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RESEARCH ARTICLE

Prognostic value of fasting glucose on the risk of heart failure and left ventricular systolic dysfunction in non-diabetic patients with ST-segment elevation myocardial infarction

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Abstract **Recent studies have shown that acute blood glucose elevation in patients with ST-segment elevation myocardial infarction (STEMI) suggests a poor prognosis. To investigate the effect of fasting blood glucose (FBG) on the risk of heart failure (HF) and left ventricular systolic dysfunction (LVSD) in non-diabetic patients undergoing primary percutaneous coronary intervention (PCI) for acute STEMI, we retrospectively recruited consecutive non-diabetic patients who underwent primary PCI for STEMI in our hospital from February 2003 to March 2015. The patients were divided into two groups according to the FBG level. A total of 623 patients were recruited with an age of 61.3 ± 12.9 years, of whom 514 (82.5%) were male. The HF risk (odds ratio 3.401, 95% confidence interval (CI) 2.144–5.395, $P < 0.001$) was significantly increased in patients with elevated FBG than those with normal FBG. Elevated FBG was also independently related to LVSD (β 1.513, 95%CI 1.282–1.785, $P < 0.001$) in a multiple logistics regression analysis. In conclusion, elevated FBG was independently associated with 30-day HF and LVSD risk in non-diabetic patients undergoing primary PCI for STEMI.**

Keywords myocardial infarction; percutaneous coronary intervention; diabetes mellitus; fasting glucose; heart failure

Introduction

ST-segment elevation myocardial infarction (STEMI) is a common cardiovascular emergency with high mortality. With the development of primary percutaneous coronary intervention (PCI) and secondary prevention drugs for coronary heart disease (CAD), the recent mortality rate of STEMI patients is decreasing [1]. However, the proportion of patients who develop heart failure (HF) after myocardial infarction (MI) has increased over time as peri-MI mortality rates declined [2]. Methods to identify STEMI patients with a high risk of HF after primary PCI must be urgently developed.

The China PEACE-Retrospective Acute Myocardial Infarction (AMI) Study, which characterized the trends in the epidemiology, treatment, and outcomes of patients with STEMI in China from 2001 to 2011, found substantial increases in the estimated national rate of hospital admission for STEMI, gaps in quality of care with the underuse of guideline-recommended therapies and the use of therapies of unknown effectiveness, and a growing burden of prevalent coronary risk factors [3]. In addition, a number of studies have shown that acute blood glucose elevation in patients with STEMI suggests a poor short-term prognosis [4–6]. Fasting blood glucose (FBG) has also been found to be related to adverse outcomes in patients with AMI and has recently been reported to be a better prognostic factor of mortality than acute blood glucose [7]. However, there is insufficient research evidence for the relevance between FBG and the risk of HF and left ventricular systolic dysfunction (LVSD) in STEMI patients undergoing primary PCI. In addition, the correlation between blood glucose and the prognosis of patients with STEMI is not the same in diabetic and non-diabetic patients; the predictive value of blood glucose for non-diabetic STEMI patients is more significant [8,9]. To this end, we conducted this large-scale, single-center study to determine the prognostic value of FBG for short-term

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HF and LVSD risk in non-diabetic STEMI patients receiving primary PCI.

Materials and methods

Study subjects

Consecutive STEMI patients who underwent primary PCI at our hospital from February 2003 to March 2015 were included. The STEMI patients were diagnosed according to the "2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation" [10]. Exclusion criteria were (1) previous definitive diagnosis of diabetes mellitus; (2) no previous diabetes mellitus history but a post-admission HbA1c $\geqslant$ 6.5%; and (3) no echocardiographic examination within 30 days of onset.

