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Original Article

Relationship between the triglyceride-glucose index and non-alcoholic fatty liver disease among individuals with atrial fibrillation

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Abstract: Background: The triglyceride-glucose (TyG) index serves as a reliable surrogate marker of insulin resistance. Accumulating evidence links an elevated TyG index to increased cardiovascular risk, but its relationship with nonalcoholic fatty liver disease (NAFLD) in atrial fibrillation (AF) patients remains underexplored. This study aimed to investigate the association between the TyG index and NAFLD incidence among AF patients. Methods: We conducted a retrospective analysis of 298 AF patients from a single tertiary care center. NAFLD was diagnosed via hepatic ultrasonography after secondary causes were excluded. TyG index was calculated as ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2]. Logistic regression models were employed to assess the independent association between the TyG index and NAFLD. Results: The NAFLD prevalence was 27.9% (83/298). Patients with NAFLD presented significantly greater TyG index values (8.90 ± 0.05 vs. 8.62 ± 0.04; P < 0.001). After multivariable adjustment, each 1-unit increment in the TyG index independently predicted NAFLD (OR 2.194, 95% CI 1.149-4.190; P = 0.017). Receiver operating characteristic curve analysis demonstrated moderate discriminative ability (AUC 0.667, 95% CI 0.601-0.732). A dose-response relationship was observed (P for trend < 0.001), with NAFLD incidence increasing progressively across TyG tertiles (12.87%, 29.81%, and 40.82%). Conclusions: An elevated TyG index is an independent risk factor for NAFLD in AF patients, suggesting a shared metabolic pathophysiology between insulin resistance, hepatic steatosis, and cardiac arrhythmia. The TyG index may serve as a readily available tool for NAFLD risk stratification in this high-risk population.

Keywords: Triglyceride-glucose index, nonalcoholic fatty liver disease, atrial fibrillation, insulin resistance, metabolic syndrome

Introduction

Nonalcoholic fatty liver disease (NAFLD) is one of the most common chronic liver disorder, with a global prevalence of approximately 25% and comparable rates reported in China [1]. NAFLD is the hepatic sign of a systemic inflammatory syndrome that is caused metabolically and is linked to a higher risk of heart diseases, such as atrial fibrillation (AF) [2]. AF, the most common type of sustained cardiac arrhythmia, affects more than 37 million people worldwide, and its incidence is increasing as the population ages [3, 4]. Cai et al. analyzed six cohort studies and reported that NAFLD was independently associated with a greater risk of AF events [5]. Insulin resistance (IR) is a key pathophysiological mechanism linking NAFLD and AF. In patients with IR, the activity of lipase increases, and the levels of long-chain fatty acids (LCFAs) in portal vein blood increase, resulting in more LCFAs in liver cells, thereby exacerbating NAFLD [6]. IR is also a risk factor for the onset of AF [7]. IR enhances the proinflammatory response of cardiomyocytes through C-reactive protein and reactive oxygen species. This results in myocardial remodeling, atrial enlargement and autonomic neuropathy, consequently facilitating the onset of AF [8, 9]. A recent systematic review and meta-analysis of

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nine observational studies revealed a significant association between NAFLD and a twofold increase in the risk of AF [10]. Consequently, as an additional risk factor for both AF and NAFLD, it is imperative to examine the relationship between IR and NAFLD in patients with AF.

The TyG index is an easy and accurate way to measure IR. It is calculated as ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2] [11]. Recent studies indicate that the TyG index predicts the development of type 2 diabetes, cardiovascular diseases such as AF, and NAFLD in the general population [6]. Nonetheless, information regarding the correlation between the TyG index and NAFLD in patients with AF is scarce. Due to the increased systemic inflammation, oxidative stress, and hepatic congestion associated with AF, it is essential to ascertain whether the TyG index can accurately detect NAFLD in this particular population.

