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The association between the triglyceride–glucose index and short-term mortality in ICU patients with sepsis-associated acute kidney injury

Heping Xu[1*†], Yan Xia[2†], Ruiyong Mo[3] and Yiqiao Liu[3]

Abstract

Background The triglyceride-glucose (TyG) index has emerged as a novel marker for insulin resistance and is commonly observed in patients suffering from sepsis-associated acute kidney injury (SA-AKI). This study explored the correlation between the TyG index and short-term all-cause mortality among SA-AKI patients.

Methods We performed a retrospective analysis of ICU patients with SA-AKI using data from the MIMIC-IV database. The primary outcomes were 28-day and 90-day all-cause mortality. Multivariate Cox proportional hazards regression, restricted cubic spline (RCS) models, and Kaplan–Meier (K–M) survival analyses were used to examine the associations between the TyG index and mortality. Subgroup and sensitivity analyses were conducted to ensure the robustness of the findings.

Results The study included 4971 SA-AKI patients, with 2873 males (57.8%), an average age of 65.4 years (± 15.8), and an average TyG index of 9.10 (± 0.70). RCS analysis revealed a U-shaped relationship between the TyG index and mortality. When the TyG index was below 9.04, the risk of mortality at both 28 days and 90 days was reduced (adjusted HRs of 0.695, 95% CI: 0.542–0.890 and 0.691, 95% CI: 0.557–0.858, respectively). In contrast, values above 9.04 were associated with increased mortality, though the relationship was not statistically significant (adjusted HRs of 1.026, 95% CI: 0.855–1.231 and 1.012, 95% CI: 0.863–1.188, respectively). K–M analysis revealed higher mortality rates for patients with either high (T3) or low (T1) TyG indices than for those with moderate (T2) TyG indices. Sensitivity analyses confirmed these associations even after excluding patients with diabetes, cerebrovascular diseases, or ICU stays of less than 2 days.

Conclusion The TyG index is significantly and nonlinearly associated with short-term all-cause mortality in SA-AKI patients; however, establishing a causal relationship between the two requires validation through larger prospective studies.

Keywords Triglyceride-glucose index, Sepsis, Acute kidney injury, All-cause mortality, Insulin resistance

[†]Heping Xu and Yan Xia are co-first authors.

*Correspondence:
Heping Xu
xhp21528@163.com

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

Introduction

Sepsis is characterized as life-threatening organ dysfunction due to a dysregulated host response to infection [1]. It impacts over 30 million individuals globally each year [2–4]. In the United States, the national weighted incidence of sepsis is estimated to be around 6%, with an in-hospital mortality rate of 15.6% [5]. Among the organs affected by sepsis, the kidneys are the most vulnerable, with studies showing that 45–70% of acute kidney injury (AKI) cases are linked to sepsis [6–9]. Furthermore, sepsis-associated acute kidney injury (SA-AKI) is closely associated with poor clinical outcomes, including extended hospital stays, higher rates of cardiovascular events, and increased mortality [10, 11]. Therefore, the early identification of patients at risk for SA-AKI and the prompt initiation of suitable interventions are crucial to preventing the progression of acute kidney injury.

Insulin resistance (IR), marked by a diminished sensitivity of peripheral tissues to insulin, is frequently observed in septic patients. Acute fluctuations in blood glucose levels can elevate the risk of mortality in these patients [12]. The triglyceride-glucose (TyG) index, derived from fasting blood glucose (FBG) and triglyceride (TG) levels, serves as a reliable surrogate marker for insulin resistance [13–16]. Previous research has demonstrated that a higher TyG index is linked to diabetes, cardiovascular diseases, and all-cause mortality [17–25]. However, there is limited evidence connecting the TyG index to short-term mortality in ICU patients with sepsis-associated acute kidney injury (SA-AKI), necessitating further study. Therefore, this study aimed to examine the associations between the TyG index and clinical outcomes in SA-AKI patients, thereby addressing a gap in the existing literature.

Methods

Data source

The data for this study were obtained from the MIMIC-IV database (version 2.2) [26, 27], a publicly accessible registry developed by the Complex Systems Monitoring Group at Beth Israel Deaconess Medical Center (BIDMC) in Boston, Massachusetts. The dataset includes detailed records of over 50,000 patients admitted between 2008 and 2019, covering demographics, laboratory results, vital signs, diagnoses, and survival data. Because the database is anonymized and does not include protected health information, the BIDMC Institutional Review Board approved a waiver of informed consent and permitted the use of the data for research purposes. Data extraction was performed by the corresponding author, He Ping Xu, who completed the CITI Program online training course (Record ID 59568270), using PostgreSQL as the data management tool.

Definitions

The triglyceride-glucose (TyG) index was calculated using the formula: ln[fasting glucose (mg/dL) × fasting triglycerides (mg/dL) / 2] [28]. Sepsis was diagnosed according to the Sepsis-3 criteria, which define sepsis as life-threatening organ dysfunction caused by a dysregulated response to infection, with a Sequential Organ Failure Assessment (SOFA) score of 2 or higher. Septic shock was identified as sepsis accompanied by a lactate level exceeding 2.0 mmol/L and the need for vasopressor treatment [1]. Acute kidney injury (AKI) was defined by an increase in serum creatinine of ≥ 0.3 mg/dL (≥ 26.5 μmol/L) within 48 h, an increase in serum creatinine to ≥ 1.5 times the baseline level within the past week, or a reduction in urine output to < 0.5 mL/kg/h for 6 h [29]. Baseline creatinine was defined as the lowest serum creatinine value within 7 days prior to ICU admission for AKI, which serves as the reference point for KDIGO staging. SA-AKI was defined as the occurrence of AKI within 7 days of sepsis onset (diagnosed according to Kidney Disease Improving Global Outcome criteria and Sepsis 3 criteria, respectively) [30].

