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Research paper

Indicators and predictors of in-hospital mortality and survival in patients with ventricular septal rupture

Jayashree Kharge *, Chirag Jagdish Parikh, Manohar Jayadevappa Suranagi, Sridhar Lakshmanasastry, Kikkeri Hemanasetty Srinivasa, Cholanahalli Nanjappa Manjunath

Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore, India

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ABSTRACT

Background: Ventricular septal rupture (VSR), a mechanical complication of myocardial infarction (MI), usually presents with rapid clinical deterioration with acute heart failure or cardiogenic shock. VSR may occur within 24 h to several days after MI and can occur in both anterior and inferior wall MI. Although guidelines recommend emergent surgery, this is associated with a high mortality rate of up to 40%. Intra-aortic balloon pump (IABP) and extracorporeal membrane oxygenation (ECMO) stabilize patients in preparation for angiography and surgery. Delayed surgery allows better septal repair in scarring tissue but also carries the risk of rupture extension and death while waiting. Percutaneous closure of the defect with appropriately designed devices results in better survival in the subacute phase.

Aims: To study the indicators and predictors of VSR in the current era of primary percutaneous coronary interventions and mechanical circulatory support.

Methods: Of total of 34,681 patients presenting with MI, the incidence of VSR was 0.45%. We sought to evaluate the predictors of survival and death in VSR. Coronary angiography (CAG) was performed, hemodynamic support provided to unstable patients, and consenting patients were referred to definitive therapy, either surgery or percutaneous device closure. The previously postulated hypotheses of triple vessel disease (TVD), diabetes mellitus (DM), and concentric left ventricular hypertrophy (LVH) due to Hypertension (HTN) being protective against VSR were explored.

Results: Of the 169 patients with VSR, we found that the group that survived was mostly men and the mean age was 61.5 years; this was in contrast to the non-survivors, who were mainly women, and the mean age was 65.2 years ($p = 0.025$); higher Killip Class was 111-1V ($p = 0.001$), lower LVEF ($p = 0.010$), apical VSR and LV aneurysm ($p = 0.015$ and $p = 0.002$, respectively) were predictors of death. 48 patients underwent CAG, with single vessel disease (SVD) with lower-grade Rentrop collateral flow being most common in the death group. 25 patients were subjected to definitive therapy with surgical patch closure or percutaneous device closure. The patients who died were older by approximately 7 years. The risk factors for coronary artery disease, such as HTN, diabetes, and smoking, were not statistically different between the two groups.

Conclusion: Prevention of myocardial infarction is more important than managing a VSR, which carries a high mortality despite advanced mechanical support and definitive interventional therapy such as emergent surgery and percutaneous device closure.

1. Introduction

The incidence of ST-elevation myocardial infarction (STEMI) is much higher in the Indian subcontinent than Western population [1]. It is estimated that approximately 3 million cases of STEMI occur every year. According to the Position statement for the management of STEMI in India, delayed hospitalization and reperfusion therapy are causes of increased 30-day mortality [2–5]. Although the incidence of ventricular septal rupture (VSR) in myocardial infarction (MI) has reduced from 1%–3% [6–9] prior to the reperfusion era, to 0.2%–0.5% [10,11]

* Corresponding author at: Dept of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bannerghatta Main Road, Jayanagar 9th Block, Bangalore 560069, India.
E-mail address: khargej@gmail.com (J. Kharge).

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following PCI or thrombolytic therapy, the mortality of VSR remains unchanged at 45% for surgically treated patients and almost 90% for those on medical therapy [12,13]. The current study aimed to analyze the incidence of post-MI VSR and identify predictors of mortality in these patients.

2. Methods

This is a prospective observational study of patients diagnosed with VSR following acute coronary syndrome (ACS) at our institute between February 1, 2016 and December 31, 2018.

