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Triglyceride-glucose index predicts adverse cardiovascular events in patients with H-type hypertension combined with coronary heart disease: a retrospective cohort study

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Abstract

Background The triglyceride–glucose index (TyG index) is a reliable surrogate for insulin resistance (IR) in individuals with type 2 diabetes mellitus and is associated with cardiovascular disease. Recent studies have reported that H-type hypertension is likewise a predictor of adverse events in patients with coronary heart disease (CHD). However, the relationship between the TyG index and prognosis in patients with H-type hypertension combined with CHD has not yet been reported. In this study, we investigated the relationship between the TyG index and adverse outcomes in patients with H-type hypertension combined with CHD.

Methods This was a single-center retrospective cohort study that included patients who were diagnosed with H-type hypertension combined with CHD between 2018 and 2023 at Beijing Anzhen Hospital of Capital Medical University. The TyG index was divided into three groups according to tertiles. Kaplan–Meier curves were used to analyze the cumulative risk of major adverse cardiovascular events (MACEs), and ROC curve analysis was performed by the log-rank test. Cox proportional hazards regression models were adopted to explore the relationship between the TyG index and MACEs. C-statistics, NRI, and IDI were used to evaluate the incremental predictive ability of the TyG index.

Results A total of 546 patients were included, with a median follow-up time of 39.00 ± 0.69 months, and 73 MACEs occurred, with higher tertiles of the TyG index associated with a higher cumulative risk of MACEs (log-rank, P = 0.001). After adjusting for confounders, the fully adjusted ORs (95% CI) for T2 and T3 of the TyG index, with the lowest tertile as reference, were 1.64 (0.80–3.36) and 2.43 (1.19–4.97), respectively. The addition of the TyG index led to a significant improvement in the overall predictive power of the baseline model. [C-statistic increased from 0.63 to 0.66, p = 0.031; continuous NRI (95% CI) 0.13 (0.001–0.276), p = 0.038; IDI (95% CI) 0.01 (0.000–0.031), p = 0.047].

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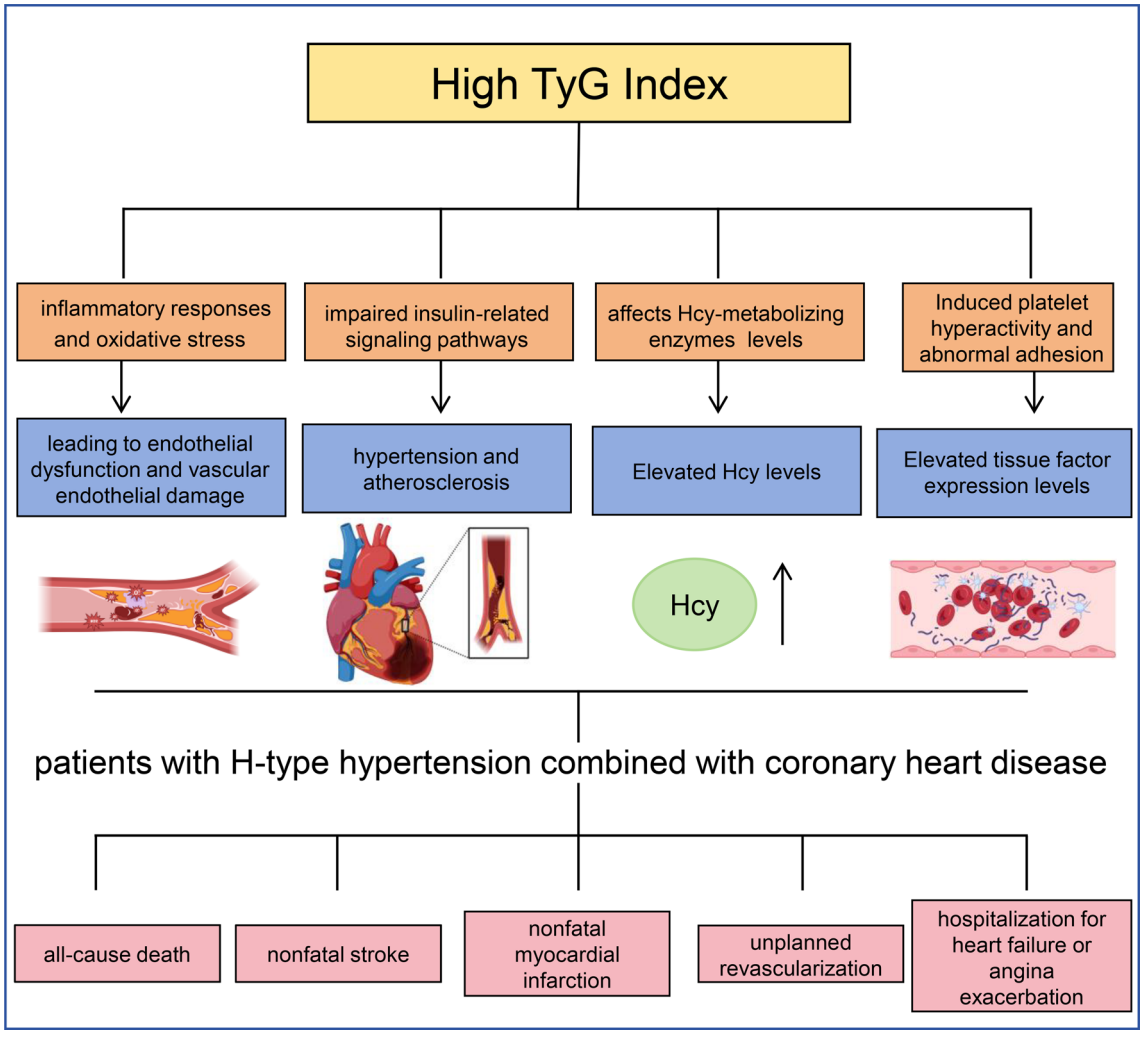
Conclusion The TyG index may be an independent risk factor for predicting adverse postoperative cardiovascular events in patients with H-type hypertension combined with CHD, indicating its potential significance in improving risk stratification strategies.

Keywords TyG index, H-type hypertension, Coronary heart disease, Major adverse cardiovascular events

Graphical Abstract



Background

Coronary heart disease (CHD) is the leading cause of morbidity and mortality worldwide and is characterized by multiple causes of lipid plaque formation, leading to chronic inflammatory, fibroproliferative disease [1]. The incidence of patients with CHD has been increasing annually, favoring a younger age group. H-type hypertension is a specific type of hypertension characterized by essential hypertension and hyperhomocysteinemia. Homocysteine (Hcy) is a sulfur-containing amino acid formed during the metabolism of methionine (Met) to cysteine. Patients with hyperhomocysteinemia have impaired Met metabolism due to defects in metabolic enzyme-encoding genes or deficiencies in certain vitamin cofactors, resulting in elevated Hcy levels. Although the H-type hypertension population represents only a fraction of the primary hypertension population, the actual number of people suffering from this disease in China is often larger than we think due to the large population base. According to epidemiological statistics, H-type hypertension patients account for about 80% of essential hypertension patients, and their risk of cardiovascular complications is 5 times higher than that of essential hypertension patients, and approximately 25–30 times greater than that in the normal population [2]. H-type hypertension not only increases the risk of damage to

brain, heart, kidney and other target organs, but also accelerates the occurrence and development of arteriosclerosis, which is easy to lead to coronary heart disease (CHD) and other adverse cardiovascular events, which brings a heavy burden to the global medical economy and health undertakings [3]. Early recognition and treatment of hyperhomocysteinemia is key to reducing the poor prognosis of patients with H-type hypertension and improve cardiovascular outcomes.

Insulin resistance (IR) is a pathological state characterized by insulin receptor abnormalities and reduced insulin sensitivity, which disrupts glucose and lipid metabolism, resulting in hyperglycemia, hypertension, dyslipidemia, and other pathologies [4, 5]. The glucose–triglyceride (TyG) index is a new metabolic surrogate index that was first used to assess IR in type 2 diabetes mellitus patients and is an independent predictor of major adverse cardiovascular events in patients with type 2 diabetes mellitus [6]. The TyG index is derived from the patient's fasting blood glucose and fasting triglyceride calculations, and it possesses the advantages of being easily accessible and inexpensive. As research continues, the TyG index is also able to predict the risk of adverse cardiovascular disease in nondiabetic populations. In recent years, studies have shown that the TyG index is significantly associated with the prognosis of hypertension [7, 8], coronary heart disease [9], diabetes mellitus [10, 11], stroke [12, 13], and heart failure [14, 15]. Several studies have shown that the TyG index is associated with hypertension and that changes in the TyG index have a linear dose–response relationship with changes in blood pressure, which can predict the risk of developing hypertension in a population with relative accuracy [8, 16]. A cohort study revealed that the TyG index was associated with poor prognosis in patients with coronary artery disease combined with hypertension, with patients with a high TyG index having a poorer prognosis [17]. A cross-sectional study revealed that the TyG index is a reliable predictor of the degree of coronary artery stenosis in patients with H-type hypertension and plays an important role in risk stratification and intervention [18]. CHD, H-type hypertension, and metabolic syndrome are independent diseases, but there is a common pathophysiological basis, in which metabolic disorders play a significant pathophysiological role [19–21]. TyG is a commonly used and novel indicator reflecting metabolic disorders, which is significantly correlated with a variety of cardiovascular diseases, and can be used as a prognostic indicator, but whether it is able to predict the prognosis of patients with H-type hypertension combined with CHD is not yet clear.

