



# Risk factors for in-hospital death in acute ST-segment elevation myocardial infarction after emergency percutaneous coronary intervention: a multicenter retrospective study

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**Background:** For some acute ST-segment elevation myocardial infarction (STEMI) cases, the risk of in-hospital death remains high even after emergency percutaneous coronary intervention (PCI). This study sought to identify predictors of in-hospital mortality in STEMI patients after PCI.

**Methods:** Patients with acute STEMI, who underwent emergency PCI at Hebei General Hospital, Baoding First Central Hospital, and Cangzhou Central Hospital, from January 2016 to December 2018, were retrospectively included in this study. The patients' general data, previous medical history, clinical data and medication data were collected and compared between the survival and mortality groups. The primary outcome was in-hospital mortality. In-hospital mortality was defined as all-cause death during admission.

**Results:** Of the 1,169 patients (876 male and 293 female) enrolled in this study, 95 (8.13%) died during hospitalization. The multivariate logistic regression analysis showed that being female [odds ratio (OR) =5.86, 95% confidence interval (CI): 2.03–16.92, P=0.001], a Killip class of 2 (OR =8.13, 95% CI: 2.03–32.61, P=0.003), a Killip class of 4 (OR =17.31, 95% CI: 3.69–81.27, P=0.001), a left main coronary artery lesion (OR =44.25, 95% CI: 3.96–494.05, P=0.002), a final TIMI flow of 1 (OR =171.83, 95% CI: 28.46–1037.51, P=0.001), a final TIMI flow of 2 (OR =72.93, 95% CI: 38.54–138.00, P=0.001), symptom onset-to-door time (SDT) (OR =1.01, 95% CI: 1.00–1.02, P=0.001), symptom onset-to-balloon dilatation time (SBT) (OR =1.01, 95% CI: 1.00–1.02, P=0.001), and the Synergy Between PCI With Taxus and CABG (SYNTAX) score (OR =1.07, 95% CI: 1.01–1.12, P=0.019) were risk factors; while postoperative  $\beta$ -receptor blockers (OR =0.10, 95% CI: 0.03–0.30, P=0.001) postoperative angiotensin-converting enzymes/angiotensin receptor blockers (OR =0.13, 95% CI: 0.04–0.44, P=0.001), BMI (OR =0.85, 95% CI: 0.74–0.98, P=0.024), the percentage of the ejection fraction (OR =0.81, 95% CI: 0.75–0.86, P=0.001), and low-density lipoprotein cholesterol (OR =0.44, 95% CI: 0.21–0.91, P=0.027) were protective factors for in-hospital mortality.

**Conclusions:** Female, Killip grade, a left main lesion, TIMI grade, SDT, SBT, and SYNTAX score were associated with a higher risk of in-hospital death. Conversely, BMI, ejection fraction, LDL-C level, and postoperative use of  $\beta$ -blocker and ACEI/ARB drugs were associated with a lower in-hospital death risk.

**Keywords:** Percutaneous coronary intervention (PCI); ST-segment elevation myocardial infarction (STEMI); hospital mortality; prognosis

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## Introduction

Acute myocardial infarction (AMI) is the main cause of death in patients with cardiovascular diseases (1,2). AMI consists of two types, that is, ST-segment elevation myocardial infarction (STEMI) and non ST-segment elevation myocardial infarction (NSTEMI). Nearly 40% of AMI cases arise from ST-segment elevation myocardial infarction (STEMI) (3), which results from the occlusion of 1 or more of the coronary arteries that supply the heart with blood.

Percutaneous coronary intervention (PCI) is the 1st-line treatment strategy for patients with acute STEMI, and PCI improves the prognosis of AMI patients (4-7). PCI reduces mortality in acute STEMI patients by about 22% compared to fibrinolysis (8); however, the mortality risk remains high for many patients in the hospital.

