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Association between triglyceride glucose-body mass index and all-cause mortality in critically ill patients with acute pancreatitis

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This study delves into the correlation between the triglyceride glucose-body mass index (TyG-BMI) index upon hospital admission and clinical outcomes among this patient population. We investigated the association between TyG-BMI at hospital admission and clinical outcomes in this patient group, and analyzed data from the Medical Information Mart for Intensive Care IV database, identifying acute pancreatitis (AP) patients admitted to ICUs and stratifying them by TyG-BMI quartiles. We assessed the relationship between TyG-BMI and mortality (both in-hospital and ICU) using Cox proportional hazards regression and restricted cubic splines. The cohort included 419 patients, average age 56.34 ± 16.62 years, with a majority being male (61.58%). Hospital and ICU mortality rates were 11.93% and 7.16%, respectively. Higher TyG-BMI was positively correlated with increased all-cause mortality. Patients in the highest TyG-BMI quartile had significantly greater risks of in-hospital and ICU mortality. An S-shaped curve in the spline analysis indicated a threshold effect at a TyG-BMI of 243 for increased in-hospital mortality risk. TyG-BMI is a reliable predictor of both in-hospital and ICU mortality in severely ill AP patients, suggesting its utility in enhancing risk assessment and guiding clinical interventions for this vulnerable population.

Keywords Acute pancreatitis, Triglyceride glucose-body mass index (TyG-BMI), All-cause mortality, Insulin resistance, MIMIC-IV

Pancreatitis represents a primary cause of hospitalization among patients with gastrointestinal disorders, entailing substantial morbidity, mortality, and socioeconomic burdens[1,2]. It also assumes a significant role in escalating hospital mortality[3]. Roughly one in five patients develop moderate to severe acute pancreatitis (AP), triggering severe complications such as necrosis of pancreatic or surrounding tissues and failure of multiple organs. Reported mortality rates fluctuate between 20% and 40%[4,5], underscoring the urgency of promptly evaluating AP severity and implementing interventions. There are several scoring systems available to evaluate AP severity, such as Ranson criteria[6], Acute Physiology and Chronic Health Evaluation II (APACHE-II)[6], Balthazar grade[7], and Bedside Index for Severity in Acute Pancreatitis (BISAP)[8]. These scoring systems typically involve a more complex assessment process that requires gathering multiple indicators. This time-consuming assessment process may lead to potential delays in identifying the optimal treatment window, thereby increasing the risk of mortality in certain patients. Thus, it is urgent to identify a simplistic, cost-efficient, and acutely sensitive predictive marker for the severity of AP.

Insulin resistance (IR) is a condition in which insulin becomes less effective in facilitating the uptake and utilization of glucose[9]. This condition is closely associated with the development of AP and is a significant risk factor for its progression[10]. The triglyceride-glucose (TyG) index, which is derived from fasting triglyceride (TG) and blood glucose (FBG) levels, has emerged as a simple surrogate marker for IR[11].

Obesity, as measured by body mass index (BMI), plays a pivotal role in the development of IR, and stands as a critical determinant in its etiology[12]. The TyG-BMI composite index, a fusion of TyG and BMI, has shown commendable agreement with the homeostatic model assessment of IR in measuring IR in Korean[9] and Chinese cohorts[13]. The predictive role of TyG-BMI index has been demonstrated in various clinical conditions, such as

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ischemic stroke[14], heart failure[15], and the necessity of percutaneous coronary intervention[16]. We hypothesize that this index is associated with prognostic outcomes in patients afflicted with AP and could potentially be utilized for early prognostic assessment. While the prognostic implications of the TyG-BMI index have been studied across a spectrum of diseases, its impact on clinical endpoints within the AP patient population has not yet been fully investigated. Therefore, the aim of our study is to ascertain the relationship between TyG-BMI and mortality in AP patients.

Methods

Database

We harnessed clinical data from the Medical Information Mart for Intensive Care (MIMIC)-IV database, an international online intensive care repository. The database contains patient-related information from the Intensive Care Units (ICUs) of Beth Israel Deaconess Medical Center from 2008 to 2019. It comprises detailed records of 299,712 hospitalized patients and 73,181 critical care patients. In our research team, the author Y.L. has completed the Collaborative Institutional Training Initiative program, (certification number 53244021).