This study sought to examine the correlation between the TyG index and NAFLD in patients with AF. By clarifying this relationship, we aimed to offer a cost-effective, easily implementable instrument for metabolic liver risk stratification in AF patients, thereby enhancing their cardiovascular and hepatic outcomes.

Methods

Study population

We conducted a retrospective analysis of AF patients who were admitted to the Cardiology Department of General Hospital of Ningxia Medical University consecutively from January 2022 to December 2024. The inclusion criteria were as follows: (1) Patients with AF; and (2) participants aged 18 years or above. The exclusion criteria consisted of: (1) participants under 18 years old; (2) Those who consumed excessive alcohol (men ≥ 30 g per day, women ≥ 20 g per day); (3) participants with missing data on fasting triglycerides, blood glucose, and NAFLD; and (4) participants infected with hepatitis B virus and/or hepatitis C virus.

Clinical and laboratory assessments

Baseline demographics, anthropometric measurements, and medical history were collected. Body mass index (BMI) was calculated as weight in kilograms divided by height in meter square. Venous blood samples were obtained after overnight fasting for determination of complete blood count, liver enzymes, renal function, lipid profile, fasting glucose, and thyroid function.

Statistical analysis

The data were analyzed using Statistical package for the social sciences (SPSS for Windows, version 27, IBM corp., Armonk, NY, USA), R version 4.0.1 and Graphpad prism 9.5 software. The normally distributed measurement data are expressed as the means ± standard deviations. The independent sample t test was used for comparisons between groups; for the measurement data that did not follow a normal distribution, nonparametric tests were used for comparisons between groups, and the median M (P25, P75) was used for representation; for the count data, percentages were used for representation, and the χ^2 test was used for comparisons between groups. Logistic regression analyses were conducted to evaluate the independent association between the TyG index and NAFLD. Restricted cubic spline regression was employed to investigate possible nonlinear associations. Receiver operating characteristic (ROC) curves were created to evaluate prediction efficacy. A two-sided P value ≤ 0.05 was considered statistically significant.

Results

Baseline characteristics

Among 298 enrolled AF patients (mean age 67.4 ± 9.2 years; 59.7% male), 83 (27.9%) fulfilled the diagnostic criteria for NAFLD. **Table 1** summarizes the baseline characteristics stratified by NAFLD status. Compared with their non-NAFLD counterparts, patients with NAFLD were younger [63 (57-70) vs. 69 (60-74) years; P = 0.003] and had a higher BMI [27.08 (24.54-29.30) vs. 24.22 (22.20-26.26) kg/m^2; P < 0.001]. No significant differences in sex distribution, ethnicity, smoking status, alcohol consumption, hypertension, diabetes, or AF type were observed between the groups.

Laboratory parameters

Table 2 presents the comparative laboratory findings of all the included patients. The TyG index values were elevated (8.90 ± 0.05 vs. 8.62 ± 0.04; P < 0.001). NAFLD patients exhibited higher white blood cell counts [6.51 (5.43-

Table 1. Comparison of baseline characteristics between the two groups

Variables	NAFLD (N = 83)	Normal (N = 215)	t/Z//X^2	*P*
Male, n (%)	54 (65.1%)	124 (57.7%)	1.358	0.244
Age/years	63 (57,70)	69 (60,74)	2.985	0.003
Ethnicity, n (%)			1.083	0.298
Hui	14 (16.9%)	48 (22.3%)		
Han	69 (83.1%)	167 (77.7%)		
Smoking, n (%)	15 (18.1%)	62 (28.8%)	3.621	0.057
Drinking, n (%)	3 (3.6%)	18 (8.3%)	2.069	0.150
Hypertension, n (%)	53 (63.9%)	134 (62.3%)	0.060	0.807
Diabetes, n (%)	22 (26.5%)	53 (24.7%)	0.109	0.741
CHD, n (%)	33 (39.8%)	113 (52.6%)	3.296	0.048
BMI (kg/m^2)	27.08 (24.54, 29.30)	24.22 (22.20, 26.26)	6.351	< 0.001
Atrial Fibrillation Type, n (%)			3.132	0.077
Paroxysmal	43 (51.8%)	87 (40.5%)		
Persistent	40 (48.2%)	128 (59.5%)		

NAFLD: nonalcoholic fatty liver disease, CHD: coronary heart disease, BMI: body mass index.