34,681 patients were included in the study; of these, 169 were diagnosed with post-MI VSR, the incidence of VSR was 0.48%. Patients were assessed for hemodynamic stability, and an electrocardiogram (ECG) diagnosis of MI was made on the basis of the Universal Definition of Myocardial Infarction. Echocardiography was performed using a GE Vivid T8 Cardiovascular Ultrasound machine (General Electric, USA) to evaluate regional wall motion abnormalities (RWMA), left ventricular ejection fraction (LVEF), and VSR. The size, location, and type of the VSR were also noted. The presence of left ventricle aneurysm, mitral regurgitation, tricuspid regurgitation, and right ventricular function were assessed. The management of VSR was at the discretion of the treating physicians and surgeons. The patients were grouped into survivors (discharged) and non-survivors (died), and they were analyzed to assess the predictors of in-hospital death and factors favoring discharge following post-MI VSR. Patients were followed up for a period of 30 days from the time of hospitalization for any further complications or death.

3. Statistical analysis

Descriptive statistics are presented as means ± standard deviations and medians ± interquartile ranges (IQRs) for continuous variables, and frequencies and percentages for categorical variables. Group comparisons for continuously distributed data were performed using the independent sample *t*-test when comparing the two groups. If data were found to be non-normally distributed, the Wilcoxon-Mann-Whitney *U* test was used for these comparisons. The chi-square test was used for group comparisons of categorical data; if the expected frequency in the contingency tables was <5 for >25% of the cells, Fisher's exact test was used instead. Statistical significance was set $p < 0.05$ (Figure 1 and 2).

4. Results

The total number of patients admitted during the study period was 34,681. Of these, 169 patients diagnosed with MI, the incidence of VSR was 0.48%. We assessed the predictors of in-hospital death and factors favoring discharge following post-MI VSR. The group that survived mainly comprised men, and the mean age was 61.5 years; this is in contrast to the non-survivors, who were mainly women, and the mean age was 65.2 years ($p = 0.025$). Moreover, the Killip Class was III-IV in the death group ($p = 0.001$), and LVEF was lower in the non-survivors ($p = 0.010$). Apical VSR and LV aneurysm were predictors of higher mortality ($p = 0.015$ and $p = 0.002$, respectively). Forty-eight patients underwent coronary angiography, single vessel disease (SVD) with lower-grade Rentrop collateral flow being the most common in the death group. Hemodynamic support included inotropes and Intra aortic balloon pump (IABP) and 25 patients were subjected to definitive therapy with surgical patch closure or percutaneous device closure.

Table 1 summarizes the association between mortality and clinical variables. There was a statistically significant association between mortality and age, sex, Killip Class, duration of chest pain, systolic BP (mmHg), diastolic BP (mmHg), and Thrombolysis in Myocardial Infarction (TIMI) risk score. The patients who died were older by approximately 7 years and were more likely to be female, have a Killip Class III–IV, a shorter duration of chest pain, lower systolic and diastolic BP, and a TIMI score > 7.

Table 2 summarizes the association between mortality and ECG, Echo, and angiography profiles. There was a statistically significant association between mortality and anterior and inferior wall MI. Those who died were more likely to have anterior wall MI and less likely to have inferior wall MI; they also tended to have relatively lower LVEF on 2D echocardiography. The apical location of VSR and presence of LV aneurysm were also predictors of higher mortality ($p = 0.015$ and 0.002, respectively). 48 patients underwent coronary angiography, with single vessel disease (SVD) being the most common, and with lower evidence of collaterals by Rentrop classification in the death group.

There was no significant association between the mode of management and outcomes (Table 3).

