Ma *et al. Thrombosis Journal* (2025) 23:47
https://doi.org/10.1186/s12959-025-00729-5

Thrombosis Journal

RESEARCH Open Access

Prognostic significance of stress hyperglycemia ratio in patients with type 2 diabetes mellitus and acute coronary syndromes

Xiaoteng Ma[1†], Huijun Chu[2†], Yan Sun[1], Yujing Cheng[1], Dai Zhang[1], Lixia Yang[1], Zhijian Wang[1], Xiaoli Liu[1] and Yujie Zhou[1*]

Abstract

Background Prognostic significance of stress hyperglycemia ratio (SHR) has not been well studied in patients with type 2 diabetes mellitus (T2DM) and acute coronary syndromes (ACS).

Methods We prospectively measured admission fasting blood glucose (AFBG) and glycated hemoglobin A1c (HbA1c), and retrospectively calculated the stress hyperglycemia ratio (SHR, = AFBG/[1.59 × HbA1c (%) − 2.59]) in 791 patients with T2DM and ACS undergoing percutaneous coronary intervention (PCI). The primary endpoint was defined as major adverse cardiovascular and cerebrovascular events (MACCE), including all-cause mortality, non-fatal stroke, non-fatal myocardial infarction, and unplanned repeat coronary revascularization.

Results The mean age of the study population was 61 ± 10 years, and 72.8% were male. Over a median follow-up of 927 days, 194 patients developed at least one primary endpoint event. The follow-up incidence of MACCE increased in parallel with SHR tertiles (15.6%, 21.9%, and 36.1%, respectively; P for trend < 0.001). The Cox proportional hazards regression analysis adjusted for multiple confounding factors showed hazard ratios for MACCE of 1.525 (95% CI: 1.009–2.305; $P = 0.045$) for the middle tertile and 2.525 (95% CI: 1.729–3.687; $P < 0.001$) for the highest tertile of SHR, with the lowest tertile as the reference. The addition of SHR to the baseline reference prediction model improved model predictive performance markedly (C-statistic: increased from 0.704 to 0.721; cNRI: 0.176 [95% CI: 0.063–0.282], $P = 0.002$; IDI: 0.030 [95% CI: 0.009–0.063], $P = 0.002$).

Conclusion SHR was independently and significantly associated with adverse cardiovascular outcomes in T2DM and ACS patients who underwent PCI, and had an incremental effect on the predictive ability of the baseline reference prediction model.

Keywords Stress hyperglycemia ratio, Major adverse cardiovascular and cerebrovascular events, Type 2 diabetes mellitus, Acute coronary syndromes, Percutaneous coronary intervention

†Xiaoteng Ma and Huijun Chu contributed equally to this paper.

*Correspondence:
Yujie Zhou
azzyj12@163.com

[1]Department of Cardiology, Beijing Anzhen Hospital, Capital Medical University, Beijing 100029, China
[2]Department of Anesthesia, Beijing Anzhen Hospital, Capital Medical University, Beijing, China

Introduction

Acute coronary syndromes (ACS) refer to a wide spectrum of obstructive coronary artery diseases, which are characterized by coronary plaque rupture/erosion and thrombus formation leading to a sudden reduction in blood supply to the heart, and include unstable angina (UA), non-ST segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Each year, an estimated more than 7 million people in the world are diagnosed with ACS 1. It has been shown that ACS are associated with high cardiovascular morbidity and mortality and impose a substantial financial burden on health care system 1. Of note, diabetes is one of the most important accomplices of ACS. Patients with ACS who have known diabetes or newly diagnosed diabetes are at higher risk of cardiovascular events and mortality than those who do not have diabetes 2, 3. Guideline-directed medical therapy and the development of percutaneous coronary intervention (PCI) techniques and materials have markedly reduced major adverse cardiovascular and cerebrovascular events (MACCE) among diabetic patients with ACS; however, these patients receiving the "so-called" optimal treatment still have high residual cardiovascular risk. Therefore, identification and management of previously unrecognized potential risk factors is critical to further improve the prognosis of such patients.

Individualized glucose-lowering therapy is generally essential for blood glucose control and stabilization in diabetic patients. Nonetheless, acute hyperglycemia on admission is common in diabetic patients with ACS and is associated with adverse clinical outcomes 4, 5. Hyperglycemia following an ACS event appears to be associated with both background glycemia and multiple stress mechanisms. A considerable number of previous studies relied on blood glucose levels on admission to identify stress hyperglycemia, with the caveat that these studies mainly included patients without diabetes. In fact, absolute hyperglycemia based on blood glucose levels on admission is not exactly equivalent to stress hyperglycemia, especially in diabetic patients 6. In the strict sense, stress hyperglycemia refers to an acute increase in blood glucose levels adjusted for background glycemia, irrespective of whether a patient has previously been diagnosed with diabetes 6, 7, 8. Of note, stress hyperglycemia that occurs after acute illness in patients with diabetes is more likely to be overlooked than in patients without diabetes 6.

Changes in blood glucose levels from baseline, rather than absolute blood glucose levels, may be of value 7, 9. Stress hyperglycemia has been shown to be a better and more powerful predictor of adverse clinical outcomes than absolute hyperglycemia in multiple populations of critically ill patients, including patients with acute myocardial infarction (MI) and patients with acute cerebral infarction 9, 10, 11, 12, 13, 14. Stress hyperglycemia mentioned above can be well evaluated using the stress hyperglycemia ratio (SHR) proposed by Roberts et al., which is calculated using the admission fasting blood glucose (AFBG) levels divided by the estimated average glucose (eAG) levels derived from the glycated hemoglobin A1c (HbA1c) 14, 15. The predictive value of SHR for adverse cardiovascular outcomes is beyond doubt 16; however, its prognostic significance has not been well studied in patients with type 2 diabetes mellitus (T2DM) and ACS.

Thus, the purpose of our study was to investigate the possible association between SHR and adverse cardiovascular outcomes in patients with T2DM and ACS.

Methods

Study population

This was a retrospective analysis of data obtained from the T2DM subgroup of a single-center prospective cohort study (ChiCTR1800017417; Registration Date: July 29, 2018) that aimed to investigate the prognostic value of the Logistic Clinical SYNTAX Score and novel risk factors for MACCE in patients with ACS undergoing PCI. The prospective cohort study was approved by the institutional review board of Beijing Anzhen Hospital, Capital Medical University, and all patients gave their written informed consent before study inclusion.

The T2DM subgroup consisted of 826 patients with T2DM and ACS undergoing elective or primary PCI. Given the purpose of this analysis, we excluded patients who had previously undergone coronary artery bypass grafting (CABG), had a creatinine clearance (CrCl) of less than 30 ml/min, were currently using glucocorticoids for connective tissue disease, or had active infections on admission. Three patients who had failed follow-up were also excluded. Eventually, a total of 791 patients were included in the present analysis.

