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The application of the triglyceride-glucose-body mass index (TyG-BMI) in predicting acute kidney injury in diabetic patients following coronary artery bypass grafting surgery

Chen Li[1†], Xiaobin Liu[1†], Xingping Lv[1†], Wei Zhou[1], Guoliang Fan[1] and Feng Zhu[1*]

Abstract

Background The triglyceride-glucose-body mass index (TyG-BMI), a marker for insulin resistance, is recognized for its predictive role in cardiovascular and metabolic diseases, including kidney disease. we explored the TyG-BMI index's association with postoperative kidney injury in coronary artery bypass grafting (CABG) patients, who are at an elevated risk for such complications, underscoring its potential as a predictor for acute kidney injury (AKI).

Methods This single-center, retrospective study included 126 patients. Patients were divided into AKI and non-AKI groups postoperatively according to the KDIGO classification criteria. Univariate logistic regression was used to screen for variables with significant differences ($P<0.01$), and multiple multivariate regression models were constructed to analyze independent risk factors in the multivariate regression model and to analyze the value of TyG-BMI in predicting AKI in diabetic patients after CABG.

Results Compared to the non-AKI group, the AKI group had statistically significant differences in preoperative fasting triglycerides, preoperative fasting glucose, preoperative and postoperative creatinine levels, ICU stay duration, and TyG-BMI levels ($P<0.05$). Based on the results of univariate regression analysis, a multivariate logistic regression model A was constructed using all significant variables, and a multivariate logistic regression model 2 was constructed using significant variables other than TyG-BMI. ROC analysis showed that model 2 had better predictive performance than model 1 (AUC = 0.836 vs. 0.766). A positive correlation was observed between TyG-BMI and AKI occurrence (Spearman's correlation coefficient: $R=0.33$, $P=0.00019$).

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Conclusion Elevated TyG-BMI levels are closely associated with AKI in diabetic patients after CABG. TyG-BMI has potentially predictive value for AKI in diabetic patients after CABG and may play a crucial role in risk stratification in clinical practice.

Keywords Triglyceride-glucose, Body mass index, Coronary artery bypass grafting, Acute kidney injury, Insulin resistance

Introduction

Coronary artery bypass grafting (CABG) is an effective treatment for patients with coronary artery disease (CAD) [1]. However, approximately one-third of patients undergoing CABG develop acute kidney injury (AKI) [2]. Clinically, kidney function is primarily assessed using serum creatinine, glomerular filtration rate, and other biochemical markers, which may not promptly indicate the onset of AKI [3]. Obesity is a global public health issue associated with various cardiovascular diseases [4], including hypertension, insulin resistance, and diabetes [5]. Despite normal serum biochemistry in obese patients, they are prone to AKI due to a higher burden of comorbidities and potential structural changes in the kidneys [6, 7]. Traditional assessments of insulin resistance (IR), such as the hyperinsulinemic-euglycemic clamp and the homeostatic model assessment of IR, are complex and time-consuming, limiting their application in research settings. The triglyceride-glucose (TyG) index, first proposed by South American researchers, has shown good correlation with both hyperinsulinemic-euglycemic clamp and homeostatic model assessment of IR. This index is easier to obtain and less costly than traditional IR assessments [8]. The triglyceride-glucose-body mass index (TyG-BMI), formally introduced in 2016, incorporates BMI as an obesity metric, enhancing predictability. Studies have found that TyG-BMI has the strongest correlation with homeostatic model assessment of IR compared to various biomarkers, suggesting it has greater diagnostic value than the TyG index alone [9]. Recently, TyG-BMI has been validated as effective in assessing atherosclerosis, prehypertension, and prediabetes, making it a burgeoning research area [10]. Few studies have examined the impact of obesity on the incidence of AKI in diabetic patients post-CABG. Therefore, our aim is to investigate the effect of the TyG-BMI index on AKI in diabetic patients undergoing CABG.

Materials and methods

General information

Data were collected from 126 diabetic patients (91 males and 35 females) who underwent CABG at Shanghai East Hospital from January 2022 to December 2023. Inclusion Criteria: 1. Age ≥ 18 years with complete clinical data.2. Underwent CABG after admission. 3. Diabetics Exclusion Criteria: Patients with chronic renal insufficiency receiving peritoneal dialysis or hemodialysis, or those with missing important clinical data.Participants were divided into two groups based on the occurrence of postoperative AKI: the AKI group ($n = 67$) and the non-AKI group ($n = 59$) (Fig. 1). AKI was defined according to the newest consensus-based KDIGO criteria as follows: small changes in serum creatinine (≥ 0.3 mg/dl or 26.5 mmol/l) when they occurred within 48 h or a maximal change in serum creatinine ≥ 1.5 times the baseline value until postoperative day 7 compared with preoperative baseline values or urine volume < 0.5 ml/kg/h for 6 h [11].

