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Triglyceride-glucose index correlates with the incidences and prognoses of cardiac arrest following acute myocardial infarction: data from two large-scale cohorts

Huiruo Liu[1†], Liangshan Wang[1†], Hong Wang[1], Xing Hao[1], Zhongtao Du[1], Chenglong Li[1] and Xiaotong Hou[1*]

Abstract

Background The triglyceride-glucose (TyG) index, renowned for its efficacy and convenience in assessing insulin resistance, has been validated as a reliable indicator for various cardiovascular conditions. The current study aims for clarifying the link of TyG with the incidences and prognoses of cardiac arrest (CA) following acute myocardial infarction (AMI).

Methods Our analysis is a multicenter, retrospective study utilizing data from the Medical Information Mart for Intensive Care IV and the eICU Collaborative Research Database. Patients with AMI for whom TyG could be calculated within the first 24 h after admission were included. The main endpoints were in-hospital and ICU mortalities. Correlations between TyG and outcomes were evaluated using logistic regression models, restricted cubic splines (RCS), as well as correlation and linear analyses. Overlap weighting (OW), inverse probability of treatment weighting (IPTW), and propensity score matching (PSM) methodologies were utilized to balance the cohorts, thereby minimizing potential biases. Subgroup analyses were performed in accordance with identified modifiers.

Results In total, 5208 individuals diagnosed with AMI, among whom 371 developed CA, were ultimately included. Higher TyG levels were observed among AMI populations with CA compared to those without [9.2 (8.7–9.7) vs. 9.0 (8.5–9.4)], and TyG demonstrated a moderate discriminatory capacity for identifying CA occurrences within entire AMI populations. Multivariate logistic regressions revealed TyG serves a significant risk indicator for both in-hospital (OR 1.711) and ICU mortalities (OR 1.520) in AMI-CA patients, and it is also associated with prolonged LOSs. RCS analyses confirmed linear relationships of ascending TyG with increased mortality risks for AMI-CA (*P* for nonlinearity: 0.592 and 0.816, respectively), which persisted following PSM, OW, and IPTW adjustments. Subgroup analyses further identified a strong link of the TyG with mortality rates among elders, females, individuals with BMI < 28 kg/m^2, and those with hypertension.

[†]Huiruo Liu and Liangshan Wang contribute equally to this manuscript.

*Correspondence:
Xiaotong Hou
xt.hou@ccmu.edu.cn

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

Conclusions Elevated TyG levels were found to apparently correlate with higher prevalence and adverse outcomes regarding CA in patients with AMI. Our findings point a fresh insight into the significance of the TyG in critically ill coronary conditions.

Keywords Triglyceride-glucose index, Acute myocardial infarction, Cardiac arrest, Insulin resistance

Introduction

Cardiac arrest (CA), referring to an abrupt cessation of cardiac activity, represents a highly prevalent and exceedingly lethal medical condition with substantial global mortality rates [1–3]. Acute myocardial infarction (AMI) persists one of the predominant causes for CA [4], with CA occurring in roughly 5.0–7.0% of AMI cases [5, 6]. Once presenting with CA, the mortalities for AMI patients exhibit a sharp surge, escalating from less than 4% to approximately 40–50% [7, 8]; despite gradual improvements regarding survival for acute myocardial infarction complicating cardiac arrest (AMI-CA) following advances in the field of acute cardiovascular care, it still remains at a rather low level [6, 9]. More worryingly, predicting the occurrence of CA following AMI continues to pose a formidable challenge.

As one of the gravely fatal complications of AMI, AMI-CA likely shares some common risk factors with atherosclerosis, such as diabetes mellitus (DM) [10, 11], obesity [12], and hyperlipidemia [13, 14]. In an Asia cohort consisting of 101,113 subjects experiencing acute coronary syndrome and an America cohort comprising 5208 patients diagnosed with AMI, CA was conformably showed to be more common in individuals exhibiting greater admission insulin resistance extent [15, 16]. Patients with ST-segment elevation AMI were potentially more susceptible to experiencing CA if having metabolic syndrome/insulin resistance syndrome [17]. Stress hyperglycemia commonly appeared in patients who occurred CA [18, 19]; and further, elevated stress hyperglycemia ratio has been confirmed to be independently associated with the incidence of CA among those presenting with ACS [20]. Moreover, diabetic status has been reported as an independent risk predictor for mortality events among those who underwent AMI-CA [21]. Collectively, the available evidence suggests that glucolipid metabolism disorders might play a role in the pathogenesis of CA following AMI, implying a possible association between insulin resistance (IR) and the occurrences and unfavorable prognoses of AMI-CA.

The triglyceride-glucose (TyG) index, renowned for its robustness and convenience regarding assessing insulin resistance (IR), has gained increasing recognition over the years owing to its diagnostic significance in cardiovascular fields [22–24]. Significant correlations between TyG index and the risks as well as adverse outcomes of multiple acute cardiovascular conditions, encompassing but not limited to acute coronary syndrome [25, 26], malignant arrhythmia [27, 28], acute heart failure [29], and cardiogenic shock [15] have already been reported; furthermore, existing evidence also linking TyG and its derivative indices to chronic heart failure [30], hypertension [31], atrial fibrillation [32, 33], etc. More importantly, researchers have substantiated the predictive potential of the TyG index in relation to CA risks and clinical outcomes within the general population [34, 35]. Based on these findings, we speculated the potential utility of TyG as a predictive tool for assessing the occurrences and prognoses of CA following AMI, while conclusive evidence is still absent.

Hereto, we supposed to investigate the links between TyG and incidences as well as subsequent outcomes of CA in patients with AMI, aiming for identifying a novel indicator for accurate identification or timely intervention in this specific patient population from an energy metabolism perspective.

Methods

Study design and population

The present multicenter, retrospective, observational study was performed across 209 medical centers to evaluate the links of TyG with CA incidences among the AMI population, as well regarding their clinical prognoses. Our analysis collected 4420 admission records from the eICU Collaborative Research Database (eICU-CRD) and 788 from the Medical Information Mart for Intensive Care IV (MIMIC-IV) database. Diagnoses were ascertained based on the codes of the International Classification of Diseases, 9th and 10th Revision, and/or diagnosis string (Table S1). Patients diagnosed with AMI were screened for eligibility. The exclusion criteria for participants were as follows: (a) age < 18 years; and (b) insufficient FBG or TG values recorded within the initial 24 h after admission. Only records pertaining to the initial ICU admission were extracted for individuals with multiple admissions.

Our analysis comprised two primary components. Firstly, we assessed the differences regarding TyG levels among AMI patients occurring and not occurring CA. Next, we grouped the overall AMI-CA populations in accordance with TyG levels or mortality status for additional analyses.

Data source

MIMIC-IV, an extensively utilized, single-center, and publicly available database, encompassing comprehensive

and high-caliber medical information of individuals admitted to the ICUs at Beth Israel Deaconess Medical Center (BIDMC). The data included in this study encompasses patient encounters, laboratory tests, and treatment records from 2008 to 2019, as documented in this database. This undertaking is approved by the institutional review boards of BIDMC, with a waiver granted for obtaining informed consent. Data extraction was conducted by one author (HRL).

eICU-CRD, a large and longitudinal database comprising more than 200,000 records of ICU admissions across 208 hospitals in the United States. The data used in this study were collected from this database between 2014 and 2015, which provides a detailed account of patient vital signs, laboratory results, and medication administrations during ICU stays. This project is freely accessible, officially managed, and recognized for its superior data quality. Data extraction was performed by one author (HRL).

