



Predictive factors of cardiac rupture in patients with ST-elevation myocardial infarction*

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Abstract: Cardiac rupture (CR) is a potentially fatal mechanical complication of ST-elevation myocardial infarction (STEMI). We aimed to determine the incidence and risk factors of CR in Chinese STEMI patients. A total of 9798 consecutive STEMI patients from four centers in China were retrospectively analyzed, among which 178 patients had CR. STEMI patients without CR were chosen as a control group. Clinical characteristics were compared between STEMI patients with CR and those without CR. The incidence of CR in STEMI patients was 1.82%, and the 30-d mortality was up to 61.2%. CR patients were significantly older, more female, and associated with a longer time from onset of pain to hospital admission than their non-CR counterparts ($P < 0.001$). More patients with anterior myocardial infarction (82.1%) were found in the CR group, and CR patients had significantly higher heart rates than the control group (91 ± 19 bpm vs. 71 ± 16 bpm; $P < 0.001$). In multiple-adjusted models, the independent risk factors of CR were advanced age, female gender, anaemia, increased heart rate, anterior myocardial infarction, increased white blood cell (WBC) count, delayed admission, and renal dysfunction. The level of hemoglobin remained a significant determinant factor of CR (OR (95% CI): 0.82 (0.75–0.89); $P < 0.001$) after adjusting for various potential confounding factors. Counts of WBC also remained a significant determinant of the CR (OR (95% CI): 1.08 (1.04–1.12); $P < 0.001$). A number of variables were independently related to CR. This study indicated, for the first time, that both hemoglobin and WBC levels were independently correlated with occurrence of CR.

Key words: ST-elevation myocardial infarction, Risk factor, Anaemia, Cardiac rupture

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1 Introduction

Cardiac rupture (CR) after ST-elevation myocardial infarction (STEMI) includes free-wall rupture (FWR), ventricular septal rupture (VSR), and papillary muscle rupture (PMR). Along with the development of primary percutaneous coronary intervention (PCI) and drug therapy, the incidence of CR has

gradually declined (Figueras *et al.*, 2008). However, there are still a number of deaths caused by CR that could not be determined until autopsy. CR is still one of the common causes of in-hospital deaths for STEMI patients (Markowicz-Pawlus *et al.*, 2007; Figueras *et al.*, 2008). The determination of CR risk factors plays an important role in the prevention of CR. Independent risk factors reported include female gender, advanced age, lower blood pressure, higher heart rate, delayed admission, and thrombolysis (Yip *et al.*, 2003; Okino *et al.*, 2005; French *et al.*, 2010; Lopez-Sendon *et al.*, 2010; Qian *et al.*, 2011). Patients with CR have new clinical characteristics in the era of reperfusion. Up to now there has been a lack of systemic

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research on the assessment of CR risk factors in Chinese STEMI patients from multiple centers. The objective of our study was to determine the incidence and risk factors of CR in Chinese STEMI patients.

2 Subjects and methods

2.1 Study population

We retrospectively analyzed a total of 9798 consecutive STEMI patients who were admitted from January 2011 to January 2013 from four centers (Chinese PLA General Hospital, Beijing Anzhen Hospital, Beijing Fuwai Hospital, and Beijing Chao-Yang Hospital). One hundred and seventy-eight of these patients with CR served as a case group, and the others without CR were chosen as a control group. Diagnosis of STEMI was based on the concurrence of symptoms (chest pain or symptoms compatible with acute heart failure or unexplained syncope) and electrocardiogram findings (ST-segment elevation ≥ 1 mm in ≥ 2 inferior leads or ≥ 2 mm in ≥ 2 precordial leads). The diagnosis of FWR was based on strict necropsy or electrical mechanical dissociation or pericardial tamponade accompanied with laboratory results (echocardiography showed massive pericardial effusion and diagnostic pericardiocentesis showed bloody fluid). VSR and PMR were established by the emergence of new systolic murmurs and the result of echocardiography. STEMI patients received aspirin, clopidogrel, statin, low molecular weight heparin (LMWH), β -blockers, and angiotensin converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) according to the STEMI guideline (Kushner *et al.*, 2009), unless there were contraindications to these drugs. We provided a comparative analysis between CR patients and non-CR patients by clinical manifestations, laboratory profiles, and the treatments during their stay in hospital. Peripheral blood samples were collected from patients within 12 h of admission for blood routines and biochemistry examinations.

