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Positive association between stress hyperglycemia ratio and pulmonary infection in patients with ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention

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Abstract

Background Previous studies have shown that the stress hyperglycemia ratio (SHR), a parameter of relative stress-induced hyperglycemia, is an excellent predictive factor for all-cause mortality and major adverse cardiovascular events (MACEs) among patients with ST-segment elevation myocardial infarction (STEMI). However, its association with pulmonary infection in patients with STEMI during hospitalization remains unclear.

Methods Patients with STEMI undergoing percutaneous coronary intervention (PCI) were consecutively enrolled from 2010 to 2020. The primary endpoint was the occurrence of pulmonary infection during hospitalization, and the secondary endpoint was in-hospital MACEs, composed of all-cause mortality, stroke, target vessel revascularization, or recurrent myocardial infarction.

Results A total of 2,841 patients were finally included, with 323 (11.4%) developing pulmonary infection and 165 (5.8%) developing in-hospital MACEs. The patients were divided into three groups according to SHR tertiles. A higher SHR was associated with a higher rate of pulmonary infection during hospitalization (8.1%, 9.9%, and 18.0%, $P < 0.001$) and in-hospital MACEs (3.7%, 5.1%, and 8.6%, $P < 0.001$). Multivariate logistic regression analysis demonstrated that SHR was significantly associated with the risk of pulmonary infection during hospitalization (odds ratio [OR] = 1.46, 95% confidence interval [CI] 1.06–2.02, $P = 0.021$) and in-hospital MACEs (OR = 1.67, 95% CI 1.17–2.39, $P = 0.005$) after adjusting for potential confounding factors. The cubic spline models demonstrated no significant non-linear relationship between SHR and pulmonary infection ($P = 0.210$) and MACEs ($P = 0.743$). In receiver operating characteristic curve, the best cutoff value of SHR for pulmonary infection was 1.073.

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Conclusions The SHR is independently associated with the risk of pulmonary infection during hospitalization and in-hospital MACEs for patients with STEMI undergoing PCI.

Keywords ST-segment elevation myocardial infarction, Percutaneous intervention, Stress hyperglycemia ratio, Pulmonary infection, Risk factor

Introduction

Infection during hospitalization, especially pulmonary infection, is a serious complication observed among patients with ST-segment elevation myocardial infarction (STEMI), which induces a tenfold increase in the 30-day mortality rate [1–4]. Early identification and intervention improve the effectiveness of treatment and outcomes.

Stress-induced hyperglycemia (SIH), defined as temporarily increased acute blood glycemia during an emergency situation, is a common state among patients with STEMI [5]. SIH is linked to infection both in vitro and in vivo [6, 7], and results in an increased release of pro-inflammatory cytokines [8, 9]. Prior studies have used admission blood glucose (ABG) as a parameter of stress hyperglycemia [10, 11]. However, the ABG level is influenced not only by acute stress but also by a chronic glycaemic condition, which limits its ability to distinguish a true acute glycaemic rise [12]. In recent years, the stress hyperglycemia ratio (SHR), calculated from the ABG adjusted by the average glycaemic status [13], has been regarded as a better index of relative SIH compared to ABG alone [12]. The SHR has been reported as a reliable predictor for poor long- and short-term prognosis in patients with STEMI [12, 14, 15], and for stroke-associated pneumonia in patients without diabetes mellitus (DM) [16]. However, the prognostic value of the SHR for pulmonary infection during hospitalization among patients with STEMI remains unclear.

Therefore, the main aim of the present study was to investigate the association between SHR and pulmonary infection during hospitalization among patients with STEMI.

Method

Study population

Patients with STEMI at Guangdong Provincial People's Hospital were consecutively enrolled between 2010 and 2020. Patients were diagnosed with STEMI according to the international guideline as follows: (1) typical chest pain or ischemia symptoms; (2) dynamic changes in the new electrocardiogram: ST-segment elevation of more than two adjacent leads or new left bundle branch block; (3) increased levels of myocardial-injury biochemical markers [17]. We excluded patients who (1) undergoing coronary artery bypass grafting, (2) with infection

before the diagnosis of STEMI, (3) were on hemodialysis at admission, (4) died within 24 h after admission, (5) readmission to hospital, (6) did not receive the percutaneous coronary intervention (PCI) and (7) were missing crucial laboratory data (ABG or glycosylated hemoglobin [HbA1c]). This study was approved by the research ethics committee of Guangdong Provincial People's Hospital and was conducted according to the guidelines stipulated in the Declaration of Helsinki.