Data collection

Data on demographics, medical history, and medication history were collected for all included patients using a standard questionnaire. The levels of HbA1c, AFBG, triglycerides, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), serum creatinine (SCr), and high-sensitivity C-reactive protein (hsCRP) in the first fasting blood samples after admission were measured at the central laboratory of Beijing Anzhen Hospital. Low-density lipoprotein cholesterol (LDL-C) levels were calculated using the Friedewald equation. CrCl was calculated using the method described by Cockcroft and Gault 17. Hypertension was defined as a blood pressure ≥ 140/90 mmHg, chronic use of antihypertensive drugs, or self-reported previous diagnosis

of hypertension. Dyslipidemia was defined as fasting TC > 200 mg/dL, LDL-C > 130 mg/dL, HDL-C < 40 mg/dL, triglyceride > 150 mg/dL, chronic use of lipid-lowering drugs, or self-reported previous diagnosis of dyslipidemia. Peripheral arterial disease (PAD) was defined as vascular diseases related to the aorta and arteries, except the coronary arteries, which were accompanied by exercise-related intermittent claudication, revascularization surgery, reduced or absent pulsation, angiographic stenosis > 50%, or a combination of these characteristics. Heart failure was defined as having signs or symptoms of heart failure, being treated for heart failure, or having left ventricular ejection fraction (LVEF) of less than 40%.

Measurement of stress hyperglycemia ratio

HbA1c was used to estimate the average blood glucose before admission using the equation "eAG levels = (1.59 × HbA1c) − 2.59" proposed by Nathan et al. 18. SHR was calculated as AFBG (mmol/L) divided by eAG (mmol/L).

Endpoints and follow-up

Follow-up visits were conducted one month and every six months after discharge. Information on adverse cardiovascular outcomes was obtained by trained personnel with no knowledge of baseline characteristics through telephone contact with the patients or their family members and was determined by careful review of the corresponding medical records. The primary endpoint was defined as MACCE, including all-cause mortality, non-fatal stroke, non-fatal MI, and unplanned repeat coronary revascularization. The most severe endpoint event was selected for the primary endpoint analysis if the patients had multiple endpoint events during follow-up (death > stroke > MI). If more than one stroke, MI, or revascularization event occurred, then the first stroke, MI or revascularization event was considered. The final follow-up was conducted in November 2019.

Statistical analyses

Continuous variables were correspondingly presented as the mean ± standard deviation (SD) or the median and interquartile range (IQR) for normal or non-normal distribution where t-test or Mann-Whitney U test was used to examine differences between two groups, and ANOVA or Kruskal–Wallis H test was applied to analyze differences among three groups. Categorical variables were expressed as counts and percentages where the Chi-squared test or Fisher's exact test was used to analyze differences between groups. SHR was analyzed as a categorical variable (the lowest tertile: < 0.7443; the middle tertile: ≥ 0.7443, < 0.8698; the highest tertile: ≥ 0.8698) and as a continuous variable for its association with MACCE. Additionally, receiver operating characteristic (ROC) curve analysis and Youden's index (sensitivity + specificity - 1) were used to determine the optimal cut-off value of SHR as a continuous variable (= 0.8150) for predicting the occurrence of MACCE. The Kaplan-Meier curve and log-rank test analysis were performed to estimate cumulative MACCE rates stratified by SHR tertiles. Hazard ratios (HRs) with the corresponding 95% confidence intervals (CIs) for MACCE were calculated using Cox proportional hazards regression analyses. Variables with a univariate significance level of ≤ 0.10 were included in the multivariate Cox proportional hazards regression model. The incremental effect of adding SHR to the baseline reference prediction model that included variables with a univariate significance level of ≤ 0.10 other than SHR to predict MACCE was evaluated using the Harrell's C statistics, continuous net reclassification improvement (cNRI), and integrated discrimination improvement (IDI). Post-hoc subgroup analyses stratified by age (≥ 60 versus < 60 years), sex (male versus female), body mass index (BMI) (≥ 25 versus < 25 kg/m^2), current smoking (yes versus no), hypertension (yes versus no), type of ACS (UA versus MI), hsCRP (≥ 2 versus < 2 mg/L), and SYNTAX score (≥ 22 versus < 22) were performed to determine the consistency of the prognostic significant of SHR as a continuous variable for MACCE. Two-tailed tests were used in all statistical tests, and $P < 0.05$ was considered statistically significant. SPSS version 24.0 (IBM Corp., Armonk, New York, US) and R software version 4.1.0 (R Foundation for Statistical Computing, Beijing, China) were used for statistical analyses. GraphPad Prism version 7.0 (GraphPad Software Inc., San Diego, California, US) was used for plotting the Kaplan-Meier curve.

Results

The mean age of the study population was 61 ± 10 years, and 576 (72.8%) patients were male. The baseline characteristics of the patients stratified by tertiles of SHR are summarized in Table 1. Patients with higher levels of SHR tended to be male, had higher rates of PAD, higher levels of systolic blood pressure, triglycerides, and AFBG, and lower levels of HbA1c. In terms of angiographic characteristics, the proportion of proximal left anterior descending artery (LAD) disease differed across tertiles of SHR. Compared with those with the middle and highest SHR tertiles, patients with the lowest SHR tertile were more likely to be prescribed β-blockers and insulin at discharge.

Over a median follow-up of 927 days (IQR: 744–1109 days), 194 patients developed at least one primary endpoint event. The follow-up incidence of MACCE increased in parallel with SHR tertiles (15.6%, 21.9%, and 36.1%, respectively; P for trend < 0.001). The baseline characteristics of the study population according to MACCE are shown in Table 2. Patients with MACCE had lower diastolic blood pressure and higher heart rate, and

Table 1 Baseline characteristics of the study population according to SHR tertiles