Data collection

Clinical data were collected from the electronic medical record system. General Information: gender, age, height, weight. Medical History: hypertension, diabetes, coronary artery disease, cardiac function classification, atrial fibrillation, hyperlipidemia, myocardial infarction, chronic lung disease, and chronic renal insufficiency. Clinical Data: white blood cell count, neutrophil percentage, hemoglobin, platelet count, total bilirubin, albumin, creatinine, triglycerides, fasting blood glucose, left atrial diameter, left ventricular end-diastolic diameter, ejection fraction, Euroscore, ICU length of stay and Mechanical ventilation. Calculations: BMI: BMI = weight (kg) / height (m)^2. TyG Index: TyG = Ln[fasting triglycerides (mg/dL) × fasting glucose (mg/dL) / 2] [12]. TyG-BMI Index: TyG-BMI = TyG index × BMI (kg/m^2) [9].

Statistical analysis

Statistical analysis was performed using R software version 4.4.0 and SPSS version 29.0. The one-sample Kolmogorov-Smirnov (K-S) test (two-tailed) was used to assess whether the measurement data conformed to a normal distribution. Measurement data with a normal distribution are presented as mean ± standard deviation (SD). The t-test was used for comparisons between two groups with normally distributed data. Measurement data with a non-normal distribution are presented as median (Q1, Q3), and the Mann-Whitney U test was used to compare the means between the two groups. The chi-square test was used for comparing categorical data.Univariate logistic regression was used to identify variables with significant differences ($P < 0.01$). These significant variables were then included in multiple multivariate regression models to analyze independent risk factors. The value of TyG-BMI in predicting acute kidney injury (AKI) in diabetic patients' post-CABG was

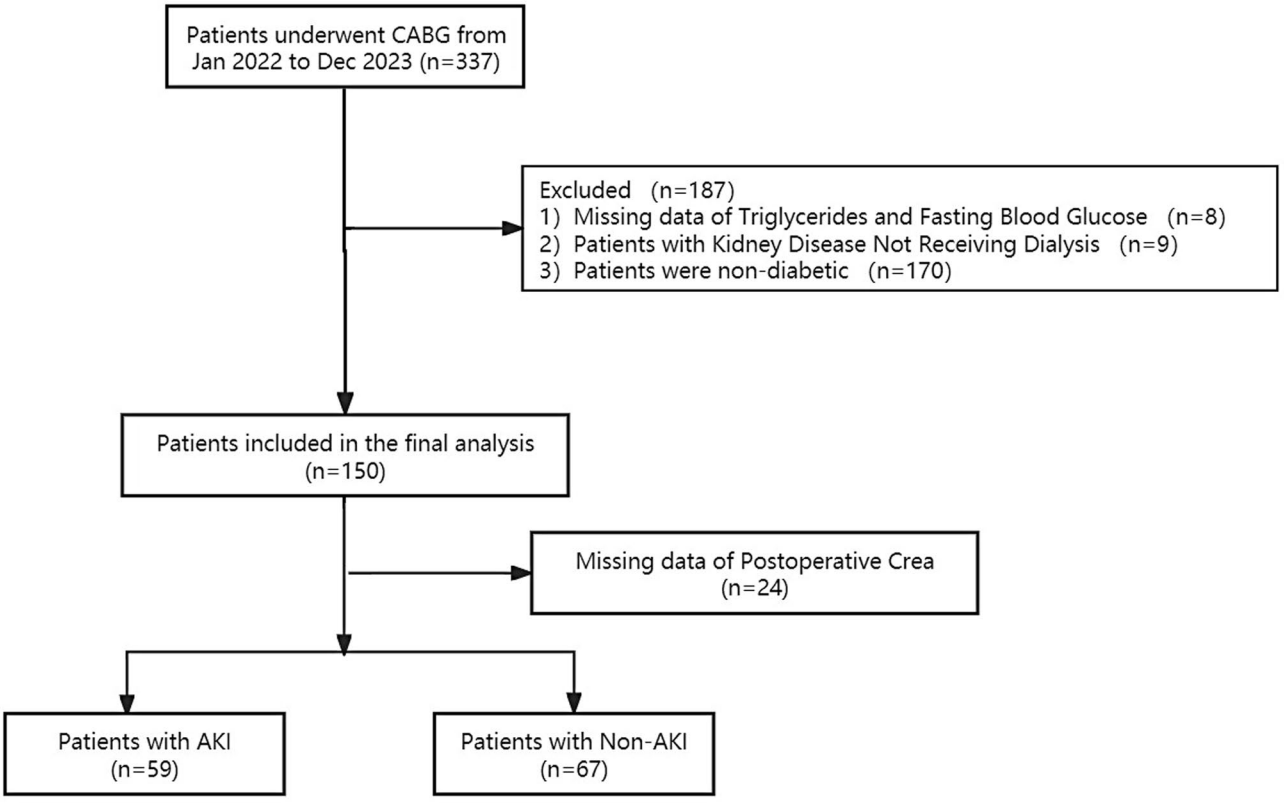

Fig. 1 Flow diagram of patient selection

analyzed using these multivariate regression models. A P-value of <0.05 was considered statistically significant. Pearson's correlation analysis was used to describe the association between TyG-BMI and AKI.