Data collection and definitions

Baseline blood samples were collected within 24 h of admission and tested for white blood cell count, hemoglobin, ABG, HbA1c, troponin I/T, cardiac enzymes, serum creatinine, blood lipids, and other routine blood tests. Relevant data were collected, including demographic information, medical history (hypertension, DM, myocardial infarction [MI] or coronary revascularization, admission status (heart rate, systolic and diastolic blood pressure, and Killip class), and coronary revascularization data. Killip class is used to assess the severity of acute myocardial infarction. Killip class I was defined as absence of congestive heart failure, class II as presence of rales and/or jugular venous distension, class III as presence of pulmonary oedema and class IV as cardiogenic shock. DM was defined based on the history of DM or newly diagnosed DM with HbA1c \geq 6.5%, fasting blood glucose \geq 7.0 mmol/L, or 2-h plasma glucose \geq 11.1 mmol/L in the oral glucose tolerance test [18]. The SHR was defined as the index calculated by the formula: $[(ABG \text{ (mg/dl)}) / (28.7 \times HbA1c \text{ (\%)} - 46.7)]$ [13].

Coronary angiography and/or PCI were performed at the interventional cardiologists' discretion and according to the current guidelines [19]. Medications, including aspirin, clopidogrel or ticagrelor, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and β -blockers, were prescribed by the cardiologists according to current guidelines and recommendations. Transthoracic echocardiography, electrocardiograph, and X-ray were performed, and chest computed tomography was performed if necessary.

Endpoints and definitions

The primary endpoint was pulmonary infection during hospitalization, which was determined based on typical medical imaging and clinical signs, symptoms or relevant

laboratory biomarkers (e.g., white blood cell count) by an experienced clinician [20]. For patients without sufficient evidence recorded for pulmonary infection diagnosis, the infection was determined using ICD-10-CM codes at discharge. The secondary endpoint was in-hospital major adverse cardiovascular events (MACEs), comprising all-cause mortality, stroke, target vessel revascularization, or recurrent MI.

Statistical analyses

Continuous variables are shown as the mean \pm SD or median (interquartile range) and were compared using independent *t*-test or Kruskal–Wallis test based on their distribution. Categorical variables are presented as numbers (percentages), and differences between groups were analyzed using Pearson's chi-squared test or Fisher's exact test. Variables with a statistical significance level of $P < 0.10$ on univariate logistic regression or with clinical importance, including age, sex, anemia, estimated glomerular filtration rate (eGFR), current smoker, DM, hypertension, chronic obstructive pulmonary disease, prior MI, prior PCI, prior stroke, transradial assessment, multi-vessel disease, and white blood cell count, were included and used for adjustment in the multivariate analysis. Receiver operating characteristic (ROC) curves were used to show the predictive value of the SHR. The best cutoff value and area under the ROC curve (AUC) were calculated. A cubic spline model was also performed to judge the effect of SHR on pulmonary infection. The logistic regression model was used to construct the cubic splines, and the knots were set at 0.75, 1, 1.25, 1.5, mainly based on the distribution of SHR. Statistical analysis was performed with SAS 9.4 (SAS Institute, Cary, NC). $P < 0.05$ were considered to indicate statistical significance, and all analyses were two-tailed.

Results

Baseline characteristics

From 2010 to 2020, 4,757 patients were diagnosed with STEMI. After excluding 42 patients undergoing coronary artery bypass grafting, 68 with infection before the diagnosis of STEMI, 25 on hemodialysis at admission, 49 subjects who died within 24 h after admission, 87 readmissions to hospital, 554 subjects who did not receive PCI and 1,091 without ABG or HbA1c, 2,841 patients were finally included. The study flow is shown in Fig. 1. The patients were divided into three groups according to the tertiles of SHR ($n = 947$, 946 and 948). The patients' baseline characteristics are shown in Table 1.

The mean age of the population was 62.27 ± 12.18 years, and 82.6% were male. 737 (83.6%) of the 882 DM diagnosis were based on medical history. For the remanent 145 newly diagnosis of DM, 15 (10.3%) participants were

diagnosed with DM based on oral glucose tolerance test, 109 (75.2%) based both on glycemia and HbA1c, 13 (9.0%) based on HbA1c and 8 (5.5%) on glycemia only. As shown in Table 1, the higher SHR group was more likely to be older, female, has a higher Killip classification, and has a history of DM, but less likely to be current smokers. Meanwhile, the patients with lower SHR had a higher rate of prior MI and PCI, and in terms of in-hospital medication usage, a higher rate of aspirin use.