8.16) vs. 5.95 (4.99-7.45) × 10^9/L; *P* = 0.016], lymphocyte counts [1.98 (1.36-2.36) vs. 1.62 (1.10-1.99) × 10^9/L; *P* < 0.001], hemoglobin levels [149 (136-160) vs. 139 (126-152) g/L; *P* = 0.001], and platelet counts [207 (178-255) vs. 179 (144-230) × 10^9/L; *P* < 0.001]. Moreover, they demonstrated lower creatinine [5.53 (4.81-6.84) vs. 6.75 (5.40-8.74) μmol/L; *P* < 0.001] and urea levels [68.30 (57.80-78.70) vs. 76.10 (64.60-90.60) mmol/L; *P* < 0.001], alongside elevated ALT [27.2 (20.4-36.5) vs. 23.8 (17.4-33.7) U/L; *P* = 0.021], albumin [40.8 (38.4-44.3) vs. 38.8 (35.2-41.4) g/L; *P* < 0.001], triglycerides [1.54 (1.23-2.09) vs. 1.09 (0.89-1.50) mmol/L; *P* < 0.001], total cholesterol [3.63 (3.21-4.44) vs. 3.52 (2.81-4.08) mmol/L; *P* = 0.032], LDL cholesterol [2.19 (1.64-2.97) vs. 1.98 (1.49-2.60) mmol/L; *P* = 0.015], and T3 levels [1.11 (0.99-1.27) vs. 1.01 (0.84-1.20) ng/mL; *P* < 0.001]. Notably, D-dimer concentrations were significantly lower [0.26 (0.16-0.51) vs. 0.43 (0.23-1.15) μg/mL; *P* < 0.001], whereas CHA_2DS_2-VASc scores were reduced [2 (2-4) vs. 3 (2-4); *P* = 0.007].

As shown in **Figure 1**, when a TyG index cutoff of ≥ 8.76 was used to define IR [12], the prevalence of NAFLD was 35.90% in the IR group, which was 1.58-fold higher than that in the non-IR group (22.65%, *P* < 0.05). This finding underscores that an elevated TyG index, as a marker of IR, is strongly associated with an increased risk of NAFLD.

When grouped by TyG index tertiles, the prevalence of NAFLD increased stepwise across T1 (12.87%), T2 (30.00%), and T3 (41.24%) (*P* for trend < 0.001). This graded association indicates that a higher TyG index tertile is strongly linked to an elevated risk of NAFLD. The prevalence of NAFLD was based on the tertiles of the TyG index. The classification of TyG tertiles was as follows: T1 (≤ 8.43), T2 (8.44-8.93), and T3 (≥ 8.94), as shown in **Figure 2**.

Association between TyG index and NAFLD

Univariate logistic regression revealed that the TyG index significantly predicted NAFLD (OR 2.756, 95% CI 1.676-4.534; *P* < 0.001). After multivariable adjustment, this association persisted (OR 2.194, 95% CI 1.149-4.190; *P* = 0.017), as shown in **Figure 3**. Additionally, BMI (OR 1.340 per kg/m^2, 95% CI 1.156-1.554; *P* < 0.001), albumin (OR 1.106 per g/L, 95% CI 1.031-1.187; *P* = 0.005), and LDL cholesterol (OR 3.156 per mmol/L, 95% CI 1.011-9.856; *P* = 0.048) emerged as independent predictors.

Dose-response relationship

Restricted cubic spline analysis demonstrated a significant nonlinear association between the TyG index and NAFLD risk (*P* for overall < 0.001; *P* for nonlinearity = 0.044), with risk acceleration beginning at TyG ≈ 8.64, as shown in **Figure 4**.