Variable	Lowest tertile *n* = 263	Middle tertile *n* = 265	Highest tertile *n* = 263	*P* value
SHR	0.6402 ± 0.0857	0.8035 ± 0.0349	1.0021 ± 0.1378	< 0.001
Age (years)	61 ± 10	62 ± 10	61 ± 10	0.244
Male sex, n (%)	184 (70.0)	186 (70.2)	206 (78.3)	0.049
BMI (kg/m^2)	26.0 ± 3.4	25.9 ± 3.1	26.1 ± 3.0	0.650
SBP at admission (mmHg)	129 ± 18	132 ± 16	134 ± 17	< 0.001
DBP at admission (mmHg)	75 ± 11	75 ± 10	76 ± 11	0.481
Heart rate at admission (bpm)	70 ± 9	69 ± 8	70 ± 10	0.739
Current smoking, n (%)	116 (44.1)	96 (36.2)	108 (41.1)	0.177
Family history of CAD, n (%)	75 (28.5)	82 (30.9)	78 (29.7)	0.830
Hypertension, n (%)	178 (67.7)	179 (67.5)	183 (69.6)	0.854
Dyslipidaemia, n (%)	213 (81.0)	222 (83.8)	226 (85.8)	0.309
Previous MI, n (%)	48 (18.3)	64 (24.2)	58 (22.1)	0.247
Past PCI, n (%)	53 (20.2)	74 (27.9)	64 (24.3)	0.113
Previous ischemic stroke or TIA, n (%)	13 (4.9)	15 (5.7)	24 (9.1)	0.117
PAD, n (%)	34 (12.9)	31 (11.7)	52 (19.8)	0.019
Heart failure, n (%)	23 (8.7)	19 (7.2)	24 (9.1)	0.690
LVEF (%)	65 (60–68)	64 (60–68)	64 (59–67)	0.502
Clinical presentation				
UA, n (%)	196 (74.5)	219 (82.6)	209 (79.5)	0.071
NSTEMI, n (%)	40 (15.2)	31 (11.7)	25 (9.5)	0.130
STEMI, n (%)	27 (10.3)	15 (5.7)	29 (11.0)	0.065
GRACE risk score	96 (77–131)	92 (74–118)	91 (73–119)	0.168
Laboratory measurements (fasting state)				
SCr (umol/L)	70.9 ± 16.3	71.5 ± 16.1	72.5 ± 14.3	0.488
TC (mmol/L)	4.02 ± 1.00	4.18 ± 1.07	4.18 ± 0.98	0.138
LDL-C (mmol/L)	2.38 ± 0.84	2.46 ± 0.84	2.43 ± 0.76	0.540
HDL-C (mmol/L)	1.01 ± 0.22	1.04 ± 0.20	1.01 ± 0.24	0.134
Triglycerides (mmol/L)	1.46 (1.04–1.89)	1.43 (1.09-2.00)	1.69 (1.09–2.45)	0.001
HsCRP (mg/L)	1.61 (0.71–4.23)	1.28 (0.69–3.25)	1.45 (0.67–3.71)	0.189
FPG (mmol/L)	7.12 (6.18–8.20)	7.90 (6.92–9.06)	8.13 (7.24–10.21)	< 0.001
HbA1c (%)	7.7 (7.0-8.7)	7.0 (6.6–7.9)	7.0 (6.4–7.8)	< 0.001
Angiographic results				
Left-main/multi-vessel disease, n (%)	236 (89.7)	245 (92.5)	230 (87.5)	0.162
Proximal LAD disease, n (%)	116 (44.1)	155 (58.5)	125 (47.5)	0.003
SYNTAX score	23 ± 11	23 ± 11	22 ± 10	0.626
Use of hypoglycemic agents before admission, n (%)	194 (73.8)	190 (71.7)	189 (71.9)	0.840
Use of medications at discharge				
Aspirin, n (%)	262 (99.6)	262 (98.9)	259 (98.5)	0.462
P2Y12 inhibitors, n (%)	263 (100)	265 (100)	263 (100)	-
Statins, n (%)	263 (100)	265 (100)	263 (100)	-
β-blockers, n (%)	207 (78.7)	186 (70.2)	178 (67.7)	0.013
ACEI/ARBs, n (%)	131 (49.8)	121 (45.7)	145 (55.1)	0.093
Hypoglycemic agents, n (%)	199 (75.7)	188 (70.9)	184 (70.0)	0.296
Insulin, n (%)	118 (44.9)	74 (27.9)	76 (28.9)	< 0.001
Oral hypoglycemic agents, n (%)	146 (55.5)	145 (54.7)	135 (51.3)	0.593

Abbreviations: ACEI/ARBs, angiotensin converting enzyme inhibitors/angiotensin receptor blockers; BMI, body mass index; CAD, coronary artery disease; DBP, diastolic blood pressure; FBG, fasting blood glucose; HbA1c, glycated hemoglobin A1c; HDL-C, high-density lipoprotein-cholesterol; HsCRP, high-sensitivity C-reactive protein; LAD, left anterior descending artery; LDL-C, low-density lipoprotein-cholesterol; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSTEMI, non ST-segment elevation myocardial infarction; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; SCr, serum creatinine; SHR, stress hyperglycemia ratio; STEMI, ST-segment elevation myocardial infarction; TC, total cholesterol; TIA, transient ischemic attack; UA, unstable angina

Table 2 Baseline characteristics of the study population grouped by MACCE

Variable	No such events *n* = 597	MACCE *n* = 194	*P* value
SHR	0.7948 ± 0.1681	0.8783 ± 0.1853	< 0.001
T1, n (%)	222 (37.2)	41 (21.1)	
T2, n (%)	207 (34.7)	58 (29.9)	
T3, n (%)	168 (28.1)	95 (49.0)	
Age (years)	61 ± 10	62 ± 10	0.454
Male sex, n (%)	436 (73.0)	140 (72.2)	0.814
BMI (kg/m^2)	26.1 ± 3.2	25.6 ± 3.1	0.067
SBP at admission (mmHg)	131 ± 17	133 ± 16	0.328
DBP at admission (mmHg)	77 ± 10	72 ± 10	< 0.001
Heart rate at admission (bpm)	69 ± 9	72 ± 10	< 0.001
Current smoking, n (%)	243 (40.7)	77 (39.7)	0.803
Family history of CAD, n (%)	172 (28.8)	63 (32.5)	0.332
Hypertension, n (%)	404 (67.7)	136 (70.1)	0.527
Dyslipidaemia, n (%)	493 (82.6)	168 (86.6)	0.189
Previous MI, n (%)	114 (19.1)	56 (28.9)	0.004
Past PCI, n (%)	123 (20.6)	68 (35.1)	< 0.001
Previous ischemic stroke or TIA, n (%)	40 (6.7)	12 (6.2)	0.802
PAD, n (%)	66 (11.1)	51 (26.3)	< 0.001
Heart failure, n (%)	39 (6.5)	27 (13.9)	0.001
LVEF (%)	65 (60–68)	62 (57–67)	0.001
Clinical presentation			
UA, n (%)	467 (78.2)	157 (80.9)	0.423
NSTEMI, n (%)	78 (13.1)	18 (9.3)	0.161
STEMI, n (%)	52 (8.7)	19 (9.8)	0.646
GRACE risk score	92 (77–118)	92 (73–132)	0.618
Laboratory measurements (fasting state)			
SCr (umol/L)	70.7 ± 14.5	74.4 ± 18.2	0.010
TC (mmol/L)	4.09 ± 1.03	4.24 ± 0.99	0.072
LDL-C (mmol/L)	2.41 ± 0.83	2.47 ± 0.75	0.314
HDL-C (mmol/L)	1.04 ± 0.22	0.97 ± 0.20	< 0.001
Triglycerides (mmol/L)	1.46 (1.04-2.00)	1.66 (1.12–2.36)	0.003
HsCRP (mg/L)	1.28 (0.65–3.16)	2.23 (0.90–5.24)	< 0.001
FBG (mmol/L)	6.87 (6.12–8.02)	7.89 (6.70–9.24)	< 0.001
HbA1c (%)	7.2 (6.6–8.1)	7.4 (6.8–8.2)	0.115
Angiographic results			
Left-main/multi-vessel disease, n (%)	525 (87.9)	186 (95.9)	< 0.001
Proximal LAD disease, n (%)	293 (49.1)	103 (53.1)	0.331
SYNTAX score	22 ± 11	25 ± 10	< 0.001
Use of hypoglycemic agents before admission, n (%)	425 (71.2)	148 (76.3)	0.167
Use of medications at discharge			
Aspirin, n (%)	597 (100)	186 (95.9)	< 0.001
P2Y12 inhibitors, n (%)	597 (100)	194 (100)	-
Statins, n (%)	597 (100)	194 (100)	-
β-blockers, n (%)	440 (73.7)	131 (67.5)	0.095
ACEI/ARBs, n (%)	293 (49.1)	104 (53.6)	0.273
Hypoglycemic agents, n (%)	415 (69.5)	156 (80.4)	0.003
Insulin, n (%)	191 (32.0)	77 (39.7)	0.049
Oral hypoglycemic agents, n (%)	323 (54.1)	103 (53.1)	0.806

