



# Risk factors for cardiac rupture after acute ST-segment elevation myocardial infarction during the percutaneous coronary intervention era: a retrospective case-control study

Zesheng Xu<sup>1</sup>, Yingkai Li<sup>1,2</sup>, Ruyan Zhang<sup>3</sup>, Yongqing Liu<sup>1</sup>, Hua Liu<sup>1</sup>, Jiancai Yu<sup>1</sup>, Xianbo Zhou<sup>1</sup>, Yihui Du<sup>1</sup>, Hongliang Cong<sup>3</sup>

<sup>1</sup>Department of Cardiology, Cangzhou Central Hospital, Tianjin Medical University Teaching Hospital, Cangzhou, China; <sup>2</sup>Department of Cardiology, Beijing Anzhen Hospital, Capital Medical University, Beijing, China; <sup>3</sup>Department of Cardiology, Tianjin Chest Hospital, Tianjin, China  
*Contributions:* (I) Conception and design: Z Xu; (II) Administrative support: Z Xu, H Cong; (III) Provision of study materials or patients: Y Li, R Zhang; (IV) Collection and assembly of data: Y Li, R Zhang, Y Liu, J Yu; (V) Data analysis and interpretation: Y Li, R Zhang, X Zhou, Y Du; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

*Correspondence to:* Dr. Hongliang Cong. Tianjin Chest Hospital, 261 Taierzhuang Road, Tianjin 300222, China. Email: hongliangcong@126.com.

**Background:** In percutaneous coronary intervention (PCI) era, more clinically valuable risk factors are still needed to determine the occurrence of cardiac rupture (CR). Therefore, we aimed to provide evidence for the early identification of CR in ST-segment elevation myocardial infarction (STEMI).

**Methods:** A total of 22,016 consecutive patients with STEMI admitted to Cangzhou Central Hospital and Tianjin Chest Hospital from January 2013 to July 2021 were retrospectively included, among which 195 patients with CR were included as CR group. From the rest 21,820 STEMI patients without CR, 390 patients at a ratio of 1:2 were included as the control group. A total of 66 patients accepted PCI in the CR group, and 132 patients who accepted PCI in the control group at a ratio of 1:2 were included. The status of first medical contact, laboratory examinations, and PCI characteristics were recorded. Multivariate logistic regression analysis was used to investigate the risk factors related to CR.

**Results:** There was a higher proportion of patients with myocardial infarction (MI) in the high lateral wall in the CR group (23.6% vs. 8.2%,  $P < 0.001$ ). The proportion of single lesions was lower in the CR group (24.2% vs. 45.5%,  $P = 0.004$ ). Female (OR = 2.318, 95% CI: 1.431–3.754,  $P = 0.001$ ), age (OR = 1.066, 95% CI: 1.041–1.093,  $P < 0.001$ ), smoking (OR = 1.750, 95% CI: 1.086–2.820,  $P = 0.022$ ), total chest pain time (OR = 1.017, 95% CI: 1.000–1.035,  $P = 0.049$ ), recurrent acute chest pain (OR = 2.750, 95% CI: 1.535–4.927,  $P = 0.001$ ), acute myocardial infarction (AMI) in the high lateral wall indicated by ECG (OR = 5.527, 95% CI: 2.798–10.918,  $P < 0.001$ ), acute heart failure (OR = 3.585, 95% CI: 2.074–6.195,  $P < 0.001$ ), and NT-proBNP level (OR = 1.000, 95% CI: 1.000–1.000,  $P = 0.023$ ) were risk factors for CR in all patients. In patients who accepted PCI, single lesion (OR = 0.421, 95% CI: 0.204–0.867,  $P = 0.019$ ), preoperative thrombolysis in myocardial infarction (TIMI) grade (OR = 0.358, 95% CI: 0.169–0.760,  $P = 0.007$ ), and postoperative TIMI grade (OR = 0.222, 95% CI: 0.090–0.546,  $P = 0.001$ ) were risk factors for CR.

**Conclusions:** Non-single lesions and preoperative and postoperative TIMI grades were risk factors for CR in patients who accepted PCI. In addition to previously reported indicators, we found that AMI in the high lateral wall maybe helpful in early and accurate identification and prevention of possible CR.

**Keywords:** Cardiac rupture (CR); ST-segment elevation myocardial infarction; acute myocardial infarction in high lateral wall; thrombolysis in myocardial infarction grades; percutaneous coronary intervention

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## Introduction

Cardiac rupture (CR) is a serious mechanical complication after acute myocardial infarction (AMI) which includes free wall rupture (FWR), ventricle septal rupture (VSR), and papillary muscle rupture (PMR). Once CR occurs in an AMI patient, it is difficult to treat and the fatality rate is extremely high. The previous incidence of CR was 2% to 6% (1). Although thrombolytic therapy and direct percutaneous coronary intervention (PCI) has been shown to reduce the incidence of CR, and the incidence of CR after AMI was further reduced (0.7–1.1%) (2), the occurrence of CR after ST-segment elevation myocardial infarction (STEMI) is still catastrophic.

Surgery, despite having a low success rate and high risk of death, is the most effective way to treat CR, though most patients do not have the opportunity for surgery. Therefore, the early and accurate identification and prevention of possible CR is of great importance. Previous studies found older age, female, lower LVEF, hyperglycemia, and thrombocytopenia were risk factors for CR after AMI (3–6). These factors were lack of specificity, which often occur in other diseases. Therefore, our study aims to find some risk factors for CR with high clinical specificity. In this study, we aimed to analyze the influencing factors of CR in STEMI and to provide evidence for the early and rapid identification of CR. We present the following article in accordance with the STROBE reporting checklist (available at <https://jtd.amegroups.com/article/view/10.21037/jtd-22-394/rc>).

