Decision Making, Management, and Midterm Outcomes of Postinfarction Ventricular Septal Rupture: Our Experience with 21 Patients

Abstract

Context: Ventricular septal rupture (VSR) is a dreaded complication following myocardial infarction. Surgical repair of VSR is associated with significant early mortality. Variable outcomes in terms of early mortality and midterm functional status have been reported from different centers. **Aims:** In our study, we attempt to review the experience of decision making and surgical repair of postinfarction VSR, and to analyze the factors contributing to the early mortality and midterm outcome after repair. **Materials and Methods:** It is a retrospective study. Data were summarized retrospectively by frequencies and percentages for categorical factors, and means and standard deviations for continuous factors. Multivariate logistic regression, odds ratios, 95% confidence intervals, and P value were calculated for different variables to determine their independent effect on operative mortality. All surviving patients answered the EQ-5D Health Questionnaire. **Results:** Preoperative renal failure, left ventricular dysfunction (moderate and severe), and Killip class (III and IV) were significantly associated with early mortality after surgery. Small residual ventricular septal defect (VSD) was not found to affect the midterm quality of life. **Conclusions:** Early surgical repair benefits the patient by preventing early end-organ damage. The renal failure left ventricular dysfunction (moderate and severe) and Killip class (III and IV) adversely affect early outcomes after surgery. Small residual ventricular septal defect (VSD) does not affect the midterm quality of life.

Keywords: *Cardiac surgery, myocardial infarction, ventricular septal rupture*

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Introduction

Ventricular septal rupture (VSR) is a dreaded complication of acute myocardial infarction (MI) associated with high mortality. Ventricular ruptures mostly involve the left ventricular (LV) free wall rather than the interventricular septum (IVS). LV free wall rupture is usually fatal, whereas surgical repair of post-MI VSR has short-term (30 days) mortality at 19%–66%.[1,2] Medical management of this condition is ineffective, with a 30 days mortality of 90%. Among 41,021 patients in the GUSTO-I trial, VSR was suspected in 140 (0.34%) and confirmed by retrospective review in 84 (0.2%). The incidence of postinfarction VSR following acute MI has been estimated at 0.2–2% in the thrombolytic era.[3] This mechanical complication usually occurs in the first week after the onset of acute MI and is generally associated with an occluded infarct-related coronary artery, in the absence of collateral circulation.[4]

In 1845, Latham first described postinfarction VSR, and in 1923, Brunn made the first antemortem diagnosis.[5,6] Sager established the clinical criteria for diagnosis and stressed the association of VSR with coronary artery disease.[7] In 1957, Cooley, *et al*. first reported the successful surgical repair of a postinfarction VSR.[8]

We report our experience in decision making, management, and midterm outcomes of postinfarction VSR referred to our institution between the years 2010–2014. In the present study, we have evaluated the clinical outcome of 21 patients treated surgically at our center, by assessing preoperative, operative, and postoperative variables.

Materials and Methods

Our series comprised 24 consecutive patients referred to our center for repair of postinfarction VSR between January 2010 and December 2014. Coronary angiography was performed in every patient. Three

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patients did not consent to the high-risk operation and were excluded from the study. The data of 21 patients who underwent repair were retrospectively analyzed. Among these, 16 (76.2%) were operated during the acute phase within 7 days after the onset of MI, 5 (23.8%) were operated during 8–21 days after MI as they were referred late from other institutions. All except 4 patients were male. Table 1 shows the preoperative clinical profile. All patients had left to right shunt demonstrated by color Doppler. Intra-aortic balloon pump (IABP) support was initiated preoperatively in 20 patients and immediately after coming off cardiopulmonary bypass (CPB) in one patient. Around 11 patient's required preoperative assisted ventilation (6 with endotracheal intubation and 5 noninvasive ventilation) while preparing for surgical repair. The location of the VSR, right ventricular (RV), and left ventricular (LV) function were assessed by 2D echocardiography and color Doppler [Table 2].