2.2 Statistical analysis

Variables following a normal distribution were expressed as mean \pm standard deviation (SD). Variables following a non-normal distribution were expressed as median (interquartile range). The means of

normally distributed variables were compared by using Student's *t*-test. For comparisons of the distributions of variables between groups, χ^2 analysis was used. The Mann-Whitney *U* test was used for those with non-normal distribution. Independent CR risk factors were calculated using multivariable logistic regression models (stepwise regression hazards analysis). Statistical analyses were performed using SPSS software (Version 19.0, SPSS Inc., USA). Statistical significance was set at $P < 0.05$.

3 Results

3.1 Incidence of CR

In the 9798 consecutive patients with STEMI, 178 patients (1.82%) developed CR. Of these CR patients, FWR occurred in 118 patients (1.20%), VSR occurred in 49 patients (0.50%), and PMR occurred in 11 patients (0.11%). The incidence of CR varied across the four hospital clusters: 2.20% (54/2450) for Chinese PLA General Hospital vs. 1.68% (43/2560) for Beijing Anzhen Hospital vs. 1.90% (60/3150) for Beijing Fuwai Hospital vs. 1.44% (21/1460) for Beijing Chao-Yang Hospital. Despite the improvements in CR treatments such as repair operation and pericardial device closure, the 30-d mortality of CR was still up to 61.2% (109/178) compared with 5.9% (567/9620) in those without CR ($P < 0.001$). And CR accounted for 16.1% (109/676) of all cause in-hospital deaths post-STEMI. In-hospital mortality was higher in patients with FWR (92/118, 78.0%) than in those with VSR or PMR (17/60, 28.3%). The presence of cardiac tamponade combined with hemodynamic instability was the common finding of FWR (68/118, 57.6%), while sudden unexpected cardiogenic shock or heart failure was the common finding of VSR or PMR (49/60, 81.7%).

3.2 Clinical characteristics and treatments before CR

Demographics, medical histories, clinical features, and treatments in patients with and without CR are shown in Table 1. The female proportion and advanced age proportion in the CR group were significantly higher than those in the control group ($P < 0.001$). The occurrence of hypertension was more frequent in CR patients ($P < 0.001$), whereas smoking

occurred less frequently in CR patients ($P=0.037$). More patients with anterior myocardial infarction (82.1%) were found in the CR group. There were also significant trends toward higher heart rate and lower blood pressure in the CR group ($P<0.001$). The initial hemoglobin levels of the CR group were significantly lower than those of the control group ((12.1 ± 1.8) g/dl vs. (13.4 ± 2.0) g/dl; $P<0.001$), while the white blood cell (WBC) counts were significantly higher in the CR group ($(11.5\pm 4.2)\times 10^9$ L⁻¹ vs. $(9.6\pm 3.7)\times 10^9$ L⁻¹; $P<0.001$). The CR patients tended to have longer time from onset of pain to first medical contact (12 (6–22) h vs. 10 (4–16) h; $P<0.001$). Both left ventricular ejection fraction and Killip class indicated that the CR group had worse heart function. Recommended

medications within the first 24 h including ACEI/ARB and β -blockers were used less frequently in CR patients ($P<0.001$). Furthermore, primary PCI treatment was used more frequently in the non-CR group ($P<0.001$).

3.3 Risk factors for post-infarct CR

Multivariable logistic regression analysis included clinical characteristics on admission (age, gender, heart rate, smoking habit, blood pressure, myocardial infarction location, Killip class, hemoglobin level, count of WBC, time to admission, and eGFR; 9798 patients with complete data). The independent predictors of CR were as below: female gender (odds ratio (OR) 2.63, 95% confidence

Table 1 Clinical characteristics and treatments in STEMI patients with and without cardiac rupture (CR)