Study population and definitions

Data extraction was conducted utilizing Structured Query Language within a PostgreSQL environment (version 14.6). Based on our analysis of the MIMIC-IV database[17], ICU admission is generally determined by the actual care received by patients, typically including those with hemodynamic instability, respiratory failure requiring mechanical ventilation, or other organ dysfunctions necessitating close monitoring and support. AP was diagnosed by the International Classification of Disease, 9th Revision (ICD-9) code 577.0 or 10th Revision (ICD-10) code K85. in patients aged over 18 years. Patients lacking recorded admission-day levels of FBG, TG, or BMI were excluded. The methodological flowchart is depicted in Fig. 1.

Data collection

The TyG-BMI index was designated as the primary variable of this investigation. Baseline characteristics of patients were extracted from the database, encompassing demographics such as age, sex, ethnicity, marital status, and BMI. We also collected the Simplified Acute Physiology Score II (SAPS II) at admission and the administration details of Vasopressin and Octreotide. The SAPS II, used for severity assessment, consists of 17 variables including physiological measurements, age, and admission type. Higher scores indicate greater severity and higher predicted mortality[18]. SAPS II is widely used for assessing severity in critically ill patients, including those with AP[19]. Comorbidities at baseline, including chronic lung disease, hypertension, acute kidney injury (AKI), heart failure, diabetes, and malignancy, were collected. Pertinent laboratory parameters at ICU admission were also gathered, encompassing red blood cell distribution width, red blood cell (RBC) count, white blood cell (WBC) count, platelet count, alanine aminotransferase (ALT) levels, blood glucose, hemoglobin concentration, creatinine, lactate, international normalized ratio, blood urea nitrogen, anion gap, serum chloride levels, potassium levels, sodium levels, and total bilirubin levels. To reduce potential biases, variables with over 15% missing values were excluded from the analysis. The "mice" package in the R software was used for processing missing data.

Definition and clinical outcomes

The TyG-BMI index was calculated using the following equation: $\ln [TG\ (mg/dL) \times FBG\ (mg/dL)/2] \times BMI$[16]. The primary endpoint was all-cause mortality during hospitalization, while the secondary endpoint was ICU mortality.

Statistical analysis

Continuous variables are presented as mean ± SD or median (interquartile range), depending on their distribution. Meanwhile, categorical variables were displayed as proportions. The Kolmogorov-Smirnov test was

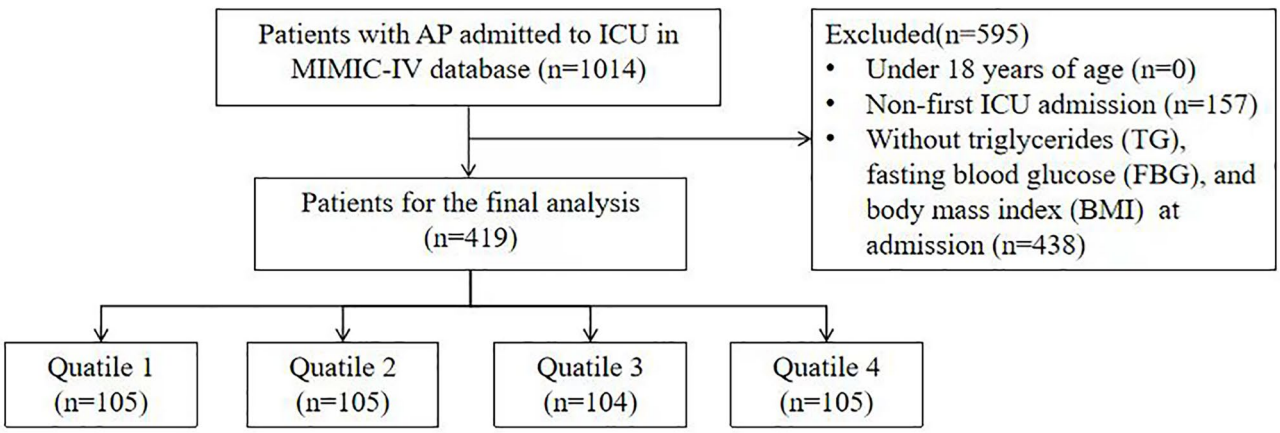

Fig. 1. Flowchart of participants through the trial.

used for testing the normality of continuous data. Normally distributed variables were analyzed utilizing t-test or ANOVA, while the Mann-Whitney U-test and Kruskal-Wallis test were reserved for non-normally distributed variables.

