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Predictive value of the triglyceride-glucose index for coronary artery bypass grafting-acute kidney injury patients

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Abstract

Background Acute kidney injury (AKI) is a common and serious complication after coronary artery bypass grafting (CABG), significantly affecting patient outcomes. The triglyceride-glucose (TyG) index, a marker of insulin resistance, has shown potential in predicting various metabolic and cardiovascular conditions. This study aimed to evaluate the predictive value of the TyG index for AKI occurrence following CABG.

Methods This retrospective, single-center study included 3,260 patients who underwent CABG. Patients were categorized into AKI and non-AKI groups based on postoperative renal function. The preoperative TyG index was calculated from fasting blood glucose and triglyceride levels. Patients were further divided into quartiles based on the TyG index. Logistic regression analysis was used to assess the relationship between TyG index and AKI risk. Subgroup analyses and spline regression were employed to explore potential interactions and non-linear relationships.

Results Of the 3,260 patients, 514 (15.8%) developed AKI. Baseline characteristics showed that AKI patients had significantly higher levels of hemoglobin (Hb), alanine aminotransferase (ALT), aspartate aminotransferase (AST), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and glucose, while brain natriuretic peptide (BNP) levels were lower compared to non-AKI patients. Logistic regression analysis confirmed that the TyG index was an independent risk factor for AKI following CABG, both as a continuous variable (OR 1.034 [95% CI 1.017–1.050], $p < 0.001$) and when grouped by quartiles. A non-linear relationship between TyG index and AKI risk was observed, with a significant increase in AKI risk when the TyG index exceeded 5.4. Subgroup analyses revealed that this association was consistent across multiple patient groups, including those stratified by age, sex, BMI, extracorporeal circulation use, and comorbidities such as hypertension, diabetes, and hyperlipidemia.

Conclusions The preoperative TyG index is a significant independent predictor of AKI after CABG, with a dose-response relationship observed across various subgroups. Monitoring the TyG index can help identify high-risk patients, potentially guiding early intervention and improving postoperative outcomes. These findings underscore

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the potential of the TyG index as a valuable tool for predicting AKI in clinical practice, warranting further validation in prospective studies.

Keywords CABG, AKI, Triglyceride-glucose

Introduction

Coronary artery bypass grafting (CABG) is one of the standard procedures for treating severe coronary artery disease [1]. However, postoperative Acute kidney injury (AKI) remains a common and serious complication that significantly affects patient outcomes [2]. The occurrence of AKI is associated with multiple factors, including the trauma from surgery, anesthesia, the use of medications, and intraoperative hemodynamic changes [3]. AKI not only prolongs hospital stay and increases healthcare costs, but it can also lead to long-term kidney dysfunction, systemic complications, and higher mortality rates [4]. Therefore, identifying high-risk patients and implementing early preventive and intervention strategies are crucial for improving postoperative outcomes in CABG patients.

In recent years, the triglyceride-glucose (TyG) index, a marker of insulin resistance (IR), has been widely used for predicting and assessing metabolic diseases [5]. The TyG index is calculated from fasting blood glucose (FBG) and triglyceride (TG) levels and effectively reflects abnormalities in glucose and lipid metabolism [6]. Previous studies have shown that the TyG index is closely associated with the occurrence and prognosis of cardiovascular diseases, diabetes, and other metabolic disorders [7, 8]. Given that insulin resistance is a key risk factor for the development of AKI, recent studies have begun to explore the relationship between the TyG index and AKI. Particularly in acutely ill patients, insulin resistance may exacerbate kidney injury and hinder recovery.

As a potential predictor of AKI, the TyG index could play a significant role in forecasting postoperative AKI following CABG. While the prognostic value of the TyG index has been widely validated in other diseases and clinical settings, its application in predicting AKI after CABG has not been sufficiently explored. Therefore, this study aims to assess whether the TyG index can serve as an effective indicator for predicting AKI after CABG, providing clinicians with a simple, cost-effective tool for early identification of high-risk patients and personalized intervention and management.

Methods

Data sources

This study employed a retrospective design, with data sourced from patients who underwent coronary artery bypass grafting (CABG) at Beijing Anzhen Hospital between January 2022 and December 2023. The study adhered to ethical guidelines and was approved by the hospital's ethics committee. As a retrospective study utilizing de-identified clinical data, the ethics committee waived the requirement for informed consent.

Study population

The inclusion criteria were as follows: patients diagnosed with coronary artery disease and scheduled for coronary artery bypass grafting (CABG); patients aged > 18 and ≤ 80 years. The exclusion criteria included: preoperative renal dysfunction, including patients with chronic kidney disease, those on dialysis, and others; patients undergoing concomitant valve surgery, major vascular surgery, or other procedures; and to ensure the completeness of clinical data for all enrolled patients, those with missing or incomplete clinical information were excluded. This approach was taken to minimize potential bias and the impact of missing data on the results.

The primary endpoint of this study was the occurrence of acute kidney injury (AKI) after CABG surgery.

AKI was defined according to the "Kidney Disease: Improving Global Outcomes" (KDIGO) guidelines [9]. AKI was defined as an increase in serum creatinine (Scr) of ≥ 0.3 mg/dL within 48 h or an increase in Scr of ≥ 1.5 times baseline within 7 days.

Data collection

All clinical data required for this study were retrieved from the electronic medical record system at Beijing Anzhen Hospital, including basic patient information, preoperative and postoperative clinical test results, triglyceride (TG) and fasting blood glucose (FBG) levels, and postoperative renal function parameters. All data were anonymized to ensure compliance with privacy protection requirements.

TyG index calculation

The TyGindex was calculated using the following formula: TyG index = TG [mg/dL]*FBG [mg/dL]/2. To assess the predictive value of the TyG index for AKI following CABG, patients were divided into four groups based on the TyG index quartiles on the first day of hospitalization. For patients with multiple measurements, the first TyG index measurement within 24 h of admission was used for analysis.