Clinical characteristics and treatment	CR group (n=178)	Control group (n=9620)	P
Demographic characteristics			
Male	98 (55.1%)	7763 (80.7%)	<0.001
Age (year)	67±10	61±13	<0.001
BMI (kg/m ²)	24.2±3.3	25.0±4.0	0.252
Hypertension	108 (60.7%)	5069 (52.7%)	0.037
Diabetes mellitus	48 (27.0%)	2309 (24.0%)	0.866
Hypercholesterolemia	40 (22.5%)	1673 (17.4%)	0.079
Current smoking	59 (33.1%)	5099 (53.0%)	<0.001
Clinical presentation in the first 24 h			
Anterior myocardial infarction	146 (82.1%)	5262 (54.7%)	<0.001
SBP (mmHg)	111±21	122±23	<0.001
DBP (mmHg)	70±13	73±14	0.014
Pulse (bpm)	91±19	77±16	<0.001
Killip class III or IV	75 (42.1%)	2097 (21.8%)	<0.001
Initial white blood cell ($\times 10^9$ L ⁻¹)	11.5±4.2	9.6±3.7	<0.001
Initial hemoglobin (g/dl)	12.1±1.8	13.4±2.0	<0.001
Time from onset to admission (h)	12 (6–22)	10 (4–16)	<0.001
Initial random glucose (mmol/L)	9.9±5.1	9.2±3.6	0.131
Initial LDL-C (mmol/L)	2.73±0.83	2.81±1.07	0.424
eGFR (ml/min)	61.6±24.8	79.6±23.5	<0.001
Left ventricular ejection fraction (%)	47±11	52±11	<0.001
Left ventricular end diastolic diameter (mm)	53±9	49±6	<0.001
In-hospital mortality	109 (61.2%)	567 (5.9%)	<0.001
Treatments in the first 24 h			
Aspirin+thienopyridine	162 (91.0%)	9004 (93.6%)	0.173
β -Blocker	121 (68.0%)	7436 (77.3%)	<0.001
ACEI/ARB	74 (41.4%)	4598 (47.8%)	0.009
Statin	164 (92.1%)	9120 (94.8%)	0.119
Primary percutaneous intervention	54 (30.3%)	6705 (69.7%)	<0.001
Thrombolysis	31 (17.4%)	1279 (13.3%)	0.116

Data are expressed as number of patients (percentage), median (range), or mean±SD. BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LDL-C: low density lipoprotein cholesterol; eGFR: estimated glomerular filtration rate; ACEI/ARB: angiotensin converting enzyme inhibitor or angiotensin receptor blocker

interval (CI) 1.77–3.92), age per 10-year increase (OR 1.30, 95% CI 1.10–1.53), heart rate per 10-beat per minute (bpm) increase (OR 1.42, 95% CI 1.29–1.56), systolic blood pressure (SBP) per 10-mmHg decrease (OR 1.27, 95% CI 1.18–1.40), anterior myocardial infarction (OR 3.23, 95% CI 2.07–5.04), hemoglobin level ≥ 12 g/dl (OR 0.49, 95% CI 0.32–0.73), count of WBC (OR 1.09, 95% CI 1.05–1.14), time to admission (OR 1.01, 95% CI 1.00–1.12), and eGFR ≤ 60 ml/min (OR 1.98, 95% CI 1.32–2.97). These are shown in Table 2.

3.4 New CR predictors: the relationships between results of blood routine and CR

We have explored new risk factors of CR: one was lower hemoglobin level, and the other was higher WBC count. Anemia had a close relationship with

gender, age, and renal function, so we analyzed the association between hemoglobin level and occurrence of CR after adjustments for the above confounding factors. We found that the lower level of hemoglobin remained a significant determinant of CR (OR (95% CI): 0.82 (0.75–0.89); $P < 0.001$; c -statistic value=0.680) after adjusting for various confounding factors. There was no significant interaction between male and female ($P = 0.420$), as shown in Table 3. Higher WBC counts also remained a significant determinant of CR (OR (95% CI): 1.08 (1.04–1.12); $P < 0.001$; c -statistic value=0.629), as shown in Table 4. The lower level of hemoglobin was also independently correlated with pericardial effusion ($P < 0.001$). Incidence of CR increased with the decline of hemoglobin level, while incidence of CR increased with the increase of WBC counts, which is shown in Fig. 1.

