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Associations of the triglyceride-glucose index with short-term mortality in patients with cardiogenic shock: a cohort study

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Abstract

Background Cardiogenic shock (CS) is a severe cardiac disorder with a high mortality rate. The triglyceride-glucose (TyG) index, a biomarker of insulin resistance, is associated with cardiovascular disease-related mortality. This study aimed to investigate the association between the TyG index and mortality in patients with CS.

Methods This retrospective cohort study analyzed 727 patients with CS from the Medical Information Mart for Intensive Care IV database. The TyG index was calculated as follows: ln[triglycerides (mg/dL) × fasting blood glucose (mg/dL)/2]. Outcomes included 28-day intensive care unit (ICU) mortality and 28-day in-hospital mortality. Kaplan-Meier survival curve models and Cox proportional hazards regression models were used to evaluate the prognostic significance of the TyG index. Receiver Operating Characteristic (ROC) curve analysis was used to determine the predictive efficacy of the TyG index for mortality. Subgroup analyses were conducted to determine the association between the TyG index and mortality across different groups.

Results Non-survivors had a significantly higher TyG index (ICU: 9.30 vs. 9.13, $p=0.008$; in-hospital: 9.29 vs. 9.13, $p=0.004$). Adjusted Cox models showed that each 1-unit increase in the TyG index increased ICU mortality risk by 24% (hazard ratio [HR] = 1.24, 95% confidence interval [CI]:1.04–1.48; $p=0.015$) and in-hospital mortality by 44% (HR = 1.44, 95% CI:1.11–1.88; $p=0.007$). The Quartile 4 TyG index ICU mortality was increased by 77% (HR = 1.77, 95% CI:1.09–2.89) compared to that for Quartile 1 and in-hospital mortality was increased by 61% (HR = 1.61, 95% CI:1.08–2.38). The area under the ROC curve (AUROC) showed a modest standalone predictive ability of 0.56, but when combined with clinical variables, the AUROC improved to 0.80 (ICU) and 0.78 (in-hospital). Subgroup analyses identified stronger associations in patients ≥ 60 years, females, non-septic, and those with acute myocardial infarction or heart failure.

Conclusions The TyG index is significantly associated with short-term mortality in patients with CS and may serve as a useful biomarker for risk stratification.

Trial registration Not applicable.

Keywords Triglyceride-Glucose Index, Short-term Mortality, Cardiogenic Shock

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Introduction

Cardiogenic shock (CS) is a complex syndrome characterized in clinical practice as a cardiac disorder that results in clinical and biochemical evidence of sustained tissue hypoperfusion [1]. Recent studies have indicated that, although the prognosis for CS has improved relative to previous years, the in-hospital mortality rate remains approximately 30–40% [2]. Among patients with CS, 40% had mixed CS, characterized by heart failure (HF) and acute coronary syndrome (ACS), whereas 35% had HF alone and 17% had ACS alone [3]. Considering the high mortality rate of CS and its highly heterogeneous etiology, it is imperative to further investigate simple and validated bioindicators associated with the risk of CS-related mortality.

Insulin resistance (IR) refers to the diminished responsiveness of tissues to insulin, leading to an impaired capacity of insulin to facilitate cellular glucose uptake or inhibit hepatic glucose production [4]. IR is widely recognized as a contributor to metabolic disorders, including hypertension, hyperlipidemia, and hyperglycemia [5]. Moreover, hyperinsulinemia resulting from IR can lead to vascular injury and coronary atherosclerosis [6]. These factors collectively contribute to the initiation and progression of cardiovascular diseases (CVD), including HF and coronary heart disease (CHD) [7, 8]. The triglyceride-glucose (TyG) index, which can rapdily and effectively assess IR, is increasingly used [9, 10]. This assessment contrasts with the Homeostasis Model Assessment of IR (HOMA-IR), which depends on the measurement of insulin, yet still has high sensitivity and specificity for assessing IR [11–13]. A growing number of studies have indicated that the TyG index is strongly associated with mortality risk from CVD, including HF and CHD [14, 15]. Currently, only one study has reported that an elevated TyG index is associated with the incidence of CS following acute myocardial infarction (AMICS) and a higher mortality risk [16]. However, whether the TyG index is a effective predictor of short-term mortality in all patients with CS remains unclear.

Consequently, we conducted a study using the Medical Information Mart for Intensive Care IV (MIMIC-IV) database to investigate the association between the TyG index and the short-term prognosis of patients with CS. This study will provide insights into the metabolic direction of intervention strategies for patients with CS.

Methods

Study cohort

This study used data from the MIMIC-IV database (version 3.0), a well-known publicly available resource managed by the Computational Physiology Laboratory at the Massachusetts Institute of Technology. The database includes patient information collected from 2008 to 2022 of hourly physiological measurements recorded by bedside monitors, which were validated by intensive care unit (ICU) nursing staff. It features a diverse cohort of ICU patients and provides extensive details on demographic factors, medical history, laboratory test results, and prescribed medications (https://doi.org/10.13026/kpb9-mt58). Access to the database was granted upon successfu l completion of the National Institutes of Health online course titled "Protecting Human Research Participants", which ensures ethical standards in research involving humans. Specifically, access was awarded to individuals who completed the Collaborative Institutional Training Initiative examination, including the author, Mo (Certification number: 65748833). In accordance with the International Classification of Diseases (ICD), 9th and 10th revisions, this study enrolled patients diagnosed with CS (ICD codes 4019 and E785). The exclusion criteria were: (1) patients < 18 years (2), patients with an ICU stay of < 24 h (3), patients with missing triglyceride data, and (4) patients with missing glucose data. As a result, 727 patients were included in this study (Fig. 1).

Data collection

PostgreSQL software (version 13.7.2) was used to extract information. The demographic characteristics included age, sex, and ethnicity. Anthropometric characteristic: body mass index (BMI). The types of admissions to the ICU included the Coronary Care Unit (CCU), Cardiovascular ICU, Medical ICU, and other specialized units. Vital signs, including temperature, heart rate, respiratory rate, systolic blood pressure (SBP), diastolic blood pressure, mean blood pressure, and peripheral capillary oxygen saturation (SpO_2) were recorded as the initial measurements at the time of admission. Comorbidities including type 2 diabetes mellitus (T2DM), hypertension, HF, acute myocardial infarction (AMI), cancer, chronic kidney disease (CKD), cirrhosis, pneumonia, stroke, hyperlipidemia, chronic obstructive pulmonary disease (COPD), acute renal failure (ARF), and sepsis were extracted from the database based on diagnostic codes. Scoring systems, including the Sequential Organ Failure Assessment (SOFA), the Simplified Acute Physiology Score II (SAPS II), and the Oxford Acute Severity of Illness Score (OASIS), were applied within 24 h of patient admission to the ICU. Laboratory blood indicators included white blood cell (WBC) count, red blood cell count, platelet (PLT) count, hemoglobin (Hb) level, blood urea nitrogen (BUN) level, creatinine level, glucose level, triglycerides (TG) level, and the triglyceride-glucose (TyG) index. All hematological indices were obtained during the first assessment following patient admission and adhered to clinical laboratory standards. Treatments included whether patients had continuous