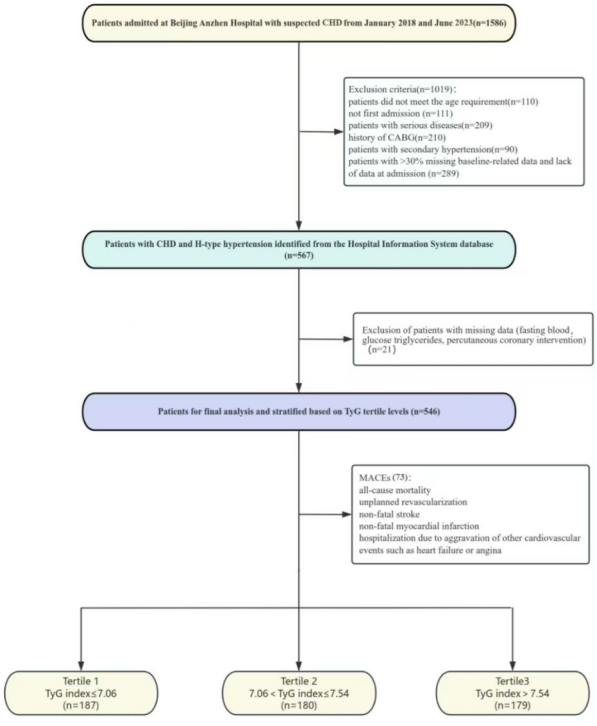


Fig. 1 Flow chart of the enrolled patients. TyG, triglyceride–glucose index; MACEs, major adverse cardiovascular events; CHD, coronary heart disease

The aim of this study was to investigate the relationship between the TyG index and the risk of MACEs in H-type hypertension combined with CHD based on accessible real clinical data. The results of this study may help to identify the high-risk patients among them at an early stage and thus provide new perspectives to improve the prognosis of patients.

Methods

Study design and participants

This was a single-center retrospective cohort study that included a total of 546 patients who were diagnosed with H-type hypertension combined with CHD from 2018 to 2023 at Beijing Anzhen Hospital of Capital Medical University. The detailed screening process is shown in Fig. 1. The inclusion criteria for this study were as follows: (1) aged 18–80 years; (2) diagnosed with both CHD and H-type hypertension. The exclusion criteria were as follows: coronary artery bypass grafting (CABG); cardiogenic shock; comorbidities with malignant tumors; other patients with a life expectancy of less than 1 year; refractory end-stage heart failure (LVEF < 30%); suspected familial hypertriglyceridemia (triglycerides > 5.65 mmol/L); severe hepatic and renal insufficiency (estimated Child–Pugh score ≥ 7 and estimated glomerular filtration rate (eGFR < 30 mL/min/1.73m^2); nonfirst admission; secondary hypertension; missing baseline-related and follow-up data; and severe structural heart disease requiring intervention. The units of FBG and TG were first converted from mmol/L to mg/dL, and the

TyG index was calculated as ln [TG (mg/dl) × glucose (mg/dl)/2]. The TyG indices for the three distinct groups were as follows: T1: ≤ 7.06; T2: > 7.06, ≤ 7.54; and T3: > 7.54. This retrospective study adhered to the principles outlined in the Declaration of Helsinki. Given its retrospective nature, the requirement for obtaining informed consent was exempted by the institutional review board, ensuring that all identifying information pertaining to patients was appropriately concealed and protected.

Data collection and definitions

All the subjects completed blood collection in the early morning under fasting conditions, and blood samples were tested on the same day in a standard laboratory. Clinical data were collected from medical records by trained clinicians who were unaware of the purpose of the study. The data included demographic characteristics, clinical risk factors, laboratory indices, peripheral arterial disease characteristics, and Percutaneous coronary intervention (PCI) procedure information. The demographic characteristics included age, sex, body mass index (BMI) and so on. Clinical risk factors included heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP), a history of smoking, alcohol consumption, diabetes mellitus, atrial fibrillation, chronic kidney disease (CKD), previous heart attack, previous stroke, and a family history of cardiovascular disease. The record of the above factors is determined by sell-reported or clinical testing.CHD was defined as the presence of at least one of the following conditions:: (1) percutaneous coronary angiography or computed tomographic angiography (CTA) examination showed that at least one coronary artery trunk or primary branch had ≥ 50% stenosis; (2) typical exertional angina symptoms with positive stress test (electrocardiogram stress test, stress echocardiography or nuclide myocardial stress imaging); (3) previously diagnosed myocardial infarction (MI) or unstable angina pectoris [22].The laboratory parameters included LVEF, RBC, WBC, PLT, NE, BNP, Glu, HbA1C%, hs-CRP, Hb, Alb, eGFR, Urea, UA, Cr, TG, TC, HDL-C, LDL-C, Hcy, ALT, and AST. The PCI procedure information included the coronary lesion site, number of coronary lesions, and number of stents. The units of FBG and TG were first converted from mmol/L to mg/dL, and the TyG index was calculated as ln [TG (mg/dl) × glucose (mg/dl)/2]. Diabetes was defined as the presence of at least one of the following conditions: HbA1C% ≥ 6.5%; random blood glucose ≥ 11.1 mmol/L; fasting blood glucose ≥ 7.0 mmol/L; blood glucose ≥ 11.1 mmol/L 2 h after the oral glucose tolerance test [23]. Weight (kg)/ [height (m)]^2 was used to calculate body mass index (BMI).H-type hypertension was defined as essential hypertension with hyperhomocysteinemia, and hyperhomocysteinemia was defined as a plasma Hcy concentration higher than 10 or 15 μmol/L [21]. With respect to the definition of hyperhomocysteinemia, the value of 15 μmol/L was selected for this study. Dyslipidemia was diagnosed as LDL-C ≥ 3.4 mmol/L, HDL-C < 1.0 mmol/L, TG ≥ 1.7 mmol/L, or the use of lipid-lowering treatment.

Follow-up and endpoint events

All patients were followed up by professionally trained personnel through telephone communication, a medical history system, or outpatient review. Follow-up lasted from patient discharge until June 2024, unless withdrawal or death occurred. The primary study endpoint was the MACEs composite, which included all-cause death, nonfatal stroke, nonfatal myocardial infarction (MI), unplanned revascularization, and hospitalization for heart failure or angina exacerbation. Nonfatal myocardial infarction is defined as concurrent clinical evidence of acute myocardial ischemia because of myocardial injury, as evidenced by the myocardial marker troponin above the 99th percentile of the upper limit of the normal reference value [24]. Nonfatal stroke includes cerebral infarction and cerebral hemorrhage, which are diagnosed based on imaging. Unscheduled repeat revascularization was defined as ischemia-driven revascularization. If more than one event occurred during follow-up (all-cause death > nonfatal stroke > nonfatal myocardial infarction > unplanned repeat revascularization), the most severe event was considered the endpoint event. If an endpoint event occurred more than once, the first event after PCI was selected. All endpoint events were independently adjudicated by at least two experienced cardiologists.

Statistical analysis

Continuous variables are expressed as the means ± standard deviations or medians (interquartile spacings), and comparisons of normally distributed data were analyzed via one-way ANOVA. Kruskal–Wallis test was used for comparison of non-normally distributed data. Categorical variables are expressed as frequency counts (frequencies), and the chi-square test or Fisher's exact test was used. We used Cox proportional risk regression models to assess the relationships between the TyG index and outcomes, calculated hazard ratios (HRs), and expressed the study results as HRs with 95% confidence intervals (CIs). Three different models were developed. Model 1 was not adjusted. Model 2 was adjusted for age and sex. Model 3 was adjusted for the variables included in Model 2, as well as for RBC, HbA1c%, Hb, and Alb. Kaplan–Meier survival curves were used to describe the incidence of mortality in the different groups and were compared via log-rank tests. Restricted cubic spline curves (RCSs) were used to investigate possible nonlinear correlations between the TyG index and clinical outcomes. Subgroup analyses were used, including age (> 65

and ≤ 65 years), sex (male and female), diabetes mellitus (yes or no), BMI (< 25 kg/m^2 or ≥ 25 kg/m^2), CVD (yes or no), glycosylated hemoglobin (> 7%, ≤ 7%), LDL-C (> 1.5 mmol/L, ≤ 1.5 mmol/L), and the likelihood ratio test was used to assess the interaction between the different subgroups. Improvements in risk identification beyond the identified clinical risk variables (RBC, HbA1C%, Hb, Alb) were assessed through the C statistic, sustained net reclassification improvement (NRI), and integrated discrimination improvement (IDI) for the TyG index. All data were analyzed by SPSS version 27.0 and R version 3.6.2 to analyze. Statistical significance was defined as a two-tailed P value < 0.05.

