Original Article

Outcomes of surgical repair of anterior or posterior ventricular septal rupture after myocardial infarction

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Background: Ventricular septal rupture (VSR) is a rare and fatal complication of myocardial infarction. Surgery is the main treatment for the condition. It is currently believed that surgery is less effective for posterior VSR than for anterior VSR. The objective of this study was to investigate the clinical outcomes of surgical treatment for myocardial infarction combined with an anterior or posterior VSR.

Methods: This was a single-center, retrospective, observational, cohort study. Clinical data of 68 patients with myocardial infarction combined with VSR were retrospectively analyzed. According to the site of the VSR, patients were divided into the anterior (43 cases) and posterior (25 cases) VSR groups, and the general clinical data, preoperative examination results, surgery, and follow-up results were compared between the two groups.

Results: Compared with the anterior VSR group, the operative time in the posterior VSR group was longer {300 [240, 360] *vs.* 360 [300, 400] min; P=0.003}, and the cardiopulmonary bypass time was longer (142.0±52.2 *vs.* 180.2±52.3 min; P=0.005), and the aortic clamp time was longer (84.0±32.5 *vs.* 115.9±39.8 min; P=0.001). There were no significant differences in the incidence of perioperative complications, including bleeding, low cardiac output, pulmonary, and cerebrovascular complications, and the incidence of perioperative death between the two groups (P>0.05). The patients were followed up for 1.0–10.5 (median, 4.2) years. There were no significant differences in the survival rate and the incidence of major adverse cardiovascular and cerebrovascular events (MACCEs) including myocardial infarction, heart failure, revascularization, and cerebrovascular events between the two groups (P>0.05).

Conclusions: The perioperative risks and medium- and long-term outcomes of the surgical repair of anterior or posterior VSR after myocardial infarction were similar.

Keywords: Myocardial infarction; ventricular septal rupture (VSR); surgical repair

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Introduction

Ventricular septal rupture (VSR) is a rare and fatal complication of myocardial infarction (1). In recent years, the development of drug percutaneous coronary interventions and surgical techniques has improved the prognosis of myocardial infarction, but patients with extensive myocardial infarction who do not receive timely revascularization are still at risk of VSR, which has an incidence of about 0.3% (2). Among these patients, anterior VSR is more common (66–78%), while posterior VSR accounts for about 17–22% of cases (3). Conservative

treatment of VSR with drugs is not effective, and surgery is the recommended treatment (4). At present, it is generally considered that the surgical repair of posterior VSR is more challenging than that of anterior VSR (4-6). This study retrospectively analyzed the data of surgically treated patients with myocardial infarction combined with VSR and discusses the methods of treatment and the impact of the different VSR sites after myocardial infarction. We present the following article in accordance with the STROBE reporting checklist (available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-577/rc).

Methods

Patient enrollment

This was a single-center, retrospective, observational, cohort study. The clinical data of 68 consecutive patients in Beijing Anzhen Hospital who received surgical treatment for myocardial infarction with VSR from January 2009 to January 2020 were analyzed. The inclusion criteria were: (I) meeting myocardial infarction diagnostic criteria (7): abnormally raised cardiac troponin (cTn) level measured by at least one laboratory test, together with clinical evidence of myocardial ischemia; (II) echocardiography suggesting the interruption of ventricular septal echo continuity or left ventricular angiography showing left-to-right shunt. The exclusion criteria were: (I) concurrent combined free wall rupture; (II) history of interventional closure of percutaneous VSR; (III) history of a malignant tumor.

VSR site determination

According to the 17-segments model recommended by the American Society of Ultrasound to locate the rupture position (8), VSR located in the middle segment of the anterior septum, the basal segment of the anterior septum, and the apical segment of the ventricular septum was defined as an anterior VSR, while VSR in the remaining parts was defined as a posterior VSR. The patients were divided into an anterior VSR group (43 cases) and a posterior VSR group (25 cases). Clinical baseline data, surgical data, perioperative complications, and follow-up results were analyzed in both groups. All data were obtained from an online hospital database, and all the imaging data were evaluated by experienced cardiologists, cardiac surgeons and echocardiologists with the position of associate chief physician or above. The baseline data of the two groups are shown in *Table 1*.