The technique used for repair was infarct exclusion in all patients. Intraoperative transesophageal echocardiography (TEE) and postoperatively, 2D echocardiography was carried out on all patients to assess ventricular function and residual VSD. Associated coronary artery bypass grafting (CABG) was undertaken in 12 patients. Only 1 patient required repair of LV aneurysm as well. The variables tested as predictors of operative mortality were LV and RV dysfunction, preoperative renal failure, location of VSD, and residual VSD. These variables were chosen after the data were analyzed.

The mean follow-up of patients discharged from the hospital was 5 years. Patients were followed-up every 6 months in the outpatient setting and through telephone interviews. All discharged patients had echocardiography during follow-up. The follow-up of patients discharged from the hospital was 100% complete. All surviving patients answered the EQ-5D Health Questionnaire. The EQ-5D is a well-regarded questionnaire that provides a generic measure of health status based on 5 dimensions: mobility, self-care, usual activities, pain/discomfort, and anxiety/depression.

Data were analyzed retrospectively for frequencies and percentages for categorical factors, and means and standard deviations for continuous factors. Multivariate logistic regression, odds ratios, 95% confidence intervals, and *P* value were calculated for different variables to determine their independent effect on operative mortality. Renal failure was defined as serum creatinine levels higher than 1.36 mg/dL or the need for renal replacement therapy (RRT). Postoperative RRT was in the form of continuous venovenous hemofiltration (CVVH) or continuous venovenous hemodiafiltration (CVVHDF). Right ventricular (RV) dysfunction was defined by Trans-Annular Plane Systolic Excursion (TAPSE) <16 mm.

Table 1: Preoperative clinical profile

Variables	No. of patients
Age (years)	
≤60	5
≥61	16
Killip Class	
II	4
III	11
IV	6
Sinus rhythm	21
Cardiogenic shock	6
Location of VSD	
Anterior	17
Posterior	4
Left ventricular function	
Normal (LVEF >0.6)	0
Mild dysfunction (LVEF 0.4-0.59)	4
Moderate dysfunction (LVEF 0.3-0.4)	11
Severe dysfunction (LVEF <0.3)	6
Right ventricular function	
Normal (TAPSE >16 mm)	14
Abnormal (TAPSE <16 mm)	7
Coronary artery disease	
Single vessel	14
Double vessel	3
Triple vessel	4
Time interval between VSD diagnosis and surgery	
<7 days	16
8-21 days	5
IABP inserted	
Preoperatively	20
Postoperatively	1

IABP=Intra-aortic balloon pump, LVEF=Left ventricular ejection fraction, TAPSE=Tricuspid Annular Plane Systolic Excursion

Table 2: Preoperative left and right ventricular function

Anterior VSD (*n*=17)		Posterior VSD (*n*=4)	
LV dysfunction	RV dysfunction (TAPSE <16 mm)	LV dysfunction	RV dysfunction (TAPSE <16 mm)
Mild 4	0	Mild 0	0
Moderate 10	2	Moderate 1	3
Severe 5	1	Severe 1	1

Results

The time from confirmation of the diagnosis of VSD to surgery varied widely from 10 h to 21 days. Sixteen (76%) patients were operated on within 7 days of VSD diagnosis; 4 of them within 48 h. Five (23.8%) patients were operated after 7 days of diagnosis because they had presented late to our centre. The mean time from onset of symptoms of MI to the time of VSD could not be recorded as most of the patients were referred from peripheral hospitals with a lack of diagnostic modalities.