Statistical analysis

All data were analyzed using SPSS version 24.0 (USA) and R software (Version 4.2.0). Descriptive statistics were first employed to summarize the basic clinical

characteristics of the patients. The baseline characteristics of the four groups based on the TyG index were then compared using chi-square tests for categorical variables and Kruskal-Wallis tests for continuous variables, as the latter were not normally distributed. In cases where continuous variables were normally distributed, one-way ANOVA was used for group comparisons.

Subsequently, multivariate regression analysis was performed to evaluate the association between the TyG index and postoperative AKI, adjusting for potential confounding factors, and calculating the odds ratio (OR) with its 95% confidence interval (CI). Additionally, logistic regression models were used to further assess the sensitivity, specificity, and predictive performance of the TyG index in forecasting AKI occurrence. Spline curves were applied to examine the non-linear relationship between the TyG index and AKI prediction. A two-tailed p-value of < 0.05 was considered statistically significant.

Ethical statement

The data used in this study were obtained from the clinical database of Beijing Anzhen Hospital, with all patient information de-identified to comply with privacy protection regulations. The study was approved by the hospital's ethics committee and adhered strictly to ethical guidelines.

Results

Baseline characteristics

A total of 6,780 patients who underwent coronary artery bypass graft (CABG) surgery were included in the data, with 5,783 patients undergoing isolated CABG. Based on the inclusion and exclusion criteria, 3,260 patients were included in this study. The patient selection flowchart is shown in Fig. 1. Table 1 compares the baseline characteristics of patients with and without AKI.

Regarding laboratory parameters, patients with AKI had significantly higher levels of hemoglobin (Hb), alanine aminotransferase (ALT), aspartate aminotransferase (AST), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and glucose (Glu), while their B-type natriuretic peptide (BNP) levels were lower compared to non-AKI patients. Subsequently, these patients were divided into four groups based on the quartiles of their TyG index. The baseline characteristics of each group are shown in Table 2.

Primary endpoint

Among the 3,260 patients, 514 (15.8%) developed AKI. Table 2 shows the differences in the cumulative incidence of AKI during hospitalization across the four groups. We also observed that the incidence of AKI gradually

