 1.31	0.022
LDL-C, mmol/L	3.05 ± 0.77	3.15 ± 0.95	0.669
HDL-C, mmol/L	1.16 (0.98, 1.31)	0.96 (0.88, 1.06)	0.006

Continued

ApoA1, g/L	1.30 (1.12, 1.44)	1.28 (1.20, 1.45)	0.839
ApoB, g/L	0.93 ± 0.19	1.09 ± 0.24	0.006
Lipoprotein(a), mg/L	117.90 (68.45, 218.30)	99.20 (59.53, 198.65)	0.625
Fibrinogen, mg/L	2.90 (2.57, 3.37)	2.81 (2.35, 3.48)	0.729
D-dimer, mg/L	0.24 (0.15, 0.42)	0.23 (0.13, 0.42)	0.621
Severe NAFLD, n (%)			<0.001
Yes	12 (15.6)	11 (68.8)	
No	65 (84.4)	5 (31.3)	

TyG, triglyceride-glucose index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; NAFLD, non-alcoholic fatty liver disease.

3.3. Spearman Correlation Analysis between Other Variables, TyG Index, and NAFLD Severity

As shown in Table 3, Spearman correlation analysis revealed that the TyG index was positively correlated with diabetes, hemoglobin, ALT, AST, ALP, GGT, FPG, HbA1c, TC, ApoB, and TG (all $P < 0.05$). Conversely, TyG was negatively correlated with HDL-C and serum chloride (both $P < 0.05$). No significant associations were found between TyG and other variables (all $P > 0.05$).

Regarding NAFLD severity, significant positive correlations were observed with BMI, diabetes, hypertension, SBP, ALT, AST, ALP, FPG, HbA1c, TG, and TyG index itself (all $P < 0.05$). Additionally, negative correlations were detected between NAFLD severity and HDL-C as well as serum chloride (both $P < 0.05$). All other clinical and biochemical variables showed no significant relationship with NAFLD severity ($P > 0.05$).

Table 3. Spearman correlation analysis between other variables, TyG index, and NAFLD severity.

Variables	TyG		NAFLD severity	
	r	P value	r	P value
Age	0.210	0.044	−0.118	0.260
Sex	−0.106	0.311	−0.185	0.075
Smoking	0.165	0.113	0.025	0.809
Diabetes	0.508	<0.001	0.286	0.005
Hypertension	0.176	0.091	0.218	0.036
Dyslipidemia	0.643	<0.01	0.172	0.099
Hyperuricemia	0.044	0.675	−0.031	0.771
Antidiabetic medications	0.146	0.162	−0.085	0.418
Antihypertensive medications	0.199	0.055	0.001	0.990

Continued

BMI	−0.020	0.846	0.313	0.002
SBP	0.046	0.661	0.239	0.021
DBP	0.156	0.135	0.106	0.312
WBC	0.189	0.070	0.065	0.533
Hemoglobin	0.375	<0.001	0.164	0.116
Platelet count	−0.004	0.968	−0.072	0.493
ALT	0.510	<0.001	0.261	0.012
AST	0.509	<0.001	0.284	0.006
Total bilirubin	−0.001	0.992	0.022	0.836
ALP	0.304	0.003	0.217	0.036
GGT	0.577	<0.001	0.130	0.213
Albumin	0.102	0.330	−0.170	0.102
Uric acid	0.116	0.267	0.117	0.264
BUN	0.146	0.163	0.071	0.496
Creatinine	−0.108	0.304	−0.071	0.502
eGFR	0.051	0.626	0.088	0.401
FPG	0.507	<0.001	0.262	0.011
HbA1c	0.460	<0.001	0.259	0.012
Serum potassium	0.143	0.171	0.055	0.602
Serum sodium	−0.193	0.064	−0.077	0.465
Serum calcium	0.292	0.004	0.080	0.446
Serum chloride	−0.295	0.004	−0.261	0.012
Triglycerides	0.944	<0.001	0.259	0.012
Total cholesterol	0.298	0.004	0.037	0.724
LDL-C	0.160	0.127	0.087	0.406
HDL-C	−0.351	<0.001	−0.205	0.049
ApoA1	0.008	0.941	−0.024	0.822
ApoB	0.391	<0.001	0.084	0.423
Lipoprotein(a)	−0.083	0.428	0.040	0.704
Fibrinogen	−0.171	0.101	0.004	0.972
D-dimer	−0.182	0.081	−0.017	0.874
TyG	–	–	0.305	0.003
NAFLD severity	0.305	0.003	–	–

TyG, triglyceride-glucose index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; NAFLD, non-alcoholic fatty liver disease.