Study methods

The patients' age, sex, body mass index (BMI), smoking history, previous history of diabetes and other medical diseases, previous MI and coronary revascularization (including PCI and coronary artery bypass graft), and family history of CAD were recorded. Blood samples for FBG level were obtained after an overnight fast of 8–12 h within 24 h of admission. Biochemical parameters such as baseline blood pressure, heart rate, liver and kidney function, electrolytes, and blood lipids of all patients were measured at the time of treatment. Emergency coronary angiography was performed through the radial or femoral artery after the final diagnosis of STEMI. The "culprit vessel" was the only target of primary PCI treatment without limiting the application and choice of stents. An intra-aortic balloon pump (IABP) was used in patients with hemodynamic instability. All patients were given a loading dose of aspirin and clopidogrel before primary PCI. Secondary prevention drug therapy for CAD, including dual antiplatelet therapy with aspirin and clopidogrel, β-receptor blockers, angiotensin converting enzyme inhibitors (ACEIs)/angiotensin II receptor blockers (ARBs), and statins, was used routinely. Each patient's 18-lead ECG was recorded preoperatively and postoperatively. Serum creatine kinase, creatine kinase myocardial band (CK-MB), and cardiac troponin I (cTnI) were routinely measured every 2 h until all of them reached peak values. These myocardial markers were detected every 6 h until 72 h after onset. Venous blood was drawn at 24 h after the onset of symptoms to detect N-terminal pro-brain natriuretic peptide (NT-proBNP). Echocardiography was performed at 15 $\pm$ 9 days after onset to determine the size and systolic function of the left ventricle. Left ventricular ejection fraction (LVEF) was measured by the biplane method. FBG tests were performed for all patients within 24 h of admission. According to FBG, patients were divided into a normal FBG group (FBG $\leqslant$ 6.1 mmol/L) and an elevated FBG group (FBG $>$ 6.1 mmol/L), and the baseline clinical characteristics, coronary angiography data, and medical and revascularization treatment were compared between groups. In addition, left ventricular size; LVSD; and risk of developing HF, arrhythmia, cardiac mechanical complications, cardiogenic shock, cardiogenic death, and all-cause mortality within 30 days were also compared between the two groups. Univariate and multivariate analyses were performed to identify the independent association of 30-day HF and LVSD.

Study definitions

Symptom-to-door time was defined as the interval between the onset of symptoms and arrival at the hospital. Door-to-balloon time was defined as the interval between a patient's arrival at the hospital and first balloon inflation as documented in the patient's medical record. Symptom-to-balloon time was defined as the interval between the onset of symptoms and first balloon inflation as documented in the patient's medical record. Successful PCI was defined as a residual stenosis $<$ 20% in target lesions in stented patients, a residual stenosis $<$ 50% in target lesions in patients with simple balloon dilatation, and thrombolysis in MI (TIMI) grade 3 forward blood flow in the target vessel. HF was judged according to the clinical characteristics of the patients, and cardiac function was classified into grades I to IV according to the Killip classification. HF was defined as the documentation of clinical symptoms of HF (dyspnea on exertion or fluid retention) or signs of HF (rales, jugular venous distension, pulmonary edema) by a physician with a Killip class $\geqslant$ 2 at any time of hospitalization. LVSD was defined as biplane LVEF $<$ 40%. Contrast-induced acute kidney injury (CI-AKI) was defined as $\geqslant$ 25% increase in serum creatinine within 72 h after the procedure [11].

Statistical analysis

Categorical data were presented as frequency and percentage. Continuous variables were reported as mean $\pm$ standard deviation or median and interquartile range. Independent *t*-test or Mann–Whitney U test and chi-square test or Fisher's exact test were performed as appropriate for intergroup comparison. Uncorrected Kaplan–Meier survival curves were used to compare the risk of 30-day HF between the normal FBG group and the elevated FBG group. Univariate and multivariate COX regression analyses were used to determine the association of 30-day HF. Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated. Binary logistic regression analysis was used to determine the independent association of LVSD. All statistical analyses were performed at a two-

sided significance level of 0.05 by using the SPSS software version 22.0 (IBM corporation).