Table 2. Comparison of laboratory indicators between the two groups

Parameters	NAFLD (N = 83)	Normal (N = 215)	*t/z*	*P*
WBC/(*10^9/L)	6.51 (5.43, 8.16)	5.95 (4.99, 7.45)	2.411	0.016
NEUT/(*10^9/L)	3.70 (2.77, 4.97)	3.60 (2.79, 4.92)	0.763	0.445
LYM/(*10^9/L)	1.98 (1.36, 2.36)	1.62 (1.10, 1.99)	3.690	< 0.001
MXD/(*10^9/L)	0.51 (0.38, 0.69)	0.49 (0.40, 0.66)	0.493	0.622
EOS/(*10^9/L)	0.09 (0.04, 0.17)	0.08 (0.04, 0.15)	1.176	0.239
BASO/(*10^9/L)	0.03 (0.02, 0.04)	0.03 (0.03, 0.04)	1.815	0.069
HGB/(g/L)	149 (136, 160)	139 (126, 152)	3.324	0.001
PLT/(*10^9/L)	207 (178, 255)	179 (144, 230)	3.581	< 0.001
CREA/(μmol/L)	5.53 (4.81, 6.84)	6.75 (5.40, 8.74)	4.366	< 0.001
UREA/(mmol/L)	68.30 (57.80, 78.70)	76.10 (64.60, 90.60)	3.481	< 0.001
FBG/(mmol/L)	5.50 (5.06, 6.35)	5.77 (4.91, 6.84)	0.690	0.490
UA/(μmol/L)	335 (273, 406)	350 (283, 431)	1.395	0.163
Total Bilirubin (g/L)	16.3 (11.6, 22.9)	16.2 (11.6, 24.4)	0.499	0.653
AST (U/L)	26.2 (20.8, 37.1)	24.5 (19.6, 33.5)	1.177	0.239
ALT (U/L)	27.2 (20.4, 36.5)	23.8 (17.4, 33.7)	2.316	0.021
GGT (U/L)	29.6 (21.3, 45.4)	27.5 (19.6, 47.4)	0.961	0.336
ALB (g/L)	40.8 (38.4, 44.3)	38.8 (35.2, 41.4)	4.460	< 0.001
TG (mmol/L)	1.54 (1.23, 2.09)	1.09 (0.89, 1.50)	5.938	< 0.001
TC (mmol/L)	3.63 (3.21, 4.44)	3.52 (2.81, 4.08)	2.148	0.032
LDL (mmol/L)	2.19 (1.64, 2.97)	1.98 (1.49, 2.60)	2.426	0.015
HDL (mmol/L)	0.95 (0.77, 1.17)	0.97 (0.79, 1.17)	0.868	0.386
TSH (uIU/mL)	2.38 (1.31, 3.98)	1.90 (1.22, 3.44)	1.497	0.134
T3 (ng/mL)	1.11 (0.99, 1.27)	1.01 (0.84, 1.20)	3.654	< 0.001
T4 (ug/dL)	8.0 (6.7, 8.8)	8.0 (6.7, 9.3)	0.543	0.587
D-D (ug/mL)	0.26 (0.16, 0.51)	0.43 (0.23, 1.15)	3.508	< 0.001
CHADS-VASc Score	2 (2, 4)	3 (2, 4)	2.717	0.007
TyG index	8.90 ± 0.05	8.62 ± 0.04	4.47	< 0.001

NAFLD: Nonalcoholic Fatty Liver Disease, WBC: white blood cell, NEUT: neutrophil, LYM: lymphocyte, MXD: medium-sized unclassified cell, EOS: eosinophil, BASO: basophil, HGB: hemoglobin, PLT: platelet, CREA: creatinine, UREA: urea, FBG: fasting blood glucose, UA: uric acid, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma-glutamyl transferase, ALB: albumin, TG: triglyceride, TC: total cholesterol, LDL: low-density lipoprotein cholesterol, HDL: high-density lipoprotein cholesterol, TSH: thyroid stimulating hormone, T3: triiodothyronine, T4: thyroxine, D-D: d-dimer, TyG index: the triglyceride-glucose index.