Inclusion and exclusion criteria

Inclusion criteria

1. Patients aged 18 years and above.
2. Patients admitted to the ICU for the first time were diagnosed with sepsis and subsequently diagnosed with acute kidney injury (AKI) within 7 days after the onset of sepsis.

Exclusion criteria

1. Patients with prior ICU admissions were excluded to avoid data duplication.
2. Patients whose survival time was less than 24 h were excluded to ensure a comprehensive assessment of their clinical status and outcomes.
3. Patients with a history of chronic kidney disease were excluded.
4. Patients with missing essential data (such as fasting serum glucose and triglyceride levels) or incomplete data were excluded, as these parameters are critical for accurately calculating the TyG index.

Data extraction

Data were collected directly from the critical care information system, electronic hospital records, laboratory results, and vital sign monitors. Structured Query Language (SQL) was employed to extract data recorded on the first day of ICU admission, including demographics (age, sex, ethnicity), comorbidities (such as myocardial infarction, congestive heart failure, cerebrovascular

disease, chronic pulmonary disease, and diabetes), the Charlson comorbidity index (CCI), the Sequential Organ Failure Assessment (SOFA) score, the Simplified Acute Physiology Score II (SAPS II), septic shock, invasive ventilation, renal replacement therapy (RRT) and laboratory results (including white blood cell count, hemoglobin, platelet count, electrolytes, blood urea nitrogen, prothrombin time, anion gap, bicarbonate, chloride, blood glucose, and triglycerides). The primary outcomes assessed were 28-day and 90-day mortality, while the secondary outcomes included hospital length of stay (LOS), ICU LOS, and in-hospital mortality.

Statistical analysis

Continuous variables are reported as means (standard deviations) or medians (interquartile ranges), while categorical variables are presented as percentages. Baseline characteristics across different TyG index categories were compared using the chi-square test for categorical data, one-way ANOVA for normally distributed continuous data, and the Kruskal-Wallis H test for non-normally distributed continuous data.

Multivariate Cox proportional hazards regression was employed to assess the associations between the TyG index and 28- and 90-day mortality. Multicollinearity was evaluated using the variance inflation factor (VIF), with a VIF greater than 5 indicating significant multicollinearity. Four models were developed for the analysis: (1) An unadjusted model. (2)A model adjusted for demographics and comorbidities. (3) A model further adjusted for vital signs. (4) A fully adjusted model incorporating laboratory test results.

Restricted cubic splines (RCSs) were used to determine cutoff values and visualize the nonlinear associations between the TyG index and 28- and 90-day mortality following ICU admission. Subgroup analyses were performed to assess interactions and effects based on age (< 65 years and ≥ 65 years), sex, ethnicity, congestive heart failure, chronic lung disease, diabetes, cerebrovascular disease, and the presence of shock.

Sensitivity analyses were conducted to further validate the findings. Logistic regression was performed on patient subgroups excluding diabetic patients, patients with cerebrovascular disease, and patients with a hospital length of stay (LOS) of less than 2 days. Statistical significance was set at $P < 0.05$. Data analyses were performed using Stata version 18.0 and the R statistical software package version 4.1.1.

Results

Baseline characteristics

A total of 4,971 critically ill patients with SA-AKI were included in the final analysis, with the screening process detailed in Fig. 1. Baseline characteristics stratified by the TyG index are shown in Table 1. Among the 4,971 patients, 2,873 (57.8%) were male, with a mean age of 65.4 ± 15.8 years and a mean TyG index of 9.10 ± 0.70. Patients with a higher TyG index (≥ 9.04) were generally younger and had a greater incidence of diabetes compared to those with a TyG index < 9.04. Additionally, patients in the higher TyG group experienced longer ICU and hospital lengths of stay (LOS). However, no statistically significant differences were observed in in-hospital mortality, 28-day mortality, or 90-day mortality between the two groups.

Associations of the TyG index with clinical outcomes in SA-AKI patients

Table 2 illustrates the associations between the TyG index and short-term all-cause mortality. The analysis was performed by stratifying the TyG index into two groups. Four Cox regression models were developed to assess the independent effect of the TyG index on short-term all-cause mortality in ICU patients with SA-AKI. After adjusting for age, sex, ethnicity, comorbidities, laboratory tests, severity scores, and shock, restricted cubic spline (RCS) analysis revealed a nonlinear association between the TyG index and prognosis, with a threshold of 9.04 (nonlinear p-value < 0.05; Fig. 2).