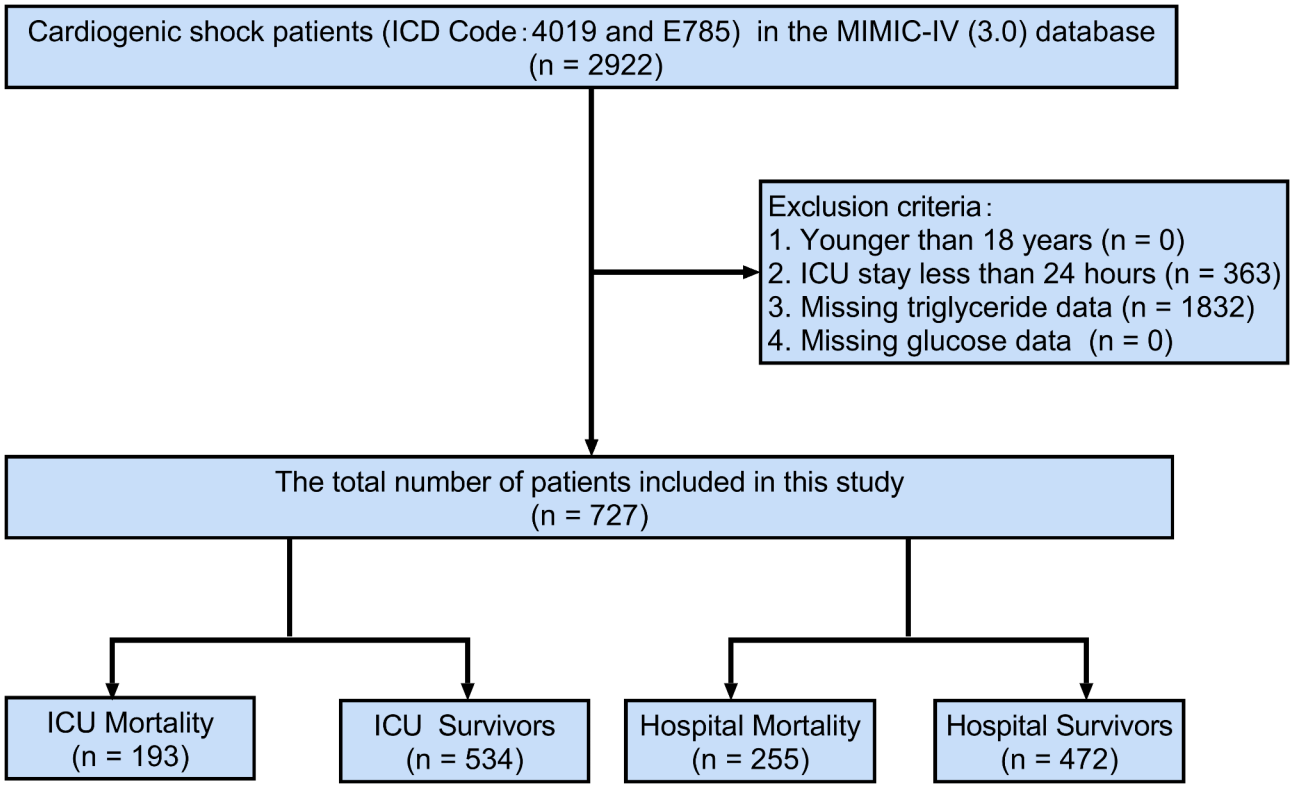

Fig. 1 Study flowchart

renal replacement therapy (CRRT) or mechanical ventilation during hospitalization.

TyG index calculation

The formula for calculating the TyG index is: TyG index = ln (TG (mg/dL) × fasting blood glucose (mg/dL) / 2) [17].

Outcomes

The outcomes of patients with CS in the MIMIC-IV database included the 28-day all-cause in-hospital mortality and 28-day all-cause mortality in the ICU.

Statistical analysis

Statistical analyses were performed using R software (version 4.3.0, Austria) and GraphPad Prism (version 9.5.1, USA). The Shapiro-Wilk test was used to assess the normality of the data. Continuous variables were identified as not normally distributed; therefore, they are presented as median (Quartile 1 - Quartile 3). The Mann-Whitney U test was used for comparisons between two groups of continuous variables, while the Kruskal-Wallis H test was used for comparisons among multiple groups. Categorical variables are expressed as n (%) and analyzed using the chi-square test or Fisher's exact test. Missing values were addressed using multiple imputation, specifically employing the "mice" package. Variables with excessive missing values (> 30%) were excluded from the analysis [18]. Statistical significance was defined as a two-sided p-value < 0.05.

Box plots illustrate the differences in the TyG index between the deceased and survivor groups of patients with CS. Patients were categorized into four groups (Quartile 1-Quartile 4) based on the quartiles of the TyG index. Kaplan-Meier survival curves were generated for each TyG quartile, and differences in probabilities of survival between groups were assessed using the log-rank test. Subsequently, Cox proportional hazard regression models were used to determine the prognostic value of the TyG index. The TyG index was analyzed as a continuous variable (per 1-unit increase) and as a categorical variable (quartile groups). Three Cox regression models were used to analyze the data. Model 1 was an unadjusted model. Model 2 was adjusted for age, sex, and ethnicity. Model 3 was further adjusted for heart rate, SpO_2, hypertension, HF, CKD, cirrhosis, COPD, ARF, sepsis, PLT count, Hb level, BUN level, creatinine level, CRRT, and ventilation status. To address potential multicollinearity among covariates, a bidirectional stepwise regression approach was applied using the Akaike Information Criterion for variable selection. Hazard ratios (HRs) with 95% confidence intervals (CIs) were reported. Receiver Operating Characteristic (ROC) curve analysis was used to assess the predictive capability of the TyG index for

28-day ICU mortality and 28-day in-hospital mortality in patients with CS. To further investigate the applicability of the TyG index across different patient subgroups, subgroup analyses were conducted based on age (< 60 years vs. ≥60 years), sex, race, BMI (< 30 kg/m² vs. ≥30 kg/m²), AMI, HF, and sepsis. In the subgroup analysis, the TyG index was categorized into a binary variable based on the optimal cutoff value from the ROC curve analysis. The variables adjusted in the subgroup analysis were the same as those in the Cox regression Model 3; however, the variables included in the subgroup stratification were excluded.

Results

Patient characteristics

The baseline characteristics of the 727 patients were stratified by the quartiles of the TyG index (Table 1). The median age of the study cohort was 67 years, predominantly male (63.14%), and mostly white ethnicity (56.95%). The distribution of the ICU types revealed that patients in the CCU accounted for the highest percentage. BMI was significantly different across quartiles. Vital signs revealed significant differences in temperature and SBP with minor variations across the quartiles. Comorbidities such as T2DM, HF, AMI, CKD, pneumonia, hyperlipidemia, and sepsis were significantly different across the quartiles (all $p < 0.05$). Notably, a higher percentage of patients with T2DM, pneumonia, and sepsis was observed in the higher quartiles. All scoring systems (SOFA, SAPS II, and OASIS) indicated worse conditions in the higher quartiles. Laboratory indicators, such as WBC count, PLT count, BUN level, glucose level, TG level, and the TyG index had significant variations across quartiles. Notably, the TyG index increased with increasing quartiles. Treatment interventions, including CRRT and mechanical ventilation, also differed significantly among the quartiles, with higher use in the higher quartiles.

Differences in the TyG index between deceased and surviving patients

Figure 2 illustrates the distribution of the TyG index among patients with CS who survived and those who died during the 28-day ICU period and the 28-day in-hospital period. For patients who died during the 28-day ICU period, the median TyG index was 9.30 (8.84, 9.89), which was significantly higher than that of the surviving patients; 9.13 (8.65, 9.66) ($p = 0.008$). Similarly, among patients who died during the 28-day in-hospital period, the TyG index was 9.29 (8.82, 9.89), which was significantly higher than that of the surviving patients; 9.13 (8.65, 9.63) ($p = 0.004$).

Associations of the TyG index with 28-Day ICU mortality and in-hospital mortality

Kaplan-Meier survival curves (Fig. 3) show that a higher TyG index was associated with a lower 28-day ICU probability of survival (log-rank test: chi-square = 10.8322, $p = 0.012$) and lower 28-day in-hospital probability of survival (chi-square = 8.2280, $p = 0.041$).

Cox regression models revealed a significant association between a high TyG index and 28-day ICU mortality and 28-day in-hospital mortality (Table 2).