Predictive performance

ROC curve analysis revealed an area under the curve (AUC) of 0.667 (95% CI 0.601-0.732) for the TyG index alone in the prediction of NAFLD, as shown in **Figure 5**. While indicating moderate discriminative capacity, this suggests potential utility for initial risk screening in resource-limited settings.

Discussion

This study aimed to examine the relationship between the TyG index and NAFLD in patients with AF. Our primary findings indicate that a raised TyG index is an independent risk factor for NAFLD in our population cohort, with each unit increase corresponding to about 2.2-fold greater odds. Moreover, we noted a graded, dose-dependent relationship in which increasing TyG tertiles were associated with heightened NAFLD risk. These data highlight the essential role of IR, as measured by the TyG index, in the pathophysiological relationship between hepatic steatosis and cardiac arrhythmogenesis.

The observed TyG index threshold of 8.64 (above which NAFLD risk increases nonlinearly) is consistent with previously reported values [13]. The primary causes of IR include impaired insulin uptake and utilization efficiency, along

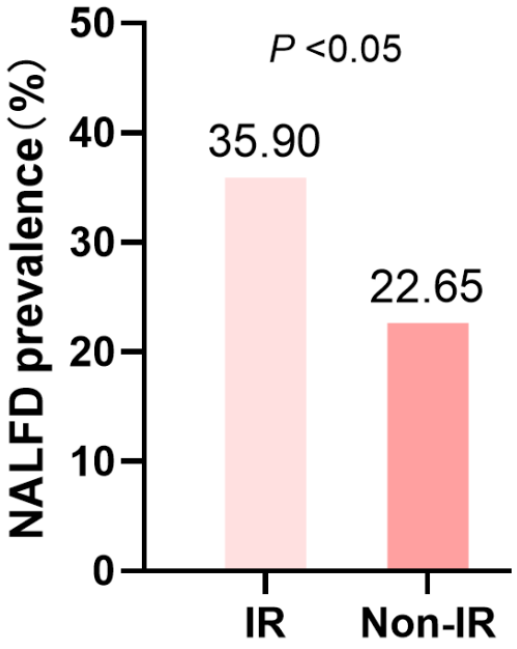


Figure 1. Based on the TyG index cutoff value of ≥ 8.76, the subjects were divided into the insulin-resistant group (n = 117) and the non-insulin-resistant group (n = 181), with respective numbers of NAFLD cases of n = 42 and n = 41.