SHR for pulmonary infection and in-hospital MACEs

During hospitalization, 323 (11.4%) patients developed the pulmonary infection and 165 (5.8%) suffered from in-hospital MACEs. The SHR was associated with pulmonary infection (8.1%, 9.9%, and 18.0%; $P < 0.001$) and in-hospital MACEs (3.7%, 5.1%, and 8.6%; $P < 0.001$), as shown in Fig. 2. In multivariate logistic regression analysis, SHR, as a continuous value, remained an independent risk factor significantly associated with pulmonary infection during hospitalization (adjusted odds ratio [OR] = 1.46, 95% confidence interval [CI] 1.06–2.02, $P = 0.021$) and in-hospital MACEs (adjusted OR = 1.67, 95% CI 1.17–2.39, $P = 0.005$) after adjusting for potential risk factors, including age, sex, anemia, eGFR, current smoker, DM and so on (Table 2). Taking SHR as a categorical value, the same results were found, in that a SHR more than 1.176 was a risk factor for a higher risk of pulmonary infection during hospitalization (adjusted OR = 1.75, 95% CI 1.25–2.46, $P = 0.001$) and in-hospital MACEs (adjusted OR = 1.68, 95% CI 1.09–2.60, $P = 0.020$) (Table 2).

Cubic spline models demonstrated no significant non-linear relationship between SHR and pulmonary infection during hospitalization ($P = 0.210$) or MACEs ($P = 0.743$) (Fig. 3).

For the interaction between DM and the SHR, significant interactions were observed both for the pulmonary infection (interaction $P = 0.012$) and the in-hospital MACEs (interaction $P = 0.009$). For further analysis, we divided these patients into 6 groups, G1: without DM, $\text{SHR} < 0.929$ (T1); G2: without DM, $0.929 \leq \text{SHR} < 1.176$ (T2); G3: without DM, $\text{SHR} \geq 1.176$ (T3); G4: with DM, T1; G5: with DM, T2 and G6: with DM, T3.

Multivariate logistic regression analysis demonstrated that DM was not significantly associated with the pulmonary infection (OR = 1.60, 95% CI 0.90–2.83, $P = 0.110$) nor the in-hospital MACEs (adjusted OR = 1.05, 95% CI 0.49–2.26, $P = 0.898$) for patients without stress hyperglycemia (T1). A SHR more than 1.176 (T3) was significantly associated with a higher risk of pulmonary infection during hospitalization (adjusted OR = 2.01, 95% CI 1.31–3.07, $P = 0.001$) but not for in-hospital MACEs