To investigate the effect of the TyG-BMI index on short-term survival, Kaplan-Meier survival analysis was executed to ascertain 28-day and 90-day all-cause mortality in patients with different TyG-BMI levels, and log-rank testing was used for comparisons between groups. The relation between the TyG-BMI index and mortality was further explored through univariate and multivariate Cox regression analyses. Model 1 served as the unadjusted reference; Model 2 was adjusted for demographic variables including age, sex, and ethnicity. Model 3 was additionally adjusted for a comprehensive range of clinical covariates such as comorbidities (heart failure, AKI, hypertension, diabetes, sepsis, chronic obstructive pulmonary disease, and malignancies), hematologic and biochemical parameters (WBC, RBC, platelet count, ALT levels), metabolic and physiologic data (serum lactate, creatinine, total bilirubin, anion gap), SAPS II, and specific treatments (Vasopressin and Octreotide administration). The Cox regression analysis and a restricted cubic spline model with four knots were leveraged to ascertain the link between TyG-BMI index and the risk of in-hospital and ICU mortality in critically ill patients with AP. All statistical analyses were carried out using the R software, version 4.0.2.

Results

This investigation incorporated 419 patients admitted for severe AP. The median age of these patients was 56.34 years, and male patients constituted 61.58%. The median value of the TyG-BMI of the participants was 297.88. The in-hospital and ICU mortality rates were 11.93% and 7.16% respectively, as detailed in Table 1.

Baseline characteristics

The patients were classified into quartiles by the level of the TyG-BMI index [quartile 1 (134.91-241.68), quartile 2 (241.68-281.39), quartile 3 (281.39-349.13), quartile 4 (349.13-683.47)], as shown in Table 1. Individuals with the uppermost quartile of the TyG-BMI index tended to be older and exhibited elevated SAPS II scores upon admission, increased prevalence of diabetes and AKI, as well as higher levels of RBC, hemoglobin, glucose, TG, creatinine, and potassium compared to individuals in lower quartiles. Despite these differences, the in-hospital and ICU mortality rates did not significantly diverge across quartiles ($P > 0.05$).

Kaplan–Meier analysis

Figure 2 illustrates Kaplan-Meier survival analysis curves assessing key outcomes among different TyG-BMI groups. Noteworthy, no significant disparities in mortality at 28 and 90 days were observed (log-rank $P > 0.05$).

Cox regression analysis

Cox proportional hazards regression was leveraged to scrutinize the correlation between the TyG-BMI and in-hospital and ICU mortality outcomes. The analytical findings showed that TyG-BMI is significantly associated with an increased risk of in-hospital mortality. This association was observed in all models: unadjusted [HR, 1.03; 95% CI: 1.01–1.06; $P = 0.020$], partially adjusted [HR, 1.04; 95% CI: 1.01–1.07; $P = 0.014$], and fully adjusted [HR, 1.05; 95% CI: 1.01–1.10; $P = 0.010$], when TyG-BMI was treated as a continuous variable. In addition, the index was identified as a significant risk factor for ICU mortality in AP patients in the fully adjusted model (HR: 1.10; 95% CI: 1.02–1.18; $P = 0.017$). The uppermost quartile of the TyG-BMI was correlated with a significant increase in the risk of in-hospital mortality [HR, 4.08; 95% CI: 1.34–12.41; $P = 0.007$] compared to the nadir quartile, as shown in the fully adjusted Cox regression (Table 2). similar results for ICU mortality were found in multivariate Cox regression analysis (Table 2).

Restricted cubic splines

The restricted cubic splines were employed to better characterize and graphically represent the relationship between the TyG-BMI index and in-hospital mortality. As illustrated in Fig. 3, a non-linear correlation was noted between TyG-BMI index and in-hospital mortality. Specifically, the mortality risk initially decreased within the lower spectrum of TyG-BMI, to a nadir at a TyG-BMI index value of approximately 243, and then elevated steeply (P for overall < 0.001, P for non-linearity $= 0.003$).