These findings indicate a significant association between the TyG index and prognosis, with differing effects below and above the threshold. Specifically, in the multivariate two-stage Cox regression model, the TyG index showed a pronounced effect depending on the threshold. For SA-AKI patients with a TyG index < 9.04, each unit increase in the TyG index was associated with a 30.5% reduction in the risk of all-cause mortality at 28 days (adjusted HR = 0.695; 95% CI: 0.542–0.890) and a 30.8% reduction at 90 days (adjusted HR = 0.691; 95% CI: 0.557–0.858). In contrast, for patients with a TyG index ≥ 9.04, each unit increase in the TyG index was associated with a 2.6% increase in the risk of all-cause mortality at 28 days (adjusted HR = 1.026; 95% CI: 0.855–1.231) and a 1.2% increase at 90 days (adjusted HR = 1.012; 95% CI: 0.863–1.188), although these associations were not statistically significant. These results highlight the complex relationship between the TyG index and mortality outcomes in critically ill SA-AKI patients, emphasizing the importance of considering the TyG index level in prognostic assessments.

Kaplan–Meier survival analysis

The study cohort was stratified into three groups based on TyG index tertiles: T1, T2, and T3. Kaplan–Meier survival analysis was conducted to assess short-term mortality in SA-AKI patients. As illustrated in Fig. 3, the short-term survival curves of the T1 and T3 groups were significantly lower than those of the T2 group (log-rank

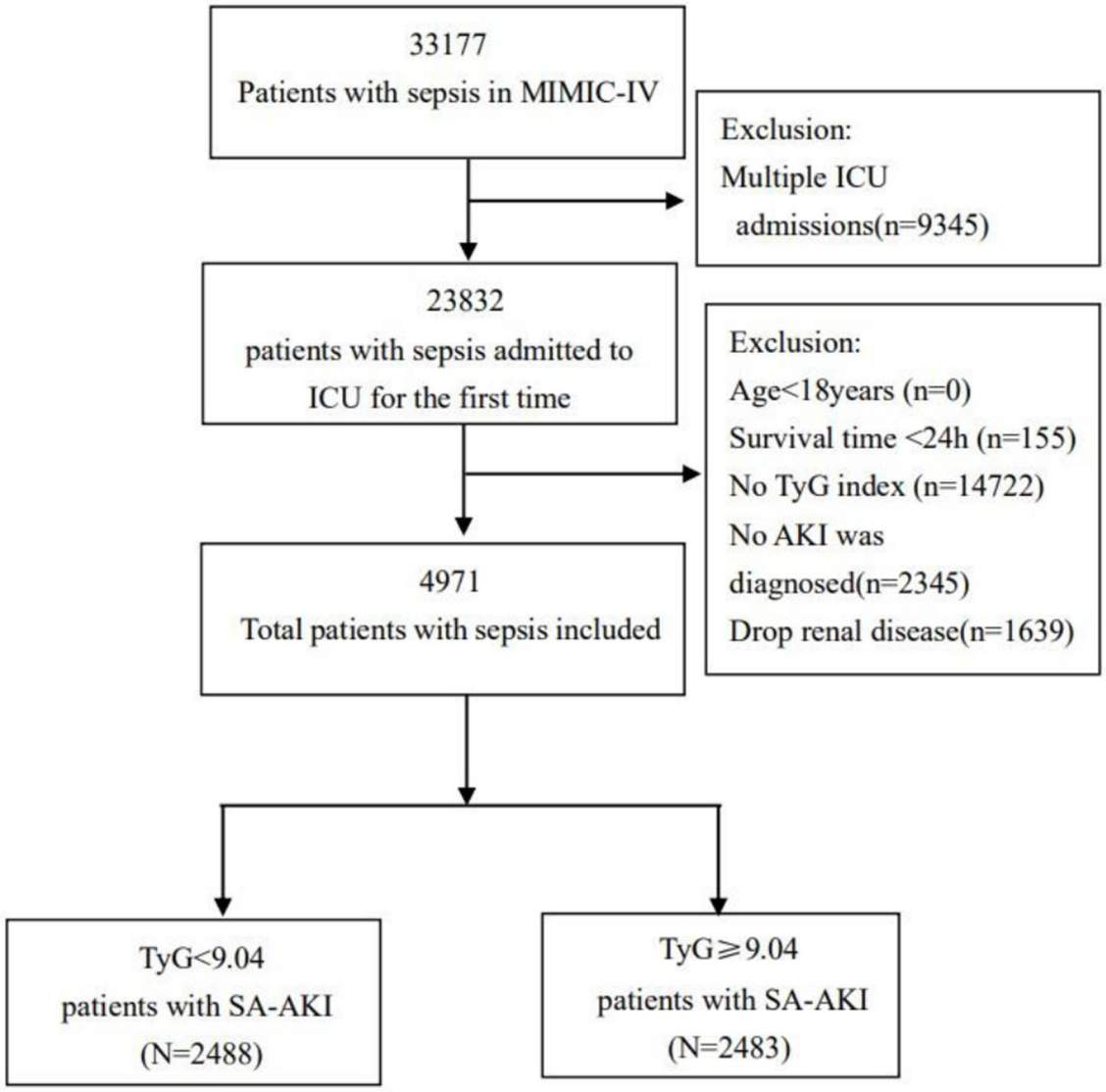

Fig. 1 Flow chart of patient selection for analysis

test, $P<0.05$). No statistically significant difference was observed between the T1 and T3 groups ($P>0.05$), suggesting that both a higher or lower TyG index at admission may be associated with increased short-term mortality risk.

