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Association between triglyceride-glucose index and in-hospital all-cause mortality under different glucose metabolism status among patients with coronary artery disease

Xuyang Meng[1,2†], Xiang Wang[1†], Baoyu Feng[3], Zinan Zhao[4], Qirui Song[5], Wei Xu[6], Chenxi Xia[1,7], Yi Li[1,2], Fang Wang[1,2*] and Chenguang Yang[1*]

Abstract

Background This current study aimed to investigate the relationship between the triglyceride-glucose (TyG) index and in-hospital all-cause mortality of coronary artery disease (CAD) in patients with different glucose metabolic statuses.

Methods Participants were divided into three groups according to tertiles of the TyG index. Glucose metabolic status was classified as normal glucose regulation, pre-diabetes mellitus, and diabetes mellitus (DM). The primary outcome was in hospital all-cause mortality.

Results We observed a significant relationship between the TyG index and in-hospital deaths of patients with CAD in this study. After adjusting for multiple factors in the logistic regression model, the TyG index was still an independent risk factor, and the T3 group (OR, 2.311; 95% CI = 1.237–4.317; $P = 0.009$) was correlated with a 2.311-fold risk compared with the T1 group. In the subgroup analysis of different glucose metabolic status, the T3 group (OR, 1.541; 95% CI: 1.013–2.344; $P = 0.043$) were associated with a significantly higher risk of in-hospital deaths in CAD patients with DM.

Conclusions An increased TyG index was correlated with a higher risk of in-hospital all-cause mortality. Our study indicated that TyG index could be a valuable predictor of in-hospital death of CAD patients, especially for individuals with DM.

Keywords Triglyceride-glucose index, Diabetes mellitus, Coronary artery disease, In-hospital all-cause mortality

†Xuyang Meng and Xiang Wang contributed equally to this work.

*Correspondence:
Fang Wang
bjh_wangfang@163.com
Chenguang Yang
yangchenguang87@126.com

Full list of author information is available at the end of the article

Background

Coronary artery disease (CAD) is the main cause of global mortality, which making it a major threat to public health [1]. Type 2 diabetes mellitus (T2DM), defined as hyperglycaemia resulting from tissue insulin resistance and relative insulin deficiency, can exacerbate the progression of atherosclerosis [2, 3]. Therefore, CAD has become one of the most common complications in patients with T2DM [4], occurring at least twice as often as in those without T2DM [5]. Meanwhile, coronary arteries in diabetic patients present extensive and diffuse atherosclerosis, longer and more complex lesions than in non-diabetic patients [6].

Insulin resistance is a critical mechanism in developing T2DM and significantly correlated with adverse cardiovascular outcomes [7, 8]. Hyperinsulinemic-euglycemic clamp test is recognized as the gold standard for measuring insulin resistance in the body, but is rarely applied in clinical settings due to its invasive and cumbersome procedure [9]. After that, the Homeostatic Model Assessment of insulin resistance (HOMA-IR) was proposed as an alternative, which was calculated by fasting insulin and glucose. However, HOMA-IR reflects only the ability of basal insulin to suppress hepatic glucose production, the concept of insulin resistance also includes impairment in the oxidation and utilization of fatty acids [10]. Therefore, Simental-Mendía et al. [11] first suggested that the triglyceride-glucose (TyG) index could be a surrogate for the HOMA-IR.

The TyG index, which has been confirmed as surrogate markers of insulin resistance [12], is related to a higher incidence of CAD [13, 14]. It is composed of fasting triglyceride (TG) and fasting blood glucose (FBG), and calculated using the formula: TyG=Ln[fasting TG (mg/dl) × FBG (mg/dl)/2] [11]. Several recent retrospective studies in China have reported that high TyG index was significantly associated with adverse clinical outcomes in patients with CAD, including all-cause death, non-fatal myocardial infarction (MI), unplanned repeat revascularization and non-fatal stroke [8, 15]. However, this compound outcome may only reflect a comprehensive effect, so it is necessary to further evaluate the relationship between the TyG index and death risk under specific conditions. The aim of the present study was to clarify the association between the TyG index and in-hospital mortality in patients with CAD, in order to provide new insights into the role of the TyG index in predicting outcomes in CAD patients.