3.4. Univariate Logistic Regression Analysis of NAFLD Severity

Based on the univariate logistic regression results in Table 4, several variables were significantly associated with the severity of NAFLD. Diabetes (OR = 4.126), hypertension (OR = 2.876), BMI (OR = 1.123), ALT (OR = 1.009), AST (OR = 1.017), ALP (OR = 1.028), FPG (OR = 1.537), HbA1c (OR = 1.517), serum chloride (OR = 0.814), TG (OR = 1.408), and HDL-C (OR = 0.115) were significantly associated with NAFLD severity. Other variables were not significantly associated with NAFLD severity in univariate analysis (P > 0.05).

Table 4. Univariate logistic regression analysis of NAFLD severity.

Variables	OR	95% CI	P value
Age	0.959	0.901 - 1.021	0.194
Male	2.578	0.893 - 7.437	0.080
Smoking	1.238	0.223 - 6.859	0.807
Diabetes	4.126	1.450 - 11.742	0.008
Hypertension	2.876	1.052 - 7.861	0.040
Dyslipidemia	2.386	0.841 - 6.769	0.102
Hyperuricemia	0.867	0.334 - 2.248	0.769
Antihypertensive medications	1.015	0.100 - 10.266	0.990
BMI	1.123	1.038 - 1.214	0.004
SBP	1.028	0.998 - 1.060	0.069
DBP	1.017	0.977 - 1.059	0.402
WBC	1.133	0.894 - 1.436	0.300
Hemoglobin	1.030	0.989 - 1.073	0.153
Platelet count	0.999	0.992 - 1.005	0.687
ALT	1.009	1.000 - 1.018	0.041
AST	1.017	1.002 - 1.033	0.031
Total bilirubin	1.045	0.938 - 1.164	0.425
ALP	1.028	1.004 - 1.053	0.020
GGT	1.007	0.992 - 1.023	0.355
Albumin	0.939	0.823 - 1.070	0.345
Uric acid	1.003	0.999 - 1.007	0.172
BUN	1.127	0.812 - 1.566	0.474
Creatinine	0.985	0.949 - 1.024	0.450
eGFR	1.008	0.993 - 1.024	0.308
FPG	1.537	1.135 - 2.081	0.005
HbA1c	1.517	1.082 - 2.127	0.016
Serum potassium	1.207	0.165 - 8.847	0.853
Serum sodium	0.940	0.734 - 1.203	0.622

Continued

Serum calcium	5.471	0.072 - 416.279	0.442
Serum chloride	0.814	0.675 - 0.981	0.031
Triglycerides	1.408	1.083 - 1.830	0.011
Total cholesterol	1.208	0.775 - 1.883	0.405
LDL-C	1.422	0.781 - 2.590	0.249
HDL-C	0.115	0.014 - 0.933	0.043
ApoA1	0.648	0.098 - 4.290	0.653
ApoB	4.678	0.459 - 47.724	0.193
Lipoprotein(a)	1.000	0.997 - 1.003	0.856
Fibrinogen	1.027	0.553 - 1.906	0.933
D-dimer	1.032	0.151 - 7.032	0.974

TyG, triglyceride-glucose index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; NAFLD, non-alcoholic fatty liver disease.

3.5. Multivariate Logistic Regression Analysis of the Association between TyG Index and NAFLD Severity

As shown in Table 5, multivariate logistic regression analyses demonstrated a significant independent association between the TyG index and the severity of NAFLD, even after adjustment for potential confounding factors. In Model 1, which adjusted for diabetes and hypertension only, a one-unit increase in the TyG index was associated with a more than threefold increase in the odds of having severe NAFLD (OR = 3.213, 95% CI: 1.594 - 6.477, P = 0.001). When the TyG index was dichotomized at the optimal cutoff value of 9.84, individuals in the high TyG group had 11.917 times greater odds of severe NAFLD compared to those in the low TyG group (95% CI: 3.506 - 40.502, P < 0.001).