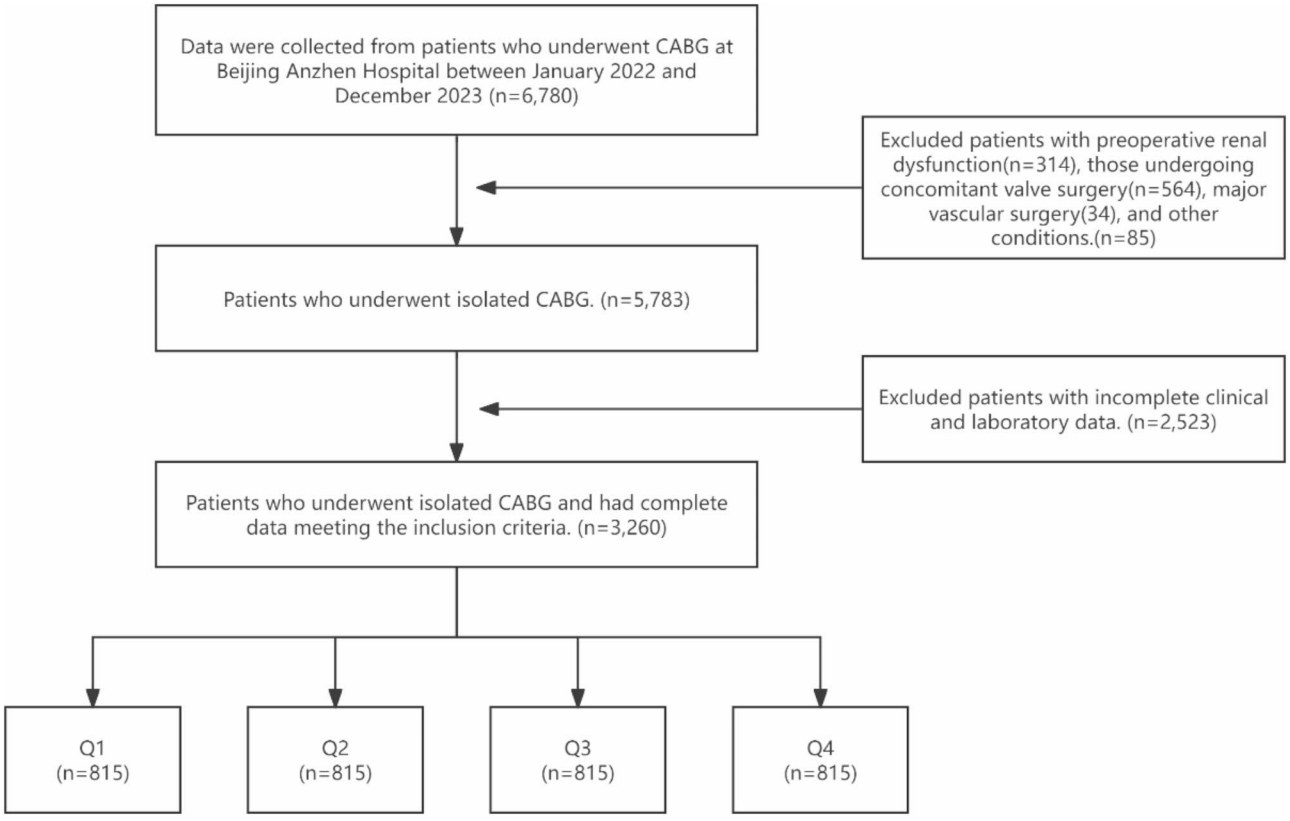

Fig. 1 Flow chart of patient selection

Table 1 Baseline characteristics of the AKI and Non-AKI groups

	All patients (*N* = 3260)	Non-AKI (*N* = 2746)	AKI (*N* = 514)	*p*. value
Gender				
Female	841 (25.8%)	666 (24.3%)	175 (34.0%)	< 0.001
Male	2419 (74.2%)	2080 (75.7%)	339 (66.0%)	
Age, years	64.0 (57.0–69.0)	63.5 (57.0–69.0)	64.0 (57.0–69.0)	0.384
BMI	25.5 (23.6–27.7)	25.6 (23.6–27.7)	25.3 (23.4–27.7)	0.143
Cardiopulmonary bypass	614 (18.8%)	536 (19.5%)	78 (15.2%)	0.0244
MI	524 (16.1%)	442 (16.1%)	82 (16.0%)	0.988
Stenocardia	2992 (91.8%)	2513 (91.5%)	479 (93.2%)	0.237
STEMI	112 (3.4%)	103 (3.8%)	9 (1.8%)	0.0313
NSTEMI	156 (4.8%)	129 (4.7%)	27 (5.3%)	0.668
hypertension	2194 (67.3%)	1847 (67.3%)	347 (67.5%)	0.953
Diabetes	1304 (40.0%)	1087 (39.6%)	217 (42.2%)	0.285
Hyperlipidemia	1737 (53.3%)	1460 (53.2%)	277 (53.9%)	0.8
Cerebral infarction	400 (12.3%)	334 (12.2%)	66 (12.8%)	0.722
COPD	74 (2.3%)	56 (2.0%)	18 (3.5%)	0.0598
PCI	336 (10.3%)	285 (10.4%)	51 (9.9%)	0.815
Hb	117.0 (99.0–136.0)	115.0 (98.0–134.0)	128.0 (104.0–145.0)	< 0.001
ALT	23.0 (15.0–38.0)	22.0 (15.0–36.0)	25.0 (16.0–44.0)	< 0.001
AST	22.0 (17.0–34.0)	22.0 (17.0–34.0)	24.0 (18.0–33.0)	0.0241
TG	1.4 (1.0–1.9)	1.4 (1.0–1.9)	1.6 (1.2–2.1)	< 0.001
TC	3.8 (3.2–4.5)	3.8 (3.2–4.4)	4.2 (3.6–4.8)	< 0.001
HDL-C	1.0 (0.8–1.1)	0.9 (0.8–1.1)	1.0 (0.9–1.2)	< 0.001
LDL-C	2.2 (1.7–2.7)	2.2 (1.7–2.7)	2.4 (1.9–3.0)	< 0.001
Urea	6.2 (4.8–8.0)	6.2 (4.8–8.0)	6.4 (5.0–8.2)	0.0889
Scr	73.1(31.2–106)	72.8(36.7–106)	73.1(31.2–106)	0.599
UA	326.6 (272.5–386.8)	327.3 (274.3–387.4)	321.1 (264.7–380.6)	0.169
CK-MB	2.8 (1.6–5.6)	2.9 (1.6–5.7)	2.6 (1.5–5.3)	0.0528
BNP	191.5 (83.0–376.0)	201.0 (87.0–388.0)	145.0 (62.0–315.0)	< 0.001
EF	60.0 (54.0–65.0)	60.0 (54.0–65.0)	60.0 (54.0–65.0)	0.768
Glu	7.2 (5.6–9.9)	7.2 (5.6–9.8)	7.4 (5.6–10.0)	0.418
TyG-index	5.3 (3.6–8.1)	5.2 (3.5–7.8)	6.0 (4.0–9.2)	< 0.001

MI: myocardial infarction; COPD: chronic obstructive pulmonary disease; PCI: percutaneous coronary intervention; Hb: hemoglobin; ALT: alanine aminotransferase; AST: aspartate aminotransferase; TG: triglycerides; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; UA: uric acid; CK-MB: creatine kinase-MB; BNP: B-type natriuretic peptide; EF: ejection fraction; Glu: Glucose; TyG-index: triglyceride glucose index

increased with higher quartiles of the TyG index (log-rank $P < 0.001$).

As shown in Table 3, logistic regression models confirmed the independent impact of the TyG index on the incidence of CABG-related AKI. Logistic regression analysis revealed that the TyG index was a significant risk factor for AKI in the unadjusted model (OR, 1.034 [95% CI, 1.017–1.050], $P < 0.001$), partially adjusted model (OR, 1.033 [95% CI, 1.016–1.049], $P < 0.001$), and fully adjusted model with the TyG index as a continuous variable (OR, 1.032 [95% CI, 1.016–1.048], $P < 0.001$).

Furthermore, when the TyG index was treated as a categorical variable, patients in the fourth quartile (Q4) had a significantly higher risk of AKI compared to those in the first quartile (Q1). In the unadjusted model, the odds ratio (OR) for AKI in Q4 was 1.89 (95% CI, 1.45–2.49, $P < 0.001$); in the partially adjusted model, OR was 1.86 (95% CI, 1.42–2.46, $P < 0.001$); and in the fully adjusted model, OR was 1.86 (95% CI, 1.42–2.46, $P < 0.001$). Compared to patients in Q1, the risk of AKI increased with higher quartiles of the TyG index.

In this study, we used spline curves to analyze the relationship between the TyG index and CABG-related acute kidney injury (CABG-AKI) (Fig. 2). The results showed that the risk of AKI significantly increased when the TyG index reached 5.4. Specifically, the relationship between the TyG index and AKI risk was non-linear, with the risk of AKI progressively rising as the TyG index increased. After reaching the critical threshold of 5.4, the risk of AKI sharply escalated, suggesting that a high TyG index may be an important predictor of postoperative AKI in CABG patients. This finding underscores the importance of monitoring and managing the TyG index in clinical practice to reduce the incidence of postoperative AKI.