Methods

Study design and population

This retrospective observational cohort study was performed in Beijing hospital in China between January 1, 2016 and December 30, 2021. A total of 19,929 participants who were diagnosed with CAD during hospitalization were enrolled. The flowchart of this study is shown in Fig. 1. After excluding patients with an estimated glomerular filtration rate (eGFR)<30 ml/min, cancer and missing FBG or TG data, we included 10 964 patients in this current analysis eventually. In order to investigate the relationship between the TyG index and in-hospital death among participants with CAD, the patients were divided into three groups according to the tertiles of the TyG index: T1 group (TyG index<6.84, $n=3656$), T2 group (6.84≤TyG index<7.38, $n=3656$), and T3 group (TyG index≥7.38, $n=3652$).

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Beijing Hospital. Informed consent was obtained from all study participants.

Measurements and definitions

Patient sociodemographic characteristics (age, sex, height, weight, smoking status, and drinking status), medical history (diabetes mellitus, hypertension, chronic kidney disease, and cancer), and laboratory test results were collected from medical records of Beijing Hospital. We also recorded the patient's systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) on hospital admission, as well as the use of antiplatelet, antihypertensive, or antilipidemic medications. Blood samples for the measurement were obtained from all the participants after at least 8 h of fasting. FBG, creatinine, total cholesterol (TC), TG, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were measured using the LABOSPECT 008 system (Hitachi, Tokyo, Japan), and the glycated hemoglobin A1c (HbA1c) level was measured by high-performance liquid chromatography (G8, TOSOH, Tokyo, Japan) in the laboratory of Beijing Hospital.

The TyG index was calculated as follows: Ln (fasting TG [mg/dL] × FBG [mg/dL]/2) [11]. Body mass index (BMI) was calculated as weight (kg) divided by the squared height (m), and eGFR was calculated according to the Chronic Kidney Disease Epidemiology Collaboration creatinine equation [16].

CAD was defined as ≥50% lumen stenosis in at least one major coronary artery (left anterior descending, left circumflex, or right coronary arteries).

The Glucose metabolism status was defined according to the American Diabetes Association (ADA) criteria [17]. Diabetes mellitus (DM) was diagnosed when patients had the following: an FBG level≥7.0 mmol/L, 2-hour plasma glucose level≥11.1 mmol/L according to the oral glucose tolerance test, HbA1c≥6.5%, or diabetes history. Pre-diabetes mellitus (pre-DM) was defined as patients without self-reported DM but with an FBG level ranging from 5.6 to 6.9 mmol/L, 2-hour plasma glucose