Data extraction

Data was extracted using PostgreSQL software (version 13.7.2) and Navicat Premium (version 16). The extracted data are presented in detail as follow: (a) demographic data, including age, sex, weight, as well as height; (b) vital signs within 24 h after admission, such as temperature, respiratory rates, heart rates, diastolic blood pressures, systolic blood pressures, also SpO_2; (c) comorbidities, including diabetes mellitus (DM), acute renal failure, hypertension, previous myocardial infarction (MI), chronic heart failure (CHF), dyslipidemia, malignant ventricular arrhythmia (MVA), chronic kidney disease, chronic obstructive pulmonary disease, stroke, also cardiogenic shock; (d) laboratory data within 24 h after admission such as white blood cells (WBC), red blood cells, platelet counts, creatinine, blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate aminotransferase (AST), hemoglobin (Hb), creatine kinase-myocardial band (CK-MB), Troponin T, lactate (LAC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), sodium, potassium, fasting blood glucose (FBG), also triglyceride (TG); (e) treatment during hospitalization, such as the utilization of vasoactive agents, coronary artery bypass grafting (CABG), percutaneous coronary intervention, statins, antiplatelet drugs, β-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, also oral anticoagulants; and (f) severity of illness scores such as acute physiology score (APS), and Glasgow coma scale (GCS) score. No personally identifiable information was disclosed. TyG index was calculated by: ln [fasting TG (mg/dl) * FBG (mg/dl)/2] [36]. BMI was calculated by: weight (kg)/[height $(m)^2$]. Mean arterial pressure (MAP) was calculated by: [2 * diastolic blood pressure (mmHg) + systolic blood pressure (mmHg)]/3.

Endpoints

The main endpoints were in-hospital and ICU mortality among patients with AMI-CA. Additional outcomes, including the length of stay in hospital (LOS-H), as well as length of stay in ICU (LOS-ICU), were showed as secondary endpoints.

Statistical analysis

Statistical analysis was performed using R software, as well as Graph prism. The continuous variables were presented as median with interquartile range (IQR, 25th-75th percentiles), and examined using Mann–Whitney U or Kruskal–Wallis tests. For the Kruskal–Wallis test, post-hoc analysis was conducted using the Dunn's test with Bonferroni correction to identify specific group differences while controlling for type I error inflation. The categorical variables were showed as absolute numbers with percentages and analyzed through chi-square tests. Missing data were imputed through the Multiple Imputation by Chained Equations algorithm, with the number of imputations determined based on the proportion of missing data and the complexity of the dataset. Variables with a missing rate exceeding 50% were excluded from the analysis. Statistical significance was established at an α level of 0.05.

Univariate and multivariate logistic models were employed to evaluate the link of TyG with mortalities. And the TyG was assessed as both a categorical and a continuous variable. Confounding factors were identified according to baseline characteristic differences and clinical relevance. The trend regarding mortalities or LOSs was evaluated through ordinal categorization of the TyG index. RCS curves were used for exploring the potential non-linear association between TyG and mortality risks in the AMI-CA population. After stratifying the overall AMI-CA cohort using the RCS-derived threshold (TyG index < 9.233 and ≥ 9.233), a propensity score matching (PSM) procedure utilizing a 1:1 ratio was conducted, yielding matched groups of 136 patients each. To further standardize the population and address potential biases, the inverse probability of treatment weighting (IPTW) and overlap weighting (OW) methods were applied. Multivariate linear and correlation analyses were applied for assessing the associations of TyG with certain continuous indicators such as LOS, APS score, GCS score, etc. The AUC comparisons between indicators were evaluated using DeLong's tests. Subgroup analyses were performed stratifying by sex, age, BMI, and hypertensive status. Multivariate logistic regression models were used which incorporated all modifiers in Model 3 except for the one used for stratification.

Results

Elevated TyG index was observed among AMI populations who occurred CA

The flowchart depicting the process of patient selection has been illustrated in Fig. 1. Overall, a sum of 5208 individuals [median age: 64.0 (IQR 55.0–74.0); male: 65.42% (n = 3407)] diagnosed with AMI at admission were ultimately enrolled, comprising 4420 participants from the eICU-CRD cohort and 788 from the MIMIC-IV cohort. Among them, 371 occurred CA. Data regarding baseline characteristics between AMI patients with and without CA have been outlined in Table 1.

Notably, AMI patients occurring CA exhibited apparently higher TyG levels compared to those who did not [9.2 (8.7–9.7) vs. 9.0 (8.5–9.4)]. Next, we evaluated the link of TyG with the risks of occurring CA among AMI patients, utilizing logistic regression analyses (Table 2). As depicted, the unadjusted model demonstrated TyG to be a risk indicator for the diagnosis of CA among individuals with AMI (OR 1.497; 95% CI 1.307–1.714). After controlling for age, sex, and BMI, this correlation was still significant (OR 1.500; 95% CI 1.298–1.733). In the fully adjusted model 3, the TyG elevation still maintained an apparent relationship with an increased incidence of CA (OR 1.237; 95% CI 1.032–1.482).

Moreover, as presented in Fig. 2, the receiver operating characteristic (ROC) curve indicated that TyG exhibited moderate performance regarding discerning CA occurrences within entire AMI populations (AUC 0.595, 95% CI 0.565–0.625), surpassing certain indicators such as age, sex, BMI, HR, MAP, hypertension, DM, CHF,

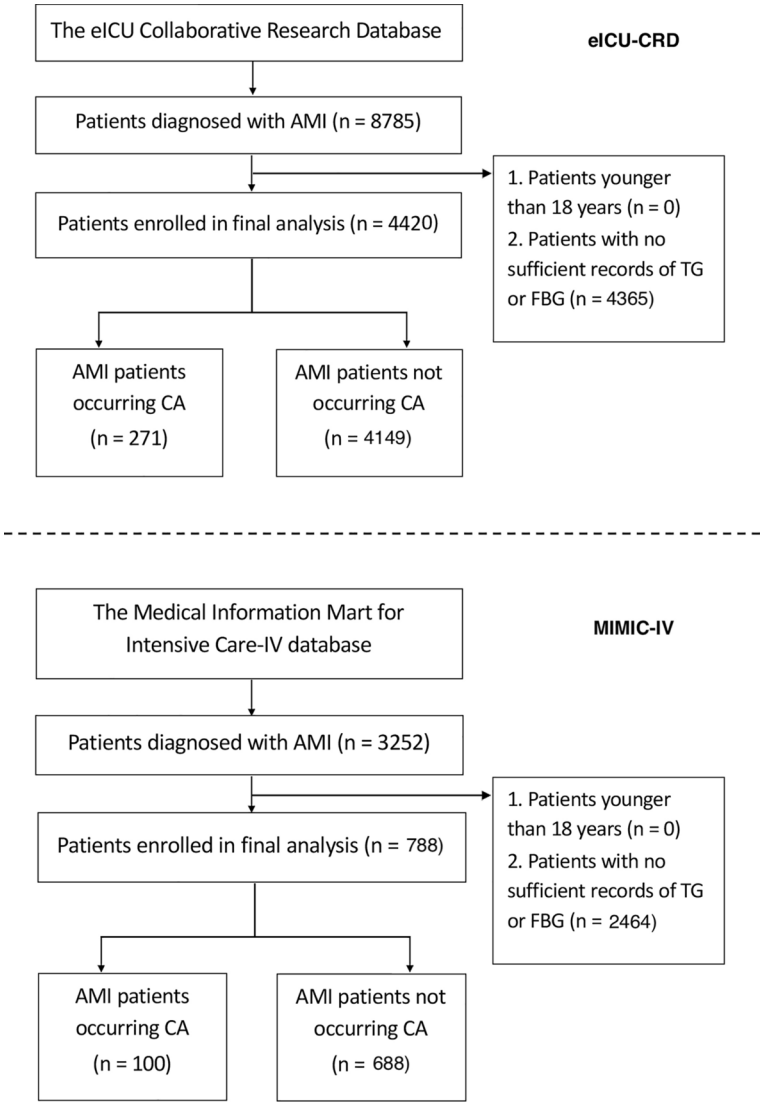

Fig. 1 Flowchart of patient selection from the eICU-CRD and the MIMIC-IV databases. TG, triglyceride; FBG, fasting blood glucose; AMI, acute myocardial infarction; CA, cardiac arrest; eICU-CRD, eICU Collaborative Research Database; MIMIC-IV, Medical Information Mart for Intensive Care-IV