Results

General information

A total of 1218 patients undergoing primary PCI for STEMI between February 2003 and March 2015 were identified. Patients with previous diabetes (n = 425) or newly diagnosed diabetes (n = 72) were excluded. Of the 721 non-diabetic patients, 98 patients who did not undergo echocardiography within 30 days of onset were excluded. Therefore, 623 patients were eventually enrolled. The mean age of patients was 61.3 ± 12.9 years, and 514 (82.5%) were male. A total of 90 (14.4%) and 53 (8.5%) patients developed HF and LVSD, respectively, within 30 days. A total of 14 (2.2%) cases of 30-day all-cause mortality were found, including 12 (1.9%) cases of cardiogenic death.

Comparison between the elevated FBG group and normal FBG group

Among 623 non-diabetic patients with STEMI, 161 (25.8%) patients were included in the elevated FBG group (FBG $>$ 6.1 mmol/L), and 462 (74.2%) patients were in the normal FBG group (FBG $\leqslant$ 6.1 mmol/L). The baseline data of these patients are shown in Table 1. Patients in the elevated FBG group had a higher heart rate at admission than did the normal FBG group. No significant differences were found between the two groups in terms of demographic data, cardiovascular disease risk factors, comorbidities, coronary angiography, PCI, and medical treatment (Table 1). Patients in the elevated FBG group had significantly higher risks of 30-day HF (odds ratio (OR) 3.401 (95% CI 2.144 to 5.395), $P < 0.001$), all-cause mortality (OR 5.412 (95% CI 1.786 to 16.397), P = 0.001), and cardiogenic death (OR 4.155 (95% CI 1.300 to 13.280), P = 0.009) compared with those in the normal FBG group.

FBG and HF

The NT-proBNP levels, CK-MB, and cTnI peaks in the elevated FBG group were significantly higher compared with those in the normal FBG group (Table 2). The 30-day HF risk in the elevated FBG group was significantly increased compared with that in the normal FBG group ($P < 0.001$, Fig. 1). Univariate COX regression analysis showed that age, previous MI, FBG, estimated glomerular filtration rate, left anterior descending artery (LAD) as the culprit vessel, and use of ACEIs/ARBs were significantly correlated with 30-day HF risk (Table 3). However, BMI; combined hypertension; previous coronary revascularization; symptom-to-balloon time; triple vessel lesion; preoperative TIMI grade flow 0; and the use of β-receptor blockers, GPIIb/IIIa receptor antagonists, and IABP were not associated with the risk of HF. Multivariate COX regression analysis showed that after the baseline clinical characteristics were adjusted, FBG remained independently associated with the 30-day HF risk in non-diabetic STEMI patients (HR 1.273, 95% CI 1.168 to 1.388, $P < 0.001$); in addition, age, previous MI, and LAD as the culprit vessel were also independently associated with the 30-day HF risk (see Table 3 for details).

FBG and left ventricular structure and systolic function

Left ventricular end-diastolic diameter was significantly increased (52.3 ± 5.8 mm vs. 49.4 ± 4.2 mm, $P <$ 0.001), and LVEF was significantly decreased (51.4% ± 12.0% vs. 59.1% ± 10.05%, $P < 0.001$) in the elevated FBG group compared with the normal FBG group (Fig. 2). The incidence of LVSD was significantly higher in the elevated FBG group than in the normal FBG group (19.3% vs. 4.8%, $P < 0.001$). Logistics regression analysis showed that age, FBG, estimated glomerular filtration rate, symptom-to-balloon time, and LAD as the culprit vessel were risk factors for developing LVSD. However, BMI; combined hypertension; previous coronary revascularization; previous MI; triple vessel lesion; preoperative TIMI grade flow 0; and the use of β-receptor blockers, ACEIs/ARBs, and GPIIb/IIIa receptor antagonists or IABP were not significantly associated with LVSD. Further multivariable logistic regression analysis showed that FBG remained independently associated with LVSD (β 1.513, 95% CI 1.282 to 1.785, $P < 0.001$); in addition, symptom-to-balloon time and LAD as the culprit vessel were also independently associated with LVSD (see Table 4 for details).