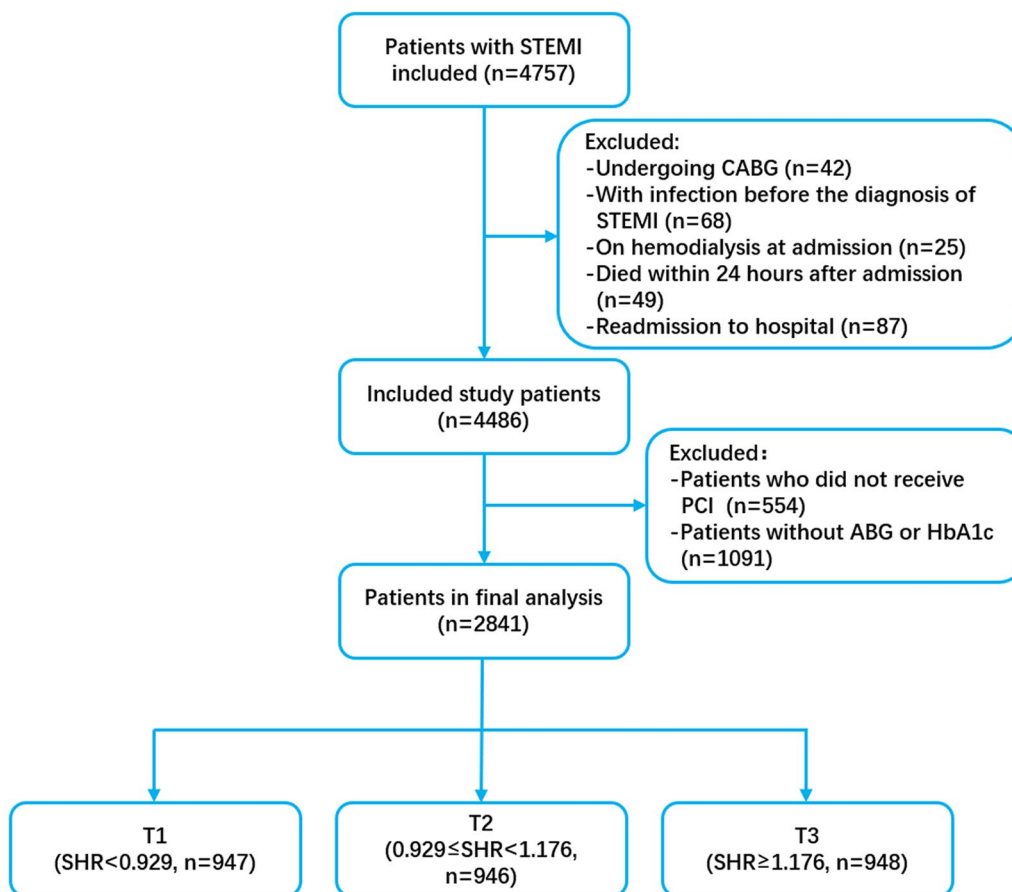


Fig. 1 Flow chart of participants. *STEMI* ST-segment elevation myocardial infarction, *CABG* coronary artery bypass grafting, *PCI* percutaneous coronary intervention, *ABG* admission blood glucose, *HbA1c* glycosylated hemoglobin, *SHR* stress hyperglycemia ratio

(adjusted OR=1.36, 95% CI 0.78–2.38, P=0.276) for patients without DM.

A cumulative effect was observed for the in-hospital MACEs since DM with high SHR (T3) associated with higher risk than that of DM with low SHR (T1) and high SHR (T3) without DM. No cumulative effect was observed for the outcome of pulmonary infection. (Additional file 1: Table S1).

Predictive value of the SHR for in-hospital events

The ROC curve of the SHR for pulmonary infection during hospitalization was drawn, and, as shown in Fig. 4A, had an AUC of 0.624 (95% CI 0.590–0.659). The best cut-off value of 1.073 was derived with a sensitivity of 64.7% and specificity of 56.8%. Subgroup analysis showed that the AUC was 0.595 (95% CI 0.538–0.652) and 0.637 (95% CI 0.593–0.680) for patients with and without DM, respectively (Fig. 4B). Additionally, an AUC of 0.629 (95% CI 0.584–0.674) was found for in-hospital MACEs (Fig. 4C).

Discussion

To the best of our knowledge, this is the first study to establish a correlation between SHR and pulmonary infection during hospitalization among patients with STEMI undergoing PCI. Our main findings were that the SHR is independently correlated with pulmonary infection and MACEs in patients with STEMI during hospitalization.

SIH and STEMI

SIH is a common status with a strong predictive value for worse clinical outcomes for patients with STEMI [5, 10, 11]. Several complex acute physiological changes, such as excessive gluconeogenesis, activation of the sympathetic-adrenergic-system, counter-regulatory hormones (e.g., catecholamine, cortisol), and proinflammatory cytokines (e.g., tumor necrosis factor- α), play roles in the development of SIH and lead to a vicious cycle [8, 9, 21, 22]. This vicious cycle is also believed to contribute to worse outcomes for critically ill patients, including all-cause mortality and pneumonia [10, 11, 16, 23]. The

Table 1 Comparison of baseline characteristics between groups stratified by tertile of SHR