MACCE indicates major adverse cardiovascular and cerebrovascular events. Other abbreviations as in Table 1

higher rates of previous MI, past PCI, PAD, and heart failure. Patients with MACCE had higher levels of SCr, triglycerides, hsCRP, AFBG, and SYNTAX score, and lower levels of HDL-C. With the exception of insulin, there was no difference in use of medications at discharge between patients with and without MACCE.

The Kaplan-Meier analysis showed that the cumulative incidence of MACCE increased with higher SHR tertiles (log-rank test, $P<0.001$) (Fig. 1). The Cox proportional hazards regression analyses used to assess the association of SHR as a categorical variable and as a continuous variable with MACCE are presented in Table 3 and S1, respectively. When SHR was analyzed as a categorical variable, the univariate analysis showed that compared with those with the lowest SHR tertile, patients with the highest SHR tertile had a significantly higher risk of MACCE (HR: 2.607, 95% CI: 1.808–3.761; $P<0.001$); the multivariate analysis showed that after adjusting for other confounding factors, HRs for MACCE were 1.525 (95% CI: 1.009–2.305; $P=0.045$) and 2.525 (95% CI: 1.729–3.687; $P<0.001$) for the middle and highest tertiles of SHR, respectively, with the lowest tertile as the reference. When considering as a continuous variable, SHR had an HR of 7.388 (95% CI: 3.769–14.484; $P<0.001$) for MACCE in the univariate analysis and had a covariable-adjusted HR of 5.370 (95% CI: 2.658–10.850; $P<0.001$) for MACCE in the multivariate analysis. Additionally, compared with those with SHR < 0.8150, patients with SHR ≥ 0.8150 were at higher risk of MACCE (adjusted HR, 2.252; 95% CI: 1.660–3.055; $P<0.001$). Notably, the addition of SHR to the baseline reference prediction model improved model predictive performance markedly (C-statistic: increased from 0.704 to 0.721; cNRI: 0.176 [95% CI: 0.063–0.282], $P=0.002$; IDI: 0.030 [95% CI: 0.009–0.063], $P=0.002$). SHR in our study includes AFBG and HbA1c in its formula. We compared the predictive ability of SHR to AFBG and HbA1c for MACCE. The C-statistics of SHR, AFBG and HbA1c were 0.657 (0.613–0.701), 0.640 (0.594–0.686), and 0.538 (0.491–0.584), respectively. According to pair-wise comparison of the C-statistics, SHR performed best.

The prognostic value of SHR as a continuous variable for MACCE was further investigated in different subgroups of the study population. Increased SHR level (per 1-unit) was consistently and significantly associated with MACCE in different subgroups, including age ≥ 60 versus < 60 years, male versus female, BMI ≥ 25 versus < 25 kg/m^2, with versus without current smoking, with versus without hypertension, UA versus MI, hsCRP ≥ 2 versus < 2 mg/L, and SYNTAX score ≥ 22 versus < 22 (Fig. 2).

Discussion

The main findings of the present study were as follows: (1) the cumulative incidence of MACCE increased gradually with rising SHR tertiles; (2) elevated SHR was independently and significantly associated with increased risk of MACCE, suggesting that SHR was a valuable indicator of early risk stratification in patients with T2DM and ACS. Compared with those with SHR < 0.8150, patients with SHR ≥ 0.8150 had a higher risk of developing MACCE and should receive intensive medical therapy at follow-up to reduce the risk of MACCE; (3) the addition of SHR to the baseline reference prediction model significantly improved the prediction performance; (4) compared with AFBG and HbA1c, SHR had better predictive ability for MACCE, which was consistent with the results of previous studies. To the best of our knowledge, this is the first study to investigate the prognostic significance of SHR in patients with T2DM and ACS.

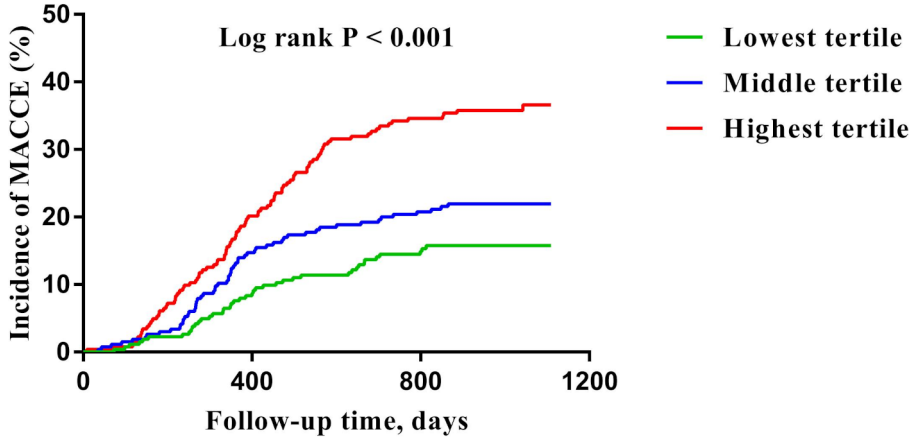

Fig. 1 Kaplan-Meier curve for the cumulative incidence of MACCE over time stratified by tertiles of SHR. MACCE was defined as a composite of all-cause mortality, non-fatal stroke, non-fatal myocardial infarction, and unplanned repeat coronary revascularization

Table 3 Univariate and multivarite Cox proportional hazards analyses for MACCE according to SHR tertiles