Subgroup analysis

To explore potential clinical heterogeneity, interaction and stratified analyses were conducted (Fig. 4). The associations between the TyG index and short-term mortality were evaluated across subgroups stratified by age (<65 and ≥65 years), sex, race, history of myocardial infarction, congestive heart failure, cerebrovascular disease, chronic lung disease, diabetes, and septic shock, in order to assess the relationship between the TyG index and short-term mortality The results indicated that for low TyG levels (<9.04), there was a significant reduction in both 28-day and 90-day mortality risk, with protective effects observed across most subgroups, particularly in patients without heart failure, diabetes, or shock. For high TyG levels (≥9.04), there was no significant effect on 28-day mortality risk overall, but a trend toward increased risk was observed at 90 days, particularly in subgroups such as males and those with chronic lung disease. These findings suggest that the TyG index may be a valuable risk prediction marker. No significant interactions were observed between the TyG index and 28-day or 90-day mortality rates across the subgroups.

Sensitivity analysis

The results of the sensitivity analyses are summarized in Table 3. These findings were consistent with the initial

Table 1 Baseline characteristics and outcomes of SA-AKI patients grouped according to the TyG index

Characteristics	TyG index			
	All patients (*N* = 4971)	< 9.04 (*N* = 2488)	≥ 9.04 (*N* = 2483)	*P*
TyG	9.10(0.70)	8.55(0.34)	9.64(0.50)	< 0.0001
Demographic				
Age, years	65.4(15.8)	67.7(15.6)	63.1(15.7)	< 0.0001
Sex (male, n)	2873(57.8)	1435(57.7)	1438(57.9)	0.866
Ethnicity (white, n)	3369(67.8)	1728(69.5)	1641(66.1)	0.011
Comorbidities				
Myocardial infarction	767(15.4)	384(15.4)	383(15.4)	0.993
Congestive heart failure	1129(22.7)	587(23.6)	542(21.8)	0.138
Chronic pulmonary disease	1178(23.7)	586(23.6)	592(23.8)	0.811
Diabetes	1347(27.1)	435(17.5)	912(36.7)	< 0.0001
Cerebrovascular disease	816(16.4)	424(17.0)	392(15.8)	0.233
Severity scores				
Charlson comorbidity index	5(4–7)	5(4–7)	5(3–6)	0.0003
First day of SOFA	7(4–10)	6(4–9)	7(4–10)	< 0.0001
SAPSII	39(31–49)	39(31–49)	40(31–50)	0.0729
Vital signs				
SBP, mmHg	112.9(104.6-124.1)	112.0(104.0-122.6)	113.8(105.4–125)	< 0.0001
DBP, mmHg	61.2(55.2–67.9)	60.7(54.5–67.0)	61.7(55.8–68.8)	< 0.0001
Heart rate, beats/min	86.6(76.5–98.9)	84.8(75.5–96.8)	88.1(77.7–101.0)	< 0.0001
Respiratory rate, beats/min	19.4(17.0-22.5)	19.1(16.7–22.0)	19.8(17.2–23.0)	< 0.0001
Temperature, °C	36.9(36.7–37.2)	36.9(36.6–37.1)	36.9(36.7–37.3)	< 0.0001
SpO2, %	97.2(95.8–98.5)	97.3(95.9–98.6)	97.1(95.6–98.5)	0.0040
Laboratory parameters				
WBC, cell/mm3	12.1(8.8–16.0)	11.6(8.5–15.5)	12.45(9.2-16.55)	< 0.0001
Hemoglobin, mg/dL	10.6(9.2–12.2)	10.5(9.2–12.1)	10.8(9.4–12.4)	< 0.0001
Platelet, cell/mm3	180.5(127–247)	177(125-240.5)	184.0(127.5–252.0)	0.0109
Sodium, mEq/L	138.5(136.0-141.0)	138.5(136.0-141.0)	138.5(136.0-141.0)	0.0342
Potassium, mEq/L	4.2(3.8–4.6)	4.10(3.8–4.5)	4.2(3.85–4.6)	0.0004
Calcium, mg/dL	8.25(7.8–8.7)	8.25(7.8–8.7)	8.25(7.8–8.6)	0.3025
BUN, mg/dL	20.0(14.0-30.5)	19.5(13.5–30.0)	20.5(14.5–31.0)	0.0008
Creatinine, mg/dL	1.0(0.75–1.4)	0.95(0.75–1.35)	1.05(0.8–1.5)	< 0.0001
PT, sec	14.6(12.9–17.3)	14.7(13.2–17.8)	14.35(12.8–16.8)	< 0.0001
Anion gap, mEq/L	14.5(12–17)	15(12.5–17.5)	14(12-16.5)	< 0.0001
Bicarbonate, mEq/L	22.5(20–25)	22.5(19.5–25)	23(20.5–25.5)	< 0.0001
Chloride, mEq/L	104.5(101–108)	104.5(100.5–108)	105(101–108)	0.1794
Outcome				
Septic shock	2801(56.3)	1341(53.9)	1460(58.8)	< 0.0001
RRT	471(9.5)	172(6.9)	299(12.0)	< 0.0001
Invasive ventilation	1769(35.6)	808(32.5)	961(38.7)	< 0.0001
LOS ICU	4.4 (2.2–9.3)	3.9(2.1–7.9)	5.1(2.4–10.8)	< 0.0001
LOS hospital	10.9(6.4–19.0)	10.3(6.1–17.4)	11.8(6.9–20.4)	< 0.0001
In-hospital mortality	981(19.7)	486(19.5)	495(19.9)	0.722
28-day mortality	1059(21.3)	543(21.8)	516(20.8)	0.369
90-day mortality	1410(28.4)	716(28.8)	694(28.0)	0.517

