BMC Nephrology

RESEARCH

Open Access

Prognostic value of glycemic gap in ST-segment elevation myocardial infarction-associated acute kidney injury

Xiaofu Zhang[1†], Yong Li[1†], Qinghuan Yang[1], Siwen Wu[1], Yang Song[1], Ziyun Luo[2*] and Jianping Xu[1*]

Abstract

Background Stress-induced hyperglycemia (SIH) is a common phenomenon in acute myocardial infarction and is associated with poor prognosis. The relationship between glycemic gap (GG), a marker of SIH, and ST-segment elevation myocardial infarction (STEMI)-associated acute kidney injury (STAAKI) remains unclear. This study aims to explore the predictive value of GG for the risk of STAAKI after percutaneous coronary intervention (PCI) in STEMI patients.

Methods This study retrospectively selected patients diagnosed with STEMI who underwent primary PCI. Logistic regression analysis was used to identify the risk factors associated with STAAKI. To examine the dose-response relationship between GG and STAAKI, restricted cubic splines (RCS) were employed. The predictive accuracy of the models was assessed using Delong test, net reclassification index (NRI) and integrated discrimination improvement (IDI).

Results This study included 595 patients, the incidence of STAAKI was 9.2%. Multivariate logistic regression showed LVEF (OR per 1% increase = 0.931, 95% CI: 0.895 ~ 0.969), NT-proBNP (OR per 1 pg/mL increase = 1.579, 95% CI: 1.212 ~ 2.057), and GG (OR per 1 mmol/L increase = 1.379, 95% CI: 1.223 ~ 1.554) as independent predictors of STAAKI. RCS analysis indicated a linear dose-response relationship between GG and STAAKI. After integrating GG, the new model could significantly improve the risk model for STAAKI ($Z = 2.77$, NRI = 0.780, and IDI = 0.095; All $P < 0.05$).

Conclusion GG is an independent risk factor for the occurrence of STAAKI after PCI in STEMI patients, and integrating GG can significantly improve risk modeling regarding STAAKI.

Clinical trial number Not applicable.

Keywords Cardiovascular disease, Glycemic gap, STEMI, Acute kidney injury, STEMI-induced acute kidney injury

†Xiaofu Zhang and Yong Li contributed equally to this work.

*Correspondence:
Ziyun Luo
312179045@qq.com
Jianping Xu
xjp_hh@163.com
[1]Department of Cardiology, The First People's Hospital of Yuhang District, Hangzhou, Zhejiang 311100, China
[2]Department of Nephrology, Yichun People's Hospital, Yichun, Jiangxi 336000, China

Introduction

Early restoration of coronary blood flow through percutaneous coronary intervention (PCI) plays a crucial role in improving the prognosis of patients with ST-segment elevation myocardial infarction (STEMI) [1]. STEMI-induced acute kidney injury (STAAKI) has become a common cause of in-hospital acute kidney injury (AKI) [2–4]. STAAKI is closely associated with poor clinical outcomes and prolonged hospital stays [4, 5]. Due to the limited therapeutic options for STAAKI in clinical practice and its high incidence, accurately identifying high-risk STEMI patients is of significant value [6].

In critically ill patients, the secretion of hormones such as cortisol, catecholamines, and glucagon significantly increases, leading to stress-induced hyperglycemia (SIH) [7]. Admission blood glucose (ABG) has been used in some studies as a marker of SIH to predict the risk of adverse events [8, 9]. However, the relationship between ABG and adverse outcomes in acute patients, with or without diabetes, is inconsistent [10, 11]. Since ABG values are also influenced by chronic blood glucose levels, they do not accurately reflect the true extent of SIH [12, 13]. Glycemic gap (GG), derived from the ABG adjusted for chronic blood glucose status via glycated hemoglobin (HbA1c), is considered a superior marker of SIH compared to ABG, as it partially eliminates the impact of chronic hyperglycemia on disease severity assessment, thus improving the accuracy of the evaluation [14–16]. A substantial body of evidence has confirmed that GG levels in acute myocardial infarction (AMI) patients are closely related to adverse cardiovascular outcomes, with GG showing superior predictive value for major adverse cardiovascular events (MACE) compared to ABG [17–19]. However, the relationship between GG and STAAKI in STEMI patients remains unclear. This study aims to explore the predictive value of GG for the risk of STAAKI after PCI in STEMI patients.

Methods

Study population

This study retrospectively selected patients who were admitted to Yichun People's Hospital from January 2021 to October 2024, diagnosed with STEMI [20]. Inclusion criteria: (1) age > 18 years; (2) successful PCI treatment within 12 h of symptom onset (TIMI = 3); (3) complete clinical data. Exclusion criteria: (1) history of myocardial infarction or coronary artery bypass grafting (CABG); (2) hemodialysis or chronic renal failure; (3) inflammatory diseases or malignant neoplasms; (4) exposure to other radiographic contrast agents or nephrotoxic medications [21] within 48 h before or 72 h after the procedure. The study flowchart was shown in Fig. 1. The study protocol was approved by the Ethics Committee of Yichun People's Hospital and was in compliance with the Helsinki