Table 1 Baseline characteristics between AMI populations with and without CA

Variables	Total AMI populations (n = 5208)	AMI populations without CA (n = 4837)	AMI populations with CA (n = 371)	*P* value
TyG index	9.0 (8.6-9.5)	9.0 (8.5-9.4)	9.2 (8.7-9.7)	< 0.001
FBG	127.0 (106.0-168.0)	126.0 (105.0-164.0)	161.0 (126.0-227.0)	< 0.001
TG	117.0 (84.0-172.0)	117.0 (84.0-171.0)	115.0 (83.0-176.0)	0.654
Demographic data				
Age, y	64.0 (55.0-74.0)	65.0 (55.0-75.0)	63.0 (55.0-72.0)	0.060
Sex, n (%)				0.022
Male	3407 (65.42)	3144 (65.00)	263 (70.89)	
Female	1801 (34.58)	1693 (35.00)	108 (29.11)	
BMI, kg/m^2	28.7 (25.1-33.0)	28.6 (25.0-32.9)	29.3 (25.8-34.1)	0.020
Vital signs				
Heart rate, bpm	77.0 (68.1-87.5)	76.8 (68.0-87.3)	79.3 (70.2-90.3)	0.002
MAP, mmHg	83.5 (75.9-92.1)	83.8 (76.0-92.3)	80.7 (74.2-89.8)	< 0.001
RR, bpm	18.5 (16.5-21.0)	18.4 (16.5-20.9)	19.5 (17.1-22.5)	< 0.001
Temperature, °C	36.7 (36.6-36.9)	36.7 (36.6-36.9)	36.7 (36.2-37.0)	< 0.001
SpO_2, %	97.0 (95.7-98.2)	97.0 (95.7-98.2)	97.5 (95.9-98.8)	< 0.001
Comorbidities, n (%)				
Hypertension	1091 (20.95)	1005 (20.78)	86 (23.18)	0.273
DM	293 (5.63)	262 (5.42)	31 (8.36)	0.018
Previous MI	1547 (29.70)	1411 (29.17)	136 (36.66)	0.002
CHF	365 (7.01)	317 (6.55)	48 (12.94)	< 0.001
ARF	703 (13.50)	583 (12.05)	120 (32.35)	< 0.001
Stroke	315 (6.05)	298 (6.16)	17 (4.58)	0.219
COPD	277 (5.32)	253 (5.23)	24 (6.47)	0.306
CKD	385 (7.39)	355 (7.34)	30 (8.09)	0.596
Dyslipidemia	649 (12.46)	611 (12.63)	38 (10.24)	0.179
MVA	113 (2.17)	107 (2.21)	6 (1.62)	0.449
Cardiogenic shock	375 (7.20)	273 (5.64)	102 (27.49)	< 0.001
Laboratory data				
WBC, K/uL	11.0 (8.6-13.9)	10.8 (8.5-13.5)	14.7 (11.3-18.0)	< 0.001
RBC, K/uL	4.3 (3.8-4.8)	4.3 (3.8-4.8)	4.3 (3.8-4.7)	0.475
PLT, K/uL	213.6 (174.0-260.0)	214.0 (174.0-260.0)	210.2 (177.1-265.8)	0.996
Hb, g/dL	13.1 (11.5-14.4)	13.1 (11.5-14.4)	13.0 (11.3-14.4)	0.651
ALT	33.0 (21.6-55.0)	31.5 (21.0-50.5)	79.0 (41.6-163.9)	< 0.001
AST	55.5 (28.0-138.0)	51.0 (27.0-123.0)	169.0 (77.1-310.0)	< 0.001
BUN	17.0 (13.0-24.0)	17.0 (13.0-24.0)	19.8 (14.8-27.0)	< 0.001
Creatinine	1.0 (0.8-1.3)	1.0 (0.8-1.3)	1.1 (0.9-1.6)	< 0.001
CK-MB, ng/mL	40.0 (10.0-118.6)	38.0 (9.6-113.4)	76.3 (14.9-218.4)	< 0.001
TnT, ng/mL	1.2 (0.3-3.6)	1.2 (0.3-3.6)	1.9 (0.5-5.7)	0.009
LAC, mmol/L	2.0 (1.4-3.3)	1.9 (1.3-3.0)	2.8 (1.7-4.8)	< 0.001
TC, mg/dL	160.0 (132.0-190.0)	160.5 (132.0-191.0)	151.0 (120.0-180.5)	< 0.001
HDL-C, mg/dL	39.0 (32.0-47.5)	39.0 (32.0-47.0)	40.0 (31.0-48.0)	0.738
LDL-C, mg/dL	92.0 (67.7-120.0)	93.0 (68.0-120.0)	88.0 (59.8-113.0)	0.024
Sodium, mEq/L	138.0 (136.0-140.0)	138.0 (136.0-140.0)	138.5 (136.0-140.7)	0.003
Potassium, mEq/L	4.1 (3.8-4.4)	4.1 (3.8-4.3)	4.1 (3.8-4.4)	0.719
GCS score	15.0 (15.0-15.0)	15.0 (15.0-15.0)	15.0 (15.0-15.0)	15.0 (15.0-15.0)
APS score	31.0 (22.0-44.0)	30.0 (22.0-41.0)	57.5 (35.0-91.0)	< 0.001
Therapy data, n (%)				
Vasoactive agents	592 (11.37)	440 (9.10)	152 (40.97)	< 0.001
PCI	1686 (32.37)	1567 (32.40)	119 (32.08)	0.899
CABG	118 (2.27)	108 (2.23)	10 (2.70)	0.564
Statins	1927 (37.00)	1780 (36.80)	147 (39.62)	0.278
Antiplatelet drugs	3567 (68.49)	3307 (68.37)	260 (70.08)	0.494

Table 1 (continued)

Variables	Total AMI populations (n = 5208)	AMI populations without CA (n = 4837)	AMI populations with CA (n = 371)	*P* value
β-blockers	2849 (54.70)	2655 (54.89)	194 (52.29)	0.333
ACEIs/ARBs	1487 (28.55)	1391 (28.76)	96 (25.88)	0.236
Oral anticoagulants	322 (6.18)	305 (6.31)	17 (4.58)	0.184

AMI acute myocardial infarction; *CA* cardiac arrest; *TyG index* triglyceride-glucose index; *FBG* fasting blood glucose; *TG* triglyceride; *BMI* body mass index; *MAP* mean blood pressure; *RR* respiratory rate; *DM* diabetes mellitus; *MI* myocardial infarction; *CHF* chronic heart failure; *ARF* acute renal failure; *COPD* chronic obstructive pulmonary disease; *CKD* chronic kidney disease; *MVA* malignant ventricular arrhythmia; *WBC* white blood cell; *RBC* red blood cell; *PLT* platelet; *Hb* hemoglobin; *ALT* alanine aminotransferase; *AST* aspartate aminotransferase; *BUN* blood urea nitrogen; *CK-MB*, creatine kinase-myocardial band; *TnT* troponin T; *LAC* lactate; *TC* total cholesterol; *HDL-C* high-density lipoprotein cholesterol; *LDL-C* low-density lipoprotein cholesterol; *GCS* glasgow coma scale; *APS* acute physiology score; *PCI* percutaneous coronary intervention; *CABG* coronary artery bypass grafting; *ACEIs/ARBs* angiotensin-converting enzyme inhibitors/angiotensin receptor blockers

Table 2 Logistic regression analyses for the correlation between the TyG index and the occurrence of CA among AMI populations

TyG index	OR	95% CI	*P* value
Model 1	1.497	1.307–1.714	< 0.001
Model 2	1.500	1.298–1.733	< 0.001
Model 3	1.237	1.032–1.482	0.021

TyG triglyceride-glucose; *CA* cardiac arrest; *AMI* acute myocardial infarction; *OR* odds ratio; *CI* confidence interval