One factor that increases patient mortality is the no-reflow phenomenon, which occurs when there is inadequate myocardial perfusion of the coronary artery without evidence of angiographic epicardial vessel obstruction, spasm, or dissection (9-11). Studies suggest that many factors may influence the risk of mortality after PCI, including age, Killip class, symptom onset to balloon time [based on a spontaneous breathing trial (SBT)], baseline thrombolysis in myocardial infarction (TIMI) flow, lesion length, reference vessel diameter, cutoff occlusion pattern, thrombus burden, SYNTAX score, mean platelet volume, platelet-lymphocyte ratio, increased creatinine, and elevated creatine kinase (4-7). During coronavirus disease 2019 (COVID-19) pandemic, a high GRACE score and COVID-19 were independent risk factors associated with higher in-hospital mortality for ST-segment elevation AMI and non-ST-segment elevation AMI (12). In relation to patient mortality, the question of whether these specific clinical features or others have predictive value in the prognosis of patients with acute STEMI after emergency PCI requires further investigation.

This study aimed to identify predictors of in-hospital mortality in STEMI patients after emergency PCI.

We present the following article in accordance with the STROBE reporting checklist (available at <https://dx.doi.org/10.21037/apm-21-2722>).

## Methods

### Patients

This study was a retrospective, multicenter, observational study. Patients with STEMI, who underwent emergency

PCI at Hebei General Hospital, Baoding No. 1 Central Hospital, and Cangzhou Central Hospital, from January 2016 to December 2018 were included in the study. To be eligible for inclusion in the study, patients had to meet the following inclusion criterion: have a diagnosis of acute STEMI with indications for PCI as per the current guidelines (13). Patients with acute STEMI who only underwent emergency coronary angiography but did not undergo coronary intervention were excluded from the study. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional ethics research committee board of Baoding No. 1 Central Hospital (No. [2021]014). Individual consent for this retrospective analysis was waived.

### Emergency PCI procedures

The procedures and protocols for emergency PCI were conducted according to the current guidelines (14). Patients with hemodynamic instability were treated with an intra-aortic balloon pump (IABP) to increase the blood flow of the coronary artery and cerebral artery, and patients with pulmonary edema were treated with a non-invasive ventilator. All patients with acute STEMI were given 3,000 units of unfractionated heparin intravenously and 600 mg of clopidogrel (Sanofi, Paris, France) before the procedure, followed by a long-term oral dose of 75 mg of clopidogrel per day for at least 12 months, or 180 mg of ticagrelor (AstraZeneca Pharmaceutical Co. Ltd, Sweden) followed by a maintenance dose of 90 mg twice a day for at least 12 months. All patients were given 300 mg of aspirin (Bayer Healthcare Co. Ltd, Germany) to chew before the procedure, and then received 100 mg of aspirin per day.

### Data collection

The collected patient data included data on age, gender, body mass index (BMI), history of smoking, history of alcohol consumption, family history of coronary heart disease, personal history of angina pectoris, hypertension, type 2 diabetes, myocardial infarction, cerebral infarction, chronic kidney disease, coronary intervention, atrial fibrillation and cerebral hemorrhage, heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), Killip grade, blood results, including white blood cell count (WBC), red blood cell count, hemoglobin, platelet count, serum potassium, serum sodium, serum chlorine, serum

creatinine, uric acid, cholesterol, triglyceride, low-density lipoprotein (LDL), high-density lipoprotein, random blood glucose, creatine kinase myocardial band (CK-MB) peak value, left ventricular ejection fraction (LVEF), chest pain at time from onset to first medical contact, time from onset to balloon dilatation, intervention information related to culprit blood vessels, lesion site, lesion diameter, lesion length, number of lesion blood vessels, the treatment of non-culprit blood vessels, collateral circulation, syntax score, thrombus score, thrombus aspiration, postoperative TIMI blood flow classification, IABP application, intraoperative slow blood flow, intraoperative ventricular fibrillation, central packing,  $\beta$ -blocker use, angiotensin-converting enzyme/angiotensin receptor blocker (ACEI/ARB) use, aldosterone use, diuretic use, and nicorandil use after admission. Commonly used drugs included angiotensin-converting enzyme inhibitors, statins,  $\beta$ -blockers, nitrates, and diuretics. For patients with AMI with a heavy intravascular thrombus load, a thrombus aspiration catheter was used to treat the thrombus (15).