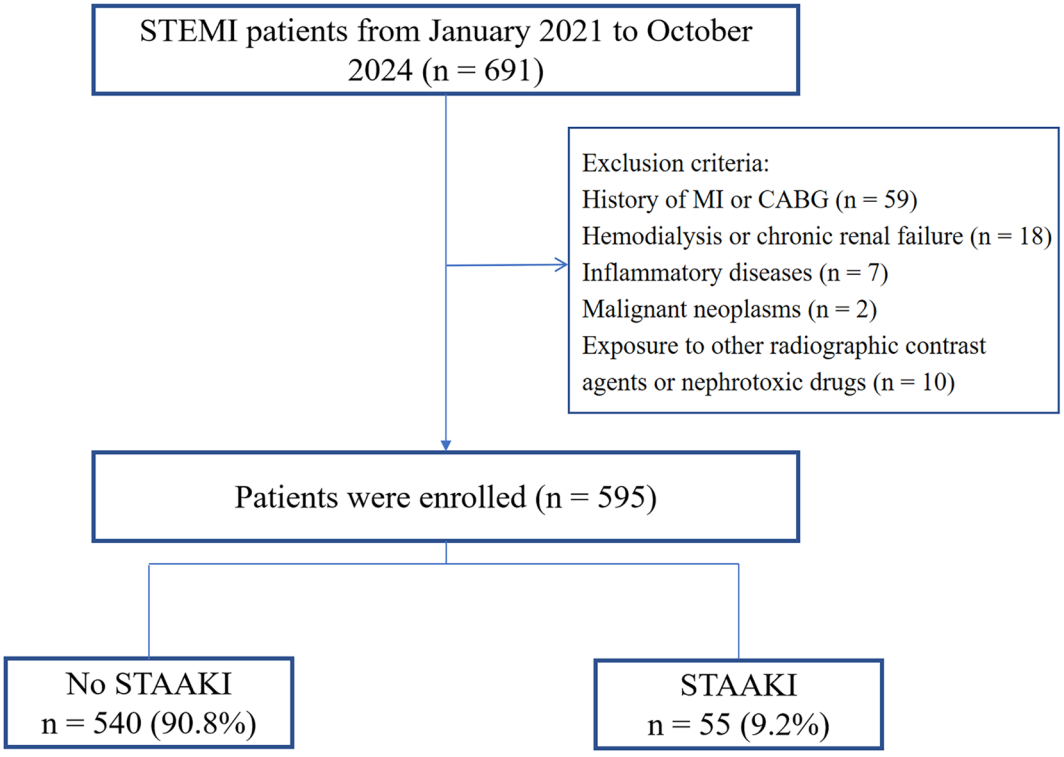

Fig. 1 The study flowchart. CABG = coronary artery bypass grafting; STAAKI = STEMI-induced acute kidney injury; STEMI = ST-segment elevation myocardial infarction

Declaration (Ethics number: 2024 – 304). Given that this study was a retrospective observational study with no harm to patients, informed consent was waived.

Data collection

Data on age, gender, risk factors (including smoking, hypertension, diabetes, and chronic kidney disease) were collected and recorded through the electronic medical record system. Serum creatinine (Scr) levels before PCI, and Scr measurements taken 48–72 h after contrast agent exposure were collected. STAAKI was defined as a Scr increase of at least 50% or 0.3 mg/dL within 48–72 h following contrast exposure [22]. In addition, other blood test results during hospitalization, including ABG, HbA1c, total cholesterol (TC), triglycerides (TG), estimated glomerular filtration rate (eGFR), peak C-reactive protein (CRP), peak high-sensitivity troponin T (hsTnT), and peak N-terminal pro B-type natriuretic peptide (NT-proBNP), were also collected and recorded. According to previous literature, A1C-derived average glucose (ADAG, mmol/L) = (1.59×HbA1c) – 2.59, and GG (mmol/L) = ABG - ADAG [23]. All PCI-related procedures were performed according to the STEMI guideline [20].

Statistical analysis

Data were analyzed using SPSS 27.0 (IBM, Chicago, USA) and R (version 4.3.1). The Kolmogorov-Smirnov test was used to assess data normality. Continuous variables with normal distribution were presented as mean ± standard deviation (SD) and analyzed using t-tests. Non-normally distributed continuous variables were expressed as median (Q25, Q75) and analyzed using the Mann-Whitney U test. Categorical variables were presented as frequencies and percentages, and analyzed using the χ^2 test. Receiver operating characteristic (ROC) curve analysis was performed to assess the diagnostic value of GG for STAAKI in STEMI patients. Comparisons between the areas under the curve (AUC) were made using the Delong test. Logistic regression analysis was used to identify the risk factors associated with STAAKI. To examine the dose-response relationship between GG and STAAKI, restricted cubic splines (RCS) were employed. The predictive accuracy of the models was assessed using the net reclassification index (NRI) and integrated discrimination improvement (IDI). A p-value < 0.05 was considered statistically significant.

Results

Patient characteristics

This study included 595 patients, of which 24.0% were female, with a mean age of 63.17 ± 13.18 years. The incidence of STAAKI was 9.2%. Patients in the STAAKI group had higher ABG, GG (t- statistic = 6.89), CRP, and NT-proBNP, and a higher prevalence of diabetes, use of diuretics and left anterior descending artery (LAD) lesion. Additionally, they had a lower left ventricular ejection fraction (LVEF) compared to those in the No STAAKI group. All differences were statistically significant ($P < 0.05$) (Table 1).