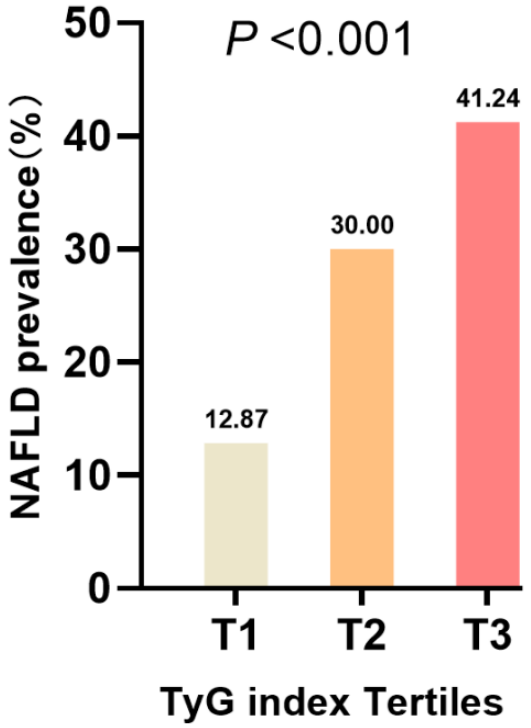
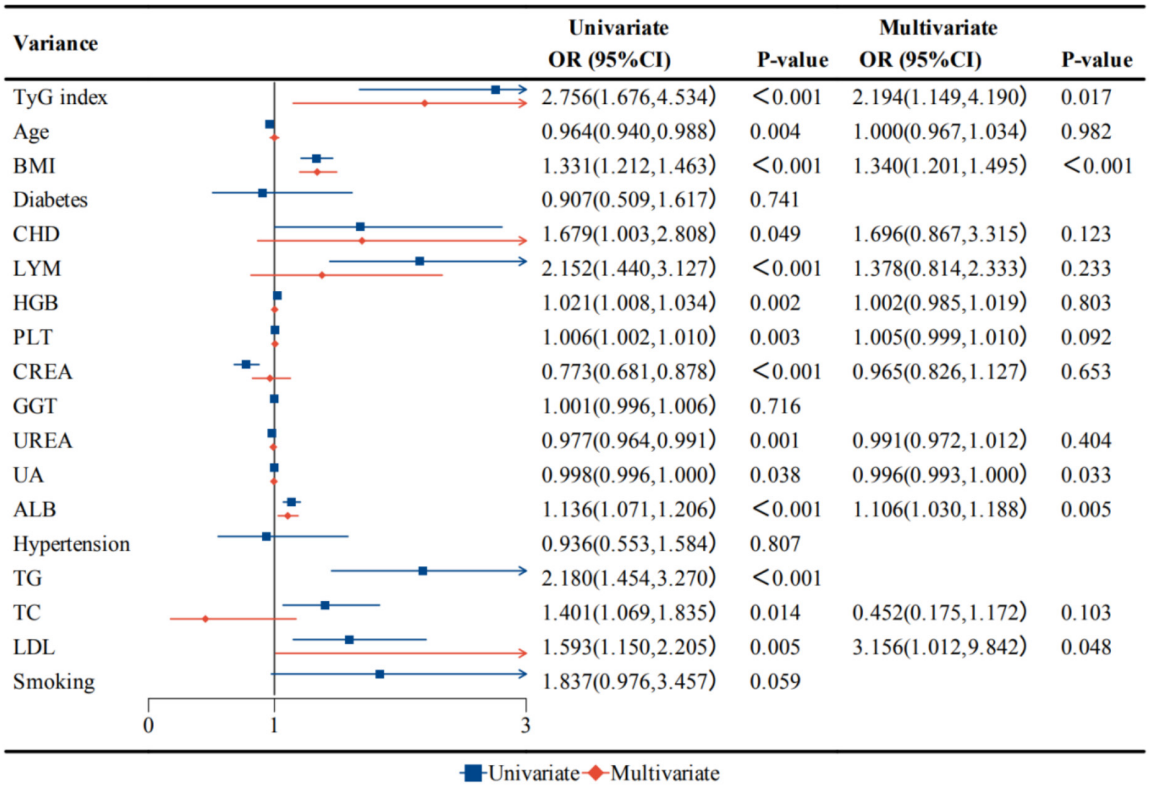


Figure 2. When grouped based on the tertiles of TyG index: T1 (≤ 8.43), T2 (8.44-8.93), and T3 (≥ 8.94). The prevalence of NAFLD was found to exhibit a progressive upward trend in T1 (n = 13), T2 (n = 31), and T3 (n = 39) stages, as calculated using the Kruskal-Wallis H test (P < 0.001).

with compensatory hyperinsulinemia [14]. First, IR compromises tissue insulin sensitivity and glucose metabolism, impairing the function of organs, including the liver and heart [15]. Second, IR is correlated with chronic inflammation, potentially contributing to NAFLD and AF [16]. Finally, IR-induced oxidative stress promotes stellate cell proliferation and inflammatory hepatic macrophage activation, thereby contributing to NAFLD development [17]. The AUC value we calculated was 0.667. While not exceptionally high, it still demonstrates a certain advantage over similar studies in the general population. This finding indicates that when combined with existing metabolic parameters, it offers additional value.