## Methods

### *Study design and patients*

In this case-control study, a total of 22,016 consecutive patients with STEMI admitted to Cangzhou Central Hospital and Tianjin Chest Hospital from January 2013 to July 2021 were retrospectively included, among which 195 patients with CR were included as CR group. From the rest 21,820 STEMI patients without CR, 390 patients at a ratio of 1:2 (7) were included as the control group. A total of 66 patients accepted PCI in the CR group, and 132 patients who accepted PCI in the control group were included at a ratio of 1:2 (*Figure 1*). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of Tianjin Chest Hospital (No. 2021LW015) and Cangzhou Central Hospital (No. 2021-187-01). Signed

informed consent was obtained from the family member of each patient.

The inclusion criteria were as follows: (I) patients who met the diagnosis of STEMI (8) according to the latest fourth edition of the Global Myocardial Infarction Definition criteria; (II) patients with CR including FWR, VSR, and PMR confirmed by ultrasound or autopsy (CR group), and (III) patients without CR after STEMI during the same time period (control group). The exclusion criteria were patients with severe kidney and liver dysfunction and severe coagulation dysfunction before admission.

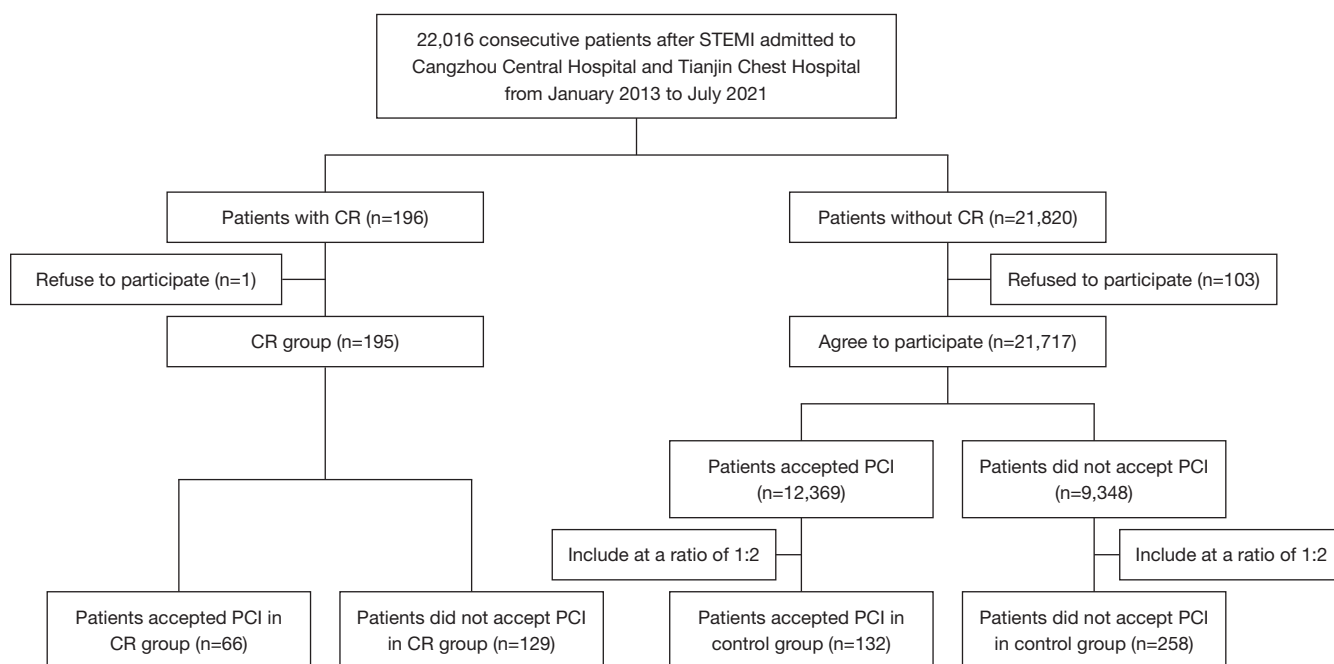
### *Clinical data collection*

Apart from the clinical data, chest pain onset time, and total chest pain time were collected. Laboratory and clinical examination results were collected including white blood cell (WBC) count, hemoglobin, platelet count, creatine kinase (CK), creatine kinase isoenzyme (CK-MB), electrocardiogram (ECG), and echocardiography results on admission. The vital signs, treatment strategies, and clinical symptoms of patients after admission were recorded. For patients who underwent PCI, the characteristics of PCI surgery, the lesion characteristics according to the patient's angiographic results, and the immediate surgical results after PCI were recorded.

### *Clinical treatment and PCI procedure*

Conservative medication, thrombolysis, and emergency coronary artery bypass graft (CABG) or PCI were determined according to the latest European Society of Cardiology (ESC) STEMI management guidelines (9) for all admitted patients. STEMI patients who underwent PCI were given 300 mg aspirin (Bayer HealthCare Manufacturing S.r.l.) and 300 mg clopidogrel bisulfate (Sanofi Winthrop Industrie) before surgery if there were no contraindications.