Logistic regression analysis of STAAKI

Univariate logistic regression revealed that ABG, GG, NT-proBNP, diabetes, LAD, diuretics and LVEF were significantly associated with the development of STAAKI ($P < 0.05$). After adjusting GG, NT-proBNP, diabetes, LAD, diuretics and LVEF, multivariate logistic regression showed LVEF (OR per 1% increase = 0.931, 95% CI: 0.895 ~ 0.969), NT-proBNP (OR per 1 pg/mL increase = 1.579, 95% CI: 1.212 ~ 2.057), and GG (OR per 1 mmol/L increase = 1.379, 95% CI: 1.223 ~ 1.554) as independent predictors of STAAKI (Table 2). RCS analysis indicated a linear dose-response relationship between GG and STAAKI both before and after adjustments (using the logistic regression), suggesting that higher GG levels are associated with an increased risk of STAAKI (Fig. 2). In the subgroup analysis, univariate logistic regression showed GG (OR per 1 mmol/L increase = 1.614, 95% CI: 1.238 ~ 2.105, $P < 0.001$) was associated with STAAKI in patients with diabetes. In patients without diabetes, GG (OR per 1 mmol/L increase = 1.365, 95% CI: 1.199 ~ 1.554, $P < 0.001$) was also associated with STAAKI (Supplementary Table 1).

ROC analysis of STAAKI

ROC analysis demonstrated that the AUC for LVEF, ABG, NT-proBNP, diabetes, and GG in predicting STAAKI were 0.673, 0.654, 0.710, 0.565, and 0.734, respectively ($P < 0.05$). The optimal cutoff value for GG was 0.603 mmol/L, yielding a sensitivity of 69.1% and specificity of 74.1%. The Delong test suggested that the AUC of GG was larger than that of ABG ($Z = 3.129$, $P = 0.002$) and diabetes ($Z = 3.116$, $P = 0.002$). (Supplementary Table 2, Table 3; Fig. 3). In the subgroup analysis, ROC analysis showed the AUC for GG was 0.717 ($P = 0.001$), the optimal cutoff value was 0.740 mmol/L, yielding a sensitivity of 66.7% and specificity of 77.9% in patients with diabetes. In patients without diabetes, the AUC for GG was 0.750 ($P < 0.001$), the optimal cutoff value was 0.459 mmol/L, yielding a sensitivity of 73.5% and specificity of 70.8% (Supplementary Table 3).

Predictive accuracy of the models

A baseline model (LVEF, and NT-proBNP) was constructed based on the multivariate logistic regression. ROC analysis showed that the baseline model had an AUC of 0.746 (95% CI: 0.689 ~ 0.803), with a sensitivity of 81.8% and specificity of 59.8%. The new model that

Table 1 Patient characteristics

	Total (*n* = 595)	**No STAAKI (*n* = 540)**	**STAAKI (*n* = 55)**	***P***
Age, years	63.17 ± 13.18	63.06 ± 13.39	64.25 ± 10.98	0.453
Female, n (%)	143 (24.03)	126 (23.33)	17 (30.91)	0.210
Heart rate, bpm	79.92 ± 14.50	79.82 ± 14.61	80.93 ± 13.49	0.589
SBP, mmHg	127.71 ± 20.13	127.71 ± 20.26	127.76 ± 18.97	0.984
DBP, mmHg	79.11 ± 14.00	79.08 ± 14.10	79.40 ± 13.13	0.870
BMI, kg/m^2	24.58 ± 3.85	24.51 ± 3.88	25.27 ± 3.49	0.164
Smoking, n (%)	276 (46.39)	256 (47.41)	20 (36.36)	0.118
Hypertension, n (%)	264 (44.37)	239 (44.26)	25 (45.45)	0.865
Diabetes, n (%)	157 (26.39)	136 (25.19)	21 (38.18)	0.037
CKD, n (%)	21 (3.53)	20 (3.70)	1 (1.82)	0.735
HGB, g/L	139.82 ± 17.02	139.92 ± 17.02	138.84 ± 17.12	0.654
Plt, 10^9/L	216.62 ± 58.71	217.39 ± 59.62	209.09 ± 48.67	0.318
Creatinine, μmol/L	67.24 ± 20.32	67.50 ± 20.61	64.67 ± 17.25	0.326
eGFR, mL/min/1.73 m^2	102.38 ± 20.63	102.56 ± 20.81	100.65 ± 18.84	0.512
Total cholesterol, mmol/L	4.26 ± 1.01	4.25 ± 1.02	4.38 ± 0.94	0.388
Triglycerides, mmol/L	1.51 ± 1.13	1.52 ± 1.17	1.45 ± 0.72	0.685
HDL-C, mmol/L	0.98 ± 0.24	0.98 ± 0.24	1.00 ± 0.16	0.516
LDL-C, mmol/L	2.74 ± 0.87	2.72 ± 0.88	2.90 ± 0.85	0.166
ABG, mmol/L	7.86 ± 3.17	7.67 ± 2.93	9.68 ± 4.63	0.003
HBA1c, %	6.55 ± 1.71	6.56 ± 1.74	6.40 ± 1.29	0.488
ADAG, mmol/L	7.82 ± 2.71	7.85 ± 2.77	7.58 ± 2.05	0.488
GG, mmol/L	0.04 ± 2.42	-0.17 ± 2.24	2.10 ± 3.09	< 0.001
Peak hs-CRP, mg/L	2.20 (0.50, 7.65)	2.10 (0.50, 7.35)	2.70 (1.50, 9.35)	0.026
Peak hs-TnT, ng/L	528.0 (96.8, 1907.5)	492.8 (82.3, 1972.5)	1041.0 (157.0, 1616.0)	0.104
Peak NT-proBNP, pg/mL	1344.0 (564.6, 3033.6)	1200.0 (507.8, 2749.8)	3000.00 (1706.5, 4387.0)	< 0.001
IABP, n (%)	16 (2.69)	14 (2.59)	2 (3.64)	0.985
LVEF, %	51.89 ± 6.99	52.32 ± 6.78	47.73 ± 7.75	< 0.001
Killip class, n (%)				0.137
I	511 (85.88)	468 (86.67)	43 (78.18)	
II	28 (4.71)	26 (4.81)	2 (3.64)	
III	1 (0.17)	1 (0.19)	0 (0.00)	
IV	55 (9.24)	45 (8.33)	10 (18.18)	
IRA-LAD, n(%)	296 (49.75)	261 (48.33)	35 (63.64)	0.031
IRA-LCX n(%)	58 (9.75)	53 (9.81)	5 (9.09)	0.863
IRA-RCA, n(%)	238 (40.00)	223 (41.30)	15 (27.27)	0.043
IRA-Left main, n(%)	3 (0.50)	3 (0.56)	0 (0.00)	1.000
Aspirin, n(%)	593 (99.66)	538 (99.63)	55 (100.00)	1.000
P2Y12, n(%)	594 (99.83)	539 (99.81)	55 (100.00)	1.000
Statins, n(%)	592 (99.50)	537 (99.44)	55 (100.00)	1.000
ACEI/ARB, n(%)	272 (45.71)	246 (45.56)	26 (47.27)	0.808
β-blockers, n(%)	516 (86.72)	469 (86.85)	47 (85.45)	0.771
Nitrates, n(%)	237 (39.83)	220 (40.74)	17 (30.91)	0.156
Heparin, n(%)	493 (82.86)	448 (82.96)	45 (81.82)	0.830
Diuretics, n(%)	298 (50.08)	263 (48.70)	35 (63.64)	0.035