A clinical evaluation was conducted after the first medical contact when the patient was admitted to hospital to determine each patient's Killip grade. Killip grade I was defined as AMI patients without clinical symptoms of heart failure; Killip grade II was defined as AMI complicated with left heart failure, and a wet rale of both lungs <50% in the lung field; Killip grade III was defined as AMI with acute pulmonary edema, large, small, dry and wet rales of the whole lung; Killip grade IV was defined as hemodynamic changes of different degrees or stages such as AMI complicated with cardiogenic shock (16). Cardiogenic shock was defined as per the Kamir-NIH study; that is, as >30 min of continuous SBP <90 mmHg, and was characterized by terminal organ hypoperfusion (17).

Left ventricular function was evaluated by transthoracic echocardiography for the 1st time after STEMI was diagnosed. The patient was placed in a supine position for the echocardiography. As per the cutting-edge method of the American Society of Echocardiography, the internal dimensions of the left ventricle (i.e., end-systolic diameter and end-diastolic diameter) were measured using at least 3 consecutive cardiac cycles. LVEF was calculated as  $LVEF = (\text{end-diastolic volume} - \text{end-systolic volume}) / \text{end-diastolic volume}$ . Left ventricular end-diastolic diameter and left ventricular end-systolic diameter were calculated using the Simpson method under 2-dimensional echocardiography (18).

The SYNTAX score, which is a scoring system based on

the degree and scope of the coronary artery disease after coronary angiography, was used to assess cardiac disease grade (19).

The thrombus score was used to evaluate the degree of a thrombotic lesion. The thrombus score was assessed after the guidewire passed through the lesion (but before balloon dilation). The thrombus score was assigned using the following scale: 0 points if there was no thrombus under multiple, persistent cardiac cycles, and the intimal dissection caused by the guidewire in the false lumen was excluded; 1 point if there was a fuzzy thrombus shadow; 2 points if the length of the confirmed thrombus image was <1/2 of the inner diameter of the blood vessel; 3 points if there was a determined thrombus, the length of which was 1/2–2 times greater than the inner diameter of the blood vessel; 4 points if there was a determined thrombus, the length of which was 2 times greater than the inner diameter of the blood vessel; and 5 points if blood vessel occlusion meant that the thrombus could not be evaluated (20).

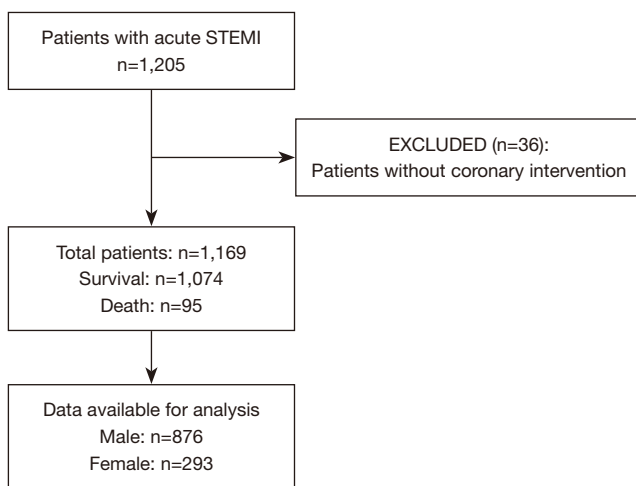
TIMI was used for blood flow classification. TIMI blood flow classification was evaluated after coronary stent implantation and non-compliant balloon dilatation. TIMI grade 0 blood flow (no perfusion) was defined as no forward blood flow after coronary artery occlusion. TIMI grade 1 blood flow (no perfusion penetration) was defined as weak anterior coronary blood flow after occlusion, and the distal coronary bed not being completely filled. TIMI grade 2 blood flow (partial reperfusion) was defined as delayed or slow forward blood flow that completely filled the distal region. TIMI grade 3 blood flow (complete perfusion) referred to normal blood flow that completely filled the distal coronary bed (21).

### *Observation outcome*

The primary outcome was in-hospital mortality. In-hospital mortality referred to all-cause death during admission.