Discussion

In the present study, we found that FBG elevation was independently associated with greater risk for 30-day HF and LVSD in STEMI patients without established diabetes who underwent primary PCI.

Among the 793 STEMI patients without previous diagnosed diabetes, HbA1c levels $\geqslant$ 6.5% identified an additional 72 patients (5.9%) as having diabetes. Another single-center registry study of 2146 AMI patients without prior history of diabetes demonstrated that the percentage of new diabetes cases diagnosed by impaired glucose tolerance test after admission was as high as 14.3% [12]. These results suggest that a considerable proportion of STEMI patients without previous diagnosis of diabetes have developed diabetes but have not yet been identified.

Table 1 Baseline characteristics of normal FBG group and elevated FBG group

	Normal FBG group (n = 462)	Elevated FBG group (n = 161)	P value
Demographic characteristics			
Age (year)	61.5±12.8	60.8±13.2	0.535
Gender (male %)	383 (82.9)	131 (81.4)	0.718
BMI (kg/m^2)	24.8±3.5	25.0±3.8	0.568
Risk factors of CAD (n (%))			
Hypertension	253 (54.8)	100 (62.1)	0.117
Hyperlipidemia	206 (44.6)	68 (42.2)	0.645
Smoking	293 (63.4)	101 (62.7)	0.924
Premature CAD family history	98 (21.2)	34 (21.1)	1.000
Complications and previous history of CAD (n (%))			
CKD	15 (3.2)	9 (5.6)	0.232
Stroke	42 (9.1)	15 (9.3)	1.000
Previous MI	26 (5.6)	8 (5.0)	0.843
Coronary revascularization	25 (5.4)	6 (3.7)	0.529
Clinical features			
Symptom-to-door time (h)	2.5 (1.5, 5.0)	3.0 (1.5, 5.4)	0.495
Admission HR (time/min)	80.7±17.0	86.1±17.2	0.001
Admission SBP (mmHg)	116.8±19.9	117.5±22.4	0.724
Anterior MI (n (%))	243 (52.6)	96 (59.6)	0.141
Laboratory examination			
Admission Hb (g/ L)	141.7±18.6	145.1±23.8	0.180
eGFR (mL/min)	75.3 (64.8, 88.1)	80.0 (66.5, 96.8)	0.034
LDL-C (mmol/L)	2.9 (2.4, 3.5)	2.9 (2.3, 3.4)	0.136
WBC (10^9/L)	9.6 (7.7, 11.6)	10.4 (8.2, 13.8)	0.017
hsCRP (mg/L)	6.5 (2.0, 12.0)	7.4 (4.8, 15.0)	0.003
HbA1c (%)	5.7 (5.4, 6.0)	5.8 (5.6, 6.2)	0.097
Coronary arteriography			
Symptom-to- balloon time (h)	5.0 (3.0, 7.7)	5.0 (3.5, 8.8)	0.178
LAD as the culprit vessel (n (%))	233 (50.4)	88 (54.7)	0.362
Multi-vessel lesion (n (%))	349 (75.5)	117 (72.7)	0.463
Triple vessel lesion (n (%))	205 (44.4)	62 (38.5)	0.229
TIMI 0 in preoperative (n (%))	326 (70.6)	120 (74.5)	0.362
TIMI 3 in postoperative (n (%))	34 (7.4)	12 (7.5)	1.000
Successful PCI (n (%))	432 (93.5)	156 (96.9)	0.116
IABP application (n (%))	10 (2.2)	7 (4.3)	0.161
Drug therapy (n (%))			
GPIIb/IIIa receptor antagonist	181 (39.2)	66 (41.0)	0.709
β–receptor blocker	414 (89.6)	140 (87.0)	0.382
ACEI/ARB	400 (86.6)	130 (80.7)	0.094
Statin	439 (95.0)	158 (98.1)	0.109