Follow-up

The patients were followed up via outpatient consultations, phone, or WeChat for 1.0–10.5 (median, 4.2) years and the incidence of major adverse cardiovascular and cerebrovascular events (MACCEs), including all-cause death, myocardial infarction, heart failure, revascularization, and cerebrovascular events, were recorded. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Institutional Ethics Committee of Beijing Anzhen Hospital (No. 2019070X) and informed consent was obtained from all the patients.

Surgical technique

All surgeries were performed with a median thoracotomy, establishment of cardiopulmonary bypass, moderate hypothermia, aortic cross clamp, and perfusion with cardioplegic fluid. If coronary artery bypass grafting (CABG) was planned, it was performed prior to VSR repair to better achieve myocardial protection and minimize cardiac management after VSR repair. It is common to use the great saphenous vein as conduit (5).

The septal rupture repair is usually exposed through an incision in the infarcted left ventricular myocardium or ventricular aneurysm but may also be accessed through an incision in the right ventricle or atrium (9,10). In this study, the location of the anterior VSR incision was parallel to the anterior descending branch 1–2 cm in the position of the ventricular aneurysm, and the large diagonal branch was carefully protected. The posterior VSR requires the heart to be elevated to be fully exposed. The incision was made at 1–2 cm parallel to the posterior descending branch in the left ventricle infarction site. The posterior descending branch is close to the posteromedial papillary muscle, and the submitral device needs to be protected. The selection of such incision allows the most direct visualization of the VSR, and it is easier to perform the resection of the ventricular aneurysm.

Some patients received direct VSR patch repair (Dagget) (11) due to evident fibrosis of the rupture site. The majority of VSR patients, however, were subjected to the classical infarct exclusion technique (David) (12) or the modified David procedure (13,14). Bovine pericardium mesh of appropriate size was selected to better preserve

Table 1 Preoperative baseline data of 68 patients with myocardial infarction complicated with VSR

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value
Age (years)	64.8±8.3	63.4±8.1	0.483
≥65-year-old	23 (53.5)	12 (48.0)	0.662
Male*	22 (51.2)	20 (80.0)	0.018*
BMI	24.3±2.7	23.7±2.8	0.381
Medical history			
Hypertension	18 (41.9)	14 (56.0)	0.260
Diabetes	15 (34.9)	6 (24.0)	0.349
Hyperlipidemia	35 (81.4)	14 (56.0)	0.265
Renal insufficiency	32 (74.4)	15 (60.0)	0.215
COPD	2 (4.7)	0 (0.0)	0.274
Peripheral vascular disease	6 (14.0)	5 (20.0)	0.514
Cerebrovascular event	6 (14.0)	3 (12.0)	0.819
Myocardial infarction	13 (30.2)	9 (36.0)	0.624
PCI	11 (25.6)	7 (28.0)	0.827
Smoking*	15 (34.9)	15 (60.0)	0.044*
Echocardiography			
LVEF (%)*	46.9±8.1	52.8±8.7	0.009*
LVEDD (mm)	52.5±5.8	53.8±5.5	0.375
LVESD (mm)	36.3±5.5	36.8±3.9	0.723
VSR diameter (mm)	12.7±7.2	14.1±6.1	0.426
SPAP (mmHg)	50.2±11.3	45.5±17.7	0.276
Coronary arteriography			
LAD lesion	42 (97.7)	21 (84.0)	0.057
LCX lesions	18 (41.9)	8 (32.0)	0.420
RCA lesions*	17 (39.5)	21 (84.0)	<0.001*
Killip classification			0.937
Class I	0 (0.0)	0 (0.0)	
Class II	28 (65.1)	16 (64.0)	
Class III	11 (25.6)	6 (24.0)	
Class IV	4 (9.3)	3 (12.0)	
Preoperative status			
MI-to-operation time (days)	36.0 [18.0, 71.0]	64.0 [24.5, 89.0]	0.173
VSR-to-operation time (days)	31.0 [13.0, 56.0]	43.0 [19.0, 63.0]	0.340
VSR-to-operation time ≤7 days	6 (14.0)	3 (12.0)	0.820
VSR-to-operation time >7 days	37 (86.0)	22 (88.0)	0.887

Table 1 (*continued*)

Table 1 (*continued*)