The conversion factor for converting glucose: 1 mmol/L = 18.018 mg/dL

BMI = body Mass Index; IABP = intra-aortic balloon pump; LVEF = left ventricular ejection fraction; CKD = chronic kidney disease; SBP = systolic blood pressure; DBP = diastolic blood pressure; LAD = left anterior descending; LCX = left circumflex artery; RCA = right coronary artery; ACEI = angiotensin-converting-enzyme inhibitor; ARB = angiotensin II receptor blocker; HDL-C = high-density leptin cholesterol; LDL-C = low-density leptin cholesterol; hs-CRP = high sensitivity C-reactive protein; hs-TnT = high sensitivity troponin T; NT-proBNP = N-terminal pro-B-type natriuretic peptide; STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury; GG = glycemic gap; ABG = admission blood glucose

Table 2 Univariate and multivariate regression analysis for STAAKI

	OR per 1 unit increase (95%CI)	*P*	OR per 1 unit increase (95%CI)	*P*
Age, years	1.007 (0.986 ~ 1.029)	0.520		
Female, n (%)	1.470 (0.802 ~ 2.694)	0.213		
Heart rate, bpm	1.005 (0.986 ~ 1.024)	0.588		
SBP, mmHg	1.000 (0.986 ~ 1.014)	0.984		
DBP, mmHg	1.002 (0.982 ~ 1.022)	0.870		
BMI, kg/m^2	1.052 (0.979 ~ 1.131)	0.164		
Smoking, n (%)	0.634 (0.357 ~ 1.126)	0.120		
Hypertension, n (%)	1.050 (0.601 ~ 1.832)	0.865		
Diabetes, n (%)	1.835 (1.030 ~ 3.269)	0.039		
CKD, n (%)	0.481 (0.063 ~ 3.658)	0.480		
HGB, g/L	0.996 (0.980 ~ 1.013)	0.653		
Plt, 10^9/L	0.998 (0.993 ~ 1.002)	0.318		
Peak hs-CRP, mg/L	1.007 (1.000 ~ 1.015)	0.058		
Creatinine, μmol/L	0.993 (0.978 ~ 1.007)	0.325		
eGFR, mL/min/1.73 m^2	0.996 (0.983 ~ 1.009)	0.512		
Peak hs-TnT, ng/L	1.144 (0.982 ~ 1.334)	0.085		
Peak NT-proBNP, pg/mL	1.658 (1.311 ~ 2.097)	< 0.001	1.579 (1.212 ~ 2.057)	0.001
Total cholesterol, mmol/L	1.126 (0.860 ~ 1.474)	0.388		
Triglycerides, mmol/L	0.945 (0.718 ~ 1.243)	0.685		
HDL-C, mmol/L	1.323 (0.415 ~ 4.217)	0.637		
LDL-C, mmol/L	1.241 (0.914 ~ 1.686)	0.166		
ABG, mmol/L	1.150 (1.073 ~ 1.233)	< 0.001		
HBA1c	0.940 (0.789 ~ 1.119)	0.487		
ADAG, mmol/L	0.962 (0.862 ~ 1.073)	0.487		
GG, mmol/L	1.389 (1.238 ~ 1.559)	< 0.001	1.379 (1.223 ~ 1.554)	< 0.001
IABP, n (%)	1.418 (0.314 ~ 6.407)	0.650		
LVEF, %	0.919 (0.886 ~ 0.953)	< 0.001	0.931 (0.895 ~ 0.969)	< 0.001
Killip class > 1, n (%)	1.814 (0.913 ~ 3.603)	0.089		
IRA-LAD, n(%)	1.871 (1.053 ~ 3.324)	0.033		
IRA-LCX n(%)	0.919 (0.351 ~ 2.405)	0.863		
IRA-RCA, n(%)	0.533 (0.287 ~ 0.989)	0.046		
ACEI/ARB, n(%)	1.071 (0.615 ~ 1.868)	0.808		
β-blockers, n(%)	0.889 (0.404 ~ 1.960)	0.771		
Nitrates, n(%)	0.651 (0.358 ~ 1.182)	0.158		
Heparin, n(%)	0.924 (0.449 ~ 1.900)	0.830		
Diuretics, n(%)	1.843 (1.037 ~ 3.275)	0.037		

