The Clinical Characteristics of Acute Myocardial Infarction with Ventricular Septal Perforation and the Prognosis Comparison of Different Treatment Methods

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Research article

Keywords: Acute myocardial infarction, Septal perforation, Risk factors, Prognosis

DOI: https://doi.org/10.21203/rs.3.rs-75606/v1

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Abstract

Background: To explore the clinical characteristics of acute myocardial infarction (AMI) with ventricular septal perforation (VSR) and the prognosis comparison of different treatment methods, as well as the analysis of related risk factors.

Methods: From January 2006 to February 2020, 29 patients with AMI and VSR diagnosed in the people's Hospital of Peking University were selected as the study object, among them, 16 cases were male (55.2%), 13 cases were female (44.8%), the average age was 64.69 ± 10.32 years old, and they were divided into survival group (n=16) and non-survival group (n=13) according to whether they survive within 30 days of surgical or drug conservative treatment. The clinical characteristics, coronary angiography and treatment of the two groups were summarized, and the prognosis and related risk factors were analyzed.

Results: There was no significant difference in the basic clinical characteristics between the two groups (P>0.05). Compared with the results of coronary angiography in the two groups, the proportion of the culprit vessel which was a simple anterior descending branch in the non-survival group was higher than that in the survival group, there was a statistical difference between the two groups (P<0.05). The peri-operative data of the two groups showed that the proportion of patients with complete revascularization, simultaneous bypass and recanalization of culprit vessels in the survival group was significantly higher than that in the non-survival group (P < 0.05). However, the incidence of postoperative low cardiac output and mortality during hospitalization in the survival group were significantly lower than those in the non-survival group (P < 0.05). Logistic regression analysis showed that complete revascularization (OR = 0.021, 95% CI 0.001-0.374, P = 0.009), recanalization of culprit vessels (OR = 0.045, 95% CI 0.004-0.548, P = 0.015) were independent risk factors for 30 day mortality. Kaplan-Meier survival curve showed that during the follow-up period, the long-term survival rate of patients with operation and complete revascularization was significantly higher than that of patients with drug conservative treatment and incomplete revascularization, there was a statistical difference between the two groups (P < 0.05).

Conclusions: Whether complete revascularization and recanalization of culprit vessels or not are independent risk factors for 30 day mortality in patients with AMI and VSR; the long-term survival rate of patients after surgery and complete revascularization is significantly higher than that of patients with conservative medical treatment and incomplete revascularization. Surgery and complete revascularization are important factors affecting the long-term prognosis of patients with AMI and VSR.

Background

VSR is a fatal complication after acute myocardial infarction (AMI); it's a great challenge to surgery, at present, the incidence of VSR is reported to be between 0.17% and 0.31% [1]. Although the prognosis of AMI patients has greatly improved in recent years, the overall prognosis of VSR patients is still poor. The 30-day mortality rate of VSR patients treated with drug conservative therapy is still close to 100%, and the
30-day mortality rate of patients treated with surgery is close to 50% [2-3]. This study conducted a retrospective analysis of 29 patients with AMI combined with VSR, aiming to explore and analyze the relevant risk factors affecting prognosis and guide clinical practice.

1 Data And Methods

1.1 Research object

From January 2006 to February 2020, 29 patients with AMI and VSR diagnosed in the Heart Surgery Department of the People's Hospital of Peking University were selected as the subjects of study, including 16 males (55.2%) and 13 females (44.8%), with an average age of 64.69 ± 10.32 years. Inclusion criteria: all patients had a clear history of severe chest pain; ST segment elevation ≥ 0.1mv (limb lead) or 0.2mv (chest lead) changes in at least two adjacent or related leads of ECG were performed in the external hospital or the People's Hospital of Peking University, and cardiac enzymes was increased; Physical examination found that systolic murmurs could be heard in the left side of the sternum 3 to 4 intercostal space; Echocardiography indicated that there was a clear left to right shunt in the ventricular level. Exclusion criteria: tumor patients or diseases that make patients in a cachexia state in a short period of time, or diseases progress rapidly, with life expectancy ≤ 6 months; combined with aortic wall hematoma or aortic dissection, aneurysm, arteriovenous malformation, etc; combined with cardiomyopathy, such as hypertrophic cardiomyopathy, etc.