Model 1: unadjusted

Model 2: Adjusted for age, sex, and body mass index

Model 3: Adjusted for age, sex, body mass index, heart rate, mean arterial pressure, respiratory rate, SpO_2, temperature, hypertension, diabetes mellitus, chronic heart failure, chronic kidney disease, stroke, previous myocardial infarction, cardiogenic shock, malignant ventricular arrhythmia, white blood cell, alanine transaminase, aspartate transaminase, blood urea nitrogen, creatinine, troponin T, creatine kinase-myocardial band, lactate, total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, sodium, potassium, vasoactive agents, coronary artery bypass grafting, and percutaneous coronary intervention

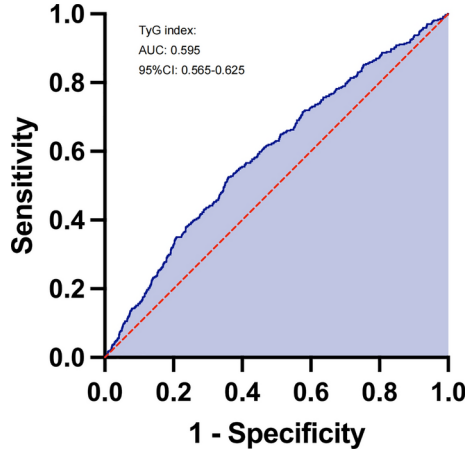

Fig. 2 ROC curve of TyG index for identifying patients diagnosed with CA from the overall AMI population. TyG, triglyceride-glucose; AUC, area under the cure; CI, confidence interval

previous MI, CKD, MVA, dyslipidaemia, TnT, TC, HDL-C, LDL-C, TG, and CABG (Table S2).

Collectively, these findings provided evidence supporting the positive correlation of elevated TyG with CA occurrences among individuals with AMI, suggesting that TyG holds potential as a valuable predictor for CA occurrence in this specific patient population.

Baseline characteristics and clinical outcomes regarding AMI-CA populations in different TyG tertiles

We then explored the correlation between TyG and clinical prognoses of AMI-CA patients. As presented in Table S3 and S4, out of 371 AMI patients who experienced CA, 98 died during hospitalization while 80 died during ICU stay. Importantly, our data revealed that both survivors at hospital discharge [9.2 (8.7–9.6) vs. 9.5 (8.9–10.0)] and survivors at ICU discharge [9.2 (8.7–9.6) vs. 9.4 (8.8–9.9)] exhibited significantly lower TyG levels compared to corresponding non-survivors. Several disparities were also observed regarding other general characteristics of survivors and non-survivors (Table S3 and S4). Then, AMI populations post-CA were further categorized into three groups based on the tertiles of the distribution of on-admission TyG index (Table 3). As illustrated, patients with higher TyG levels tended to exhibit overweight and younger age, along with higher levels of heart rate, WBC, ALT, AST, BUN, creatinine, and LAC, but lower levels of HDL-C. Moreover, among those in the highest TyG tertile, there were more patients that were concomitant with DM, ARF, and CS, also higher utilization rates of vasoactive agents and reduced frequencies of PCI. Meanwhile, the T3 group demonstrated greater APS scores upon admission.

The clinical endpoints for each of the three groups were documented in Table 4. Compared to individuals with lower TyG, those exhibiting higher TyG showed obviously elevated in-hospital (19.4% vs. 23.4% vs. 36.6%) and ICU (15.3% vs. 17.7% vs. 31.7%) mortality rates, as well as longer LOS-H (5.2 days vs. 5.1 days vs. 7.8 days) and LOC-ICU (2.8 days vs. 3.0 days vs. 5.1 days). Next, the trend analyses were executed (Fig. 3), manifesting an apparent upward trend in both in-hospital and ICU mortality as TyG tertiles increased, with all *P* for trend < 0.020. Meanwhile, a discernibly increasing trend was also seen for LOS-H and LOS-ICU across these three groups, with all *P* for trend < 0.010.

Collectively, these findings suggested a possible link between elevated TyG and unfavourable prognoses among AMI populations presenting with CA.

Table 3 Baseline characteristics of AMI patients who occurred CA stratified according to TyG tertiles

Characteristics	T1 (n = 124)	T2 (n = 124)	T3 (n = 123)	*P* value
TyG index	8.6 (8.2-8.8)	9.2 (9.1-9.4)	9.9 (9.7-10.3)	< 0.001
FBG	128.5 (113.0-157.0)	161.0 (130.3-208.8)	232.0 (176.0-335.0)	< 0.001
TG	73.0 (58.0-93.8)	123.5 (99.3-159.5)	188.0 (145.0-264.0)	< 0.001
Demographic data				
Age, y	64.5 (56.3-74.0)	65.0 (56.0-72.0)	61.0 (53.0-67.0)	0.019
Sex, n (%)				0.079
Male	85 (68.6)	97 (78.2)	81 (65.9)	
Female	39 (31.5)	27 (21.8)	42 (34.2)	
BMI, kg/m^2	27.8 (25.1-33.9)	28.6 (25.9-32.9)	30.5 (26.4-35.8)	0.025
Vital signs				
Heart rate, bpm	76.5 (69.2-85.8)	79.1 (70.9-91.8)	85.3 (71.1-93.3)	0.009
MAP, mmHg	80.3 (72.9-87.8)	82.2 (75.1-90.6)	80.0 (74.2-89.9)	0.560
Temperature, °C	36.7 (36.4-36.9)	36.7 (36.4-37.0)	36.5 (34.1-37.1)	0.213
SpO_2, %	97.4 (95.9-99.0)	97.2 (95.1-98.7)	97.6 (96.3-98.7)	0.317
Comorbidities, n (%)				
Hypertension	23 (18.6)	30 (24.2)	33 (26.8)	0.289
DM	5 (4.0)	8 (6.5)	18 (14.6)	0.007
Previous MI	47 (37.9)	42 (33.9)	47 (38.2)	0.731
CHF	11 (8.9)	17 (13.7)	20 (16.3)	0.213
ARF	34 (27.4)	33 (26.6)	53 (43.1)	0.008
Stroke	6 (4.8)	6 (4.8)	5 (4.1)	0.945
COPD	7 (5.7)	11 (8.9)	6 (4.9)	0.399
CKD	9 (7.3)	7 (5.7)	14 (11.4)	0.234
Dyslipidaemia	10 (8.1)	13 (10.5)	15 (12.2)	0.561
MVA	2 (1.6)	2 (1.6)	2 (1.6)	0.999
Cardiogenic shock	24 (19.4)	35 (28.2)	43 (35.0)	0.022
Laboratory data				
WBC, K/uL	12.4 (10.3-16.0)	14.9 (11.5-17.6)	16.2 (13.1-21.0)	< 0.001
RBC, K/uL	4.3 (3.7-4.6)	4.4 (3.9-4.7)	4.3 (3.7-4.8)	0.343
PLT, K/uL	210.8 (179.1-269.9)	219.2 (173.0-256.5)	207.0 (177.3-259.8)	0.771
Hb, g/dL	12.8 (11.1-14.0)	13.3 (11.9-14.5)	12.8 (10.8-14.9)	0.165
ALT	63.0 (37.5-135.5)	71.0 (38.2-145.5)	113.3 (49.6-214.6)	0.002
AST	116.8 (56.5-208.4)	170.0 (71.5-295.5)	224.0 (112.0-477.0)	< 0.001
BUN	18.0 (14.0-25.5)	18.6 (14.424.3)	22.0 (16.7-29.7)	0.006
Creatinine	1.0 (0.8-1.5)	1.1 (0.9-1.5)	1.3 (1.0-1.9)	< 0.001
CK-MB, ng/mL	42.2 (15.4-189.5)	78.5 (14.1-216.0)	101.5 (15.8-251.9)	0.435
TnT, ng/mL	1.1 (0.4-3.5)	2.2 (0.7-6.1)	1.3 (0.7-8.8)	0.236
LAC, mmol/L	2.2 (1.4-3.3)	2.5 (1.7-4.1)	3.7 (2.3-6.0)	< 0.001
TC, mg/dL	147.0 (115.0-179.0)	149.0 (124.5-180.8)	155.0 (128.3-181.0)	0.612
HDL-C, mg/dL	43.0 (36.0-54.8)	37.0 (31.5-45.8)	34.5 (27.0-44.3)	< 0.001
LDL-C, mg/dL	89.0 (57.0-113.0)	84.5 (59.9-111.8)	87.8 (67.5-115.3)	0.828
Sodium, mEq/L	138.5 (136.5-140.8)	138.0 (136.1-140.3)	138.8 (135.5-141.0)	0.642
Potassium, mEq/L	4.1 (3.9-4.4)	4.1 (3.8-4.4)	4.0 (3.6-4.3)	0.134
GCS score	15.0 (14.0-15.0)	15.0 (15.0-15.0)	15.0 (15.0-15.0)	0.611
APS score	41.0 (28.0-66.0)	54.0 (30.5-84.0)	79.0 (55.0-104.8)	< 0.001
Therapy data, n (%)				
Vasoactive agents	41 (33.1)	47 (37.9)	64 (52.0)	0.007
PCI	46 (37.1)	46 (37.1)	27 (22.0)	0.013
CABG	2 (1.6)	3 (2.4)	5 (4.1)	0.480
Statins	55 (44.4)	45 (36.3)	47 (38.2)	0.399
Antiplatelet drugs	93 (75.0)	86 (69.4)	81 (65.9)	0.285
β-blockers	69 (55.7)	63 (50.8)	62 (50.4)	0.656
ACEIs/ARBs	36 (29.0)	33 (26.6)	27 (22.0)	0.435