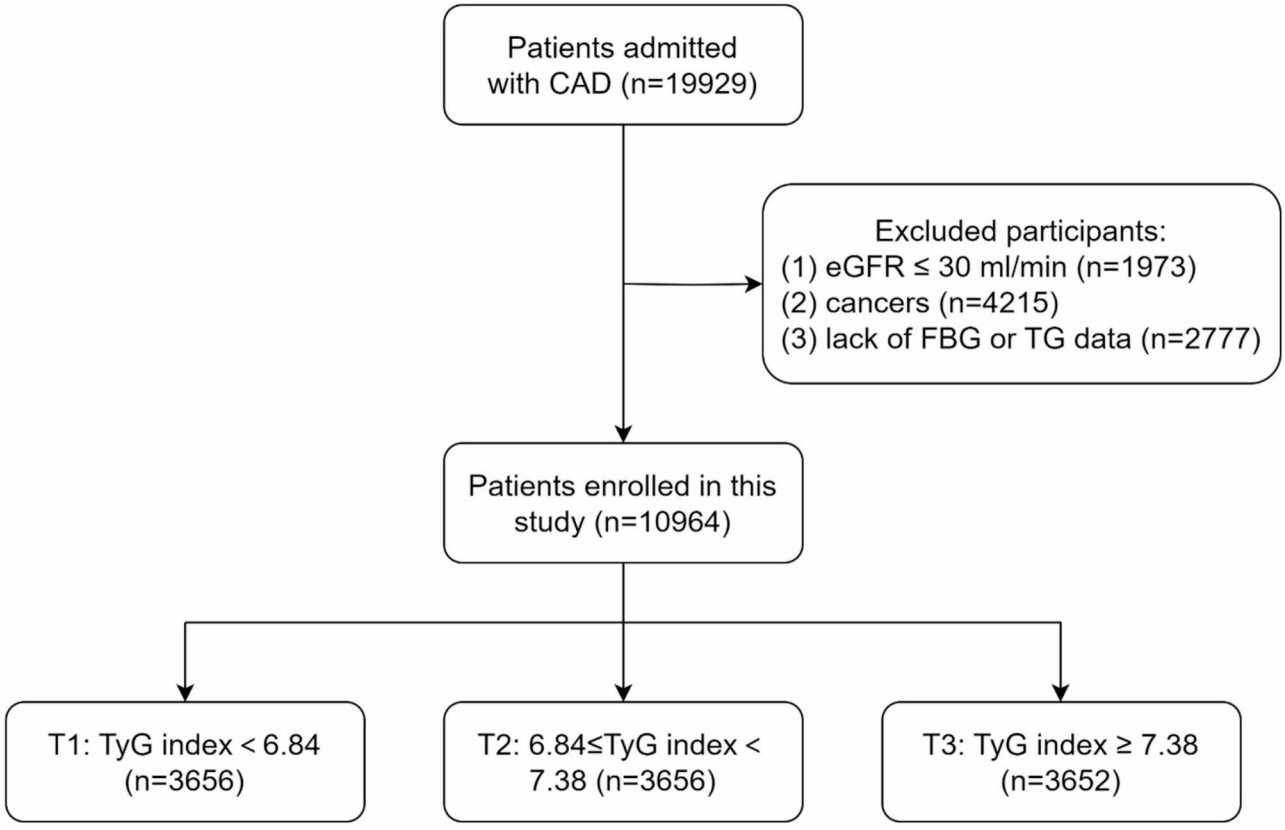

Fig. 1 Flowchart of study patients
CAD, coronary artery disease; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; TG, triglyceride; TyG, triglyceride–glucose

level ranging from 7.8 to 11.0 mmol/L, or HbA1c level ranging from 5.7 to 6.4%. Patients with no history of diabetes or an HbA1c≤5.7% were regarded as normoglycemia (NGR).

The primary outcome of this study was in-hospital mortality, defined as all-cause death occurring within 30 days of admission, as recorded in medical records.

Statistical analysis

Continuous variables are shown as mean±standard deviation or median with interquartile range (25–75%). Categorical variables are summarized as number (*n*) or percentage (%). The Kruskal–Wallis test was used to compare the baseline variables of the TyG index tertiles when appropriate, and the chi-square test was performed to compare the categorical variables among groups.

To clarify the association between the TyG index and in-hospital death, we calculated Odds ratios (ORs) and 95% confidence intervals (CIs) using a logistic regression analysis. In this study, Model 1 was unadjusted. Model 2 was adjusted for age and sex. Model 3 was further adjusted for BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antilipidemic drug use, and antihypertensive drug use based on model 2. Restricted cubic splines were used to examine the shape of the associations between the baseline TyG index and in-hospital death.

All statistical analyses were performed using SAS, version 9.4 (SAS Institute, Inc., Cary, NC), R, version 4.0.3 (R Foundation for Statistical Computing) and SPSS Statistics, version 26.0 (IBM Corporation). All *P*values were 2-sided, and statistical significance was defined as $P<0.05$.

Results

Baseline characteristics

The mean age of the 10,964 patients with CAD was 68±11 years and a total of 4,985 (45.5%) patients were diagnosed with DM. Table 1 shows the baseline characteristics based on tertiles of the TyG index. The age, sex, BMI, SBP, DBP, HR, FBG, HbA1c, HDL-C, LDL-C, TC, TG, eGFR, smoking status, drinking status, hypertension, acute coronary syndrome (ACS), NGR, pre-DM, DM, use of antiplatelets, use of antihypertensive drugs, use of antilipidemic drugs and use of angiotensin converting enzyme inhibitor (ACEI)/angiotensin receptor blocker (ARB) drugs were markedly different among the three groups (all, $P<0.05$).

