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Association between the triglyceride glucose index and the risk of acute respiratory failure in patients with acute pancreatitis

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Abstract

Background The triglyceride glucose (TyG) index serves as a dependable marker for insulin resistance and has shown a significant correlation with the severity of acute pancreatitis (AP). However, no research exists regarding the association between the TyG index and the development of acute respiratory failure (ARF) in AP. This study assesses the association between TyG index and ARF in patients with AP.

Methods Retrospective cohort analysis was conducted with the MIMIC-IV 2.2 critical care data. The endpoint focused on ARF during hospitalization. Statistical analysis encompassed univariate and multivariate logistic regressions, alongside restricted cubic spline (RCS) analysis to explore potential nonlinear associations. Receiver operating characteristic (ROC) curve analysis was employed to identify the optimal TyG index cutoff, leading to the classification of patients into Low TyG and High TyG groups. Propensity score matching (PSM) and inverse probability of treatment weighting (IPTW) were subsequently applied to minimize the influence of confounding factors, thereby further clarifying the relationship between the TyG index and ARF in patients with AP.

Results A total of 758 patients were involved in this study, the incidence of ARF was 21.64%. Logistic regression analyses demonstrated a significant association between the TyG index and the incidence of ARF in patients with AP. The RCS model illustrated a nonlinear relationship between a higher TyG index and an increased risk of ARF. The cutoff value of TyG index was 9.099 for ARF in patients with AP based on the ROC curve analysis. Furthermore, following PSM and IPTW, multivariate logistic regression analysis indicated that the High TyG group exhibited a significantly higher risk of ARF compared to the Low TyG group ($P < 0.05$).

Conclusions The TyG index is associated with ARF risk in AP patients and may aid in early risk assessment.

Keywords Triglyceride glucose index, Insulin resistance, Acute pancreatitis, Acute respiratory failure, MIMIC-IV database

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Introduction

Acute pancreatitis (AP), increasingly recognized in clinical practice, has a global pooled incidence of 34 cases per 100,000 general population per year, and studies suggest that persistent organ failure and infected pancreatic necrosis further increase the mortality risk associated with AP [1]. In a study by Schepers involving 639 pancreatitis patients, 240 developed multiple organ failure, with 92% experiencing respiratory failure, and a mortality rate of 37%, while respiratory failure had the longest duration, with a median of 19 days (IQR 7–39), surpassing both cardiovascular and renal failure [2]. In AP, the respiratory system is primarily affected, followed by secondary involvement of the renal and cardiovascular systems [3]. The exact cause of respiratory failure in AP remains unclear. However, one potential mechanism is the direct injury of pulmonary vascular endothelial cells by trypsin released after pancreatic damage, leading to increased vascular permeability [4]. Additionally, trypsin can activate phospholipase A2, which damages pulmonary surfactant lecithin, accelerating its degradation and causing alveolar collapse [5].

Acute respiratory failure (ARF) is a serious complication in patients with AP. Several prediction models have been developed to assess the risk of acute respiratory distress syndrome (ARDS). For instance, the Lung Injury Prediction Score (LIPS) has been used to evaluate the ARDS risk in non-AP patients [6–8]. Moreover, the United States Critical Illness and Investigation Group developed and validated LIPS, which effectively identifies patients at higher risk of ARDS from various causes. Another model, the Early Acute Lung Injury (EALI) score, has been proposed to detect early ARDS in patients [6, 9–11]. Recent studies, including those by Lan Li et al., have introduced simplified models such as the simplified Lung Injury Prediction Score (sLIPS) and the simplified Early Acute Lung Injury score (sEALI), which may offer practical applications in predicting respiratory failure in AP patients [12]. However, the use of these models often requires certain exclusion criteria, such as excluding patients with respiratory failure, fluid overload, increased left atrial pressure, or congestive heart failure.

Emerging evidence suggests that insulin resistance (IR) plays a critical role in AP and its complications. Pancreatitis can lead to decreased pancreatic insulin secretion and increased peripheral IR. Studies have shown that IR-induced metabolic disturbances can exacerbate oxidative stress, intensify systemic inflammation, promote foam cell formation, impair endothelial function, and stimulate smooth muscle cell proliferation [13]. Given that the pathophysiology of ARF in AP involves diffuse alveolar damage, microvascular injury, and inflammatory cell infiltration [14], IR may serve as a crucial contributing factor. Additionally, persistent IR may elevate cardiac workload by increasing sympathetic nervous system activity, inducing renal sodium retention, and raising blood pressure, this mechanism could also contribute to the onset of ARF [15].

The triglyceride glucose (TyG) index, introduced by Luis E. Simental-Mendia et al., provides a simple and accessible method for evaluating insulin resistance (IR) [16]. Recent research has increasingly focused on exploring the relationship between the TyG index and AP. For instance, Jin Myung Park et al. investigated 373 AP patients and found that the TyG index was an independent predictor of severe AP [17], a finding supported by Wei Yimin et al. [18]. However, while the TyG index has been associated with AP severity, its relationship with AP-related complications, particularly ARF, remains largely unexplored.

This study aims to investigate the association between the TyG index and ARF in AP patients using the MIMIC-IV database. By evaluating the predictive value of the TyG index for ARF, our findings may provide novel insights into early risk stratification and targeted interventions for AP management.

Methods

Data selection

This study utilized a retrospective observational design, drawing upon data from the publicly accessible Medical Information Mart for Intensive Care IV 2.2 (MIMIC-IV) critical care dataset, specifically focusing on patients treated at the Beth Israel Deaconess Medical Center (BIDMC) between 2008 and 2019. The MIMIC-IV database includes data on both ICU and general ward patients, covering the entire hospital stay for each patient, from admission through discharge. The dataset contains a comprehensive range of variables, including demographic characteristics, laboratory results, vital signs, medication usage, treatments, and patient outcomes. Ethical considerations were paramount in our study. Approval for the project was obtained from the Institutional Review Boards (IRBs) of both MIT and BIDMC, with the requirement for informed consent waived due to the de-identified nature of the data. To ensure adherence to regulatory standards, researcher Ruxian Sun (Record ID: 61,773,273) obtained a Collaborative Institutional Training Initiative (CITI) license and the necessary permissions for accessing the MIMIC-IV database. The study rigorously adhered to the STROCSS guidelines.

Patients diagnosed with AP were included in this study based on the International Classification of Diseases, 9th and 10th Revisions. The exclusion criteria were as follows: (1) patients with multiple admissions for AP had only their data from the first admission extracted; (2) patients with a hospital stay of less than 24 h; (3) patients with severe diseases such as end-stage renal disease,

cirrhosis, or malignant tumors; (4) patients without sufficient data (TG and FBG) on the first day of admission; (5) patients with abnormal data, abnormal data were defined as values falling outside the clinically accepted range. The outliers removed included one patient with a height of 5 m and another with a weight of 4 tons; (6) patients with more than 20% missing data. Ultimately, a total of 758 patients were enrolled in this study and grouped into four groups based on the quartiles of the TyG index (Fig. 1).

Data collection

The data were collected using Structured Query Language (SQL) in conjunction with PostgreSQL version 14.2 to extract baseline patient characteristics. These encompassed demographic information (age, gender, body mass index (BMI)), vital signs (systolic blood pressure (SBP), diastolic blood pressure (DBP), saturation of peripheral oxygen (SpO_2)). Information on comorbidities based on the International Classification of Diseases, 9th and 10th Revisions was obtained from the MIMIC-IV database, include ARF, hyperlipidemia, diabetes, hypertension, cerebrovascular disease, congestive heart failure, renal disease, sepsis. Laboratory test results (including white blood cells (WBC), red blood cells (RBC), platelets, hemoglobin, sodium, calcium, international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (APTT), alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, creatinine, blood urea nitrogen (BUN), albumin, amylase, lipase, triglyceride (TG), fasting blood glucose (FBG) collected within the first 24 h of admission. The information regarding both the duration of hospitalization and in-hospital mortality was extracted concurrently. Additionally, the TyG index was calculated using the formula: Ln [Triglycerides (mg/dl) × Glucose (mg/dl)/2].