Results

General information

A total of 126 diabetic patients who underwent CABG were included in this study. The baseline clinical characteristics and laboratory measurements of the patients are shown in Table 1. The patients were divided into two groups based on the occurrence of postoperative AKI. There were statistically significant differences between the preoperative fasting triglycerides, preoperative fasting blood glucose, ICU stay duration, preoperative creatinine, postoperative creatinine, and TyG-BMI ($P<0.05$). Subjects in the AKI group had a significantly higher TyG-BMI relative to subjects in the non-AKI group (191.27 [166.00, 207.84] vs. 108.62 [155.11, 192.26], $P<0.001$). There were no statistically significant differences between the groups in terms of gender, age, BMI, history of hypertension, NYHA classification, history of coronary artery disease, history of atrial fibrillation, history of hyperlipidemia, history of myocardial infarction, history of chronic lung disease, history of chronic renal insufficiency, Euroscore, preoperative white blood cell count, preoperative left atrial diameter, preoperative ejection fraction (EF%), preoperative neutrophil percentage, preoperative hemoglobin, preoperative platelet count, preoperative total bilirubin, preoperative albumin, preoperative left ventricular end-diastolic diameter, and mechanical ventilation time ($P>0.05$).

Univariate logistic regression analysis

Univariate logistic regression analysis indicated that preoperative albumin level, history of chronic kidney disease, preoperative white blood cell count, preoperative creatinine level, postoperative creatinine level, ICU stay duration, and TyG-BMI were risk factors for AKI in diabetic patients after CABG (Table 2).

Comparison of multivariate logistic regression models

Multivariate logistic regression model A analysis showed that TyG-BMI (OR 1.03, 95% CI 1.02–1.06, $P<0.001$), preoperative creatinine level (OR 0.94, 95% CI 0.90–0.98, P =0.007), and postoperative creatinine (D2) (OR 1.04, 95% CI 1.01–1.07, P =0.013) were significant risk factors for AKI in diabetic patients post-CABG, and multivariate logistic regression model B analysis showed that preoperative creatinine level (OR 0.95, 95% CI 0.92–0.99, P =0.019) and postoperative creatinine (D2) (OR 1.04, 95% CI 1.01–1.07, P =0.02) (Fig. 2). Based on the results

Table 1 Comparison of baseline data and clinical indicators between two groups

	Overall population		
Variable	**Non-AKI Group (*n* = 59)**	**AKI Group (*n* = 67)**	**P value**
Male (%)	42 (71.1)	49 (73.1)	0.844
Age (years) (median[IQR])	67.50 [60.00, 74.00]	68.00 [56.75, 76.25]	0.910
BMI (kg/m^2) (median[IQR])	23.90 [22.00, 25.10]	24.10 [21.80, 27.20]	0.391
NYHA (%)			0.863
I	9 (52.9)	8(47.0)	
II	23 (41.0)	33 (58.9)	
III	12 (42.8)	16 (57.1)	
IV	1 (33.3)	2(66.7)	
Hypertension (%)	38 (64.4)	51 (76.1)	0.173
CAD (%)	56 (94.9)	66 (98.5)	0.340
AF (%)	3 (5.0)	3 (4.5)	1.000
Hyperlipidemia (%)	4 (6.7)	4 (5.9)	1.000
MI (%)	4 (6.7)	9 (13.4)	0.254
Chronic lung disease (%)	5 (8.5)	1 (1.5)	0.098
Chronic kidney disease (%)	2 (3.3)	8 (11.9)	0.102
Euroscore (median [IQR])	5.00 [4.00, 7.00]	6.00 [4.00, 7.00]	0.435
White blood cell count (x10^9/L) (median [IQR])	6.32 [5.07, 8.05]	6.75 [5.51, 7.98]	0.122
Neutrophil percentage (%) (median [IQR])	61.60 [53.50, 66.20]	62.90 [54.60, 67.70]	0.238
Hemoglobin (g/L) (median [IQR])	131.00 [115.00, 138.00]	123.00 [109.00, 138.00]	0.103
Platelet (x10^9/L) (median [IQR])	201.00 [165.00, 241.00]	195.00 [156.00, 248.00]	0.845
TBil (μmol/L) (median [IQR])	10.20 [7.20, 13.20]	10.10 [7.30, 13.20]	0.989
ALB (g/L) (median [IQR])	40.07 [37.30, 43.20]	38.87 [35.74, 41.90]	0.118
LAD (mm) (median [IQR])	37.00 [35.00, 41.00]	38.00 [36.00, 42.00]	0.205
LVEDD (mm) (median [IQR])	47.00 [44.00, 50.00]	48.00 [43.00, 52.00]	0.512
LVEF (%) (median [IQR])	62.00 [48.00, 67.00]	60.00 [51.00, 65.00]	0.496
TG (mmol/L) (median [IQR])	1.17 [0.94, 1.64]	1.63 [1.22, 2.42]	< 0.001
FBG (mmol/L) (median [IQR])	5.90 [4.70, 7.20]	8.40 [6.70, 10.60]	< 0.001
TyG-BMI	108.62 [155.11, 192.26]	191.27 [166.00, 207.84]	< 0.001
Preop Crea (μmol/L) (median [IQR])	75.30 [68.10, 91.00]	92.00 [64.20, 111.40]	< 0.001
Postop Crea (μmol/L) (D0) (median [IQR])	61.50 [51.00, 76.40]	78.90 [57.00, 91.80]	0.008
Postop Crea (μmol/L) (D1) (median [IQR])	79.00 [65.00, 94.00]	93.00 [71.00, 123.00]	0.001
Postop Crea (mmol/L) (D2) (median [IQR])	58.70 [76.70, 94.00]	95.90 [76.00, 138.00]	< 0.001
ICU length of stay (minutes) (median[IQR])	1429.00 [1238.00, 2737.00]	2385.00 [1406.00, 2817.00]	0.024
Mechanical ventilation (minutes) (median [IQR])	311.00 [215.00, 516.00]	343.00 [245.00, 567.00]	0.274