Discussion

The objective of this investigation was to scrutinize the correlation of TyG-BMI index with clinical prognoses amongst a demographic of acutely ill patients afflicted with AP. Following adjustment for confounders, our findings indicated that escalated TyG-BMI values were closely linked with an upsurge in all-cause mortality both within ICU settings and overall hospitalization among these individuals. As such, the TyG-BMI index might be instrumental as a prognostic indicator for healthcare providers, potentially serving as an independent marker of mortality in the context of AP. Our findings align with studies demonstrating the prognostic value of TyG-BMI in various clinical contexts. TyG-BMI has shown associations with cardiovascular outcomes[20–22], stroke risk[23,24], and mortality in critical care settings[25,26]. It has also been linked to coronary artery disease severity[16,20] and long-term outcomes in heart failure[21]. Our study extends the utility of TyG-BMI to AP.

The pathogenic mechanisms underlying AP significantly involve inflammation within the peripancreatic adipose tissue[27]. IR, as a protracted, low-grade inflammatory state, is typified by heightened systemic concentrations of pro-inflammatory cytokines[28,29]. This increase in pro-inflammatory cytokines further intensifies inflammation following the initial trigger and magnifies damage to the pancreas and other organs, resulting in systemic inflammatory response syndrome, organ failure, and local complications. Obesity and diabetes, which serve as representative markers of metabolic disorders, have been strongly linked to the