There is no consensus on the standard percentage of missing values for excluding variables from analysis. In our study, we chose a threshold of 60%, aligning with Zheng et al.'s practice of omitting variables with over 60% missing values in their analysis [19]. To handle missing data, we employed multiple imputation using a random forest algorithm, implemented through the 'mice' package in R software, where the algorithm was trained using other non-missing variables [20, 21].

Clinical outcomes

The endpoint of the study focused on patients with AP who were diagnosed with ARF according to the International Classification of Diseases, 9th and 10th Revisions, during their hospitalization.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) or median and interquartile range (IQR) according to their distribution, while categorical variables were presented as proportions. The normality of continuous parameters was assessed using the Kolmogorov-Smirnov test. Normally distributed continuous variables were analyzed using the t-test or ANOVA, whereas non-normally distributed variables were assessed using the Mann-Whitney U test or Kruskal-Wallis test.

In this study, our analysis was centered on patients diagnosed with AP, categorized into four groups (Q1: 6.62–8.42; Q2: 8.42–8.88; Q3: 8.88–9.63; Q4: 9.63–13.87) based on quartiles of the recorded TyG index. To investigate the association between the TyG index and ARF in patients with AP, we performed both univariable and multivariable logistic regression analyses. Odds ratio

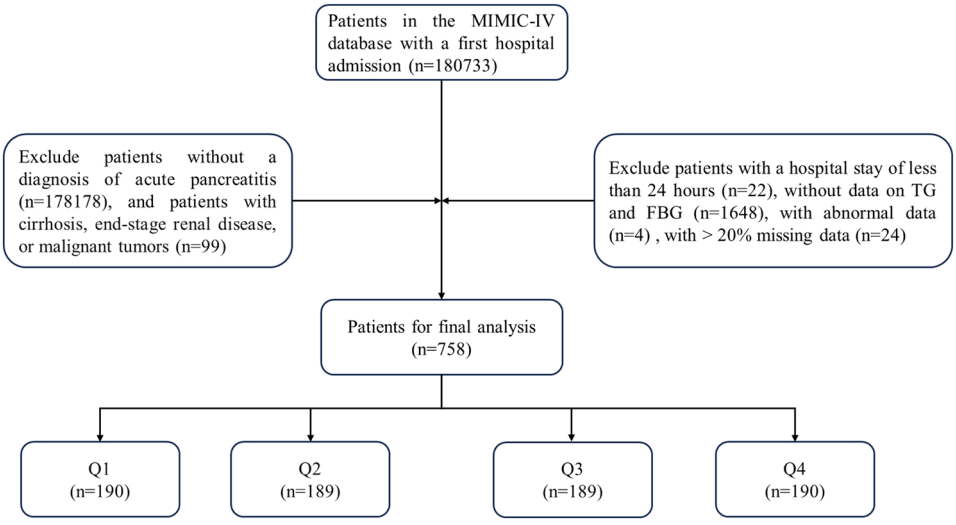

Fig. 1 Flow chart of patients selection for analytic

(OR) and 95% confidence interval (CI) were utilized as measures of rate estimates.

The multivariate analysis comprised three models: model 1 (unadjusted), model 2 (adjusted for age, gender, BMI), and model 3 (adjusted for age, gender, BMI, cerebrovascular disease, congestive heart failure, renal disease, hypertension, hyperlipidemia, diabetes, CAD, sepsis, WBC, RBC, platelet, hemoglobin, amylase, sodium, creatinine, lipase, heparin, pantoprazole, albumin, calcium, ALP, ALT, AST, BUN, bilirubin, INR, PT, APTT). The TyG index was included in the models both as a continuous variable and as categorical. The lowest quartile of the TyG index served as the reference group in all three models. P values for trends were computed based on quartile levels. We further analyzed the nonlinear association between the TyG index and ARF using a restricted cubic spline (RCS) regression model with five knots.

Furthermore, subgroup analyses were conducted to explore heterogeneity in the relationship between the TyG index and ARF across different subgroups. Stratified analyses were performed based on age (< 65 and ≥ 65 years), gender (female and male), BMI (< 30 and ≥ 30), hypertension (No and Yes), diabetes (No and Yes), CAD (No and Yes), hyperlipidemia (No and Yes), cerebrovascular disease (No and Yes), congestive heart failure (No and Yes), renal disease (No and Yes), and sepsis (No and Yes). Interactions between variables were assessed using the likelihood ratio test.

Additionally, we employed receiver operating characteristic (ROC) curve analysis to determine the optimal cutoff value for the TyG index and to compare its predictive performance for acute respiratory failure (ARF) in acute pancreatitis (AP) patients against FBG and TG. The area under the curve (AUC) was calculated for each parameter. Based on the cutoff value, patients were categorized into a Low TyG group (TyG index < cutoff value) and a High TyG group (TyG index ≥ cutoff value). Given the baseline differences between these groups in the original cohort, propensity score matching (PSM) was applied to minimize confounding effects. The R package Matching was used to generate propensity scores and perform 1:1 nearest neighbor matching with a caliper width of 0.05. Standardized mean differences (SMDs) were used to assess the balance of baseline characteristics after matching. McNemar's test was applied to compare categorical variables in the matched cohort, while paired analyses were conducted for continuous variables. To further control for confounders, inverse probability of treatment weighting (IPTW) was implemented using propensity scores derived from logistic regression via the R package WeightIt. Logistic regression analyses were conducted in the original cohort, the matched cohort (using conditional logistic regression), and the weighted cohort.

All analyses were performed using R software (version 4.3.3), and a two-tailed p-value < 0.05 was considered statistically significant.

Results

In total, 758 patients were enrolled in this study, 325 (42.88%) were female, and 433 (57.12%) were male. Their median age was 54 (IQR: 42–67) years. The median TyG index value was 8.88 (IQR: 8.42–9.63). The incidence of ARF was 21.64%.

Baseline characteristics

Table 1 presents the baseline characteristics of patients divided into quartiles based on the TyG index (Q1: 6.62–8.42; Q2: 8.42–8.88; Q3: 8.88–9.63; Q4: 9.63–13.87). The median TyG index of the four groups were 8.11 (IQR: 7.87–8.29), 8.65 (IQR: 8.55–8.76), 9.22 (IQR: 9.02–9.39), and 10.42 (IQR: 9.94–11.45), respectively. Among the patients in the Q4 group, a younger age and higher BMI at admission were observed. Additionally, they exhibited a higher incidence of diabetes, hypertension and sepsis. Furthermore, this group demonstrated higher levels of hemoglobin, AST, creatinine, BUN, TG, FBG, as well as lower levels of platelet, sodium, calcium, ALP, albumin. They also had a higher frequency of heparin and Pantoprazole use, a higher length of hospital stay and in-hospital mortality, compared with the lower TyG index group (all $P < 0.05$). Moreover, Q4 group accounted for a higher proportion of males. With a higher TyG index, there was a gradual increase in the incidence of ARF (9.47% vs. 14.29% vs. 22.75% vs. 40.00%; $P < 0.001$).

Table 2 presents a comparison of baseline characteristics between non-ARF patients and ARF patients. The ARF group had a higher BMI and showed a higher incidence of diabetes, hypertension, congestive heart failure, and sepsis, as well as greater use of heparin and pantoprazole. In terms of laboratory indicators, ARF patients had higher levels of WBC, sodium, INR, PT, APTT, AST, bilirubin, creatinine, BUN, TG, and FBG, but lower levels of RBC, calcium, and albumin (all $P < 0.05$). Additionally, the ARF group exhibited a significantly higher TyG index than the non-ARF group (9.50 [IQR: 8.82–10.22] vs. 8.76 [IQR: 8.34–9.38]; $P < 0.001$).