All values are presented as the mean±SD or n (%) or as the median (interquartile range). P value represents mean differences between groups. FBG, fasting blood glucose; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; MI, myocardial infarction; HR, heart rate; SBP, systolic blood pressure; Hb, hemoglobin; eGFR, estimated glomerular filtration rate; LDL-C, low density lipoprotein cholesterol; WBC, white blood cell; hsCRP, high sensitive C-reactive protein; HbA1C, glycated hemoglobin; LAD, left anterior descending artery; TIMI, thrombolysis in MI; PCI, percutaneous coronary intervention; IABP, intra-aortic balloon pump; GPIIb/IIIa, glycoprotein IIb/IIa; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blockers.

Therefore, diabetes screening is necessary for all patients admitted for STEMI and will help in the early diagnosis and prompt treatment of dysglycemia.

Acute hyperglycemia on admission is significantly associated with poor prognosis of STEMI [5,13,14]. In recent years, blood glucose in the acute phase has high

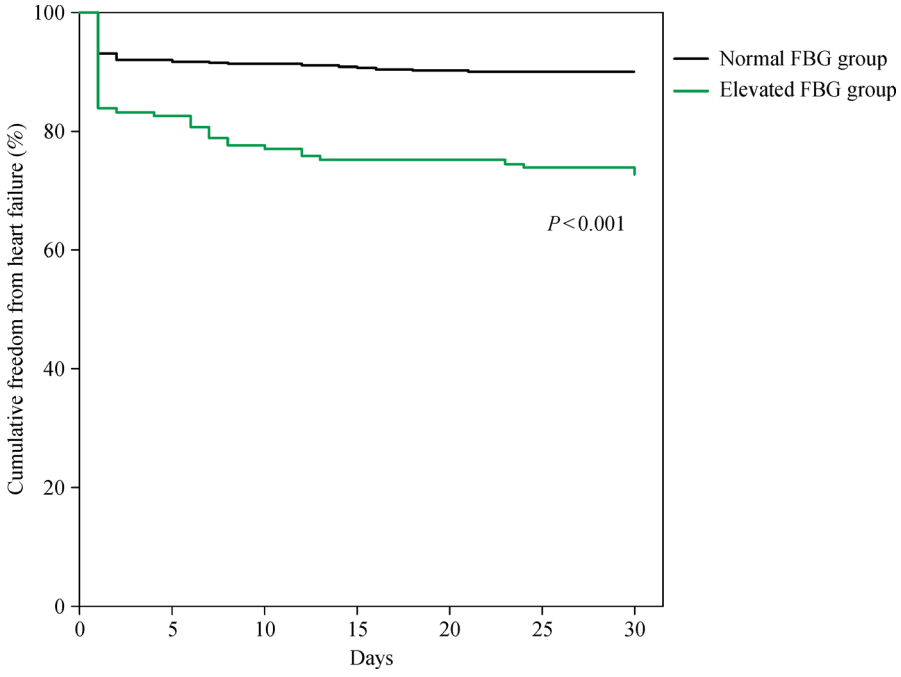

Fig. 1 Kaplan–Meier estimates of freedom from heart failure. The risk of 30-day HF was significantly increased in patients in the elevated FBG group compared with those in the normal FBG group, $P < 0.001$. HF, heart failure; FBG, fasting blood glucose.

Table 2 Comparison of HF, MI complications, and prognosis in different FBG groups at 30 days