Coronary angiography and PCI were performed after local anesthesia with 2% lidocaine hydrochloride (Tianjin Biochemical Pharmacy, Tianjin, China). The transradial approach, transfemoral artery approach, or other access routes were determined by the surgeon. The interventional therapy for revascularization (PCI, thrombus aspiration, intracoronary thrombolysis) was adopted according to the patient's intraoperative condition. Complete revascularization or target vessel revascularization was determined according to the latest ESC STEMI management guidelines (9). Patients



**Figure 1** Flow diagram. PCI, percutaneous coronary intervention; CR, cardiac rupture.

were routinely given dual antiplatelet therapy (DAPT) after the operation. Other cardiovascular system drugs such as  $\beta$ -inhibitors and angiotensin-converting enzyme inhibitors (ACEIs) or other drugs were determined by the patient's clinical condition. A 90-minute accelerated infusion regimen of alteplase was adopted in patients treated with thrombolytic therapy, with 15 mg injected intravenously, followed by continuous intravenous drip (0.75 mg/kg, maximum 50 mg) within 30 minutes, and the remaining (0.5 mg/kg, maximum 35 mg) was given by continuous intravenous drip within 60 minutes (9).

### Definitions

Chest pain onset time was the time from the onset of chest pain to admission in hospital for the current myocardial infarction (MI). Total chest pain time was the total duration of chest pain symptoms for the current MI. The location of MI was determined by ST-segment elevation leads of ECG: 4 or more consecutive leads of ST-segment elevation in leads V1 to V6 indicated anterior wall MI; ST segment elevation in leads I and aVL indicated high lateral MI; ST segment elevation in leads II, III, and aVF and ST segment elevation in leads V3R–V5R indicated inferior wall MI and right ventricular MI; ST segment elevation in leads V1–V3

indicated anteroseptal MI. The blood perfusion grading after angiography was based on the thrombolysis in myocardial infarction (TIMI) blood flow grading standard (10).

### Statistical analysis

All analyses were performed using SPSS 21.0 software (SPSS, Inc., Chicago, IL, USA). Categorical variables were described using number (percentage) and were compared by the  $\chi^2$  test. For normally distributed data, quantitative variables are shown as mean  $\pm$  standard deviation (SD) and were compared by the t test. For variables of skewed distribution, quantitative variables are shown as median (Q1, Q3) and were compared by Wilcoxon rank-sum test. Potential risk factors for whole cannulation success were investigated first by univariate and multivariate logistic regression models. Multivariate logistic regression analysis was used to investigate the risk factors related to CR. Enter method was used for the selection of significant factors from the multiple logistic regression analysis. A two-tailed probability value of  $P < 0.05$  was considered as statistically significant.

### Results

A total of 22,016 patients with acute MI were admitted to

**Table 1** Baseline data in the 2 groups

Variables	CR group (n=195)	Control group (n=390)	P value
Female, n (%)	107 (54.9)	116 (29.7)	<0.001
Age	71.90±8.36	63.15±11.80	<0.001
Hypertension, n (%)	129 (66.2)	233 (59.7)	0.132
Diabetes mellitus, n (%)	55 (28.2)	116 (29.7)	0.700
Smoking, n (%)	78 (40)	133 (34.1)	0.161
Previous MI, n (%)	10 (5.1)	30 (7.7)	0.247
Previous PCI, n (%)	21 (10.8)	43 (11.0)	0.925
Previous CABG*, n (%)	0	6 (1.5)	0.186
Previous stroke, n (%)	49 (25.1)	73 (18.7)	0.072
Previous bleeding disorder*, n (%)	4 (2.1)	5 (1.3)	0.490
Previous thrombotic diseases*, n (%)	2 (1.0)	8 (2.1)	0.508

\*, Fisher's exact test was used. CR, cardiac rupture; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft.

Cangzhou Central Hospital and Tianjin Chest Hospital from January 2013 to July 2021. Among them, 196 cases of CR occurred in all patients, with an incidence rate of 0.89%.

### Baseline data

Age was significantly different between the 2 groups (71.90±8.36 *vs.* 63.15±11.80 years,  $P<0.001$ ). There was a higher proportion of female patients in the CR group (54.9%) than the control group (29.7%) ( $P<0.001$ ). There were no significant differences between the 2 groups of patients in terms of other data including hypertension, diabetes mellitus, previous MI, previous PCI, and previous CABG (Table 1).

### Status of first medical contact

Although there was no difference in the chest pain onset time [9 (4, 24) *vs.* 10 (4, 24),  $P=0.748$ ], the total chest pain time in the CR group was longer than in the control group [7 (3, 15) *vs.* 5 (2, 10),  $P<0.001$ ]. Acute heart failure occurred in more patients of the CR group (45.1% *vs.* 11.3%,  $P<0.001$ ). The CR group had more patients with cardiac shock (4.6% *vs.* 1.3%,  $P=0.019$ ). There were more patients with recurrent acute chest pain in the CR group (24.6% *vs.* 10.0%,  $P<0.001$ ). The CR group also had more patients with MI in the high lateral wall as indicated on the ECG

(23.6% *vs.* 8.2%,  $P<0.001$ ) (Table 2).

### Laboratory examination and clinical treatment

The counts of WBCs and neutrophils in the CR group were higher than those in the control group ( $P<0.001$ ). The hemoglobin level and left ventricular ejection fraction (LVEF) of the CR group were lower ( $P<0.001$ ). CK, amino-terminal pro-brain natriuretic peptide (NT-proBNP), and CK-MB levels in the CR group were higher ( $P<0.05$ ). A higher proportion of patients in the CR group used diuretics (54.9% *vs.* 30.3%,  $P<0.001$ ) and positive inotropic drugs (26.7% *vs.* 12.8%,  $P<0.001$ ) compared with the control group (Table 3).