Relationship between TyG level and ARF risk

Table 3 presents the risk of ARF in patients categorized into different quartiles based on TyG levels. When analyzing the TyG index as a continuous variable, logistic regression analysis revealed a statistically significant association between ARF risk and the TyG index in unadjusted model (OR: 1.539; 95% CI: 1.342–1.769; $P < 0.001$), partially adjusted model (OR: 1.608; 95% CI: 1.387–1.872;

Table 1 Baseline characteristics of patients with acute pancreatitis grouped according to TyG index quartiles[a]

Categories	Overall (*N* = 758)	Q1 (*N* = 190)	Q2 (*N* = 189)	Q3 (*N* = 189)	Q4 (*N* = 190)	*P*-value
Demographic						
Age, years	54 (42–67)	56 (39–72)	58 (48–74)	54 (43–67)	50 (41–59)	< 0.001
Gender, n (%)						< 0.001
Female	325 (42.88%)	102 (53.68%)	83 (43.92%)	84 (44.44%)	56 (29.47%)	
Male	433 (57.12%)	88 (46.32%)	106 (56.08%)	105 (55.56%)	134 (70.53%)	
BMI, kg/m2	28.60 (24.63–32.88)	28.10 (23.92–31.87)	28.00 (24.50–32.50)	28.70 (25.20–33.20)	29.20 (25.82–33.68)	0.033
Vital signs						
SBP, mmHg	113 (104–125)	112 (102–123)	114 (104–126)	112 (104–125)	116 (104–126)	0.227
DBP, mmHg	70 (63–78)	69 (62–77)	70 (64–80)	70 (62–76)	71 (66–80)	0.010
SpO2, %	100 (90–100)	100 (100–100)	100 (93–100)	100 (89–100)	90 (85–100)	< 0.001
Laboratory tests						
WBC, K/uL	10.80 (7.60–15.60)	9.00 (6.45–13.17)	11.20 (8.50–16.00)	11.60 (8.20–16.60)	11.45 (7.90–16.40)	< 0.001
RBC, m/uL	3.98 (3.49–4.51)	3.96 (3.50–4.39)	4.09 (3.67–4.56)	3.84 (3.34–4.45)	4.08 (3.45–4.54)	0.022
Platelet, K/uL	226 (167–303)	229 (172–291)	245 (179–308)	234 (164–317)	209 (158–263)	0.011
Hemoglobin, g/dL	12.22 ± 2.22	11.91 ± 1.88	12.44 ± 1.94	11.92 ± 2.49	12.61 ± 2.42	0.002
Sodium, mEq/L	139 (136–141)	139 (137–141)	139 (137–141)	139 (136–141)	137 (134–141)	0.004
Calcium, mg/dL	8.40 (7.70–8.90)	8.40 (8.00-8.90)	8.60 (8.00-9.10)	8.50 (7.90–8.90)	7.80 (7.03–8.50)	< 0.001
INR	1.20 (1.10–1.40)	1.20 (1.10–1.40)	1.20 (1.10–1.40)	1.20 (1.10–1.40)	1.20 (1.10–1.30)	0.810
PT, s	13.50 (12.50–15.10)	13.60 (12.60-15.17)	13.60 (12.50–15.00)	13.60 (12.60–15.30)	13.20 (12.33–14.78)	0.352
APTT, s	29.10 (26.33–32.58)	29.35 (26.92–31.87)	29.00 (26.00-32.80)	28.10 (25.80–32.10)	29.80 (26.72–33.98)	0.130
ALP, IU/L	88 (62–141)	88 (62–150)	95 (64–142)	99 (66–149)	80 (56–114)	0.016
ALT, IU/L	39(20–113)	35 (18–127)	40 (18–136)	42 (23–95)	38 (21–93)	0.882
AST, IU/L	45 (24–115)	35 (22–115)	40 (22–96)	44 (26–111)	54 (28–149)	0.041
Bilirubin, mg/dL	0.80 (0.50–1.50)	0.70 (0.40–1.30)	0.80 (0.50–1.40)	0.80 (0.50–1.70)	0.80 (0.50–1.70)	0.324
Creatinine, mg/dL	0.90 (0.70–1.20)	0.80 (0.60-1.00)	0.80 (0.70–1.10)	0.90 (0.70–1.20)	1.00 (0.70–1.70)	< 0.001
BUN, mg/dL	13 (9–21)	11 (8–17)	13 (9–20)	15 (10–23)	17 (10–28)	< 0.001
Albumin, g/dL	3.30 (2.80–3.80)	3.40 (2.90–3.80)	3.40 (2.90–3.80)	3.30 (2.90–3.80)	3.00 (2.60–3.60)	< 0.001
Amylase, IU/L	126 (54–342)	139 (66–453)	142 (58–408)	110 (44–296)	112 (50–268)	0.003
Lipase, IU/L	205 (82–785)	186 (83–705)	234 (73–914)	194 (76–712)	227 (90–791)	0.461
TG, mg/dL	125 (85–218)	71 (56–86)	108 (88–124)	158 (130–202)	411 (261–1291)	< 0.001
FBG, mg/dL	113 (94–150)	92 (80–104)	108 (95–123)	123 (105–152)	169 (127–222)	< 0.001
TyG index	8.88 (8.42–9.63)	8.11 (7.87–8.29)	8.65 (8.55–8.76)	9.22 (9.02–9.39)	10.42 (9.94–11.45)	< 0.001
Comorbidities, n (%)						
Hyperlipidemia	196 (25.86%)	42 (22.11%)	48 (25.40%)	51 (26.98%)	55 (28.95%)	0.478
Diabetes	49 (6.46%)	5 (2.63%)	8 (4.23%)	11 (5.82%)	25 (13.16%)	< 0.001
Hypertension	409 (53.96%)	82 (43.16%)	108 (57.14%)	103 (54.50%)	116 (61.05%)	0.004
CAD	70 (9.23%)	18 (9.47%)	19 (10.05%)	17 (8.99%)	16 (8.42%)	0.955
Cerebrovascular disease	23 (3.03%)	4 (2.11%)	5 (2.65%)	11 (5.82%)	3 (1.58%)	0.072
Congestive heart failure	68 (8.97%)	18 (9.47%)	16 (8.47%)	17 (8.99%)	17 (8.95%)	0.990
Renal disease	75 (9.89%)	20 (10.53%)	19 (10.05%)	20 (10.58%)	16 (8.42%)	0.884
Sepsis	64 (8.44%)	12 (6.32%)	14 (7.41%)	13 (6.88%)	25 (13.16%)	0.059
CCI	3 (1–5)	3 (1–5)	3 (2–5)	3 (1–5)	3 (1–4)	0.442
Medication, n (%)						
Heparin	675 (89.05%)	155 (81.58%)	167 (88.36%)	175 (92.59%)	178 (93.68%)	< 0.001
Pantoprazole	292 (38.52%)	58 (30.53%)	77 (40.74%)	64 (33.86%)	93 (48.95%)	0.001
Events						
LOS hospital, days	7 (4–16)	5 (3–10)	7 (4–13)	9 (5–19)	11 (5–23)	< 0.001
Hospital death, n (%)	26 (3.43%)	2 (1.05%)	10 (5.29%)	2 (1.06%)	12 (6.32%)	0.004

Table 1 (continued)

Categories	Overall (*N* = 758)	Q1 (*N* = 190)	Q2 (*N* = 189)	Q3 (*N* = 189)	Q4 (*N* = 190)	*P*-value
Outcome						
ARF, n (%)	164 (21.64%)	18 (9.47%)	27 (14.29%)	43 (22.75%)	76 (40.00%)	< 0.001

[a] TyG index: Q1 (6.62–8.42), Q2 (8.42–8.88), Q3 (8.88–9.63), Q4 (9.63–13.87)

Continuous variables are presented as mean ± SD if normally distributed, and median (interquartile range) if not normally distributed. Categorical variables are presented as number of patients (%). TyG index: triglyceride glucose index; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; SpO2: pulse blood oxygen saturation; WBC: white blood cell; RBC: red blood cell; INR: international normalized ratio; PT: prothrombin time; APTT: activated partial thromboplastin time; ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; TG: triglyceride; FBG: fasting blood glucose; CAD: coronary artery disease; CCI: charlson comorbidity index; LOS: length of stay; ARF: acute respiratory failure

$P < 0.001$), and fully adjusted model (OR: 1.399; 95% CI: 1.144–1.713; $P = 0.001$). Additionally, when treating the TyG index as a nominal variable, the highest quartile (Q4) showed a significant association with ARF risk in unadjusted model (Q2 vs. Q1: [OR: 1.593; 95% CI: 0.851–3.046; $P = 0.150$]; Q3 vs. Q1: [OR: 2.814;95% CI: 1.579-5.200; $P < 0.001$]; Q4 vs. Q1: [OR: 6.370; 95% CI: 3.692–11.508; $P < 0.001$]), partially adjusted model (Q2 vs. Q1: [OR: 1.397; 95% CI: 0.738–2.699; $P = 0.309$]; Q3 vs. Q1: [OR: 2.772; 95% CI: 1.534–5.188; $P < 0.001$]; Q4 vs. Q1: [OR: 6.854; 95% CI: 3.845–12.774; $P < 0.001$]), and fully adjusted model [Q2 vs. Q1: [OR: 0.959; 95% CI: 0.434–2.151; $P = 0.917$]; Q3 vs. Q1: [OR: 2.661; 95% CI: 1.281–5.761; $P = 0.010$]; Q4 vs. Q1: [OR: 3.618; 95% CI: 1.710–7.993; $P = 0.001$]). Across all models, there was a consistent trend of increasing ARF risk with higher TyG index levels (all P for trend < 0.001).