	Normal FBG group (n = 462)	Elevated FBG group (n = 161)	P value
HF (n (%))	46 (10.0)	44 (27.3)	<0.001
NT-proBNP (pg/mL)	420.0 (200.0, 1400.0)	1380.0 (337.3, 4206.8)	<0.001
CK-MB peak (ng/mL)	183.3 (94.3, 381.1)	334.9 (130.4, 587.4)	<0.001
cTnI peak (ng/mL)	70.8 (32.5, 149.7)	116.2 (50.2, 204.6)	<0.001
LVEDD (mm)	49.4±4.2	52.3±5.8	<0.001
LVEF (%)	59.1±10.0	51.4±12.0	<0.001
Recurrent MI (n (%))	7 (1.5)	1 (0.6)	0.687
Cardiac shock (n (%))	19 (4.1)	15 (9.3)	0.016
Malignant arrhythmia (n (%))	34 (7.4)	29 (18.0)	<0.001
Cardiac mechanical complication (n (%))	19 (4.1)	13 (8.2)	0.044
Cardiac death (n (%))	5 (1.1)	7 (4.3)	0.009
CI-AKI (n (%))	28 (6.1)	24 (14.9)	0.001
All-cause mortality (n (%))	5 (1.1)	9 (5.6)	0.001

All values are presented as the mean±SD or n (%) or as the median (interquartile range). P value represents mean differences between groups. HF, heart failure; NT-proBNP, N-terminal pro-brain natriuretic peptide; CK-MB, creatine kinase myocardial band; cTnI, cardiac troponin I; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; MI, myocardial infarction; CI-AKI, contrast-induced acute kidney injury.

predictive value for the prognosis of nondiabetic AMI patients [8]. A recent study demonstrated that non-diabetic patients with hyperglycemia have significantly higher mortality and comorbidity, including cardiogenic shock and cardiac arrhythmias, compared with diabetic patients [15]. This study also showed that admission hyperglycemia is independently associated with the in-hospital death in non-diabetic patients but not in diabetic patients [15], suggesting that acute-phase blood glucose tests have a great clinical significance for non-diabetic STEMI patients and that more attention should be paid to the detection and assessment of acute-phase blood glucose in this population.

Furthermore, hyperglycemia during the acute phase of AMI is associated with a surge of catecholamines and decreased insulin sensitivity, which reflect a compromised

Table 3 Independent association of risk of HF within 30 days of the patient

	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Age	1.038 (1.020 – 1.056)	<0.001	1.032 (1.011 – 1.052)	0.002
Hypertension	1.210 (0.792–1.848)	0.379	1.004 (0.646–1.560)	0.986
Previous MI	2.954 (1.608–5.429)	<0.001	2.990 (1.585–5.639)	0.001
FBG	1.308 (1.213–1.411)	<0.001	1.273 (1.168–1.388)	<0.001
eGFR	0.984 (0.975–0.994)	0.001	0.993 (0.982–1.005)	0.244
Symptom-to-balloon time	1.012 (0.975–1.049)	0.542	1.006 (0.966–1.046)	0.787
LAD as the culprit vessel	2.416 (1.531–3.812)	<0.001	2.261 (1.422–3.595)	0.001
β-receptor blocker	1.498 (0.692–3.239)	0.305	1.373 (0.629–2.997)	0.426
ACEI/ARB	0.524 (0.324–0.848)	0.008	0.730 (0.425–1.253)	0.253

All values are presented as the median (interquartile range). HR, hazard ratio; CI, confidence interval; MI, myocardial infarction; FBG, fasting blood glucose; eGFR, estimated glomerular filtration rate; LAD, left anterior descending artery; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blockers.