### PCI surgery characteristics

As shown in Table 4, the proportion of single lesions in the CR group was lower than that in the control group (24.2% *vs.* 45.5%,  $P=0.004$ ). For the treatment of infarct-related artery (IRA), no significant differences were observed between the 2 groups in the application of thrombus aspiration, stent implantation, percutaneous transluminal coronary angioplasty (PTCA), and intracoronary thrombolysis. The preoperative TIMI blood flow grade (0.20±0.40 *vs.* 0.45±0.76,  $P=0.003$ ) and postoperative TIMI blood flow grade (2.55±0.92 *vs.* 2.96±0.23,  $P=0.001$ ) in the CR group

**Table 2** Status of first medical contact

Variables	CR group (n=195)	Control group (n=390)	P value
Duration of chest pain			
Chest pain onset time (h)	9 (4, 24)	10 (4, 24)	0.748
Total chest pain time (h)	7 (3, 15)	5 (2, 10)	<0.001
In-hospital vital signs			
SBP (mmHg)	120.88±20.47	123.73±19.35	0.099
DBP (mmHg)	73.29±13.20	75.14±13.07	0.109
HR (beats/min)	80.42±16.86	77.85±15.09	0.063
Temperature (°C)	36.35±0.42	36.54±2.11	0.201
In-hospital symptoms, n (%)			
Recurrent acute chest pain	48 (24.6)	39 (10.0)	<0.001
Dyspnea	74 (37.9)	176 (45.1)	0.098
Syncope*	5 (2.6)	5 (1.3)	0.313
ECG expression predicts AMI site, n (%)			
Anterior wall	114 (58.5)	206 (52.8)	0.196
Ventricle septal	4 (2.1)	19 (4.9)	0.098
High lateral wall	46 (23.6)	32 (8.2)	<0.001
Inferior wall and right ventricle	78 (40.0)	176 (45.1)	0.238
In-hospital clinical event, n (%)			
Acute heart failure	88 (45.1)	44 (11.3)	<0.001
Cardiac shock*	9 (4.6)	5 (1.3)	0.019
Infection	35 (17.9)	49 (12.6)	0.080
Hemorrhage	5 (2.6)	15 (3.8)	0.421
Fever	57 (29.2)	137 (35.1)	0.153
Stroke in-hospital*	1 (0.5)	2 (0.5)	1.000

\*, Fisher's exact test was used. CR, cardiac rupture; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; ECG, electrocardiogram; AMI, acute myocardial infarction.

were lower than the control group.

### Risk factors for CR

Multivariate logistic regression analysis of all patients showed that female (OR =2.318, 95% CI: 1.431–3.754, P=0.001), age (OR =1.066, 95% CI: 1.041–1.093, P<0.001), smoking (OR =1.750, 95% CI: 1.086–2.820, P=0.022), total chest pain time (OR =1.017, 95% CI: 1.000–1.035, P=0.049), recurrent acute chest pain (OR =2.750, 95% CI: 1.535–4.927, P=0.001), AMI in the high

lateral wall indicated by ECG (OR =5.527, 95% CI: 2.798–10.918, P<0.001), acute heart failure (OR =3.585, 95% CI: 2.074–6.195, P<0.001), and NT-proBNP level (OR =1.000, 95% CI: 1.000–1.000, P=0.023) were risk factors for CR (Figure 2). In patients who received PCI, multivariate logistic regression analysis showed that non-single lesion (OR =0.421, 95% CI: 0.204–0.867, P=0.019), preoperative TIMI grade (OR =0.358, 95% CI: 0.169–0.760, P=0.007), and postoperative TIMI grade (OR =0.222, 95% CI: 0.090–0.546, P=0.001) were risk factors for CR (Figure 3).

**Table 3** Laboratory examination and clinical treatment

Variables	CR group (n=195)	Control group (n=390)	P value
Laboratory tests			
WBC ( $\times 10^9/L$ )	11.85 $\pm$ 4.49	9.89 $\pm$ 3.17	<0.001
Neutrophils ( $\times 10^9/L$ )	9.39 $\pm$ 4.26	7.44 $\pm$ 3.18	<0.001
Absolute lymphocyte counts ( $\times 10^9/L$ )	1.39 $\pm$ 0.70	1.46 $\pm$ 0.60	0.190
Hemoglobin (g/L)	128.48 $\pm$ 17.97	134.55 $\pm$ 17.96	<0.001
Platelet count ( $\times 10^9/L$ )	228.24 $\pm$ 65.76	224.06 $\pm$ 62.77	0.455
CK (U/L)	1,109.00 (517.00, 1,958.00)	783.50 (273.25, 1,488.50)	<0.001
CK-MB (U/L)	109.32 (50.00, 220.57)	73.10 (32.00, 151.85)	<0.001
Ccr ( $\mu\text{mol/L}$ )	90.46 $\pm$ 52.27	85.28 $\pm$ 26.56	0.194
TG (mmol/L)	1.48 $\pm$ 0.92	1.58 $\pm$ 0.81	0.176
TC (mmol/L)	4.51 $\pm$ 0.92	4.58 $\pm$ 0.98	0.404
HDL-C (mmol/L)	1.09 $\pm$ 0.27	1.06 $\pm$ 0.27	0.287
LDL-C (mmol/L)	2.84 $\pm$ 0.76	2.98 $\pm$ 0.86	0.062
NT-proBNP (pg/mL)	3,606.00 (1,184.00, 7,750.81)	1,147.50 (353.15, 2,648.00)	<0.001
LVEF	43.93 $\pm$ 9.83	49.92 $\pm$ 8.85	<0.001
Clinical treatment, n (%)			
PCI	66 (33.8)	132 (33.8)	1.000
Thrombolysis	8 (4.1)	19 (4.9)	0.676
DAPT	190 (97.4)	386 (99.0)	0.168
$\beta$ -blocker	123 (63.1)	277 (71.0)	0.051
ACEI/ARB	120 (61.5)	260 (66.7)	0.220
Diuretics	107 (54.9)	118 (30.3)	<0.001
Positive inotropic drugs	52 (26.7)	50 (12.8)	<0.001