Further stratified and interaction analyses revealed that the association between the TyG index and AKI risk was

Table 2 Baseline characteristics according to TyG quartiles

	Overall (*N* = 3260)	Q1 (0.89–3.58) (*N* = 815)	Q2 (3.58–5.31) (*N* = 815)	Q3 (5.31–8.09) (*N* = 815)	Q4 (8.09–85.43) (*N* = 815)	*p*. value
Gender						< 0.001
Female	841 (25.8%)	165 (20.2%)	209 (25.6%)	230 (28.2%)	237 (29.1%)	
Male	2419 (74.2%)	650 (79.8%)	606 (74.4%)	585 (71.8%)	578 (70.9%)	
age	62.6 (8.74)	62.3 (9.20)	63.0 (8.81)	62.5 (8.34)	62.7 (8.58)	0.445
BMI	25.2 (3.08)	25.7 (3.22)	25.9 (3.16)	26.2 (3.18)	25.7 (3.18)	< 0.001
Cardiopulmonary bypass	614 (18.8%)	162 (19.9%)	160 (19.6%)	163 (20.0%)	129 (15.8%)	0.091
MI	524 (16.1%)	130 (16.0%)	125 (15.3%)	129 (15.8%)	140 (17.2%)	0.775
stenocardia	2992 (91.8%)	755 (92.6%)	740 (90.8%)	751 (92.1%)	746 (91.5%)	0.562
STEMI	112 (3.4%)	21 (2.6%)	35 (4.3%)	27 (3.3%)	29 (3.6%)	0.296
NSTEMI	156 (4.8%)	39 (4.8%)	40 (4.9%)	38 (4.7%)	39 (4.8%)	0.997
hypertension	2194 (67.3%)	547 (67.1%)	528 (64.8%)	546 (67.0%)	573 (70.3%)	0.125
Diabetes	1304 (40.0%)	326 (40.0%)	318 (39.0%)	308 (37.8%)	352 (43.2%)	0.142
hyperlipidemia	1737 (53.3%)	438 (53.7%)	433 (53.1%)	445 (54.6%)	421 (51.7%)	0.679
cerebral infarction	400 (12.3%)	98 (12.0%)	91 (11.2%)	111 (13.6%)	100 (12.3%)	0.503
COPD	74 (2.3%)	20 (2.5%)	18 (2.2%)	17 (2.1%)	19 (2.3%)	0.964
PCI	336 (10.3%)	89 (10.9%)	79 (9.7%)	90 (11.0%)	78 (9.6%)	0.655
Hb	117 (23.4)	115 (22.4)	117 (23.1)	119 (23.9)	117 (23.9)	< 0.001
ALT	43.3 (128)	46.1 (169)	34.8 (42.9)	49.3 (162)	43.1 (96.7)	0.291
AST	42.5 (192)	49.8 (286)	32.6 (53.1)	48.1 (211)	39.3 (135)	0.0291
TG	1.64 (1.03)	0.964 (0.278)	1.34 (0.365)	1.66 (0.506)	2.60 (1.53)	< 0.001
TC	3.95 (1.02)	3.62 (0.898)	3.81 (0.944)	4.01 (0.991)	4.36 (1.10)	< 0.001
HDL-C	0.992 (0.232)	1.06 (0.259)	0.997 (0.225)	0.979 (0.217)	0.934 (0.209)	< 0.001
LDL-C	2.32 (0.854)	2.13 (0.775)	2.27 (0.825)	2.38 (0.865)	2.50 (0.904)	< 0.001
Urea	6.87 (3.32)	6.43 (2.51)	6.63 (2.68)	7.03 (3.39)	7.39 (4.29)	< 0.001
Scr	73.1(15.1)	70.8(14.7)	71.1(14.9)	73.4(1.51)	76.9(15.1)	< 0.001
UA	334 (91.2)	343 (91.4)	338 (90.1)	338 (95.3)	317 (85.8)	< 0.001
CK-MB	7.56 (34.6)	7.50 (23.3)	8.61 (58.4)	7.69 (21.8)	6.45 (18.7)	0.0048
BNP	308 (414)	321 (440)	303 (381)	307 (436)	300 (398)	0.335
EF	58.5 (9.19)	58.7 (9.14)	58.4 (8.94)	58.6 (9.30)	58.2 (9.40)	0.382
Glu	8.18 (3.44)	5.90 (1.56)	7.06 (2.12)	8.61 (2.88)	11.2 (4.09)	< 0.001
AKI	514 (15.8%)	100 (12.3%)	110 (13.5%)	134 (16.4%)	170 (20.9%)	< 0.001

MI: myocardial infarction; COPD: chronic obstructive pulmonary disease; PCI: percutaneous coronary intervention; Hb: hemoglobin; ALT: alanine aminotransferase; AST: aspartate aminotransferase; TG: triglycerides; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; UA: uric acid; CK-MB: creatine kinase-MB; BNP: B-type natriuretic peptide; EF: ejection fraction; Glu: Glucose; TyG-index: triglyceride glucose index; AKI: acute kidney injury

Table 3 The incidence of AKI in the different groups

Variable	Model 1		Model 2		Model 3	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
AKI incidence						
Continuous variable per 1 unit	1.034 (1.017–1.050)	< 0.001	1.033 (1.016–1.049)	< 0.001	1.032 (1.016–1.048)	< 0.001
Quartile[a]						
Q1	**Ref.**	**Ref.**	**Ref.**	**Ref.**	**Ref.**	**Ref.**
Q2	1.12(0.84–1.49)	0.4541	1.10(0.82–1.47)	0.5285	1.11(0.83–1.49)	0.4742
Q3	1.40(1.06–1.86)	0.0178	1.37(1.04–1.82)	0.0277	1.37(1.04–1.83)	0.0281
Q4	1.89(1.45–2.49)	< 0.001	1.86(1.42–2.46)	< 0.001	1.86(1.42–2.46)	< 0.001

Logistic proportional TyG index for AKI incidence. Model 1 was unadjusted, Model 2 was adjusted for gender, age and BMI, Model 3 was adjusted for the variables in model 2 and further adjusted for cardiopulmonary bypass, MI, stenocardia, hypertension, Diabetes, hyperlipidemia, and cerebral infarction. a TyG index Triple Quartile Q1: 0.89–3.58; Q2: 3.58–5.31; Q3: 5.31–8.09; Q4: 8.09–85.4

significant across various subgroups of CABG patients (Fig. 3). The analysis indicated that the link between the TyG index and AKI risk was consistent across multiple subgroups, particularly in patients aged < 65 years, women, those with low or high BMI, those who did or did not undergo cardiopulmonary bypass, patients with perioperative angina, and those with or without a history of hypertension, diabetes, and hyperlipidemia. A