Unadjusted model (Model 1): For each 1-unit increase in the TyG index, the risk of ICU mortality increased by 27% (HR = 1.27, 95% CI: 1.08–1.49, $p = 0.003$). Compared to Quartile 1, the risk of mortality in Quartiles 3 and 4 increased by 92% and 81%, respectively. For each 1-unit increase in the TyG index, the risk of in-hospital mortality increased by 25% (HR = 1.25, 95% CI: 1.08–1.44, $p = 0.002$), with Quartiles 3 and 4 showing increases in risk of 52% and 65%, respectively.

Adjusted for age, sex, and ethnicity (Model 2): For each 1-unit increase in the TyG index, the risk of mortality increased by 34% (HR = 1.34, 95% CI: 1.15–1.58, $p < 0.001$), with Quartiles 3 and 4 demonstrating increases in risk of 104% and 108%, respectively. Additionally, each 1-unit increase in the TyG index was associated with a 32% increase in the risk of in-hospital mortality (HR = 1.32, 95% CI: 1.15–1.53, $p < 0.001$), and Quartiles 3 and 4 showed increases in risk of 62% and 91%, respectively.

Further adjusted for multiple clinical variables (Model 3): for each 1-unit increase in the TyG index, the risk of mortality remained elevated by 24% (HR = 1.24, 95% CI: 1.04–1.48, $p = 0.015$). The risk of mortality in Quartile 3 increased by 100%, whereas that in Quartile 4 showed a 77% increase. For each 1-unit increase in the TyG index, the risk of in-hospital mortality increased by 44% (HR = 1.44, 95% CI: 1.11–1.88, $p = 0.007$) and that in Quartile 4 increased by 61% (HR = 1.61, 95% CI: 1.08–2.38, $p = 0.018$).

The predictive ability of the TyG index for 28-day ICU mortality and in-hospital mortality

Figure 4a illustrates the predictive ability of the TyG index as a singular indicator of 28-day ICU mortality and in-hospital mortality in patients with CS. The area under the ROC (AUROC) curve for the TyG index predicting 28-day ICU mortality was 0.56 (95% CI: 0.52–0.61), with an optimal cutoff value of 9.22, sensitivity of 0.55 (95% CI: 0.51–0.59), and specificity of 0.59 (95% CI: 0.52–0.66). Similarly, the AUROC curve for the TyG index predicting 28-day in-hospital mortality was also 0.56 (95% CI: 0.52–0.61), with the same optimal cutoff value of 9.22, sensitivity of 0.56 (95% CI: 0.51–0.60), and specificity of 0.56 (95% CI: 0.50–0.62).

Table 1 Baseline characteristics of individuals classified by quartiles of the TyG index

Characteristics	Total (*n* = 727)	Quartile 1 (*n* = 182)	Quartile 2 (*n* = 182)	Quartile 3 (*n* = 182)	Quartile 4 (*n* = 181)	*P*
Demographic characteristics						
Age, years	67.00 (57.00, 76.00)	69.00 (60.25,78.75)	67.50 (58.00,76.75)	67.00 (57.25,74.75)	64.00 (53.00,72.00)	< 0.001
Male, n (%)	459 (63.14)	113 (62.09)	112 (61.54)	113 (62.09)	121 (66.85)	0.695
Ethnicity, White, n (%)	414 (56.95)	106 (58.24)	108 (59.34)	99 (54.40)	101 (55.80)	0.770
Anthropometric Characteristic						
BMI, kg/m^2	28.51 (24.69, 32.88)	26.90 (23.78,30.82)	27.22 (23.84,31.48)	30.10 (24.70,35.25)	30.12 (27.00,35.34)	< 0.001
ICU types, n (%)						0.060
CCU	390 (53.65)	109 (59.89)	106 (58.24)	93 (51.10)	82 (45.30)	
CVICU	116 (15.96)	23 (12.64)	23 (12.64)	28 (15.38)	42 (23.20)	
MICU	124 (17.06)	25 (13.74)	30 (16.48)	33 (18.13)	36 (19.89)	
Others	97 (13.34)	25 (13.74)	23 (12.64)	28 (15.38)	21 (11.60)	
Vital signs						
Temperature, ℃	36.72 (36.44, 37.06)	36.64 (36.44,36.89)	36.67 (36.44,37.00)	36.78 (36.44,37.11)	36.78 (36.50,37.28)	0.002
Heart rate, bpm	92.00 (78.00, 108.50)	92.00 (75.00,106.00)	90.50 (78.00,108.75)	94.00 (79.00,111.75)	93.00 (80.00,108.00)	0.635
Respiratory rate, bpm	20.00 (16.00, 25.00)	20.00 (16.00,24.00)	20.00 (17.00,24.00)	21.00 (16.00,25.75)	20.00 (16.00,26.00)	0.924
SBP, mmHg	111.00 (96.00, 126.18)	107.00 (93.25,120.75)	110.50 (96.00,125.00)	114.00 (98.00,131.75)	111.00 (96.00,126.00)	0.035
DBP, mmHg	67.00 (56.00, 79.00)	67.00 (54.25,79.00)	67.00 (55.00,81.00)	69.00 (57.00,80.75)	67.00 (57.00,77.60)	0.829
MBP, mmHg	79.00 (68.00, 90.50)	78.00 (67.00,90.00)	78.00 (68.00,91.00)	80.00 (69.00,93.75)	78.78 (69.00,88.00)	0.576
SpO_2, %	97.00 (94.00, 100.00)	97.00 (94.00,99.00)	97.00 (93.00,100.00)	97.00 (94.00,100.00)	97.00 (93.00,100.00)	0.961
Comorbidity						
T2DM, n (%)	247 (33.98)	41 (22.53)	50 (27.47)	74 (40.66)	82 (45.30)	< 0.001
Hypertension, n (%)	167 (22.97)	39 (21.43)	35 (19.23)	42 (23.08)	51 (28.18)	0.216
Heart failure, n (%)	560 (77.03)	154 (84.62)	140 (76.92)	132 (72.53)	134 (74.03)	0.030
AMI, n (%)	287 (39.48)	56 (30.77)	81 (44.51)	73 (40.11)	77 (42.54)	0.038
Cancer, n (%)	72 (9.90)	16 (8.79)	23 (12.64)	16 (8.79)	17 (9.39)	0.556
CKD, n (%)	213 (29.30)	60 (32.97)	52 (28.57)	63 (34.62)	38 (20.99)	0.021
Cirrhosis, n (%)	31 (4.26)	12 (6.59)	10 (5.49)	3 (1.65)	6 (3.31)	0.088
Pneumonia, n (%)	289 (39.75)	56 (30.77)	74 (40.66)	77 (42.31)	82 (45.30)	0.029
Stroke, n (%)	57 (7.84)	21 (11.54)	14 (7.69)	8 (4.40)	14 (7.73)	0.092
Hyperlipidemia, n (%)	327 (44.98)	99 (54.40)	82 (45.05)	75 (41.21)	71 (39.23)	0.019
COPD, n (%)	80 (11.00)	17 (9.34)	24 (13.19)	23 (12.64)	16 (8.84)	0.430
ARF, n (%)	516 (70.98)	126 (69.23)	120 (65.93)	131 (71.98)	139 (76.80)	0.134
Sepsis, n (%)	592 (81.43)	128 (70.33)	134 (73.63)	165 (90.66)	165 (91.16)	< 0.001
Scoring System						
SOFA	7.00 (4.00, 10.00)	6.00 (4.00,9.00)	6.00 (4.00,10.00)	8.00 (5.00,10.75)	9.00 (6.00,12.00)	< 0.001
SAPA II	44.00 (33.00, 55.00)	41.00 (32.00,51.75)	43.00 (32.00,53.00)	45.00 (33.00,55.75)	48.00 (38.00,58.00)	0.001
OASIS	36.00 (30.00, 42.50)	34.00 (28.00,41.00)	35.00 (29.00,41.00)	37.00 (30.25,42.00)	38.00 (33.00,44.00)	< 0.001
Laboratory blood indicators						
WBC, K/uL	13.40 (9.55, 18.40)	10.98 (7.93,14.93)	13.70 (9.90,17.70)	13.70 (10.80,18.20)	15.50 (11.20,21.10)	< 0.001
RBC, m/uL	3.80 (3.17, 4.43)	3.63 (3.08,4.47)	3.78 (3.08,4.29)	3.89 (3.17,4.46)	3.85 (3.30,4.48)	0.322
PLT, K/uL	206.00 (145.00, 266.00)	178.50 (128.00,231.75)	225.50 (169.25,281.50)	215.50 (156.25,269.97)	206.00 (145.00,261.00)	< 0.001
Hb, g/dL	11.10 (9.30, 13.20)	10.70 (9.20,13.07)	11.30 (9.20,13.17)	11.10 (9.22,13.28)	11.30 (9.70,13.40)	0.321
BUN, mg/dL	28.00 (18.00, 45.00)	33.00 (19.25,50.00)	29.00 (18.00,45.50)	27.00 (18.00,42.75)	26.00 (18.00,38.00)	0.016
Creatinine, mg/dL	1.40 (1.00, 2.20)	1.50 (1.10,2.10)	1.30 (0.90,2.18)	1.40 (1.00,2.20)	1.50 (1.10,2.20)	0.411
Glucose, mg/dL	155.00 (118.00, 209.50)	113.50 (94.25,138.75)	146.00 (119.00,176.75)	170.50 (140.00,225.75)	249.00 (171.00,331.00)	< 0.001
TG, mg/dL	118.00 (83.00, 176.00)	75.00 (59.00,86.00)	105.50 (86.00,124.75)	141.00 (112.00,177.50)	235.00 (170.00,376.00)	< 0.001
TyG index	9.19 (8.70, 9.70)	8.39 (8.18,8.54)	8.95 (8.81,9.08)	9.40 (9.30,9.51)	10.24 (9.96,10.57)	< 0.001
Treatment						