### *Statistical analysis*

All data were processed by SPSS 21.0 (IBM, Armonk, NY, USA). The independent sample *t*-, chi-square, and Mann-Whitney U-tests were used as applicable to assess variant differences between the mortality and survival groups. A single-sample Kolmogorov-Smirnov test was used for the normality test. Continuous variables of normal distribution are expressed by mean  $\pm$  standard deviation, while continuous variables of non-normal distribution are



**Figure 1** The flow diagram of the study.

expressed as median (interquartile range). The classification variable is represented by the n (%). The missing data were replaced by the median. Univariable logistic regression analyses were used to screen out factors with a P value <0.20. The backward step-by-step iteration method was used for the multivariable binary logistic regression analysis to finally determine the risk factors related to hospital death. Odds ratios (ORs) were calculated with 95% confidence intervals (CIs). A P value <0.05 was considered statistically significant.

## Results

### Baseline characteristics

A total of 1,205 patients with acute STEMI met the inclusion criteria. Of these patients, 36 without coronary intervention, were excluded. Ultimately, 1,169 patients (876 male and 293 female) with acute STEMI, who underwent emergency PCI were included in this study as observation subjects. After the emergency PCI, a total of 1,074 patients survived during hospitalization, and 95 died during hospitalization (aged 59.6±11.4 years and 66.3±13.3 years, respectively,  $P<0.001$ ). *Figure 1* shows a flow diagram of the study. The specific clinical features of all subjects are shown in *Table 1*.

Most patients survived after undergoing emergency PCI (1,074/1,169, 91.2%), but the proportion of females in the survival group was significantly lower than that in the mortality group (24.0% vs. 36.8%,  $P=0.006$ ). Patients' mean SBP and DBP were higher in the survival group than the

mortality group ( $P<0.001$ ). Patients in the survival group were more likely to have a history of smoking (48.2% vs. 36.8%,  $P=0.033$ ), but less likely to have a history of cerebral infarction (14.3% vs. 22.1%,  $P=0.042$ ), and atrial fibrillation (1.6% vs. 5.3%,  $P=0.011$ ) than those in the mortality group. Patients' WBC was lower in the survival group than the mortality group ( $10.64\pm 3.38$  vs.  $12.45\pm 5.11$ ,  $P<0.001$ ), and LDL cholesterol was higher in the survival group than the mortality group ( $2.98\pm 0.85$  vs.  $2.67\pm 0.70$ ,  $P<0.001$ ).

At admission, fewer survival patients had a Killip class  $\geq 2$  class ( $P<0.001$ ), left main coronary artery ( $P<0.001$ ), or left anterior descending coronary artery (LAD,  $P=0.01$ ) infarction than mortality patients. Survival patients had more mid and distal culprit artery occlusion than mortality patients (35.5% vs. 28.4%, 13.9% vs. 6.4%, respectively,  $P<0.001$ ), and their distribution of thrombus score fell between 0–3 more often ( $P=0.005$ ) than that of the mortality patients. LVEF, symptom onset-to-door time (SDT), SBT (i.e., the time of the crossing of the wire), and IABP application were lower in the survival group than the mortality group ( $P<0.001$ ). After coronary angiography, the survival patients had lower SYNTAX scores than mortality patients ( $20.71\pm 7.67$  vs.  $29.31\pm 9.92$ ,  $P=0.003$ ). A final TIMI flow of 3 was evident in more patients in the survival group than the mortality group (94.4% vs. 60.0%,  $P<0.001$ ) after coronary stent implantation and non-compliant balloon dilatation. However, patients in the survival group were administered more  $\beta$ -blockers and ACEI/ARBs after admission ( $P<0.001$ ) than those in the mortality group.