Categories	Overall (*N* = 419)	Q1 (*N* = 105)	Q2 (*N* = 105)	Q3 (*N* = 104)	Q4 (*N* = 105)	*P*-value
Demographic						
Age, years	56.34 ± 16.62	60.72 ± 18.38	56.46 ± 16.99	53.59 ± 15.65	54.57 ± 14.51	0.010
Male, n (%)	258 (61.58)	55 (52.38)	65 (61.90)	65 (62.50)	73 (69.52)	0.086
Ethnicity, n (%)	0.983					
White	282 (67.3)	69 (65.71)	70 (66.67)	71 (68.27)	72 (68.57)	
Black	32 (7.64)	9 (8.57)	8 (7.62)	6 (5.77)	9 (8.57)	
Other	105 (25.06)	27 (25.71)	27 (25.71)	27 (25.96)	24 (22.86)	
BMI, kg/m^2, mean ± SD	30.78 ± 7.31	24.70 ± 3.86	29.41 ± 5.78	31.62 ± 5.31	37.40 ± 7.37	< 0.001
Marital status (%)	0.578					
Married	189 (45.11)	43 (40.95)	46 (43.81)	48 (46.15)	52 (49.52)	
Single	161 (38.42)	39 (37.14)	40 (38.10)	41 (39.42)	41 (39.05)	
Divorced	40 (9.55)	11 (10.48)	12 (11.43)	8 (7.69)	9 (8.57)	
Other	29 (6.92)	12 (11.43)	7 (6.67)	7 (6.73)	3 (2.86)	
Laboratory tests						
RBC, m/μL, mean ± SD	3.77 ± 0.88	3.68 ± 0.87	3.79 ± 0.84	3.58 ± 0.87	4.04 ± 0.90	0.001
WBC, K/μL, mean ± SD	14.46 ± 8.80	13.63 ± 9.24	15.28 ± 9.97	14.46 ± 7.93	14.46 ± 7.91	0.605
Platelet, K/μL, mean ± SD	220.20 ± 132.33	211.02 ± 125.14	233.19 ± 132.93	220.10 ± 149.09	216.50 ± 121.50	0.661
Hemoglobin, g/dL, mean ± SD	11.53 ± 2.57	11.22 ± 2.67	11.66 ± 2.40	10.98 ± 2.65	12.27 ± 2.40	0.001
Glucose, mg/dL, mean ± SD	159.60 ± 111.63	137.86 ± 159.40	145.62 ± 60.75	162.49 ± 87.44	192.47 ± 107.89	0.002
TG, mg/dL, mean ± SD	482.80 ± 1106.02	160.00 ± 245.59	288.02 ± 614.79	482.12 ± 1173.05	1001.07 ± 1643.44	< 0.001
Creatinine, mg/dL, mean ± SD	1.64 ± 1.73	1.44 ± 1.65	1.26 ± 1.38	1.81 ± 1.82	2.06 ± 1.91	0.003
ALT, IU/L, mean ± SD	395.84 ± 1626.18	627.93 ± 2302.70	350.20 ± 1493.74	435.72 ± 1703.61	169.87 ± 333.07	0.230
Albumin, g/dL, mean ± SD	2.90 ± 0.62	2.97 ± 0.68	2.97 ± 0.61	2.87 ± 0.61	2.80 ± 0.58	0.117
Sodium, mEq/L, mean ± SD	138.10 ± 5.97	138.17 ± 5.48	138.49 ± 5.20	137.57 ± 6.71	138.15 ± 6.42	0.733
Potassium, mEq/L, mean ± SD	4.15 ± 0.80	4.13 ± 0.88	3.96 ± 0.66	4.16 ± 0.91	4.33 ± 0.69	0.009
Calcium, mg/dl, mean ± SD	104.36 ± 7.27	103.99 ± 6.59	104.20 ± 6.65	104.18 ± 8.57	105.07 ± 7.18	0.713
INR, mean ± SD	1.52 ± 0.94	1.73 ± 1.38	1.45 ± 0.88	1.47 ± 0.67	1.44 ± 0.63	0.079
Bilirubin, total, mg/dL, mean ± SD	1.52 ± 0.94	1.73 ± 1.38	1.45 ± 0.88	1.47 ± 0.67	1.44 ± 0.63	0.079
RDW	14.81 ± 1.74	15.02 ± 1.77	14.50 ± 1.58	15.00 ± 1.94	14.70 ± 1.62	0.092
Aniongap, mean ± SD	15.96 ± 4.94	15.76 ± 5.11	16.12 ± 4.53	16.05 ± 5.08	15.92 ± 5.10	0.956
Lactate, mean ± SD	2.31 ± 1.91	2.46 ± 2.40	2.07 ± 1.24	2.11 ± 1.34	2.60 ± 2.31	0.120
TyG index	9.60 ± 1.21	8.80 ± 0.83	9.32 ± 0.89	9.81 ± 1.01	10.49 ± 1.35	< 0.001
TyG-BMI index	297.88 ± 83.41	207.07 ± 27.76	260.71 ± 10.24	310.05 ± 18.48	413.83 ± 57.48	< 0.001
Comorbidities						
Hypertension, n, %	188 (44.87)	38 (36.19)	46 (43.81)	49 (47.12)	55 (52.38)	0.119
Diabetes, n, %	123 (29.36)	15 (14.29)	24 (22.86)	37 (35.58)	47 (44.76)	< 0.001
HF, n (%)	52 (12.41)	15 (14.29)	11 (10.48)	10 (9.62)	16 (15.24)	0.528
AKI, n (%)	270 (64.44)	59 (56.19)	58 (55.24)	74 (71.15)	79 (75.24)	0.002
COPD, n (%)	16 (3.82)	7 (6.67)	2 (1.90)	5 (4.81)	2 (1.90)	0.218
MT, n, %	28 (6.68)	13 (12.38)	5 (4.76)	6 (5.77)	4 (3.81)	0.055
Sepsis, n, (%)	279 (66.59)	67 (63.81)	61 (58.10)	73 (70.19)	78 (74.29)	0.067
Medicine						
Vasopressin	94 (22.43)	19 (18.10)	21 (20.00)	29 (27.88)	25 (23.81)	0.336
Octreotide	36 (8.59)	11 (10.48)	7 (6.67)	11 (10.58)	7 (6.67)	0.575
SAPSII, mean ± SD	36.61 ± 16.35	36.26 ± 16.07	33.17 ± 15.12	37.23 ± 16.21	39.80 ± 17.44	0.031
Events						
Hospital mortality	50 (11.93)	12 (11.43)	7 (6.67)	19 (18.27)	12 (11.43)	0.079
ICU mortality	30 (7.16)	9 (8.57)	4 (3.81)	10 (9.62)	7 (6.67)	0.381

Table 1. Baseline characteristics of acute pancreatitis patients grouped according to TyG-BMI index quartile. *TyG-BMI index: Q1 (134.91-241.68), Q2 (241.68-281.39), Q3 (281.39-349.13), Q4 (349.13-683.47). *TyG-BMI index* triglyceride glucose-body mass index, *BMI* body mass index, *RBC* red blood cell, *WBC* white blood cell, *TG* triglyceride, *ALT* alanine aminotransferase, *RDW* red blood cell distribution width, *INR* malignant tumor, *TyG* triglyceride-glucose, *HF* heart failure, *MT* malignant tumor, *COPD* chronic obstructive pulmonary disease, *AKI* acute kidney injury, *SAPS II* Simplified Acute Physiology Score II, *ICU* malignant tumor.