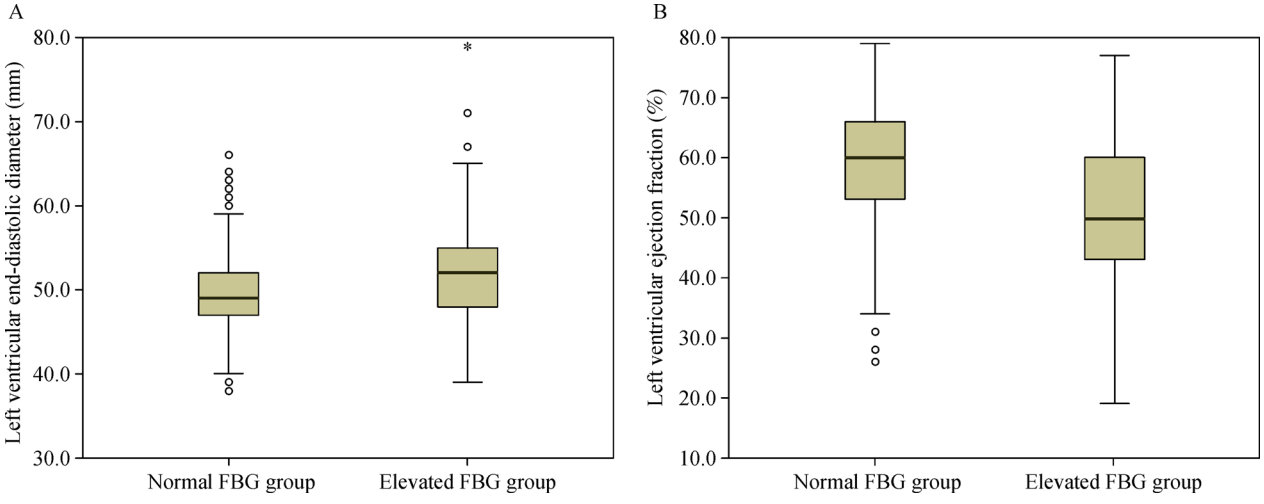

Fig. 2 Comparison of left ventricular size and systolic function in different FBG groups. Left ventricular end-diastolic diameter (LVEDD) was significantly increased (52.3 ± 5.8 mm vs. 49.4 ± 4.2 mm, $P < 0.001$), and left ventricular ejection fraction (LVEF) was significantly decreased (51.4% ± 12.0% vs. 59.1% ± 10.05%, $P < 0.001$) in patients in the elevated FBG group compared with those in the normal FBG group. FBG, fasting blood glucose.

Table 4 Independent association of left ventricular systolic dysfunction

	β (95% CI)	*P* value	β (95% CI)	*P* value
Age	1.011 (0.989–1.034)	0.326	1.006 (0.980–1.034)	0.649
FBG	1.541 (1.320–1.799)	<0.001	1.513 (1.282–1.785)	<0.001
eGFR	0.996 (0.985–1.008)	0.532	0.997 (0.984–1.011)	0.692
Symptom-to-balloon time	1.068 (1.025–1.113)	0.002	1.080 (1.030–1.131)	0.001
LAD as the culprit vessel	4.517 (2.226–9.163)	<0.001	4.493 (2.152–9.384)	<0.001

All values are presented as the median (interquartile range). CI, confidence interval; FBG, fasting blood glucose; eGFR, estimated glomerular filtration rate; LAD, left anterior descending artery.

metabolic state related to myocardial damage [16]. More importantly, acute hyperglycemia is not only a predictor of infarct size; hyperglycemia itself can also lead to further aggravation of myocardial injury. The mechanism by which hyperglycemia is associated with poor outcome of AMI patients remains unclear. Extensive myocardial damage can cause a rise in stress hormones, leading to an insulin-resistant state and reactive hyperglycemia in

non-diabetic patients with AMI [17]. In addition, hyperglycemia itself may activate and promote systemic inflammation [18], oxidative stress response, and free radical generation [19] and cause endothelial dysfunction [20], a hypercoagulable state [21], an impaired ischemic preconditioning [22], coronary microvascular dysfunction [23], and aggregate myocardial injury. Therefore, elevated FBG may be a predictor of large infarct size and may be correlated with HF in patients with STEMI. In summary, elevated FBG during the acute phase of STEMI may reflect the acute stress of large MI, exacerbate myocardial damage, and exaggerate LV remodeling by the mechanism mentioned above.