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value
Cardiogenic shock	10 (23.3)	3 (12.0)	0.413
Pulmonary edema	19 (44.2)	6 (24.0)	0.096
Pulmonary infection	5 (11.6)	2 (8.0)	0.638
Vasoactive agent	15 (34.9)	8 (32.0)	0.809
CRRT	1 (2.3)	2 (8.0)	0.275
IABP	13 (30.2)	6 (24.0)	0.581
ECMO	0 (0.0)	0 (0.0)	–
Ventilator support	3 (7.0)	0 (0.0)	0.177
Emergency surgical status	10 (23.3)	4 (16.0)	0.476

Data are n (%), mean ± standard deviation, or median [first quartile, third quartile]. *, anterior VSR *vs.* posterior VSR, P<0.05. VSR, ventricular septal rupture; BMI, body mass index; COPD, chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; SPAP, systolic pulmonary artery pressure; LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery; MI, myocardial infarction; CRRT, continuous renal replacement therapy; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation.

the left ventricular geometry. The pericardium was sutured to the lower part of the non-infarcted myocardium in the ventricular septum and the non-infarcted myocardium in the anterior wall of the left ventricle to isolate the left ventricle from the infarcted myocardium, and the fragile tissue was continuously reinforced and sutured. Finally, two pieces of Teflon felt were used to close the left ventricular incision. Modified infarct exclusion technology allows better preservation of the left ventricular geometry and effectively reduces the rate of postoperative residual shunt recurrence.

Statistical analysis

Categorical variables were expressed as frequencies and percentages, and the comparison between groups was performed using Pearson's chi-square test or Fisher's exact test; the rank-sum test was used for ranking variables. The distribution of continuous variables was tested using the Shasharo-Wilk test, and the F-test was used for homogeneity of variance. Continuous variables showing normal distribution and homogeneity of variance were expressed as means and standard deviations, and independent-sample Student's *t*-tests were used for between-group comparisons. Non-normally distributed continuous variables were represented as medians and quartiles, and the Mann-Whitney U test was used for comparisons between two groups. The baseline characteristics are shown in *Tables 1-3*, with differences in univariate analyses in binary logistic regression to calibrate the risk factors affecting perioperative complications and death. Meanwhile, univariate Cox regression analysis was performed on risk factors potentially affecting medium and long-term prognosis, and risk factors with P values <0.2 were included in multivariate Cox regression analysis to calibrate for hazard ratios (HRs) for end-point events. All tests were two-tailed, and a P value <0.05 was considered significant. SPSS version 22.0 (IBM, Armonk, NY, USA) software was used for the statistical analysis of data.

Results

From January 2009 to January 2020, a total of 139 patients diagnosed with myocardial infarction complicated with VSR were admitted to Beijing Anzhen Hospital. Sixty-eight of these patients were included in this study. Thirty patients were excluded because of preoperative death due to rapid hemodynamic deterioration, four patients as they underwent percutaneous ventricular septal intervention, 21 patients due to their advanced age and the presence of complications, while 16 patients were excluded as they refused surgery because of cost or other reasons. Thus, a total of 68 patients undergoing surgical repair of VSR were included, including 43 patients with anterior VSR and 25 patients with posterior VSR (*Figure 1*).

Table 2 Surgical data of 68 patients with myocardial infarction complicated with VSR

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value
Operation time (min)*	300 [240, 360]	360 [300, 400]	0.003*
CPB time (min)*	142.0±52.2	180.2±52.3	0.005*
Aortic clamp time (min)*	84.0±32.5	115.9±39.8	0.001*
Combined CABG	36 (83.7)	23 (92.0)	0.548
Number of grafts*	2 [1, 2]	2 [1, 3]	0.037*
Combined aneurysm resection	38 (88.4)	17 (68.0)	0.082
Combined mitral valve surgery	2 (4.7)	4 (16.0)	0.251
Combined tricuspid valvuloplasty	7 (16.3)	5 (20.0)	0.954

Data are n (%), mean ± standard deviation, or median [first quartile, third quartile]. *, anterior VSR *vs.* posterior VSR, P<0.05. VSR, ventricular septal rupture; CPB, cardiopulmonary bypass; CABG, coronary artery bypass grafting.