	Univariate analysis		Multivariate analysis	
Variables	**HR (95% CI)**	***P* value**	**HR (95% CI)**	***P* value**
SHR				
T1	ref		ref	ref
T2	1.466 (0.983–2.187)	0.061	1.525 (1.009–2.305)	0.045
T3	2.607 (1.808–3.761)	< 0.001	2.525 (1.729–3.687)	< 0.001
BMI	0.955 (0.911–1.001)	0.057	0.941 (0.893–0.991)	0.022
DBP at admission	0.965 (0.952–0.979)	< 0.001	0.978 (0.964–0.992)	0.003
Heart rate at admission	1.032 (1.017–1.046)	< 0.001	1.017 (1.001–1.034)	0.032
Previous MI	1.525 (1.118–2.081)	0.008	0.970 (0.670–1.403)	0.870
Past PCI	1.763 (1.312–2.367)	< 0.001	1.627 (1.142–2.319)	0.007
PAD	2.393 (1.737–3.296)	< 0.001	1.524 (1.054–2.203)	0.025
Heart failure	1.945 (1.295–2.921)	0.001	0.903 (0.554–1.471)	0.682
SYNTAX score	1.026 (1.013–1.039)	< 0.001	1.012 (0.998–1.027)	0.099
SCr	1.013 (1.005–1.021)	0.002	1.008 (0.999–1.018)	0.081
HDL-C	0.223 (0.109–0.457)	< 0.001	0.398 (0.185–0.855)	0.018
Triglycerides	1.107 (1.046–1.172)	< 0.001	1.066 (1.000-1.136)	0.049
HsCRP	1.039 (1.017–1.061)	< 0.001	1.020 (0.994–1.047)	0.128
Discharged with β-blockers	0.755 (0.559–1.020)	0.067	0.797 (0.586–1.083)	0.147
Discharged with insulin	1.345 (1.009–1.794)	0.043	1.184 (0.864–1.622)	0.293

HR indicates hazard ratio; 95% CI, 95% confidence interval. Other abbreviations as in Tables 1 and 2

Stress hyperglycemia is a special type of acute hyperglycemia. Acute hyperglycemia on admission is prevalent in patients with ACS 19, and it is related, at least in part, to the overactivated neurohormonal systems following an ACS event 20. The excessive release of stress hormones such as cortisol and catecholamines, which can significantly raise blood glucose, has been demonstrated to be associated with poor prognosis in ACS patients 20. During MI, cortisol can have various deleterious effects, for example, increasing sensitivity to catecholamines and stimulating mineralocorticoids receptors present in the myocardium 21. The study of Swieszkowski and colleagues including 149 patients with MI showed that there was a positive correlation between serum cortisol and blood glucose on admission in both patients with and without diabetes, and that both serum cortisol and blood glucose on admission were associated with mortality in univariate analysis, but only a significant association between serum cortisol and mortality was found in multivariate analysis 22. Both in patients with and without diabetes, acute hyperglycemia has been shown to induce oxidative stress and inflammation 23, 24, 25, 26, and thus lead to endothelial dysfunction 27, 28 and increased procoagulant and prothrombotic effects 29, 30. Also, acute hyperglycemia abolishes ischemic preconditioning through multiple mechanisms such as increase in nitrative stress, activation of the mTOR pathway and inhibition of Akt phosphorylation 31, 32, 33. Of note, high random blood glucose on admission was shown to be independently associated with in-hospital mortality in non-diabetic patients with MI but not in diabetic MI patients 34, 35. O'Sullivan et al. reported that patients with a first MI and fasting hyperglycemia on admission but no prior history of glucose intolerance had significantly more in-hospital complications than those with normal fasting blood glucose, and there was no significant difference in in-hospital prognosis between patients previously known to have diabetes and those with fasting hyperglycemia 36.

With the advancement of knowledge, stress hyperglycemia has been defined as an acute upward fluctuation in blood glucose adjusted for background glycemia 7, and it is easily identified in ACS patients without diabetes because a high admission blood glucose (≥ 7.8 mmol/L) represents a marked blood glucose elevation in non-diabetic patents and is positively associated with admission serum cortisol (as a surrogate marker for the severity of stress) in patients who had stress hyperglycemia and normal glucose post-discharge, but not in stress hyperglycemia patients who had diabetes/impaired glucose tolerance on post-discharge testing 6, 37. Unfortunately, it is very challenging to use high admission blood glucose to identify stress hyperglycemia in diabetic patients. Thus, the concept of acute hyperglycemia adjusted for background glycemia, that is, SHR, has been proposed in recent years. The current view is that SHR can reflect "true stress hyperglycemia" during hospitalization irrespective of diabetes status 8. SHR in our study includes AFBG and HbA1c in its formula. AFBG measured immediately after acute illness can more accurately reflect the impact of disease-related stress mechanisms on blood glucose on admission on the basis of minimizing the influence of food and drink. HbA1c is generally considered to effectively reflect the average blood glucose levels in the past 8 to 12 weeks. Therefore, it is reasonable to apply HbA1c to represent the background glycemia during stressful events or severe disease states.

The prognostic value of SHR in patients with coronary artery disease has been demonstrated in a considerable