### Risk factors for in-hospital mortality after emergency PCI for patients with acute STEMI

The risk factors for in-hospital mortality are shown in *Table 2*. The multivariate logistic regression analysis showed that being female (OR =5.86, 95% CI: 2.03–16.92,  $P=0.001$ ), a Killip class of 2 (OR =8.13, 95% CI: 2.03–32.61,  $P=0.003$ ), a Killip class of 4 (OR =17.31, 95% CI: 3.69–81.27,  $P=0.001$ ), a left main coronary artery lesion (OR =44.25, 95% CI: 3.96–494.05,  $P=0.002$ ), a final TIMI flow of 1 (OR =171.83, 95% CI: 28.46–1037.51,  $P=0.001$ ), a final TIMI flow of 2 (OR =72.93, 95% CI: 38.54–138.00,  $P=0.001$ ), SDT (OR =1.01, 95% CI: 1.00–1.02,  $P=0.001$ ), SBT (OR =1.01, 95% CI: 1.00–1.02,  $P=0.001$ ), and SYNTAX score (OR =1.07, 95% CI: 1.01–1.12,  $P=0.019$ ) were risk factors for in-hospital mortality. However, the postoperative application of  $\beta$ -receptor blockers (OR =0.10, 95% CI: 0.03–0.30,  $P=0.001$ ) and ACEI/ARB (OR =0.13,

**Table 1** Baseline characteristics

Characteristics	Total (n=1,169)	Survival (n=1,074)	Mortality (n=95)	P
<b>Demography</b>				
Age (years)	1,169	59.6±11.4	66.3±13.3	<0.001
Sex (%)				0.006
Male	876	816 (76.0)	60 (63.2)	
Female	293	258 (24.0)	35 (36.8)	
BMI (kg/m <sup>2</sup> )	1,169	25.50±3.25	24.99±3.93	0.115
History of smoking	553	518 (48.2)	35 (36.8)	0.033
History of drinking	319	297 (27.7)	22 (23.2)	0.346
History of diabetes mellitus	240	214 (19.9)	26 (27.4)	0.085
RBG (mmol/L)	1,169	8.06±2.83	9.25±3.51	0.056
History of hypertension	567	516 (48.0)	51 (53.7)	0.292
SBP (mmHg)	1,169	129.74±22.48	106.44±28.53	<0.001
DBP (mmHg)	1,169	80.25±13.93	66.28±18.58	<0.001
Heart rate (bpm)	1,169	75.60±15.35	76.08±22.12	0.776
History of chronic kidney disease	13	11 (1.0)	2 (2.1)	0.336
Family history for CAD	113	108 (10.1)	5 (5.3)	0.130
History of angina	523	474 (44.1)	49 (51.6)	0.162
History of cerebral infarction	175	154 (14.3)	21 (22.1)	0.042
Prior myocardial infarction	77	70 (6.5)	7 (7.4)	0.749
History of coronary intervention	74	66 (6.1)	8 (8.4)	0.383
History of atrial fibrillation	22	17 (1.6)	5 (5.3)	0.011
History of cerebral hemorrhage	37	33 (3.1)	4 (4.2)	0.544
<b>Serum biochemical indicators</b>				
WBC (10 <sup>9</sup> /L)	1,169	10.64±3.38	12.45±5.11	<0.001
RBC (10 <sup>12</sup> /L)	1,169	4.52±0.56	4.46±0.66	0.367
Hemoglobin (g/L)	1,169	139.65±17.30	136.20±18.28	0.24
Platelet count (10 <sup>9</sup> /L)	1,169	226.98±60.31	237.24±90.48	0.258
Serum potassium concentration (mmol/L)	1,169	3.99±0.48	4.11±0.51	0.023
Serum sodium concentration (mmol/L)	1,169	138.85±3.25	138.79±3.61	0.712
Serum chloride concentration (mmol/L)	1,169	102.73±4.67	102.13±8.07	0.294
Creatinine (μmol/L)	1,169	73.20±21.11	84.49±41.55	0.023
Uric acid (μmol/L)	1,169	312.20±87.88	342.48±114.83	0.012
Total cholesterol (mmol/L)	1,169	4.56±1.00	4.42±1.14	0.312
Triglyceride (mmol/L)	1,169	1.66±1.06	1.52±0.53	0.687
HDL cholesterol (mmol/L)	1,169	1.03 ± 0.26	1.03±0.25	0.99
LDL cholesterol (mmol/L)	1,169	2.98±0.85	2.67±0.70	<0.001
CK-MB (ng/mL)	1,169	168.57±71.58	241.12±128.89	0.056

**Table 1** (continued)

Table 1 (continued)