In Model 2, which included a broader set of covariates (diabetes, hypertension, HbA1c, BMI, ALT, AST, ALP, serum chloride, and HDL-C), the TyG index remained a robust correlate. A one-unit increase in TyG was associated with a fourfold increase in the odds of severe NAFLD (OR = 4.092, 95% CI: 1.825 - 9.175, P = 0.001), and participants with a TyG index > 9.84 had 18.114 times the odds of severe NAFLD (95% CI: 4.452 - 73.708, P < 0.001) compared to the reference group. To further address potential concerns regarding overadjustment given that the TyG index is mathematically derived from fasting glucose and triglyceride levels, an alternative parsimonious model was constructed. In Model 3, which adjusted only for BMI, hypertension, and ALT (selected based on clinical relevance and univariate significance), the TyG index remained independently associated with severe NAFLD (adjusted OR per one-unit increase = 3.876, 95% CI: 1.892 -

7.941, P < 0.001; high vs. low TyG group OR = 14.527, 95% CI: 4.216 - 50.053, P < 0.001). The consistency of these findings across models with varying degrees of adjustment supports the robustness of the observed association.

Table 5. Multivariate logistic regression analysis of the association between TyG index and NAFLD severity.

Variables	Model 1			Model 2		
	OR	95% CI	P value	OR	95% CI	P value
TyG	3.213	1.594 - 6.477	0.001	4.092	1.825 - 9.175	0.001
TyG grouping						
≤9.84	Ref			Ref		
>9.84	11.917	3.506 - 40.502	<0.001	18.114	4.452 - 73.708	<0.001

Model 1: adjusted for diabetes and hypertension only; Model 2: adjusted for diabetes, hypertension, glycated hemoglobin A1c, body mass index, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, serum chloride, and high-density lipoprotein cholesterol. TyG, triglyceride-glucose index; NAFLD, non-alcoholic fatty liver disease; OR, odds ratio; CI, confidence interval; Ref, reference group.

3.6. Sensitivity Analysis Using Ordinal Logistic Regression

As a sensitivity analysis to assess the robustness of the binary classification of NAFLD severity, ordinal logistic regression was performed treating NAFLD severity as a four-level ordinal outcome (none, mild, moderate, severe). The proportional odds assumption was not violated (score test, P = 0.321). In the unadjusted ordinal model, a one-unit increase in the TyG index was associated with a 2.847-fold increase in the odds of being in a higher NAFLD severity category (95% CI: 1.412 - 5.741, P = 0.003). After adjusting for the same covariates as in Model 2 (diabetes, hypertension, HbA1c, BMI, ALT, AST, ALP, serum chloride, and HDL-C), the association remained significant (adjusted OR = 3.516, 95% CI: 1.578 - 7.832, P = 0.002). These findings are consistent with the primary binary logistic regression results and support the robustness of the association between the TyG index and NAFLD severity regardless of the analytical approach.

3.7. ROC Curve Analysis and Diagnostic Performance of the TyG Index for Severe NAFLD

As shown in Figure 1, the ROC curve was constructed to evaluate the discriminatory power of the TyG index for identifying severe NAFLD. The apparent AUC was 0.704 (95% CI: 0.576 - 0.833, P = 0.003). After bootstrap internal validation with 1000 resamples, the bias-corrected AUC was 0.689 (95% CI: 0.561 - 0.817), suggesting minimal overoptimism. The optimal cutoff value determined by Youden's index was 9.84. At this threshold, the TyG index demonstrated a sensitivity of 47.8% (95% CI: 26.8% - 69.4%), specificity of 92.9% (95% CI: 84.1% - 97.6%), positive predictive value (PPV) of 68.8% (95% CI: 41.3% - 89.0%), and negative predictive value (NPV) of 84.4% (95% CI: 74.4% - 91.7%). The high specificity and NPV

suggest that the TyG index may be particularly useful for ruling out severe NAFLD in obese patients. Detailed diagnostic performance metrics are summarized in Table 6.

Table 6. Diagnostic performance of the TyG index at the optimal cutoff (9.84) for identifying severe NAFLD.