Figure 2 illustrates the restricted cubic splines regression model, revealing a non-linear relationship between TyG levels and ARF risk across unadjusted, partially adjusted, and fully adjusted models (all P for nonlinear < 0.001).

Subgroup analysis

A subgroup analysis was performed to confirm the relationship between TyG level and ARF risk in subgroups stratified by age, gender, BMI, hypertension, diabetes, CAD, hyperlipidemia, cerebrovascular disease, congestive heart failure, renal disease and sepsis. The TyG index displayed a significant association with an increased risk of ARF in subgroups defined by age < 65 years (OR: 1.31; 95% CI: 1.06–1.63), age ≥ 65 years (OR: 1.90; 95% CI: 1.10–3.28), male (OR: 1.43; 95% CI: 1.13–1.82), BMI < 30 kg/m^2 (OR: 1.34; 95% CI: 1.04–1.71), BMI ≥ 30 kg/m^2 (OR: 1.46; 95% CI: 1.08–1.98), absence of hypertension (OR: 1.79; 95% CI: 1.26–2.53), absence of diabetes (OR: 1.47; 95% CI: 1.19–1.82), absence of CAD (OR: 1.37; 95% CI: 1.12–1.69), absence of hyperlipidemia (OR: 1.41; 95% CI: 1.13–1.77), absence of cerebrovascular disease (OR: 1.40; 95% CI: 1.14–1.72), absence of congestive heart failure (OR: 1.35; 95% CI: 1.10–1.67), absence of renal disease (OR: 1.42; 95% CI: 1.15–1.74), absence of sepsis (OR: 1.36; 95% CI: 1.10–1.68). Interaction analyses were conducted to examine the impact of the aforementioned covariates, revealing an interaction between the TyG index and diabetes (P for interaction = 0.034) (Fig. 3).

ROC analysis

The optimal cutoff value of the TyG index for predicting ARF in AP patients was determined to be 9.099, corresponding to a sensitivity of 67.7% and a specificity of 65.9%. The AUC values for the TyG index, TG, and FBG were 0.692 (95% CI: 0.648–0.737), 0.665 (95% CI: 0.619–0.712), and 0.660 (95% CI: 0.614–0.706), respectively. These findings suggest that the TyG index is a superior predictor of ARF in AP patients compared to TG or FBG alone (Fig. 4).

PSM analysis

Based on the result of the ROC curve analysis, 443 patients with a TyG index < 9.099 were classified into the Low TyG group, whereas the remaining 315 patients were classified into the High TyG group. Furthermore, PSM was conducted between the two groups. Prior to matching, disparities in age, gender, BMI, calcium, creatinine, BUN, albumin, diabetes and heparin existed between the groups. However, after matching, a significant reduction in imbalance was observed, resulting in a high degree of comparability in baseline variables between the two groups (Fig. 5). Table 4 summarizes the characteristics of the cohort before and after PSM. Following PSM, the incidence of ARF remained significantly higher in the High TyG group compared to the Low TyG group (24.27% vs. 14.56%; $P = 0.013$). Multivariate logistic regression analysis indicated a stronger correlation between the High TyG group and ARF compared to the Low TyG group (OR: 3.644; 95% CI: 2.186–6.185; $P < 0.001$). The association remained robust after PSM (OR: 2.925; 95% CI: 1.538–5.786; $P = 0.001$) and IPTW (OR: 3.393; 95% CI: 2.429–4.792; $P < 0.001$) (Fig. 6).

Discussion

This study represents the first retrospective investigation into the relationship between the TyG index and ARF in AP patients. By analyzing data from 758 patients extracted from the MIMIC-IV database, we identified a heightened risk of ARF in individuals with elevated TyG

Table 2 Baseline characteristics of the Non-ARF and ARF groups

Categories	Overall (*N* = 758)	Non-ARF (*N* = 594)	ARF (*N* = 164)	*P*-value
Demographic				
Age, years	54 (42–67)	54 (41–66)	55 (46–70)	0.047
Gender, n (%)				0.260
Female	325 (42.88%)	261 (43.94%)	64 (39.02%)	
Male	433 (57.12%)	333 (56.06%)	100 (60.98%)	
BMI, kg/m^2	28.60 (24.63–32.88)	28.50 (24.50–32.20)	29.10 (26.20–34.50)	0.007
Vital signs				
SBP, mmHg	113 (104–125)	113 (104–125)	115 (103–128)	0.570
DBP, mmHg	70 (63–78)	70 (63–78)	71 (66–80)	0.135
SpO_2, %	100 (90–100)	100 (94–100)	87 (80–90)	< 0.001
Laboratory tests				
WBC, K/uL	10.80 (7.60–15.60)	10.15 (7.20-13.78)	15.15 (10.67–19.83)	< 0.001
RBC, m/uL	3.98 (3.49–4.51)	4.00 (3.54–4.55)	3.87 (3.34–4.45)	0.044
Platelet, K/uL	226 (167–303)	230 (172–299)	213 (156–314)	0.104
Hemoglobin, g/dL	12.22 ± 2.22	12.28 ± 2.16	12.01 ± 2.41	0.168
Sodium, mEq/L	139 (136–141)	138 (136–141)	140 (136–142)	0.021
Calcium, mg/dL	8.40 (7.70–8.90)	8.50 (7.90-9.00)	7.80 (7.00-8.62)	< 0.001
INR	1.20 (1.10–1.40)	1.20 (1.10–1.30)	1.30 (1.20–1.50)	< 0.001
PT, s	13.50 (12.50–15.10)	13.30 (12.33–14.70)	14.30 (13.00–16.00)	< 0.001
APTT, s	29.10 (26.33–32.58)	29.00 (26.20–32.30)	29.80 (26.98–33.82)	0.049
ALP, IU/L	88 (62–141)	89 (62–148)	84 (64–118)	0.142
ALT, IU/L	39 (20–113)	38 (19–105)	42 (23–138)	0.078
AST, IU/L	45 (24–115)	40 (22–105)	66 (33–169)	< 0.001
Bilirubin, mg/dL	0.80 (0.50–1.50)	0.75 (0.50–1.40)	0.85 (0.50–1.72)	0.044
Creatinine, mg/dL	0.90 (0.70–1.20)	0.80 (0.60–1.08)	1.10 (0.70-2.00)	< 0.001
BUN, mg/dL	13 (9–21)	12 (8–18)	21 (14–35)	< 0.001
Albumin, g/dL	3.30 (2.80–3.80)	3.40 (2.90–3.88)	2.80 (2.40–3.10)	< 0.001
Amylase, IU/L	126 (54–342)	123 (54–336)	131 (55–396)	0.678
Lipase, IU/L	205 (82–785)	207 (83–772)	200 (78–892)	0.706
TG, mg/dL	125 (85–218)	118 (81–190)	192 (105–352)	< 0.001
FBG, mg/dL	113 (94–150)	109 (91–140)	137 (108–188)	< 0.001
TyG index	8.88 (8.42–9.63)	8.76 (8.34–9.38)	9.50 (8.82–10.22)	< 0.001
Comorbidities, n (%)				
Hyperlipidemia	196 (25.86%)	151 (25.42%)	45 (27.44%)	0.601
Diabetes	49 (6.46%)	30 (5.05%)	19 (11.59%)	0.003
Hypertension	409 (53.96%)	308 (51.85%)	101 (61.59%)	0.027
CAD	70 (9.23%)	53 (8.92%)	17 (10.37%)	0.572
Cerebrovascular disease	23 (3.03%)	15 (2.53%)	8 (4.88%)	0.120
Congestive heart failure	68 (8.97%)	34 (5.72%)	34 (20.73%)	< 0.001
Renal disease	75 (9.89%)	54 (9.09%)	21 (12.80%)	0.159
Sepsis	64 (8.44%)	25 (4.21%)	39 (23.78%)	< 0.001
CCI	3 (1–5)	3 (1–4)	4 (2–6)	< 0.001
Medication, n (%)				
Heparin	675 (89.05%)	514 (86.53%)	161 (98.17%)	< 0.001
Pantoprazole	292 (38.52%)	191 (32.15%)	101 (61.59%)	< 0.001
Events				
LOS hospital, days	7 (4–16)	6 (4–11)	21 (14–34)	< 0.001
Hospital death, n (%)	26 (3.43%)	6 (1.01%)	20 (12.20%)	< 0.001