Table 1 (continued)

Characteristics	Total (*n* = 727)	Quartile 1 (*n* = 182)	Quartile 2 (*n* = 182)	Quartile 3 (*n* = 182)	Quartile 4 (*n* = 181)	*P*
CRRT, n (%)	187 (25.72)	40 (21.98)	40 (21.98)	45 (24.73)	62 (34.25)	0.022
Ventilation, n (%)	667 (91.75)	160 (87.91)	160 (87.91)	174 (95.60)	173 (95.58)	0.003

Data are presented as Median (Quartile1, Quartile3) or n (%).

Abbreviations: TyG: Triglyceride-glucose; BMI: Body mass index; ICU: Intensive Care Unit; CCU: Coronary Care Unit; CVICU: Cardiovascular Intensive Care Unit; MICU: Medical Intensive Care Unit; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MBP: Mean Blood Pressure; SpO_2: Peripheral capillary oxygen saturation; T2DM: Type 2 diabetes mellitus; AMI: Acute myocardial infarction; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; ARF: Acute renal failure; SOFA: Sequential Organ Failure Assessment; SAPS II: Simplified Acute Physiology Score II; OASIS: Oxford Acute Severity of Illness Score; WBC: White Blood Cells; RBC: Red Blood Cells; PLT: Platelets; Hb: Hemoglobin; BUN: Blood Urea Nitrogen; TG: Triglyceride; CRRT: Continuous Renal Replacement Therapy

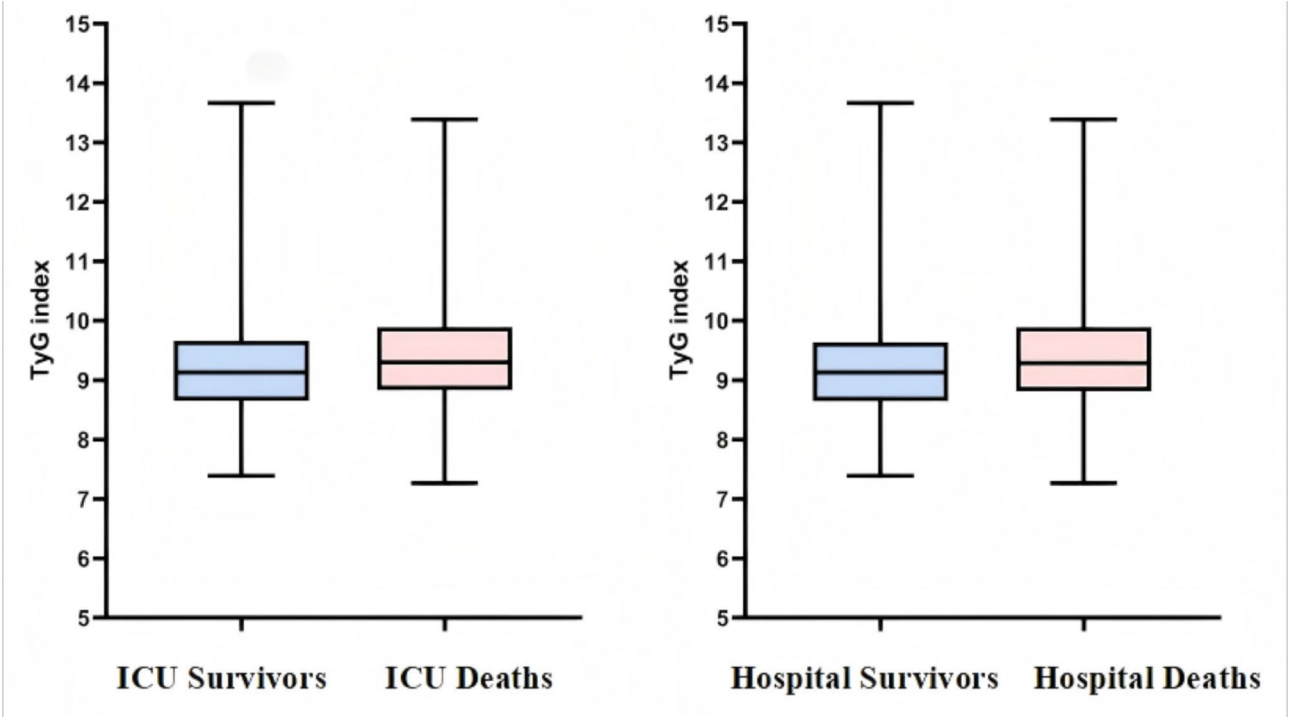

Fig. 2 Distribution of the triglyceride-glucose (TyG) index in survivors vs. non-survivors. Box plots display the TyG index for survivors and nonsurvivors during the 28-day intensive care unit (ICU) and in-hospital periods. Non-survivors had a higher median (Q1-Q3) TyG index: ICU (9.30 [8.84, 9.89] vs. 9.13 [8.65, 9.66], $p = 0.008$) and in-hospital (9.29 [8.82, 9.89] vs. 9.13 [8.65, 9.63], $p = 0.004$)

Figure 4b shows the results off Model 3 in the Cox regression analysis, demonstrating that the predictive performance significantly improved when multiple variables (age, sex, ethnicity, heart rate, SpO_2, hypertension, HF, CKD, cirrhosis, COPD, ARF, sepsis, PLT count, Hb level, BUN level, creatinine level, CRRT, and ventilation status) were considered. The AUROC curve for the model predicting 28-day ICU mortality was 0.80 (95% CI: 0.76–0.83), while the AUROC curve for the model predicting 28-day in-hospital mortality was 0.78 (95% CI: 0.74–0.81).

Subgroup analysis

For patients with CS, a high TyG index (> 9.22) was associated with a significantly increased risk of ICU mortality. Subgroup analyses indicated that this association was particularly pronounced among patients ≥ 60 years, females, individuals with white ethnicity, individuals with a BMI < 30 kg/m², those with AMI, patients with HF, and patients without sepsis. Interaction analyses revealed that the interaction among the different subgroups was significant only within the sepsis subgroup (Fig. 5).