Table 3 (continued)

Characteristics	T1 (n = 124)	T2 (n = 124)	T3 (n = 123)	*P* value
Oral anticoagulants	7 (5.7)	7 (5.7)	3 (2.4)	0.380
Bystander CPR > 15min	6 (4.8)	7 (5.7)	5 (4.1)	0.846
Unwitnessed	2 (1.6)	1 (0.8)	2 (1.6)	0.814

Table 4 Clinical outcomes of AMI patients who occurred CA stratified according to TyG tertiles

Clinical outcomes	T1 (n = 124)	T2 (n = 124)	T3 (n = 123)	*P* value
Primiary outcomes, n (%)				
In-hospital mortality	24 (19.4)	29 (23.4)	45 (36.6)	0.006
ICU mortality	19 (15.3)	22 (17.7)	39 (31.7)	0.003
Secondary outcomes, days				
LOS-H	5.2 (2.9–10.9)	5.1 (3.2–13.8)	7.8 (4.6–18.8)	0.002
LOS-ICU	2.8 (1.6–6.7)	3.0 (1.9–6.3)	5.1 (2.6–9.1)	< 0.001

AMI acute myocardial infarction; *CA* cardiac arrest; *TyG* triglyceride-glucose; *T* tertiles; *ICU* intensive care unit; *LOS-H* length of stay in hospital; *LOS-ICU* length of stay in intensive care unit

Correlations of the TyG index with clinical endpoints among AMI-CA populations

To further assess the association of the TyG index with mortalities of AMI-CA populations, logistic analysis was conducted (Table 5). Univariate logistic analysis revealed an apparent link of mortality with TyG when examined as a continuous variable, with an OR of 1.680 regarding in-hospital mortality (95% CI 1.226–2.303) and an OR of 1.481 regarding ICU mortality (95% CI 1.064–2.061). Moreover, our data showed individuals in the highest TyG tertile exhibited a 2.404- and 1.844-fold increased risk of in-hospital (95% CI 1.350–4.281) and ICU mortality (95% CI 1.007–3.376), respectively, compared to those in the lowest tertile. After controlling for influential factors, these relationships remained statistically significant in both the partially adjusted (OR_{HOS}: 1.914, OR_{ICU}: 1.682) and fully adjusted (OR_{HOS}: 1.711, OR_{ICU}: 1.520) models when analysing the TyG index as continuous variable. Consistent results were obtained when TyG analysed as categorical variable (T3 *vs.* T1: Model 2: OR_{HOS}: 2.927, OR_{ICU}: 2.221; Model 3: OR_{HOS}: 2.728, OR_{ICU}: 2.133), and a robust tendency for mortality risk to increase with elevated TyG tertiles was obtained (all *P* for trend < 0.05).

Additionally, we performed correlation analyses, revealing apparent links of TyG levels with certain variables such as LOS-H, LOS-ICU, WBC, CS, and APS

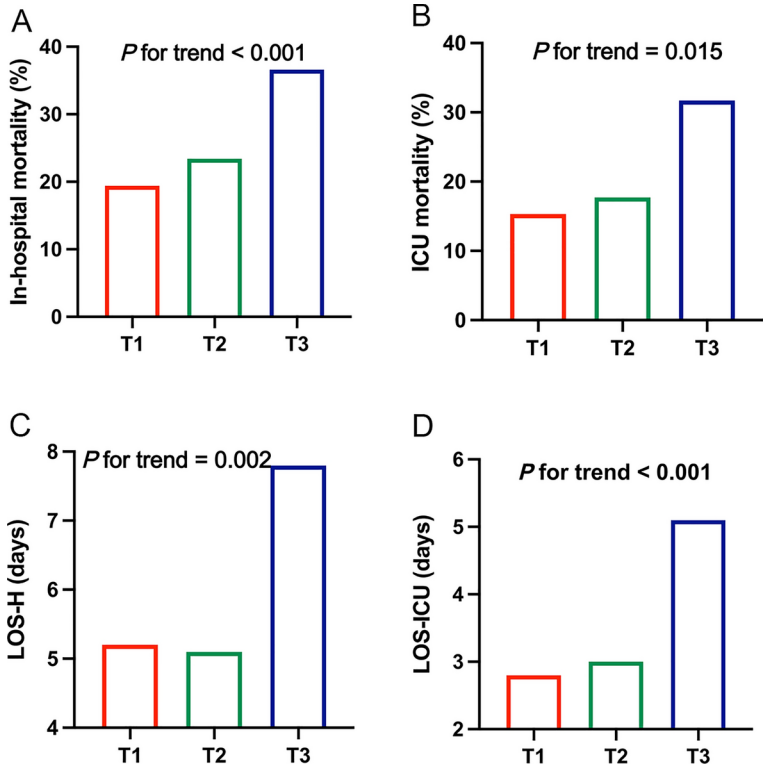

Fig. 3 Endpoints stratified by tertiles of the TyG index in the AMI-CA patient population. **A** in-hospital mortality; **B** ICU mortality; **C** LOS-H; **D** LOS-ICU. ICU, intensive unit care; LOS-H, length of stay in hospital; LOS-ICU, length of stay in intensive unit care; T, tertile

Table 5 Logistic regression analyses for the correlation between TyG index and mortality in AMI populations post-CA

Variables	Model 1		Model 2		Model 3	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
In-hospital mortality						
Per 1 Unit increase	1.680 (1.226-2.303)	0.001	1.914 (1.370-2.673)	< 0.001	1.711 (1.152-2.540)	0.008
Tertile 1	1 (Reference)	–	1 (Reference)	–	1 (Reference)	–
Tertile 2	1.272 (0.692–2.340)	0.439	1.393 (0.744–2.607)	0.300	1.368 (0.664–2.819)	0.396
Tertile 3	2.404 (1.350–4.281)	0.003	2.927 (1.593–5.377)	< 0.001	2.728 (1.303–5.711)	0.008
P for trend	0.003	–	< 0.001	–	0.008	–
ICU mortality						
Per 1 Unit increase	1.481 (1.064–2.061)	0.020	1.682 (1.188–2.382)	0.003	1.520 (1.004–2.301)	0.048
Tertile 1	1 (Reference)	–	1 (Reference)	–	1 (Reference)	–
Tertile 2	1.056 (0.553–2.014)	0.869	1.170 (0.602–2.277)	0.643	1.122 (0.523–2.406)	0.768
Tertile 3	1.844 (1.007–3.376)	0.047	2.221 (1.175–4.197)	0.014	2.133 (1.010–4.506)	0.047
P for trend	0.047	–	0.014	–	0.033	–

TyG triglyceride-glucose; *AMI* acute myocardial infarction; *CA* cardiac arrest; *OR* odds ratio; *CI* confidence interval; *ICU* intensive care unit.