ABG and RCA were not included in the multivariate regression analysis considering the interference of correlation BMI = body Mass Index; IABP = intra-aortic balloon pump; LVEF = left ventricular ejection fraction; CKD = chronic kidney disease; SBP = systolic blood pressure; DBP = diastolic blood pressure; LAD = left anterior descending; LCX = left circumflex artery; RCA = right coronary artery; ACEI = angiotensin-converting-enzyme inhibitor; ARB = angiotensin II receptor blocker; HDL-C = high-density leptin cholesterol; LDL-C = low-density leptin cholesterol; hs-CRP = high sensitivity C-reactive protein; hs-TnT = high sensitivity troponin T; NT-proBNP = N-terminal pro-B-type natriuretic peptide; STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury; GG = glycemic gap; ABG = admission blood glucose

integrating GG (LVEF, NT-proBNP, and GG) had an AUC of 0.809 (95% CI: 0.755 ~ 0.863), with a sensitivity of 76.4% and specificity of 75.2%. The Delong test suggested that the AUC of the new model was significantly larger than that of the baseline model ($Z = 2.77$, $P = 0.006$). NRI and IDI for the new model were 0.780 (0.5157 ~ 1.0446), $P < 0.001$, and 0.095 (0.0493 ~ 0.1397), $P < 0.001$, respectively. These findings indicate that the new model significantly improves the risk model for STAAKI in STEMI patients (Table 4; Fig. 4, Supplementary Table 4).

Discussion

The main findings of this study are as follows: first, elevated GG is an independent risk factor for STAAKI in STEMI patients; second, there is a linear dose-response relationship between GG and STAAKI; and third, integrating GG significantly improves the risk model for STAAKI.

Compared to the general population, the incidence of STAAKI after coronary angiography is significantly higher in patients with acute myocardial infarction (AMI) and diabetes [24, 25]. In our study, the incidence

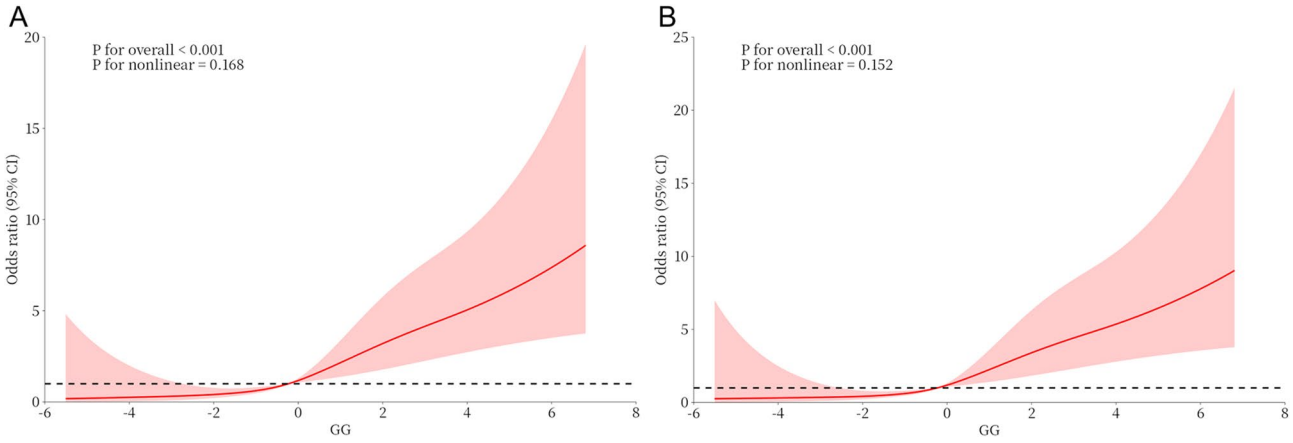

Fig. 2 Dose-response relationship between GG and STAAKI. (**A**) a unadjusted dose-response relationship between GG and STAAKI; (**B**) an adjusted dose-response relationship between GG and STAAKI. STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury; GG = glycemic gap

Table 3 ROC curve for STAAKI

	AUC	95%CI	*P*	Cut-off	Sensitivity	Specificity
ABG, mmol/L	0.654	0.578 ~ 0.731	< 0.001	6.64	0.764	0.498
GG, mmol/L	0.734	0.658 ~ 0.809	< 0.001	0.603	0.691	0.741
Diabetes	0.565	0.482 ~ 0.648	0.112	-	0.382	0.748

GG = glycemic gap; ABG = admission blood glucose; STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury

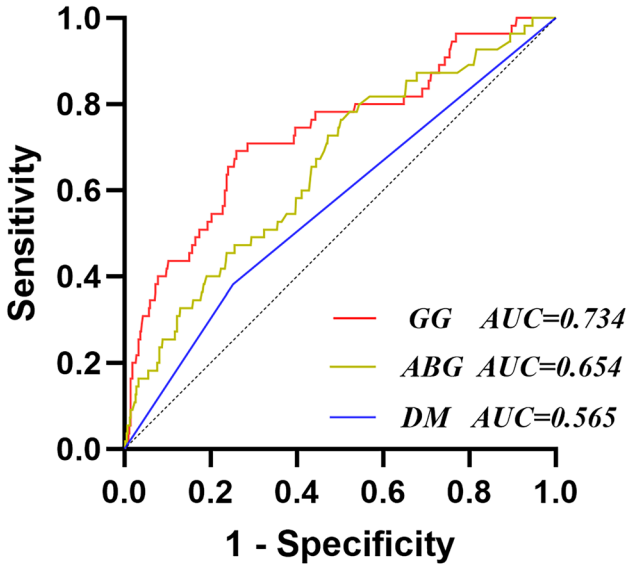

Fig. 3 Receiver operating characteristic analysis (ROC) of GG for identifying STAAKI. STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury; GG = glycemic gap; ABG = admission blood glucose; DM = diabetes

of STAAKI in STEMI patients after primary PCI was 9.2%, which is consistent with previous research findings [26, 27]. Clinically, the management of STAAKI remains focused on prevention. Therefore, identifying more risk factors for STAAKI and optimizing risk stratification holds significant clinical value.