In both cross-sectional population studies and longitudinal cohort studies, nonalcoholic fatty liver disease was associated with a significantly increased risk of AF [18]. A meta-analysis encompassing 19 studies demonstrated that NAFLD independently correlated with a high risk of AF [5]. The observed association between the TyG index and NAFLD in AF patients may arise through multiple mechanisms. IR promotes hepatic lipogenesis by impairing insulin signaling, leading to excessive triglyceride accumulation and NAFLD development [19]. Concurrently, IR triggers systemic inflammation, oxidative stress, and endothelial dysfunction, collectively promoting the remodeling of atrial structure and electrical activity, thereby increasing the risk of AF onset [20]. This bidirectional relationship forms a vicious cycle: NAFLD exacerbates IR, amplifying susceptibility to AF, and vice versa. Our finding of lower CHA_2DS_2-VASC scores among NAFLD patients may appear counterintuitive but likely reflects the younger age of this subgroup, underscoring the need for lifelong risk assessment rather than short-term prediction models.

Clinically, incorporating the TyG index into routine assessments of AF patients facilitates early identification of those with occult NAFLD, enabling timely lifestyle and pharmacological interventions to mitigate hepatic and cardiovascular sequelae [21]. Given its simplicity and widespread availability, the TyG index has particular advantages in resource-limited settings



Variance	Univariate OR (95%CI)	P-value	Multivariate OR (95%CI)	P-value
TyG index	2.756(1.676,4.534)	<0.001	2.194(1.149,4.190)	0.017
Age	0.964(0.940,0.988)	0.004	1.000(0.967,1.034)	0.982
BMI	1.331(1.212,1.463)	<0.001	1.340(1.201,1.495)	<0.001
Diabetes	0.907(0.509,1.617)	0.741		
CHD	1.679(1.003,2.808)	0.049	1.696(0.867,3.315)	0.123
LYM	2.152(1.440,3.127)	<0.001	1.378(0.814,2.333)	0.233
HGB	1.021(1.008,1.034)	0.002	1.002(0.985,1.019)	0.803
PLT	1.006(1.002,1.010)	0.003	1.005(0.999,1.010)	0.092
CREA	0.773(0.681,0.878)	<0.001	0.965(0.826,1.127)	0.653
GGT	1.001(0.996,1.006)	0.716		
UREA	0.977(0.964,0.991)	0.001	0.991(0.972,1.012)	0.404
UA	0.998(0.996,1.000)	0.038	0.996(0.993,1.000)	0.033
ALB	1.136(1.071,1.206)	<0.001	1.106(1.030,1.188)	0.005
Hypertension	0.936(0.553,1.584)	0.807		
TG	2.180(1.454,3.270)	<0.001		
TC	1.401(1.069,1.835)	0.014	0.452(0.175,1.172)	0.103
LDL	1.593(1.150,2.205)	0.005	3.156(1.012,9.842)	0.048
Smoking	1.837(0.976,3.457)	0.059		

Figure 3. Forest Plot of Univariate and Multivariate Logistic Regression Analyses for Identifying Risk Factors Associated with NAFLD. BMI: body mass index, CHD: coronary heart disease, LYM: lymphocyte, HGB: hemoglobin, PLT: platelet, CREA: creatinine, UREA: urea, UA: uric acid, ALB: albumin, TG: triglyceride, TC: total cholesterol, LDL: low-density lipoprotein cholesterol.