Variables	Overall (n = 2841)	T1 (n = 947) SHR < 0.929	T2 (n = 946) 0.929 ≤ SHR < 1.176	T3 (n = 948) SHR ≥ 1.176	P value
Age, years	62.27 ± 12.18	61.67 ± 11.82	61.81 ± 12.53	63.32 ± 12.12	0.005
Male, n (%)	2347(82.6%)	810(85.5%)	794(83.9%)	743(78.4%)	< 0.001
SBP, mm Hg	122 ± 22	123 ± 21	122 ± 21	120 ± 22	0.019
DBP, mm Hg	74 ± 14	74 ± 13	74 ± 13	73 ± 13	0.045
Heart rate	80 ± 16	77 ± 14	80 ± 15	83 ± 17	< 0.001
Killip classification					< 0.001
I	1973 (69.5%)	692 (73.1%)	687 (72.7%)	594 (62.7%)	
II	586 (20.6%)	184 (19.4%)	186 (19.7%)	216 (22.8%)	
III	157 (5.5%)	40 (4.2%)	47 (5.0%)	70 (7.4%)	
IV	124 (4.4%)	31 (3.3%)	25 (2.6%)	68 (7.2%)	
Medical history, n (%)					
Current smoker	1179 (41.5%)	423 (44.7%)	409 (43.3%)	347 (36.6%)	< 0.001
Hypertension	1471(51.8%)	468 (49.4%)	488 (51.6%)	515 (54.3%)	0.101
Diabetes mellitus	882 (31.0%)	257 (27.1%)	245 (25.9%)	380 (40.1%)	< 0.001
Hyperlipidemia	361 (12.7%)	129 (13.6%)	127 (13.4%)	105 (11.1%)	0.180
Atrial fibrillation	91 (3.2%)	24 (2.5%)	37 (3.9%)	30 (3.2%)	0.235
COPD	65 (2.3%)	22 (2.3%)	26 (2.7%)	17 (1.8%)	0.379
Prior MI	698 (24.6%)	267 (28.2%)	208 (22.0%)	223 (23.5%)	0.005
Prior PCI	358 (12.6%)	141 (14.9%)	108 (11.4%)	109 (11.5%)	0.034
Prior stroke	202 (7.1%)	60 (6.3%)	61 (6.4%)	81 (8.5%)	0.109
Laboratory measurements					
WBC	11.66 ± 3.99	10.97 ± 3.60	11.77 ± 3.78	12.23 ± 4.45	< 0.001
Albumin	35.10 ± 4.43	35.15 ± 4.64	35.28 ± 4.26	34.86 ± 4.37	0.112
LVEF, %	51.26 ± 11.81	52.44 ± 11.54	51.77 ± 11.35	49.56 ± 12.32	< 0.001
Hemoglobin, g/L	134.19 ± 19.29	134.33 ± 18.90	135.64 ± 18.12	132.61 ± 20.67	0.003
Anemia	916 (32.3%)	302 (32.0%)	290 (30.7%)	324 (34.2%)	0.247
eGFR, mL/min	84.23 ± 31.60	85.21 ± 31.23	87.72 ± 30.78	79.75 ± 32.28	< 0.001
Admission blood glucose	8.81 ± 4.10	6.48 ± 2.16	8.20 ± 2.89	11.75 ± 4.80	< 0.001
HbA1c, %	6.10 (5.70–7.00)	6.20 (5.80–7.00)	6.00 (5.60–6.70)	6.10 (5.50–7.40)	< 0.001
Total cholesterol, mmol/L	4.89 ± 1.24	4.90 ± 1.23	4.92 ± 1.19	4.84 ± 1.31	0.372
LDL-C, mmol/L	3.21 ± 1.00	3.25 ± 0.98	3.23 ± 0.96	3.17 ± 1.06	0.193
Medication use					
Aspirin	2802 (98.6%)	941 (99.4%)	938 (99.2%)	923 (97.4%)	< 0.001
Clopidogrel	2496 (88.0%)	838 (88.5%)	814 (86.3%)	844 (89.1%)	0.145
Statins	2780 (97.9%)	928 (98.0%)	928 (98.1%)	924 (97.6%)	0.701
β-blockers	2324 (81.8%)	781 (82.5%)	775 (81.9%)	768 (81.1%)	0.738
ACEI/ARB	2254 (79.3%)	758 (80.0%)	745 (78.8%)	751 (79.2%)	0.782
Angiography					
Transradial assessment	2436 (85.9%)	851 (89.9%)	819 (86.8%)	766 (81.0%)	< 0.001
Multi-vessel stenosis, n (%)	2087 (73.5%)	707 (74.7%)	679 (71.8%)	701 (73.9%)	0.335
No. of stents	1.49 ± 2.04	1.54 ± 0.91	1.52 ± 3.31	1.41 ± 0.84	0.304
Length of stents, mm	37.43 ± 24.60	39.62 ± 26.13	36.22 ± 23.20	36.47 ± 24.27	0.004
Contrast volume, mL	115.66 ± 41.24	118.69 ± 44.78	112.34 ± 36.73	115.91 ± 41.57	0.005
Length of hospital stay, days	6.00 (5.00 ~ 9.00)	6.00 (5.00 ~ 8.00)	7.00 (6.00 ~ 8.00)	7.00 (6.00 ~ 11.00)	< 0.001