Table 1 Baseline characteristics comparison of the TyG index among the three groups

	Total ($n=10964$)	T1 ($n=3656$)	T2 ($n=3656$)	T3 ($n=3652$)	*P*-value
Age (years)	68 ± 11	70 ± 11	68 ± 11	65 ± 11	<0.001
Male (*n*, %)	6945 (63.3%)	2367 (64.7%)	2243 (61.4%)	2335 (63.9%)	0.007
BMI (kg/m^2)	25.51 ± 3.44	24.53 ± 3.38	25.68 ± 3.28	26.31 ± 3.44	<0.001
SBP (mmHg)	136 ± 19	135 ± 19	136 ± 19	137 ± 19	<0.001
DBP (mmHg)	77 ± 12	76 ± 12	77 ± 12	78 ± 12	<0.001
HR (beats/min)	77 ± 13	76 ± 13	76 ± 13	78 ± 13	<0.001
HDL-C (mg/dL)	1.07 ± 0.27	1.17 ± 0.29	1.06 ± 0.25	0.96 ± 0.23	<0.001
LDL-C (mg/dL)	2.19 ± 0.83	1.96 ± 0.72	2.19 ± 0.81	2.41 ± 0.90	<0.001
TC (mg/dL)	3.81 ± 0.97	3.54 ± 0.83	3.76 ± 0.92	4.14 ± 1.05	<0.001
TG (mg/dL)	1.42 ± 0.91	0.77 ± 0.19	1.24 ± 0.28	2.24 ± 1.11	<0.001
FBG (mmol/l)	6.38 ± 2.28	5.25 ± 0.88	6.02 ± 1.44	7.85 ± 3.03	<0.001
HbA1c (%)	6.68 ± 1.34	6.20 ± 0.96	6.55 ± 1.66	7.29 ± 1.59	<0.001
eGFR (ml/min)	84.46 ± 17.31	83.67 ± 16.40	84.36 ± 16.71	85.34 ± 18.68	<0.001
Smoking (*n*, %)	4589 (41.9%)	1448 (39.6%)	1487 (40.7%)	1654 (45.3%)	<0.001
Drinking (*n*, %)	6048 (55.2%)	1973 (54.0%)	1987 (54.3%)	2088 (57.2%)	0.011
Hypertension (*n*, %)	7745 (70.6%)	2408 (90.7%)	2645 (72.3%)	2692 (73.7%)	<0.001
ACS history (*n*, %)	544 (5.0%)	147 (4.0%)	168 (4.6%)	229 (6.3%)	<0.001
Stroke history (*n*, %)	1089 (9.9%)	381 (10.4%)	363 (9.9%)	345 (9.5%)	0.390
Glucose metabolism state					<0.001
NGR (*n*, %)	2401 (21.9%)	1238 (33.9%)	799 (21.9%)	364 (10.0%)	
Pre-DM (*n*, %)	3578 (32.6%)	1387 (37.9%)	1324 (36.2%)	867 (23.7%)	
DM (*n*, %)	4985 (45.5%)	1031 (28.2%)	1533 (41.9%)	2421 (66.3%)	
Medications					
Antiplatelets (*n*, %)	7830 (71.4%)	2531 (69.2%)	2609 (71.4%)	2690 (73.7%)	<0.001
Antihypertensive drugs (*n*, %)	7827 (71.4%)	2481 (67.9%)	2641 (72.2%)	2705 (74.1%)	<0.001
Antilipidemic drugs (*n*, %)	8779 (80.1%)	2903 (79.4%)	2909 (79.6%)	2967 (81.2%)	<0.001
ACEI/ARB (*n*, %)	4598 (41.9%)	1354 (37.0%)	1516 (41.5%)	1728 (47.3%)	<0.001
Statins (*n*, %)	8668 (79.1%)	2871 (78.5%)	2882 (78.8%)	2915 (79.8%)	0.365

TyG, triglyceride–glucose; FBG, fasting blood glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglyceride; HR, heart rate; HbA1c, glycated hemoglobin A1c; eGFR, estimated glomerular filtration rate; ACS, acute coronary syndrome; NGR, normoglycemia; Pre-DM, Pre-diabetes mellitus; DM, diabetes mellitus; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker

Table 2 Associations between the TyG index and in-hospital death

	Model 1			Model 2			Model 3		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value	OR	95% CI	*P*-value
TyG index	1.217	0.906–1.634	0.193	1.749	1.297–2.360	<0.001	1.946	1.308–2.895	0.001
T1	Reference			Reference			Reference		
T2	1.000	0.611–1.636	1.000	1.283	0.779–2.113	0.328	1.593	0.864–2.936	0.136
T3	1.223	0.764–1.956	0.402	2.061	1.271–3.343	0.003	2.311	1.237–4.317	0.009

Model 1: unadjusted

Model 2: adjusted for age and sex

Model 3: adjusted for sex, age, BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antilipidemic drug use, and antihypertensive drug use

TyG, triglyceride–glucose; OR, odds ratio; CI, confidence interval; BMI, body mass index; eGFR, estimated glomerular filtration rate

Clinical outcomes

The overall in-hospital all-cause mortality rate was 0.94% ($n=103$), with 51 deaths due to cardiovascular causes, 14 due to cerebrovascular causes, and 38 due to various other causes.