Results

Baseline characteristics

Finally, 546 subjects who were diagnosed with H-type hypertension combined with CHD at Beijing Anzhen Hospital of Capital Medical University were included. The median follow-up time of the subjects was 39.00 ± 0.69 months, and among the 546 eligible subjects, 73 (13.37%) experienced MACEs during follow-up. The mean age of the subjects was 62.00 (53.0, 68.0) years, and 469 (85.90%) of them were male.

All the subjects were divided into 3 groups according to the TyG index, and the baseline characteristics of the 3 groups are shown in Table 1. The patients in the 3 groups in terms of MACE, age, diabetes mellitus status, stroke status, DBP, erythrocytes, leukocytes, BNP, glucose, glycated hemoglobin, hemoglobin, Alb, urea, UA, TG, TC, HDL, LDL, ALT, and oral hypoglycemic drugs were significantly different (P < 0.05), and the other indices were not significantly different. The prevalence of MACEs and previous medical history (diabetes, stroke) was greater in patients with a higher baseline TyG index but relatively younger patients. In addition, DBP values and laboratory indices (including erythrocytes, leukocytes, BNP, glucose, glycosylated hemoglobin, hemoglobin, Alb, urea, UA, TG, TC, HDL, LDL, and ALT) were greater in the T3 group than in the other two groups. The percentage of patients taking oral hypoglycemic drugs was significantly greater in the T3 group than in the other two groups.

Risk factors for MACEs

The baseline characteristics of the MACEs and non–MACEs groups are shown in Table 2. A total of 73 (13.37%) patients developed MACEs during the follow-up period. Compared with subjects without endpoints, those with endpoint events had elevated concentrations of erythrocytes, glycosylated hemoglobin, Hb, and Alb (P < 0.05).

Relationship between the TyG index and cardiovascular events in patients with H-type hypertension and CHD

During a median follow-up time of 39.00 ± 0.69 months, 73 patients (13.37%) experienced at least one endpoint event. The Kaplan–Meier curves for the cumulative risk of cardiovascular events by TyG index tertiles are shown in Fig. 2, with higher TyG indices corresponding to higher cumulative risk probabilities (log-rank test, p = 0.001) and the highest tertile of TyG indices corresponding to the highest probability of cardiovascular cumulative risk, regardless of the monthly follow-up time node.

The relationships between the TyG index and MACEs in patients with H-type hypertension and CHD are shown in Table 3. The risk of MACEs increased significantly with increasing TyG index tertiles, adjusted for different models. Model 3 revealed a 2.43-fold greater risk for patients with the highest TyG index than for those with the lowest TyG index (T2 vs. T1: HR 1.64, 95% CI 0.80–3.36, P = 0.175; T3 vs. T1: HR 2.43, 95% CI 1.19–4.97, P = 0.015). To visualize the relationship between the TyG index and cardiovascular risk, we constructed a RCS model (Fig. 3). The RCS model revealed a nonlinear relationship between the TyG index and MACEs (P for nonlinearity = 0,044). The critical value of the TyG index for predicting MACEs was 7.40. The RCS curve had an S shape, and when the TyG index exceeded 7.40, the HR of MACEs increased significantly as the TyG index increased (HR/SD1.65, 95% CI 1.35–2.38; P = 0.011). For TyG index ≤ 7.40, the HR per SD was 1.45 (95% CI 0.45 to 4.68, P = 0.531).

ROC curve analysis of the value of the TyG index

The ROC curve analysis results are shown in Fig. 4. The AUC of the baseline risk model (included) that predicted the occurrence of MACEs in patients with H-type hypertension with CHD was 0.661 (P < 0.05). There was a slight increase in the AUC of the new model in which the TyG index was added to the baseline risk model (AUC: baseline risk model + TyG index, 0.661 vs baseline risk model, 0.629, P < 0.05).

The incremental predictive value of the TyG index

The incremental predictive value of the TyG index for MACEs is shown in Table 4. The addition of the TyG index had a significant effect on predicting the prognostic value of MACEs when the baseline risk model was compared with the established risk factors [C statistic: baseline risk model 0.63 (0.592–0.662) vs. new model with TyG addition 0.66 (0.621–0.681), P = 0.031; NRI: 13% improvement, P = 0.038; IDI: 1% improvement, P = 0.047].

Table 1 Baseline characteristics according to tertiles of the triglyceride–glucose index

Viable	Total	TyG index level			P value
		T1 (≤ 7.06)	T2 (> 7.06, ≤ 7.54)	T3 (> 7.54)	
N (%)	546	187	180	179	–
Age, y	62.00 (53.00,68.00)	64.00 (55.00,69.00)	62.50 (54.00,68.00)	60.00 (49.00,66.00)	0.000**
Male, n (%)	469 (85.90)	169 (90.37)	153 (85.00)	147 (82.12)	0.070
BMI, kg/m^2	26.22 (24.20,28.30)	25.35 (23.30,27.20)	26.73 (24.80,29.10)	27.04 (24.90,29.40)	0.957
HR, bpm	70.00 (67.00,78.00)	70.00 (68.00,77.30)	70.00 (66.00,78.00)	70.00 (67.00,78.00)	0.286
SBP, mmHg	130.00 (120.00,140.00)	130.00 (120.00,140.00)	130.00 (121.00,140.80)	130.00 (120.00,140.00)	0.286
DBP, mmHg	78.00 (70.00,85.00)	75.00 (70.00,84.00)	80.00 (72.00,85.00)	79.00 (70.00,85.00)	0.000**
Risk factors					
smoker, n (%)	333 (60.99)	110 (58.82)	112 (62.22)	111 (62.01)	0.552
Drinker, n (%)	165 (30.28)	59 (31.72)	54 (30.00)	52 (29.05)	0.853
AF, n (%)	14 (2.56)	6 (3.21)	5 (2.78)	3 (1.68)	0.635
Diabetes mellitus, n (%)	139 (25.46)	22 (11.76)	43 (23.89)	74 (41.34)	0.000**
CKD, n (%)	40 (7.33)	10 (5.35)	15 (8.33)	15 (8.38)	0.440
previous stroke, n (%)	35 (6.41)	17 (9.09)	13 (7.22)	5 (2.79)	0.042*
previous MI, n (%)	73 (13.37)	27 (14.44)	23 (12.78)	23 (12.85)	0.869
Family history of CVD, n (%)	33 (6.04)	10 (5.35)	12 (6.67)	11 (6.15)	0.867
LVEF, (%)	62.00 (56.00,65.00)	62.00 (56.00,65.00)	61.00 (55.00,65.00)	63.00 (57.00,66.00)	0.088
Laboratory tests					
RBC,10^{12}/L	4.54 ± 0.54	4.44 ± 0.52	4.56 ± 0.57	4.62 ± 0.51	0.005**
WBC,10^9/L	7.15 (6.20,8.50)	6.77 (5.80,7.90)	7.33 (6.30,8.70)	7.47 (6.30,8.80)	0.000**
PLT,10^9/L	227.56 ± 59.05	222.74 ± 59.63	227.56 ± 57.67	232.48 ± 59.73	0.320
NE, %	67.28 ± 8.45	67.21 ± 8.74	67.75 ± 8.80	66.88 ± 7.81	0.642
BNP, pg/ml	63.00 (26.00,161.50)	65.00 (33.50,184.50)	81.50 (26.80,239.80)	49.50 (21.00,125.80)	0.040*
Glu, mmol/L	5.67 (5.00,7.10)	5.12 (4.60,5.70)	5.72 (5.10,6.60)	7.16 (5.60,9.10)	0.000**
HbA1C%, %	6.00 (5.60,6.90)	5.70 (5.40,6.10)	6.00 (5.60,6.70)	6.70 (6.00,7.80)	0.000**
hs-CRP, mg/L	1.63 (0.70,4.00)	1.28 (0.60,3.90)	1.58 (0.80,4.80)	1.81 (0.90,3.80)	0.085
Hb, g/L	143.00 (131.00,153.00)	141.00 (127.80,149.30)	144.00 (131.00,156.00)	144.00 (132.00,153.00)	0.031*
Alb, g/L	43.21 ± 3.92	42.50 ± 3.70	43.01 ± 4.13	44.15 ± 3.77	0.000**
eGFR, mL/min/1.73 m^2	86.210 (67.6,97.6)	89.280 (69.0,96.6)	84.060 (68.7,97.0)	83.840 (65.3,99.5)	0.642
Urea, mmol/L	6.05 (4.80,7.60)	5.66 (4.50,7.00)	6.07 (4.70,7.90)	6.40 (5.40,8.00)	0.000**
UA, μmol/L	378.05 ± 103.38	364.04 ± 100.03	369.00 ± 100.18	401.78 ± 106.37	0.001**
Cr, μmol/L	83.20 (72.30,101.10)	81.60 (70.20,96.30)	83.75 (73.30,100.90)	86.70 (73.70,103.30)	0.081
TG, mmol/L	1.53 (1.20,2.10)	1.02 (0.80,1.20)	1.59 (1.40,1.80)	2.50 (2.00,3.10)	0.000**
TC, mmol/L	3.91 (3.40,4.60)	3.64 (3.20,4.20)	3.88 (3.40,4.40)	4.31 (3.70,5.10)	0.000**
HDL-C, mmol/L	0.98 (0.80,1.20)	1.04 (0.90,1.20)	0.96 (0.80,1.10)	0.92 (0.80,1.10)	0.000**
LDL-C, mmol/L	2.22 (1.70,2.80)	2.12 (1.60,2.50)	2.21 (1.80,2.80)	2.40 (1.80,3.20)	0.003**
Hcy, μmol/l	23.20 (19.50,32.10)	23.60 (19.20,34.30)	24.10 (19.70,33.80)	22.20 (19.50,30.00)	0.208
ALT, U/L	20.00 (14.00,31.00)	18.00 (13.00,28.00)	20.00 (14.00,31.00)	22.00 (15.00,34.30)	0.012*
AST, U/L	20.00 (16.00,28.00)	20.00 (16.00,27.00)	20.00 (16.00,29.00)	20.00 (16.00,29.00)	0.850
Discharge prescription, n (%)					
DAPT	476 (87.18)	165 (88.24)	154 (85.56)	157 (87.71)	0.720
ACEI/ARBs	191 (34.98)	62 (33.16)	57 (31.67)	72 (40.22)	0.191
Beta-blocker	305 (55.86)	100 (53.48)	102 (56.67)	103 (57.54)	0.710
CCB	130 (23.81)	48 (25.67)	37 (20.56)	45 (25.14)	0.454
Diuretics	54 (9.89)	12 (6.42)	19 (10.56)	23 (12.85)	0.112
Statins	462 (84.62)	161 (86.10)	150 (83.33)	151 (84.36)	0.759
Nitrate	278 (50.92)	104 (55.61)	82 (45.56)	92 (51.40)	0.154
Oral hypoglycemic agents	55 (10.07)	4 (2.14)	16 (8.89)	35 (19.55)	0.000**
Insulin	7 (1.28)	2 (1.07)	1 (0.56)	4 (2.23)	0.350
Number of diseased vessels, n (%)					0.109
1-vessel disease	157 (28.75)	61 (32.62)	47 (26.11)	49 (27.37)	
2-vessel disease	200 (36.63)	59 (31.55)	65 (36.11)	76 (42.46)	