Subgroup analysis of the TyG index with regard to hospital mortality indicated that a high TyG index was more strongly associated with an increased risk of mortality in patients > 60 years, females, individuals of white ethnicity, those with a BMI < 30 kg/m², and patients without sepsis. The interaction analysis revealed that interaction among the different subgroups was significant only within the sepsis subgroup (Fig. 6).

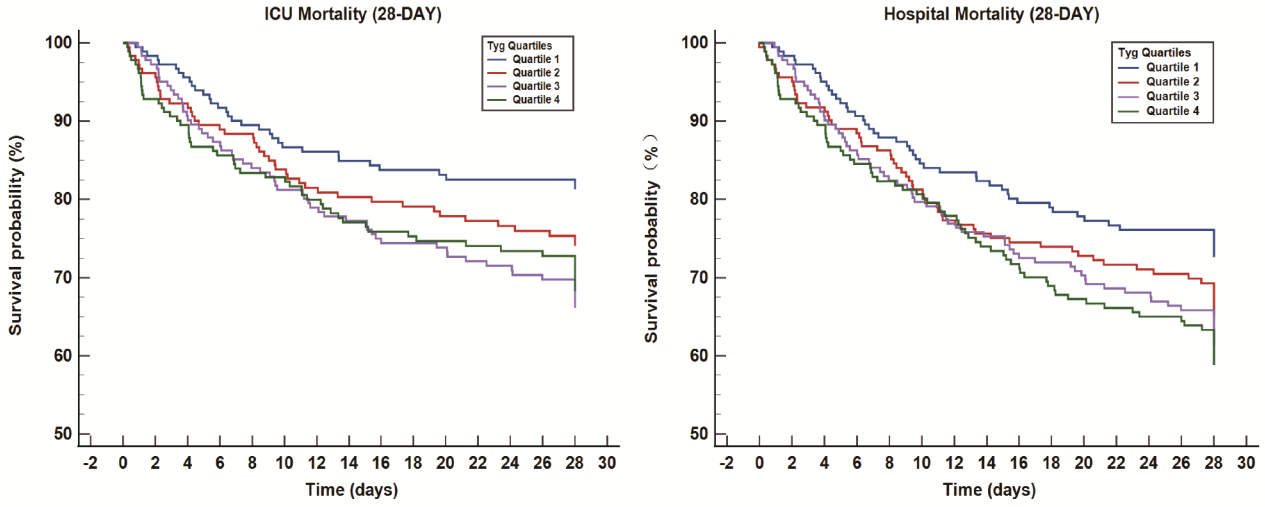

Fig. 3 Kaplan-Meier survival curves by triglyceride-glucose (TyG) index quartiles. Kaplan-Meier survival curves indicated that a higher TyG index is associated with a lower 28-day intensive care unit (ICU) probability of survival (log-rank test: chi-square = 10.8322, $p = 0.012$) and lower 28-day in-hospital probability of survival (chi-square = 8.2280, $p = 0.041$)

Table 2 Cox regression analysis of the association between the TyG index and mortality in cardiogenic shock

TyG index	Model 1			Model 2			Model 3		
	HR	95% CI	*P*	HR	95% CI	*P*	HR	95% CI	*P*
ICU Mortality									
Per 1 Unit increase	1.27	1.08 ~ 1.49	0.003	1.34	1.15 ~ 1.58	< 0.001	1.24	1.04 ~ 1.48	0.015
TyG Quartiles									
Quartile 1	1.00	Reference		1.00	Reference		1.00	Reference	
Quartile 2	1.44	0.92 ~ 2.26	0.108	1.50	0.96 ~ 2.35	0.078	1.43	0.91 ~ 2.26	0.125
Quartile 3	1.92	1.25 ~ 2.93	0.003	2.04	1.33 ~ 3.12	0.001	2.00	1.27 ~ 3.16	0.003
Quartile 4	1.81	1.17 ~ 2.79	0.007	2.08	1.34 ~ 3.22	0.001	1.77	1.11 ~ 2.83	0.016
Hospital Mortality									
Per 1 Unit increase	1.25	1.08 ~ 1.44	0.002	1.32	1.15 ~ 1.53	< 0.001	1.44	1.11 ~ 1.88	0.007
TyG Quartiles									
Quartile 1	1.00	Reference		1.00	Reference		1.00	Reference	
Quartile 2	1.35	0.93 ~ 1.96	0.120	1.40	0.96 ~ 2.04	0.077	1.21	0.82 ~ 1.78	0.327
Quartile 3	1.52	1.05 ~ 2.18	0.026	1.62	1.12 ~ 2.34	0.010	1.48	1.00 ~ 2.21	0.052
Quartile 4	1.65	1.15 ~ 2.37	0.007	1.91	1.32 ~ 2.76	< 0.001	1.61	1.08 ~ 2.38	0.018

Model 1: unadjusted.

Model 2: adjusted for age, gender and race.

Model 3: adjusted for age, gender, race, heart rate, saturation of peripheral oxygen, hypertension, heart failure, chronic kidney disease, cirrhosis, chronic obstructive pulmonary disease, acute renal failure, sepsis, platelets, hemoglobin, blood urea nitrogen, creatinine, continuous renal replacement therapy and ventilation.

TyG: Triglyceride-glucose; HR: Hazard Ratio; CI: Confidence interval; ICU: Intensive care unit

Discussion

In this study, the MIMIC-IV database was used to investigate the relationship between the TyG index and mortality in patients with CS. Our findings show that the TyG index is significantly associated with 28-day ICU mortality and 28-day in-hospital mortality among patients with CS. Although the standalone predictive capacity of the TyG index is modest, combining it with clinical parameters significantly enhanced its prognostic accuracy. Our study also found that this association was particularly pronounced among patients ≥ 60 years, females, individuals with white ethnicity, individuals with a BMI < 30 kg/m², and those with AMI and HF. These results underscore the importance of the TyG index as a simple and effective biomarker for identifying patients with CS at an elevated risk of mortality, highlighting its broad applicability in clinical practice.

Multiple studies have demonstrated that the TyG index serves as an independent predictor of mortality among patients in the ICU, those with AMI, HF, or ischemic stroke [19–23]. To the best of our knowledge, only one study has reported a relationship between the TyG index