Table 2 describes the results of the logistic regression analysis. The univariate logistic regression analysis indicated that there was no significant correlation between TyG index and in-hospital death. However, after adjusting for potential risks in model 2 (OR, 1.749; 95% CI 1.297–2.360; $P<0.001$) and model 3 (OR, 1.946; 95% CI 1.308–2.895; $P=0.001$), TyG index was considered as an independent risk factor for in-hospital all-cause mortality in patients with CAD. Using the T1 group as a reference, the multivariate logistic regression analysis revealed that the highest value of TyG index group (T3 group) was correlated with an increased risk of in-hospital death when adjusting for age and sex in model 2 (OR,

2.061; 95% CI 1.271–3.343; *P*=0.003). After adjusting for factors such as sex, age, BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antilipidemic drug use, and antihypertensive drug use, we found that the T3 group had a 2.3-fold risk of in-hospital all-cause mortality comparing with the T1 group (OR, 2.311; 95% CI 1.237–4.317; *P*=0.009).

The results of the restricted cubic splines are presented in Fig. 2, which showed that an elevated TyG index might be associated with an increased risk of in-hospital all-cause mortality (non-linear *P*=0.367).

Subgroup analyses

Table 3 shows the relationship between the TyG index and in-hospital death according to different diabetes statuses, including NGR, pre-DM, and DM. When adjusted for sex, age, BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antihypertensive drug use, and antilipidemic drug use in model 3, the TyG index as a continuous variable was an independent risk factor for in-hospital death in subgroup of DM (OR, 1.805; 95% CI 1.063–3.065; *P*=0.029). In DM subgroup, it was found that the T3 group (OR, 2.645; 95% CI 1.043–6.706;