CR, cardiac rupture; WBC, white blood cells; CK, creatine kinase; CK-MB, creatine kinase-MB; Ccr, chemokine receptor; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; NT-proBNP, amino-terminal pro-brain natriuretic peptide; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; DAPT, dual antiplatelet therapy; ACEI/ARB, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers.

## Discussion

In this study, the incidence of CR in all STEMI patients was 0.89%, and the incidence of CR in patients who underwent direct PCI within 12 hours was 0.53%, which was similar to the results of previous studies (11-13). The development of PCI has reduced the mortality of STEMI, especially the incidence of CR. Timely implementation of direct PCI, early complete recanalization of IRA, and reducing the ischemic time can minimize reperfusion injury, save the myocardium as much as possible, and reduce the occurrence

of CR (13). Although some studies (14,15) have reported that early and timely surgical treatment or interventional occlusion treatment results in the short-term survival of patients with CR, the mortality rate is still high. For catastrophic left ventricular FWR, even with extracorporeal membrane oxygenation (ECMO)-supported surgery, the mortality rate remains high (16). Therefore, how to quickly and accurately identify possible CR in the early stage has become a key clinical issue. Rapid and accurate identification of early post-MI CR can prevent catastrophic events in patients (17).

**Table 4** PCI surgery characteristics

Variables	CR group (n=66)	Control group (n=132)	P value
Door to balloon time	63.15±31.92	63.93±21.29	0.838
Lesion complexity, n (%)			
Single lesion	16 (24.2)	60 (45.5)	0.004
Culprit lesion, n (%)			
LM*	3 (4.5)	2 (1.5)	0.336
LAD	39 (59.1)	71 (53.8)	0.479
LCX	8 (12.1)	14 (10.6)	0.749
RCA	16 (24.2)	45 (34.1)	0.157
Surgical strategy, n (%)			
Thrombus aspiration	18 (27.3)	46 (34.8)	0.283
Stent implantation	59 (89.4)	123 (93.2)	0.357
PTCA	46 (69.7)	92 (69.7)	1.000
Intracoronary thrombolysis*	0 (0)	1 (0.8)	1.000
TIMI flow grade			
Preoperative TIMI grade	0.20±0.40	0.45±0.76	0.003
Postoperative TIMI grade	2.55±0.92	2.96±0.23	0.001

\*, Fisher's exact test was used. PCI, percutaneous coronary intervention; CR, cardiac rupture; LM, left main; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; PTCA, percutaneous transluminal coronary angioplasty; TIMI, thrombolysis in myocardial infarction.

In this study, age and the proportion of women in the CR group were higher than the control group, which was similar to some previous reports (3,18,19). A high incidence of CR in elderly patients may be due to the hyperextension and ventricular wall expansion of the MI area, as well as decreased ventricular wall compliance in the early stage of STEMI. Female patients have less heart fat and weaker myocardial fibers than men, which may be the main factor for CR after STEMI.

The total chest pain time of patients and the recurrent acute chest pain rates in the CR group were higher than the control group, which is similar to the findings of Hao *et al.* (19). CR after STEMI may occur due to the severity of illness or larger MI size, which showed longer total chest pain time in the clinic. On the other hand, CR after STEMI may cause higher recurrent acute chest pain rates due to recurrent MI. Reperfusion therapy should be performed as soon as possible after MI to shorten the ischemic time and reduce the occurrence of CR. In this study, there were no statistical differences between the chest pain onset time and the door to balloon time in patients treated with PCI.

Almost all patients eligible for reperfusion therapy received reperfusion therapy at hospital admission or prehospital, which is the reason why the overall incidence of CR in this study was lower than that in the drug treatment era. The rates of acute heart failure and cardiac shock in the CR group were higher than the control group, and the LVEF of the CR group was lower, which is similar to the conclusion of Fu *et al.* (4). The possible reasons include more severe disease, poorer cardiac function, wider and deeper infarcts, and thinner myocardium in the necrotic area in the CR group.