SHR stress hyperglycemia ratio, SBP systolic blood pressure, DBP diastolic blood pressure, COPD chronic obstructive pulmonary disease, MI myocardial infarction, PCI percutaneous coronary intervention, WBC white blood cell, LVEF left ventricular ejection fraction, eGFR estimated glomerular filtration rate, HbA1c glycosylated hemoglobin, LDL-C low-density lipoprotein cholesterol, ACEI/ARB angiotensin converting enzyme inhibitor/angiotensin receptor blocker

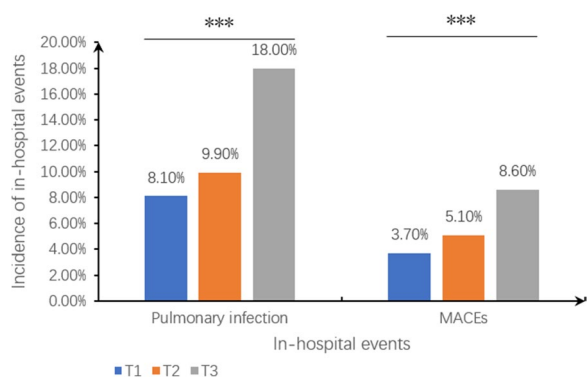


Fig. 2 Incidence of in-hospital events in different SHR groups. *** $P \leq 0.001$. SHR stress hyperglycemia ratio, MACEs major adverse cardiovascular events

relationship between SIH and the development of long- and short-term MACEs in patients with STEMI has been proven by prior studies [12, 14, 15]. In this research, we drew the same conclusion that SHR, reflecting SIH, is independently associated with the risk of MACEs during hospitalization. Despite firm evidence of the relationship

between SIH and MACEs, the correlation between SIH and pulmonary infection during hospitalization for patients with STEMI remains unclear.

SHR with pulmonary infection in patients with STEMI

As an important complication of STEMI, infection during hospitalization is associated with a worse clinical outcome [1–4], and several risk factors [24, 25] and scores [4, 26, 27] are utilized for infection prediction and prevention. Unfortunately, these predictive tools are largely complex or unable to be interfered.

In this research, we used the SHR, a simple and readily available parameter, on behalf of SIH, and found that it is independently associated with the risk of pulmonary infection during hospitalization for patients with STEMI. Although the underlying mechanism of this finding remains unclear, we hypothesize that the high pulmonary infection rate among high SHR patients with STEMI is attributed to the harmful effects of stress hyperglycemia. Glucose is considered a pro-inflammatory mediator, and elevation of blood glucose not only induces the generation of inflammatory cytokines, inflammatory processes,

Table 2 Multivariable logistic regression analysis for the SHR as categorical variable and continuous variable

Variables	Pulmonary infection			In-hospital MACEs		
	OR	95% CI	P value	OR	95% CI	P value
T1	Reference			Reference		
T2	1.21	0.84–1.74	0.296	1.32	0.83–2.11	0.238
T3	1.75	1.25–2.46	0.001	1.68	1.09–2.60	0.020
SHR*	1.46	1.06–2.02	0.021	1.67	1.17–2.39	0.005

Adjust for Age, Gender, eGFR, WBC, Anemia, Current smoker, DM, Hypertension, COPD, Prior MI, Prior PCI, Prior stroke, PCI assessment and Multi-vessel stenosis

SHR stress hyperglycemia ratio, MACEs major adverse cardiovascular events, OR odds ratio, CI confidence interval, eGFR estimated glomerular filtration rate, WBC white blood cell, DM Diabetes mellitus, COPD chronic obstructive pulmonary disease, MI myocardial infarction, PCI percutaneous coronary intervention