Table 1 (continued)

Viable	Total	TyG index level			P value
		T1 (≤ 7.06)	T2 (> 7.06, ≤ 7.54)	T3 (> 7.54)	
3-vessel disease	189 (34.62)	67 (35.83)	68 (37.78)	54 (30.17)	
Location of target lesions, n (%)					
LM	34 (6.23)	12 (6.42)	12 (6.67)	10 (5.59)	0.906
LAD	428 (78.39)	150 (80.21)	138 (76.67)	140 (78.21)	0.710
LCX	224 (41.18)	79 (42.25)	74 (41.11)	71 (40.11)	0.918
RCA	364 (66.67)	120 (64.17)	131 (72.78)	113 (63.13)	0.102
Number of stents, n (%)					0.530
0	117 (21.47)	41 (22.04)	42 (23.33)	34 (18.99)	
1	235 (43.12)	81 (43.55)	77 (42.78)	77 (43.02)	
2	134 (24.59)	47 (25.27)	43 (23.89)	44 (24.58)	
≥ 3	59 (10.81)	17 (9.09)	18 (10.00)	24 (13.41)	
MACE, n (%)	73 (13.37)	17 (9.09)	23 (12.78)	33 (18.44)	0.031*

The data are expressed as the means ± SDs, medians with interquartile ranges or percentages. *P < 0.05, **P < 0.01

TyG index triglyceride–glucose; BMI, body mass index; HR heart rate; SBP, systolic blood pressure; DBP diastolic blood pressure; AF atrial fibrillation; MI, myocardial infarction; DM diabetes mellitus; CKD, chronic kidney disease; MI, myocardial infarction; CVD cardiovascular disease; UA, unstable angina; LVEF, left ventricular ejection fraction; RBC, red blood cell; WBC, white blood cell; PLT, platelet; NE, neutrophil; BNP Brain Natriuretic Peptide; FPG, fasting blood glucose; HbA1c% hemoglobin A1c%; hs-CRP, high sensitivity C-reactive protein; Hb hemoglobin; Albumin; eGFR, estimated glomerular filtration rate; UA uric acid; Cr: creatinine; TG receptor blocker; TC total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT Alanine transaminase; AST Aspartate Aminotransferase; Hcy Homocysteine; DAPT dual antiplatelet therapy; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; CCB calcium channel blocker; PCI, percutaneous coronary intervention; LM: left main artery; LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary artery; MACEs major adverse cardiovascular events

Subgroup analyses

As shown in Fig. 5, subgroup analysis was used to further confirm the risk stratification value of the TyG index for MACEs. The subgroup analyses were generally consistent with the main findings of this study. There was no significant difference in the ability of the TyG index to predict the incidence of MACEs according to sex (female or male), BMI (< 28 or ≥ 28 kg/m2), type 2 diabetes (with or without), CVD (with or without), or the HbA1c level (> 7 or ≤ 7) (all P values for interaction > 0.05). Notably, there was an interaction effect between age、type 2 diabetes and LDL-C level on the Tyg index and MACEs (P for interaction = 0.048、P for interaction = 0.047 and P for interaction = 0.035).

Discussion

In this study, we found a relationship between the TyG index and cardiovascular risk in patients with H-type hypertension combined with CHD for the first time. The results of this study are summarized as follows: (1) the incidence of adverse cardiovascular events was significantly greater in patients with a higher TyG index than in those with a lower TyG index during the established observation period; (2) The TyG index was significantly associated with MACEs after adjusting for confounders, regardless of whether it was analyzed as a continuous or categorical variable; (3) the patient's TyG index showed a nonlinear relationship with the probability of risk of MACEs and was associated with a variety of cardiovascular risk factors; and (4) the inclusion of the TyG index in the baseline model significantly enhanced the predictive efficacy of the model for Mace risk. This study demonstrated that the TyG index, as a simple and easy-to-measure method for estimating IR, can improve the accuracy and pertinence of the risk stratification of patients with H-type hypertension undergoing PCI, thereby identifying high-risk patients among them. Our findings expand the understanding of the TyG index as a tool for risk stratification and identifying individuals at higher risk of cardiovascular disease, especially in patients with dual cardiovascular disease. Most previous studies have focused on single cardiovascular diseases. Patients with multiple comorbid cardiovascular diseases, though less common, are at particularly high risk and face complex clinical challenges. Therefore, improving diagnosis and risk stratification for these patients is especially important.

Hypertension and diabetes mellitus are important and common risk factors for cardiovascular disease and can accelerate the onset and progression of coronary heart disease [25, 26]. H-type hypertension is a special type of hypertension that substantially increases the risk of coronary artery disease compared with essential hypertension and is a new risk factor for CVD [21]. In addition, a cross-sectional study revealed that H-type hypertension was significantly associated with metabolic syndrome, including IR, but failed to explain the causal relationship [19]. It has been reported that hypertension and hyperhomocysteinemia have a synergistic detrimental effect on the prognosis of patients with coronary artery disease and that the underlying mechanisms are intricate and cover multiple levels of effects, including Hcy, including