Continuous variables are presented as mean ± SD if normally distributed, and median (interquartile range) if not normally distributed. Categorical variables are presented as number of patients (%). ARF: acute respiratory failure; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; SpO2: pulse blood oxygen saturation; WBC: white blood cell; RBC: red blood cell; INR: international normalized ratio; PT: prothrombin time; APTT: activated partial thromboplastin time; ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; TG: triglyceride; FBG: fasting blood glucose; TyG index: triglyceride glucose index; CAD: coronary artery disease; CCI: charlson comorbidity index; LOS: length of stay

Table 3 Multivariate logistic regression analyses of TyG index and incidence of ARF in patients with acute pancreatitis

Categories	Model 1		Model 2		Model 3	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Continuous variable per unit	1.539 (1.342–1.769)	< 0.001	1.608 (1.387–1.872)	< 0.001	1.399 (1.144–1.713)	0.001
Quartile[a]						
Q1 (*N* = 190)	Ref.		Ref.		Ref.	
Q2 (*N* = 189)	1.593 (0.851–3.046)	0.150	1.397 (0.738–2.699)	0.309	0.959 (0.434–2.151)	0.917
Q3 (*N* = 189)	2.814 (1.579-5.200)	< 0.001	2.772 (1.534–5.188)	< 0.001	2.661 (1.281–5.761)	0.010
Q4 (*N* = 190)	6.370 (3.692–11.508)	< 0.001	6.854 (3.845–12.774)	< 0.001	3.618 (1.710–7.993)	0.001
P for trend	< 0.001		< 0.001		< 0.001	

Model 1: unadjusted

Model 2: adjusted for age, gender, BMI, hyperlipidemia, diabetes

Model 3: adjusted for age, gender, BMI, cerebrovascular disease, congestive heart failure, renal disease, hypertension, hyperlipidemia, diabetes, CAD, sepsis, WBC, RBC, platelet, amylase, sodium, creatinine, lipase, heparin, pantoprazole, albumin, calcium, ALP, ALT, AST, BUN, bilirubin, INR

a TyG index: Q1 (6.62–8.42), Q2 (8.42–8.88), Q3 (8.88–9.63), Q4 (9.63–13.87)

TyG index: triglyceride glucose index; ARF: acute respiratory failure; OR: odds ratio; BMI: body mass index; WBC: white blood cell; RBC: red blood cell; INR: international normalized ratio; ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; CAD: coronary artery disease

index levels. Notably, even after adjusting for potential confounding variables, this correlation remained statistically significant. To reinforce the robustness of our findings, we determined the optimal cutoff point through ROC curve analysis, subsequently stratifying the population accordingly. Following this, we mitigated the impact of confounding factors on the outcomes using PSM and IPTW methods. Despite these adjustments, our results consistently demonstrated that surpassing the TyG index cutoff point nearly triples the risk of ARF development in patients. Importantly, this study introduced a straightforward approach to assess IR for improving risk stratification in critically ill patients with AP.

Previous studies have demonstrated a correlation between the TyG index and the severity of pancreatitis [17, 18]. Similarly, investigations had highlighted the respiratory system, particularly the lungs, as one of the commonly affected organ systems in SAP [22]. Consequently, our objective was to explore the relationship between the TyG index and AP complicated by ARF, aiming to offer a novel warning system for identifying early-stage ARF complica19tions in AP patients and facilitating timely clinical interventions. Encouragingly, our study revealed a persistent and statistically significant association between the TyG index and ARF across various models, including unadjusted, partially adjusted, and fully adjusted models. Furthermore, analysis using the RCS regression model supported this association and revealed a nonlinear trend.

Numerous studies have demonstrated the reliability and practicality of the TyG index as a surrogate marker for IR [16, 23–27]. Additionally, the TyG index is positively associated with TG levels. Recent studies indicate a significant increase in the incidence, severity, and recurrence of hypertriglyceridemic acute pancreatitis (HTG-AP) [28]. Hypertriglyceridemia exacerbates the severity and complications of AP in a dose-dependent manner, leading to elevated heart rate and maximum C-reactive protein (CRP) levels, highlighting the systemic inflammatory impact of high TG levels [29–32]. However, persistently elevated plasma triglycerides, even at mild to moderate levels, may predispose the pancreas to AP by exceeding the storage capacity in adipose tissue, resulting in TG accumulation, including within the pancreas. Pancreatic lipase can then release cytotoxic free fatty acids (FFA) from pancreatic tissue [33]. Given the pancreas's high protein production relative to its size, energy derived from β-oxidation of FFA can lead to the production of cytotoxic by-products, favoring inflammation [34]. Situated near metabolically active adipose tissue in the abdomen, the pancreas lacks protective fibrous capsules, making it more susceptible to reaching the "acute pancreatitis threshold" when additional stressors are present [35]. Thus, hypertriglyceridemia likely exacerbates the inflammatory response in AP, potentially leading to respiratory failure. This study suggests that the TyG index may offer a more physiologically plausible means of predicting AP complicated by respiratory failure.

In subgroup analysis, no significant association between the TyG index and ARF was observed in women or individuals with underlying conditions such as hypertension, diabetes, coronary heart disease, hyperlipidemia, cerebrovascular disease, heart failure, kidney disease, and sepsis. Notably, a significant interaction was observed between diabetes and the TyG index in the diabetic group, which strongly influenced the results. Firstly, the lack of significant correlation between the TyG index and ARF in the female population might be attributed to the estrogen-mediated reduction in IR. Studies have reported a significantly lower incidence of diabetes in individuals receiving estrogen replacement therapy compared to those on a placebo, suggesting that estrogen exerts anti-diabetic effects in women at risk of the disease [36, 37]. Moreover, 17β estradiol (E2), a gonadal