Metric	Value	95% CI
AUC (apparent)	0.704	0.576 - 0.833
AUC (bootstrap bias-corrected)	0.689	0.561 - 0.817
Sensitivity, %	47.8	26.8 - 69.4
Specificity, %	92.9	84.1 - 97.6
Positive predictive value, %	68.8	41.3 - 89.0
Negative predictive value, %	84.4	74.4 - 91.7
Positive likelihood ratio	6.70	2.45 - 18.31
Negative likelihood ratio	0.56	0.38 - 0.83

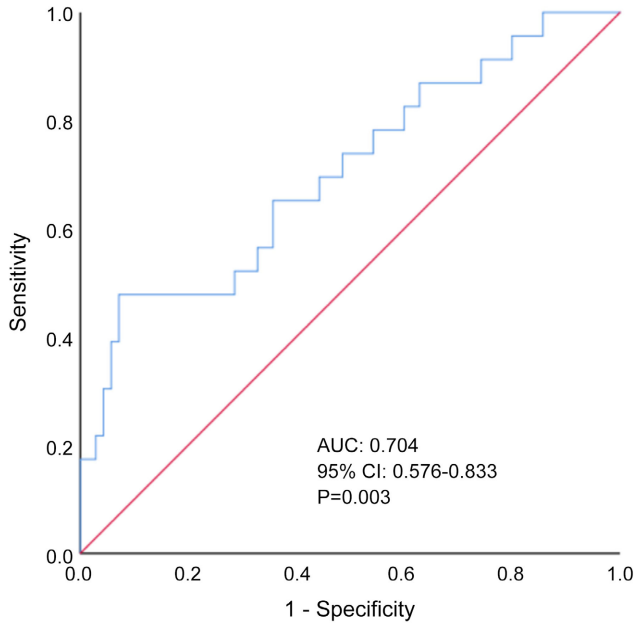

Figure 1. ROC curve assessing the discriminatory ability of the TyG index for identifying severe NAFLD. TyG, triglyceride-glucose index; NAFLD, non-alcoholic fatty liver disease; ROC, receiver operating characteristic curve; AUC, area under the curve; CI, confidence interval.

4. Discussion

This study demonstrated a significant association between the TyG index and the severity of NAFLD in obese individuals. Patients in the high TyG group exhibited more pronounced glucose metabolism disorders, elevated liver enzymes, and a higher proportion of severe NAFLD compared to those in the low TyG group. Multivariate logistic regression analysis confirmed that the TyG index remained

an independent correlate of NAFLD severity after adjusting for multiple confounding factors. ROC curve analysis further supported its moderate discriminatory ability. These findings suggest that the TyG index may serve as a simple and non-invasive tool for identifying obese patients at high risk of severe NAFLD, offering promising clinical utility. The moderate correlation between the TyG index and HbA1c (ρ = 0.460) observed in our study suggests that while these markers share common metabolic pathways, they capture distinct aspects of glycemic dysregulation. The low variance inflation factors in the fully adjusted model further indicate that multicollinearity did not materially affect our estimates.

In recent years, as research into the pathogenesis of NAFLD has advanced, increasing attention has been paid by international scholars to the role of metabolic indicators in assessing NAFLD risk—particularly the TyG index. Numerous studies have demonstrated that the TyG index not only reflects IR but is also closely associated with the development and progression of NAFLD, showing good discriminatory performance [16] [17] [25]. For example, Wang *et al.* conducted a cross-sectional study involving 11,987 non-obese Japanese individuals and found a positive and nonlinear association between TyG and NAFLD risk, suggesting that TyG may be important indicators for early screening and intervention in non-obese populations [17]. In addition, Cai *et al.* conducted a retrospective study involving 654 snoring patients and found a significant positive association between the TyG index and NAFLD risk, with a clear dose-response relationship, suggesting that the TyG index may serve as an effective indicator for screening NAFLD in snoring populations [26]. Furthermore, Yetim *et al.* conducted a retrospective study in Turkey involving 79 obese adolescents and found that the TyG index was significantly higher in the NAFLD-positive group and positively correlated with liver fat content, suggesting that the TyG index may serve as an important diagnostic indicator for NAFLD in obese adolescents and was incorporated into a predictive model to improve diagnostic accuracy [27]. Besides, Wang *et al.* conducted a cross-sectional study in a high-altitude region of China involving 1,384 adults and found a significant positive association between the TyG index and NAFLD risk, suggesting that the TyG may serve as a preferred indicator for NAFLD screening in high-altitude populations [28]. Additionally, Nayak *et al.*, in a systematic umbrella review including 32 meta-analyses, found that the TyG index was closely associated with various diseases [16]. Specifically, the TyG index was significantly elevated in NAFLD patients, with a 2.36-fold higher risk compared to individuals without NAFLD (OR = 2.36, 95% CI: 1.88 - 2.97). Moreover, the TyG index showed strong associations with metabolic syndrome, obstructive sleep apnea, and T2DM, suggesting that TyG may serve as a valuable diagnostic and predictive biomarker for IR-related metabolic diseases, including NAFLD. However, due to considerable heterogeneity among the included studies, further high-quality research is needed to confirm its clinical reliability. Compared with previous studies, the present research offers several distinctive advantages. First, it focuses on obese individuals—a population highly susceptible to NAFLD but