SIH generally refers to a transient increase in blood glucose levels caused by the activation of the sympathetic

Table 4 Incremental value of GG for STAAKI

	NRI		IDI	
	Estimate (95% CI)	*P*	Estimate (95% CI)	*P*
LVEF + NT-proBNP	Reference	-	Reference	-
LVEF + NT-proBNP + GG	0.780 (0.5157 ~ 1.0446)	< 0.001	0.095 (0.0493 ~ 0.1397)	< 0.001

NT-proBNP = N-terminal pro-B-type natriuretic peptide; LVEF = left ventricular ejection fraction; GG = glycemic gap; STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury

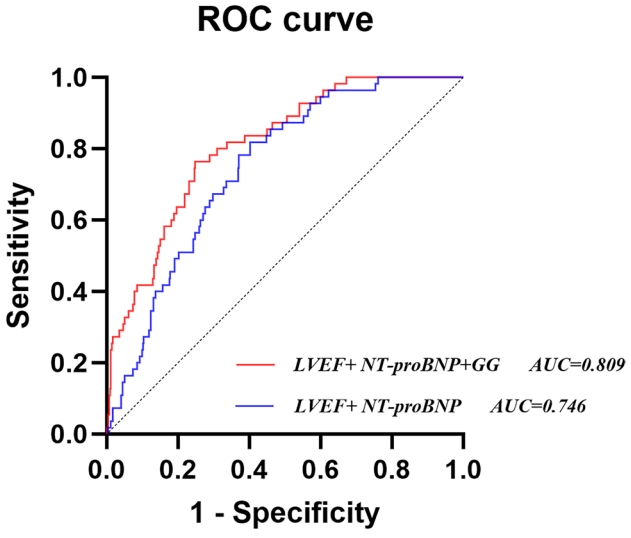

Fig. 4 Receiver operating characteristic analysis (ROC) of models for identifying STAAKI. STAAKI = ST-segment elevation myocardial infarction-induced acute kidney injury; GG = glycemic gap; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide

nervous system during critical conditions. It has been identified as an important indicator of the severity of various diseases and conditions, including surgery, acute ischemic stroke, and acute myocardial infarction (AMI) [28, 29]. Currently, the main indicators for SIH include ABG and GG. However, ABG levels are influenced by both acute physiological stress and chronic baseline blood glucose levels, and therefore, they do not accurately reflect the extent of SIH in acute disease states [12, 13]. GG quantifies the relative increase in chronic blood glucose during acute disease states, truly reflecting the acute hyperglycemic condition, and has a strong correlation with disease severity and prognosis [14–19]. For AKI patients, an elevated ABG level does not necessarily indicate stress-induced hyperglycemia [30]. A large body of evidence has confirmed that GG levels are more predictive of adverse cardiovascular outcomes than ABG levels in AMI patients [17–19, 31, 32]. However, the relationship between GG levels and STAAKI in STEMI patients remains unclear. Our study innovatively found that elevated GG was an independent risk factor for STAAKI in STEMI patients, and RCS showed a linear dose-response relationship between GG and STAAKI. In fact, the close relationship between SIH and renal injury has been established in many studies. A rapid increase in blood glucose can cause osmotic diuresis, leading to renal hypoperfusion and damage [33]. Moreover, SIH can trigger abnormal activation of the sympathetic nervous system, excessive release of catecholamines and cortisol, oxidative stress, inflammation, endothelial dysfunction, thrombosis, and ischemia-reperfusion injury, all of which may result in further cardiac injury, reduced renal reperfusion, and ultimately lead to AKI [34–36]. In our study, both ABG and diabetes were also found to be associated with the risk of STAAKI, but consistent with previous studies [17–19], the ROC results indicated that GG had a significantly stronger predictive ability for STAAKI than ABG and diabetes. Furthermore, our study also demonstrated that GG could notably improve the risk model for STAAKI. Therefore, our findings provide additional information for risk stratification of STAAKI after PCI in STEMI patients. GG, as a simple and effective biomarker, could be widely used for screening high-risk populations of STAAKI after PCI in STEMI patients. Clinically, patients with elevated GG levels may require more attention and strategic management, such as hydration and choice of surgical approach.

Limitations

First, due to the inherent limitations of retrospective studies, the current findings could not demonstrate an exact causal relationship between elevated GG and STAAKI. Second, the sample size of the present study was valid and all were patients diagnosed with STEMI; therefore, some of the findings may need to be replicated in other diseases. Third, although our study has demonstrated that there is a relationship between GG and STAAKI. However, the specific mechanism regarding the risk of elevated GG and the occurrence of STAAKI is not clear, which may require more basic research to clarify.

Conclusions

GG is an independent risk factor for the occurrence of STAAKI after PCI in STEMI patients, and integrating GG can significantly improve risk modeling regarding STAAKI.