Characteristics	Total (n=1,169)	Survival (n=1,074)	Mortality (n=95)	P
Vascular lesions				
Lesion diameter (mm)	1,169	3.04±0.38	3.10±0.52	0.440
Lesion length (mm)	1,169	28.87±10.55	28.55±11.29	0.543
Killip class				<0.001
1	974	939 (87.4)	35 (36.9)	
2	124	110 (10.2)	14 (14.7)	
3	26	18 (1.7)	8 (8.4)	
4	45	7 (0.7)	38 (40.0)	
Infarct-related artery				
Left main coronary artery	10	3 (0.3)	7 (7.4)	<0.001
LAD	553	496 (46.2)	57 (60.0)	0.01
LCX	139	130 (12.1)	9 (9.5)	0.448
RCA	467	445 (41.4)	22 (23.1)	0.004
Number of coronary artery lesions				0.08
Single vessel	380	354 (32.9)	26 (27.4)	
Double vessel	397	368 (34.3)	29 (30.5)	
Triple vessel	392	352 (32.8)	40 (42.1)	
Occlusion site culprit artery				
Ostial	21	13 (1.2)	8 (8.4)	<0.001
Proximal	585	531 (49.4)	54 (56.8)	
Mid	408	381 (35.5)	27 (28.4)	
Distal	155	149 (13.9)	6 (6.4)	
Collateral circulation	71	66 (6.1)	5 (5.3)	0.73
Non-culprit blood vessel treatment	53	47 (4.4)	6 (6.3)	0.384
Thrombus score				
0	6	6 (0.6)	0 (0)	0.005
1	15	15 (1.4)	0 (0)	
2	98	96 (8.9)	2 (2.1)	
3	479	450 (41.9)	29 (30.5)	
4	392	348 (32.4)	44 (46.3)	
5	179	159 (14.8)	20 (21.1)	
Thrombus aspiration	394	369 (34.4)	25 (26.3)	0.112
SYNTAX score	1,169	20.71±7.67	29.31±9.92	0.003
EF (%)	1,169	54.64±8.05	47.17±8.52	<0.001
SDT (minutes)	1,169	104.21±69.01	280.40±162.94	<0.001
SBT (minutes)	1,169	247.53±104.41	470.32±201.37	<0.001
IABP	32	14 (1.3)	18 (18.9)	<0.001

Table 1 (continued)

Table 1 (continued)

Characteristics	Total (n=1,169)	Survival (n=1,074)	Mortality (n=95)	P
Intraoperative slow reflow phenomenon	127	86 (8.0)	41 (43.2)	<0.001
Intraoperative ventricular fibrillation	43	38 (3.5)	5 (5.3)	0.392
Intraoperative acute cardiac tamponade	6	2 (0.2)	4 (4.2)	<0.001
Final TIMI flow				<0.001
0	17	1 (0.1)	16 (16.9)	
1	12	2 (0.2)	10 (10.5)	
2	69	57 (5.3)	12 (12.6)	
3	1,071	1,014 (94.4)	57 (60.0)	
Mechanical complication	1,169	0 (0)	1 (1.05)	0.441
Medication after admission				
β-blocker	841	809 (75.3)	32 (33.7)	<0.001
ACEI/ARB	664	644 (60.0)	20 (21.1)	<0.001
Spironolactone	450	417 (38.8)	33 (34.7)	0.432
Diuretic	265	241 (22.4)	24 (25.3)	0.529
Nicorandil	144	137 (12.8)	7 (7.4)	0.126

BMI, body mass index; RBG, random plasma glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure; CAD, coronary artery disease; WBC, white blood cell; RBC, red blood cell; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CK-MB, creatine kinase-MB; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; EF, ejection fraction; SDT, symptom to door time; SBT, symptom to balloon time; IABP, intra-aortic balloon pump; TIMI, thrombolysis in myocardial infarction; ACEI/ARB, angiotensin-converting enzyme/angiotensin receptor blocker.

95% CI: 0.04–0.44,  $P=0.001$ ), BMI (OR =0.85, 95% CI: 0.74–0.98,  $P=0.024$ ), the percentage of ejection fraction (OR =0.81, 95% CI: 0.75–0.86,  $P=0.001$ ), and LDL-C (OR =0.44, 95% CI: 0.21–0.91,  $P=0.027$ ) were protective factors for in-hospital mortality.