often underrepresented in prior literature. Second, we investigated not only the association between the TyG index and NAFLD severity but also identified an optimal clinical cutoff value (9.84) through ROC curve analysis, providing a practical reference for personalized risk stratification. Most importantly, our multivariate regression models accounted for numerous potential confounding factors, thus reinforcing the independence and clinical relevance of the TyG index as a risk stratification marker. In summary, this study not only confirms the generalizability of international findings but also extends the clinical application of the TyG index to the assessment of NAFLD severity in obese patients. These findings lay the groundwork for further exploration into the biological mechanisms linking TyG and NAFLD progression, which may inform future preventive and therapeutic strategies.

Current evidence suggests that the TyG index is not only significantly associated with the risk of NAFLD but also closely related to its severity, indicating a potential biological basis for its role in the development and progression of NAFLD. Exploring the underlying biological mechanisms linking the TyG index and NAFLD can enhance our understanding of its clinical applicability and scientific rationale. Firstly, as a logarithmic transformation of the product of fasting glucose and TG levels, the TyG index serves as a reliable surrogate marker for IR. IR is a fundamental metabolic feature in the pathogenesis of NAFLD [15]. When insulin sensitivity declines, lipolysis in adipose tissue increases, leading to an elevated influx of free fatty acids (FFAs) into the liver [29]. These FFAs are converted into triglycerides, promoting hepatic lipid accumulation. Furthermore, IR impairs hepatic glucose metabolism, enhancing gluconeogenesis and de novo lipogenesis, which further aggravates hepatic steatosis [30]. Secondly, hyperglycemia itself exerts hepatotoxic effects. Chronic elevation of blood glucose levels can induce oxidative stress, inflammatory responses, and mitochondrial dysfunction, activating pathways such as JNK and NF-κB, which promote hepatocyte apoptosis and inflammation, accelerating the progression from simple steatosis to NASH [31]. Additionally, elevated TG levels reflect disordered lipoprotein metabolism, and this lipid overload may further activate Kupffer cells and hepatic stellate cells, facilitating hepatic fibrosis [32] [33]. In the context of metabolic syndrome, the TyG index is also associated with various systemic pathological conditions, including chronic low-grade inflammation, endothelial dysfunction, and gut microbiota dysbiosis—all of which may contribute synergistically to the development of NAFLD [34]-[36]. Studies have shown that elevated TyG levels may compromise intestinal barrier integrity, allowing endotoxins such as lipopolysaccharides (LPS) to enter the portal circulation, thereby triggering hepatic inflammation and worsening liver injury [37] [38]. In summary, the TyG index reflects multiple key mechanisms implicated in NAFLD, including IR, dysregulated lipid and glucose metabolism, chronic inflammation, and oxidative stress. Its simplicity, affordability, and reproducibility make it a promising metabolic biomarker. Future research combining longitudinal cohort studies and mechanistic experiments is essential

to clarify the causal relationship and underlying pathways between the TyG index and NAFLD, thereby promoting its standardized clinical use and precision management.