Continuous variables are presented as the means (SDs) or medians (quartiles), whereas categorical variables are presented as absolute numbers (percentages). TyG, triglyceride–glucose; SOFA, Sequential Organ Failure Assessment score; SAPS II, simplified acute physiology score II; SBP, systolic blood pressure; DBP, diastolic blood pressure; SpO2, pulse oxygen saturation; WBC, white blood cell; BUN, blood urea nitrogen; PT, RRT, renal replacement therapy; prothrombin time; LOS, length of stay

results through various sensitivity analyses. After excluding high-risk diabetic patients, the impact of the TyG index on 28-day and 90-day all-cause mortality remained minimal. When TyG < 9.04, the adjusted hazard ratio (HR) for 28-day mortality in Sensitivity Analysis 1 was 0.657 (95% CI: 0.502–0.861), and the adjusted HR for 90-day mortality was 0.676 (95% CI: 0.534–0.854). In Sensitivity Analysis 2, the adjusted HR for 28-day mortality was 0.663 (95% CI: 0.506–0.870), and the adjusted HR for 90-day mortality was 0.667 (95% CI: 0.528–0.843).

Table 2 Relationships between the TyG index and all-cause mortality according to different models

TyG index	Model1		Model2		Model3		Model4	
	HR (95% CI) *P* value		HR (95% CI) *P* value		HR (95% CI) *P* value		HR (95% CI) *P* value	
28-day mortality								
TyG index (< 9.04)	0.700(0.552,0.887)	0.003	0.762(0.598,0.971)	0.028	0.708(0.554,0.905)	0.006	0.695(0.542,0.890)	0.004
TyG index (≥ 9.04)	1.306(1.109,1.539)	0.001	1.123(0.942,1.340)	0.197	1.026(0.856,1.229)	0.781	1.026(0.855,1.231)	0.781
90-day mortality								
TyG index (< 9.04)	0.691(0.562,0.849)	< 0.0001	0.750(0.608,0.925)	0.007	0.696(0.562,0.860)	0.001	0.691(0.557,0.858)	0.001
TyG index (≥ 9.04)	1.257(1.089,1.451)	0.002	1.115(0.955,1.301)	0.169	1.013(0.865,1.186)	0.874	1.012(0.863,1.188)	0.881

OR, Odds ratio; CI, Confidence interval

Model 1: Unadjusted model

Model 2: Adjusted for age, sex, ethnicity, myocardial infarction, congestive heart failure, cerebrovascular disease, chronic pulmonary disease, diabetes, the Charlson comorbidity index, the SOFA score, the SAPSII score, invasive ventilation, RRT and septic shock

Model 3: Adjusted for variables included in Model 2 + SBP, respiratory rate, heart rate, temperature and SpO2

Model 4: Adjusted for variables included in Model 3 + white blood cell count, hemoglobin, platelet count, calcium, blood urea nitrogen, creatinine, potassium and prothrombin time