Model 1: unadjusted.

Model 2: Adjusted for age, sex, and body mass index.

Model 3: Adjusted for age, sex, body mass index, heart rate, mean arterial pressure, diabetes mellitus, previous myocardial infarction, cardiogenic shock, chronic heart failure, malignant ventricular arrhythmia, unwitnessed, bystander cardiopulmonary resuscitation > 15min, creatine kinase-myocardial band, alanine transaminase, aspartate transaminase, creatinine, lactate, haemoglobin, blood urea nitrogen, percutaneous coronary intervention, coronary artery bypass grafting, white blood cell, red blood cell, high density lipoprotein cholesterol, oral anticoagulants, antiplatelet drugs, vasoactive agents, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, β-blockers, and statins.

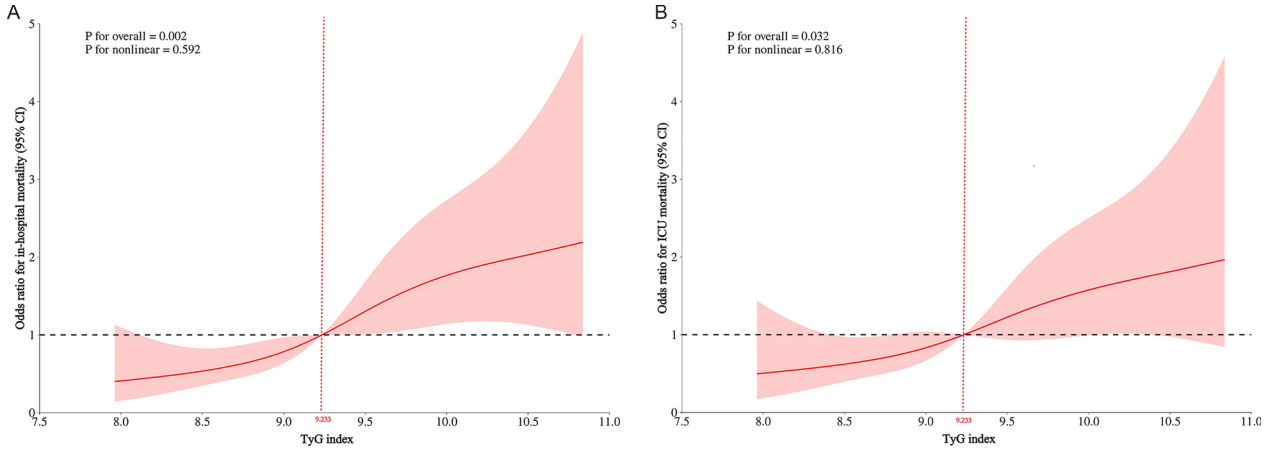

Fig. 4 RCS curves of the TyG index in relation to mortality in the AMI-CA patient population. **A** in-hospital mortality; **B** ICU mortality. ICU, intensive unit care; TyG, triglyceride-glucose; CI, confidence interval

score (all $P<0.05$); however, no obvious relationships were found with HDL-C, LDL-C, TC, CK-MB, MVA or GCS score (Table S5). Meanwhile, the results of multiple linear analyses showed a significant association between TyG and LOS-ICU ($\beta=1.051$, $P=0.031$) as well as APS score ($\beta=11.325$, $P<0.001$), while also indicating a near-significant correlation with LOS-H ($\beta=1.617$, $P=0.065$) (Table S6).

Collectively, these findings demonstrated that TyG was obviously related to unfavorable prognoses among AMI-CA populations, establishing it as an independent risk indicator, particularly in relation to mortality and LOS outcomes.

AMI-CA patients with higher TyG index demonstrated increased risk of mortality and prolonged length of stay

After controlling for age, sex, and BMI, the RCS curves showed significant linear links of elevated TyG levels (> 9.233) with heightened risks of in-hospital (*P* for nonlinearity = 0.592) and ICU mortalities (*P* for nonlinearity = 0.816), as shown in Fig. 4.

AMI-CA patients were stratified in accordance with the TyG threshold of 9.233, as determined by RCS, and several dissimilarities regarding general characteristics between groups were observed, as summarized in Table S7. To address potential confounding factors, we conducted additional PSM, IPTW, and OW analyses. Baseline characteristics of enrolled population following 1:1 PSM have been presented in Table S8, wherein each arm

comprised 136 individuals and nearly all potential confounders were effectively balanced. Moreover, the general characteristics of the IPTW- and OW-adjusted cohorts were elaborated in Table S9, with a substantial balance across most confounding variables.

As shown in Table S10, among the original cohort that categorized into higher and lower TyG groups, those suffering from AMI-CA with higher TyG exhibited an elevated risk for in-hospital (19.5% vs. 33.3%) and ICU (17.3% vs. 25.8%) mortality, along with significantly longer LOS-H (5.1 days vs. 7.3 days) and LOS-ICU (2.8 days vs. 4.9 days). Elevated mortality rates remained significantly associated with increasing TyG levels in the PSM- (HOS: 11.8% vs. 28.7%, ICU: 10.3% vs. 22.1%), IPTW- (HOS: 25.9% vs. 36.3%, ICU: 22.0% vs. 30.5%) and OW-adjusted (HOS: 17.3% vs. 36.6%, ICU: 15.2% vs. 28.0%) cohorts. Interestingly, the results regarding LOSs also exhibited consistency. In detail, both the cohorts adjusted using IPTW (HOS: 9.6 days vs. 13.4 days, ICU: 5.1 days vs. 7.6 days) and OW (HOS: 3.1 days vs. 4.4 days, ICU: 1.7 days vs. 2.8 days) showed significantly prolonged LOS-H and LOS-ICU among patients with higher TyG index; similar findings were observed for LOS-H (5.2 days *vs.* 6.7 days) and LOS-ICU (2.8 days *vs.* 4.3 days) following PSM. Additionally, logistic regression analyses were conducted to investigate the association between elevated TyG and in-hospital and ICU mortalities in the original cohort as well as cohorts adjusted using PSM, OW, and IPTW methods, revealing significant correlations with all P values being less than 0.05 (Fig. 5).

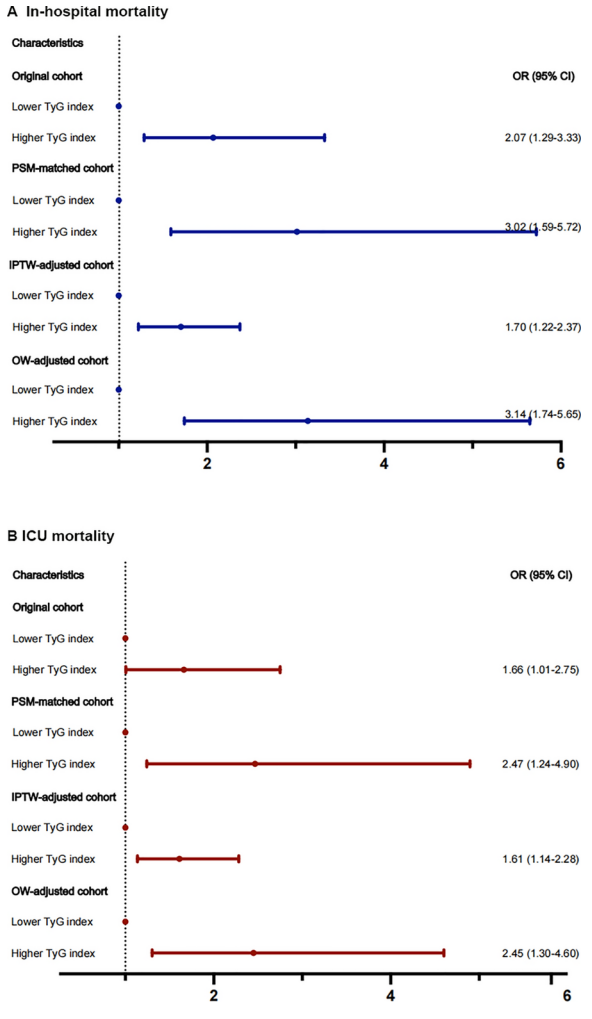

Fig. 5 Logistic regression model to explore mortality risks associated with higher TyG index in the original, PSM-, OW-, and IPTW-adjusted cohorts. **A** in-hospital mortality; **B** ICU mortality. TyG, triglyceride-glucose; ICU, intensive unit care; PSM, propensity score matching; IPTW, inverse probability of treatment weighting; OW, overlap weighting; OR, odds ratio; CI, confidence interval

Collectively, these findings suggested that higher TyG was positively correlated with increased mortalities and prolonged LOSs in AMI-CA patients, even after controlling for various statistical methods.