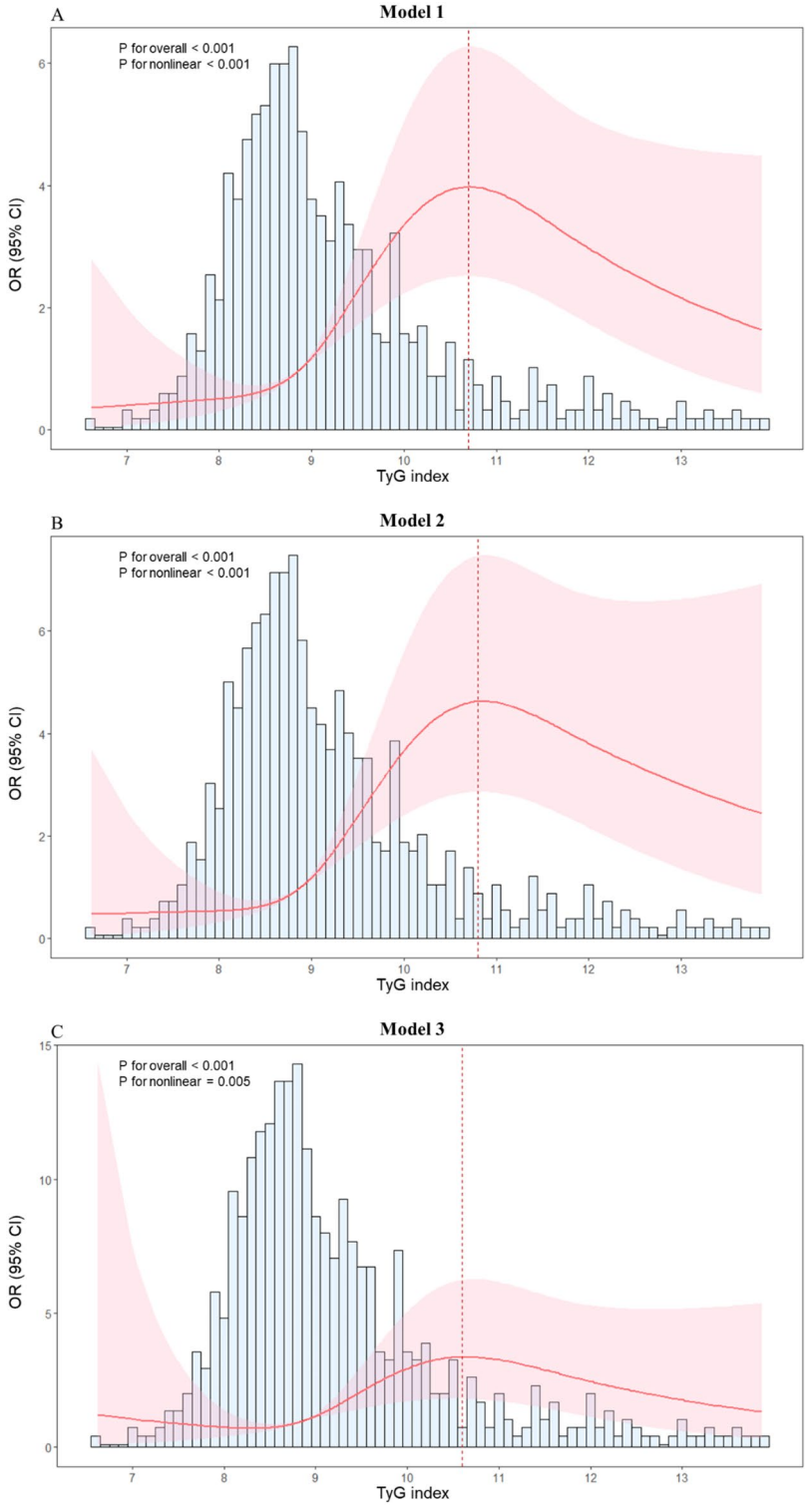

Fig. 2 Restricted cubic spline regression analysis of TyG index with ARF. The heavy central lines represent the estimated adjusted odds ratios, with shaded ribbons indicating the corresponding 95% confidence intervals. The histogram illustrates the distribution of patients. The dashed line marks the inflection point derived from threshold effect analysis of the TyG index on ARF in acute pancreatitis patients. A: Model 1 was unadjusted. B: Model 2 adjusted for age, gender, BMI, hyperlipidemia, diabetes. C: Model 3 adjusted for age, gender, BMI, cerebrovascular disease, congestive heart failure, renal disease, hypertension, hyperlipidemia, diabetes, CAD, sepsis, WBC, RBC, platelet, amylase, sodium, creatinine, lipase, heparin, pantoprazole, albumin, calcium, ALP, ALT, AST, BUN, bilirubin, INR. TyG index: triglyceride glucose index; ARF: acute respiratory failure; OR: odds ratio; BMI: body mass index; CAD: coronary artery disease; WBC: white blood cell; RBC: red blood cell; ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: urea nitrogen; INR: international normalized ratio

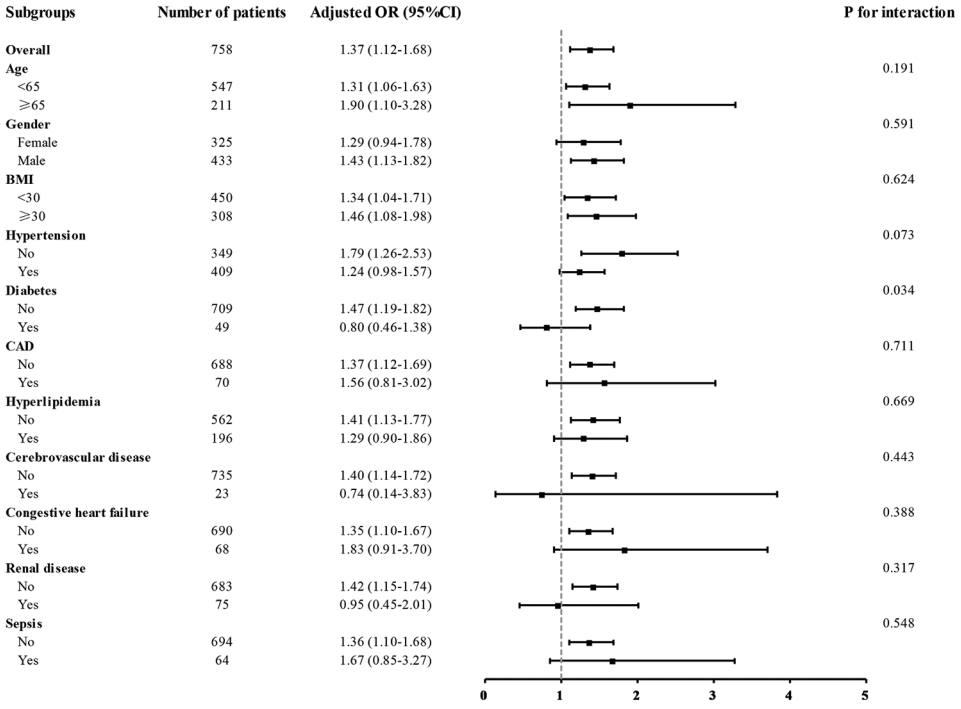

Fig. 3 Subgroup analyses for the association of TyG index with ARF. OR: odds ratio; CI: confidence interval; BMI: body mass index; CAD: coronary artery disease; TyG index: triglyceride glucose index; ARF: acute respiratory failure

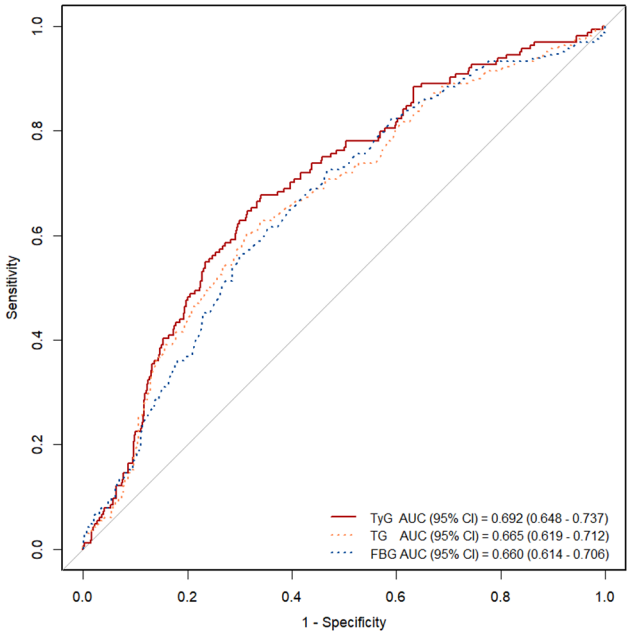

Fig. 4 ROC curve for TyG, TG and FBG. AUC: area under the curve; CI: confidence interval; ROC: receiver operating characteristic; TyG: triglyceride glucose index; TG: triglyceride; FBG: fasting blood glucose

hormone, has been identified as a crucial hormonal signal for energy homeostasis, promoting islet adaptation to metabolic stress, enhancing islet cell survival, maintaining lipid homeostasis, and stimulating glucose-stimulated insulin biosynthesis and secretion. Consequently, E2 confers protection against pancreatic β-cell failure in most rodent models of diabetes [38]. Furthermore, E2 can reverse menopause-induced alterations in glucose and insulin metabolism, leading to increased insulin secretion and reduced IR, whereas androgen progestins may counteract this potentially beneficial IR effect [39]. This elucidates why the TyG index is more applicable to male patients than female patients. Elevated TG and FBG levels may occur in patients with the aforementioned underlying conditions, particularly in diabetic patients where blood glucose levels are significantly elevated during AP onset, thereby significantly interfering with the TyG index. Consequently, no significant association between the TyG index and ARF was discerned.