Table 3 Postoperative data of 68 patients with myocardial infarction complicated with VSR

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value
Erythrocytes (units)	6 [3, 16]	8 [3, 12]	0.505
Plasma (mL)	400 [0, 800]	800 [100, 1,100]	0.388
Platelets (units)	0 [0, 2]	0 [0, 2]	0.800
ICU stay time (hours)	68.0 [38.0, 111.0]	73.0 [43.0, 104.5]	0.638
Ventilator use time (hours)	42.0 [21.0, 76.0]	50.0 [26.3, 89.0]	0.288
Drainage volume within 24 h after surgery (mL)	335 [173, 678]	370 [185, 625]	0.655
Postoperative hospital stay (day)	10.0 [6.0, 14.0]	8.0 [6.5, 10.0]	0.362
Postoperative echocardiography			
LVEF (%)	47.6±8.4	48.3±5.7	0.101
LVEDD (mm)	48.5±6.1	51.7±4.4	0.345
LVESD (mm)	35.9±7.0	37.4±4.3	0.216
SPAP (mmHg)	29.8±6.6	26.8±4.9	0.268
Residual VSR shunt	10 (23.3)	2 (8.0)	0.207
Shunt diameter (mm)	0 [0, 1]	0 [0, 0]	0.112

Data are n (%), mean ± standard deviation, or median [first quartile, third quartile]. VSR, ventricular septal rupture; ICU, intensive care unit; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; SPAP, systolic pulmonary artery pressure.

The mean age of patients with VSR after myocardial infarction was 64.3±8.2 years. There was no statistically significant difference in age between the anterior and posterior VSR groups (64.8±8.3 *vs.* 63.4±8.1 years; P=0.483), and there was no significant difference in the proportion of patients older than 65 years old between the two groups [23 (53.5%) *vs.* 12 (48.0%); P=0.662].

Emergency surgery due to hemodynamic instability was performed within 7 days after VSR in 13.2% (9/68) of patients. There was no significant difference in the proportion of patients undergoing emergency surgery between the anterior and posterior VSR groups [6 (14.0%) *vs.* 3 (12.0%); P=0.200]. The overall perioperative mortality within 7 days of VSR was 44.4% (4/9), and there was no

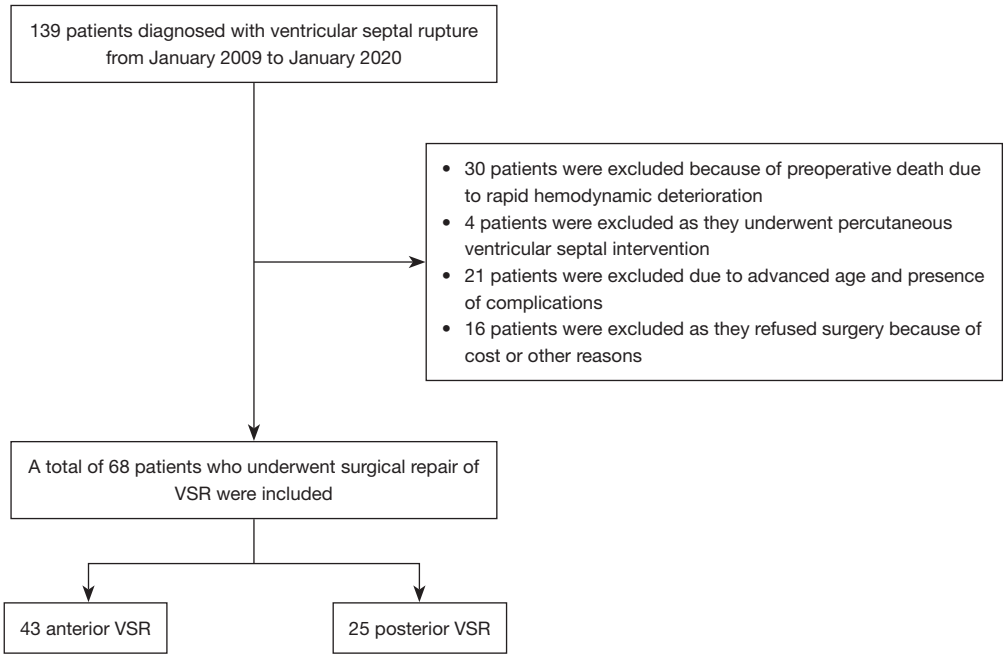

Figure 1 Flow chart of participants in the present study. VSR, ventricular septal rupture.

significant difference between the anterior and posterior VSR groups [2 (33.3%) *vs.* 2 (66.6%); P=0.813]. A high proportion, 86.8% (59/68), of patients underwent surgical treatment more than 7 days after VSR, and this fraction was not significantly different between the anterior and posterior VSR groups [37 (86.0%) *vs.* 22 (88.0%); P=0.887]. The perioperative mortality in patients that underwent surgery more than 7 days after VSR was 8.5% (5/59), and there was no significant difference between the anterior and posterior VSR groups [4 (10.8%) *vs.* 1 (4.5%); P=0.401].