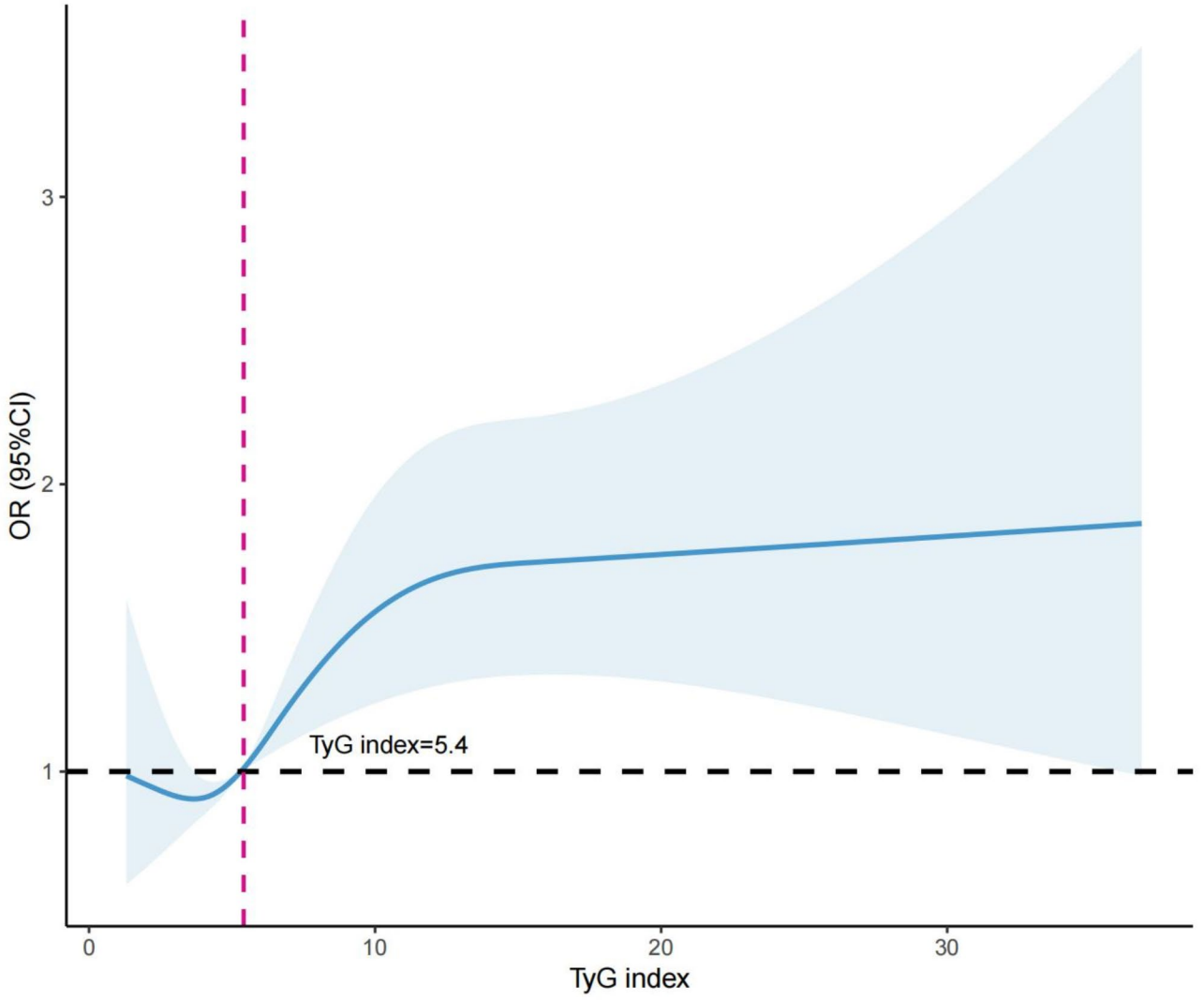

Fig. 2 Spline Chart for the TyG index hazard ratio

significant dose-response relationship was observed in these subgroups. These findings suggest that the TyG index may influence AKI risk in different ways depending on patient clinical characteristics, further validating its potential value as a predictor of AKI in CABG patients.

Discussion

In this study, we explored the relationship between the TyG index and acute kidney injury (AKI) in patients undergoing coronary artery bypass grafting (CABG). The TyG index, an indirect marker of insulin resistance, reflects the degree of metabolic disturbance, and insulin resistance itself is considered one of the potential risk factors for the development of AKI [10]. Through a retrospective analysis of 3,260 CABG patients, we identified a significant dose-response relationship between the TyG index and the risk of AKI, with this association being consistent across multiple subgroups. This finding provides a novel perspective for predicting and managing postoperative AKI in CABG patients, particularly in high-risk populations.

In addition to the focus on AKI, our study also aligns with other research demonstrating the broader relevance of the TyG index in various health conditions. For instance, higher TyG index levels have been positively correlated with an increased incidence of periodontitis, with the TyG index emerging as the most significant factor associated with periodontitis in non-diabetic men [11]. Furthermore, a significant independent association between the TyG index and skeletal muscle mass loss (SMM-L) [12] suggests that it may serve as a valuable marker for muscle health. The TyG index has also been identified as an important prognostic indicator for arthritis [13] and is significantly correlated with sensorineural hearing loss (SNHL) [14], highlighting its potential relevance to auditory health.