This study is the first to predict the risk of CR based on the location of the infarction indicated on the ECG. The proportion of patients with high lateral MI in the CR group was higher than in the control group. High lateral MI indicated on the ECG is a form of FWR, which was reported as the most frequent site among all the CRs including FWR, VSR, and PMR (20). A previous meta-analysis (21) indicated that anterior wall MI may be a risk factor for CR, which is inconsistent with our study. We speculate that there are several reasons for this. First, the

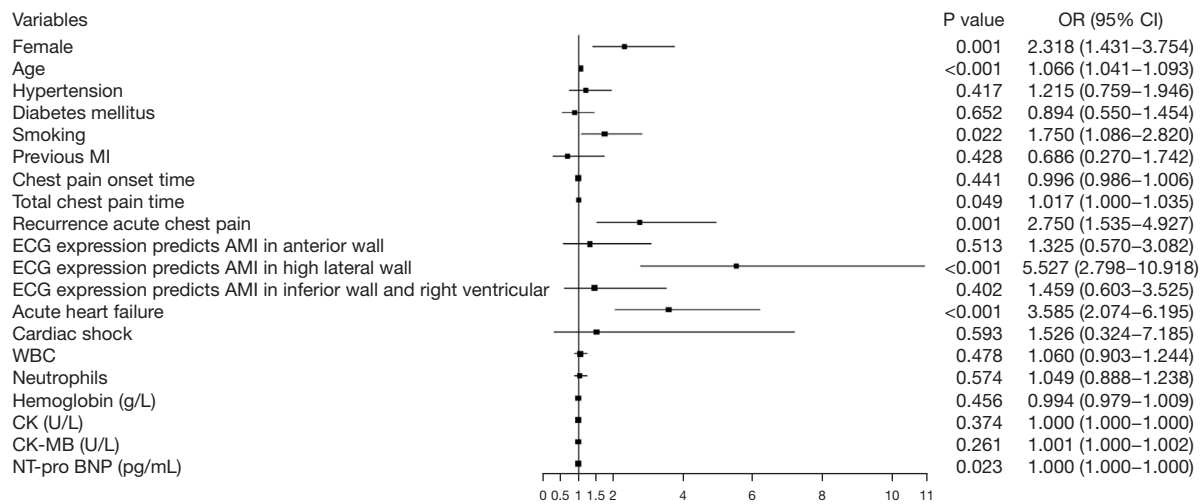


Figure 2 Multivariate logistic regression analysis of risk factors for CR in all patients. CR, cardiac rupture.

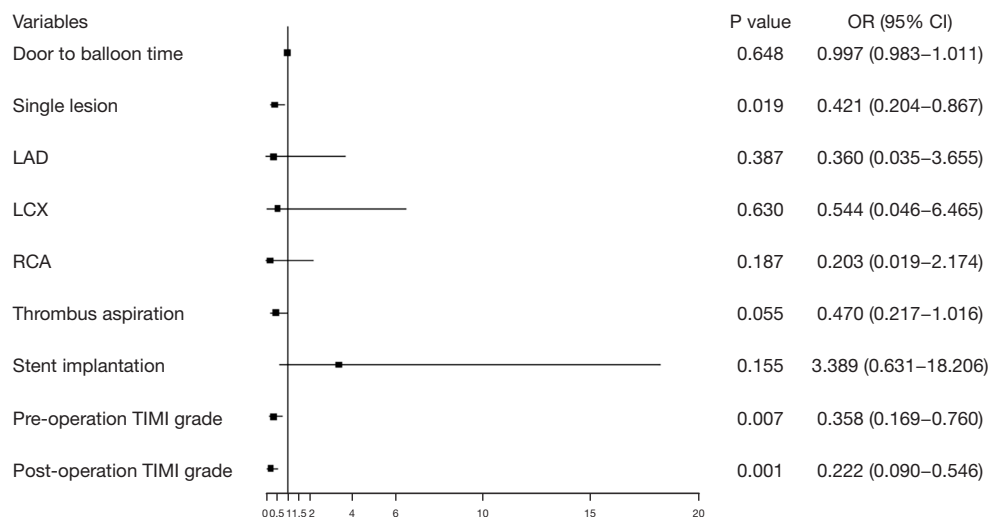


Figure 3 Multivariate logistic regression analysis of risk factors for CR in patients who accepted PCI. CR, cardiac rupture; PCI, percutaneous coronary intervention.

promotion of main vessel stenting in emergent PCI reduces the proportion of CR in anterior wall MI, leading to a relative increase in the proportion of CR in the lateral MI. Second, although revascularization can be achieved by PCI or other treatments, the high lateral MI indicated on the ECG occurred in the left ventricle near the atrioventricular sulcus. Abnormal shear stress may occur during myocardial contraction in this area, which is due to its special anatomical structure, namely, the connection between the myocardium and the annulus fiber (left and right fiber triangle) (22). Therefore, these reasons make high lateral

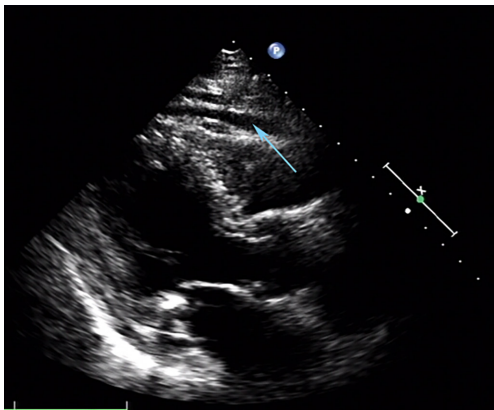
MI indicated on the ECG a risk factor for CR. Third, the IRA of high lateral MI may be the diagonal branch or obtuse marginal branch that lacks effective collateral circulation, thus infarction or accidental injury during emergency PCI may increase the possibility of CR (23).

In this study, the levels of WBCs and neutrophils in patients with CR were higher than the control group, and the hemoglobin level was lower than the control group, which is consistent with a previous study (24). A recent study suggested that elevated WBCs and neutrophils are risk factors for septal perforation (25). MI is related to



inflammatory responses, which can mediate ischemic events to delay the healing of myocardial tissue, promote tissue damage, and cause CR by releasing inflammatory factors (26,27). The early myocardial hemorrhage in CR often leads to a decrease in hemoglobin level (28). In this study, the levels of CK, CK-MB, and NT-proBNP in patients with CR were higher than the control group, which was similar to the results of Lu *et al.* (5), and may be due to the release of more cardiac enzymes, poorer cardiac function, and larger infarct area in patients with CR.