1.2 Research method

Collect the general data (gender, age, etc.) of the patients, laboratory indexes (including myocardial injury markers, triglyceride (TG), cholesterol (Chol), low density lipoprotein (LDL) were collected, LDL), echocardiographic results (such as the location and size of the perforation, the end diastolic diameter of left ventricle, whether there is ventricular aneurysm, etc.), previous history (smoking history, hypertension history, old myocardial infarction history, diabetes history, etc.), coronary artery disease and perioperative situation, etc. Patients were divided into survival group (n = 16) and non-survival group (n = 13) according to whether they survived within 30 days after operation or drug conservative treatment. Summarize the clinical characteristics, coronary angiography and treatment of the two groups of patients, and analyze the prognosis and related risk factors.

1.3 Observation index

To record and compare the general data, hospital laboratory indexes, coronary angiography, treatment, 30-day mortality related risk factors and long-term survival rate between the two groups.

1.4 Statistical methods
SPSS 22.0 statistical software was used to process the data. The measurement data of normal distribution is represented by mean ± standard deviation (x ± s), and the comparison between groups is represented by t test; the measurement data of non normal distribution is represented by M (Q1, Q3), and the comparison between two groups is represented by non parameter test. The use case (percentage) of count data was expressed, and χ² test was used for comparison between the two groups. The influencing factors of 30-day mortality were analyzed by binary regression and Kaplan Meier (K-M) method. P < 0.05 was statistically significant.

2 Results

2.1 Comparison of general data between the two groups

There was no significant difference between the two groups in gender, age, BMI, smoking, hypertension and diabetes, previous myocardial infarction (P > 0.05); there was no significant difference in blood pressure, heart rate, cholesterol, triglyceride, low-density lipoprotein and high-density lipoprotein between the two groups (P > 0.05). See Table 1 for details
## Table 1
Patients' Characteristics

| Variables                  | Total, n = 29 | Survivors, n = 16(%) | Nonsurvivors, n = 13(%) | P value |
|----------------------------|---------------|-----------------------|--------------------------|---------|
| Age (y)                    | 64.69 ± 10.32 | 64.13 ± 11.15         | 65.38 ± 9.61             | 0.482   |
| Gender (Female)            | 13(44.8)      | 6(37.5)               | 7(53.8)                  | 0.467   |
| BMI                        | 24.23 ± 3.61  | 22.53 ± 3.23          | 26.33 ± 2.97             | 0.673   |
| Smoking history            | 14(48.3)      | 10(62.5)              | 4(30.8)                  | 0.139   |
| Systemic hypertension      | 16(55.2)      | 9(56.3)               | 7(53.8)                  | 1.0     |
| Diabetes mellitus II       | 11(37.9)      | 7(43.8)               | 4(30.8)                  | 0.702   |
| Hyperlipidemia             | 8(27.6)       | 5(31.3)               | 3(23.1)                  | 0.697   |
| P/h/o CAD                  | 7(24.1)       | 4(25.0)               | 3(23.1)                  | 1.0     |
| HR (beats per minute)      | 91.59 ± 20.23 | 91.88 ± 17.97         | 91.23 ± 23.46            | 0.934   |
| SBP (mmHg)                 | 111.45 ± 17.90| 106.75 ± 14.25        | 117.23 ± 20.68           | 0.118   |
| DBP (mmHg)                 | 73.31 ± 11.97 | 70.75 ± 12.26         | 76.46 ± 11.26            | 0.692   |
| LDL (mmol/L)               | 2.04(1.48, 2.57) | 1.87(1.44, 2.15)      | 2.37(1.62, 2.83)         | 0.125   |
| HDL (mmol/L)               | 0.79 ± 0.33   | 0.74 ± 0.27           | 0.85 ± 0.39              | 0.483   |
| Triglyceride (mmol/L)      | 1.20 ± 0.46   | 1.33 ± 0.54           | 1.04 ± 0.29              | 0.091   |
| Cholesterol (mmol/L)       | 3.32 ± 1.17   | 3.01 ± 0.96           | 3.69 ± 1.33              | 0.122   |

BMI: body mass index; CAD: coronary artery disease; P/h/o: past history of;
HR: Heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure;
LDL: Low density lipoprotein; HDL: High density lipoprotein;

* Statistically significant.