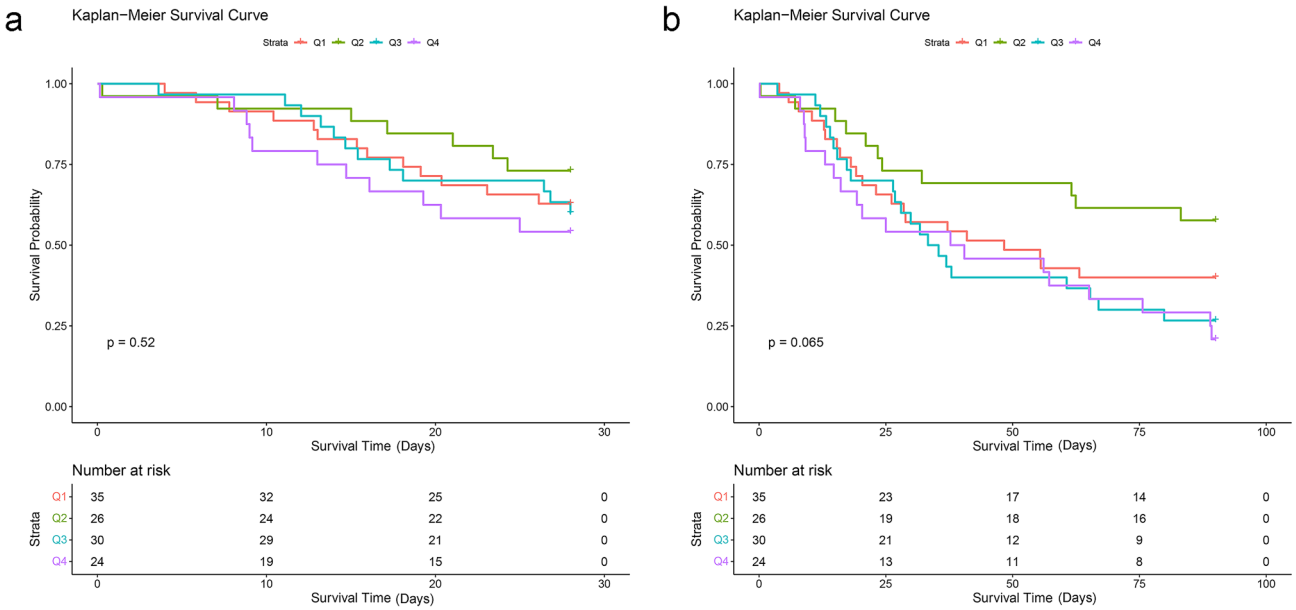

Fig. 2. Kaplan–Meier survival analysis curves for all-cause mortality at 28 days (**a**), and 90 days (**b**).

Categories	Model 1		Model 2		Model 3	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Hospital mortality						
Continuous variable per 10 units	1.03 (1.01–1.06)	0.020	1.04 (1.01–1.07)	0.014	1.05 (1.01–1.10)	0.010
Quartile						
Q1	Ref.		Ref.		Ref.	
Q2	0.67 (0.26–1.71)	0.406	0.71 (0.28–1.81)	0.466	0.98 (0.30–3.24)	0.973
Q3	1.89 (0.92–3.89)	0.085	1.89 (0.90–3.97)	0.093	1.72 (0.61–4.85)	0.307
Q4	1.63 (0.73–3.64)	0.230	1.79 (0.79–4.07)	0.164	4.08 (1.34–12.41)	0.013
ICU mortality						
Continuous variable per 10 units	1.02 (0.99–1.06)	0.197	1.04 (0.99–1.09)	0.094	1.10 (1.02–1.18)	0.017
Quartile						
Q1	Ref.		Ref.		Ref.	
Q2	0.50 (0.15–1.64)	0.254	0.54 (0.16–1.78)	0.312	0.72 (0.16–3.24)	0.664
Q3	1.22 (0.50–3.01)	0.660	1.18 (0.46–2.98)	0.734	0.89 (0.23–3.46)	0.861
Q4	1.21 (0.45–3.25)	0.705	1.38 (0.49–3.83)	0.542	6.53 (1.13–37.84)	0.036