5. Discussion

VSR continues to challenge medical and surgical teams to improve prognosis and reduce cardiac morbidity and mortality. We found a statistically significant association between mortality and older age, female sex, lower Killip Class (III–IV) at presentation, shorter duration of chest pain, lower systolic BP (mmHg) and diastolic BP (mmHg) at presentation, and higher TIMI risk score (>7) following VSR. Patients who died were older by approximately 7 years. The associated risk factors for coronary artery disease, including hypertension, diabetes, and smoking, were not statistically different between the two groups. The proposed

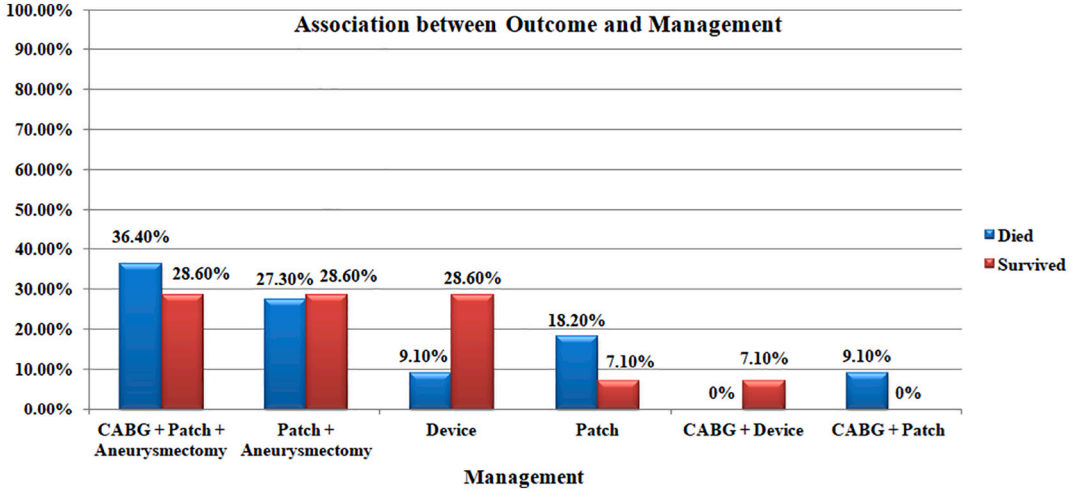

Fig. 1. Survival and death in patients based on management of VSR.

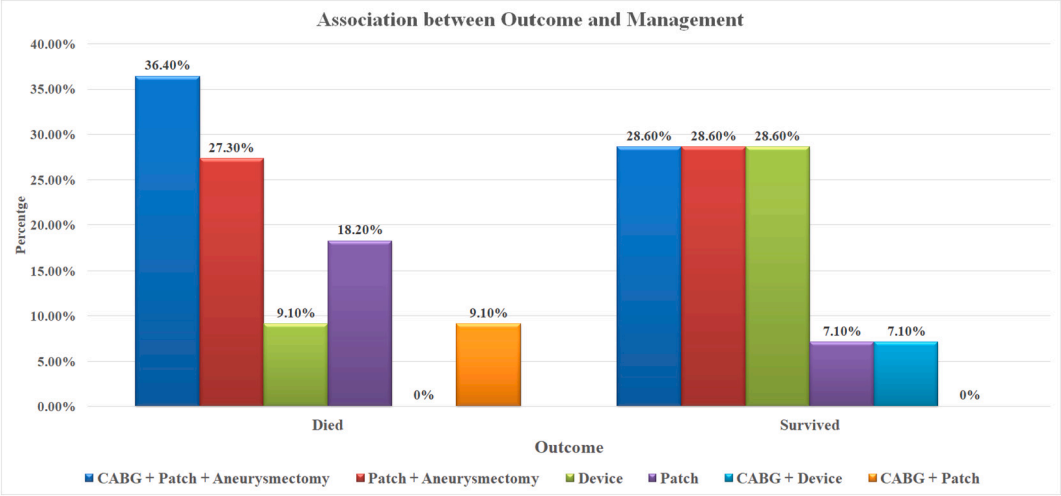

Fig. 2. Outcome of survival and death in patients undergoing surgery and percutaneous device.

Table 1
Association between outcome and demographic variables.