### 2.2 Comparison of myocardial enzymology, echocardiography and coronary angiography between the two groups

Comparing the myocardial enzyme indexes of the two groups, we found that there was statistical difference in creatine kinase (CK-MB) immediately after admission (P < 0.05), but no significant difference in other indexes (P > 0.05).
There was no significant difference in VSR perforation location, perforation size, ejection fraction and left ventricular end diastolic diameter between the two groups (P > 0.05).

Comparing the results of coronary angiography between the two groups, it was found that 75% (12 / 16) of the patients in the survival group had coronary angiography before operation, all of them were lesions of two or more coronary arteries; 61.5% (8 / 13) of the patients in the non-survival group had coronary angiography before operation, and 5 of them had simple lesions of anterior descending artery. The proportion of patients in the non-surviving group who had a simple anterior descending vessel was higher than that in the surviving group, and there was a statistical difference between the two (P < 0.05). See Table 2 for details.
| Variables                              | All, (n = 29) | Survivors, (n = 16) | Nonsurvivors, (n = 13) | P value |
|----------------------------------------|---------------|---------------------|------------------------|---------|
| Time from AMI to VSR (days)            | 5.0 (2.0, 10.0) | 6.0 (2.0, 13.25)    | 2.0 (1.5, 10.0)        | 0.478   |
| Time from AMI to VSR repair (days)     | 19.71 ± 13.34 | 22.38 ± 12.27       | 14.38 ± 14.61          | 0.22    |
| MYO                                    | 67.50 (21.05, 199.50) | 37.1 (20.48, 134.25) | 112.1 (24.45, 337.9)  | 0.236   |
| CK-MB                                  | 2.70 (1.14, 15.04) | 1.76 (1.10, 7.78)   | 4.40 (2.55, 34.58)     | 0.027*  |
| cTnI                                    | 0.46 (0.05, 5.40) | 0.20 (0.05, 5.02)   | 2.78 (0.28, 9.30)      | 0.136   |
| NYHA class (IV)                        | 11 (37.93)     | 6 (37.5)            | 5 (38.46)              | 1.0     |
| AMI location (inferior)                | 24 (82.76)     | 12 (75.0)           | 12 (92.31)             | 0.343   |
| EF at presentation (%)                 | 47.0 (41.35, 58.10) | 45.0 (35.55, 52.73) | 55.0 (42.95, 61.2)     | 0.125   |
| VSR location (Proximal apex)          | 25 (86.21)     | 13 (81.25)          | 12 (92.31)             | 0.606   |
| VSR Size (cm)                          | 1.15 ± 0.49    | 1.05 ± 0.50         | 1.27 ± 0.48            | 0.228   |
| LVED                                    | 5.70 (5.05, 6.20) | 5.65 (5.0, 5.90)    | 5.70 (5.10, 6.50)      | 0.263   |
| Ventricular aneurysm                   | 24 (82.76)     | 15 (93.75)          | 9 (69.23)              | 0.144   |
| Pulmonary artery pressure (mmHg)       | 45.0 (29.0, 60.0) | 45.0 (33.0, 68.0)   | 42.50 (11.75, 59.50)   | 0.412   |
| Mitral regurgitation (Whether or not)  | 19 (65.52)     | 10 (62.5)           | 9 (69.23)              | 1.0     |
| Tricuspid regurgitation (moderate to severe) | 11 (37.93) | 4 (25)              | 7 (53.85)              | 0.143   |
| Angiographic data                      | 5 (25)         | 0 (0)               | 5 (62.5)               | 0.003*  |

AMI: acute myocardial infarction; VSR: ventricular septal rupture;
MYO: Myoglobin; CK-MB: Creatine kinase; cTnI: Troponin I;
NYHA: New York Heart Association; EF: ejection fraction; LVED: Left ventricular end diastolic diameter; IABP: intraaortic balloon counter pulsation; ECMO: extracorporeal membrane oxygenator

* Statistically significant.
### 2.3 Comparison of perioperative data between the two groups