Table 2 Baseline clinical characteristics of patients stratified by MACEs

Indicators	Overall	Maces	Non-Maces	P value
N (%)	546	73	473	
Age, y	62.00 (53.00,68.00)	58.50 (49.00,67.00)	62.00 (53.00,68.00)	0.206
Male	469 (85.90)	45 (83.33)	424 (86.18)	0.568
BMI, kg/m^2	26.22 (24.20,28.30)	26.01 (23.90,27.80)	26.23 (24.20,28.40)	0.358
Heart rate, bpm	70.00 (67.00,78.00)	72.00 (67.00,80.00)	70.00 (67.00,78.00)	0.429
SBP, mmHg	130.00 (120.00,140.00)	130.00 (120.00,143.80)	130.00 (120.00,140.00)	0.820
DBP, mmHg	78.00 (70.00,85.00)	77.50 (70.00,85.30)	78.50 (70.00,85.00)	0.880
Risk factors				
Smoker, n (%)	333 (61.10)	31 (57.41)	305 (61.99)	0.145
Drinker, n (%)	165 (30.28)	15 (27.78)	150 (30.55)	0.677
AF, n (%)	14 (2.56)	1 (1.85)	13 (2.64)	0.727
Diabetes mellitus, n (%)	139 (25.46)	15 (27.78)	124 (25.20)	0.680
CKD, n (%)	40 (7.33)	5 (9.26)	35 (7.11)	0.566
Previous stroke, n (%)	35 (6.41)	2 (3.70)	33 (6.71)	0.392
Previous MI, n (%)	73 (13.37)	9 (16.67)	64 (13.01)	0.453
Family history of CVD, n (%)	33 (6.04)	2 (3.70)	31 (6.30)	0.447
LVEF, (%)	62.00 (56.00,65.00)	62.00 (55.00,68.00)	62.00 (56.00,65.00)	0.430
Laboratory tests				
RBC,10^{12}/L	4.59 (4.20,4.90)	4.47 (4.00,4.70)	4.61 (4.30,4.90)	0.033*
WBC,10^9/L	7.15 (6.20,8.50)	7.74 (6.60,9.30)	7.11 (6.10,8.50)	0.112
PLT,10^9/L	220.00 (186.00,264.00)	222.00 (198.80,252.80)	220.00 (186.00,266.00)	0.979
NE, %	67.28 ± 8.45	68.69 ± 7.30	67.14 ± 8.55	0.235
BNP, pg/ml	63.00(26.00,161.50)	88.00 (29.80,260.80)	62.00 (26.00,155.50)	0.209
Glu, mmol/L	5.67(5.00,7.10)	5.89 (5.10,7.70)	5.63 (5.00,6.90)	0.180
HbA1C%, %	6.00(5.60,6.90)	6.30 (5.80,7.70)	6.00 (5.60,6.80)	0.030*
hs-CRP, mg/L	1.63(0.70,4.00)	2.48 (0.80,5.90)	1.50 (0.70,3.80)	0.068
Hb, g/L	143.00(131.00,153.00)	142.00 (122.50,148.30)	143.00 (131.00,153.00)	0.035*
Alb, g/L	43.40(40.80,45.80)	42.25 (39.50,44.90)	43.50 (40.90,45.90)	0.046*
eGFR, mL/min/1.73 m^2	86.21(67.60,97.60)	86.81 (55.00,100.10)	86.16 (68.80,97.30)	0.501
Urea, mmol/L	6.05(4.80,7.60)	6.17 (4.60,9.40)	6.02 (4.80,7.50)	0.467
UA, µmol/L	366.50(311.30,441.70)	389.60 (313.70,438.40)	363.75 (310.50,442.00)	0.286
Cr, µmol/L	83.20(72.30,101.10)	89.60 (71.40,124.10)	83.05 (72.30,100.40)	0.208
Glu, mmol/L	5.67(5.00,7.10)	5.89 (5.10,7.70)	5.63 (5.00,6.90)	0.180
TG, mmol/L	1.53(1.20,2.10)	1.53 (1.20,2.00)	1.53 (1.10,2.10)	0.600
TC, mmol/L	3.91(3.40,4.60)	3.93 (3.50,4.50)	3.90 (3.40,4.60)	0.558
HDL-C, mmol/L	0.98(0.80,1.20)	1.00 (0.80,1.20)	0.98 (0.80,1.10)	0.903
LDL-C, mmol/L	2.22(1.70,2.80)	2.31 (1.90,2.70)	2.20 (1.70,2.80)	0.562
Hcy, µmol/L	23.20(19.50,32.10)	23.00 (19.50,33.00)	23.25 (19.50,32.10)	0.715
ALT, U/L	20.00(14.00,31.00)	17.00 (13.50,30.00)	20.00 (14.00,31.80)	0.186
AST, U/L	20.00(16.00,28.00)	19.00 (14.80,22.30)	20.00 (16.00,28.00)	0.168
Discharge prescription, n (%)				
DAPT	476 (87.18)	46 (85.19)	430 (87.40)	0.644
ACEI/ARBs	191 (34.98)	17 (31.48)	174 (35.37)	0.570
Beta blocker	305 (55.86)	31 (57.41)	274 (55.69)	0.809
CCB	130 (23.81)	13 (24.07)	117 (23.78)	0.962
Diuretics	54 (9.89)	4 (7.41)	50 (10.16)	0.520
Statins	462 (84.62)	47 (87.04)	415 (84.35)	0.603
Nitrate	278 (50.92)	31 (57.41)	247 (50.20)	0.315
Oral hypoglycemic agents	55 (10.07)	4 (7.41)	51 (10.37)	0.493
Insulin	7 (1.28)	1 (1.85)	6 (1.22)	0.695
Number of diseased vessels, n (%)				0.291
1-vessel disease	157 (28.75)	11 (20.37)	146 (29.67)	
2-vessel disease	200 (36.63)	24 (44.44)	176 (35.77)	

Table 2 (continued)

Indicators	Overall	Maces	Non-Maces	P value
3-vessel disease	189 (34.62)	19 (35.19)	170 (34.55)	
Location of target lesions, n (%)				
LM	34 (6.23)	3 (5.56)	31 (6.30)	0.830
LAD	428 (78.39)	40 (74.07)	388 (78.86)	0.417
LCX	320 (58.82)	37 (68.52)	283 (57.76)	0.127
RCA	364 (66.67)	39 (72.22)	325 (66.06)	0.362
Number of stents, n (%)				0.350
0	117 (21.47)	13 (24.07)	104 (21.18)	
1	235 (43.12)	21 (38.89)	214 (43.58)	
2	134 (24.59)	13 (24.07)	121 (24.64)	
≥ 3	59 (10.81)	7 (12.96)	52 (10.57)	

The data are expressed as the means ± SDs, medians with interquartile ranges or percentages. *P < 0.05, **P < 0.01

BMI, body mass index; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; AF, atrial fibrillation; MI, myocardial infarction; DM, diabetes mellitus; CKD, chronic kidney disease; MI, myocardial infarction; CVD, cardiovascular disease; UA, unstable angina; LVEF, left ventricular ejection fraction; RBC, red blood cell; WBC, white blood cell; PLT, platelet; NE, neutrophil; BNP, brain natriuretic peptide; FPG, fasting blood glucose; HbA1c%, hemoglobin A1c%; hs-CRP, high-sensitivity C-reactive protein; Hb, hemoglobin; Alb, Albumin; eGFR, estimated glomerular filtration rate; UA, uric acid; Cr, creatinine; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine transaminase; AST, aspartate Aminotransferase; Hcy, homocysteine; DAPT, dual antiplatelet therapy; ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; CCB, calcium channel blocker; PCI, percutaneous coronary intervention; LM: left main artery; LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary artery; MACEs major adverse cardiovascular events