Table 2 Univariate and multivariate analyses of clinical characteristics for cardiac rupture (CR) patients

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	<i>P</i> value	OR	95% CI	<i>P</i> value
Age per extra 10 years	1.44	1.26–1.63	<0.001	1.30	1.10–1.53	<0.001
Female gender	3.42	2.52–4.63	<0.001	2.63	1.77–3.92	<0.001
Current smoking	0.44	0.32–0.60	<0.001	0.76	0.52–1.10	0.143
HR per extra 10 bpm	1.53	1.42–1.65	<0.001	1.42	1.29–1.56	<0.001
SBP per extra 10 mmHg	0.79	0.74–0.85	<0.001	0.78	0.71–0.85	<0.001
Anterior myocardial infarction	3.80	2.56–5.63	<0.001	3.23	2.07–5.04	<0.001
Killip class III or IV	2.62	1.93–3.55	<0.001	0.85	0.57–1.25	0.398
Hemoglobin level ≥ 12 g/dl	0.36	0.26–0.49	<0.001	0.49	0.32–0.73	<0.001
Count of WBC	1.11	1.08–1.15	<0.001	1.09	1.05–1.14	<0.001
Time to admission	1.65	1.16–2.35	0.005	1.01	1.00–1.02	0.015
eGFR ≤ 60 ml/min	0.26	0.19–0.35	<0.001	1.98	1.32–2.97	<0.001

HR: heart rate; SBP: systolic blood pressure; WBC: white blood cell; eGFR: estimated glomerular filtration rate

Table 3 Association between anemia and cardiac rupture (CR) evaluated with logistic regression analysis, with adjustments for age, gender, and other confounding factors

Gender	HGB (g/dl)	Model 1		Model 2		<i>P</i> value of interaction
		OR (95% CI)	<i>P</i> value	OR (95% CI)	<i>P</i> value	
Male	13.5 \pm 1.9	0.785 (0.717–0.859)	<0.001	0.756 (0.655–0.872)	<0.001	0.420
Female	12.2 \pm 1.9	0.801 (0.726–0.884)	<0.001	0.862 (0.739–0.998)	0.045	

Model 1 regression was done with adjustment for age; Model 2 regression was done with adjustment for age, systolic blood pressure, heart rate, estimated glomerular filtration rate, anterior myocardial infarction, and count of white blood cell. HGB: hemoglobin

Table 4 Association between higher count of WBC and cardiac rupture (CR) evaluated with logistic regression analysis, with adjustments for age, gender and other confounding factors

Gender	WBC ($\times 10^9$ L ⁻¹)	Model 1		Model 2		<i>P</i> value of interaction
		OR (95% CI)	<i>P</i> value	OR (95% CI)	<i>P</i> value	
Male	9.65 \pm 3.66	1.115 (1.063–1.169)	<0.001	1.066 (1.012–1.122)	<0.001	0.480
Female	9.70 \pm 4.12	1.091 (1.026–1.160)	<0.001	1.091 (1.026–1.160)	<0.001	

Model 1 regression was done with adjustment for age; Model 2 regression was done with adjustment for age, systolic blood pressure, heart rate, estimated glomerular filtration rate, admission time, and hemoglobin. WBC: white blood cell

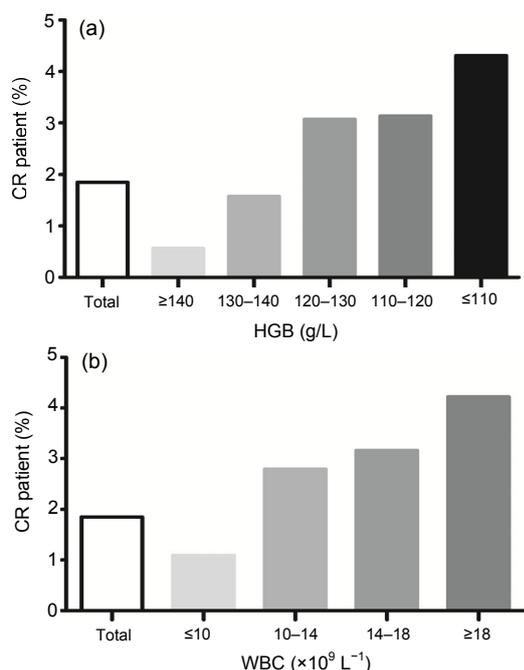


Fig. 1 Relations between cardiac rupture (CR) and anaemia (a) and white blood cell (WBC) levels (b)

(a) CR incidence increases with the decline of hemoglobin (HGB): 22 (0.56%), 35 (1.57%), 47 (3.07%), 29 (3.13%), and 45 (4.31%) for ≥ 140 , 130-140, 120-130, 110-120, and ≤ 110 g/L HGB, respectively. (b) CR incidence increases with the increase of WBC levels: 63 (1.09%), 75 (2.79%), 28 (3.16%), and 12 (4.22%) for $\leq 10 \times 10^9$, 10×10^9 - 14×10^9 , 14×10^9 - 18×10^9 , and $\geq 18 \times 10^9 L^{-1}$ WBC, respectively