Around 11 (52.38%) patients were in Killip class III at the time of VSD diagnosis, 4 were in class II, and 6 were in

class IV. Fifteen (71%) patients had some deterioration in clinical status from the time of VSD diagnosis to the time of surgical repair. Seventeen (81%) patients required inotropic support preoperatively. IABP was used in all patients; preoperatively in twenty (95%) and postoperatively in one (5%) patient. Nearly 6 patients required invasive ventilation in the preoperative period. Coronary angiography showed single vessel disease (SVD) in 14 (66.67%), double vessel disease (DVD) in 3 (14.28%), and triple vessel disease in 4 (19.04%) patients. Figure 1 shows the distribution of coronary artery disease according to the VSR location. SVD was significantly more common in anterior VSRs compared to posterior VSRs in this series, similar to the findings of Davies *et al*[9]. In the group with SVD, 85% had total occlusion of the infarcted artery. Reported figures range from 57% to 82%[3,10].Preoperative echocardiography revealed moderate left ventricular dysfunction in 11 (52.38%) with left ventricular ejection fraction (LVEF = 30–40%) and severe impairment in 6 (28.5%) patients. Right ventricular dysfunction was noted in 7 (33.3%) preoperatively.

There was no statistically significant relationship between the location of VSD (anterior and posterior) and mortality. Of the 17 patients who underwent concomitant CABG, 14 (82.35%) survived [Table 3]. Out of the remaining 4 patients who did not undergo revascularization, 2 (50%) patients survived. The hospital (30 days) mortality was 23%. One mortality was intraoperative and 4 occurred in the intensive care unit. Low cardiac output was the cause of death.

For the 16 patients discharged from hospital, total cumulative survival was 100% at a mean followup of 5 years. Nearly 6 (37.5%) of the surviving patients were in New York Heart Association (NYHA) class I, 9 (56.25%) were in class II, and 1 (6.25%) was in class III [Table 4]. No reoperation has been performed postdischarge. All surviving patients were followed up by telephone interviews using EQ5D questionnaire: 15 patients indicated having no
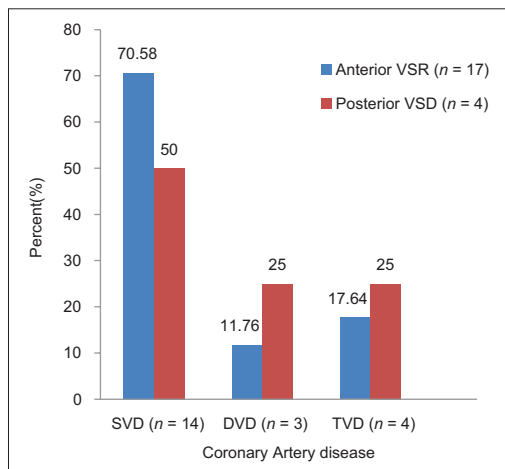
problems in any of the dimensions, and 1 reported moderate discomfort as well as anxiety/depression and pain. None of the interviewed patients reported having extreme problems in any of the five areas assessed.

The predictors of mortality were renal failure (*P* = 0.02), left ventricular dysfunction (moderate and severe) (*P* = 0.02), and Killip class III and IV (*P* = 0.01). Table 5 shows the risk analysis for predictors of operative mortality. There was no statistically significant relationship between the

Table 3: Operative mortality

Factors	Numbers (%)
Age (years)	
≤60	0
≥61	5
Anterior VSD	3/17 (17%)
Posterior VSD	2/4 (50%)
Left ventricular function	
Mild dysfunction (LVEF 0.41-0.59)	0/4 (0%)
Moderate dysfunction (LVEF 0.30-0.40)	3/11 (27%)
Severe dysfunction (LVEF <0.30)	2/6 (33%)
Right ventricular dysfunction	
Normal (TAPSE >16 mm)	14/21 (66.66%)
Abnormal (TAPSE <16 mm)	7/21 (33.33%)
Coronary artery disease	
Single-vessel	3/14 (21.4%)
Multi-vessel	2/7 (28.5%)
Cardiogenic shock	
Yes	3/6 (50%)
No	2/15 (13.3%)
Renal failure	
Yes	3/6 (50%)
No	2/15 (13.3%)
Residual VSD	0/21 (0%)