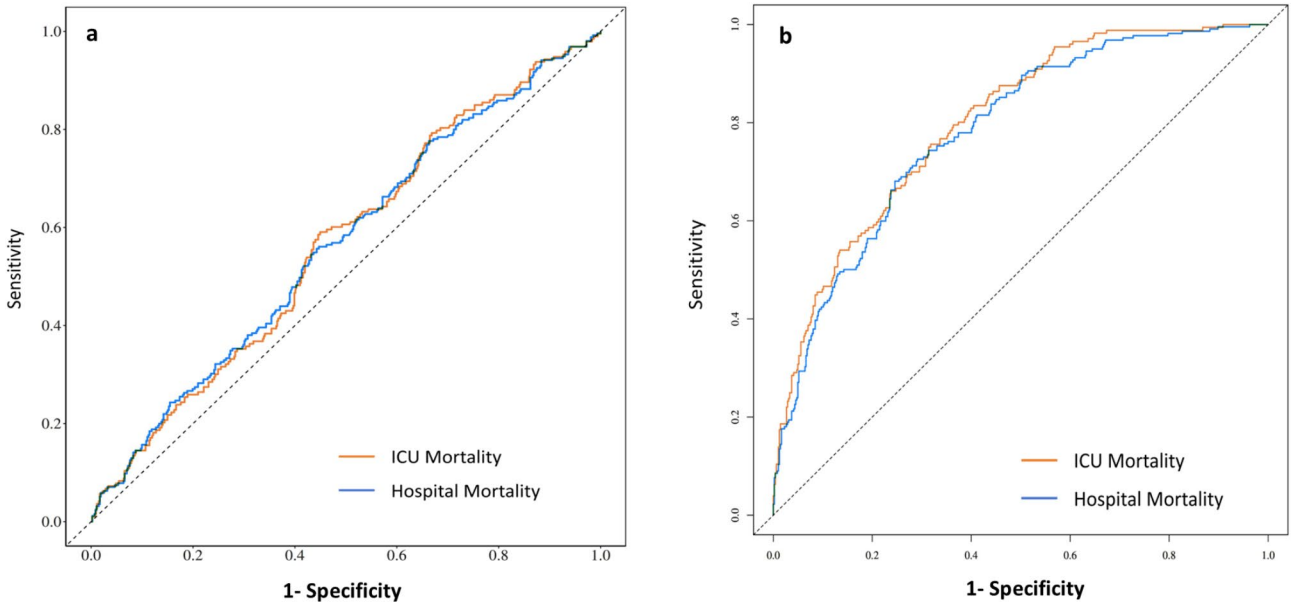

Fig. 4 Predictive ability of the triglyceride-glucose (TyG) index for mortality. Figure **4a** Receiever operating characteristic (ROC) curve for the TyG index alone. The area under the ROC curve (AUROC) for predicting 28-day intensive care unit (ICU) mortality was 0.56 (95% condifendence interval [CI]: 0.52–0.61) with an optimal cutoff value of 9.22, sensitivity of 0.55 (95% CI: 0.51–0.59), and specificity of 0.59 (95% CI: 0.52–0.66). For in-hospital mortality, the AUROC curve was also 0.56 (95% CI: 0.52–0.61) with the same cutoff value, sensitivity of 0.56 (95% CI: 0.51–0.60), and specificity of 0.56 (95% CI: 0.50–0.62). Figure **4b**: ROC curve for the combined model (Model 3) incorporating the TyG index and multiple clinical variables. The AUROCcurve for predicting 28-day ICU mortality was 0.80 (95% CI: 0.76–0.83), and for in-hospital mortality, it was 0.78 (95% CI: 0.74–0.81)

and CS. Liu et al. [16] reported that an elevated TyG index is associated with and an increased risk of mortality in patients with AMICS. The current study and that by Liu et al. [16] examined the relationship between the TyG index and CS, highlighting the potential role of the TyG index in predicting risk of mortality among patients with CS. However, there is a notable difference between our study and that of Liu et al. Their study focused specifically on AMICS and explored the association between the TyG index and the incidence and prognosis of AMICS [16]. In contrast, our study investigated the broader context of CS, encompassing various etiologies beyond AMI, such as HF and sepsis. Additionally, our study demonstrated that the TyG index has a stronger association with mortality in patients without sepsis, a finding that warrants further discussion. However, the role of the TyG index in the prognosis of patients with sepsis remains controversial. Some studies have indicated that an elevated TyG index is associated with poor outcomes [24, 25], whereas others have suggested that an elevated TyG index may serve as a protective prosnostic factor [26]. The controversy may have arisen due to differences in cohort selection or the complex relationship between the TyG index and patients with sepsis. Future studies involving larger-scale clinical cohorts are necessary to fully elucidate the relationship between the TyG index and sepsis.

The significant association between the TyG index and mortality in patients with CS indicates that IR, as assessed using the TyG index, may play a crucial role in the pathophysiology and prognosis of CS. This relationship can be elucidated through several potential mechanisms, including impaired myocardial metabolism, endothelial dysfunction, inflammation, hyperglycemia, autonomic dysfunction, and the presence of comorbidities. IR reduces the ability of insulin to promote glucose uptake and utilization in myocardial cells, leading to a metabolic shift from glucose to fatty acid oxidation [27, 28]. This shift reduces the efficiency of energy production in the heart during periods of increased demand, such as ischemia or HF, resulting in greater vulnerability of myocardial cells to ischemic injury and dysfunction, which can worsen clinical outcomes in patients with CS [29]. IR is closely linked to endothelial dysfunction, characterized by reduced nitric oxide production and increased oxidative stress [30]. This dysfunction can result in decreased coronary blood flow, increased vascular stiffness, and impaired microcirculatory function, all of which contribute to the preogression of CVD [31–33]. IR is associated with inflammation [34, 35], which fosters the development and progression of atherosclerosis. This inflammation can result in instability and rupture of plaque instability, potentially triggering ACS and leading to CS [36]. Furthermore, the pro-inflammatory environment associated with IR can intensify myocardial injury and

Variables	n(%)	Low TyG	High TyG	HR(95%CI)	P	P for interaction
All patients	727 (100.00)	80/375	113/352	1.57 (1.16 ~ 2.12)	0.004	
Age						0.359
< 60 yrs	224 (30.81)	17/98	29/126	1.25 (0.66 ~ 2.37)	0.490	
⩾60 yrs	503 (69.19)	63/277	84/226	1.83 (1.28 ~ 2.60)	<.001	
Gender						0.659
Female	268 (36.86)	35/141	50/127	1.84 (1.15 ~ 2.93)	0.011	
Male	459 (63.14)	45/234	63/225	1.46 (0.96 ~ 2.21)	0.075	
Race						0.312
White	414 (56.95)	42/223	57/191	1.85 (1.20 ~ 2.88)	0.006	
Non−white	313 (43.05)	38/152	56/161	1.19 (0.78 ~ 1.83)	0.419	
BMI						0.860
< 30 kg/m^2	560 (77.03)	67/299	87/261	1.58 (1.12 ~ 2.23)	0.009	
⩾ 30 kg/m^2	167 (22.97)	13/76	26/91	1.60 (0.80 ~ 3.20)	0.186	
AMI						0.566
No	440 (60.52)	53/236	64/204	1.37 (0.93 ~ 2.02)	0.113	
Yes	287 (39.48)	27/139	49/148	1.76 (1.05 ~ 2.93)	0.032	
Heart Failure						0.700
No	167 (22.97)	18/72	41/95	1.54 (0.85 ~ 2.79)	0.156	
Yes	560 (77.03)	62/303	72/257	1.54 (1.07 ~ 2.23)	0.020	
Sepsis						0.010
No	135 (18.57)	6/102	7/33	6.54 (1.49 ~ 28.71)	0.013	
Yes	592 (81.43)	74/273	106/319	1.34 (0.98 ~ 1.84)	0.071	

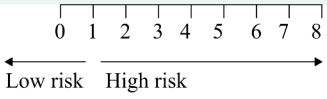

Fig. 5 Subgroup analysis of the triglyceride-glucose (TyG) index for intensive care unit (ICU) mortality

compromise cardiac function [37], further worsening the prognosis of patients with CS. An increased TyG index is frequently associated with hyperglycemia, a common characteristic among critically ill patients [38], including those with CS. Stress-induced hyperglycemia and an elevated blood glucose level due to acute illness are associated with increased mortality and adverse outcomes in critically ill patients [39, 40]. Hyperglycemia can impair immune function, elevate the oxidative stress level, and aggravate myocardial injury [41], thereby contributing to the higher mortality observed in patients with CS and an elevated TyG index. IR can also affect cardiac autonomic function, resulting in increased sympathetic activity and diminished parasympathetic tone [42, 43]. The TyG index is strongly correlated with numerous comorbidities, such as hypertension, T2DM, and obesity, which are well-established risk factors for CVD and adverse outcomes in CS [44, 45]. An elevated TyG index may signify the presence of multiple comorbidities [44]. The TyG index affects the prognosis of patients with CS through multiple mechanisms; thus, further studies are essential for a comprehensive exploration.