In the survival group, the proportion of patients undergoing coronary artery bypass, complete revascularization and revascularization of culprit vessels was much higher than that in the non-survival group \( (P < 0.05) \); the emergency operation rate in the survival group was lower than that in the non-survival group \( (P < 0.05) \); The survival time and long-term survival time of the patients in the survival group were significantly longer than those in the non-survival group, and there was a statistical difference between the two groups \( (P < 0.05) \); but the incidence of low cardiac output and death in the hospital in the survival group is lower than the non-surviving group, there is a significant statistical difference between the two \( (P < 0.05) \). See Table 3 for details

| Variables                | All, \( n = 29 \) | Survivors, \( n = 16 \) | Nonsurvivors, \( n = 13 \) | \( P \) value |
|--------------------------|-------------------|-------------------------|-----------------------------|--------------|
| IABP insertion           | 20(68.97)         | 11(68.75)               | 9(69.23)                    | 1.0          |
| ECMO support             | 2(0.07)           | 0 (0)                   | 2(0.15)                     | 0.192        |

AMI: acute myocardial infarction; VSR: ventricular septal rupture;
MYO: Myoglobin; CK-MB: Creatine kinase; cTnI: Troponin I;
NYHA: New York Heart Association; EF: ejection fraction; LVED: Left ventricular end diastolic diameter; IABP: intraaortic balloon counter pulsation; ECMO: extracorporeal membrane oxygenator

* Statistically significant.
Table 3
Perioperative data

| Variables                              | All, (n = 29) | survivors, (n = 16) | Nonsurvivors, (n = 13) | P value |
|----------------------------------------|---------------|--------------------|------------------------|---------|
| Technique of repair                    |               |                    |                        |         |
| Daivd                                  | 9(37.5)       | 5(31.25)           | 4(50)                  | 0.412   |
| Dagget                                 | 15(62.5)      | 11(68.75)          | 4(50)                  | 0.412   |
| Concomitant CABG                       | 18(75)        | 15(93.75)          | 3(37.5)                | 0.007*  |
| Urgent operation                       | 13(54.17)     | 6(37.5)            | 7(87.5)                | 0.033*  |
| Complete revascularization             | 19(65.52)     | 15(93.75)          | 4(30.77)               | 0.001*  |
| Criminal recanalization                | 19(65.52)     | 15(93.75)          | 4(30.77)               | 0.001*  |
| Aortic cross-clamp time(min)           | 172.0(91.0,226.0) | 170.0(128.75,241.25) | 172.0(0.0,219) | 1.0     |
| Cardiopulmonary bypass time(min)      | 115.0(50.0,153.5) | 117.5(63.0,171)    | 69.0(0.0,147)          | 1.0     |
| CRRT (Whether or not)                 | 11(45.83)     | 5(31.25)           | 6(75)                  | 0.082   |
| Postoperative VIS                      | 6(25)         | 5(31.25)           | 1(12.5)                | 0.621   |
| Duration of mechanical ventilation (hrs) | 135.50(24.0,397.50) | 131.5(15.75,467.75) | 91.0(0.0,179) | 0.783   |
| Length of ICU stay (days)              | 8.0(3.0,18.75) | 8.0(3.0,25.75)     | 3.0(0.0,8.5)           | 0.58    |
| Length of hospital stay(days)          | 23.0(14.5,35.0) | 33.50(27.25,59.0)  | 14.0(5.0,15.5)         | 0.001*  |
| The survival time of the patients(months) | 2.7(0.50,21.42) | 16.98(12.0,73.63)  | 0.50(0.17,0.52)        | 0.001*  |
| Postoperative low cardiac output syndrome | 6(25)        | 0(0)               | 6(75)                  | 0.001*  |
| Death in hospital                      | 10(34.48)     | 0(0)               | 10(76.92)              | 0.001*  |
| Open the chest twice                   | 4(13.79)      | 3(18.75)           | 1(7.69)                | 0.606   |
| Tracheotomy                            | 6(20.69)      | 5(31.25)           | 1(7.69)                | 0.183   |

CABG: coronary artery bypass grafting; ICU: intensive care unit; VIS: Vasoactive-Inotropic Score; CRRT: continuous renal replacement therapy

* Statistically significant.
| Variables                                      | All, \(n = 29\) | survivors, \(n = 16\) | Nonsurvivors, \(n = 13\) | \(P\) value |
|-----------------------------------------------|-----------------|-----------------------|---------------------------|-------------|
| Operative bleeding volume > 400 ml            | 20(83.33)       | 14(87.5)              | 6(75)                     | 0.578       |

CABG: coronary artery bypass grafting; ICU: intensive care unit; VIS: Vasoactive-Inotropic Score; CRRT: continuous renal replacement therapy

* Statistically significant.