Abbreviations

PCI	Percutaneous coronary intervention
STEMI	ST-segment elevation myocardial infarction
STAAKI	STEMI-induced acute kidney injury
SIH	Stress-induced hyperglycemia
ABG	Admission blood glucose
GG	Glycemic gap
AMI	Acute myocardial infarction
MACE	Major adverse cardiovascular events

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-04167-3.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Acknowledgements

None.

Author contributions

XFZ, ZYL, YL, QHY, SWW, YS, and SQ collected or analyzed the data. XFZ and YL wrote the manuscript. XFZ, ZYL and JPX directed the entire research work and corrected the articles.

Funding

This work was partly supported by Medical and Health Technology Project of Hangzhou (Grant B20231533) and Zhejiang Provincial Medical and Health Technology Project (Grant 2024KY1447).

Data availability

The datasets generated during and/or analyzed during the current study are available by request form Ziyun Luo (312179045@qq.com) or Jianping Xu (xjp_hh@163.com).

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committee of Yichun People's Hospital and was in compliance with the Helsinki Declaration (Ethics number: 2024 – 304). Given that this study was a retrospective observational study with no harm to patients, informed consent was waived.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 16 February 2025 / Accepted: 8 May 2025
Published online: 15 May 2025

References

1. Dérimay F, Rioufol G. ST-segment elevation myocardial infarction and primary percutaneous coronary intervention: can we go beyond the clock?? Circ Cardiovasc Imaging. 2022;15(11):e014906. https://doi.org/10.1161/CIRCIMAGING.122.014906.
2. Mettler FA Jr, Mahesh M, Bhargavan-Chatfield M, et al. Patient exposure from radiologic and nuclear medicine procedures in the united States: procedure volume and effective dose for the period 2006–2016. Radiology. 2020;295(2):418–27. https://doi.org/10.1148/radiol.2020192256.
3. Rear R, Bell RM, Hausenloy DJ. Contrast-induced nephropathy following angiography and cardiac interventions. Heart. 2016;102(8):638–48. https://doi.org/10.1136/heartjnl-2014-306962.
4. Schmucker J, Fach A, Becker M, et al. Predictors of acute kidney injury in patients admitted with ST-elevation myocardial infarction - results from the Bremen STEMI-Registry. Eur Heart J Acute Cardiovasc Care. 2018;7(8):710–22. https://doi.org/10.1177/2048872617708975.
5. Mehran R, Dangas GD, Weisbord SD. Contrast-Associated acute kidney injury. N Engl J Med. 2019;380(22):2146–55. https://doi.org/10.1056/NEJMra1805256.
6. Chandiramani R, Cao D, Nicolas J, Mehran R. Contrast-induced acute kidney injury. Cardiovasc Interv Ther. 2020;35(3):209–17. https://doi.org/10.1007/s12928-020-00660-8.
7. Mifsud S, Schembri EL, Gruppetta M. Stress-induced hyperglycaemia. Br J Hosp Med (Lond). 2018;79(11):634–9. https://doi.org/10.12968/hmed.2018.79.11.634.
8. Stranders I, Diamant M, van Gelder RE, et al. Admission blood glucose level as risk indicator of death after myocardial infarction in patients with and without diabetes mellitus. Arch Intern Med. 2004;164(9):982–8. https://doi.org/10.1001/archinte.164.9.982.
9. Deckers JW, van Domburg RT, Akkerhuis M, Nauta ST. Relation of admission glucose levels, short- and long-term (20-year) mortality after acute myocardial infarction. Am J Cardiol. 2013;112(9):1306–10. https://doi.org/10.1016/j.amjcard.2013.06.007.
10. Schuetz P, Kennedy M, Lucas JM, et al. Initial management of septic patients with hyperglycemia in the noncritical care inpatient setting. Am J Med. 2012;125(7):670–8. https://doi.org/10.1016/j.amjmed.2012.03.001.
11. Tiruvoipati R, Chiezey B, Lewis D, et al. Stress hyperglycemia May not be harmful in critically ill patients with sepsis. J Crit Care. 2012;27(2):153–8. https://doi.org/10.1016/j.jcrc.2011.06.011.
12. Chen G, Li M, Wen X, et al. Association between stress hyperglycemia ratio and In-hospital outcomes in elderly patients with acute myocardial infarction. Front Cardiovasc Med. 2021;8:698725. https://doi.org/10.3389/fcvm.2021.698725. Published 2021 Jul 20.
13. Yang J, Zheng Y, Li C, et al. The impact of the stress hyperglycemia ratio on Short-term and Long-term poor prognosis in patients with acute coronary syndrome: insight from a large cohort study in Asia. Diabetes Care. 2022;45(4):947–56. https://doi.org/10.2337/dc21-1526.
14. Dorn AY, Sun PY, Sanossian N, et al. Admission glycemic gap in the assessment of patients with intracerebral hemorrhage. Clin Neurol Neurosurg. 2021;208:106871. https://doi.org/10.1016/j.clineuro.2021.106871.
15. Lou R, Jiang L, Zhu B. Effect of glycemic gap upon mortality in critically ill patients with diabetes. J Diabetes Investig. 2021;12(12):2212–20. https://doi.org/10.1111/jdi.13606.
16. Jensen AV, Baunbæk Egelund G, Bang Andersen S, et al. The glycemic gap and 90-Day mortality in Community-acquired pneumonia. A prospective cohort study. Ann Am Thorac Soc. 2019;16(12):1518–26. https://doi.org/10.1513/AnnalsATS.201901-007OC.