All parameters	Outcome		
	Died (n = 138)	Survived (n = 31)	p value
Age (years)*	65.35 ± 10.09	58.84 ± 7.64	<0.001†
Sex*			0.002‡
Male	52 (37.7%)	21 (67.7%)	
Female	86 (62.3%)	10 (32.3%)	
Diabetes mellitus	80 (58.0%)	13 (41.9%)	0.105‡
Hypertension	8 (49.3%)	12 (38.7%)	0.287‡
Tobacco use	0 (7.2%)	3 (9.7%)	0.708§
Smoking	6 (18.8%)	10 (32.3%)	0.099‡
Past H/O MI	1 (8.0%)	3 (9.7%)	0.723§
Killip Class*			<0.001‡
I–II	73 (52.9%)	28 (90.3%)	
III–IV	65 (47.1%)	3 (9.7%)	
Duration of chest pain*			0.023‡
<12 h	47 (36.2%)	4 (13.3%)	
12–24 h	26 (20.0%)	5 (16.7%)	
>24	57 (43.8%)	21 (70.0%)	
Dyspnea (present)	40 (29.0%)	12 (38.7%)	0.289‡
Syncope (present)	6 (4.3%)	0 (0.0%)	0.594§
Pulse (BPM)	94.36 ± 18.88	93.87 ± 16.17	0.671‖
Systolic BP (mmHg)*	97.16 ± 39.49	121.68 ± 28.58	0.003‖
Diastolic BP (mmHg)*	54.96 ± 32.93	77.61 ± 12.73	<0.001‖
TIMI score category*			0.0012
≤7	62 (44.9%)	24 (77.4%)	
>7	76 (55.1%)	7 (22.6%)	

The following variables - Age, Gender, Killip Class, Duration of chest pain, Blood Pressure and TIMI Risk Score were significantly associated ($p < 0.05$) with the variable 'Outcome'.

* Significant at $p < 0.05$.
† t-Test.
‡ Chi-Squared Test.
§ Fisher's Exact Test.
‖ Wilcoxon-Mann-Whitney U Test.

Table 2
Association between outcome and ECG, echo and angiography profile.

	Outcome		
	Died (n = 138)	Survived (n = 31)	p value
ECG, echo and angiography profile			
ECG: anterior wall (present)*	117 (84.8%)	21 (67.7%)	0.027†
Inferior wall (present)*	26 (18.8%)	12 (38.7%)	0.017†
Others (present)	86 (62.3%)	18 (58.1%)	0.660†
2D ECHO: LVEF*			<0.001†
<30%	17 (12.3%)	0 (0.0%)	
30–40%	85 (61.6%)	14 (45.2%)	
40–50%	35 (25.4%)	12 (38.7%)	
>50	1 (0.7%)	5 (16.1%)	
2D ECHO: MR*			0.008‡
Absent	72 (52.2%)	7 (22.6%)	
Mild	57 (41.3%)	20 (64.5%)	
Moderate	7 (5.1%)	4 (12.9%)	
Severe	2 (1.4%)	0 (0.0%)	
2D ECHO: TR*			0.003‡
Absent	78 (56.5%)	7 (22.6%)	
Mild	27 (19.6%)	13 (41.9%)	
Moderate	28 (20.3%)	10 (32.3%)	
Severe	5 (3.6%)	1 (3.2%)	
2D ECHO: aneurysm (present)*	35 (25.4%)	17 (54.8%)	0.001†
VSR 2D ECHO: location			0.144‡
Apical	112 (81.2%)	21 (67.7%)	
Apical/Mid	1 (0.7%)	1 (3.2%)	
Mid	10 (7.2%)	2 (6.5%)	
Basal	15 (10.9%)	7 (22.6%)	
VSR 2D ECHO: number			0.426‡
Single	130 (94.2%)	28 (90.3%)	
Multiple	8 (5.8%)	3 (9.7%)	
VSR 2D ECHO: type			0.766‡
Simple	122 (88.4%)	27 (87.1%)	
Complex	16 (11.6%)	4 (12.9%)	
Coronary angiogram: number of vessels			0.894‡
None	0 (0.0%)	1 (4.5%)	
SVD	17 (65.4%)	14 (63.6%)	
DVD	7 (26.9%)	5 (22.7%)	
TVD	2 (7.7%)	2 (9.1%)	

The following variables were significantly associated ($p < 0.05$) with the variable 'Outcome': Anterior Wall on ECG, LVEF, mitral regurgitation LV apical aneurysm on 2D ECHO.