* SHR as continuous variable

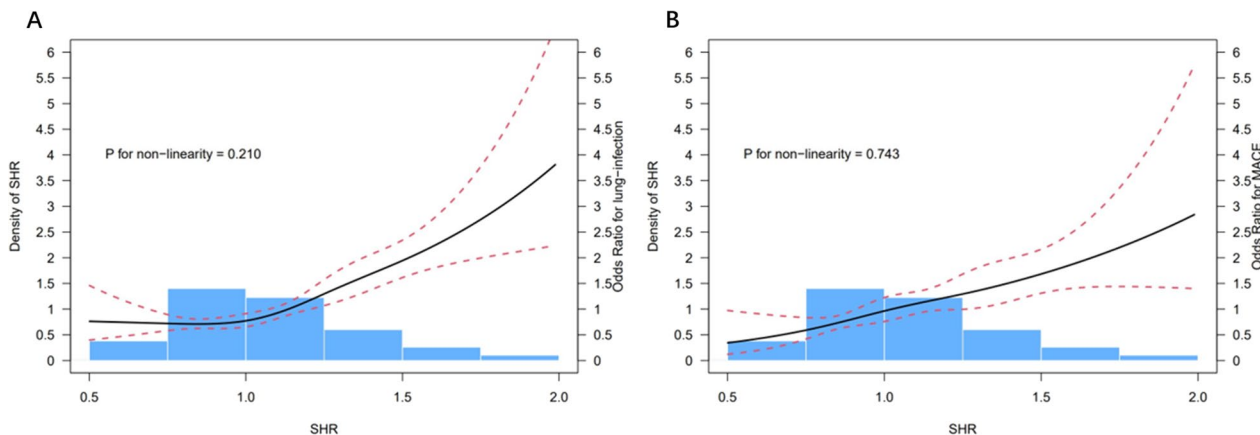


Fig. 3 **A** Cubic spline models for the association between SHR and pulmonary infection. **B** Cubic spline models for the association between SHR and in-hospital MACEs. SHR stress hyperglycemia ratio, MACEs major adverse cardiovascular events

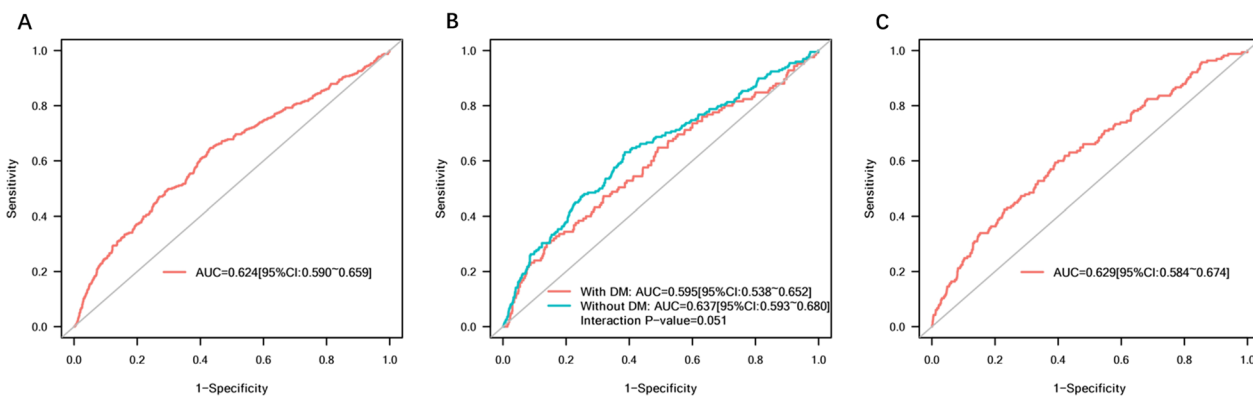


Fig. 4 **A** ROC curve of SHR for pulmonary infection among STEMI patients. **B** ROC curve of SHR for pulmonary infection among STEMI patients with or without DM. **C** ROC curve of SHR for in-hospital MACEs among STEMI patients. *ROC curve* receiver operating characteristic curve, *AUC* area under the ROC curve, *DM* diabetes mellitus, *SHR* stress hyperglycemia ratio, *STEMI* ST-segment elevation myocardial infarction, *MACEs* major adverse cardiovascular events

and insulin resistance [8, 9, 21], but also decreases vascular endothelial nitric oxide, which leads to vasoconstriction and low organ perfusion [28]. Moreover, glucose directly inhibits the function of T lymphocytes, immunoglobulin, and complement, disrupts the immune system, and further increases the risk of pulmonary infection [28]. In diabetic animal models, hyperglycemia can also lead to lower bacterial clearance and a higher rate of infection-related mortality [29–31]. Moreover, patients with high SHR have been found to be more likely to suffer from in-hospital MACEs [12, 32–34], which may increase the usage of invasive procedures, such as mechanical ventilation, thereby increasing the risk of pulmonary infection [35, 36]. This result provides a potential standard for admission blood glucose control for patients with STEMI during the acute phase, although further studies are needed.