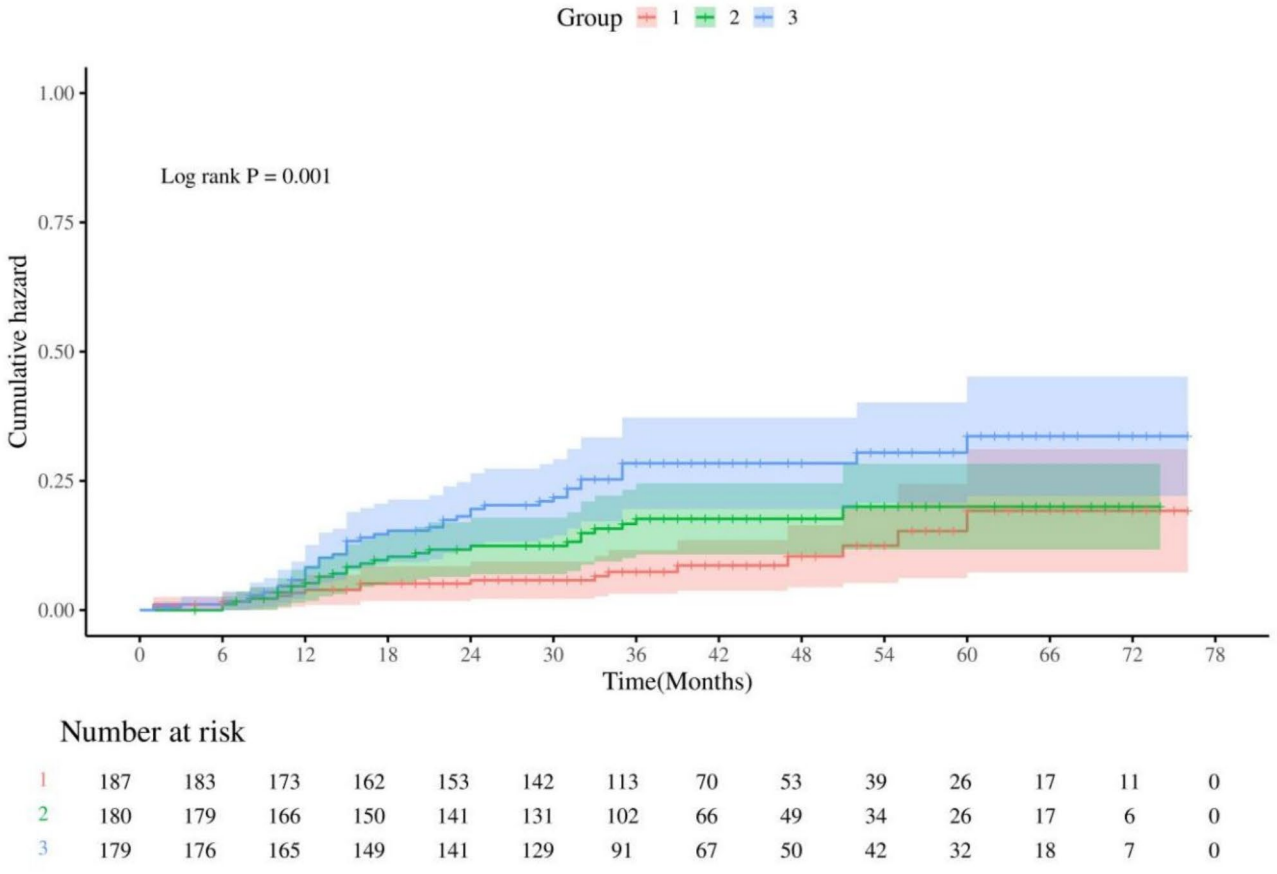


Fig. 2 Kaplan–Meier curve illustrating the cumulative risk of cardiovascular events according to different TyG index levels. TyG index triglyceride–glucose index

Table 3 Cox regression models for the relationship between the triglyceride–glucose index and major adverse cardiovascular events

Variables	Model 1		Model 2		Model 3	
	HR (95%CI)	*P*	HR (95%CI)	*P*	HR (95%CI)	*P*
TYG	1.55 (1.19 ~ 2.04)	0.001	1.53 (1.15 ~ 2.04)	0.003	1.40 (1.01 ~ 2.09)	0.046
Tertile 1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Tertile 2	1.69 (0.92 ~ 3.09)	0.092	1.69 (0.92 ~ 3.10)	0.093	1.64 (0.80 ~ 3.36)	0.175
Tertile 3	2.71 (1.54 ~ 4.74)	<.001	2.69 (1.51 ~ 4.80)	<.001	2.43 (1.19 ~ 4.97)	0.015

Model 1: unadjusted for covariates

Model 2: adjusted for age and sex

Model 3: adjusted for age, sex, RBC count, HbA1c%, Hb, and Alb

HR, hazard ratio; CI, confidence interval

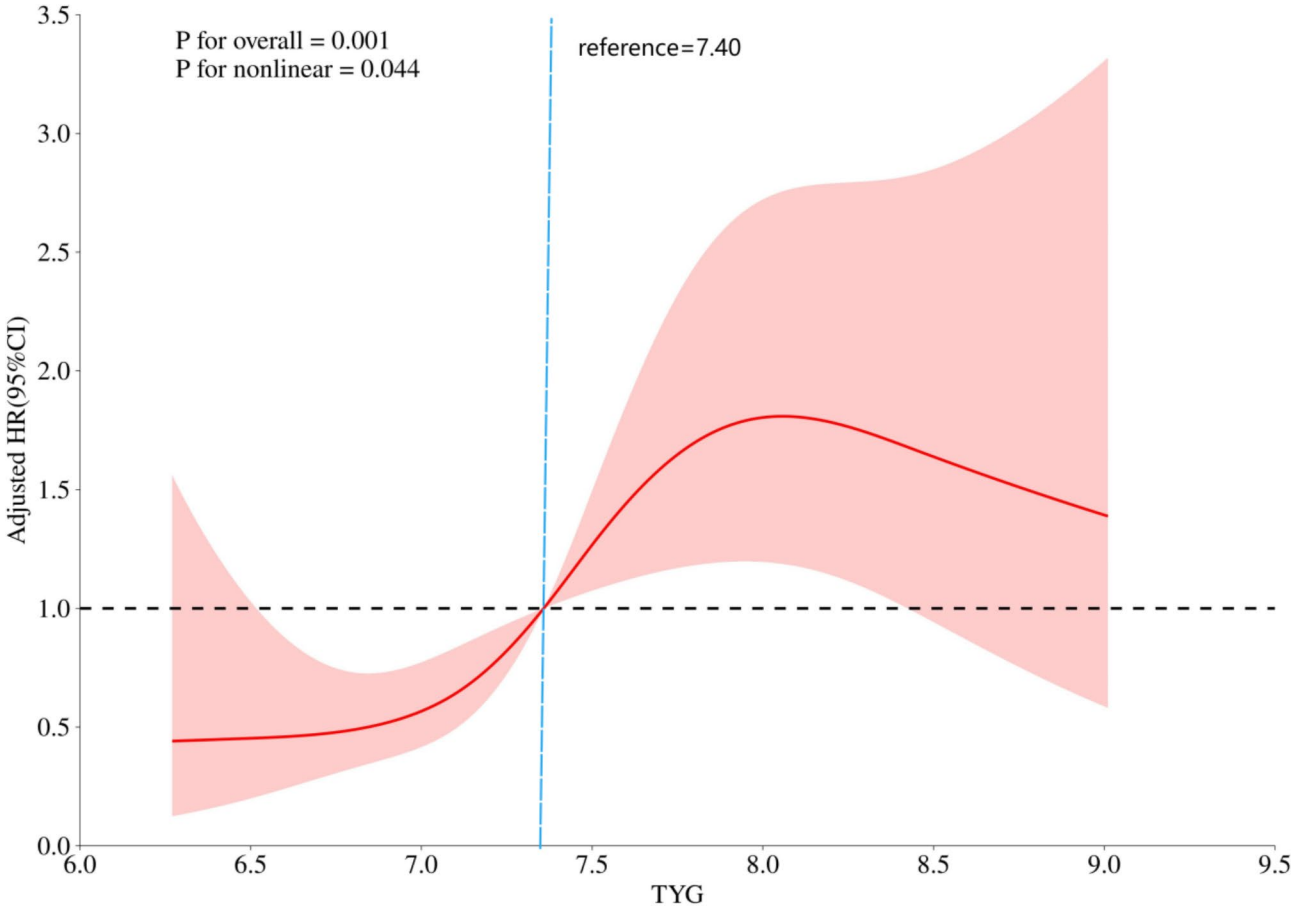


Fig. 3 The restricted cubic spline of MACEs and the TyG index. TyG index, triglyceride–glucose index; HR, hazard ratio; CI, confidence interval; MACEs, major adverse cardiovascular events

participation in and exacerbation of the process of endothelial damage in blood vessels, stimulation of vascular smooth muscle proliferation, and induction of lipid metabolism disorders, as well as contributing to the coagulation system and platelet dysfunction [21]. A study of 5724 patients in the United States reported that the interaction of hypertension and plasma Hcy increased the risk of all-cause mortality and CVD death in middle-aged and older adults. For every 1 μmol/L increase in plasma Hcy in hypertensive patients, the risk of all-cause mortality and CVD mortality increased by 8% and 7%, respectively. The corrected risk ratios for all-cause and CVD deaths were 1.08 (1.06–1.10) and 1.07 (1.04–1.10), respectively [27]. Patients with H-type hypertension combined with CHD tend to have a worse prognosis. Therefore, it is particularly important to identify such patients early and stratify their risk of adverse cardiovascular events. Given prior evidence that the TyG index predicts poor outcomes in several cardiovascular diseases, we evaluated its association with the risk of cardiovascular events in our study population to investigate its predictive role.