### 2.4 Regression analysis of risk factors influencing 30-day mortality

Logistic regression analysis showed that complete revascularization (or = 0.021, 95% CI 0.001–0.374, \(P = 0.009\)) and recanalization of culprit vessels (or = 0.045, 95% CI 0.004–0.548, \(P = 0.015\)) were independent risk factors for 30-day mortality. See Table 4 for details.

| Table 4                                                                 |
|------------------------------------------------------------------------|
| Associated predictors of 30-day mortality in patients                  |
| Variables                                      | OR value | the 95% confidence interval | \(P\) value |
| Gender                                        | .719     | 0.076 ~ 6.793               | .773       |
| Age                                           | 1.008    | 0.907 ~ 1.120               | .886       |
| Smoking(Whether or not)                        | 1.972    | 0.136 ~ 28.501              | .618       |
| Complete revascularization                     | 0.021    | 0.001 ~ 0.374               | 0.009*     |
| Criminal recanalization                        | 0.045    | 0.004 ~ 0.548               | 0.015*     |
| AMI location (inferior)                        | 3.552    | 0.328 ~ 38.495              | .297       |

* Statistically significant.

### 2.5 The long-term follow-up of the patients

Telephone follow-up was conducted on surviving patients in the hospital, and the follow-up time was from 3 months to 136 months. The K-M survival curve showed that during the follow-up period, the long-term survival rate of patients after surgery and complete revascularization was significantly higher than that of patients with conservative drug treatment and incomplete revascularization, and the difference was statistically significant (\(P < 0.05\)).
3 Discussion

AMI with VSR is a rare but dangerous complication, which often occurs in acute ST segment elevation myocardial infarction \(^4\). VSR mostly occurs within 1 week after AMI. The patient has a sudden left ventricular shunt at the ventricular level, increased cardiac load, and rapid heart failure, cardiogenic shock, etc., which in turn causes severe pulmonary congestion and/or hemodynamic deterioration. The effect of conservative drug treatment is extremely poor. At the same time, due to the poor texture of myocardial tissue, the difficulty of suture and the high incidence of low cardiac output after myocardial infarction, surgical operations also face great challenges. In this study, the clinical characteristics and different treatment methods of patients with AMI combined with VSR were compared, the relevant risk factors affecting the 30-day mortality of the patients were analyzed, and the long-term survival rates of the patients were compared.

Previous studies have found that the risk factors of AMI combined with VSR include: old age, female, smoking, combined history of hypertension, anterior wall or large area myocardial infarction, lack of good coronary collateral circulation, etc. \(^5-6\). In this study, there was no significant difference between the two groups of patients in terms of age, gender, smoking, history of hypertension, anterior myocardial infarction, and previous myocardial infarction (P>0.05).

Early diagnosis of echocardiography in AMI patients to diagnose and guide clinical treatment is of great value to the prognosis of patients. Echocardiography can be used to determine the location, size, end diastolic diameter of left ventricle, whether there is ventricular aneurysm, pulmonary artery pressure and whether there is bicuspid valve regurgitation, etc, and to preliminarily evaluate the cardiac function and prognosis of patients \(^7\). In this study, echocardiography showed that most of the perforations were located near the apex of the interventricular septum, but there was no significant difference between the two groups in the location, size, ejection fraction, end diastolic diameter of the left ventricle, whether the VSR was associated with aneurysm, pulmonary artery pressure and whether the bicuspid or tricuspid valve returned (P > 0.05).

If the condition of the VSR patient allows, complete coronary angiography before surgery can determine the relevant coronary artery disease, determine whether to perform coronary artery bypass grafting during the same period and determine the corresponding target vessel site. Some studies suggest that if the stenosis degree of the main coronary artery and its branches is more than 50% and the blood supply area is non-infarcted area, coronary artery bypass grafting should be performed at the same time \(^8,9\). Previous literature studies have found that VSR is mostly single vessel disease, and the infarct related vessels are mostly anterior descending \(^10\). We compared the coronary angiography results of the two groups of patients, and found that the proportion of patients in the survival group who underwent coronary angiography before surgery was 75% (12/16), all of which were coronary vascular lesions of 2 or more; The ratio of pulse angiography was 61.5% (8/13), including 5 cases of anterior descending branch lesions alone. The proportion of patients in the non-surviving group who had a simple anterior descending vessel was higher than that in the surviving group, and there was a statistical difference.
between the two groups (P < 0.05), which is consistent with the results of previous studies, and most of the infarct-related blood vessels are completely occluded, lacking effective collateral circulation, the infarct size is large, the degree of myocardial ischemia in the patient is more serious than that in the survival group, and eventually the 30-day mortality rate of the patient is more obvious than that in the survival group Rise. Therefore, patients with VSR should actively improve coronary angiography before surgery to clarify coronary vascular lesions to decide whether to perform coronary artery bypass grafting during the same period.