The preoperative baseline data (*Table 1*), operation-related baseline characteristics (*Table 2*), and postoperative conditions (*Table 3*) were compared between the two groups. In comparison with the anterior VSR group, the posterior VSR group was characterized by a larger proportion of male patients, a higher left ventricular ejection fraction, a longer operative time with extracorporeal circulation and the use of an aortic clamp; these differences were statistically significant (P<0.05). The systolic pulmonary artery pressure, the rate of preoperative intra-aortic balloon pump (IABP) and ventilator utilization, and the emergency operation status were higher in the anterior VSR group than in the posterior VSR group, although these differences were not statistically significant (P>0.05). The intervals between myocardial infarction and operation and between VSR and operation, as well as the volumes of transfusion erythrocytes and plasma required, the length of intensive care unit (ICU) stay, and the duration of mechanical ventilation in the posterior VSR group were higher than those in the anterior VSR group, but the differences were not statistically significant (P>0.05).

A comparison of postoperative complications between the two groups (*Table 4*) showed that there was no statistically significant difference in complications involving bleeding and low cardiac output, nor in the rate of pulmonary and cerebrovascular complications, and postoperative in-hospital mortality (P>0.05). After multi-factor adjustment, there were no significant differences between the two groups in perioperative death [odds ratio (OR) =1.033; 95% confidence interval (CI): 0.095 to 11.279; P=0.979], bleeding complications (OR =3.101; 95% CI: 0.466 to 20.647; P=0.242), low cardiac output complications (OR =0.839; 95% CI: 0.134 to 5.245; P=0.851), pulmonary complications (OR =1.796; 95% CI: 0.292 to 11.053; P=0.528) and cerebrovascular complications (OR =1.032; 95% CI: 0.091 to 9.805; P=0.998) (*Table 5*).

In this study, the 59 patients who survived surgery completed the postoperative follow-up (37 patients in the anterior VSR group, 22 patients in the posterior VSR group), with an overall follow-up time of 1.0–10.5 (median, 4.2) years. The follow-up results showed that there were no significant differences in the incidence of all-cause death

Table 4 Postoperative complications in 68 patients with myocardial infarction combined with VSR

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value
Perioperative death	6 (14.0)	3 (12.0)	0.820
VSR-to-surgery time <7 days	2 (33.3)	2 (66.6)	0.371
VSR-to-surgery time >7 days	4 (11.4)	1 (4.8)	0.401
Bleeding complications			
Gastrointestinal hemorrhage	2 (4.7)	2 (8.0)	0.574
Pericardial tamponade	1 (2.3)	2 (8.0)	0.627
Second thoracotomy for hemostasis	7 (16.3)	6 (24.0)	0.645
Low cardiac output complications			
Heart failure	7 (16.3)	4 (16.0)	0.976
CRRT	6 (14.0)	4 (16.0)	0.820
IABP	23 (53.5)	13 (52.0)	0.906
ECMO	3 (7.0)	1 (4.0)	0.602
Pulmonary complications			
Secondary tracheal intubation	4 (9.3)	0 (0.0)	0.114
Tracheotomy	5 (11.6)	2 (8.0)	0.638
Pulmonary infection	6 (14.0)	5 (20.0)	0.517
Myocardial infarction	0 (0.0)	0 (0.0)	–
Cerebrovascular complications	1 (2.3)	0 (0.0)	0.446

Data are n (%). VSR, ventricular septal rupture; CRRT, continuous renal replacement therapy; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation.