* Significant at $p < 0.05$.
† Chi-Squared Test.
‡ Fisher's Exact Test.

protection mechanism responsible for the reduced incidence of VSR in patients with hypertension, diabetes, and coronary artery disease with collaterals has been explored previously, and it was concluded that the primary prevention of MI is the key to preventing mechanical complications of VSR.

5.1. Hypertension (HTN) and VSR

Hypertension defined as per the ACC/AHA Hypertension guidelines, 2018. The estimated cases of hypertension are increasing in prevalence, with an estimated 1.3 billion cases worldwide [14]. HTN has been

postulated to be of significance in the pathogenesis of VSR, and it is found in approximately 40%–50% of patients with VSR [15,16].

All 41 patients studied by Roberts et al., who died of rupture, whether LV free wall or septal rupture post infarction, had LV thickening consistent with a history of hypertension [17]. However, Shapira et al. documented that only one third of their study population had HTN, suggesting that HTN is not a major factor for VSR [18].

Moreyra et al. [19] suggested that the increased wall thickness due to concentric hypertrophy may confer protection against septal rupture following AMI. The Becker and van Mantgem [20] classification of free wall rupture, which is also applied to VSR, is described as follows:

Becker type 1 ruptures are slit-like tears through normal thickness myocardium, which occur abruptly within 24 h of MI, and are typically related to intramural hematomas dissecting through tissue planes. These type 1 ruptures typically occur in the setting of a relatively small inferior MI involving the margins of the posterior descending artery distribution, due to shear stress generated by the adjacent hyperkinetic myocardium supplied by the non-infarct left anterior descending (LAD) artery. These VSRs are said to be present either at, or shortly after clinical presentation. Becker type 2 ruptures typically result in sub-acute erosion of the infarcted myocardium, and are associated with neutrophilic infiltration and coagulation necrosis. Becker type 3 ruptures result from perforation of the thinned aneurysmal myocardium in the late phase post-MI, and occur more frequently in the absence of reperfusion therapy.

We believe that hypertensive patients with MI may present with Becker's type 1, slit-like rupture as a result of a hypertrophied septum, which may progress to developing intramyocardial dissection hematoma and subsequent VSR a week of more post-MI. Thrombolytic therapy may hasten the process of VSR, leading to delayed recognition and increased mortality in hypertensive patients with VSR. HTN can worsen prognosis in post-MI VSR, and that hypertrophied LV offers no protection against VSR.

5.2. Diabetes, ischemic preconditioning, and collaterals in VSR

Type 2 diabetes mellitus (DM) is predicted to increase from 415 million to 642 million by 2040 [21]. The prevalence of diabetes in VSR varies between 11% and 39% [9,10,13,22,23].

Moreyra et al. speculated that DM, with associated multiple vessel disease and collateral blood supply, protects against septal rupture, which would explain the lower incidence of DM in patients with post-MI VSR. This hypothesis was further supported by Pretre et al., who described the development of VSR in a particularly vulnerable myocardium following an abrupt closure of a single vessel [23,24] following acute MI, supporting the theory of absence of collaterals leading to VSR. However, the incidence of DM in their study was only 15% [25,26]. Interestingly, Zbinden et al. determined the influence of DM on coronary collateral flow by accurate means of collateral flow measurement in a large population with variable degrees of coronary artery disease. They concluded that the quantitatively measured collateral flow index (CFI) did not differ between diabetic and non-diabetic patients with stable coronary artery disease. It is said that the vascular endothelial growth factor-induced chemotaxis of monocytes, which has a key role in arteriogenesis, is attenuated in patients with DM [27]. The VSR post MI can occur irrespective of a previous history of coronary artery disease and diabetes with collaterals.