Surgical operation is an effective method to reduce the hospital mortality of VSR patients, especially for the patients with complicated condition and large perforation area, and it is difficult to intervene through intervention means. Due to the high risk of ventricular septal perforation, the timing of surgery is particularly important. However, when is the best operation time for VSR has always been controversial. Previous studies have suggested that the shorter the time from diagnosis to operation, the higher the operative mortality rate. Papalexopoulou and other researchers believe that under the condition of stable hemodynamics, surgery should be delayed as much as possible; however, current European guidelines recommend that patients with VSR should undergo surgery as soon as possible to reduce the incidence of heart failure. In the early period of perforation, the edges of the perforation were unclear and the surrounding tissues were brittle, which increased the difficulty of surgical repair and the risk of repair failure; and the purpose of delayed surgery was to reduce the difficulty of repair. It may cause some patients to die due to the worsening of circulatory conditions while waiting for surgery, and lose the chance of surgery. Amaoutakis et al.’s large sample study found that the mortality of surgery within 1 week of AMI combined with VSR was 54.1%, while the mortality of AMI combined with VSR for more than 1 week before surgery was only 18.4%. We compared the perioperative data of the two groups and found that the emergency operation rate of the survival group was lower than that of the non-survival group, there was a statistical difference between the two groups (P < 0.05); from the diagnosis of VSR to the operation time, the average time of the survival group was 22.38 ± 12.27 days, the average time of the non-survival group was 14.38 ± 14.61 days, although there was no significant statistical difference between the two groups (P > 0.05), however, the time from the diagnosis of the VSR to the operation in the survival group was significantly longer than that in the non-survival group, and the emergency surgery rate of the survival group was significantly lower than that of the non-survival group. The above two points further confirmed the importance of the timing of VSR surgery.

Previous studies have shown that the rapid deterioration of preoperative circulatory state is an important risk factor for poor perioperative prognosis of patients, while critical preoperative state is closely related to the time of perforation after myocardial infarction, the degree of interventricular septum involvement and perforation. In general, VSR usually occurs in the first week after AMI, with an average time of 3-5 days. This study shows that the overall average time of perforation is 5 days (2.0, 10.0). Compared with the time from AMI to VSR diagnosis in the two groups, the average time of survival group is 6 days (2.0, 13.25), while the average time of non-survival group is 2 days (1.5, 10.0), Although
there was no significant statistical difference between the two groups (P>0.05), it also supported the previous point to some extent.

Previous studies have found that complete revascularization and revascularization of culprit vessels in VSR patients can reduce 30-day mortality and improve long-term prognosis\textsuperscript{[20,21,22]}. Lundblad et al. also found that simultaneous bypass during operation can reduce the early and long-term mortality of VSR patients\textsuperscript{[23]}; perotta et al. also reported that simultaneous bypass can reduce the mortality of VSR patients\textsuperscript{[24]}. In this study, we found that the proportion of patients in the survival group who underwent coronary artery bypass, complete revascularization and culprit revascularization at the same time was much higher than that in the non-survival group, and there was a significant statistical difference between the two groups (P < 0.05), which was consistent with the results reported in the previous studies, and fully reflected the importance of the simultaneous revascularization and culprit revascularization in patients with AMI and VSR during the operation. Logistic regression analysis showed that complete revascularization (or = 0.021, 95% confidence interval 0.001-0.374, P = 0.009) and recanalization of culprit vessels (or = 0.045, 95% confidence interval 0.004-0.548, P = 0.015) were independent risk factors for 30-day mortality of VSR patients. Telephone follow-up of surviving patients in the hospital was conducted between 3 months and 136 months. The KM survival curve shows that during the follow-up period, the long-term survival rate of patients after surgical operation and complete revascularization is significantly higher than that of conservative medical treatment and patients who have not undergone complete revascularization despite surgical treatment. There are obvious statistics between the two groups of patients Academic differences (P <0.05). The results of this study further confirm the previous research. The reason may be that complete revascularization and revascularization of culprit vessels can effectively improve the blood supply of myocardial tissue, thus reducing the 30-day mortality rate of VSR patients and improving the long-term prognosis, but there is no retrospective study to confirm the direct causal relationship between them. If we want to confirm this, we need to conduct a large-scale prospective control trial, and the patients in the control group must deliberately ignore the same period of coronary artery bypass grafting, it is obvious that this prospective control trial is never possible\textsuperscript{[25]}. From the K-M survival curve, it can be seen that the long-term prognosis of VSR patients treated by surgery and complete revascularization is better than that of patients treated by drug conservative therapy and surgery but not complete revascularization (P < 0.05). It is also confirmed once again that drug treatment can only be used as a transitional treatment for the VSR patients, but not as an independent treatment. Surgical surgery is still the preferred treatment option for patients with AMI and VSR.