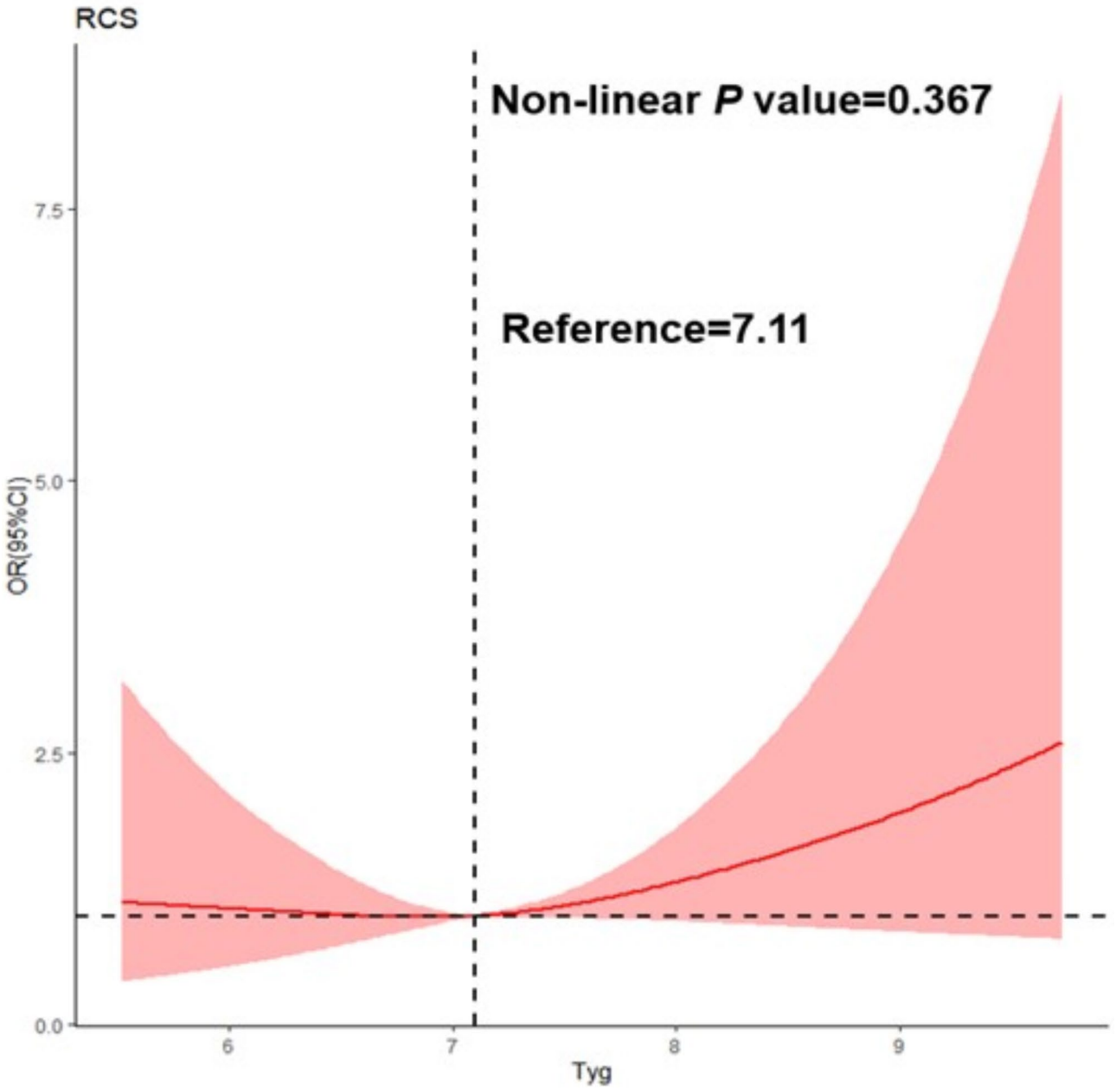

Fig. 2 Restricted cubic splines for the odds ratio of in hospital all cause death
TyG, triglyceride–glucose; OR, odds ratio; CI, confidence interval

Table 3 Associations between the TyG index and in-hospital death according to different diabetes statuses

Glucose metabolism state	Model 1			Model 2			Model 3		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value	OR	95% CI	*P*-value
NGR									
TyG index	0.450	0.160–1.264	0.130	0.768	0.264–2.237	0.629	1.088	0.293–4.046	0.900
T1	Reference			Reference			Reference		
T2	0.420	0.117–1.512	0.185	0.549	0.150–2.011	0.365	0.798	0.161–3.939	0.781
T3	0.307	0.040–2.388	0.260	0.580	0.072–4.648	0.608	0.224	0.013–3.948	0.307
Pre-DM									
TyG index	0.407	0.168–0.988	0.047	0.743	0.286–1.929	0.542	0.648	0.153–2.745	0.556
T1	Reference			Reference			Reference		
T2	0.760	0.305–1.896	0.557	1.025	0.405–2.597	0.958	0.927	0.284–3.025	0.900
T3	0.144	0.019–1.121	0.064	0.320	0.040–2.540	0.281	0.364	0.031–4.228	0.419
DM									
TyG index	1.253	0.884–1.775	0.204	1.701	1.182–2.447	0.004	1.805	1.063–3.065	0.029
T1	Reference			Reference			Reference		
T2	1.418	0.665–3.024	0.366	1.717	0.797–3.698	0.167	2.094	0.786–5.578	0.139
T3	1.585	0.785–3.199	0.199	2.452	1.196–5.030	0.014	2.645	1.043–6.706	0.040

Model 1: unadjusted

Model 2: adjusted for age and sex

Model 3: adjusted for sex, age, BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antilipidemic drug use, and antihypertensive drug use

TyG, triglyceride–glucose; OR, odds ratio; CI, confidence interval; BMI, body mass index; eGFR, estimated glomerular filtration rate; NGR, normoglycemia; Pre-DM, pre-diabetes mellitus; DM, diabetes mellitus

P=0.040) had a higher risk of in-hospital all-cause mortality by 2.6-fold than that of the T1 group after adjusting for potential risks in model 3.