Table 5 Postoperative complications of 68 patients with myocardial infarction combined with VSR after multivariate analysis

Variables	Anterior VSR (n=43)	Posterior VSR (n=25)	P value	Calibrated OR	95% CI, P value
Perioperative death	6 (14.0)	3 (12.0)	0.820	1.033	0.095–11.279, 0.979
Bleeding complications	7 (16.3)	7 (28.0)	0.249	3.101	0.466–20.647, 0.242
Low cardiac output complications	25 (58.1)	13 (52.0)	0.623	0.839	0.134–5.245, 0.851
Pulmonary complications	8 (18.6)	6 (24.0)	0.596	1.796	0.292–11.053, 0.528
Myocardial infarction	0 (0.0)	0 (0.0)	–	–	–
Cerebrovascular event	1 (2.3)	0 (0.0)	0.446	1.032	0.091–9.805, 0.998

Data are n (%). VSR, ventricular septal rupture; OR, odds ratio; CI, confidence interval.

(HR =2.652; 95% CI: 0.199 to 35.338; P=0.460), heart failure (HR =1.588; 95% CI: 0.150 to 16.766; P=0.701), myocardial infarction (HR =1.041; 95% CI: 0.099 to 10.446; P=0.996), and cerebrovascular events (HR =1.045; 95% CI: 0.109 to 11.703; P=0.974) between the two groups. There were no differences in revascularization between the two groups (*Table 6*).

Discussion

VSR is a severe complication of myocardial infarction and manifests clinically by worsening hemodynamics

Table 6 Follow-up results of 59 patients with myocardial infarction combined with VSR after discharge

Variables	Anterior VSR (n=37)	Posterior VSR (n=22)	P value	Calibrated HR	95% CI, P value
All-cause death	7 (18.9)	1 (4.5)	0.131	2.652	0.199–35.338, 0.460
Heart failure	6 (16.2)	2 (9.1)	0.466	1.588	0.150–16.766, 0.701
Myocardial infarction	1 (2.7)	0 (0.0)	0.457	1.041	0.099–10.446, 0.996
Re-revascularization	0 (0.0)	0 (0.0)	–	–	–
Cerebrovascular event	2 (5.4)	0 (0.0)	0.278	1.045	0.109–11.703, 0.974

Data are n (%). VSR, ventricular septal rupture; HR, hazard ratio; CI, confidence interval.

and cardiogenic shock (1,2). Surgery is recognized as the primary treatment for VSR. It is generally believed that the effect of posterior VSR is worse than that of anterior VSR (4-6). However, this study suggests that the perioperative risks as well as the medium- and long-term outcomes of the surgical repair of anterior or posterior VSR after myocardial infarction are similar.

VSR is usually seen in patients with coronary atherosclerotic heart disease with a single-vessel lesion. Although it may occur within a few hours to 2 weeks after myocardial infarction it typically takes place within 2 to 4 days (5). When myocardial tissue is severely ischemic, it undergoes atrophy and necrosis, leading to the formation of scar tissue. The scarred myocardium has difficulty withstanding the pressure in the heart chamber, and normal myocardial contractions may cause rupture at the scar edge, resulting in VSR (15). It has been documented the development of VSR is related to the distribution of the coronary arteries. The anterior descending branch, the circumflex branch, and the right coronary artery can all give rise to the ventricular septal branches, and VSR may occur in the anterior, inferior, and lateral walls (16). Anterior wall infarction is more likely to cause apical VSR, while inferior and lateral wall infarctions are more likely to result in the rupture of the basement membrane at the junction of the septum and posterior wall. In the patients included in this study, VSR was located in the anterior (63.2% of cases) and in the posterior (36.8%) aspect of the ventricle. This result is similar to that obtained in a previous investigation (3,6).

VSR carries a high rate of fatality, and the effects of conservative treatment with drugs are not good. Without active surgical treatment, it has been reported that 24% of patients died within 1 week of VSR, and 80% of patients died within 1 month (17). A total of 139 patients with VSR were admitted to our center from January 2009 to January 2020, of which 51.1% (71/139) did not undergo surgery. In these patients, anterior VSR accounted for 64.8% (46/71) and posterior VSR accounted for 35.2% (25/71). In addition, 42.3% (30/71) died within 1 week of VSR, 60.6% (43/71) died within 2 weeks, 77.5% (55/71) died within 1 month, and only 7.0% (5/71) were alive during the follow-up period. These results are similar to those reported in previous studies (17,18). The reasons for these patients not receiving surgical treatment are as follows: first, some patients died before surgery in the acute VSR phase; second, some patients may have had complications other than heart disease, increasing the risk of surgery; third, some patients and their families refused surgical treatment because of cost or other reasons.