## Discussion

In the research, there were 95 (8.13%) patients died during hospitalization. Of those, 42 patients died of cardiogenic shock, 32 patients died of malignant arrhythmias, 22 patients died of mechanical complications and 8 patients had severe bleeding events.

This study examined the short-term outcomes of a cohort of STEMI patients who underwent PCI to identify risk factors for in-hospital mortality. The results suggest that female gender, a Killip class of 2 or 4, a left main lesion, a TIMI flow of 1 or 2, longer SDT, longer SBT, and a higher SYNTAX score were all risk factors for hospital death after emergency PCI in STEMI patients, while the use of β-blockers and ACEI/ARB drugs after the procedure, a high

BMI, LVEF, and high LDL-C were protective factors.

Stehli *et al.* (22) and Lee *et al.* (17) showed that gender is an independent risk factor for the short- and long-term mortality of patients with AMI. Notably, the risk of death in female patients is significantly higher than in male patients. Thus, analyses of both male and female AMI patients might lead to the omission or inaccuracy of some risk factors. This study examined the 2 genders separately to further identify the factors associated with in-hospital death after emergency PCI in patients with acute ST-elevation myocardial infarction. The results showed that males and females had different risk factors for in-hospital death after emergency PCI for acute ST-elevation myocardial infarction. A factor that increases patient mortality is the no-reflow phenomenon (9–11), and its association with mortality was observed only in females.

With the establishment of chest pain centers, the rate of STEMI patients receiving early PCI has increased significantly, thus reducing the mortality rate. We found that both SDT and SBT were predictors of mortality. However, some registered data from home and abroad show

**Table 2** Analyses of variables associated with in-hospital mortality

Variable	Univariable analysis			Multivariable analysis		
	OR	95% CI	P	OR	95% CI	P
Sex						
Male (reference)						
Female	1.85	1.19–2.86	0.006	5.86	2.03–16.92	0.001
BMI (kg/m <sup>2</sup> )	0.95	0.89–1.02	0.15	0.85	0.74–0.98	0.024
SDT (minutes)	1.015	1.01–1.02	<0.001	1.01	1.00–1.02	0.001
SBT (minutes)	1.012	1.01–1.014	<0.001	1.01	1.00–1.02	0.001
WBC (10 <sup>9</sup> /L)	1.12	1.07–1.18	<0.001	1.12	1.00–1.26	0.053
LDL cholesterol (mmol/L)	0.61	0.46–0.80	<0.001	0.44	0.21–0.91	0.027
Killip						
1 (reference)						
2	3.42	1.78–6.54	<0.001	8.13	2.03–32.61	0.003
3	11.92	4.86–29.29	<0.001	5.51	0.90–33.56	0.064
4	145.64	60.78–348.99	<0.001	17.31	3.69–81.27	0.001
Left main coronary artery	28.39	7.22–111.73	<0.001	44.25	3.96–494.05	0.002
Final TIMI flow						
0	284.63	37.09–2184.63	<0.001	171.83	28.46–1037.51	0.001
1	88.94	19.04–415.50	<0.001	72.93	38.54–138.00	0.001
2	3.75	1.90–7.37	<0.001	1.01	0.22–4.78	0.986
3 (reference)						
Medication after admission						
β-Blocker	0.17	0.11–0.26	<0.001	0.10	0.03–0.30	0.001
ACEI/ARB	0.18	0.10–0.29	<0.001	0.13	0.04–0.44	0.001
SYNTAX score	1.12	1.09–1.45	<0.001	1.07	1.01–1.12	0.019
CK-MB	1.04	0.94–1.15	0.056	1.01	1.00–1.02	0.043
EF (%)	0.89	0.86–0.92	<0.001	0.81	0.75–0.86	0.001

BMI, body mass index; SDT, symptom to door time; SBT, symptom to balloon time; LDL, low-density lipoprotein; CK-MB, creatine kinase-MB; EF, ejection fraction; TIMI, thrombolysis in myocardial infarction; ACEI/ARB, angiotensin-converting enzyme/angiotensin receptor blocker.

that STEMI patients still have a high risk of death (23,24). In patients with a symptom duration >120 min, myocardial perfusion was found to have no significant benefit, and door to balloon time (DBT) in patients with SBT >90 min had little effect on mortality (25). This study found that as the time from onset to first medical contact increased, the risk of death in hospital also gradually increased; the risk of death increased by 1% for every 1-minute extension.