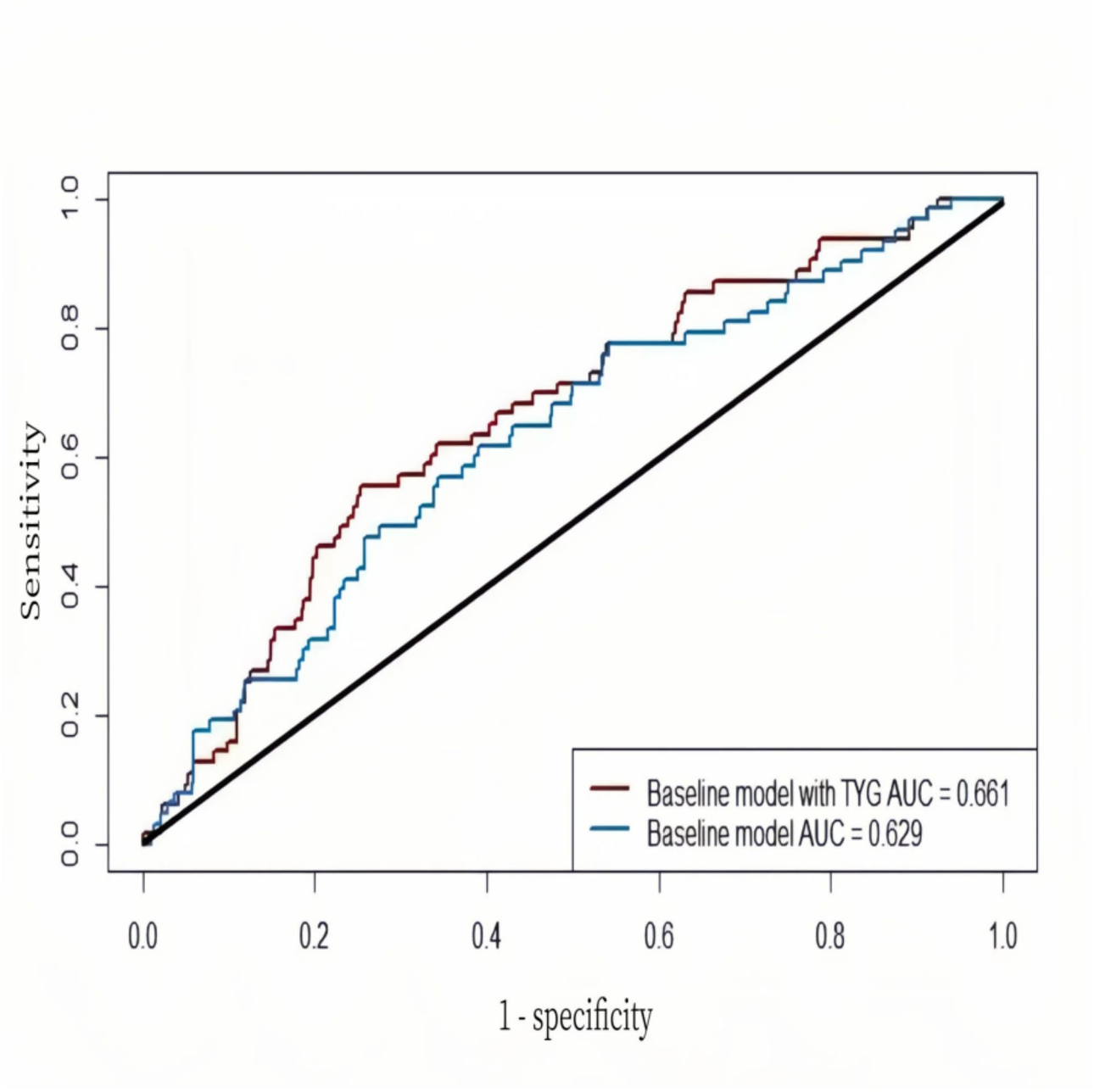


Fig. 4 Time-dependent ROC curves for the prediction of MACEs. ROC curve receiver operating characteristic curve, TyG index triglyceride–glucose index, MACEs major adverse cardiovascular events, AUC area under the receiver operating characteristic curves

Table 4 Evaluation of predictive models of the TyG index for MACEs

	C-statistic (95% CI)	P value	NRI (95% CI)	P value	IDI (95% CI)	P value
Baseline	0.63 (0.59–0.66)	Ref	Ref		Ref	
+ TYG	0.66 (0.62–0.68)	0.031	0.13 (0.00–0.28)	0.038	0.01 (0.00–0.03)	0.047

CI, confidence interval; IDI, integrated discrimination improvement; NRI, net reclassification improvement

IR is the main feature of type 2 diabetes. It is closely related to various pathological processes such as endothelial dysfunction, glucose and lipid metabolism disorder, oxidative stress and systemic inflammation, and has been proven to be a risk factor for a variety of cardiovascular events [28–30]. IR has been shown to promote CVD in both diabetic and nondiabetic patients and is predictive of poor CVD prognosis [31, 32]. Therefore,

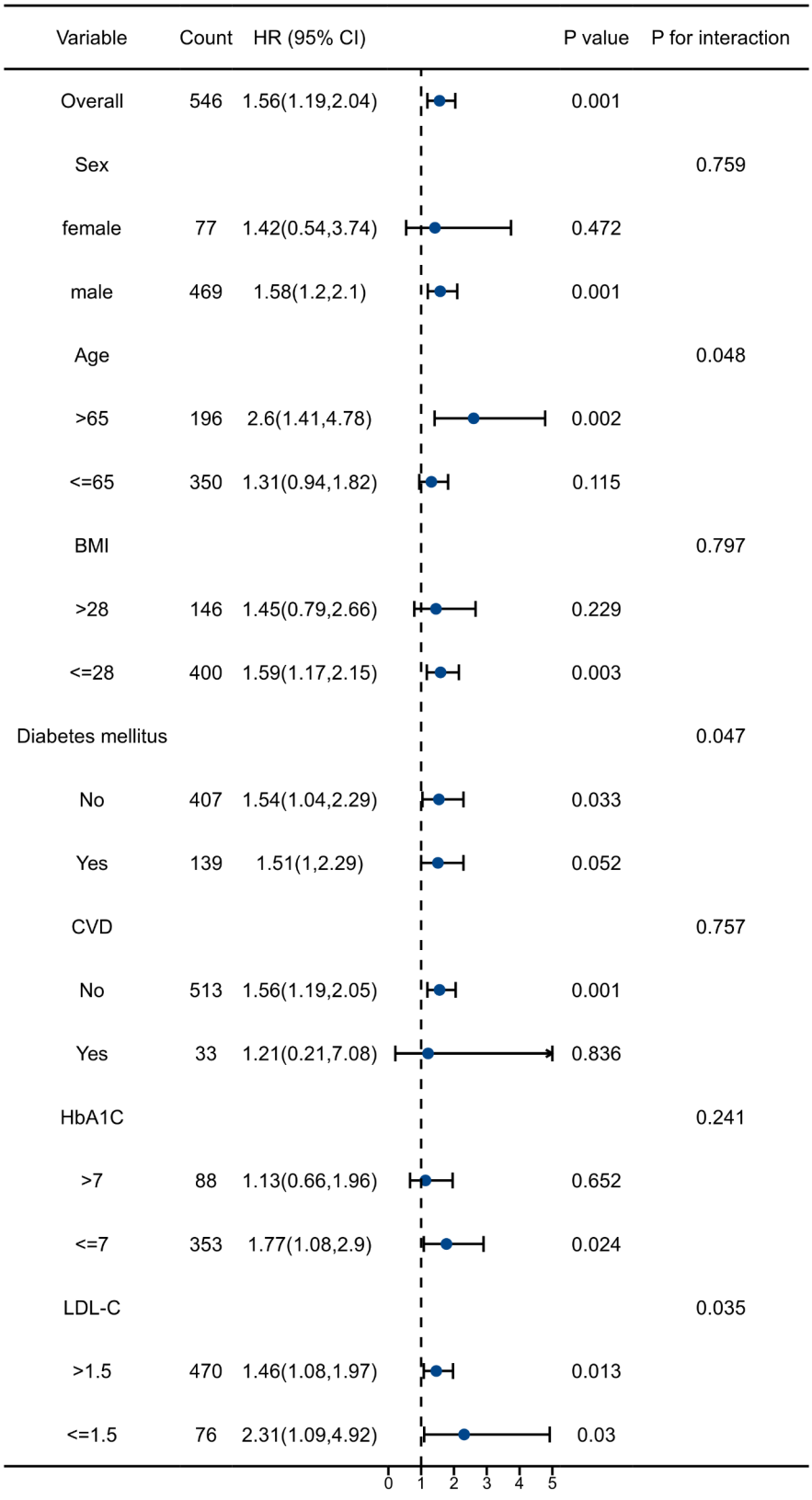


Fig. 5 Subgroup and interaction analysis for the association between the TyG index (per standard deviation) and MACEs

accurate identification of IR is clinically important for optimizing primary and secondary prevention strategies for cardiovascular risk and implementing more precise risk stratification. The glucose-triglyceride (TyG) index was first proposed in 2008 by Simental-Mendia et al. It is used mainly to identify IR in individuals with type 2 diabetes mellitus and is superior to the traditional Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) in terms of accuracy and simplicity [33, 34]. With increasing research in recent years, the TyG index has been closely associated with the prognosis of heart failure, stroke, hypertension, CHD, atrial fibrillation, CKD, metabolic syndrome, atherosclerosis, etc. [35–37]. A large cohort study involving 15,056 patients by Xin et al. revealed that the TyG index and the risk of hypertension were positively correlated [38]. In addition, the TyG index is significantly associated with the incidence of coronary obstructive disease in hypertensive patients, and this association is independent of differences in sex, age, diabetes status, and coronary artery calcium score [39]. Several recent studies have shown that the TyG index predicts a higher risk of MACEs within 1 year in patients with hypertension combined with CHD, and that this trend is particularly pronounced in diabetic patients [17]. In a cohort study of 2531 patients, Wang et al. reported that the TyG index predicted the 3-year incidence of mortality in patients with diabetes mellitus combined with acute coronary syndrome, even after adjusting for potential confounders [40]. In patients with H-type hypertension, the TyG index was significantly correlated with the extent of coronary artery lesions, and after adjusting for the effects of relevant confounders, the TyG index was significantly correlated with the severity of coronary artery lesions [18, 41]. In addition, a registry study revealed that the TyG index can be highly accurate in assessing the risk of arterial stiffness in patients with H-type hypertension and that maintaining a low TyG index may be a preventive measure for arterial stiffness in patients with H-type hypertension [42]. In a cross-sectional study, the TyG index in postmenopausal women was significantly associated with the risk of developing H-type hypertension, with a positive linear correlation [43], which may be due to an increase in IR as well as a decrease in estrogen levels in the body. The above studies show that the TyG index has a strong ability to predict the risk of cardiovascular events, but the effect of the TyG index on the prognosis of patients with H-type hypertension combined with CHD is not clear.