Although surgery is recognized as the primary treatment for VSR, there is controversy about the optimal time for surgery. It is generally believed that a long wait before the surgery can reduce perioperative mortality (6,19). Arnaoutakis *et al.* (20) reported that the overall perioperative mortality rate of patients with myocardial infarction combined with VSR was 42.9%. The mortality rate in VSR patients undergoing surgery within 7 days of myocardial infarction was 54.1%, while the mortality rate in patients with delayed surgery was significantly lower, at 18.4%. This difference is due to the formation of scar tissue around the rupture after the acute phase, which is conducive to the repair of the rupture. However, hemodynamic instability in some patients necessitates early intervention. In the acute stage of rupture, the infarcted myocardium is relatively thin and brittle, and suturing increases the risk of myocardial tear, leading to postoperative residual shunt. Therefore, it is critical to maintain stable hemodynamics preoperatively and pass through the acute phase smoothly. In this study, there was no significant difference in perioperative mortality between the two groups in terms of surgical treatment within 7 days of VSR and after 7 days.

The operative paths and modes of anterior and posterior

VSR are different (21,22). Anterior VSR can be repaired directly through an incision for the removal of the ventricular aneurysm. Posterior VSR sometimes results in mitral regurgitation due to ischemia involving the posteromedial papillary muscles. At the same time, repairing posterior VSR requires the heart to be elevated, making this procedure more difficult than anterior VSR. In this study, the operative time, CPB time, and aortic clamp time were significantly longer in the posterior VSR group than in the anterior VSR group. Meanwhile, the proportion of mitral valve surgery in the posterior VSR group was significantly higher than in the anterior VSR group, which was consistent with the pathophysiological changes caused by VSR.

Previous studies (6,23) have shown that patients with posterior VSR had higher perioperative mortality and lower long-term survival rates than patients with anterior VSR. However, Deja and coworkers observed that there was no statistically significant difference in perioperative mortality between patients with anterior and posterior VSR (24). Papadopoulos and collaborators did not observe a significant difference in the long-term survival rate between these two groups of patients (25). The results of our study showed that the perioperative risks and long-term follow-up outcomes in patients with posterior VSR were not significantly different from the patients with anterior VSR, which is at variance with previous reports or convictions. Two possible explanations can be offered for this discrepancy. The first possibility is that critically ill patients with posterior VSR may die before surgical treatment is possible, and the expectation of poor prognosis and difficulty of operation in these patients may lead to a delay in the treatment or referral to treatment. The hemodynamics of the patients undergoing surgery were relatively stable, and the patients were carefully selected, contributing to a better long-term prognosis after surgery. The second possibility is that isolated single-vessel coronary lesions may predominate in the posterior VSR, reducing the long-term risk of ischemic events after the surgery (26).

Some studies (3,27) have shown that repair of VSR accompanied by CABG can improve prognosis. In this study, the median follow-up time was 4.2 years, and a total of 7 cases (11.9%) died during follow-up. At the same time, the incidence of MACCEs was low and the medium and long-term prognosis was good, which may be related to the fact that the vast majority of patients in this study underwent CABG. Therefore, we suggest that CABG should be performed while repairing VSR to improve prognosis.

The findings of this study indicate that the perioperative risks and long-term outcomes of surgical repair for myocardial infarction with anterior or posterior VSR are similar. Therefore, cardiac surgeons should reassess the perception of the difficulty of surgical repair and poor prognosis associated with posterior VSR. Aggressive management of posterior VSR can avoid delayed referral and surgical repair and bring greater benefits to patients with posterior VSR. It is worth noting that the surgical repair of VSR should be accompanied by revascularization as much as possible to reduce the incidence of long-term adverse events.

Limitations of the study

Some limitations of this study should be acknowledged. Firstly, this study is a single-center retrospective study with a relatively small sample size, which may have resulted in selection bias. Secondly, the surgical procedures employed are not yet standardized. Developments and improvements in surgical techniques may affect the clinical outcomes. Finally, the time span of this study was long, potentially leading to information bias.

Conclusions

In summary, the perioperative risks, and medium- and long-term outcomes of the surgical repair of anterior or posterior VSR after myocardial infarction were found to be similar. This conclusion needs to be confirmed by larger clinical studies.

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Footnote

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-577/coif). The authors have no conflicts of interest to declare.

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 Institutional Ethics Committee of Beijing Anzhen Hospital (No. 2019070X) and informed consent was taken from all the patients.

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