Table 2. Cox proportional hazard ratios (HR) for all-cause mortality. *Model 1: Unadjusted model; †Model 2: adjusted for gender, age, race; ‡Model 3: adjusted for gender, age, race, heart failure, acute kidney injury, hypertension, diabetes, chronic pulmonary diseases, sepsis, malignant tumor, WBC, RBC, platelet, alanine aminotransferase, serum lactate, creatinine, total bilirubin, anion gap, SAPS II, the use of Vasopressin and Octreotide. *CI* confidence interval, *TyG-BMI* triglyceride glucose-body mass index, *HR* hazard ratio.

occurrence and progress of AP[30,31]. The association between TyG-BMI and mortality in our study could be attributed to its capture of both lipid metabolism and obesity[32], which are crucial factors in the pathogenesis of AP[33]. Additionally, insulin resistance, reflected by TyG-BMI[13,34], may exacerbate the inflammatory response in AP[34].

IR is a pivotal contributor to the pathogenesis of type 2 diabetes mellitus, dyslipidemic disorders, adiposity, and cardiovascular pathologies[35]. Consequently, the TyG index was conceived as a substitute indicator for assessing IR[36,37]. Additionally, BMI is frequently utilized to evaluate obesity and its correlation with IR. A study utilizing data from the Korean National Health and Nutrition Examination Survey has shown that the TyG-BMI index exhibits superior accuracy in delineating IR compared to other metrics[9]. The TyG-BMI index has been recognized as an efficacious marker for assessing IR[16], and stands as a prognostic element for ischemic cerebrovascular events, heart failure, and the necessity for percutaneous coronary intervention[14–16]. However, there is limited research on the prognostic value of TyG-BMI in determining the prognosis of patients with AP. To our knowledge, this is the inaugural analysis to probe the association of TyG-BMI index with in-hospital