Data are expressed as the median (25th–75th percentiles), median M (Q25, Q75), or number (percentage). BMI, body mass index; CAD, coronary heart disease; AF, atrial fibrillation; MI, myocardial infarction; TBiL, total bilirubin; Alb, albumin; LAD, left atrial diameter; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; TG, triglyceride; FBG, fasting blood-glucose; TyG, Triglyceride-Glucose; Crea, creatinine; eGFR, estimated glomerular filtration rate; perop, preoperative; postop, postoperative; D0, the day after surgery; D1, the first day after surgery; D2, the second day after surgery

of univariate regression analysis, multivariate Logistic regression model A was constructed using all significant variables, and model B was constructed using significant variables excluding TyG-BMI. ROC analysis indicated that model A had better predictive performance compared to model B (AUC = 0.836 vs. 0.766) (Fig. 3). In model 2, TyG-BMI and preoperative creatinine level were independent risk factors, but they did not have good predictive value when used alone (AUC = 0.689 and 0.596, respectively) (Fig. 4). Further, the fit curve of Model 2 is plotted, where each point in the graph represents an observation from the dataset. The x-axis indicates the predicted probability of an observation developing AKI. The y-axis represents the actual AKI label, where 1 indicates that AKI occurred and 0 indicates that AKI did not occur. The color of the scatter points represents the actual occurrence of AKI, with red indicating observations where AKI did not actually occur (AKI_Flag = 0) and green indicating observations where AKI did occur (AKI_Flag = 1). It can be intuitively observed from the image that green points are likely more concentrated in areas of higher predicted probability. The curve in the graph represents the trend of the predicted probability as the x-axis changes, indicating that the model performs well in predicting the probability of AKI (Fig. 5).

Table 2 Logistic regression analysis of AKI in patients

	β	SE	Wald X^2value	*P* value	OR value	95%CI
Hypertension	0.60	0.39	1.57	0.12	1.83	(0.86 3.96)
CAD	1.26	1.17	1.08	0.28	3.54	(0.44 72.57)
Chronic kidney disease	1.35	0.81	1.66	0.10	3.86	(0.92 26.34)
Chronic lung disease	-1.81	1.11	-1.63	0.10	0.16	(0.00 1.05)
AF	-0.13	0.84	-0.16	0.87	0.88	(0.16 4.89)
Hyperlipidemia	-0.14	0.73	-0.19	0.85	0.87	(0.20 3.85)
MI	0.76	0.63	1.20	0.23	2.13	(0.65 8.24)
Preop WBC	0.17	0.10	1.65	0.10	1.18	(0.97 1.46)
Preop N%	0.01	0.01	1.04	0.30	1.01	(1.00 1.04)
Preop Hemoglobin	-0.02	0.01	-1.50	0.13	0.98	(0.96 1.00)
Preop Platelet	0.00	0.00	0.51	0.60	1.00	(1.00 1.00)
Preop TBil	0.02	0.04	0.42	0.67	1.02	(0.94 1.10)
Preop ALB	-0.08	0.04	-1.86	0.06	0.92	(0.84 1.00)
Preop LAD	0.05	0.04	1.24	0.22	1.05	(0.97 1.13)
Preop LVEDD	0.02	0.03	0.91	0.36	1.02	(0.97 1.08)
Preop LVEF	-0.01	0.02	-0.54	0.59	0.10	(0.96 1.03)
Preop TG	1.20	0.34	3.59	< 0.001	3.34	(1.80 6.77)
Preop FBG	0.45	0.10	4.53	< 0.001	1.57	(1.31 1.94)
Preop Crea	0.01	0.01	2.12	0.03	1.01	(1.00 1.02)
Postop Crea (D0)	0.02	0.01	2.66	0.01	1.02	(1.01 1.04)
Postop Crea (D1)	0.02	0.01	3.10	< 0.001	1.02	(1.01 1.04)
Postop Crea (D2)	0.02	0.01	3.55	< 0.001	1.02	(1.01 1.03)
Euroscore	0.08	0.08	0.96	0.34	1.08	(0.92 1.27)
ICU length of stay	0.00	0.00	1.92	0.05	1.00	(1.00 1.00)
Mechanical ventilation	0.00	0.00	1.36	0.17	1.00	(1.00 1.00)
TyG-BMI	0.03	0.01	3.68	< 0.001	1.03	(1.01 1.04)