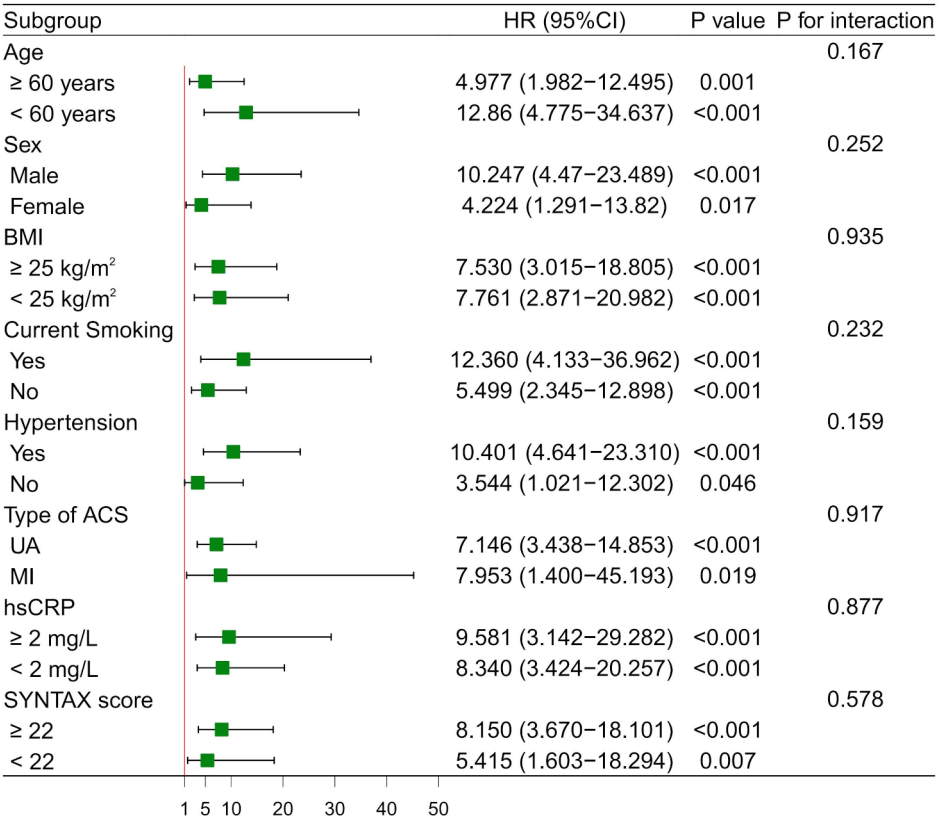

Fig. 2 Subgroup analyses of SHR as a continuous variable for MACCE. HR was evaluated by per 1-unit increase in the SHR. BMI indicates body mass index; ACS, acute coronary syndrome; Hs-CRP, high-sensitivity C-reactive protein; HR, hazard ratio; 95% CI, 95% confidence interval

number of studies 38, 39, 40, 41, 42, 43, 44. The studies of Li M et al. and Lin Z et al. both showed that there was a significant linear relationship between SHR and poor prognosis 38, 39. Li Y and colleagues reported a significant association between SHR and in-hospital mortality in patients with chronic kidney disease and ACS 40. A meta-analysis of 26 cohort studies involving 8,7974 acute MI patients showed that patients with the upper quantile of SHR had a significantly greater hazard of the composite of all-cause mortality, recurrent myocardial infarction, ischemia-driven target vessel revascularization, cardiogenic shock and stroke, and long-term and in-hospital all-cause mortality compared to those with lower SHR irrespective of baseline diabetic status 41. Of note, microvascular obstruction is not uncommon in patients with ACS undergoing PCI and has been shown to be associated with poor cardiovascular outcomes 45, 46. As we known, diabetes itself is closely related to microvascular dysfunction. Intriguingly, Bo K and colleagues found that SHR was independently associated with the presence and extent of microvascular obstruction in diabetic patients with acute MI undergoing PCI 47. The study of Zhang Y et al. which included 3,812 three-vessel disease patients with ACS more than one half of whom underwent PCI showed that the predictive value of SHR for cardiovascular death was found exclusively in patients with diabetes, but not in those without diabetes 42. However, the study of Zeng G et al. showed that elevated SHR was independently associated with increased risk of the composite of all-cause death, non-fatal MI, and unplanned revascularization in ACS patients irrespective of diabetic status 43. It should be noted that the median SYNTAX score of the patients included in the study of Zeng G et al. was less than 22, indicating non-complex coronary lesions, which was different from the study of Zhang Y et al. and our study, both of which included ACS patients with complex coronary lesions (the mean SYNTAX score of our study population was 23). Consistent with the study by Zhang Y et al., we did not find that SHR was predictive of MACCE in non-diabetic patients with ACS when analyzing the raw data, which is not reported in this manuscript. Our study showed that higher SHR was associated

with a significantly higher risk of MACCE in T2DM and ACS patients. Similarly, Wang L et al. reported that high SHR was independently associated with increased mortality risk in T2DM and multivessel disease patients (ACS patients accounted for more than 70%), and adding SHR to the original models significantly improved the C-statistic and IDI 44. Therefore, we speculate that T2DM may have discrepant effects on the prognostic value of SHR in ACS patients with non-complex versus complex coronary lesions, which needs to be confirmed by well-designed studies.

There are several limitations to the study that should be noted. First, given the retrospective observational nature, the current analysis cannot confirm a causal relationship between SHR and the risk of MACCE. Second, due to the observational nature of this study, the influence of unknown or unmeasured confounding factors on the results of the multivariate COX proportional hazards regression analyses cannot be ruled out. Third, T2DM has been shown to be closely associated with heart failure development; however, the primary endpoint of our study did not include heart failure-related endpoint events. Fourth, our study included only the Chinese population, so the findings and conclusions need to be extrapolated with caution to other ethnic groups. Fifth, the use of hypoglycemic agents before admission may affect the baseline levels of AFPG and HbA1c; however, there were no significant differences across tertiles of SHR in the use of hypoglycemic agents prior to admission.

Conclusions

SHR, easily measurable in clinical practice, was independently and significantly associated with an increased risk of MACCE in T2DM and ACS patients who underwent PCI, suggesting that SHR was a valuable indicator in early risk stratification of such patients. Our study showed that an SHR value of 0.8150 was the critical threshold for poor prognosis. Optimizing medical management based on SHR may reduce the risk of subsequent adverse cardiovascular events, which needs to be confirmed by well-designed studies.

Abbreviations

ACS	Acute coronary syndromes
AFBG	Admission fasting blood glucose
BMI	Body mass index
CABG	Coronary artery bypass grafting
CI	Confidence interval
cNRI	Continuous net reclassification improvement
CrCl	Creatinine clearance
eAG	Estimated average glucose
HbA1c	Glycated hemoglobin A1c
HDL-C	High-density lipoprotein cholesterol
HR	Hazard ratio
Hs-CRP	High-sensitivity C-reactive protein
IDI	Integrated discrimination improvement
IQR	Interquartile range
LAD	Left anterior descending artery
LDL-C	Low-density lipoprotein cholesterol
LVEF	Left ventricular ejection fraction
MACCE	Major adverse cardiovascular and cerebrovascular events
MI	Myocardial infarction
NSTEMI	Non-ST segment elevation myocardial infarction
PAD	Peripheral artery disease
PCI	Percutaneous coronary intervention
SCr	Serum creatinine
SD	Standard deviation
SHR	Stress hyperglycemia ratio
STEMI	ST-segment elevation myocardial infarction
T2DM	Type 2 diabetes mellitus
TC	Total cholesterol
UA	Unstable angina

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12959-025-00729-5.

Supplementary Material 1

Author contributions

Xiaoteng Ma and Huijun Chu analyzed the data and drafted the manuscript. Xiaoteng Ma, Huijun Chu, Yan Sun, Yujing Cheng, and Dai Zhang prospectively collected the demographic data, laboratory data, angiographic and interventional data of the enrolled patients. Lixia Yang, Zhijian Wang, and Xiaoli Liu proposed amendments to the first draft. Xiaoteng Ma and Yujie Zhou designed the study and revised the manuscript. All authors contributed to the acquisition of data and final approval of the version to be published.

Funding

This work was supported by the National Key Research and Development Program of China (2022YFC3602500), Youth Program of National Natural Science Foundation of China (82200405), General Program of National Natural Science Foundation of China (82370449), General Program of Beijing Municipal Natural Science Foundation (7232039), Capital's Funds for Health Improvement and Research (2022-2-1052), and Beijing Hospitals Authority Incubating Program (PX2021027).