4 Discussion

CR remains a major cause of mortality after STEMI and persists as a clinic challenge (Gao *et al.*, 2012). The current incidence of rupture reported in the literature is between 1% and 3% of patients suffering STEMI (Okino *et al.*, 2005; French *et al.*, 2010; Lopez-Sendon *et al.*, 2010). However, autopsy research detected CR in 30% to 65% of patients who died of acute myocardial infarction (AMI) (Hutchins *et al.*, 2002), which indicated that CR might have been globally underestimated by misdiagnosis. Our study showed the incidence of CR was 1.8%, slightly lower than reported before, partly because of the strict diagnostic criteria in our study. Some sudden unexpected deaths were not diagnosed as CR due to lack of autopsy. The percentage of sudden deaths due to rupture remained high in the first month post-infarction (Pouleur *et al.*, 2010). CR was the second

cause of 30-d mortality for STEMI in our study (16.1%, 109/676), while pump failure was the first cause, which was in accordance with previous reports confirmed by autopsy (Shamshad *et al.*, 2010).

It is necessary to have early and effective risk stratifications in STEMI patients according to risk factors of CR. Our research based on multicenter database was more credible than previous studies, and has investigated some new independent risk factors of CR. We further analyzed some new risk factors such as higher WBC count and lower level of hemoglobin. In particular, hemoglobin level decline independently correlated to occurrence of CR in Chinese STEMI patients after adjustments for other CR risk factors. In CR patients, leukocytes in filtration and hemorrhage are key histopathology (Lewis *et al.*, 1969; Kawano *et al.*, 1994; van den Borne *et al.*, 2009). The intramyocardial dissecting hematoma might appear as a component of the rupture during the remodeling process, and increases the likelihood of CR (Pliam and Sternlieb, 1993; Vargas-Barrón *et al.*, 2013). Both intramyocardial dissecting hematoma and intramyocardial hemorrhage are associated with hemoglobin level decline. On the other hand, pericardial effusion may be present for a few days prior to a fatal rupture (Figueras *et al.*, 2010), implying that CR is a dynamic process. Pericardial effusion was also independently correlated with anemia in our study. In our previous research, we have indicated that risk of CR after AMI was related to the risk of hemorrhage (Qian *et al.*, 2013). Hemorrhage of the myocardium often leads to hemoglobin level decline and bloody pericardial effusion, which could explain why anemia was closely associated with occurrence of CR. Both inflammatory cells in filtration and matrix metalloproteinase-9 (MMP-9) expression were significantly more severe in ruptured than in non-ruptured hearts with acute MI (Fang *et al.*, 2007; van den Borne *et al.*, 2009; Gao *et al.*, 2010), which could explain why the CR group had higher WBC counts in our study.

The prevention of mechanical complications should be emphasized in the acute phase of STEMI. We aimed to establish a risk evaluation model providing a good discrimination in Chinese STEMI patients. An early and effective intervention strategy for CR prevention is necessary. Early use of β -blockers and ACEI/ARB could help to prevent CR for patients with high CR risk (Figueras *et al.*, 2008;

Pedrazzini *et al.*, 2008). Primary PCI therapy rather than thrombolysis could reduce the incidence of CR (Yip *et al.*, 2003; Okino *et al.*, 2005; Figueras *et al.*, 2008; Qian *et al.*, 2011). The gradual decline in the incidence of CR over a 30-year period was associated with progressive implementation of primary PCI (Figueras *et al.*, 2008). Such knowledge will help facilitate personalized decision-making for patients who are at high risk of CR.

This study has some limitations. CR is an uncommon clinical condition, and thus involves a limited number of cases. This study included a few cases diagnosed based on clinical manifestations rather than necropsy. This was a retrospective cohort study, so retrospective bias exists in the statistics.

5 Conclusions

The incidence of CR is low in Chinese STEMI patients, but is associated with poor prognosis. A number of variables are independently related to CR. This study pointed out, for the first time, that both hemoglobin and WBC levels were strongly and independently correlated with occurrence of CR.