This study makes several innovative contributions to the prediction of mortality in patients with CS using the TyG index. First, a diverse cohort of patients with CS with various etiologies was investigated, rather than limiting the focus to AMI. This broad scope allowed exploration of the generalizability of the TyG index across different underlying conditions, thereby enhancing its applicability in clinical practice. Second, this study revealed an independent association between the TyG index and short-term mortality in patients with CS, providing detailed subgroup specificity. Finally, the findings of this study have significant practical implications for clinical practice. As a simple and easy to calculate biomarker, the TyG index can be readily implemented in clinical settings to provide early identification of patients at high-risk and guide targeted interventions aimed at improving outcomes for patients with CS.

Despite providing valuable insights into the relationship between the TyG index and mortality in patients with CS, this study has several limitations that should be acknowledged. First, although there was adjustment for multiple confounders in the statistical models, unmeasured or residual confounding factors may have

Variables	n(%)	Low TyG	High TyG	HR (95%Cl)	P	P for interaction
All patients	727 (100.00)	113/375	142/352	1.40 (1.08 ~ 1.82)	0.011	
Age						0.395
< 60 yrs	224 (30.81)	23/98	38/126	1.20 (0.69 ~ 2.09)	0.509	
⩾ 60 yrs	503 (69.19)	90/277	104/226	1.61 (1.19 ~ 2.18)	0.002	
Gender						0.378
Female	268 (36.86)	46/141	62/127	1.82 (1.20 ~ 2.76)	0.005	
Male	459 (63.14)	67/234	80/225	1.25 (0.88 ~ 1.77)	0.216	
Race						0.492
White	414 (56.95)	62/223	71/191	1.52 (1.05 ~ 2.21)	0.026	
Non−white	313 (43.05)	51/152	71/161	1.19 (0.82 ~ 1.72)	0.369	
BMI						0.716
< 30 kg/m^2	560 (77.03)	95/299	107/261	1.36 (1.01 ~ 1.83)	0.040	
⩾ 30 kg/m^2	167 (22.97)	18/76	35/91	1.65 (0.90 ~ 3.00)	0.104	
AMI						0.504
No	440 (60.52)	76/236	83/204	1.28 (0.92 ~ 1.78)	0.146	
Yes	287 (39.48)	37/139	59/148	1.55 (0.99 ~ 2.42)	0.053	
Heart Failure						0.847
No	167 (22.97)	25/72	54/95	1.53 (0.93 ~ 2.52)	0.097	
Yes	560 (77.03)	88/303	88/257	1.30 (0.95 ~ 1.78)	0.105	
Sepsis						0.028
No	135 (18.57)	9/102	7/33	3.33 (1.03 ~ 10.80)	0.045	
Yes	592 (81.43)	104/273	135/319	1.21 (0.93 ~ 1.59)	0.162	

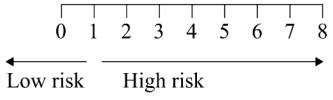

Fig. 6 Subgroup analysis of the triglyceride-glucose (TyG) index for in-hospital mortality

influenced the observed associations. Second, due to the limitations of the database, the TyG index was exclusively used as an indicator of IR and other biomarkers related to IR, such as HOMA-IR, were not investigated. Third, the TyG index was only measured at the time of patient admission and dynamic monitoring was not performed. Future studies should incorporate dynamic monitoring of the TyG index to evaluate its effects on mortality in patients with CS. Finally, the MIMIC-IV database only records all-cause mortality, which limits the ability to conduct stratified analyses.

Conclusion

In conclusion, our study demonstrated that the TyG index is significantly associated with short-term mortality in patients with CS, highlighting its potential as a useful biomarker for risk stratification in clinical practice.

Abbreviations

CS	Cardiogenic Shock
HF	Heart Failure
ACS	Acute Coronary Syndrome
IR	Insulin Resistance
CVD	Cardiovascular Diseases
CHD	Coronary Heart Disease
TyG	Triglyceride-Glucose
HOMA-IR	Homeostasis Model Assessment of IR
ICU	Intensive Care Unit
MIMIC-IV	Medical Information Mart for Intensive Care IV
ICD	International Classification of Diseases
BMI	Body Mass Index
SBP	Systolic Blood Pressure
SpO_2	Peripheral Capillary Oxygen Saturation
T2DM	Type 2 Diabetes Mellitus
AMI	Acute Myocardial Infarction
CKD	Chronic Kidney Disease
COPD	Chronic Obstructive Pulmonary Disease
ARF	Acute Renal Failure
SOFA	Sequential Organ Failure Assessment
SAPS II	Simplified Acute Physiology Score II
OASIS	Oxford Acute Severity of Illness Score
WBC	White Blood Cell
PLT	Platelet
Hb	Hemoglobin
BUN	Blood Urea Nitrogen
TG	Triglycerides
CRRT	Continuous Renal Replacement Therapy
AUROC	Area under the Receiver Operating Characteristic Curve
HR	Hazard Ratio
CI	Confidence Interval

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Author contributions
D.M. was responsible for the design and conceptualization of the study, in addition to drafting and revising the manuscript. D.M. and P.Z. contributed to data collecting, statistical analysis, and result interpretation. M.W. was responsible for the data results visualization. H.D. and J.G. read and revised the manuscript. All authors read and approved the final manuscript.

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Data availability
The datasets produced and analyzed in this research are accessible in the MIMIC-IV database (https://doi.org/10.13026/kpb9-mt58).

Declarations

Ethics approval and consent to participate
The study was performed according to the guidelines of the Helsinki Declaration. The use of the MIMIC-IV database was approved by the review committee of Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center. The data is publicly available (in the MIMIC-IV database), therefore, the ethical approval statement and the requirement for informed consent were waived for this study.

Competing interests
The authors declare no competing interests.