Subgroup analysis

We further estimated the prognostic significance of TyG for mortality endpoints among different subgroups of our study population stratified based on age, sex, BMI, and hypertensive status (Fig. 6). Our findings revealed significant associations of elevated TyG with higher risks of in-hospital and ICU mortality in AMI patients occurring CA, particularly among elders, females, those BMI < 28 kg/m^2, and those with hypertension. Notably, the TyG index appeared a more pronounced association with ICU mortality risks in female patients as opposed to males. Moreover, no interactions were obtained of the TyG with other stratified factors.

Discussion

In this multi-center, observational cohort comprising 5208 patients diagnosed with AMI, individuals presenting with CA exhibited significantly higher TyG levels compared to those without CA. The TyG index demonstrated a moderate capacity for identifying patients at risk of occurring CA within entire AMI populations. Furthermore, increasing TyG levels were found to be closely linked with higher risks of in-hospital and ICU mortality regarding AMI-CA, even after rigorous adjustment for potential confounding factors and utilization of multiple statistical approaches. Importantly, an apparent relationship of TyG with LOS was also observed. On the whole, our study underscores a previously unrecognized relevance of TyG in relation to AMI-CA, especially concerning its prognostic implications for survival and LOS, thereby augmenting understanding of its role in such critically ill cardiovascular conditions.

The consequences of this indicator within the cardiovascular sphere have elicited pronounced attention in recent years, ascribable to its facile acquisition and

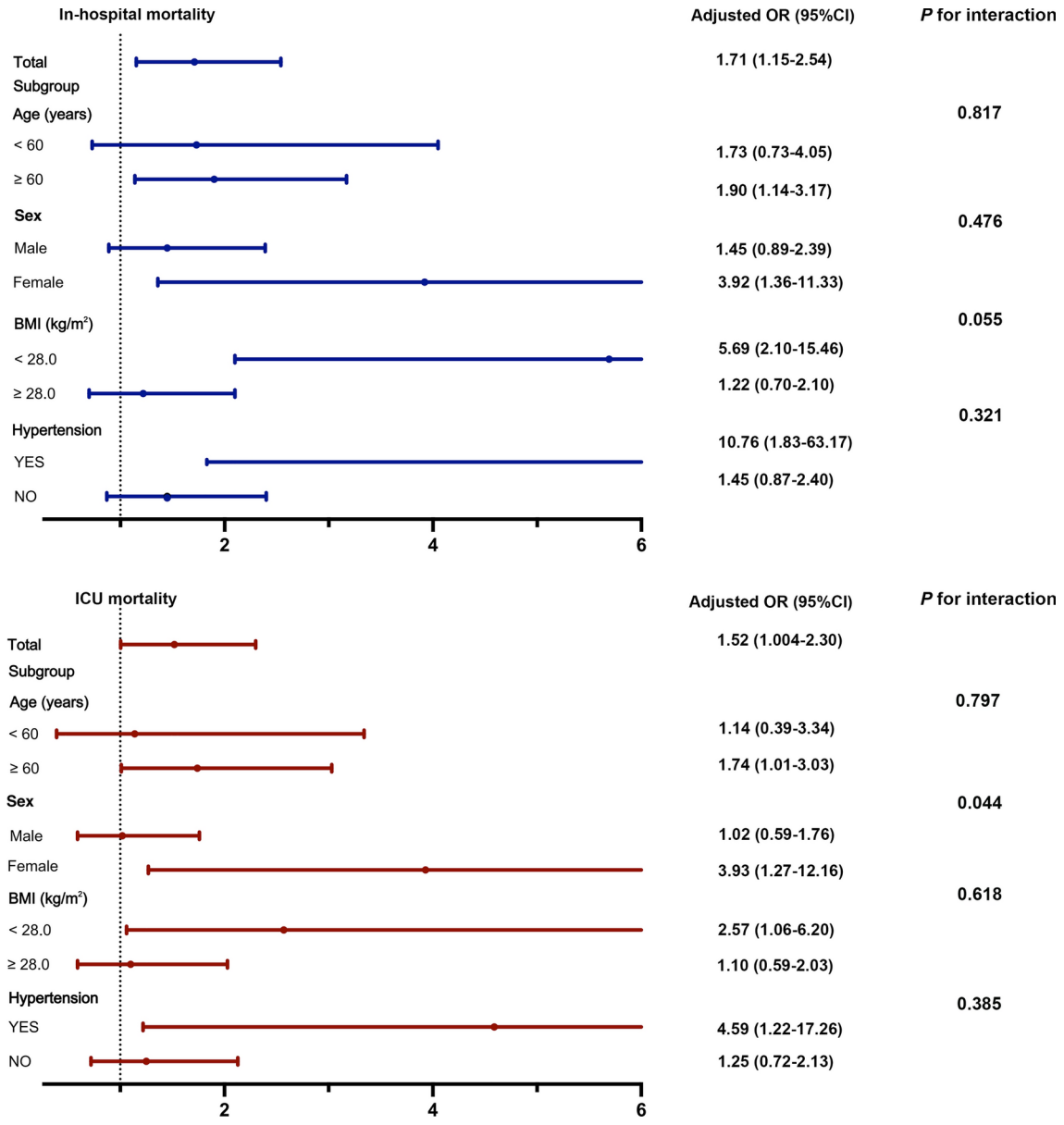

Fig. 6 Subgroup analyses for the correlation between the TyG index and mortalities, based on age, sex, BMI, and hypertension. BMI, body mass index; ICU, intensive care unit; OR, adds ratio; CI, confidence interval

trustworthy manifestation of IR, even eclipsing certain conventional testing methodologies such as the insulin clamping technique and the homeostasis model assessments of IR [36]. As recently subjected to meta-analyses, TyG has been established as a cogent marker for assessing adverse prognoses of coronary artery disorders [37–39]. From the pathological perspective, the underlying mechanisms of IR involving in coronary disorders are multifaceted, encompassing the induction of a pro-inflammatory state, augmentation of oxidative stress, disruption of endothelial function, perturbation of myocardial metabolism, disturbance in the cardiac autonomic system, and so forth [40–43], all of which may be exacerbated in the setting of acute critical illness such as AMI, leading to a cascade of events that culminate in increased mortality or comorbidity. Just as the theory goes, the TyG index, serving as an indicator of IR, has showed great relationships with number of coronary lesions [44], development of coronary artery stenosis or calcification [44, 45], intracoronary thrombus burden [46], as well regarding chronic total occlusion [46]. Moreover, the correlation between TyG and multiple cardiovascular risk elements such as metabolic syndrome [47], DM [48], CKD [49], hypertension [50], depression [51], or obstructive sleep apnea has already been identified [52]. Accordingly, it is neither complicated nor difficult to elucidate that TyG

possesses significant diagnostic and prognostic value in relation to several cardiovascular circumstances, likewise including its predictive capacity for AMI-CA as investigated by our cohort. Furthermore, elevated TyG levels have been confirmed to correlate with adverse outcomes in both general and surgical ICU cohorts [53, 54], some of whom may exhibit overlap with the specific critically cardiac patients focused on in our study.