This study has several limitations. First, as a single-center retrospective design, it cannot establish causal relationships definitively. Although multivariable adjustments and subgroup analyses were performed, residual confounding factors may still affect the results. Second, the reported blood glucose and lipid levels correspond to the initial measurements taken after admission, and it is unclear whether these were obtained from fasting patients. While the database indicates whether patients have been diagnosed with hyperlipidemia, it does not differentiate between types of lipid abnormalities, such as hypertriglyceridemia. Moreover, the triglyceride levels extracted reflect current values during hospitalization rather than historical levels, which may not accurately represent a patient's history of hypertriglyceridemia.

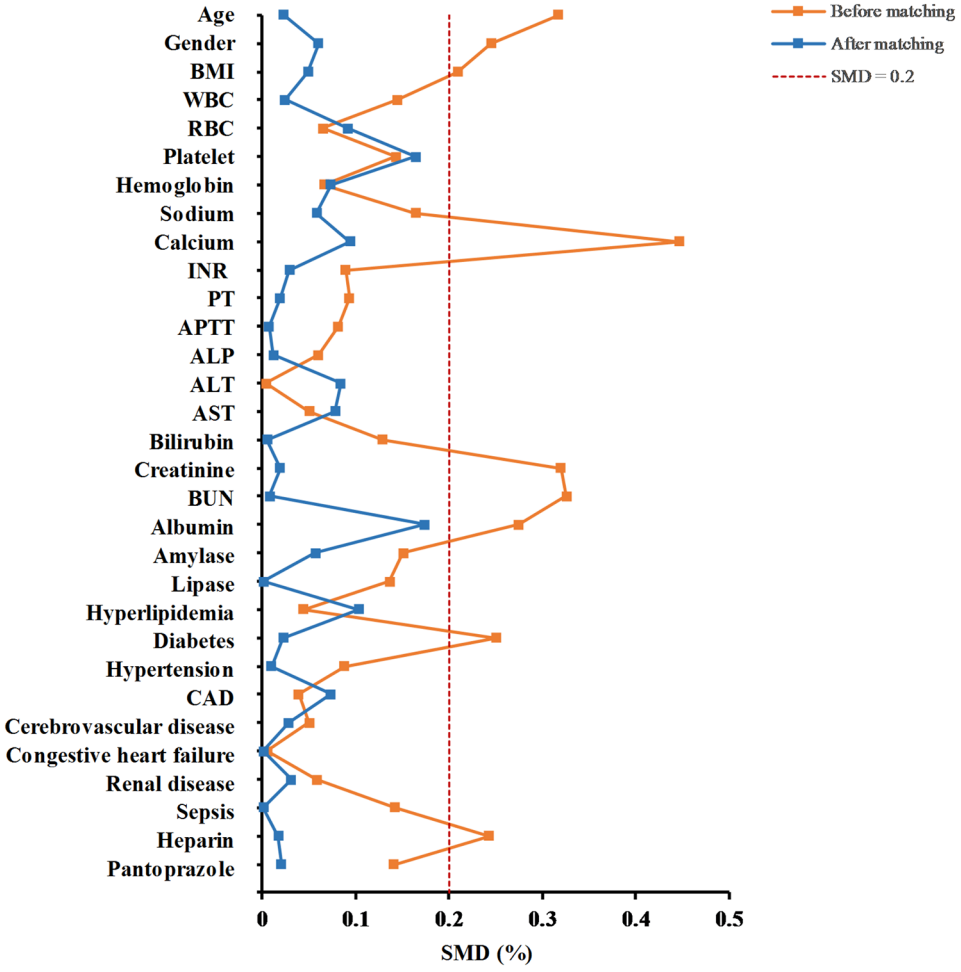

Fig. 5 The equilibrium of each variable was assessed following the propensity score matching analysis. The standardized mean differences of all variables were presented. SMD: standardized mean difference

Third, the MIMIC-IV database lacks detailed information on the etiology of acute pancreatitis (e.g., biliary, alcoholic, or hypertriglyceridemia-induced), which may introduce bias, as different etiologies could influence both the severity of pancreatitis and the risk of acute respiratory failure. Additionally, the limited sample size and the low number of deaths in the cohort hinder the ability to conduct an effective Cox regression analysis to assess the impact of TyG on the prognosis of AP patients or those with AP complicated by ARF. The retrospective nature of the study also prevented precise determination of the timing of respiratory failure onset, and reliance on final diagnosis data may have affected the accuracy of the results. Finally, this study analyzed only baseline TyG index levels and did not consider dynamic changes during hospitalization, either in general wards or the ICU. Therefore, future studies should explore the predictive value of changes in the TyG index.

Conclusion

In summary, we propose that metabolic abnormalities and systemic inflammatory responses may underlie the relationship between the TyG index and ARF. During the pathogenesis of AP, these factors can lead to pancreatic dysfunction and systemic organ damage, ultimately culminating in ARF. Our findings propose the TyG index as a potential marker for assessing ARF risk in AP patients. Future studies could delve into the association between the TyG index and other pancreatitis complications, as well as explore the potential benefits of correcting metabolic abnormalities to enhance patient outcomes. Furthermore, clinical trials are needed to validate the TyG index's efficacy in ARF risk assessment, representing a crucial avenue for future research.

Table 4 Baseline characteristics between the low TyG[a] and high TyG[b] groups before and after PSM