Discussion

In this large cohort of patients with CAD, TyG index was independently associated with in-hospital death. After adjusting for potential risk factors such as sex, age, BMI, smoking, drinking, hypertension, eGFR, antiplatelet drug use, antilipidemic drug use, and antihypertensive drug use, the TyG index was still an independent risk predictor for in-hospital all-cause mortality. Additionally, the highest tertile of the TyG index group (T3 group) was correlated with a 2.311-fold risk of in-hospital death compared with the lowest tertile of the TyG index group (T1 group). For distinct diabetes statuses, an elevated TyG index had a predictive role for in-hospital all-cause mortality in CAD patients with diabetes but not in pre-DM or NGR patients.

Both type 1 diabetes mellitus (T1DM) and T2DM are associated with a higher risk of CAD, but the mechanisms differ. In T1DM, the absolute lack of insulin leads to persistent hyperglycemia, resulting in inflammation, oxidative stress, and the accumulation of advanced glycation end products (AGEs), thereby leading to endothelial dysfunction and promoting the development of atherosclerosis [18, 19]. The primary characteristic of T2DM is insulin resistance, leading to elevated levels of both blood glucose and insulin. Hyperinsulinemia increases the synthesis of molecules that promote the development of atherosclerosis [20]. Insulin resistance causes lipid abnormalities, impairs insulin's anti-inflammatory effects, and disrupts the delicate balance of endothelial function through various pathways, playing a critical role in both the initiation and progression of atherosclerosis [20]. Therefore, insulin resistance correlates well with a major risk factor for cardiovascular disease in many populations [10, 21, 22]. As a valuable tool for assessing insulin resistance in clinical practice [23], the TyG index has been demonstrated by many studies to have a positive relationship with the incidence of CAD. Park et al. [24] consecutively enrolled 1,250 asymptomatic individuals without traditional cardiovascular risk factors and confirmed that TyG index has significant value in predicting subclinical CAD and independent association with an increased risk of non-calcified or mixed coronary plaques. In a meta-analysis, it was also found that a higher TyG index was associated with an increased CAD incidence compared to a lower TyG index [13]. Besides, a study of 234 patients with ACS showed that the TyG index may serve as a potential predictor of calcification patterns and plaque vulnerability [25].

An elevated TyG index reflects disorders in both glucose and lipid metabolism, which can lead to vascular endothelial damage and increased inflammation, and is therefore regarded as a valuable predictor of CAD severity [26]. Wang et al. [27] reviewed the coronary angiography (CAG) results of 2,792 CAD patients, and reported that an increased TyG index was associated with a higher risk of multi-vessel CAD. Another cohort study observed that the TyG index was related to the stenosis severity and number of stenosed coronary arteries [28]. These

findings suggested that the TyG index is expected to be a useful indicator of CAD severity before CAG is performed in clinical practice.

Numerous studies have also established a robust association between TyG index and prognostic risk assessment of different types of CAD. Erdoğan et al. [29] reported that TyG index assisted in the prognostic stratification of patients with chronic coronary syndrome and emerged as a more effective predictor of major adverse cardiac events (MACEs) when compare to atherogenic index of plasma. Karadeniz et al. [30] enrolled 646 patients with ST-elevation myocardial infarction and 1,048 patients with non-ST-elevation myocardial infarction to show that TyG index can predict the incidence of 5-years MACEs in all acute coronary syndrome patients, suggesting that TyG index may be a useful marker for the clinical outcomes in these patients. In addition, A study of 8,862 patients with three-vessel CAD found that an elevated TyG index was significantly associated with an increased risk of MACEs during a median follow-up of 7.5 years [31]. However, the relationship between TyG index and the "hard" endpoint of death in previous studies remains inconclusive. Most studies did not find a significant correlation between TyG index and death in CAD patients [32]. Only a few sample size sample studies in high-risk subgroups of patients with ACS have reported a positive correlation between TyG index levels and mortality [33, 34]. In the present study, we found that the level of LDL-C and HbA1c, the prevalence of hypertension and DM increased with the rise of TyG index level. More importantly, the TyG index had fair prognostic ability of in-hospital all-cause mortality in CAD patients.