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Compliance with ethics guidelines

Geng QIAN, Chen WU, Yun-dai CHEN, Chen-chen TU, Jin-wen WANG, and Yong-an QIAN declare that they have no conflict of interest.

This article does not contain any studies with human or animal subjects performed by any of the authors.

References

- Fang, L., Gao, X.M., Moore, X.L., *et al.*, 2007. Differences in inflammation, MMP activation and collagen damage account for gender difference in murine cardiac rupture following myocardial infarction. *J. Mol. Cell. Cardiol.*, **43**(5):535-544. [doi:10.1016/j.yjmcc.2007.06.011]
- Figueras, J., Alcalde, O., Barrabes, J.A., *et al.*, 2008. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation*, **118**(25):2783-2789. [doi:10.1161/CIRCULATIONAHA.108.776690]
- Figueras, J., Barrabes, J.A., Serra, V., *et al.*, 2010. Hospital outcome of moderate to severe pericardial effusion complicating ST-elevation acute myocardial infarction. *Circulation*, **122**(19):1902-1909. [doi:10.1161/CIRCULATIONAHA.109.934968]
- French, J.K., Hellkamp, A.S., Armstrong, P.W., *et al.*, 2010. Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). *Am. J. Cardiol.*, **105**(1):59-63. [doi:10.1016/j.amjcard.2009.08.653]
- Gao, X.M., Ming, Z., Su, Y., *et al.*, 2010. Infarct size and post-infarct inflammation determine the risk of cardiac rupture in mice. *Int. J. Cardiol.*, **143**(1):20-28. [doi:10.1016/j.ijcard.2009.01.019]
- Gao, X.M., White, D.A., Dart, A.M., *et al.*, 2012. Post-infarct cardiac rupture: recent insights on pathogenesis and therapeutic interventions. *Pharmacol. Ther.*, **134**(2):156-179. [doi:10.1016/j.pharmthera.2011.12.010]
- Hutchins, K.D., Skurnick, J., Lavenhar, M., *et al.*, 2002. Cardiac rupture in acute myocardial infarction: a reassessment. *Am. J. Forensic Med. Pathol.*, **23**(1):78-82. [doi:10.1097/00000433-200203000-00017]
- Kawano, H., Miyauchi, K., Okada, R., *et al.*, 1994. Histopathological study of cardiac rupture following myocardial infarction with and without thrombolytic therapy. *J. Cardiol.*, **24**(4):249-255.
- Kushner, F.G., Hand, M., Smith, S.C., *et al.*, 2009. 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (updating the 2004 guideline and 2007 focused update) and ACC/AHA/SCAI guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update): a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *Circulation*, **120**(22):2271-2306. [doi:10.1161/CIRCULATIONAHA.109.192663]
- Lewis, A.J., Burchell, H.B., Titus, J.L., 1969. Clinical and pathologic features of postinfarction cardiac rupture. *Am. J. Cardiol.*, **23**(1):43-53. [doi:10.1016/0002-9149(69)90240-9]
- Lopez-Sendon, J., Gurfinkel, E.P., Lopez de Sa, E., *et al.*, 2010. Factors related to heart rupture in acute coronary syndromes in the global registry of acute coronary events. *Eur. Heart J.*, **31**(12):1449-1456. [doi:10.1093/eurheartj/ehq061]
- Markowicz-Pawlus, E., Nozynski, J., Sedkowska, A., *et al.*, 2007. Cardiac rupture risk estimation in patients with acute myocardial infarction treated with percutaneous coronary intervention. *Cardiol. J.*, **14**(6):538-543.
- Okino, S., Nishiyama, K., Ando, K., *et al.*, 2005. Thrombolysis increases the risk of free wall rupture in patients with acute myocardial infarction undergoing percutaneous coronary intervention. *J. Interv. Cardiol.*, **18**(3):167-172. [doi:10.1111/j.1540-8183.2005.04110.x]
- Pedrazzini, G., Santoro, E., Latini, R., *et al.*, 2008. Causes of death in patients with acute myocardial infarction treated with angiotensin-converting enzyme inhibitors: findings from the Gruppo Italiano per lo Studio della Sopravvivenza