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References

1. Waksman R, Pahuja M, van Diepen S, Proudfoot AG, Morrow D, Spitzer E, et al. Standardized definitions for cardiogenic shock research and mechanical circulatory support devices: scientific expert panel from the shock academic research consortium (SHARC). Circulation. 2023;148(14):1113–26.
2. Berg DD, Bohula EA, Morrow DA. Epidemiology and causes of cardiogenic shock. Curr Opin Crit Care. 2021;27(4):401–8.
3. Jentzer JC, Ahmed AM, Vallabhajosyula S, Burstein B, Tabi M, Barsness GW, et al. Shock in the cardiac intensive care unit: changes in epidemiology and prognosis over time. Am Heart J. 2021;232:94–104.
4. Faerch K, Vaag A, Holst JJ, Hansen T, Jørgensen T, Borch-Johnsen K. Natural history of insulin sensitivity and insulin secretion in the progression from normal glucose tolerance to impaired fasting glycemia and impaired glucose tolerance: the Inter99 study. Diabetes Care. 2009;32(3):439–44.
5. Hill MA, Yang Y, Zhang L, Sun Z, Jia G, Parrish AR, et al. Insulin resistance, cardiovascular stiffening and cardiovascular disease. Metab Clin Exp. 2021;119:154766.
6. Reaven GM. Insulin resistance, the insulin resistance syndrome, and cardiovascular disease. Panminerva Med. 2005;47(4):201–10.
7. Lopaschuk GD, Karwi QG, Tian R, Wende AR, Abel ED. Cardiac energy metabolism in heart failure. Circul Res. 2021;128(10):1487–513.
8. Martín-Saladich Q, Simó R, Aguadé-Bruix S, Simó-Servat O, Aparicio-Gómez C, Hernández C et al. Insights into insulin resistance and calcification in the myocardium in type 2 diabetes: A coronary artery analysis. Int J Mol Sci. 2023;24(4).
9. Ramdas Nayak VK, Satheesh P, Shenoy MT, Kalra S. Triglyceride glucose (TyG) index: A surrogate biomarker of insulin resistance. J Pak Med Assoc. 2022;72(5):986–8.
10. Brito ADM, Hermsdorff HHM, Filgueiras MS, Suhett LG, Vieira-Ribeiro SA, Franceschini S, et al. Predictive capacity of triglyceride-glucose (TyG) index for insulin resistance and cardiometabolic risk in children and adolescents: a systematic review. Crit Rev Food Sci Nutr. 2021;61(16):2783–92.
11. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, Martínez-Abundis E, Ramos-Zavala MG, Hernández-González SO, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab. 2010;95(7):3347–51.
12. Vasques AC, Novaes FS, de Oliveira Mda S, Souza JR, Yamanaka A, Pareja JC, et al. TyG index performs better than HOMA in a Brazilian population: a hyperglycemic clamp validated study. Diabetes Res Clin Pract. 2011;93(3):e98–100.
13. Calcaterra V, Montalbano C, de Silvestri A, Pelizzo G, Regalbuto C, Paganelli V et al. WITHDRAWN: triglyceride glucose index as a surrogate measure of insulin sensitivity in a Caucasian pediatric population. J Clin Res Pediatr Endocrinol. 2019.
14. Ni W, Jiang R, Xu D, Zhu J, Chen J, Lin Y, et al. Association between insulin resistance indices and outcomes in patients with heart failure with preserved ejection fraction. Cardiovasc Diabetol. 2025;24(1):32.
15. Liang S, Wang C, Zhang J, Liu Z, Bai Y, Chen Z, et al. Triglyceride-glucose index and coronary artery disease: a systematic review and meta-analysis of risk, severity, and prognosis. Cardiovasc Diabetol. 2023;22(1):170.
16. Liu H, Wang L, Zhou X, Wang H, Hao X, Du Z, et al. Triglyceride-glucose index correlates with the occurrence and prognosis of acute myocardial infarction complicated by cardiogenic shock: data from two large cohorts. Cardiovasc Diabetol. 2024;23(1):337.
17. Simental-Mendia LE, Rodriguez-Moran M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord. 2008;6(4):299–304.
18. Sterne JA, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338:b2393.
19. Liao Y, Zhang R, Shi S, Zhao Y, He Y, Liao L, et al. Triglyceride-glucose index linked to all-cause mortality in critically ill patients: a cohort of 3026 patients. Cardiovasc Diabetol. 2022;21(1):128.
20. Su X, Zhou Y, Chang J, Zhao X, Li H, Sang H. Association between triglyceride-glucose index and all-cause mortality in critically ill patients with acute myocardial infarction: analysis of the MIMIC-IV database. Front Endocrinol (Lausanne). 2025;16:1447053.
21. Zhang K, Han Y, Gao YX, Gu FM, Cai T, Gu ZX, et al. Association between the triglyceride glucose index and length of hospital stay in patients with heart failure and type 2 diabetes in the intensive care unit: a retrospective cohort study. Front Endocrinol (Lausanne). 2024;15:1354614.
22. Zhai G, Wang J, Liu Y, Zhou Y. Triglyceride-Glucose index linked to In-Hospital mortality in critically ill patients with heart disease. Rev Cardiovasc Med. 2022;23(8):263.
23. Zhang R, Shi S, Chen W, Wang Y, Lin X, Zhao Y, et al. Independent effects of the triglyceride-glucose index on all-cause mortality in critically ill patients with coronary heart disease: analysis of the MIMIC-III database. Cardiovasc Diabetol. 2023;22(1):10.
24. Zhang S, Fan T, Wang L, Chen N, Ma L. Impact of the triglyceride-glucose index on 28-day mortality in non-diabetic critically ill patients with sepsis: a retrospective cohort analysis. BMC Infect Dis. 2024;24(1):785.
25. Zheng R, Qian S, Shi Y, Lou C, Xu H, Pan J. Association between triglyceride-glucose index and in-hospital mortality in critically ill patients with sepsis: analysis of the MIMIC-IV database. Cardiovasc Diabetol. 2023;22(1):307.
26. Cao Y, He L, Su Y, Luo J, Ding N. Triglyceride-glucose index and clinical outcomes in sepsis: A retrospective cohort study of MIMIC-IV. J Cell Mol Med. 2024;28(16):e70007.
27. Ormazabal V, Nair S, Elfeky O, Aguayo C, Salomon C, Zuniga FA. Association between insulin resistance and the development of cardiovascular disease. Cardiovasc Diabetol. 2018;17(1):122.
28. Zhang L, Keung W, Samokhvalov V, Wang W, Lopaschuk GD. Role of fatty acid uptake and fatty acid beta-oxidation in mediating insulin resistance in heart and skeletal muscle. Biochim Biophys Acta. 2010;1801(1):1–22.
29. Karwi QG, Sun Q, Lopaschuk GD. The contribution of cardiac fatty acid oxidation to diabetic cardiomyopathy severity. Cells. 2021;10(11).
30. Kim JA, Montagnani M, Koh KK, Quon MJ. Reciprocal relationships between insulin resistance and endothelial dysfunction: molecular and pathophysiological mechanisms. Circulation. 2006;113(15):1888–904.
31. Jia G, Sowers JR. Endothelial dysfunction potentially interacts with impaired glucose metabolism to increase cardiovascular risk. Hypertension. 2014;64(6):1192–3.
32. Higashi Y, Noma K, Yoshizumi M, Kihara Y. Endothelial function and oxidative stress in cardiovascular diseases. Circ J. 2009;73(3):411–8.
33. Cersosimo E, DeFronzo RA. Insulin resistance and endothelial dysfunction: the road map to cardiovascular diseases. Diabetes Metab Res Rev. 2006;22(6):423–36.
34. Olefsky JM, Glass CK. Macrophages, inflammation, and insulin resistance. Annu Rev Physiol. 2010;72:219–46.

35. Glass CK, Olefsky JM. Inflammation and lipid signaling in the etiology of insulin resistance. Cell Metab. 2012;15(5):635–45.
36. Biasucci LM, Leo M, De Maria GL. Local and systemic mechanisms of plaque rupture. Angiology. 2008;59(2 Suppl):S73–6.
37. Frangogiannis NG. The inflammatory response in myocardial injury, repair, and remodelling. Nat Rev Cardiol. 2014;11(5):255–65.
38. Gearhart MM, Parbhoo SK. Hyperglycemia in the critically ill patient. AACN Clin Issues. 2006;17(1):50–5.
39. Li L, Zhao M, Zhang Z, Zhou L, Zhang Z, Xiong Y, et al. Prognostic significance of the stress hyperglycemia ratio in critically ill patients. Cardiovasc Diabetol. 2023;22(1):275.
40. Zhang C, Shen HC, Liang WR, Ning M, Wang ZX, Chen Y, et al. Relationship between stress hyperglycemia ratio and allcause mortality in critically ill patients: results from the MIMIC-IV database. Front Endocrinol (Lausanne). 2023;14:1111026.
41. Gonzalez P, Lozano P, Ros G, Solano F. Hyperglycemia and oxidative stress: an integral, updated and critical overview of their metabolic interconnections. Int J Mol Sci. 2023;24(11).
42. Paolillo S, Rengo G, Pellegrino T, Formisano R, Pagano G, Gargiulo P, et al. Insulin resistance is associated with impaired cardiac sympathetic innervation in patients with heart failure. Eur Heart J Cardiovasc Imaging. 2015;16(10):1148–53.
43. Poon AK, Whitsel EA, Heiss G, Soliman EZ, Wagenknecht LE, Suzuki T, et al. Insulin resistance and reduced cardiac autonomic function in older adults: the atherosclerosis risk in communities study. BMC Cardiovasc Disord. 2020;20(1):217.
44. Gounden V, Devaraj S, Jialal I. The role of the triglyceride-glucose index as a biomarker of cardio-metabolic syndromes. Lipids Health Dis. 2024;23(1):416.
45. Su W, Wang J, Chen K, Yan W, Gao Z, Tang X, et al. A higher TyG index level is more likely to have enhanced incidence of T2DM and HTN comorbidity in elderly Chinese people: a prospective observational study from the reaction study. Diabetol Metab Syndr. 2024;16(1):29.

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