Evidence accumulated for decades identifies insulin resistance as a major cardiovascular risk factor in patients with diabetes [10], based on which, we performed a subgroup analysis according to the presence of concomitant diabetes in patients with CAD and found that a higher TyG index was related with an increased risk of in-hospital death in the DM subgroup, but not in the NGR or pre-DM subgroup. This result is basically consistent with previous studies showing that the TyG index has a stronger predictive value in patients with CAD and diabetes than in their non-diabetic counterparts [31]. A study of non-diabetic patients with acute myocardial infarction also illustrated that TyG index was not a reliable predictor of MACE and all-cause mortality at a 1-year follow-up [35]. This kind of result might be explained by the overall low insulin resistance in NGR patients. Notably, the results of our study in the pre-DM subgroup were also negative. Although studies have shown that TyG index may be associated with CAD severity or risk of heart failure in pre-DM participants, its prediction of MACEs has not been reported previously [25, 36]. Regarded as a risk factor for future CVD, there is no consensus on whether early pharmacological intervention should be used for pre-DM [37]. From our findings, the decision to initiate medication for such patients should be made with caution.

Strength and limitations

To the best of our knowledge, our study represents the largest cohort investigation to date assessing in-hospital all-cause mortality in patients diagnosed with CAD using the TyG index. This study, while offering valuable insights, is not without its limitations. First, this study involved a single-center and only enrolled the Asian population; therefore, the results should be interpreted cautiously. Second, this research is retrospective and non-randomized in design, which may affect the ability to establish causal relationships and requires a cautious interpretation of the findings. Third, the medication durations and dosages of hypoglycemic drugs, antiplatelet drugs, antilipidemic drugs, and antihypertensive drugs for this cohort were not collected, which might have resulted in bias for these factors in the logistic models.

Conclusions

An increased TyG index was correlated with a higher risk of in-hospital death. Our study indicated that TyG index as an estimation index for evaluating IR could be a valuable predictor of in-hospital all-cause mortality of CAD patients, especially for individuals with DM.

Abbreviations

CAD	Coronary artery disease
CVD	Cardiovascular disease
T2DM	Type 2 diabetes mellitus
TyG	Triglyceride-glucose
HOMA-IR	Homeostatic Model Assessment of insulin resistance
TG	Triglyceride
FBG	Fasting blood glucose
MI	Myocardial infarction
eGFR	Estimated glomerular filtration rate
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
HR	Heart rate
TC	Total cholesterol
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
HbA1c	Glycated hemoglobin A1c
BMI	Body mass index
DM	Diabetes mellitus
Pre-DM	Pre-diabetes mellitus
NGR	Normoglycemia
OR	Odds ratio
CI	Confidence interval
ACS	Acute coronary syndrome
CAG	Coronary angiography
MACEs	Major adverse cardiac events
ACEI	Angiotensin converting enzyme inhibitor
ARB	Angiotensin receptor blocker

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-024-20551-4.

Supplementary Table 1: Baseline characteristics according to survivor and non-survivor groups

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Author contributions
QS, XW, WX, BF and XM designed the study, CY and FW reviewed and revised the manuscript. BF and QS coded and analyzed the data. XM wrote the manuscript. ZZ and XM collected the data. CX and YL interpreted the data. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This cohort study conformed to the Declaration of Helsinki and was approved by the Ethics Committee of Beijing Hospital. Written informed consent was obtained from all the patients.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Cardiology, Beijing Hospital, National Center of Gerontology, Institute of Geriatric Medicine, Chinese Academy of Medical Sciences, No. 1 Dongdan Dahua Road, Dongcheng District, Beijing 100730, China
[2]Graduate School of Peking Union Medical College, Chinese Academy of Medical Sciences, Beijing 100730, China
[3]Department of Clinical Trial Center, Beijing Tiantan Hospital, Capital Medical University, Beijing 100070, China
[4]Department of Pharmacy, Institute of Geriatric Medicine, Beijing Hospital, National Center of Gerontology, Chinese Academy of Medical Sciences, Beijing Key Laboratory of Assessment of Clinical Drugs Risk and Individual Application (Beijing Hospital), Beijing 100730, China
[5]Hypertension Center, State Key Laboratory of Cardiovascular Disease of China, National Center for Cardiovascular Diseases of China, Fuwai Hospital, Chinese Academy of Medical Sciences, Peking Union Medical College, Beijing 100037, China
[6]Emergency Center, State Key Laboratory of Cardiovascular Disease of China, National Center for Cardiovascular Diseases, National Clinical Research Center of Cardiovascular Diseases, Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100037, China
[7]Peking University Fifth School of Clinical Medicine, Beijing 100730, PR China

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