CAD, coronary heart disease; AF, atrial fibrillation; MI, myocardial infarction; TBiL, total bilirubin; Alb, albumin; LAD, left atrial diameter; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; TG, triglyceride; FBG, fasting blood-glucose; TyG, Triglyceride-Glucose; Crea, creatinine; BMI, body mass index; perop, preoperative; postop, postoperative; D0, the day after surgery; D1, the first day after surgery; D2, the second day after surgery

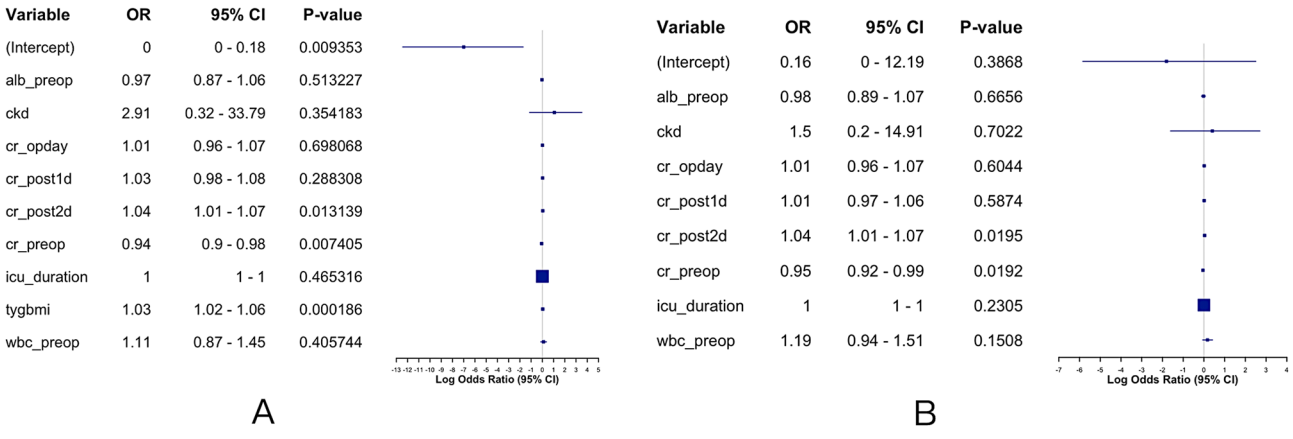

Fig. 2 Forest plots for Multivariate Logistic Regression Model

Correlation between TyG-BMI and AKI

A positive correlation was observed between TyG-BMI and AKI occurrence (Spearman's correlation coefficient: $R = 0.33$, $P = 0.00019$). The relationship was statistically significant, suggesting that higher TyG-BMI values may be associated with an increased risk of AKI (Fig. 6).

Discussion

This study explored the relationship between the TyG-BMI index and the occurrence of AKI in diabetic patients after CABG. Logistic regression analysis showed that patients with higher TyG-BMI index had a relatively higher risk of renal impairment, indicating that the