This research found no significant non-linear relationship between the SHR and pulmonary infection or MACEs during hospitalization. Similarly, in the research of Yang et al. [34], although a J-shape relationship was confirmed between the SHR and major adverse cardiovascular and cerebrovascular events, MACE, cardiac death, and MI at a 2-year follow-up, neither a U nor J-shape relationship was found for in-hospital cardiac death and MI. These consistent findings highlight that the influence of the SHR on different outcomes in different periods may vary and have diverse mechanisms, which cannot yet be fully explained. Further studies are needed to clarify this phenomenon.

SHR with infection in patients with STEMI with or without DM

The SHR was also found to be independently associated with the risk of pulmonary infection during hospitalization among patients with STEMI with or without DM. In the ROC curve of SHR for pulmonary infection, the

AUC of patients without DM was slightly larger than that of patients with DM ($P=0.051$). This tendency is consistent with previous studies that focused on other aspects of STEMI outcomes [12, 14]. As an explanation, chronic hyperglycemia of DM may induce antioxidant defenses and protect tissues and cells from the oxidative stress of acute hyperglycemia [37–39].

Although a strong association between SIH and STEMI prognosis has been declared, the optimal management for SIH is still debated. Moreover, clinical trials of SIH therapies have yielded conflicting results. Indeed, Kosiborod et al. [40] showed an association between glucose normalization and better survival in patients with acute myocardial infarction and SIH. In contrast, a meta-analysis of three clinical studies revealed few benefits of intensive blood glucose control, but an increased risk of serious hypoglycemia in patients with acute myocardial infarction and DM [41]. In this research, the SHR was proven to be independently associated with the risk of pulmonary infection and MACEs during hospitalization for patients with STEMI undergoing PCI. As the SHR, a better index of relative stress hyperglycemia, was shown to be a better predictor of worse outcomes, we propose that glycemic targets based on SHR instead of ABG may be applied to the management of acute hyperglycemia in further studies.

Strengths and limitations

To the best of our knowledge, this is the first study to establish the correlation between SHR and pulmonary infection during hospitalization among patients with STEMI who underwent PCI. However, our study has several limitations. First, as this is an observational study, we could not adjust for all potential confounders. Second, only patients with STEMI in one center

were included, which may limit the generalization of our conclusion. Third, blood glucose levels are prone to fluctuation, and the variation in durations from the onset of symptoms to the measurement of ABG and SHR may have impacted the results. Fourth, because of the small sample size of patients with STEMI without PCI, the impact of SHR on outcomes in these patients remained unclear. Therefore, further studies are needed to confirm the association between the SHR and pulmonary infection during hospitalization in patients with STEMI without PCI treatment.

Conclusion

The SHR is independently associated with the risk of pulmonary infection during hospitalization and in-hospital MACEs for patients with STEMI who underwent PCI.

Abbreviations

STEMI	ST-segment elevation myocardial infarction
SIH	Stress-induced hyperglycemia
ABG	Admission blood glucose
SHR	Stress hyperglycemia ratio
DM	Diabetes mellitus
PCI	Percutaneous coronary intervention
HbA1c	Glycosylated hemoglobin
MI	Myocardial infarction
MACEs	Major adverse cardiovascular events
eGFR	Estimated glomerular filtration rate
ROC	Receiver operating characteristic
AUC	Area under the ROC curve
OR	Adjusted odds ratio
CI	Confidence interval

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12933-023-01799-3>.

Additional file 1: Table S1. Interaction and cumulative effects analysis between diabetes and SHR. **Figure S1.** Kaplan–Meier analyses for in-hospital pulmonary infection among the three groups. **Figure S2.** Kaplan–Meier analyses for in-hospital MACEs among the three groups. **Table S2.** Multivariable cox regression analysis for the SHR as categorical variable and continuous variable.

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Not applicable.

Author contributions

ZL, XL and YL contributions to the conception and design of the work; YZ, YD, LZ, WC and SK contributions to the acquisition of the data; XL and CD contributions to the analysis of the data; ZL, XL and YZ interpretation of data; ZL have drafted the work; PH, YL and YZ substantively revised it. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and analyzed during the present study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was conducted according to the guidelines stipulated in the Declaration of Helsinki and approved by the research ethics committee of Guangdong Provincial People's Hospital.

Consent for publication

Not applicable.

Competing interests

All authors declare that they have no competing interests.

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