Data availability

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

Declarations

Ethics approval and consent to participate

This study was approved by the institutional review board of Beijing Anzhen Hospital, Capital Medical University. Given the retrospective nature of this study, the requirement for informed consent was waived.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 1 November 2024 / Accepted: 29 April 2025
Published online: 12 May 2025

References

1. Bhatt DL, Lopes RD, Harrington RA. Diagnosis and treatment of acute coronary syndromes: A review. Jama. 2022;15(7):662–75. https://doi.org/10.1001/jama.2022.0358.
2. Aggarwal B, Shah GK, Randhawa M, Ellis SG, Lincoff AM, Menon V. Utility of glycated hemoglobin for assessment of glucose metabolism in patients with

ST-Segment elevation myocardial infarction. Am J Cardiol. 2016;1(5):749–53. https://doi.org/10.1016/j.amjcard.2015.11.060.
3. Avogaro A, Bonora E, Consoli A, Del Prato S, Genovese S, Giorgino F. Glucose-lowering therapy and cardiovascular outcomes in patients with type 2 diabetes mellitus and acute coronary syndrome. Diab Vasc Dis Res. 2019;16(5):399–414. https://doi.org/10.1177/1479164119845612.
4. Kosiborod M, Rathore SS, Inzucchi SE, et al. Admission glucose and mortality in elderly patients hospitalized with acute myocardial infarction: implications for patients with and without recognized diabetes. Circulation. 2005;14(23):3078–86. https://doi.org/10.1161/circulationaha.104.517839.
5. Dziewierz A, Giszterowicz D, Siudak Z, Rakowski T, Dubiel JS, Dudek D. Admission glucose level and in-hospital outcomes in diabetic and non-diabetic patients with acute myocardial infarction. Clin Res Cardiol. 2010;99(11):715–21. https://doi.org/10.1007/s00392-010-0175-1.
6. Koraćević G, Zdravković M, What is stress hyperglycemia? A suggestion for an, improvement of its definition. Acta Endocrinol (Buchar). 2021;17(4):548–51. https://doi.org/10.4183/aeb.2021.548.
7. Dungan KM, Braithwaite SS, Preiser JC. Stress hyperglycaemia. Lancet. 2009;373(9677):1798–807. https://doi.org/10.1016/s0140-6736(09)60553-5.
8. Roberts GW, Quinn SJ, Valentine N, et al. Relative hyperglycemia, a marker of critical illness: introducing the stress hyperglycemia ratio. J Clin Endocrinol Metab. 2015;100(12):4490–7. https://doi.org/10.1210/jc.2015-2660.
9. Marenzi G, Cosentino N, Milazzo V, et al. Prognostic value of the acute-to-Chronic glycemic ratio at admission in acute myocardial infarction: A prospective study. Diabetes Care. 2018;41(4):847–53. https://doi.org/10.2337/dc17-1732.
10. Lee TF, Burt MG, Heilbronn LK, et al. Relative hyperglycemia is associated with complications following an acute myocardial infarction: a post-hoc analysis of HI-5 data. Cardiovasc Diabetol. 2017;12(1):157. https://doi.org/10.1186/s12933-017-0642-3.
11. Lee TF, Drake SM, Roberts GW, et al. Relative hyperglycemia is an independent determinant of In-Hospital mortality in patients with critical illness. Crit Care Med. 2020;48(2):e115–22. https://doi.org/10.1097/ccm.0000000000004133.
12. Roberts G, Sires J, Chen A, et al. A comparison of the stress hyperglycemia ratio, glycemic Gap, and glucose to assess the impact of stress-induced hyperglycemia on ischemic stroke outcome. J Diabetes. 2021;13(12):1034–42. https://doi.org/10.1111/1753-0407.13223.
13. Xia Z, Gu T, Zhao Z, et al. The stress hyperglycemia ratio, a novel index of relative hyperglycemia, predicts short-term mortality in critically ill patients after esophagectomy. J Gastrointest Oncol. 2022;13(1):56–66. https://doi.org/10.21037/jgo-22-11.
14. Deng Y, Wu S, Liu J, et al. The stress hyperglycemia ratio is associated with the development of cerebral edema and poor functional outcome in patients with acute cerebral infarction. Front Aging Neurosci. 2022;14:936862. https://doi.org/10.3389/fnagi.2022.936862.
15. Luo J, Xu S, Li H, et al. Prognostic impact of stress hyperglycemia ratio in acute myocardial infarction patients with and without diabetes mellitus. Nutr Metab Cardiovasc Dis. 2022;32(10):2356–66. https://doi.org/10.1016/j.numecd.2022.07.004.
16. Armillotta M, Bergamaschi L, Paolisso P, Pizzi C. Oct. Editorial commentary: beyond coronary anatomy in acute myocardial infarction: could stress hyperglycemia ratio be a new prognostic index and therapeutic target? Trends Cardiovas Med. 2024;34(7):466–7. https://doi.org/10.1016/j.tcm.2023.12.006
17. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976;16(1):31–41. https://doi.org/10.1159/000180580.
18. Nathan DM, Kuenen J, Borg R, Zheng H, Schoenfeld D, Heine RJ. Translating the A1C assay into estimated average glucose values. Diabetes Care. 2008;31(8):1473–8. https://doi.org/10.2337/dc08-0545.
19. Deedwania P, Kosiborod M, Barrett E, et al. Hyperglycemia and acute coronary syndrome: a scientific statement from the American heart association diabetes committee of the Council on nutrition, physical activity, and metabolism. Circulation. 2008;25(12):1610–9. https://doi.org/10.1161/circulationaha.107.188629.
20. de la Perez RA, Swieszkowski SP, Cintora FM, et al. Neuroendocrine system regulatory mechanisms: acute coronary syndrome and stress hyperglycaemia. Eur Cardiol. 2018;13(1):29–34. https://doi.org/10.15420/ecr.2017:19:3.
21. McAlpine HM, Morton JJ, Leckie B, Rumley A, Gillen G, Dargie HJ. Neuroendocrine activation after acute myocardial infarction. Br Heart J. 1988;60(2):117–24. https://doi.org/10.1136/hrt.60.2.117.
22. Swieszkowski SP, Costa D, Aladio JM, et al. Neurohumoral response and stress hyperglycemia in myocardial infarction. J Diabetes Complications. 2022;36(12):108339. https://doi.org/10.1016/j.jdiacomp.2022.108339.
23. Marfella R, Quagliaro L, Nappo F, Ceriello A, Giugliano D. Acute hyperglycemia induces an oxidative stress in healthy subjects. J Clin Invest. 2001;108(4):635–6. https://doi.org/10.1172/jci13727.
24. Esposito K, Nappo F, Marfella R, et al. Inflammatory cytokine concentrations are acutely increased by hyperglycemia in humans: role of oxidative stress. Circulation. 2002;15(16):2067–72. https://doi.org/10.1161/01.cir.0000034509.14906.ae.
25. 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;12(14):1681–7. https://doi.org/10.1001/jama.295.14.1681.
26. Chang CM, Hsieh CJ, Huang JC, Huang IC. Acute and chronic fluctuations in blood glucose levels can increase oxidative stress in type 2 diabetes mellitus. Acta Diabetol. 2012;49(Suppl 1):S171–7. https://doi.org/10.1007/s00592-012-0398-x.
27. Kawano H, Motoyama T, Hirashima O, et al. Hyperglycemia rapidly suppresses flow-mediated endothelium-dependent vasodilation of brachial artery. J Am Coll Cardiol. 1999;34(1):146–54. https://doi.org/10.1016/s0735-1097(99)00168-0.
28. Beckman JA, Goldfine AB, Gordon MB, Creager MA. Ascorbate restores endothelium-dependent vasodilation impaired by acute hyperglycemia in humans. Circulation. 2001;27(12):1618–23. https://doi.org/10.1161/01.cir.103.12.1618.
29. 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;23(3):304–10. https://doi.org/10.1016/j.jacc.2006.08.053.
30. Undas A, Wiek I, Stêpien E, Zmudka K, Tracz W. Hyperglycemia is associated with enhanced thrombin formation, platelet activation, and fibrin clot resistance to Lysis in patients with acute coronary syndrome. Diabetes Care. 2008;31(8):1590–5. https://doi.org/10.2337/dc08-0282.
31. Kersten JR, Schmeling TJ, Orth KG, Pagel PS, Warltier DC. Acute hyperglycemia abolishes ischemic preconditioning in vivo. Am J Physiol. 1998;275(2):H721–5. https://doi.org/10.1152/ajpheart.1998.275.2.H721.
32. Yang Z, Tian Y, Liu Y, Hennessy S, Kron IL, French BA. Acute hyperglycemia abolishes ischemic preconditioning by inhibiting Akt phosphorylation: normalizing blood glucose before ischemia restores ischemic preconditioning. Oxid Med Cell Longev. 2013;2013:329183. https://doi.org/10.1155/2013/329183.
33. Baranyai T, Nagy CT, Koncsos G, et al. Acute hyperglycemia abolishes cardioprotection by remote ischemic perconditioning. Cardiovasc Diabetol. 2015;18:14:151. https://doi.org/10.1186/s12933-015-0313-1.
34. Sewdarsen M, Vythilingum S, Jialal I, Becker PJ. Prognostic importance of admission plasma glucose in diabetic and non-diabetic patients with acute myocardial infarction. Q J Med. 1989;71(265):461–6.
35. Kim EJ, Jeong MH, Kim JH, et al. Clinical impact of admission hyperglycemia on in-hospital mortality in acute myocardial infarction patients. Int J Cardiol. 2017;1:236:9–15. https://doi.org/10.1016/j.ijcard.2017.01.095.
36. O'Sullivan JJ, Conroy RM, Robinson K, Hickey N, Mulcahy R. In-hospital prognosis of patients with fasting hyperglycemia after first myocardial infarction. Diabetes Care. 1991;14(8):758–60. https://doi.org/10.2337/diacare.14.8.758.
37. Cheung NW, Wong KYC, Kovoor P, McLean M. Stress hyperglycemia: A prospective study examining the relationship between glucose, cortisol and diabetes in myocardial infarction. J Diabetes Complications. 2019;33(4):329–34. https://doi.org/10.1016/j.jdiacomp.2018.12.015.
38. Li M, Cui X, Zhang Y, et al. The relative and combined ability of triglyceride-glucose index and stress hyperglycemia ratio to predict major adverse cardio-cerebral events in patients with multivessel coronary artery disease. Diabetol Metab Syndr. 2024;28(1):234. https://doi.org/10.1186/s13098-024-01471-0.
39. Lin Z, Song Y, Yuan S, He J, Dou K. Prognostic value of the stress-hyperglycaemia ratio in patients with moderate-to-severe coronary artery calcification: insights from a large cohort study. Diabetes Obes Metab. 2024;26(11):4933–44. https://doi.org/10.1111/dom.15894.
40. Li Y, Shen N, Xie E, et al. Predicting the impact of stress-induced hyperglycemia on in-hospital mortality in patients with chronic kidney disease and acute coronary syndrome: A retrospective study. J Diabetes Complications. 2024;38(12):108895. https://doi.org/10.1016/j.jdiacomp.2024.108895.