Variables	n (%)	TyG < 5.4	TyG ⩾ 5.4	OR (95% CI)	P	P for interaction
All patients	3260 (100.00)	217/1668	297/1592	1.54 (1.22 ~ 1.95)	<.001	
Age						0.289
<65	1772 (54.36)	106/897	160/875	1.71 (1.23 ~ 2.36)	0.001	
⩾65	1488 (45.64)	111/771	137/717	1.39 (0.99 ~ 1.96)	0.058	
Gender						0.581
Female	841 (25.80)	63/386	112/455	1.96 (1.29 ~ 2.96)	0.002	
Male	2419 (74.20)	154/1282	185/1137	1.36 (1.02 ~ 1.81)	0.036	
BMI						0.887
<24	2525 (77.45)	174/1332	225/1193	1.46 (1.12 ~ 1.91)	0.005	
⩾24	735 (22.55)	43/336	72/399	1.69 (1.02 ~ 2.79)	0.041	
Cardiopulmonarybypass						0.878
No	2646 (81.17)	180/1336	256/1310	1.57 (1.22 ~ 2.02)	<.001	
Yes	614 (18.83)	37/332	41/282	1.50 (0.76 ~ 2.98)	0.243	
Stenocardia						0.993
No	268 (8.22)	14/137	21/131	1.23 (0.45 ~ 3.37)	0.682	
Yes	2992 (91.78)	203/1531	276/1461	1.56 (1.22 ~ 1.98)	<.001	
Hypertension						0.946
No	1066 (32.70)	74/565	93/501	1.66 (1.09 ~ 2.52)	0.018	
Yes	2194 (67.30)	143/1103	204/1091	1.53 (1.15 ~ 2.03)	0.004	
Diabetes						0.303
No	1956 (60.00)	120/1011	177/945	1.79 (1.31 ~ 2.43)	<.001	
Yes	1304 (40.00)	97/657		1.29 (0.89 ~ 1.85)	0.176	
Hyperlipidemia						0.633
No	1523 (46.72)	102/776	135/747	1.51 (1.06 ~ 2.14)	0.022	
Yes	1737 (53.28)	115/892	162/845	1.57 (1.14 ~ 2.16)	0.005	
Cerebralinfarction						0.343
No	2860 (87.73)	187/1470	261/1390	1.59 (1.24 ~ 2.04)	<.001	
Yes	400 (12.27)	30/198	36/202	1.28 (0.65 ~ 2.51)	0.472	
COPD						0.631
No	3186 (97.73)	210/1630	286/1556	1.52 (1.20 ~ 1.93)	<.001	
Yes	74 (2.27)	7/38	11/36	8.93 (0.94 ~ 84.64)	0.056	
PCI						0.674
No	2924 (89.69)	197/1498	266/1426	1.53 (1.20 ~ 1.96)	<.001	
Yes	336 (10.31)	20/170	31/166	1.78 (0.77 ~ 4.11)	0.174	

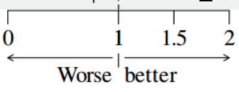

Fig. 3 Forest plots for the primary endpoint in different subgroups

Moreover, research indicates that individuals with higher TyG index levels face an increased risk of endothelial dysfunction [15], while also showing a positive correlation with intestinal abnormalities [16], pointing to its role in gastrointestinal health. Additionally, the TyG index has been recognized as an independent marker for peripheral artery disease [17], further emphasizing its role in vascular health. These findings underline the broader clinical significance of the TyG index across various health domains, extending beyond its established link to cardiovascular risk and AKI.

Therefore, further understanding the relationship between insulin resistance and AKI is crucial for refining predictive models for postoperative AKI in CABG patients. Insulin resistance refers to a reduced response of tissues to insulin, resulting in impaired physiological effects, particularly on glucose metabolism in the liver, muscle, and adipose tissue [18, 19]. Insulin resistance is commonly associated with metabolic syndrome and type 2 diabetes, often accompanied by hyperglycemia, dyslipidemia, and obesity [20, 21]. Beyond being a risk factor for diabetes and cardiovascular disease, insulin resistance may also contribute to AKI through multiple mechanisms, including altered renal blood flow, inflammation, and increased oxidative stress.

Insulin resistance promotes the development of AKI through multiple mechanisms. First, insulin resistance triggers a systemic inflammatory response, with pro-inflammatory cytokines (such as TNF-α and IL-6) secreted by adipocytes exacerbating renal inflammation and damaging renal tubules and epithelial cells [22, 23]. Second, insulin resistance is associated with

hyperglycemia and lipid metabolism disorders, leading to increased oxidative stress [24, 25]. The resulting free radicals damage tubular cells, further aggravating kidney injury. In addition, insulin resistance impairs endothelial function, reducing renal perfusion, particularly after CABG, where microvascular injury may contribute to the development of AKI. Finally, insulin resistance alters the tubules ability to reabsorb sodium and water, causing fluid retention and increasing renal workload [26, 27]. Long-term insulin resistance is also linked to the progression of diabetic nephropathy, which further accelerates kidney injury, especially in CABG patients.

Our study demonstrates a non-linear relationship between the TyG index and the risk of AKI, with a sharp increase in risk once the TyG index reaches 5.4. This threshold is particularly significant, as it not only supports previous research linking the TyG index to various cardiovascular conditions but also provides a specific marker for predicting AKI risk in patients undergoing coronary artery bypass grafting (CABG). The sharp rise in AKI risk at this threshold highlights the importance of the TyG index as a tool for identifying high-risk patients. Furthermore, this finding offers valuable insights for early screening and potential interventions aimed at reducing AKI risk in the CABG patient population. Then clinicians can implement early screening and targeted interventions, potentially mitigating the risk of acute kidney injury and improving patient outcomes in the CABG setting.

Previous studies have established the TyG index as a significant marker for cardiovascular risk. For example, Huang et al. demonstrated that a higher TyG index was associated with an increased risk of myocardial ischemia in patients with coronary artery disease (CAD) [28]. Similarly, Orlando et al. found that patients with higher TyG indices had more severe CAD [29, 30]. While these studies focused on ischemic heart disease, our study extends these findings by identifying a specific link between the TyG index and AKI in CABG patients, providing a new insight into its role in renal outcomes.