The presence of LVSD after MI is an important predictor of poor short- and long-term prognosis in patients with STEMI [24]. Therefore, the early identification of high-risk STEMI patients who may develop LVSD after PCI is of paramount importance and can facilitate more aggressive early treatment to improve patient prognosis. This study showed that FBG was linearly and negatively correlated with LVEF and that patients with elevated FBG had a significantly increased risk of LVSD after PCI. Cardiac enzyme peaks were significantly higher in patients with elevated FBG at admission compared with those with normal FBG, consistent with previous results [25], indicating that FBG elevation on admission is positively correlated with infarct size in patients with STEMI. These findings suggest that FBG is significantly associated with the degree of myocardial injury and can be used to predict infarct size and degree of injury after PCI in patients with STEMI.

This study showed that patients with elevated FBG still had a significantly higher risk of HF and LVSD than those with normal FBG, even with primary PCI as their reperfusion strategy. This finding suggests that acute-phase FBG is of high value in predicting the risk of HF and LVSD after PCI and can be used for risk assessment for non-diabetic STEMI patients. For patients with STEMI, FBG results at admission should be valued, and non-diabetic patients with elevated FBG should be considered at high risk for HF and LVSD after PCI. In terms of treatment, in addition to early opening of the culprit vessel and optimizing secondary prevention medication, echocardiography should be performed as soon as possible. Furthermore, anti-remodeling therapy, such as ACEIs/ARBs, angiotensin receptor neprilysin inhibitors, β-receptor blockers, and aldosterone antagonists, should be used early where appropriate to reduce the risk of HF and prevent the deterioration of LVSD in patients with STEMI. In addition, FBG and HbA1c should be used as routine markers for patients with STEMI, and attention should be paid to the analysis of the results.

CI-AKI is a complication of coronary angiography that occurs commonly in the setting of AMI and is associated with severe adverse outcomes, including permanent renal impairment and high mortality [26,27]. In this sample of non-diabetic patients undergoing primary PCI for STEMI, we found that the rate of CI-AKI was significantly higher in patients with elevated FBG than those with normal FBG. These findings support the results of a previous study, which showed that elevated glucose levels are associated with greater risk for CI-AKI in AMI patients without established diabetes or impaired renal function [28,29]. Our findings together with the previous report imply that non-diabetic patients with elevated FBG during the acute phase of STEMI should be recognized as a high-risk group for CI-AKI; furthermore, post-procedural renal function should be closely monitored, and prophylactic therapies used to prevent CI-AKI should be considered in this patient group.

For non-diabetic STEMI patients, research on whether hypoglycemic intervention is necessary for elevated FBG at admission is insufficient. However, prior studies showed no clear beneficial evidence for hypoglycemic intervention in the acute phase of STEMI for patients with diabetes [30,31]. Therefore, there is no clear evidence-based support for the use of hypoglycemic therapy in STEMI patients with elevated FBG, and the optimal treatment strategy for such patients remains to be further clarified by large-scale prospective studies.

Some limitations in the present study need to be considered and addressed in the future. First, this is a retrospective study, and the sample size was small. In particular, despite our results showing that patients in the elevated FBG group had significantly higher risks of all-cause mortality and cardiogenic death compared with those in the normal FBG group, the amount of data was too small to make a convincing conclusion. No further analysis was performed. Second, the end point is insufficient. We mainly focused on the observation of 30-day HF and LVSD. In the future, we can expand the sample for further research and increase the endpoint events.

In summary, elevated FBG was independently associated with greater risk for 30-day HF and LVSD in STEMI patients without established diabetes who underwent primary PCI. The early detection of post-procedural LVSD and optimal anti-remodeling therapies of heart in those patients can prevent the deterioration of LV systolic function, thereby improving their overall prognosis.

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Compliance with ethics guidelines

Hui Wang, Yang Zhang, Zhujun Shen, Ligang Fang, Zhenyu Liu, and Shuyang Zhang declare that they have no conflict of interests. All procedures were in accordance with the ethical standards of the

responsible committee on human experimentation (institutional and national) and the *Helsinki Declaration* of 1975, as revised in 2000 (5). Informed consent was obtained from all patients that were included in the study.

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