Contrary to other studies, Moreyra et al., in their study spanning over 17 years (1990–2007) found a lower incidence of diabetes and hypertension. As a result, they speculated that thickened septum due to hypertension conferred protection against VSR and also reported chronic renal disease as an additional independent risk factor for VSR. In our study, diabetes, hypertension, and renal dysfunction were all predictors of VSR.

Of the 169 patients with post-MI VSR, 102 were referred from other centers, and 67 developed VSR after hospitalization for STEMI. However, only 48 patients underwent coronary angiography (CAG) for various reasons: 31 had single vessel disease (SVD), 12 had double vessel disease (DVD), only 4 had triple vessel disease (TVD) and 1 had no significant stenosis in any coronary artery. Given that CAG was not performed in all patients, the presence of SVD and absence of collateral circulation causing VSR remain inconclusive.

In a retrospective cohort of post-MI VSR by Pradhan et al., the 30-day mortality was 80.4% and the mean time to surgery was 7.7 days (2.4). Advanced age, previous cerebrovascular accident, and surgical repair were found to be modifiers of the 30-day risk of death [28].

In a study by Ronco et al. of 475 patients from 26 different centers worldwide, the early mortality rate for surgically treated ventricular septal rupture was 40.4% [29].

Shi et al. [30] identified 96 cases of post-MI VSR and divided the cases into an acute-phase survivor group ($n = 46$, survived $\geq$2 weeks after admission) and a non-survivor group ($n = 50$, died within 2 weeks of admission). Percutaneous closure was considered in acute-phase survivors. The overall acute-phase mortality rate was 52%, while female sex and Killip Class III–IV at admission were associated with an increased risk of acute-phase death. Of the 46 patients who survived $\geq$2 weeks, 20 underwent interventional occlusion, and the procedure was successful in 19. Percutaneous closure in the acute-phase survivor group improved the immediate (21% in-hospital mortality rate) and long-term (53% mortality) outcomes. Therefore, irrespective of surgical closure or device closure, mortality remains high in the acute phase of post-MI VSR. Female sex and severe cardiac dysfunction at admission were also found to be associated with a high rate of acute-phase deaths, and they hypothesized that the survivors with left ventricular aneurysm and increased LVEDV had decreased pressure gradient between the left and right ventricles, preventing expansion of the rupture.

In a recent retrospective study of 50 cases by Zhang et al. [31], in an univariable analyses that the heart rate (HR), white blood cell (WBC) count, neutrophils count, serum glucose, serum creatinine, serum lactic acid, and the closure of rupture were significantly associated with very high mortality where in only 14 patients survived.

Taken together, these studies suggest that the survival is poor in immediate post-MI VSR, and that mechanical circulatory support may be helpful in maintaining the hemodynamics to roll in the patients into the subacute phase for improved survival, either by surgery or percutaneous closure. This is the largest study (34,000 patients with acute MI) to study the incidence of VSR and the predictors of early mortality and survival in the first thirty days.

6. Limitations

Not all patients underwent definitive surgical repair and very few underwent device closure. In addition, preoperative hemodynamic and mechanical circulatory support were not used consistently to stabilize the patients prior to surgery. A prospective study comparing percutaneous device closure and surgery in VSR while establishing an ideal time for intervention for both procedures would be interesting.

7. Conclusion

The apprehension of surgeons and cardiologists in the acute phase of post MI VSR, doubts of patients in accepting high-risk procedures, and the disease itself, continue to contribute to increased mortality. The primary prevention of MI remains key to preventing VSR.

Diseases can rarely be eliminated through early diagnosis or good treatment, but prevention can eliminate the disease.

–Denis Parsons Burkitt

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ahjo.2022.100095.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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