Despite its valuable findings, this study has several limitations that warrant consideration. First, the cross-sectional design precludes any conclusions about causal relationships between the TyG index and NAFLD severity. Longitudinal studies are needed to verify whether a high TyG index can identify the future progression of NAFLD over time. Second, liver ultrasonography was used for disease assessment, which, while non-invasive and widely accessible, may lack the sensitivity of advanced imaging techniques such as magnetic resonance imaging (MRI) or the diagnostic accuracy of liver biopsy. As a result, some cases of mild or early-stage steatosis might have been underestimated. Third, the sample size was relatively small (n = 93), with only 23 cases in the severe NAFLD group and 16 in the high TyG group. This limited sample size may reduce statistical power and result in wide confidence intervals for some estimates (e.g., OR for high TyG group: 95% CI: 4.452 - 73.708). Additionally, the fully adjusted Model 2 included nine covariates with only 23 events, which may raise concerns regarding potential overfitting despite acceptable VIF values. Additionally, all participants were recruited from a single tertiary hospital in China, which may introduce selection bias and limit applicability to broader or more diverse populations. Finally, although multiple confounding factors were adjusted for, the influence of unmeasured variables such as dietary patterns, physical activity, and genetic predisposition could not be completely ruled out. Therefore, while our findings provide important insights into the relationship between the TyG index and NAFLD severity in obese individuals, future multicenter prospective studies with larger cohorts and more comprehensive assessments are needed to validate and extend these results.

5. Conclusion

This study demonstrates a significant association between the TyG index and the severity of NAFLD among obese individuals. As a simple, cost-effective, and non-invasive marker, the TyG index holds strong potential for identifying high-risk patients with severe NAFLD in clinical settings. Future large-scale prospective studies are warranted to further validate its role in screening, risk stratification, and treatment monitoring.

Declarations

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Puren Hospital Affiliated to Wuhan University of Science and Technology (ethics approval number :(2024) Annual Audit No.01501). All study procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to data collection, with full disclosure of the study's objectives, procedures, potential risks, and data usage.

All personal information was anonymized to ensure participant confidentiality and data security.

Authors' Contributions

Li Tian (L.T.): Conceptualization, Methodology, Software, Investigation, Data curation, Formal analysis, Visualization, Writing-original draft, Writing-review & editing.

Pan Sheng (P.S.): Conceptualization, Validation, Funding acquisition, Project administration, Supervision.

All authors have read and approved the final manuscript.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no competing interests.

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Supplementary Materials

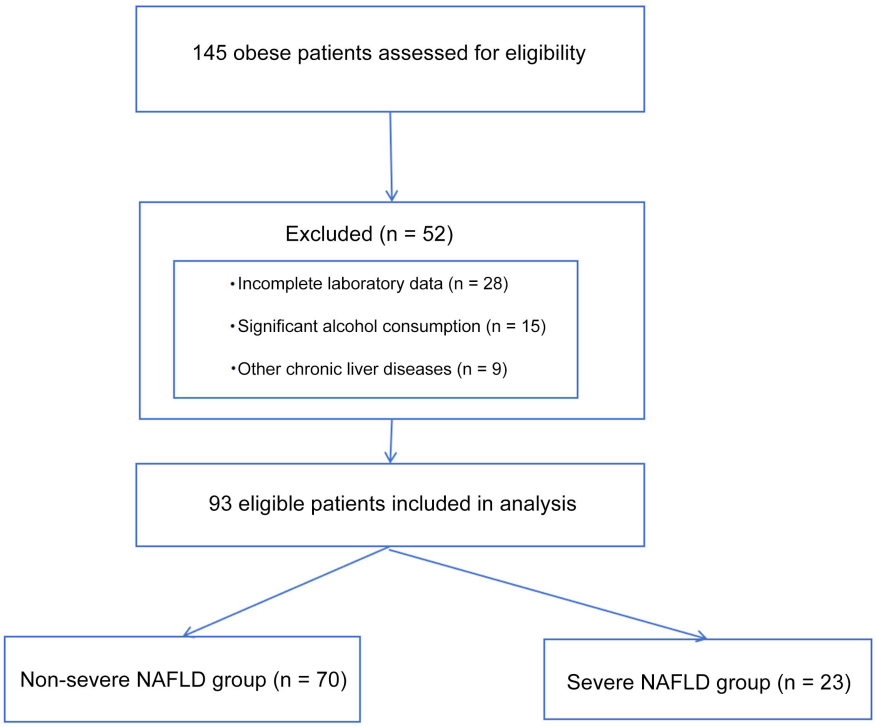

Figure S1. Flowchart of participant selection process.

A total of 145 obese patients were initially screened for eligibility. Of these, 52 were excluded for the following reasons: incomplete laboratory data (n = 28), significant alcohol consumption history (n = 15), and other chronic liver diseases (n = 9). A total of 93 eligible patients were ultimately included in the analysis and categorized into the non-severe NAFLD group (n = 70) and the severe NAFLD group (n = 23). NAFLD, non-alcoholic fatty liver disease.