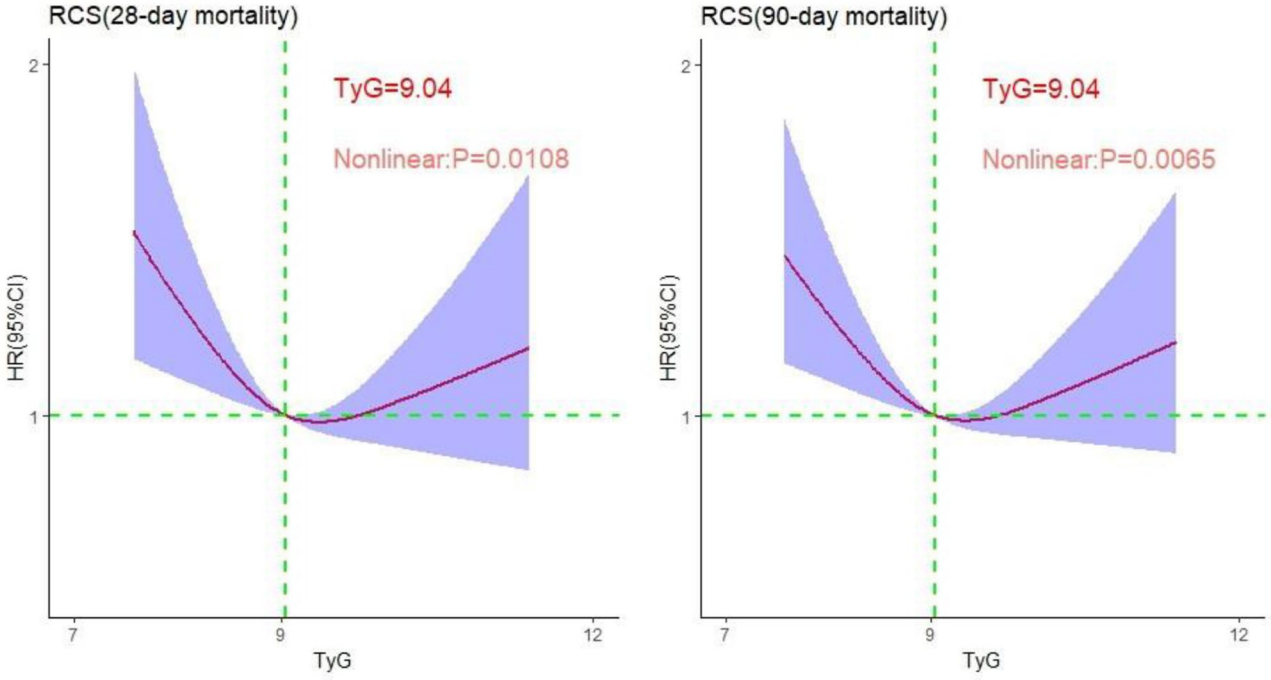

Fig. 2 Nonlinear relationships between TyG and short-term mortality rates in patients with SA-AKI

Furthermore, the association persisted after excluding patients with an ICU length of stay (LOS) of 2 days or less. In Sensitivity Analysis 3, the adjusted HR for 28-day mortality was 0.708 (95% CI: 0.542–0.925), and the adjusted HR for 90-day mortality was 0.717 (95% CI: 0.568–0.906).However, for patients with TyG ≥ 9.04, the three sensitivity analyses showed no statistical significance.

Discussion

In this study, we analyzed a large cohort of ICU patients with sepsis-associated acute kidney injury (SA-AKI) and identified a non-linear relationship between the triglyceride-glucose (TyG) index and 28-day and 90-day all-cause mortality. This relationship persisted even after adjusting for multiple confounding factors, indicating that both high and low TyG index values are associated with increased mortality, with a critical threshold identified at 9.04. These findings underscore the complex role of insulin resistance in SA-AKI outcomes. The association remained robust across various sensitivity analyses, further validating our results.

Previous studies have focused primarily on the TyG index in critically ill cardiovascular patients, with most studies indicating that an elevated TyG index is associated with increased in-hospital and ICU mortality [23–25]. For instance, Zheng et al. [31] found that a higher TyG index was linked to increased all-cause in-hospital

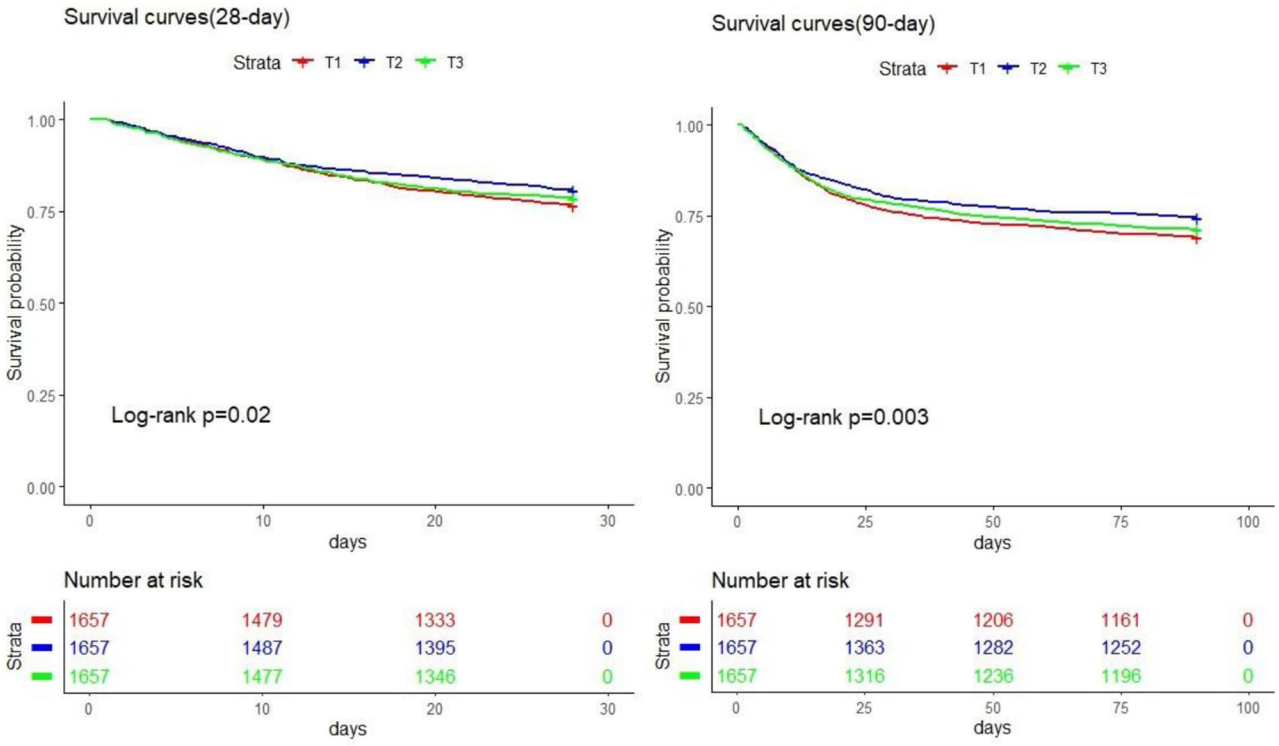

Fig. 3 Kaplan–Meier plots for short-term mortality by ICU admission TyG strata

mortality in critically ill adult septic patients. Similarly, Gao et al. [32] reported a U-shaped relationship between the TyG index and in-hospital or ICU mortality in critically ill pediatric patients. However, no prior studies have specifically examined the relationship between the TyG index and SA-AKI. Our study extends these findings to critically ill SA-AKI patients and reveals a non-linear relationship between TyG index and 28-day and 90-day all-cause mortality.