Patients in the CR group who underwent emergency

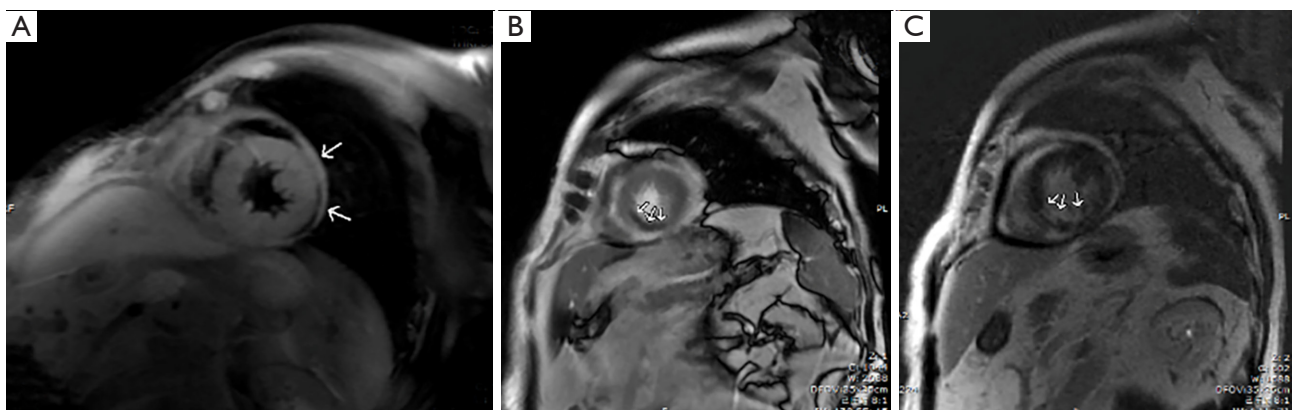


**Figure 4** Ultrasound images of patients with CR. Long axis view of the left ventricle next to the sternum. The blue arrow shows the hemopericardium. CR, cardiac rupture.

PCI had more complex lesions and lower preoperative and postoperative TIMI blood flow grades than the control group, which was similar to the conclusions of Rencuzogullari *et al.* (29). Poor coronary artery conditions and complex lesions before STEMI can aggravate the rapid deterioration of cardiac function and increase the risk of CR after MI. In addition, in the CR group, the low preoperative and postoperative TIMI blood flow grades and unimproved blood perfusion, even no-reflow in some patients, can lead to the failure of effective recovery of myocardial injury and subsequent CR. The results of the multivariate logistic regression analysis were similar to previous studies (3,4,18,19), indicating female, age, smoking, total chest pain time, recurrent acute chest pain, acute heart failure, and NT-proBNP level can be used to predict CR after STEMI.

It is worth noting that in this study, 6 patients were found to have early CR through myocardial magnetic resonance imaging (MRI) (Figures 4,5) and preventive measures were taken to prevent the occurrence of catastrophic events. Although MRI is expensive, a previous case report (30) also indicated that myocardial MRI could diagnose early CR, which is due to its ability to detect early intramyocardial hemorrhage.

There were some limitations in this study. The small sample size of CR patients was one of the limitations. In addition, early recognition of CR by MRI can be applied to more patients, however its use is limited due to its expensive cost. This is a dual-center retrospective study with a low level of evidence, however, a prospective study cannot



**Figure 5** Cardiac magnetic resonance images. (A) Fat-suppressed T1-weighted black blood sequence. The high signal of pericardial effusion suggests bloody effusion (white arrows). (B) Short-axis film sequence (FIESTA Cine) showed a strip-like low signal in the subendocardium of the inferior wall and the inferior septal wall at the proximal apical region (white arrows), suggesting a local infarction with minimal intramuscular bleeding. (C) Short-axis delayed enhanced images (8 min) showed partially transmural delayed enhancement area of the subendocardium of the inferior wall and the inferior septal wall at the proximal apical region (white arrows), suggesting MI. MI, myocardial infarction.

be conducted due to the particularity and ethics of CR. Therefore, this study is still commendable.

## Conclusions

The early and accurate identification and prevention of possible CR is of great importance. In patients who received PCI, non-single lesions and preoperative and postoperative TIMI grades were risk factors for CR. In addition to previously reported indicators, we found that AMI in the high lateral wall as indicated by ECG is a risk factor for CR in all patients.

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*Ethical Statement:* The authors are accountable for all 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. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of Tianjin Chest Hospital (No. 2021LW015) and Cangzhou Central Hospital (No. 2021-187-01). Signed informed consent was obtained from the family member of each patient.