Out-of-hospital sudden cardiac death constitutes around 50% of all cardiac fatalities [55], and CA plus cardiogenic shock even contributes to 60% to 80% of short-term mortality in AMI populations [56, 57]. Despite a noticeable decline regarding mortality has been reported over the past decades, CA continues to pose a seriously severe situation with poor survival outcomes, as evidenced by an in-hospital mortality of 66.4% in 2014 in England and a 30-day mortality of 98.8% in 2020 in China [58, 59]. As anticipated, in our study, individuals with AMI occurring CA likewise exhibited notably high mortality rates, with in-hospital mortality reaching nearly 26.4% and ICU mortality approaching approximately 21.6%. Even worse, roughly 50% of cardiovascular cases manifested CA as their initial symptom, with survival rates falling below 20% [60]. That is to say, CA always presents a life-threatening yet difficult-to-predict health issue. Several credible predicators have been identified for CA occurrences following AMI [10], such as advanced age, female, nonsmoking, prior DM, and prior renal failure; however, these factors alone are not sufficiently robust to accurately assess individual patient CA risk due to the relatively low incidences [61]. Herein, we found that TyG was remarkably higher in patients who developed CA compared to individuals who did not among AMI populations. Moreover, this index exhibited superior discriminatory power in identifying individuals presenting with CA from the overall AMI population in comparison to several established risk factors, including age, sex, BMI, previous MI, DM, CKD, malignant arrhythmia, etc. These findings suggested that TyG might serve as a novel indicator correlated with the occurrence of CA while requires prospective validation among AMI cohorts.

Several well-established scoring systems, including those widely utilized for stratifying the general ICU population such as the Sequential Organ Failure Assessment (SOFA) score, Acute Physiology and Chronic Health Evaluation (APACHE)-III and APACHE-IV scores, as well as those specifically designed to assess the prognoses of individuals post-CA such as the Post Arrest Triage score and the Cardiac Arrest Hospital Prognostic score, are frequently employed in clinical practice for evaluating the severity of the AMI-CA. Although not directly incorporating glucose metabolism-related variables, these systems encompass events such as initial rhythm [62], CHF [63], CKD [49], and GCS scores [35], that have been reported to exhibit correlations with elevated TyG levels. However, the practical application of the aforementioned scores continues to pose challenges in real-world settings due to the necessity of gathering a multitude of physiological parameters. These systems exhibited reliable predictive capacity based on existing risk indicators such as age, sex, renal insufficiency, initial rhythm, bystander cardiopulmonary resuscitation, unwitnessed status, utilization of epinephrine, out-of-hospital CA, restoration of spontaneous circulation time, etc. In the current analysis, we integrated as numerous above potential affecting factors as possible, thereby additionally enhancing the credibility of these findings. Interestingly, within our cohort, we observed a significant positive link of the TyG with APS score, which is an integral component of APACHE used for assessing acute physiological abnormalities. Notably, IR can excessively activate the sympathetic nervous system [64]. What's more, researchers have previously substantiated a discernible correlation of the TyG with prolonged QT intervals in AMI patients which is associated with an increased risk of malignant dysrhythmia [62, 65], the primary prelude of CA, thus partially clarifying the relationship between the TyG index and unfavorable outcomes in patients with AMI-CA. Regarding LOS-H and LOS-ICU, a clear increasing trend was observed with higher tertiles of TyG in the current study, and there was an apparent difference for LOS between the high and low TyG groups, even following balancing via PSM, OW, and IPTW. Hence, we currently consider TyG index as a predictor for LOS, whereas further relevant studies are still needed. Moreover, we did not observe any brain injury in relation to the elevated TyG index, which was inconsistent with previous findings in general post-CA populations that uncovered a strong correlation between increased GCS scores and higher TyG levels [35]. One explanation was that AMI could result in more pronounced ischemia–reperfusion injury, potentially exerting a more direct and severe impact on the brain, thereby obscuring the effects of glucose metabolism disturbance. Taken together, we proposed that individuals with elevated TyG presenting with AMI-CA were likely to encounter more severe clinical conditions.

To our knowledge, the present study represents the pioneering effort to evaluate the association of TyG with CA occurrences and their prognoses among populations with AMI. It synthesizes records from two extensive cohorts comprising data sourced from 209 medical centers, providing a fresh insight into the significance of this indicator in critically ill cardiovascular conditions. Potential confounders were carefully considered, and various statistical methods were employed to adjust or match the cohorts in order to enhance the reliability of our findings. Given the strong association observed between

the TyG index and the risks regarding AMI-CA, implementing dietary interventions or adjusting therapeutic strategies to mitigate this index might potentially contribute to improved prognoses for this specific critically ill coronary population. In contrast to standalone FBG or TG metrics, the TyG index is capable of simultaneously integrating information pertaining to glycolipid metabolism, providing a more comprehensive evaluation of IR, which is a critical factor in the progression of acute coronary disorders. More importantly, although FBG is better at identifying individuals with CA from the overall AMI population compared to TyG, while FBG or TG alone cannot afford to predict mortality outcomes in those occurring AMI-CA, as no consistent conclusions have been drawn regarding the association between elevated TG or FBG levels and mortalities or LOSs (Table S11 and Figure S1). Additionally, since both FBG and TG are routinely measured in AMI patients, the derived TyG index can provide broader predictive value regarding risks of CA following AMI without requiring additional complex tests, highlighting its clinical advantages.

However, several limitations remain. Firstly, the retrospective design restricts the external validity of our findings, necessitating further validation through prospective cohort studies in the AMI-CA patient population. Additionally, interventional trials are essential to establish the causality between elevated TyG and specific events. Secondly, despite our extensive efforts to control for confounders, several variables were not included in our analysis, such as the restoration of spontaneous circulation, the location of CA, and the rapid response team, which have been previously validated as significant predictors for prognoses in these populations [66, 67]; for instance, patients with in-hospital CA are likely to have more comorbidities but lower mortality rates as a result of timely and professional medical interventions compared to out-of-hospital cases [68, 69], which can lead to certain selection biases. Moreover, integrating pre-illness TyG values with disease-onset TyG could potentially enhance the clinical significance of our findings; however, the retrospective nature of our analysis utilizing public databases poses challenges in obtaining these measurements prior to admission. Thirdly, this study predominantly relies on historical data from the period of 2008–2019, which may introduce minor discrepancies with current clinical practices and trends in disease progression, given that the treatment strategies and medical techniques for CA have evolved significantly in recent years. Finally, a positive relationship has been reported between elevated TyG and increased long-term major adverse cardiovascular outcomes following ST-elevation myocardial infarction [70], hence, additional studies are warranted to verify the potential correlation of TyG with long-term prognoses among these specific critically ill cardiovascular patients focused by our study.

Conclusion

An elevated TyG index demonstrates to correlate with higher prevalence and unfavourable prognosis regarding CA following AMI. Individuals with AMI-CA who have elevated TyG levels exhibit increased mortality risks and prolonged LOS. These findings reveal the previously unrecognized value of TyG as a significant risk indicator for AMI-CA. Further comprehensive research remains essential to ascertain whether identifying or intervening on elevated TyG index can yield enhanced clinical outcomes in these critically ill coronary populations.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12933-025-02641-8.

Supplementary Material 1.

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None.

Author contributions
HRL and LSW contributed to drafting manuscript and creating figures and tables. XH and ZTD contributed to data curation. CLL and HW contributed to review of all revisions. XTH, LSW and ZTD provided resources and performed the statistical analysis. All authors have read and approved the final manuscript.

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

Declarations

Ethics and consent to participate
Not applicable.

Consent to Publish
Not applicable.

Conflict of interests
The authors declare no competing interests.

Author details
[1]Centre for Cardiac Intensive Care, Beijing Institute of Heart, Lung and Blood Vessel Diseases, Beijing Anzhen Hospital, Capital Medical University, Beijing 100029, People's Republic of China

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