This study examined the relationship between the TyG index and mortality in patients with SA-AKI, revealing a nonlinear relationship between the TyG index and both 28-day and 90-day all-cause mortality. Elevated mortality rates may be associated with both high and low TyG index values, with a critical threshold of 9.04. Kaplan–Meier survival analysis confirmed significantly higher mortality rates in patients with extreme TyG index values compared to those with intermediate values.

The mechanisms underlying the relationship between the TyG index and mortality risk in critically ill septic patients with SA-AKI are complex. Insulin resistance, reflected by the TyG index, can induce oxidative stress, leading to glomerular endothelial cell injury, basement membrane thickening, and mesangial cell proliferation, ultimately causing renal insufficiency [33]. In the hyperinsulinemic state, insulin promotes sodium reabsorption and increases the glomerular filtration rate, potentially resulting in kidney damage [34, 35]. Additionally, the inflammatory cascade effect of sepsis further impairs immune function, exacerbating adverse outcomes [36, 37].

Conversely, a low TyG index may indicate malnutrition, severe illness, or insufficient metabolic reserves, all of which can also contribute to increased mortality [38]. Recent studies have shown that the neutrophil-to-lymphocyte ratio (NLR) is closely associated with acute kidney injury in critically ill patients, with higher or lower NLR reflecting immune imbalance and inflammation, which can further affect prognosis [39–42]. Therefore, fluctuations in TyG index may not only reflect metabolic disturbances but also be associated with immune-inflammatory states, which significantly influence the prognosis of SA-AKI patients.

In conclusion, the TyG index may serve as a valuable prognostic marker in ICU patients with SA-AKI, offering potential clinical implications for patient management. However, this conclusion requires further validation through multi-center, large-scale prospective studies. It is important to note that the retrospective design of this study limits the ability to establish causal relationships, and while multivariable adjustments and subgroup analyses were performed, potential confounders may still affect the results. Additionally, the retrospective nature of this study may introduce selection bias and residual confounding factors, which could impact external validity. Another limitation is that we only collected data from the