In summary, our study found that whether complete revascularization and revascularization of criminals are independent risk factors that affect the 30-day mortality rate in patients with AMI and VSR; surgery and complete revascularization can significantly improve the hospital survival rate and the long-term prognosis of the VSR patients. Since this study is a single-center retrospective study, it is inevitable that there is a certain bias, and the sample size is small, and the statistical power will also be affected to a certain extent. In the future, a larger sample size and longer follow-up time for multiple centers will be needed to further validate our findings.
4 Limitations

Our study had several limitations. Due to its retrospective design, we were unable to certify that all potential confounding factors had been recorded. Our single-centre experience may not be applicable to other institutes.

5 Conclusion

Whether complete revascularization and recanalization of culprit vessels or not are independent risk factors for 30 day mortality in patients with AMI and VSR, the long-term survival rate of patients after surgery and complete revascularization is significantly higher than that of patients with conservative medical treatment and incomplete revascularization. Surgery and complete revascularization are important factors affecting the long-term prognosis of patients with AMI and VSR.

Abbreviations

AMI: acute myocardial infarction
VSR: ventricular septal perforation
TG: triglyceride
Chol: cholesterol
LDL: low density lipoprotein
CK-MB: creatine kinase

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

We received explicit consent from the patient

Availability of data and material

Data will be made available on request
Competing interests
The authors declare that they have no competing interests

Funding
This research was supported by the Research and Development Fund of the Peking University People's Hospital, Fund Approval Number RDY2019-32. The funding source (RDY2019-32) had no role in the study design, data collection, analysis, or interpretation. The authors declare that there are no potential conflicts of interest.

Authors' contributions
Zhou Zhao is the corresponding author. Chun Fu drafted the manuscript, Jian Liu and Youzhong An provided valuable statistical analysis for this study and participated in its design and coordination. All authors read and approved the final manuscript.

Acknowledgements
Not applicable

References
1. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. Circulation. 2000; 101:27-32.
2. Coskun KO, Coskun ST, Popov AF, et al. Experiences with surgical treatment of ventricle septal defect as a post infarction complication. J Cardiothorac Surg. 2009;4:3.
3. Pang PY, Sin YK, Lim CH, et al. Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. J Cardiothorac Surg. 2013;8:44
4. Shao Ying, Liu Yin. Characteristics and treatment progress of ventricular septal perforation after acute myocardial infarction–J. Tianjin Pharmaceuticals 2017,45:1149-1152.
5. Moreyra AE, Huang MS, Wilson AC, et al. Trends in incidence and mortality rates of ventricular septal rupture during acute myocardial infarction–J Am J Cardiol 2010, 106:1095-1100.
6. Lopez-Sendon 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 [J]. Eur Heart J, 2010, 31(12):1449-1456.
7. GUERET P, KHALIFE K, JOBIC Y, et al. Echocardiographic assessment of the incidence of mechanical complications during the early phase of myocardial infarction in the reperfusion era—a French
muhieentre prospective registry[J]. Archives of Cardiovascular Diseases 2008;101(1):41-47

8. Labrousse L, Choukroun E, Chevalier J et al. Surgery for postinfarction ventricular septal defect (VSD): risk factors for hospital death and long term results[J]. Eur J Cardiothorac Surg 2002; 21(4):725-732.