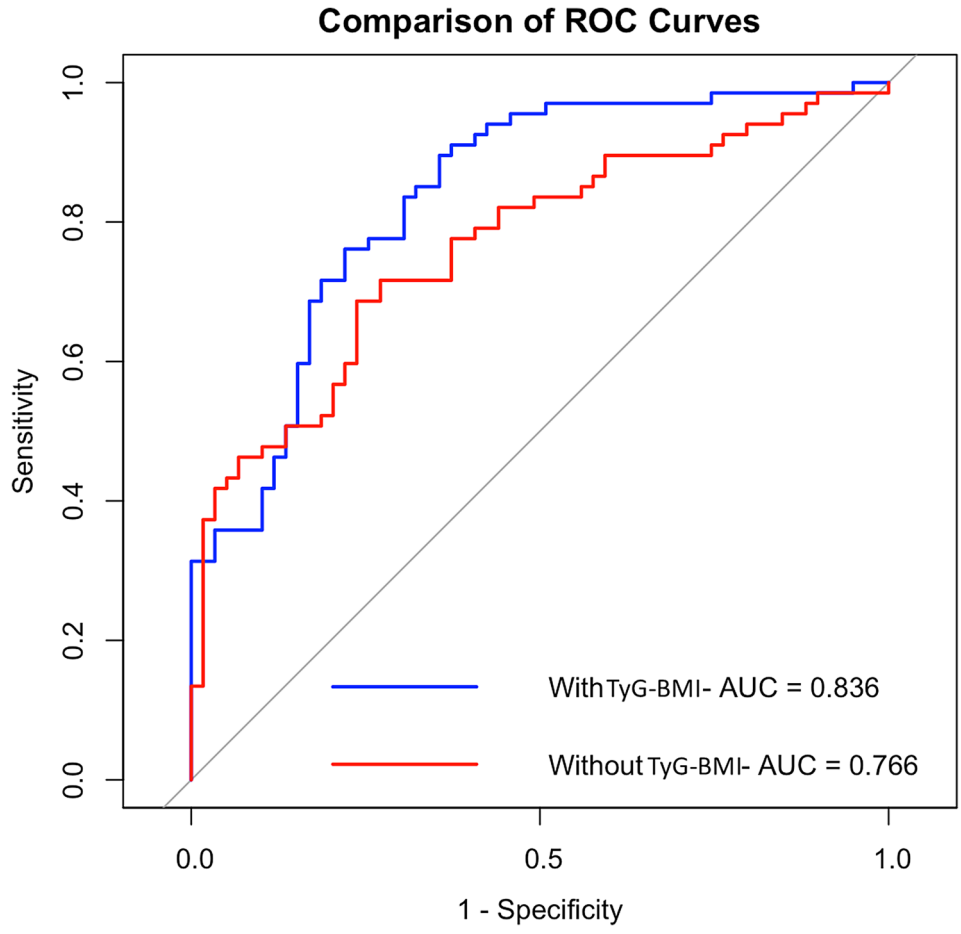

Fig. 3 The Comparison of ROC curve for predicting AKI

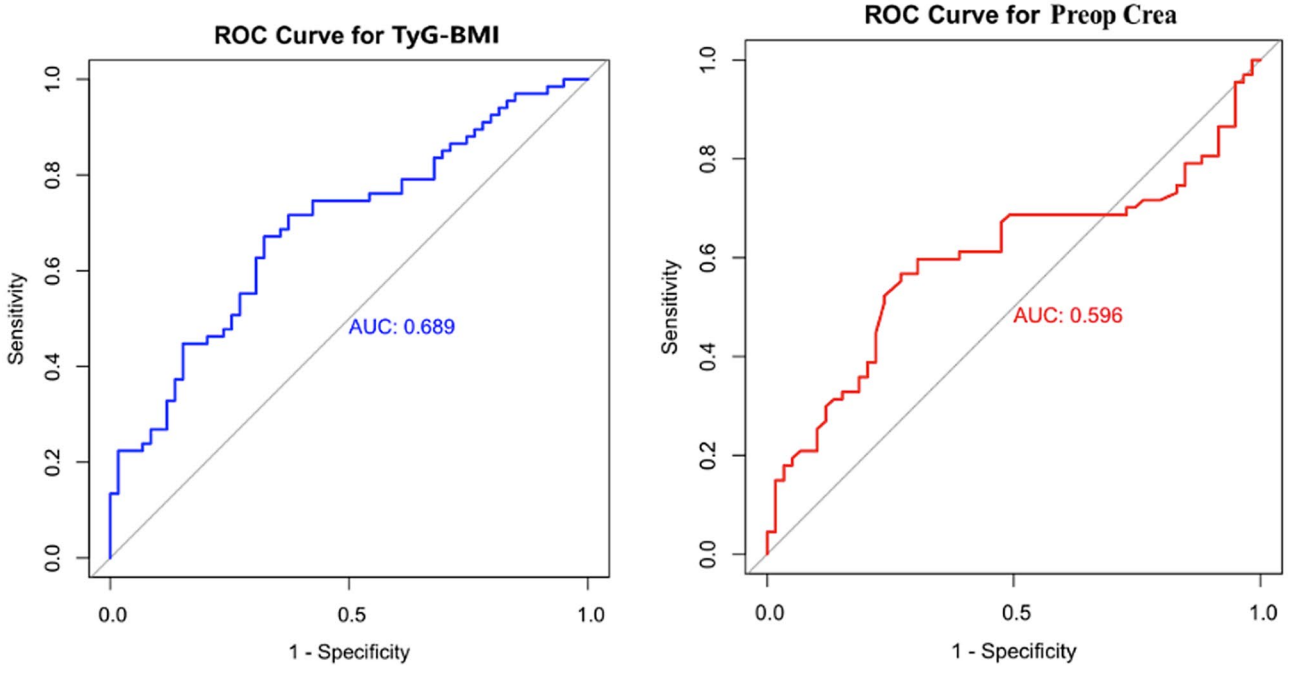

Fig. 4 The ROC curve of TyG-BMI and Crea

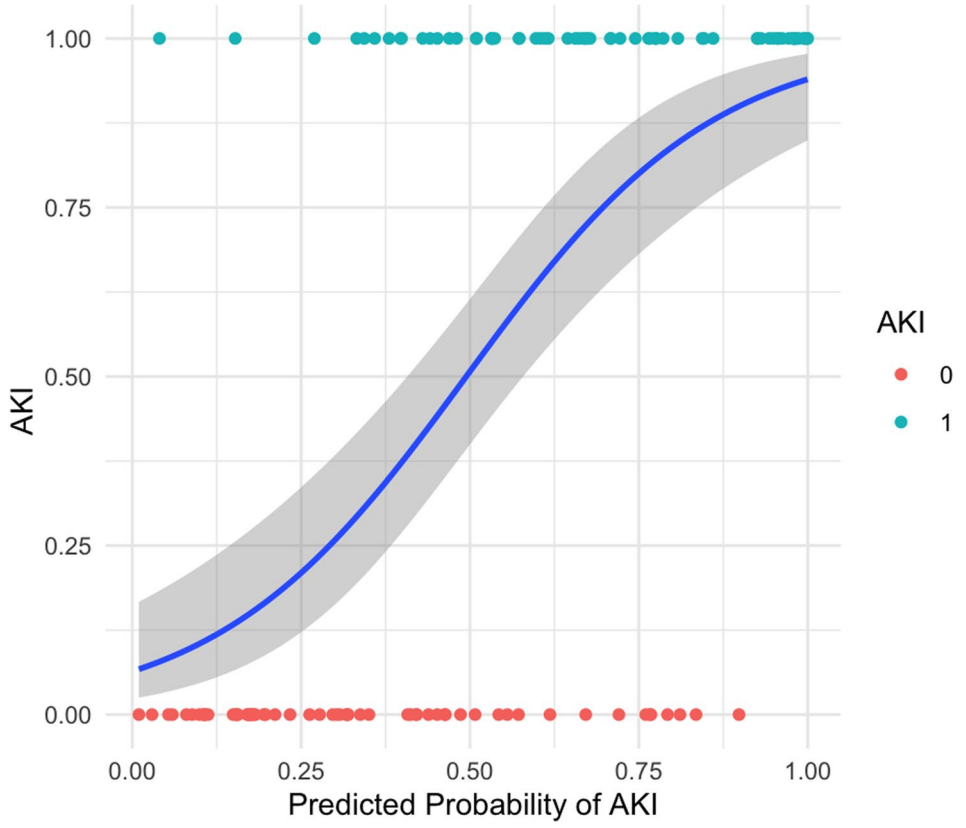

Fig. 5 Fit curve of Model2 for AKI

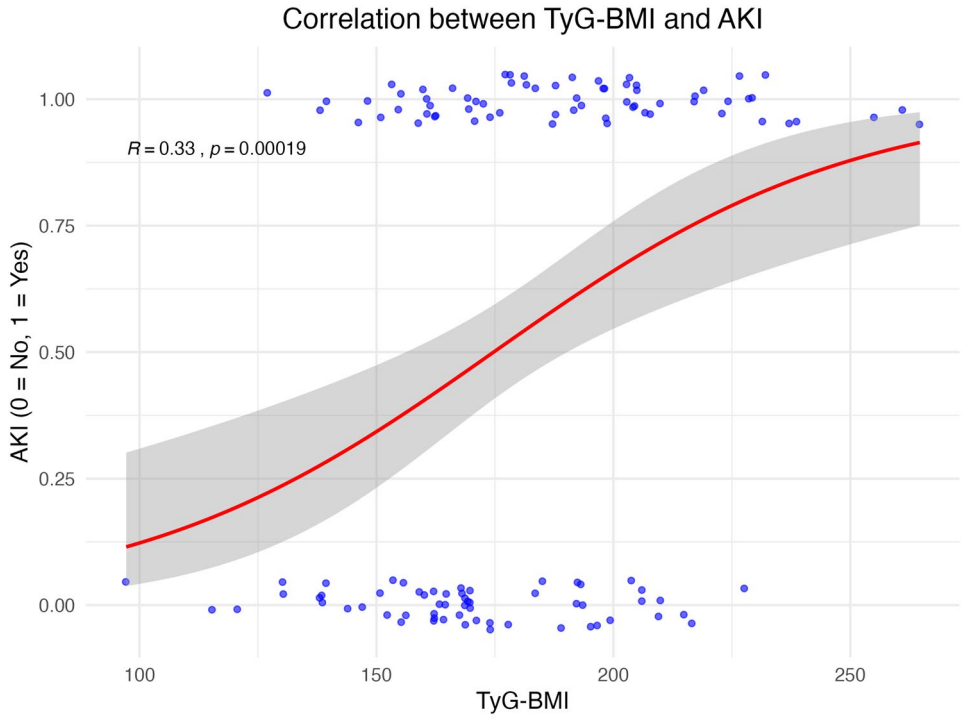

Fig. 6 Correlatioin betweenTyG-BMI and AKI

TyG-BMI index is significantly associated with early renal injury in diabetic patients after CABG.

Previous studies have found that patients with a BMI greater than 40 kg/m² exhibit a significantly increased incidence of postoperative AKI [13]. Of course, there are several notable differences between their research and ours. Their study cohort included a substantial number of obese patients, whereas our study did not include any patients with a BMI equal to or exceeding 40 kg/m². They did not examine the impact of baseline hemoglobin on AKI, which are established independent risk factors for AKI [14]. In an analysis of 432 patients undergoing surgery with cardiopulmonary bypass, a BMI of 30 kg/m² or greater was identified as an independent risk factor for AKI [15]. Furthermore, several other studies have also found that BMI is an independent predictive factor for the development of AKI in patients undergoing cardiovascular surgery [16–17].