TAPSE=Tricuspid Annular Plane Systolic Excursion

Table 4: Follow-up and current postoperative status

Variables	Percentage
Residual VSD	3/16 (18.75%)
Current NYHA class	
I	6/16 (37.5%)
II	9/16 (56.25%)
III	1/16 (6.25%)
IV	0%

Post-discharge mean follow-up: 5 years. All surviving patients answered the EQ-5D questionnaire

Figure 1: Distribution of coronary artery disease according to VSR location

Table 5: Predictors of postoperative mortality

Variable	Odds ratio	95% Confidence interval	*P*
Renal failure	23.82	1.49-380.2	0.02
Left ventricular dysfunction (moderate and severe)	0.05	0.00-0.63	0.02
Killip Class (class III and IV)	0.18	0.02-1.73	0.010

location of VSD (anterior and posterior) and mortality. All the patients who did not survive were in Killip class III and IV preoperatively. Killip classes III and IV were found to have adverse impact on the mortality (*P* = 0.01). Residual VSD was noted in 3/16 (18.75%) cases postoperatively. The residual VSD did not adversely affect the outcome (*P* = 0.67).

Discussion

Improvement in the technique of rapid thrombolysis has brought down the incidence of postinfarction VSR. However, poor access to tertiary care hospitals still poses a threat for a post-MI complication such as VSR in the developing nations. Despite the declining trend, VSR remains one of the most challenging conditions encountered by cardiac surgeons. Post-MI VSR is associated with high early mortality.[3] Early mortality following repair of postinfarction VSD has been reported to be between 19% and 66%.[1,2] Our rate of early mortality was 23.8%.

The optimal timing of surgical repair for VSRs is critical. A longer interval before surgery has been reported to be associated with improved survival.[11,12] Pang *et al.* reported a study on 38 consecutive VSR repairs, however, it has opined that delayed VSR repair should be reserved only for a carefully selected group of patients who remain hemodynamically stable.[12]

Even with high rates of hospital mortality, we feel urgent surgical intervention is required for this condition, given the positive midterm outcomes of patients discharged from the hospital. Five patients were treated after a period of 7 days because they were clinically stable. These patients were closely observed for any signs of deterioration that would prompt urgent surgery. This approach was employed by treating surgeons to allow fibrosis of the infarcted myocardium. This strategy of delay must be approached with caution given that deterioration may occur rapidly, and this deterioration has been shown to adversely affect survival.[13,14]

Although a short time from MI to surgery has been reported to be a risk factor for mortality, the optimal timing to surgery and the most appropriate approach to VSD patients have not been established yet.[15,16]

Fifteen patients in this study underwent concomitant CABG at the time of VSD repair. There was no significant survival advantage for these patients. CABG remains a controversial topic in surgery for postinfarction VSD. A number of studies have demonstrated no significant survival benefit, which is in agreement with our observations.[17-21] Moore *et al.*[22] had reported that RV dysfunction has a negative impact on early and late survival. As RV failure ensues, the left-sided cardiac chambers are unable to fill, leading to biventricular failure. This exacerbates the low cardiac output state, contributing to a higher mortality rate. However, we did not find a significant association between RV dysfunction

and mortality. This could be due to the small number of subjects in our study which lacked statistical power.

We did not find a significant difference in survival between patients with anterior or posterior defects. The location of the defect also did not influence the rates of residual or recurrent VSD. Worse outcomes have been noted for patients with posterior defects in some series, which may reflect RV dysfunction resulting from an inferior infarction and the technical difficulties associated with the repair of these lesions.[19-21] Our observations are consistent with studies that have not demonstrated a significant difference in mortality associated with the location of the VSD.[4,14,17,23-25]