41. Karakasis P, Stalikas N, Patoulias D, et al. Prognostic value of stress hyperglycemia ratio in patients with acute myocardial infarction: A systematic review with bayesian and frequentist meta-analysis. Trends Cardiovasc Med. 2024;34(7):453–65. https://doi.org/10.1016/j.tcm.2023.11.006.
42. Zhang Y, Guo L, Zhu H, et al. Effects of the stress hyperglycemia ratio on long-term mortality in patients with triple-vessel disease and acute coronary syndrome. Cardiovasc Diabetol. 2024;25(1):143. https://doi.org/10.1186/s12933-024-02220-3.
43. Zeng G, Song Y, Zhang Z, et al. Stress hyperglycemia ratio and long-term prognosis in patients with acute coronary syndrome: A multicenter, nationwide study. J Diabetes. 2023;15(7):557–68. https://doi.org/10.1111/1753-0407.13400.
44. Wang L, Wang C, Lang JC, et al. The relative and combined ability of stress hyperglycemia ratio and N-terminal pro-B-type natriuretic peptide to predict all-cause mortality in diabetic patients with multivessel coronary artery disease. Cardiovasc Diabetol. 2024;11(1):93. https://doi.org/10.1186/s12933-024-02186-2.
45. Ozaki Y, Tanaka A, Tanimoto T, et al. Thin-cap fibroatheroma as high-risk plaque for microvascular obstruction in patients with acute coronary syndrome. Circulation Cardiovasc Imaging. 2011;4(6):620–7. https://doi.org/10.1161/circimaging.111.965616.
46. Benenati S, Montorfano M, Pica S, et al. Coronary physiology thresholds associated with microvascular obstruction in myocardial infarction. Heart (British Cardiac Society). 2024;29(4):271–80. https://doi.org/10.1136/heartjnl-2023-323169.
47. Bo K, Li W, Zhang H, et al. Association of stress hyperglycemia ratio with left ventricular function and microvascular obstruction in patients with ST-segment elevation myocardial infarction: a 3.0 T cardiac magnetic resonance study. Cardiovasc Diabetol. 2024;27(1):179. https://doi.org/10.1186/s12933-024-02271-6.

Publisher's note