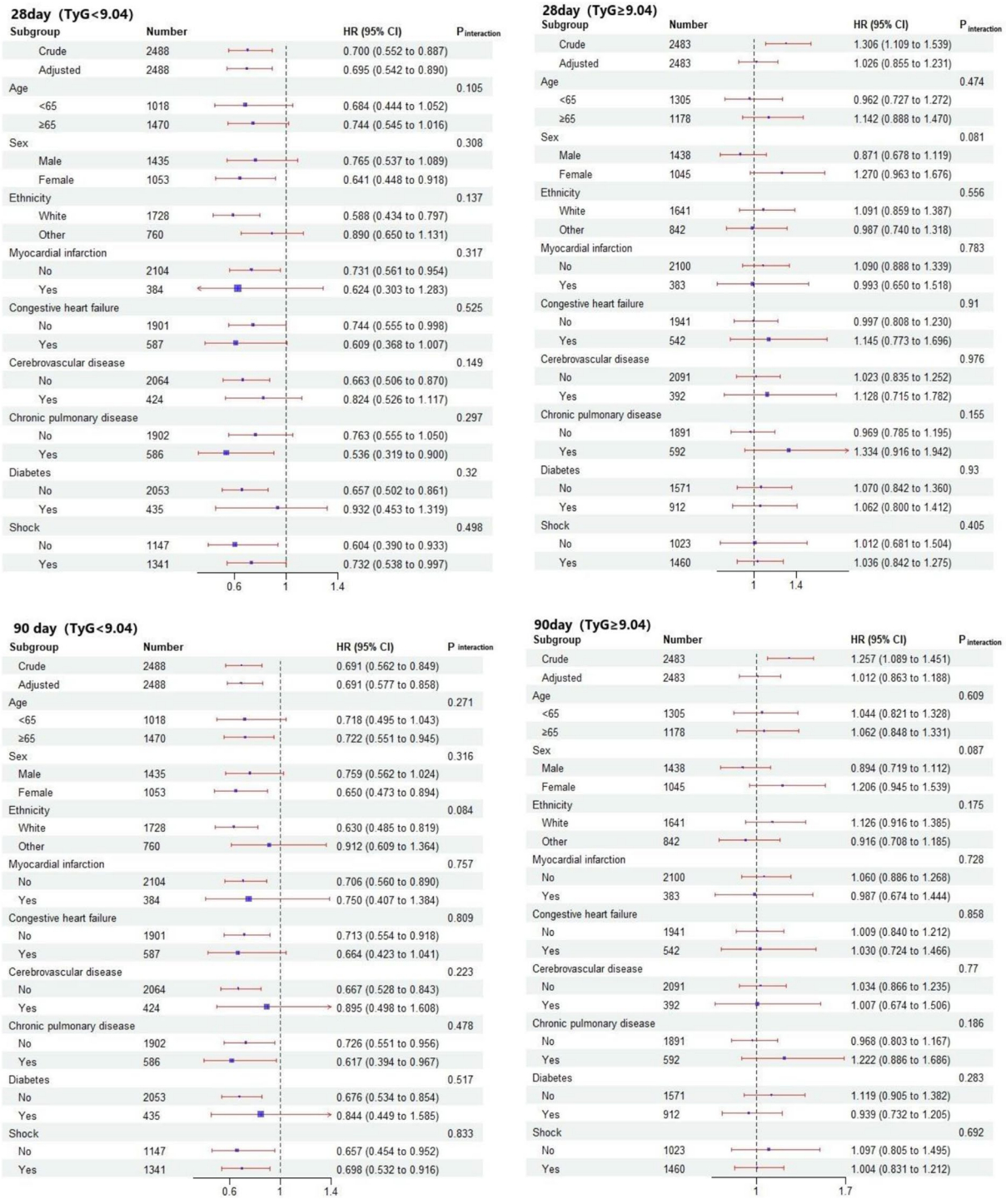

Fig. 4 Effect size of the TyG score on short-term mortality in prespecified and exploratory subgroups. The effect size was adjusted for age, sex, ethnicity, myocardial infarct, congestive heart failure, cerebrovascular disease, chronic pulmonary disease, diabetes, Charlson comorbidity index, SOFA score, SAPSII score, invasive ventilation, RRT, septic shock, SBP, respiratory rate, heart rate, temperature SpO2, white blood cell count, hemoglobin, platelet count, potassium, blood urea nitrogen, calcium, and creatinine, with the exception of the subgroup variable

first 24 h of hospitalization, including fasting glucose and triglycerides, which could lead to missing TyG data and affect the accuracy of the results. Furthermore, since the TyG index is calculated based on a single measurement of triglycerides and fasting glucose, it primarily reflects baseline values and does not capture dynamic changes in insulin resistance. Finally, the findings from this single-center study need to be confirmed by multi-center

Table 3 Sensitivity analyses

TyG index		28-day mortality		90-day mortality	
		HR(95%CI)	*P*	HR(95%CI)	*P*
Excluding participants with diabetes(*N* = 3624)					
TyG index (< 9.04)	2053	0.657(0.502,0.861)	0.002	0.676(0.534,0.854)	0.001
TyG index(≥ 9.04)	1571	1.070(0.842,1.360)	0.581	1.119(0.905,1.382)	0.300
Excluding participants with cerebrovascular disease(*N* = 4155)					
TyG index (< 9.04)	2064	0.663(0.506,0.870)	0.003	0.667(0.528,0.843)	0.001
TyG index(≥ 9.04)	2091	1.023(0.835,1.252)	0.829	1.034(0.866,1.235)	0.711
Exclude participants with LOS ICU ≤ 2 days(*N* = 3948)					
TyG index (< 9.04)	1932	0.708(0.542,0.925)	0.011	0.717(0.568,0.906)	0.005
TyG index(≥ 9.04)	2016	1.069(0.881,1.298)	0.497	1.022(0.862,1.211)	0.805

OR, Odds ratio; CI, Confidence interval; Ref, Reference; Adjusted for age, Sex, Ethnicity, Myocardial infarct, Congestive heart failure, Cerebrovascular disease, Chronic pulmonary disease, Diabetes, Charlson comorbidity index, SOFA score, SAPSII score, Invasive ventilation, RRT, Septic shock, SBP, Respiratory rate, Heart rate, Temperature, SpO2, White blood cell count, Hemoglobin, Platelet count, Creatinine, Potassium, Blood urea nitrogen, Calcium and prothrombin time

studies to ensure generalizability. Future research should focus on larger, more rigorously designed studies to provide stronger evidence to support the use of TyG as a predictive marker.

Conclusion

In summary, our study demonstrated that the TyG index is significantly and nonlinearly associated with short-term all-cause mortality in SA-AKI patients; however, establishing a causal relationship between the two requires validation through larger prospective studies.

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Author contributions

H.X and Y.X were responsible for conceptualization, methodology, and formal analysis. R.M and Y.L conducted the visualization and investigation. The original draft of the manuscript was written by H.X and Y.X, while R.XM and Y.L contributed to the review and editing of the manuscript. Funding for the research was acquired by H.X.

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Data availability

The datasets used and generated in this study are included in this published article.

Declarations

Ethics approval and consent to participate

This study uses data from the MIMIC-IV database, an anonymized public dataset approved by the Institutional Review Boards (IRBs) of the Massachusetts Institute of Technology (MIT) and Beth Israel Deaconess Medical Center (BIDMC). The requirement for informed consent was waived due to the thorough anonymization and de-identification of all patient information. All research procedures comply with the ethical standards set forth in the Declaration of Helsinki. Clinical trial number: not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Emergency Medicine, Hainan General Hospital/Hainan Affiliated Hospital of Hainan Medical University, No. 19, Xiuhua Road, Xiuying District, Haikou City, Hainan Province 570311, China
[2]Department of General Practice, The First Affiliated Hospital of Hainan Medical University, Haikou City, Hainan Province 570103, China
[3]Department of Emergency Medicine, Hainan Affiliated Hospital of Hainan Medical University, Haikou City, Hainan Province 570311, China

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