- nell'Infarto (GISSI)-3 trial. *Am. Heart J.*, **155**(2):388-394. [doi:10.1016/j.ahj.2007.10.015]
- Pliam, M.B., Sternlieb, J.J., 1993. Intramyocardial dissecting hematoma: an unusual form of subacute cardiac rupture. *J. Card. Surg.*, **8**(6):628-637. [doi:10.1111/j.1540-8191.1993.tb00422.x]
- Pouleur, A.C., Barkoudah, E., Uno, H., et al., 2010. Pathogenesis of sudden unexpected death in a clinical trial of patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *Circulation*, **122**(6):597-602. [doi:10.1161/CIRCULATIONAHA.110.940619]
- Qian, G., Wang, Z.F., Liu, H.B., et al., 2011. Clinical manifestations of 43 patients with acute myocardial infarction complicated by free wall rupture. *Chin. J. Cardiovasc. Dis.*, **39**(9):812-815 (in Chinese).
- Qian, G., Liu, H.B., Wang, J.W., et al., 2013. Risk of cardiac rupture after acute myocardial infarction is related to a risk of hemorrhage. *J. Zhejiang Univ.-Sci. B (Biomed. & Biotechnol.)*, **14**(8):736-742. [doi:10.1631/jzus.B1200306]
- Shamshad, F., Kenchaiah, S., Finn, P.V., et al., 2010. Fatal myocardial rupture after acute myocardial infarction complicated by heart failure, left ventricular dysfunction, or both: the valsartan in acute myocardial infarction trial (VALIANT). *Am. Heart J.*, **160**(1):145-151. [doi:10.1016/j.ahj.2010.02.037]
- van den Borne, S.W., Cleutjens, J.P., Hanemaaijer, R., et al., 2009. Increased matrix metalloproteinase-8 and -9 activity in patients with infarct rupture after myocardial infarction. *Cardiovasc. Pathol.*, **18**(1):37-43. [doi:10.1016/j.carpath.2007.12.012]
- Vargas-Barrón, J., Roldán, F.J., Romero-Cárdenas, A., et al., 2013. Intramyocardial dissecting hematoma and postinfarction cardiac rupture. *Echocardiography*, **30**(1):106-113. [doi:10.1111/echo.12017]
- Yip, H.K., Wu, C.J., Chang, H.W., et al., 2003. Cardiac rupture complicating acute myocardial infarction in the direct percutaneous coronary intervention reperfusion era. *Chest*, **124**(2):565-571. [doi:10.1378/chest.124.2.565]

中文概要:

本文题目: 急性 ST 段抬高型心肌梗死继发心脏破裂的预测因素分析

Predictive factors of cardiac rupture in patients with ST-elevation myocardial infarction

研究目的: 心脏破裂是急性 ST 段抬高型心肌梗死最严重的并发症之一, 病情凶险, 死亡率高。通过明确心肌梗死后出现心脏破裂并发症的危险因素, 早期识别心脏破裂高危人群, 有利于在急性心肌梗死的早期采取干预措施以减少心脏破裂的发生。

创新要点: 既往缺乏对中国 ST 段抬高型心肌梗死继发心脏破裂的系统风险评估, 本研究采用回顾性队列研究的方法, 完善并健全中国多中心的急性心肌梗死数据库, 样本总量达到 9798 例, 为国内同类研究中样本量最多。既往的国外文献报道: 女性、高龄、心梗后就诊时间延迟、前壁心肌梗死、溶栓治疗等是心肌梗死后心脏破裂的独立危险因素。本研究在中国人群验证以上危险因素的基础上首次提出就诊时血色素下降及白细胞计数的升高与随后发生的心脏破裂密切相关。新的心脏破裂预测因素的存在有利于对心脏破裂更精细化的危险分层, 早期识别心脏破裂高危人群。

研究方法: 建立中国多中心心肌梗死数据库, 比较发生心脏破裂与未发生心脏破裂患者临床特点的差异, 筛选敏感特异的心脏破裂的独立预测因素。

重要结论: 中国人群急性 ST 段抬高型心肌梗死后心脏破裂的发生率是 1.82%, 独立预测因素主要包括: 高龄、女性、就诊时静息心率增快、前壁心肌梗死、心功能不全、就诊时间延迟、肾功能不全、贫血与白细胞计数升高。本文第一次提出就诊时贫血状态及白细胞计数的升高与心脏破裂的发生直接相关。

关键词: ST 段抬高型心肌梗死; 危险因素; 贫血; 心脏破裂