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## References

1. Figueras J, Alcalde O, Barrabés JA, et al. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation* 2008;118:2783-9.
2. López-Sendón J, Gurfinkel EP, Lopez de Sa E, et al. Factors related to heart rupture in acute coronary syndromes in the Global Registry of Acute Coronary Events. *Eur Heart J* 2010;31:1449-56.
3. Qian G, Jin RJ, Fu ZH, et al. Development and validation of clinical risk score to predict the cardiac rupture in patients with STEMI. *Am J Emerg Med* 2017;35:589-93.
4. Fu Y, Li KB, Yang XC. A risk score model for predicting cardiac rupture after acute myocardial infarction. *Chin Med J (Engl)* 2019;132:1037-44.
5. Lu Q, Liu P, Huo JH, et al. Cardiac rupture complicating acute myocardial infarction: the clinical features from an observational study and animal experiment. *BMC Cardiovasc Disord* 2020;20:409.
6. Shoji K, Yanishi K, Kawamata H, et al. New risk factors for early- and late-onset cardiac rupture in ST-elevation myocardial infarction patients after primary percutaneous coronary intervention. *J Cardiol* 2022;79:400-7.
7. Ptaszyńska-Kopczyńska K, Sobolewska D, Koźuch M, et al. Efficacy of invasive treatment and the occurrence of cardiac rupture in acute ST-elevation myocardial infarction. *Kardiologia Pol* 2011;69:795-800.
8. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). *J Am Coll Cardiol* 2018;72:2231-64.
9. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J* 2018;39:119-77.
10. TIMI Study Group. The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. *N Engl J Med* 1985;312:932-6.
11. Bajaj A, Sethi A, Rathor P, et al. Acute Complications of

- Myocardial Infarction in the Current Era: Diagnosis and Management. *J Investig Med* 2015;63:844-55.
12. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal Trends and Outcomes of Mechanical Complications in Patients With Acute Myocardial Infarction. *JACC Cardiovasc Interv* 2019;12:1825-36.
  13. Honda S, Asaumi Y, Yamane T, et al. Trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. *J Am Heart Assoc* 2014;3:e000984.
  14. Khazi FM, Zamkan B, AbdelAziz T, et al. Ischemic posterior wall cardiac rupture: early intervention is key for success. *Asian Cardiovasc Thorac Ann* 2019;27:681-4.
  15. Molek P, Włodarczyk A, Gajos G, et al. Simultaneous cardiac free-wall rupture and ventricular septal rupture following acute myocardial infarction treated with emergency balloon inflation. *Pol Arch Intern Med* 2019;129:830-2.
  16. Matteucci M, Formica F, Kowalewski M, et al. Meta-analysis of surgical treatment for postinfarction left ventricular free-wall rupture. *J Card Surg* 2021;36:3326-33.
  17. Yoneyama K, Ishibashi Y, Koeda Y, et al. Association between acute myocardial infarction-to-cardiac rupture time and in-hospital mortality risk: a retrospective analysis of multicenter registry data from the Cardiovascular Research Consortium-8 Universities (CIRC-8U). *Heart Vessels* 2021;36:782-9.
  18. Leitman M, Tsatskin L, Hendler A, et al. Cardiac Rupture: New Features of the Old Disease. *Cardiology* 2016;133:257-61.
  19. Hao Z, Ma J, Dai J, et al. A real-world analysis of cardiac rupture on incidence, risk factors and in-hospital outcomes in 4190 ST-elevation myocardial infarction patients from 2004 to 2015. *Coron Artery Dis* 2020;31:424-9.
  20. Xue X, Kan J, Zhang JJ, et al. Comparison in Prevalence, Predictors, and Clinical Outcome of VSR Versus FWR after Acute Myocardial Infarction: The Prospective, Multicenter Registry MOODY Trial-Heart Rupture Analysis. *Cardiovasc Revasc Med* 2019;20:1158-64.
  21. Hao W, Lu S, Guo R, et al. Risk factors for cardiac rupture complicating myocardial infarction: a PRISMA meta-analysis and systematic review. *J Investig Med* 2019;67:720-8.
  22. Partridge JB, Anderson RH. Left ventricular anatomy: its nomenclature, segmentation, and planes of imaging. *Clin Anat* 2009;22:77-84.
  23. Asakura K, Homma T, Akutsu N, et al. Cardiac Rupture Due to Side Branch Occlusion After Stent Implantation-The Crime of Jailed Stent. *Circ J* 2020;84:295.
  24. Sulzgruber P, El-Hamid F, Koller L, et al. Long-term outcome and risk prediction in patients suffering acute myocardial infarction complicated by post-infarction cardiac rupture. *Int J Cardiol* 2017;227:399-403.
  25. Zhang XY, Bian LZ, Tian NL. The Clinical Outcomes of Ventricular Septal Rupture Secondary to Acute Myocardial Infarction: A Retrospective, Observational Trial. *J Interv Cardiol* 2021;2021:3900269.
  26. Usui S, Chikata A, Takatori O, et al. Endogenous muscle atrophy F-box is involved in the development of cardiac rupture after myocardial infarction. *J Mol Cell Cardiol* 2019;126:1-12.
  27. Ishikawa S, Noma T, Fu HY, et al. Apoptosis inhibitor of macrophage depletion decreased M1 macrophage accumulation and the incidence of cardiac rupture after myocardial infarction in mice. *PLoS One* 2017;12:e0187894.
  28. Qian G, Wu C, Chen YD, et al. Predictive factors of cardiac rupture in patients with ST-elevation myocardial infarction. *J Zhejiang Univ Sci B* 2014;15:1048-54.
  29. Rencuzogullari I, Çağdaş M, Karabağ Y, et al. Association of the SYNTAX Score II with cardiac rupture in patients with ST-segment elevation myocardial infarction undergoing a primary percutaneous coronary intervention. *Coron Artery Dis* 2018;29:97-103.
  30. Zhong W, Liu Z, Fan W, et al. Transcatheter closure for the treatment of pseudoventricular aneurysm after acute myocardial infarction: a case report. *Ann Transl Med* 2020;8:1528.

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