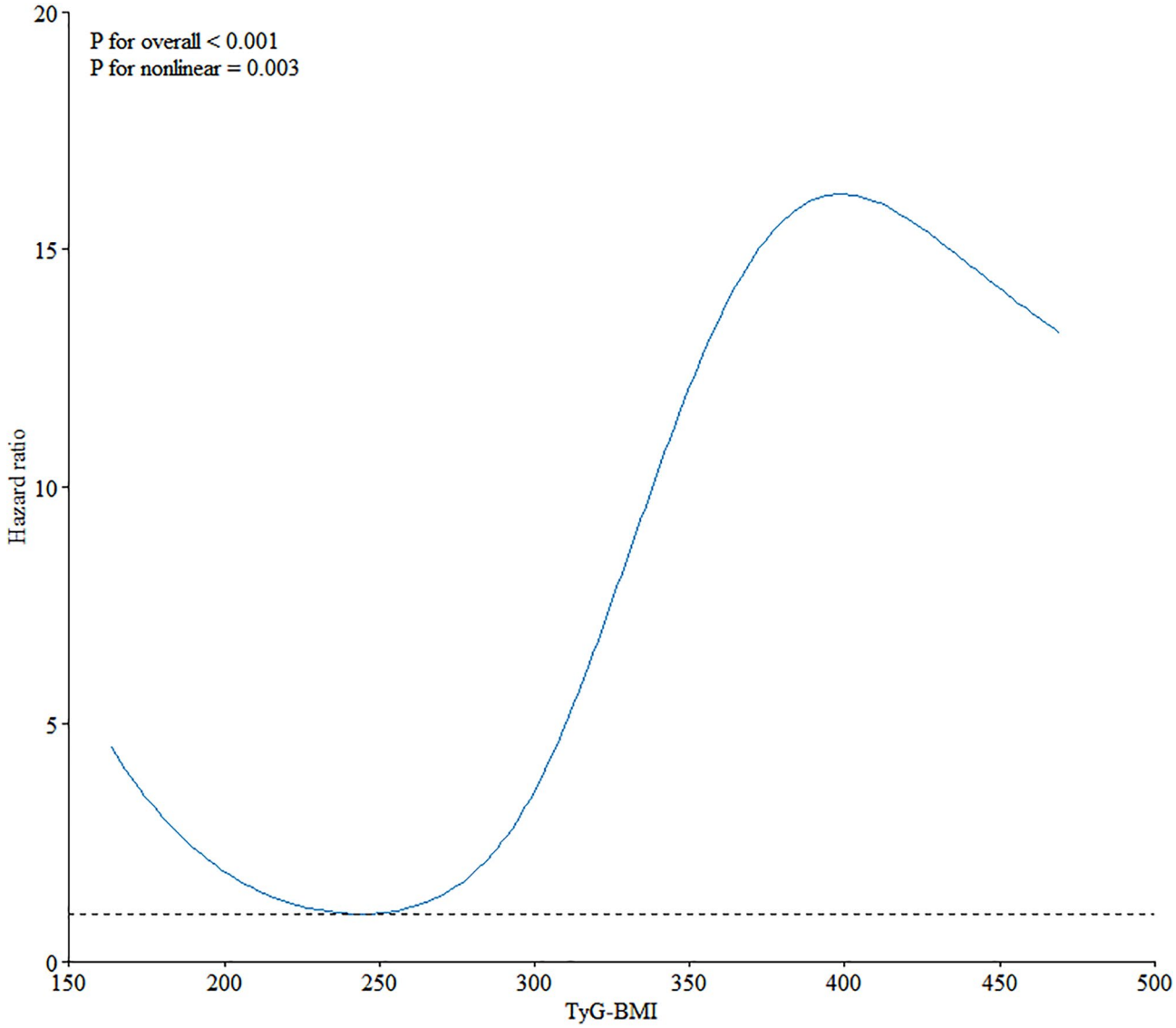

Fig. 3. Relationship between the TyG-BMI index and in-hospital mortality.

and ICU mortality in individuals with AP, using data from the MIMIC-IV database. Clinically, TyG-BMI could be a valuable tool for risk stratification in AP patients due to its simplicity and reliance on readily available parameters. However, further research is needed to establish optimal cut-off values and validate its use in diverse populations.

Nonetheless, our investigation has several limitations. Firstly, the study is a single-center, observational study, which hinders us from inferring causation. Secondly, due to the insufficient sample size, subgroup analysis was not conducted. We look forward to conducting subgroup analysis in a larger prospective study. Thirdly, TyG-BMI measurements were limited to the time of ICU admission in this study, and the changes during the course of hospitalization were not measured. Fourthly, there are limitations related to the MIMIC-IV database and our assessment tools. The database contains a mixture of ICD-9 and ICD-10 codes, with ICD-9 not specifying pancreatitis etiology, preventing consistent analysis of etiology for all cases. Additionally, while the SAPS II used for severity assessment is widely validated for critically ill patients, it may not capture some pancreatitis-specific factors. The database lacked variables required for pancreatitis-specific scoring systems, which might have provided more targeted prognostic information. Finally, due to the lack of longer follow-up data in the MIMIC-IV database, our study focuses solely on in-hospital and ICU mortality, which may have affected the overall prognostic assessment. Hence, additional studies are warranted to corroborate our observations.

Conclusion

Overall, our study reveals a significant association between higher TyG-BMI index and increased mortality in critically ill AP patients. This expands the index's applicability to acute care settings and suggests its potential as a simple risk stratification tool. Future prospective, multi-center studies are needed to validate these findings,

establish the index's clinical utility, and explore its integration with established pancreatitis-specific scoring systems in AP management.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

All authors contributed to the study conception and design. Yang Zhu was responsible for the initial drafting of the manuscript. Yang Zhu, Ye Li, Xuan Li, Sheng Huang, and Yihui Li participated in the material preparation, data collection, and analysis. All authors critically reviewed and provided feedback on previous versions of the manuscript. Yang Zhu made significant contributions to revising and finalizing the manuscript. All authors read and approved the final manuscript.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

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