17. Liu HH, Zhang M, Guo YL, et al. Association of acute glycemic parameters at admission with cardiovascular mortality in the oldest old with acute myocardial infarction. J Geriatr Cardiol. 2024;21(3):349–58. https://doi.org/10.26599/1671-5411.2024.03.008.
18. Zhu Y, Liu K, Meng S et al. Augmented glycaemic gap is a marker for an increased risk of post-infarct left ventricular systolic dysfunction. Cardiovasc Diabetol. 2020;19(1):101. Published 2020 Jul 4. https://doi.org/10.1186/s12933-020-01075-8
19. Liao WI, Lin CS, Lee CH, et al. An elevated glycemic gap is associated with adverse outcomes in diabetic patients with acute myocardial infarction. Sci Rep. 2016;6:27770. https://doi.org/10.1038/srep27770. Published 2016 Jun 13.
20. Ibanez B, James S, Agewall S, et al. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European society of cardiology (ESC). Eur Heart J. 2018;39(2):119–77. https://doi.org/10.1093/eurheartj/ehx393.
21. Patel JB, Sapra A. Nephrotoxic medications. StatPearls. Treasure Island (FL). 2023;21. StatPearls Publishing.
22. van der Molen AJ, Reimer P, Dekkers IA, et al. Post-contrast acute kidney injury - Part 1: definition, clinical features, incidence, role of contrast medium and risk factors: recommendations for updated ESUR contrast medium safety committee guidelines. Eur Radiol. 2018;28(7):2845–55. https://doi.org/10.1007/s00330-017-5246-5.
23. Wu S, Yang YM, Zhu J, et al. Impact of glycemic gap on 30-day adverse outcomes in patients with acute ST-segment elevation myocardial infarction. Atherosclerosis. 2022;360:34–41. https://doi.org/10.1016/j.atherosclerosis.2022.10.003.
24. Toprak O, Cirit M, Yesil M, et al. Impact of diabetic and pre-diabetic state on development of contrast-induced nephropathy in patients with chronic kidney disease. Nephrol Dial Transpl. 2007;22(3):819–26. https://doi.org/10.1093/ndt/gfl636.
25. Rihal CS, Textor SC, Grill DE, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation. 2002;105(19):2259–64. https://doi.org/10.1161/01.cir.0000016043.87291.33.
26. Luo Z, Li Y, Xie M, Yi S, Xu S, Luo J. Soluble suppression of tumorigenicity 2 associated with contrast-induced acute kidney injury in patients with STEMI. Int Urol Nephrol Published Online September. 2024;12. https://doi.org/10.1007/s11255-024-04204-4.
27. Zhu Y, He H, Qiu H, Zhang X, Wang L, Li W. Prognostic nutritional index combined with Triglyceride-Glucose index to contrast a nomogram for predicting contrast-Induced kidney injury in type 2 diabetes mellitus patients with acute coronary syndrome after percutaneous coronary intervention. Clin Interv Aging. 2023;18:1663–73. https://doi.org/10.2147/CIA.S429957. Published 2023 Oct 3.
28. Marik PE, Raghavan M. Stress-hyperglycemia, insulin and Immunomodulation in sepsis. Intensive Care Med. 2004;30(5):748–56. https://doi.org/10.1007/s00134-004-2167-y.
29. Li WA, Moore-Langston S, Chakraborty T, Rafols JA, Conti AC, Ding Y. Hyperglycemia in stroke and possible treatments. Neurol Res. 2013;35(5):479–91. https://doi.org/10.1179/1743132813Y.0000000209.
30. Fiaccadori E, Sabatino A, Morabito S, et al. Hyper/hypoglycemia and acute kidney injury in critically ill patients. Clin Nutr. 2016;35(2):317–21. https://doi.org/10.1016/j.clnu.2015.04.006.
31. Wang CH, Chang JL, Huang CH, et al. The association between long-term glycaemic control, glycaemic gap and neurological outcome of in-hospital cardiac arrest in diabetics: A retrospective cohort study. Resuscitation. 2018;133:18–24. https://doi.org/10.1016/j.resuscitation.2018.09.017.
32. Kojima T, Hikoso S, Nakatani D, et al. Impact of hyperglycemia on Long-Term outcome in patients with ST-Segment elevation myocardial infarction. Am J Cardiol. 2020;125(6):851–9. https://doi.org/10.1016/j.amjcard.2019.12.034.
33. Marik PE, Preiser JC. Toward Understanding tight glycemic control in the ICU: a systematic review and metaanalysis. Chest. 2010;137(3):544–51. https://doi.org/10.1378/chest.09-1737.
34. Monnier L, Mas E, Ginet C, et al. Activation of oxidative stress by acute glucose fluctuations compared with sustained chronic hyperglycemia in patients with type 2 diabetes. JAMA. 2006;295(14):1681–7. https://doi.org/10.1001/jama.295.14.1681.
35. Worthley MI, Holmes AS, Willoughby SR, et al. The deleterious effects of hyperglycemia on platelet function in diabetic patients with acute coronary syndromes mediation by superoxide production, resolution with intensive insulin administration. J Am Coll Cardiol. 2007;49(3):304–10. https://doi.org/10.1016/j.jacc.2006.08.053.

36. Ceriello A, Esposito K, Piconi L, et al. Oscillating glucose is more deleterious to endothelial function and oxidative stress than mean glucose in normal and type 2 diabetic patients. Diabetes. 2008;57(5):1349–54. https://doi.org/10.2337/db08-0063.

Publisher's note