Meng et al. found that higher TyG indices were linked to an increased risk of heart failure and left ventricular dysfunction in individuals without prior heart failure or CAD [31]. While this finding underscores the role of the TyG index in heart failure, our study uniquely connects the TyG index with AKI risk specifically in the context of coronary revascularization, a relationship that had not been clearly established in prior research. Several studies have already highlighted the role of the TyG index in coronary artery revascularization outcomes. For example, Wang et al. demonstrated a positive correlation between higher TyG index levels and major adverse cardiovascular events (MACE) in patients with in-stent restenosis [32], while Wu et al. found an association between TyG index and adverse cardiovascular outcomes in CABG patients [33]. However, our study adds a novel perspective by specifically examining the TyG index as a predictor of AKI risk in CABG patients, a critical postoperative complication with significant morbidity and mortality. Finally, Yang et al. reported that an elevated TyG index was linearly associated with AKI risk in critically ill heart failure patients, with higher TyG index levels predicting a greater need for dialysis in those with AKI [34]. Our study supports this finding, but our approach extends the knowledge base by focusing on AKI in the specific cohort of CABG patients, who may be at heightened risk for both cardiovascular and renal complications. Furthermore, the non-linear relationship we identified (with a sharp increase in risk at a TyG index ≥ 5.4) offers new insight into the threshold beyond which AKI risk escalates, which has not been well-explored in the literature.

Through logistic regression models, we confirmed that the TyG index remains an independent predictor of AKI even after adjusting for potential confounders such as age, gender, BMI, diabetes, and hypertension. Additionally, stratified analyses showed that the relationship between the TyG index and AKI risk was significant across various clinical subgroups, particularly in patients under 65 years old, women, those with lower or higher BMI, and patients who did not undergo cardiopulmonary bypass. In these subgroups, a significant dose-response relationship between the TyG index and AKI risk was observed. These findings suggest that the TyG index may be associated with individualized differences in the occurrence of AKI after CABG, particularly among patients with varying age, gender, body composition, and underlying conditions. The differences across subgroups may reflect the diverse pathways by which metabolic disturbances contribute to renal injury, indicating that the TyG index could serve as a risk assessment tool in different clinical settings.

Through spline curve analysis, we further revealed a non-linear relationship between the TyG index and AKI risk. The spline curve showed that the risk of AKI sharply increased when the TyG index reached 5.4. This threshold has important clinical implications, suggesting that clinicians should closely monitor increases in the TyG index, particularly during postoperative surveillance, to identify high-risk patients early and implement interventions to reduce the incidence of AKI.

The findings of this study underscore the significant predictive value of the TyG index as a simple and actionable metabolic marker in CABG patients. Since the TyG index reflects core features of metabolic syndrome, such as insulin resistance and lipid metabolism abnormalities, it may serve as an early warning sign for the development of AKI. Therefore, regular monitoring of the TyG index, along with individualized management of high-risk

patients, could help reduce the incidence of AKI following CABG, ultimately improving patient outcomes. Specifically, combining the TyG index with other traditional AKI predictors, such as urine output and BNP levels, may provide a more accurate means of identifying high-risk patients in clinical practice.

Our findings emphasize the importance of the TyG index in CABG patients, particularly in personalized management and preventive interventions. For patients with higher TyG indices, clinicians may consider adopting additional protective measures, such as stricter blood glucose and lipid control, early administration of renal protective agents, and optimized postoperative fluid and circulatory management. These measures could help decrease the incidence of AKI and improve postoperative recovery.

This study has several limitations. First, as a retrospective analysis, we cannot rule out the potential for selection bias and information bias. Second, although we controlled for various potential confounders, there may still be unknown confounding factors that were not fully adjusted for. Third, the TyG index, used as a metabolic marker in this study, does not yet have a fully defined mechanism of action. Future prospective studies and basic experimental research will be valuable in further elucidating its role in the development of AKI.

Conclusion

This study demonstrates a significant dose-response relationship between the TyG index and postoperative AKI following CABG, with the TyG index identified as an independent risk factor for AKI. Further stratified and interaction analyses revealed differential effects of the TyG index across patient subgroups with varying clinical characteristics, highlighting its high predictive value. Future research should focus on exploring the specific mechanisms through which the TyG index contributes to AKI development and validate its potential application in clinical practice.

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Author contributions
B.H, Ts.L, K.Z and Xj.H collected data. B.H and D.L wrote this manuscript. Y.L conducted the literature review. R.D designed the study and revised the manuscript.

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Data availability
The datasets analyzed in the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Capital Medical University Affiliated Anzhen Hospital (Approval Number: KS2023062). As this is a retrospective study utilizing de-identified clinical data, the requirement for written informed consent was waived by the Ethics Committee. The study protocol adhered to all relevant ethical guidelines and regulations. Participants were provided with detailed information about the study's purpose, procedures, potential risks, and benefits, and they were given the opportunity to ask questions before providing consent. Ensuring the confidentiality and anonymity of all participants was a top priority throughout the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Bakaeen FG, Gaudino M, Whitman G et al. 2021: The American Association for Thoracic Surgery Expert Consensus Document: coronary artery bypass grafting in patients with ischemic cardiomyopathy and heart failure[J]. J Thorac Cardiovasc Surg. 2021;162(3):829–850.
2. Ostermann M, Kunst G, Baker E, et al. Cardiac surgery associated AKI prevention strategies and medical treatment for CSA-AKI[J]. J Clin Med. 2021;10(22):5285.
3. Drozdzal S, Lechowicz K, Szostak B, et al. Kidney damage from nonsteroidal anti-inflammatory drugs-myth or truth? Review of selected literature[J]. Pharmacol Res Perspect. 2021;9(4):e817.
4. See CY, Pan HC, Chen JY, et al. Improvement of composite kidney outcomes by AKI care bundles: a systematic review and meta-analysis[J]. Crit Care. 2023;27(1):390.
5. Zhang F, Sun Y, Bai Y, et al. Association of triglyceride-glucose index and diabesity: evidence from a National longitudinal study[J]. Lipids Health Dis. 2024;23(1):412.
6. Sun Y, Ji H, Sun W et al. Triglyceride glucose (TyG) index: A promising biomarker for diagnosis and treatment of different diseases[J]. Eur J Intern Med. 2024.
7. Qiu X, Aimaiti G, Chen Y et al. Associations of TyG index with coronary heart disease risk and coronary artery sclerosis severity in OSA[J]. Diabetol metab syndr. 2024;16(1):301.
8. Feng Q, Jiang M, Peng X et al. Adjustment of the ACEF score by the triglyceride glucose index improves the prediction of clinical outcomes in patients undergoing percutaneous coronary intervention[J]. Cardiovasc diabetol. 2024;23(1):440.
9. James M, Bouchard J, Ho J, et al. Canadian society of nephrology commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury[J]. Am J Kidney Dis. 2013;61(5):673–85.
10. Nayak SS, Kuriyakose D, Polisetty LD, et al. Diagnostic and prognostic value of triglyceride glucose index: a comprehensive evaluation of meta-analysis[J]. Cardiovasc Diabetol. 2024;23(1):310.
11. Tsai KZ, Lin YP, Lai SW et al. Non-insulin-based insulin resistance indices and localized periodontitis in physically active young male adults: CHIEF oral health study[J]. Endocr metab immune Disord drug targets. 2023;23(7):937–46.
12. Li Z, Tong X, Ma Y et al. Association between the triglyceride glucose index and low skeletal muscle mass: a cross-sectional study[J]. BMJ open. 2024;14(1):e77484.