Infarct exclusion technique and septal reconstruction are the two techniques used for surgical repair in VSR. We followed infarct exclusion in all patients. It includes ventriculotomy through the infarcted LV parallel to the IVS for both anterior and posterior defects. A limited infarctectomy was performed. The patch was fixed to the noninfarcted region of the septum and viable LV wall, excluding the infarcted tissue from the LV cavity. The advantages of an exclusion technique include the restoration of LV geometry and avoidance of tension on the friable muscle.[23,26]

In our study, none of the patients were thrombolyzed when they were presented to us. This is mainly due to the unavailability of trained medical specialists in the peripheral hospitals in our state. A number of centers have reported the earlier onset of VSR in those receiving reperfusion therapy in acute MI.[3,27-29] Several clinical trials and overview analyses by Becker *et al.* have suggested that the survival benefit conferred by thrombolytic therapy may be offset by a paradoxical increase in early deaths from cardiac rupture. This large registry experience included over 350,000 patients with MI. This suggests that thrombolytic therapy accelerates cardiac rupture, typically within 24 to 48 h of treatment. They opined that the possibility that rupture represents an early hemorrhagic complication of thrombolytic therapy should be investigated.[27] Rhydwen *et al.* studied the influence of thrombolytic therapy on the patterns of VSR after acute MI in 29 patients over a 5-years period. They observed that thrombolytic therapy resulted in an earlier presentation of VSR after acute MI. However, thrombolysis reduces the overall incidence and in-hospital mortality of this life-threatening complication.[3,26,30-32] An earlier presentation of a VSD, usually within the first 24 h, is possibly associated with a worse prognosis irrespective of thrombolytic therapy because if a VSD occurs early the MI is more likely to be larger and hemorrhagic than if the VSD occurs late.[33] There is a reduction in the number of patients developing a late postinfarction VSD after thrombolytic therapy, while the number of patients developing an early postinfarction VSD is not reduced to the same degree. Rhydwen *et al.*

however, were of the opinion that the acceleration in postinfarction VSD formation after thrombolytic therapy may not be a hemorrhagic complication or reperfusion injury. The role of increased collagen breakdown due to increased plasmin levels remains unclear. This mechanism requires further investigation, although the low incidence of this complication makes this difficult.

Three (18.75%) of the surviving 16 patients have a residual VSD detected postoperatively. We did not detect an increase in mortality associated with a residual shunt. This observation is in agreement with Deja and colleagues and Davies *et al.*[17,29] Two of the patients with a residual VSD are currently in NYHA class I-II and one in NYHA class III.

In our series, 3 out of 6 patients who died (50%) and 2 out of surviving 15 patients (13.5%) had preoperative renal failure. RRT was used in 4 patients. Preoperative renal failure was found to be strongly associated with mortality ($P = 0.02$).[11]

Short-term mortality with post-MI-VSD is significant. However, those who survive the surgical repair often have a good long-term functional outcome. Indeed, in our series with a mean follow-up of 5 years, all 16 patients who survived until discharge are alive. On EQ-5D questionnaire: 15 patients indicated having no problems in any of the dimensions and 1 reported moderate discomfort as well as anxiety/depression and pain. Our findings are supported by a few of the studies that reported favorable long-term functional outcomes on successful post-MI-VSD.[17,18,24,29,33]

The small sample size is the limitation of our study and therefore lacks statistical power to identify potential risk factors contributing to early mortality and residual VSD.

Since the occurrence of postinfarction VSR is relatively rare, the small sample size underpowered the statistical analysis and could have limited the number of statistically significant variables. A prospective multicenter study incorporating a larger sample size would be useful to assess the prognostic value of the risk factors identified.

Surgical correction of this condition has been associated with significant adverse outcomes. Despite this, good midterm functional outcomes, quality of life, and survival are observed in patients discharged from the hospital. Residual VSD is a relatively common complication post-surgery; however, this may not adversely affect survival and does not appear to affect the midterm functional status if the shunt is small.

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Conflicts of interest

There are no conflicts of interest.

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