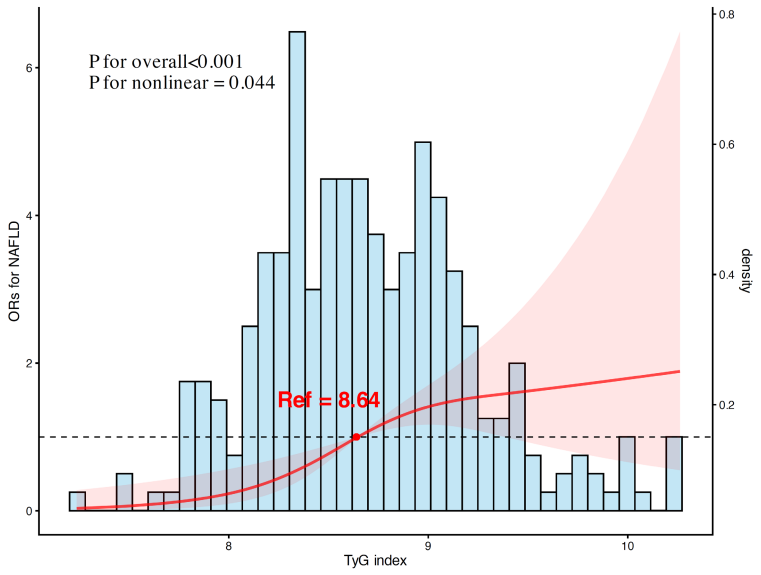


Figure 4. NAFLD prevalence distribution odds ratio and histogram based on TyG index.

where advanced imaging or specialized biomarkers may be inaccessible. Future studies should evaluate whether targeted reduction of the TyG index through lifestyle modifications or pharmacotherapy can concurrently improve the burden of both NAFLD and AF.

Limitations

This study has several limitations that warrant consideration. The cross-sectional design prevents the determination of causality or temporal correlations. Secondly, hepatic ultrasonography, although effective for extensive screening, exhibits insufficient sensitivity for mild steatosis and is unable to differentiate between simple steatosis and non-alcoholic steatohepatitis. Ultimately, our single-center cohort may restrict generalizability to wider groups, needing multi-ethnic validation.

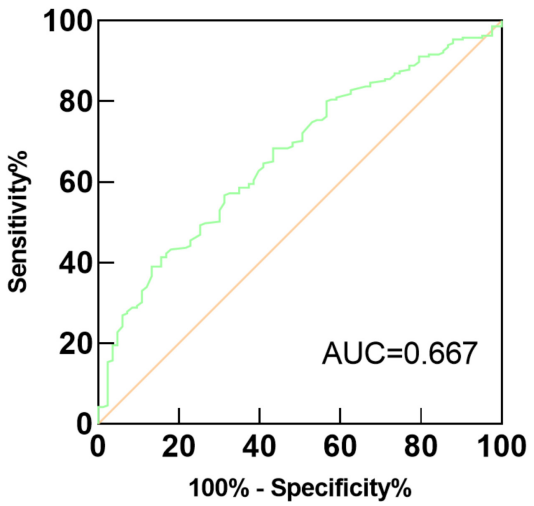


Figure 5. NAFLD Receiver operative characteristic curves and corresponding areas under the curve.

Conclusions

An increased TyG index serves as a notable independent predictor of NAFLD in AF patients, underscoring the pivotal role of IR in driving metabolic-cardiovascular comorbidity. These findings support the incorporation of the TyG index into comprehensive risk assessment frameworks for AF patients, potentially informing tailored preventative efforts aimed at common metabolic abnormalities. Longitudinal studies are necessary to determine if therapeutic manipulation of the TyG index can lead to enhanced hepatic and cardiovascular outcomes.

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Disclosure of conflict of interest

None.

Abbreviations

NAFLD, nonalcoholic fatty liver disease; TyG index, the triglyceride-glucose index; AF, atrial fibrillation; IR, Insulin resistance; ROC, receiver operating characteristic; AUC, area under the curve; LCFA, long-chain fatty acids; CHD, coronary heart disease; BMI, body mass index; WBC, white blood cell; NEUT, neutrophil; LYM, lymphocyte; MXD, medium-sized unclassified cell; EOS, eosinophil; BASO, basophil; HGB, hemoglobin; PLT, platelet; CREA, creatinine; UREA, urea; FBG, fasting blood glucose; UA, uric acid; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; ALB, albumin; TG, triglyceride; TC, total cholesterol; LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; TSH, thyroid stimulating hormone; T3, triiodothyronine; T4, thyroxine; D-D, d-dimer.

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