13. Liu Y, Yao J, Xue X, et al. Triglyceride-glucose index in the prediction of new-onset arthritis in the general population aged over 45: the first longitudinal evidence from CHARLS[J]. Lipids Health Dis. 2024;23(1):79.
14. Wang Y, Liu H, Nie X, et al. L-shaped association of triglyceride glucose index and sensorineural hearing loss: results from a cross-sectional study and Mendelian randomization analysis[J]. Front Endocrinol (Lausanne). 2024;15:1339731.
15. Li Y, Yi M, Wang X et al. Association between triglyceride-glucose index and endothelial dysfunction[J]. Endocrine. 2024;85(2):717–23.
16. Zhu JY, Liu MY, Sun C. Assessment of the triglyceride glucose index in adult patients with chronic diarrhea and constipation[J]. World J Clin Cases. 2024;12(6):1094–103.
17. Caliskan S, Boyuk F. Is triglyceride-glucose index a valuable parameter in peripheral artery disease?[J]. Cureus. 2023;15(2):e35532.
18. Wang J, Yan S, Cui Y et al. The diagnostic and prognostic value of the triglyceride-glucose index in metabolic dysfunction-associated fatty liver disease (MAFLD): a systematic review and meta-analysis[J]. Nutrients. 2022;14(23).
19. Chang M, Shao Z, Shen G. Association between triglyceride glucose-related markers and the risk of metabolic-associated fatty liver disease: a cross-sectional study in healthy Chinese participants[J]. BMJ open. 2023;13(5):e70189.
20. Pan Y, Zhao M, Song T, et al. Role of triglyceride-glucose index in type 2 diabetes mellitus and its complications[J]. Diabetes Metab Syndr Obes. 2024;17:3325–33.
21. Tahapary DL, Pratisthita LB, Fitri NA, et al. Challenges in the diagnosis of insulin resistance: focusing on the role of HOMA-IR and tryglyceride/glucose index[J]. Diabetes Metab Syndr. 2022;16(8):102581.
22. Yao L, Li B, Zhang F, et al. Correlation between prognosis and peripheral blood levels of NLRP3 and triglyceride-glucose index after myocardial ischemia-reperfusion injury[J]. J Cardiothorac Surg. 2024;19(1):553.
23. Bonfante I, Chacon-Mikahil M, Brunelli DT, et al. Obese with higher FNDC5/Irisin levels have a better metabolic profile, lower lipopolysaccharide levels and type 2 diabetes risk[J]. Arch Endocrinol Metab. 2017;61(6):524–33.
24. Soni R, Mathur K, Rathod H et al. Hyperglycemia-driven insulin signaling defects promote Parkinson's disease-like pathology in mice[J]. ACS Pharmacol Transl Sci. 2024;7(12):4155–64.
25. Narongkiatikhun P, Choi YJ, Hampson H, et al. Unraveling diabetic kidney disease: the roles of mitochondrial dysfunction and immunometabolism[J]. Kidney Int Rep. 2024;9(12):3386–402.
26. Roy D, Ghosh M, Rangra NK. Herbal approaches to diabetes management. Pharmacological mechanisms and omics-driven discoveries[J]. Phytother Res. 2024.
27. Rosas-Martinez L, Rodriguez-Munoz R, Namorado-Tonix M et al. Peroxisome proliferator-activated receptor alpha stimulation preserves renal tight junction components in a rat model of early-stage diabetic nephropathy[J]. Int J Mol Sci. 2024;25(23).
28. Huang Z, Tang R, Ding Y, et al. Association of the triglyceride glucose index with myocardial ischemia in patients with minimal to moderate coronary artery disease[J]. Sci Rep. 2024;14(1):26093.
29. Jia L, Shang S, Yang Y, et al. The synergy of serum SFRP5 levels and the TyG index in predicting coronary artery disease and prognosing major adverse cardiovascular events[J]. Lipids Health Dis. 2023;22(1):194.
30. Siverio-Morales O, Mora-Fernandez C, Hernandez-Carballo C, et al. Predictive value of triglyceride-glucose index for the evaluation of coronary artery disease severity and occurrence of major adverse cardiovascular events[J]. Am J Physiol Heart Circ Physiol. 2025;328(1):H14–20.
31. Meng X, Feng B, Yang C, et al. Association between the triglyceride-glucose index and left ventricular myocardial work indices in patients with coronary artery disease[J]. Front Endocrinol (Lausanne). 2024;15:1447984.
32. Wang YF, Kong XH, Tao HM, et al. Triglyceride-glucose index as a predictor of major adverse cardiovascular events in post-PCI patients diagnosed with in-stent restenosis[J]. Diabetes Metab Syndr Obes. 2024;17:2737–46.
33. Wu Z, Guo D, Chen S, et al. Combination of the triglyceride-glucose index and EuroSCORE II improves the prediction of long-term adverse outcomes in patients undergoing coronary artery bypass grafting[J]. Diabetes Metab Res Rev. 2023;39(8):e3710.
34. Yang Z, Gong H, Kan F et al. Association between the triglyceride glucose (TyG) index and the risk of acute kidney injury in critically ill patients with heart failure: analysis of the MIMIC-IV database[J]. Cardiovasc diabetol. 2023;22(1):232.

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