Similar to the results of Dudek *et al.* (26), we found that all-cause mortality within 1 year in the SBT group >3 hours was higher than that in the SBT group <3 hours. Thus, the prolonged opening time of mechanical reperfusion is an important factor affecting the prognosis of STEMI patients. This may be due to the prolongation of total ischemia time, which leads to increased myocardial cell necrosis, decreased viable myocardial cells, and the deterioration of cardiac



function. It is important to seek medical attention when suspicious symptoms occur so that the onset to first medical contact time can be shortened.

In our study subjects, we also found that the risk of in-hospital death in female patients with AMI undergoing emergency PCI was 5.86 times higher than that in male patients. These results are similar to those of previous clinical observation studies, such as those of Tsai *et al.* (6). In another observational study on gender differences (22), it was also observed that the mortality rate of female patients with AMI within 30 days was significantly higher than that of male patients. Researchers have speculated that this increased risk may be due to atypical chest pain symptoms in female patients, which results in female patients having a significantly longer time from onset to first medical contact than male patients.

Recently, Kim *et al.* (27) confirmed that even after emergency PCI, 14% of patients with acute STEMI have left ventricular dysfunction, which is a well-known factor for mortality after AMI. As expected, we found that as LVEF increased, there was a downward trend in the risk of death for in-hospital patients. TIMI and SYNTAX are also well-known factors for mortality (19,28).

In relation to obese patients, the traditional view has always been that obesity is a risk factor for STEMI. However, similar to the results of Kim *et al.* (29), we found that a high BMI was a protective factor for the risk of in-hospital death among STEMI patients (i.e., the higher the BMI, the lower the risk of in-hospital death among STEMI patients). Another Chinese study found the same results through observations and a study of 35,964 AMI patients. Increased BMI has been found to be independently related to decreased in-hospital mortality (30). This BMI paradox has also been observed in previous studies (31-33). The present study was not designed to explore the reasons underlying this paradox; however, previous studies have hypothesized that overweight/obese people have higher metabolic reserves, cachexia is rare in overweight/obese people, overweight/obesity is more common young patients, and high-weight people have larger surface distribution volumes and better drug tolerance for the same dose (31-33). Future studies should seek to examine this paradox more closely.

In this study, high LDL-C was a protective factor, which is counterintuitive given the role of LDL-C levels in the development of AMI (4-7). This LDL-C paradox has been reported in many studies (34-37). Sia *et al.* (34) reported that high LDL-C was protective in STEMI, while low HDL-C was protective in STEMI. Bambat *et al.* (35)

reported that high LDL-C was protective in patients with acute coronary syndromes. This paradox was observed in a registry study of 115,492 AMI patients (36). Wang *et al.* (37) speculated that the LDL-C paradox may be mediated by malnutrition; however, nutrition could not be analyzed in the present study. As for the BMI paradox, the present study was not designed to determine the reasons for this paradox; thus, further studies need to be conducted to examine this issue. Although obesity and LDL are risk factors for acute myocardial infarction, once an acute myocardial infarction occurs, it is essential for patients to strengthen nutrition, so as to get them through the critical period.

This study had some limitations. It was a retrospective multicenter study; thus, selectivity bias may have inevitably been included in the results. As the exact cause of death was often unknown, overall survival had to be used instead of cardiac death as the outcome. Further accurate, randomized, and controlled studies may lead to more effective conclusions.

## Conclusions

In conclusion, female gender, Killip grade, a left main lesion, TIMI blood flow grade, time from onset to first medical contact, time from onset to balloon dilatation, and SYNTAX score were associated with a higher risk of hospital death. Conversely, BMI, ejection fraction, LDL-C level, and postoperative use of  $\beta$ -blocker and ACEI/ARB drugs were associated with a lower in-hospital death risk.

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aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional ethics research committee board of Baoding No. 1 Central Hospital (No. [2021]014). Individual consent for this retrospective analysis was waived.

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