9. Prêtre R, Ye Q, Grinenfelder J et al. Role of myocardial revascularization in postinfarction ventricular septal rupture[J]. Ann Thorac Surg 2000;69(1):51-55.

10. Hayashi T, Hirano Y, Takai H et al. Usefulness of ST—segment elevation in the inferior leads in predicting ventricular septal rupture in patients with anterior wall acute myocardial infarction[J]. Am J Cardiol 2005;96(8):1037—1041.

11. Noguchi K, Yamaguchi A, Naito K, Yuri K, Adachi H. Short-term and long-term outcomes of postinfarction ventricular septal perforation. Gen Thorac Cardiovasc Surg 2012;60:261—7.

12. Papalexopoulou N, Young CP, Attia RQ. What is the best timing of surgery in patients with post-infarct ventricular septal rupture? Interact Cardiovasc Thorac Surg. 2013;16(2):193-196. doi:10.1093/icvts/ivs444.

13. O’Gara PT, Kushner FG, Ascheim DD, Casey Jr DE, Chung MK, De Lemos JA, et al. 2013 ACCF/AHA Guideline for the management of ST-elevation myocardial infarction. Circulation 2013;127:e362—425.

14. Ryan TJ, Antman EM, Brooks NH, Califf RM, Hillis LD, Hiratzka LF, et al. 1999 update: ACC/AHA guidelines for the management of patients with acute myocardial infarction. J Am Coll Cardiol 1999;34:890—911.

15. Muehrcke DD, Daggett WM Jr, Buckley MJ, Akins CW, Hilgenberg AD, Austen WG. Postinfarct ventricular septal defect repair: effect of coronary artery bypass grafting. Ann Thorac Surg. 1992;54:876-82.

16. Maoutakis G, Zhang Y, Geoh TJ et al. Surgical repair of ventricular septal defect in the post-infarct era: myocardial infarction outcomes from the society of thoracic surgeons national database[J]. Ann Thorac Surg. 2012;94(2):436—444.

17. Cerin G. Surgical treatment of ventricular septal defect complicating acute myocardial infarction. Experience of a north Italian referral hospital. Cardiovasc Surg. 2003;11(2):149-154. doi:10.1016/s0967-2109(02)00190-4

18. Menon V, Webb JG, Hillis LD, et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: A report from the SHOCK Trial Registry. J Am Coll Cardiol. 2000;36(3 SUPPL. A):1110-1116.

19. Poulsen SH, Praestholm MM, Munk K et al. Ventricular septal rupture complicating acute myocardial infarction: clinical characteristics and contemporary outcome[J]. Ann Thorac Surg. 2008;85(5):1591—1596.

20. Barker TA, Ramnarine IR, Woo EB, Grayson AD, Au J, Fabri BM, et al. Repair of post-infarct ventricular septal defect with or without coronary artery bypass grafting in the northwest of England: a 5-year multi-institutional experience. Eur J Cardiothorac Surg. 2003;24:940-6.
21. Pretre R, Ye Q, Grunenfelder J, Zund G, Turina MI. Role of myocardial revascularization in postinfarction ventricular septal rupture. Ann Thorac Surg. 2000;69:51-5.

22. Cox FF, Plokker HW, Morshuis WJ, Kelder JC, Vermeulen FE. Importance of coronary revascularization for late survival after postinfarction ventricular septal rupture. A reason to perform coronary angiography prior to surgery. Eur Heart J. 1996;17:1841-5.

23. Lundblad R, Abdelnoor M, Geiran OR, Svennevig JL. Surgical repair of postinfarction ventricular septal rupture: risk factors of early and late death. J Thorac Cardiovasc Surg. 2009;137(4):862–8.

24. Perotta S, Lentini S. In patients undergoing surgical repair of post-infarction ventricular septal defect, does concomitant revascularization improve prognosis? Interact Cardiovasc Thorac Surg. 2009;9(5):879–87.

25. Runar Lundblad, MD, PhD, Michel Abdelnoor, PhD, Surgical repair of postinfarction ventricular septal rupture: Risk factors of early and late death. The Journal of Thoracic and Cardiovascular Surgery April 2009.

Figures
Figure 1

Comparison of long-term survival rate between the operating group and the non-operating group
Figure 2

Comparison of long-term survival rate between patients with complete revascularization and those without revascularization