Categories	Original Cohort		*P*-value	Matched Cohort		*P*-value
	Low TyG (*N* = 443)	High TyG (*N* = 315)		Low TyG (*N* = 206)	High TyG (*N* = 206)	
Demographic						
Age, years	57 (43–71)	50 (41–61)	< 0.001	53 (37–66)	52 (42–63)	0.873
Gender, n (%)			0.001			0.544
Female	212 (47.86%)	113 (35.87%)		83 (40.29%)	77 (37.38%)	
Male	231 (52.14%)	202 (64.13%)		123 (59.71%)	129 (62.62%)	
BMI, kg/m^2	28.00 (24.40–32.00)	29.20 (25.60–33.70)	0.002	28.35 (24.60-32.08)	28.70 (25.55–32.80)	0.258
Vital signs						
SBP, mmHg	113.00 (103–125)	114 (104–126)	0.404	112 (102–124)	113 (105–125)	0.328
DBP, mmHg	70 (63–78)	70 (64–78)	0.504	70 (64–78)	70 (62–77)	0.398
SpO_2, %	100 (94–100)	91 (87–100)	< 0.001	100 (91–100)	100 (88–100)	0.006
Laboratory tests						
WBC, K/uL	10.40 (7.45–15.15)	11.40 (7.85–16.45)	0.055	11.70 (7.32–16.10)	11.20 (7.80–15.90)	0.844
RBC, m/uL	4.00 (3.58–4.54)	3.95 (3.43–4.50)	0.182	3.99 ± 0.73	4.06 ± 0.80	0.359
Platelet, K/uL	233 (174–308)	215 (157–290)	0.016	218 (162–292)	229 (171–308)	0.109
Hemoglobin, g/dL	12.16 ± 2.00	12.31 ± 2.49	0.362	12.22 ± 2.05	12.38 ± 2.43	0.459
Sodium, mEq/L	139 (137–141)	138 (135–141)	0.002	139 (137–141)	138 (135–141)	0.356
Calcium, mg/dL	8.50 (8.00–9.00)	8.10 (7.35–8.70)	< 0.001	8.30 (7.70–8.80)	8.40 (7.70–8.90)	0.282
INR	1.20 (1.10–1.40)	1.20 (1.10–1.30)	0.267	1.20 (1.10–1.40)	1.20 (1.10–1.30)	0.205
PT, s	13.60 (12.55–15.20)	13.30 (12.40–14.90)	0.080	13.60 (12.70-15.28)	13.30 (12.20-14.78)	0.107
APTT, s	29.20 (26.40–32.30)	29.00 (26.10-33.35)	0.817	29.00 (26.80-32.05)	28.60 (26.05–32.30)	0.603
ALP, IU/L	95 (64–148)	84 (59–128)	0.018	88 (62–143)	83 (58–130)	0.206
ALT, IU/L	36 (18–129)	41 (21–98)	0.606	40 (19–112)	39 (21–92)	0.610
AST, IU/L	39 (22-105.50)	52 (28–129)	0.004	47 (24–117)	45 (24–109)	0.799
Bilirubin, mg/dL	0.70 (0.50–1.40)	0.80 (0.50–1.70)	0.164	0.80 (0.50–1.60)	0.80 (0.50–1.40)	0.337
Creatinine, mg/dL	0.80 (0.60-1.00)	1.00 (0.70–1.60)	< 0.001	0.80 (0.60–1.10)	0.90 (0.70–1.28)	0.015
BUN, mg/dL	12 (8–19)	15 (10–26)	< 0.001	13 (8–21)	14 (9–23)	0.205
Albumin, g/dL	3.40 (2.90–3.80)	3.10 (2.70–3.65)	< 0.001	3.20 (2.80–3.70)	3.35 (2.82–3.80)	0.134
Amylase, IU/L	137 (63–421)	108 (46–269)	< 0.001	124 (65–324)	106 (40–218)	0.008
Lipase, IU/L	202 (73–843)	209 (88–725)	0.976	186 (76–647)	188 (84–710)	0.652
TG, mg/dL	92 (70–119)	254 (186–446)	< 0.001	94 (72–122)	225 (169–347)	< 0.001
FBG, mg/dL	102 (86–121)	146 (113–203)	< 0.001	101 (86–117)	141 (112–199)	< 0.001
TyG index	8.50 (8.18–8.76)	9.87 (9.39–10.68)	< 0.001	8.52 (8.20–8.77)	9.65 (9.34–10.31)	< 0.001
Comorbidities, n (%)						
Hyperlipidemia	111 (25.06%)	85 (26.98%)	0.550	44 (21.36%)	53 (25.73%)	0.296
Diabetes	17 (3.84%)	32 (10.16%)	< 0.001	11 (5.34%)	10 (4.85%)	0.823
Hypertension	231 (52.14%)	178 (56.51%)	0.235	110 (53.40%)	111 (53.88%)	0.921
CAD	43 (9.71%)	27 (8.57%)	0.595	14 (6.80%)	18 (8.74%)	0.462
Cerebrovascular disease	15 (3.39%)	8 (2.54%)	0.503	7 (3.40%)	6 (2.91%)	0.778
Congestive heart failure	40 (9.03%)	28 (8.89%)	0.947	18 (8.74%)	18 (8.74%)	0.995
Renal disease	47 (10.61%)	28 (8.89%)	0.434	24 (11.65%)	22 (10.68%)	0.754
Sepsis	30 (6.77%)	34 (10.79%)	0.050	20 (9.71%)	20 (9.71%)	0.996
CCI	3 (1–5)	3 (1–4)	0.477	2 (1–4)	3 (1–4)	0.310
Medication, n (%)						
Heparin	381 (86.00%)	294 (93.33%)	0.001	187 (90.78%)	188 (91.26%)	0.863
Pantoprazole	158 (35.67%)	134 (42.54%)	0.055	77 (37.38%)	79 (38.35%)	0.839
Events						
LOS hospital, days	6 (4–12)	10 (5–22)	< 0.001	7 (4–14)	8 (4–21)	0.082
Hospital death, n (%)	13 (2.93%)	13 (4.13%)	0.374	8 (3.88%)	9 (4.37%)	0.804

Table 4 (continued)

Categories	Original Cohort		*P*-value	Matched Cohort		*P*-value
	Low TyG (*N* = 443)	High TyG (*N* = 315)		Low TyG (*N* = 206)	High TyG (*N* = 206)	
Outcome						
ARF, n (%)	53 (11.96%)	111 (35.24%)	< 0.001	30 (14.56%)	50 (24.27%)	0.013

[a] Low TyG: TyG index < 9.099; [b] High TyG: TyG index ≥ 9.099

Continuous variables are presented as mean ± SD if normally distributed, and median (interquartile range) if not normally distributed. Categorical variables are presented as number of patients (%). TyG index: triglyceride glucose index; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; SpO2: pulse blood oxygen saturation; WBC: white blood cell; RBC: red blood cell; INR: international normalized ratio; PT: prothrombin time; APTT: activated partial thromboplastin time; ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; TG: triglyceride; FBG: fasting blood glucose; CAD: coronary artery disease; CCI: Charlson comorbidity index; LOS: length of stay; ARF: acute respiratory failure

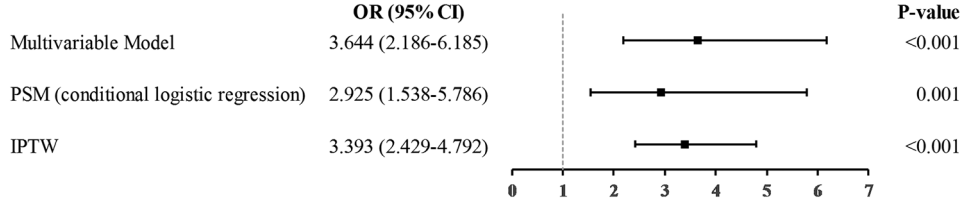

Fig. 6 The association between the High TyG group and ARF in comparison to the Low TyG group. Low TyG: TyG index < 9.099; High TyG: TyG index ≥ 9.099. OR: odds ratio; CI: confidence interval; PSM: propensity score matching, IPTW: inverse probability of treatment weighting; ARF: acute respiratory failure

Abbreviations

TyG	Triglyceride glucose
AP	Acute pancreatitis
ARF	Acute respiratory failure
IR	Insulin resistance
MIMIC-IV	Medical Information Mart for Intensive Care IV
RCS	Restricted cubic spline
ROC	Receiver operating characteristic
PSM	Propensity score matching
IPTW	Inverse probability of treatment weighting
IQR	Interquartile range
CITI	Collaborative Institutional Training Initiative
SQL	Structured Query Language
BMI	Body mass index
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
SpO2	Saturation of peripheral oxygen
WBC	White blood cells
RBC	Red blood cells
INR	International normalized ratio
PT	Prothrombin time
APTT	Activated partial thromboplastin time
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BUN	Blood urea nitrogen
TG	Triglyceride
FBG	Fasting blood glucose
SD	Standard deviation
OR	Odds ratio
CI	Confidence interval
AUC	Area under the curve
CAD	Coronary artery disease
SMD	Standardized mean difference
MODS	Multiple organ dysfunction syndrome
SAP	Severe acute pancreatitis
LIPS	Lung Injury Prediction Score
EALI	Early Acute Lung Injury
ARDS	Acute respiratory distress syndrome
HTG-AP	Hypertriglyceridemic acute pancreatitis
CRP	C-reactive protein
FFA	Free fatty acids
17β	Estradiol E2

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Author contributions
Jiao Lv designed the study. Ruxian Sun extracted and analyzed the data. Jiao Lv, Yuanjun Zhou and Changyan Tao drafted the manuscript. Yan Cai, Hongfeng Yang, Juan Xu and Jun Chen revised the manuscript. The final version was approved by all authors.

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Data availability
The datasets analysed during the current study are available in the MIMIC-IV repository, https://physionet.org/content/mimiciv/2.2/.

Declarations

Ethics approval and consent to participate
The data was extracted from Medical Information Mart for Intensive Care IV (MIMIC-IV, Version 2.2). The identification information was concealed and privacy of patients in MIMIC-IV were protected. Thus, there were no additional consent procedures from institutional ethics committee.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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