In the present study, our findings revealed significant correlations between the TyG index and traditional cardiovascular risk factors (age, blood pressure, diabetes mellitus, previous history of stroke, LDL-C level, etc.). In the Cox proportional risk regression model, we applied 3 adjusted and unadjusted models for regression analysis, and almost fully adjusted Model 3 represented the most comprehensive risk prediction model and showed the predictive value of the TyG index in relation to the incidence of MACEs in patients. To confirm the reliability and stability of the findings, we performed subgroup analyses. In the subgroup analysis, we observed a significant interaction effect of the TyG index with age and LDL-C. The TyG index can be used as a prognostic indicator in elderly (> 65 years) patients with H-type hypertension combined with CHD. Age was independently associated with H-type hypertension and coronary heart disease, which was consistent with previous studies. On the one hand, with the increase of age, the metabolic function of the human body declines, including folic acid and vitamin B12, resulting in the occurrence of hypercysteinemia; In addition, aging leads to vascular endothelial dysfunction, vascular regulation of blood pressure decline, leading to hypertension, coronary heart disease and its complications [44, 45]. In addition, the predictive value of TyG-represented IR seemed to be more favorable in patients with LDL-C > 1.5 [HR (95% CI) LDL-C > 1.5 mmol/L 1.46 (1.08 to 1.97) vs LDL-C ≤ 1.5 mmol/L 2.31 (1.09 to 4.92), P for interaction = 0.035]. The formation and deposition of LDL is an important factor in coronary artery stenosis and myocardial ischemia. When LDL is subjected to oxidative stress, its structure is changed to form oxidized low-density lipoprotein, which induces the proliferation and migration of vascular smooth muscle cells. In addition, there is an interaction between LDL and Hcy, which together promote the development of atherosclerosis [46, 47]. By adding the TyG index to the established conventional hazard model, we found a significant improvement in risk prediction ability in terms of the C statistic, NRI, and IDI. In this study, in addition to the TyG index, RBC, HbA1C%, Hb, and Alb were also significantly correlated with MACEs. The RBC reflects the oxygen-carrying capacity of blood. When there are too many RBCs, they may increase blood viscosity and promote the formation of blood clots, thereby increasing the risk of cardiovascular disease. Conversely, too few RBCs may lead to hypoxia in the heart muscle, affecting heart function. HbA1C% is primarily used to assess glycemic control in diabetics, but it is susceptible to various factors such as red blood cell lifespan, anemia, and blood transfusions. Hb, an essential component of red blood cells, is responsible for transporting oxygen. Anemia can lead to hypoxia in the heart muscle, affecting heart function and thereby increasing the risk of cardiovascular disease. In addition, anemia may also cause hemodynamic changes, increasing cardiac load and further exacerbating the development of cardiovascular disease. Albumin (Alb) is primarily used for assessing the diagnosis and treatment of liver function, malnutrition, nephrotic syndrome, and other diseases. In contrast, as a new

cardiovascular disease risk assessment indicator, the TyG index has the advantages of comprehensive assessment, high sensitivity, simple calculation, and wide application range. Therefore, we use it as a predictor of adverse outcomes in patients.

Notably, the critical value of the TyG index associated with poor prognosis varied across studies, largely because of the heterogeneity of the study populations. For example, in a registry study of older adults with H-type hypertension, patients with a TyG index greater than 9 were significantly associated with the risk of first stroke. This may be related to the fact that the elderly population itself has a reduced ability to metabolize blood glucose and lipids, and a lack of attention has led to undertreatment [48]. In patients with hypertension combined with coronary artery disease, a TyG index greater than 8.69 was significantly associated with a poor prognosis. This is because IR-associated pathologies are important causes of the exacerbation of hypertension and coronary artery disease [49]. The relatively low critical value of the TyG index in the population included in this study compared with the above studies may be related to the early intensification of lipid and glucose-lowering therapy for such patients in the medical center and the fact that a portion of the patients had better control of lipids and glucose prior to referral to this cardiovascular disease center.

Although the underlying mechanism by which the TyG index is associated with poor prognostic risk in patients with H-type hypertension combined with CHD has not been clarified, we hypothesized that it may be due to IR. First, IR is associated with metabolic abnormalities such as hyperglycemia, dyslipidemia, hypertension, and obesity. IR induces the production of glycosylation products and oxygen free radicals, which further triggers inflammatory responses and oxidative stress, leading to endothelial dysfunction and vascular endothelial damage [50]; IR leads to impaired insulin-related signaling pathways, which leads to hypertension and atherosclerosis; IR affects Hcy-metabolizing enzymes and insulin levels to increase Hcy levels, and increased Hcy levels promote the development of CVD; and IR leads to abnormal platelet function, including the induction of an overactive state and abnormal adhesion, and contributes to increased levels of thromboxane a2-mediated tissue factor expression [30]. These pathological factors strongly increase the likelihood of arterial thrombosis and inflammatory activity. These findings explain why a high TyG index predicts the risk of cardiovascular events in patients with H-type hypertension combined with coronary artery disease.

However, this study has some limitations. Firstly, this is a retrospective cohort study conducted in a single center in China, and the causal relationship cannot be definitively determined. The selection of samples and the collection of data are based on the specific conditions of the center, which may introduce selection bias and geographical limitations. Therefore, the external effects of the findings should be further examined; the TyG index in our study was measured only once at baseline, and we failed to consider the changes in the TyG index during the follow-up period, which may have led to biased analysis; The sample size collected in this study was limited, and further prospective multicenter studies with larger sample sizes and more time points are needed to improve reliability in the future.

Conclusion

In conclusion, our study revealed the predictive value of the TyG index for poor prognosis in patients with H-type hypertension combined CHD, which can help healthcare professionals make more rational clinical decisions. For the treatment strategy for high-risk patients, more emphasis should be placed on the control of cardiovascular risk factors. In addition to appropriate supplementation with folic acid and B vitamins, regular detection and purposeful reduction of TyG index levels are beneficial for reducing the incidence of poor prognosis in patients.

Abbreviations

TyG	Triglyceride-glucose
CHD	Coronary heart disease
MACEs	Major adverse cardiovascular events
CVD	Cardiovascular disease
IR	Insulin resistance
BMI	Body mass index
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
HR	Heart rate
PCI	Percutaneous coronary intervention
CKD	Chronic kidney disease
OMI	Old myocardial infarction
ACEI	Angiotensin converting enzyme inhibitor
ARB	Angiotensin receptor blocker
CCB	Calcium channel blockers
TC	Total cholesterol
TG	Triglyceride
LDL-C	Low-density lipoprotein cholesterol
HDL-C	High-density lipoprotein cholesterol
Hs-CRP	Hypersensitive C-reactive protein
Hcy	Homocysteine
Scr	Serum creatinine
FBG	Fasting blood glucose
HbA1c	Glycosylated hemoglobin
LAD	Left atrial diameter
LVEF	Left ventricular ejection fraction
RCS	Restricted cubic spline
LM	Left main artery
LAD	Left anterior descending artery
LCX	Left circumflex artery
RCA	Right coronary artery
T2DM	Type 2 diabetes mellitus
DM	Diabetes mellitus
CABG	Coronary artery bypass grafting
HRs	Hazard ratios
CI	Confidence interval
FBG	Fasting blood glucose
SD	Standard deviation
NRI	Net reclassification improvement
IDI	Integrated discrimination improvement

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Author Contribution
CL and SZ were involved in the design of the study, the analysis of the data, and the drafting of the paper.LY contributed to interpretation of the results.HP substantively revised the manuscript.All authors collaboratively participated in the data collection and analysis processes.All authors read and approved the final manuscript.

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Availability of data and materials
The data used in this study can be obtained from the first author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was conducted in accordance with the Declaration of Helsinki. Given that this is a retrospective study, ethical approval and consent to participate do not apply to the study.

Consent for publication
All authors have agreed to publish.

Competing interests
The authors declare no competing interests.

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