Insulin resistance is a systemic disorder of glucose and lipid metabolism characterized by hyperinsulinemia, hyperglycemia, and hyperlipidemia. Studies have shown that hyperglycemia is an independent predictor of AKI [18]. Hyperglycemia may cause abnormalities in glomerular blood flow and vascular permeability, including capillary occlusion, tissue hypoxia, and glomerulosclerosis, leading to proteinuria and AKI [19]. Additionally, dyslipidemia has been shown to be crucial in the development and progression of kidney disease. While the mechanisms by which lipids damage renal blood vessels, mesangial cells, and tubular cells are not fully understood, the "lipid nephrotoxicity hypothesis" was first proposed by Moorhead [20]. Current research indicates that hypertriglyceridemia is an independent risk factor for early AKI [21]. Insulin resistance plays a vital role in the development of cardiovascular disease, diabetes, and renal impairment. Insulin exerts unique effects on renal microcirculation by regulating metabolic and growth pathways [22]. Insulin resistance often impairs renal blood flow and glomerular filtration, potentially causing inflammation and fibrosis [22]. Insulin resistance can occur in patients with renal impairment, with or without diabetes. Notably, insulin resistance is an important and independent risk factor for kidney disease [23]. In this study, patients who developed AKI postoperatively had higher BMI values, hyperglycemia, and hyperlipidemia, consistent with previous research results that such patients are more prone to insulin resistance.

Approximately 2 million patients undergo cardiac surgery annually, with about 20–30% of patients developing cardiac surgery-associated AKI [24]. AKI is characterized by a sudden decline in kidney function, lasting hours to days, marked by a rapid increase in serum creatinine, reduced urine output, or both. Among patients developing AKI after cardiac surgery, only a small proportion (2–3%) require renal replacement therapy. Although cardiac surgery aims to improve quality of life and survival rates, the development of cardiac surgery-associated AKI is significantly associated with higher hospitalization costs and increased short-term and long-term postoperative mortality [25, 26]. Multiple registry and retrospective cohort studies have reported significant associations between cardiac surgery-associated AKI and chronic kidney disease, end-stage renal disease, heart failure, and the later development of major adverse cardiovascular events [27, 28]. The increase in creatinine levels can only be observed within days after renal injury, potentially leading to an underestimation of AKI occurrence. Previous studies have reported significant associations between the TyG index and decreased eGFR and diabetic nephropathy. However, the relationship between the TyG-BMI index and postoperative AKI has not been explored. This study shows that patients with AKI postoperatively had longer ICU stays, similar to previous research findings. The high incidence of AKI in this study is attributed to the older age of the patients, poor glycemic control, and elevated preoperative creatinine levels. The TyG-BMI of patients developing AKI postoperatively was significantly higher than that of patients who did not develop AKI, indicating that TyG-BMI is a risk factor for postoperative acute kidney injury. The ROC curve area for TyG-BMI alone was 0.689. Adding the TyG-BMI variable to the prediction model increased the area under the curve from 0.766 to 0.836, indicating that the TyG-BMI index has some value in predicting the occurrence of acute kidney injury in diabetic patients post-CABG. Although the correlation strength was weak to moderate, The relationship was statistically significant, suggesting that higher TyG-BMI values may be associated with an increased risk of AKI.

Based on the above data, the TyG-BMI index appears to be a reliable and effective indicator for predicting the incidence of AKI. Our findings show a close association between high TyG-BMI levels and an increased incidence of AKI. AKI may eventually lead to chronic renal impairment, uremia, or death. Our findings help identify clinically high-risk patients and take measures to prevent them from developing AKI. To date, clinical research on the relationship between acute kidney injury and the TyG-BMI marker in post-cardiac surgery patients is limited. Our investigation fills this gap.

Limitations

This study has several limitations. Firstly, the results are derived from a retrospective study, and selection bias is inevitable. Secondly, we only measured fasting triglycerides and glucose at admission, which were not continuously monitored parameters. Thirdly, we could only establish the correlation between TyG-BMI and early

renal injury post-CABG in diabetic patients; whether there is a causal relationship requires further prospective research.

Conclusions

This study found that higher TyG-BMI scores were linked to a greater chance of developing AKI after heart surgery, suggesting TyG-BMI could help identify patients at risk for AKI. This could lead to better prevention and treatment strategies. However, further research is needed to validate the accuracy and reliability of TyG-BMI in predicting AKI post-cardiac surgery.

Author contributions

(I) Conception and design: C Li, F Zhu; (II) Administrative support: F Zhu, G Fan; (III) Provision of study materials or patients: X Liu, X Lv; (IV) Collection and assembly of data: X Liu, X Lv; (V) Data analysis and interpretation: W Zhou, X Liu; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Approval for this study was obtained from the Committee on Ethics of Biomedical Research at Shanghai East Hospital, Tongji University School of Medicine, Shanghai (No. 2024-YS-043), with a waiver for individual patient consent granted. This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). Given the observational nature of the study, individual patient consent was waived by the Ethics Committee.

Consent for publication

All participating authors agree to publication of the article.

Competing interests

The authors declare no competing interests.

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