Original Article
Ventricular septal rupture complicating acute myocardial infarction in the modern era with mechanical circulatory support: a single center observational study

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Abstract: Ventricular septal rupture (VSR) is a rare but devastating complication after acute myocardial infarction (AMI). While the incidence has decreased, the mortality rate from VSR has remained extremely high. The use of mechanical circulatory support with intra-aortic balloon pump (IABP) and extracorporal membrane oxygenation (ECMO) may be useful in providing hemodynamic stability and time for myocardial scarring. However, the optimal timing for surgical repair remains an enigma. Retrospective analysis of 14 consecutive patients diagnosed with VSR after AMI at Montefiore Medical Center between January 2009 and June 2015. A chart review was performed with analysis of baseline characteristics, hemodynamics, imaging, percutaneous interventions, surgical timing, and outcomes. The survival group had a higher systolic BP (145 vs 98, p<0.01), higher MAP (96 vs 76, p=0.03), and lower HR (75 vs 104, p=0.05). Overall surgical timing was 6.5 ± 3.7 days *after indexed myocardial infarction* with a significant difference between survivors and non-survivors (9.8 vs 4.3, p=0.01). The number of pre-operative days using IABP was longer in survivors (6.5 vs 3.2, p=0.36) as was post-operative ECMO use (4.5 vs 2 days, p=0.35). The overall 30-day mortality was 71.4% with a 60% surgical mortality rate. Hemodynamics at the time of presentation and a delayed surgical approach of at least 9 days showed significant association with improved survival. Percutaneous coronary intervention (PCI) was more common in non-survivors. The use of IABP in the pre-operative period and post-operative ECMO use likely provide a survival benefit.

Keywords: Ventricular septal rupture, acute myocardial infarction, cardiogenic shock, mechanical circulatory support, ventricular septal rupture repair, surgical mortality

Introduction

Prior to the use of thrombolysis and percutaneous coronary intervention (PCI), the incidence of VSR after AMI was as high as 1-3% [1-5]. After reperfusion therapies became the standard of practice in the treatment of AMI, the incidence of VSR decreased to 0.17-0.31% [6-9]. However, despite the improvements in early diagnosis and treatment of both AMI and VSR, the mortality rate from VSR remains extremely high ranging from 45-80% [6-9].

As was shown in the SHOCK trial and validated by GUSTO-I and APEX-AMI, VSR typically occurs much earlier ranging from 8-24 hours after AMI and did not significantly differ in those receiving thrombolysis from those who did not [6-8, 10]. However, the improved early detection of VSR may be the result of other factors including near universal access to echocardiography and alterations in tissue pathology as a result of reperfusion injury combined with fibrinolysis [6, 11, 12]. Despite improvements and promising results in non-surgical managements of VSR such as transcatheter closure devices [13], surgical repair of septal defects remain the cornerstone of treatment.

Current guidelines from the American College of Cardiology Foundation and American Heart Association (ACCF/AHA) recommend emergent

surgical repair regardless of hemodynamic stability at the time of diagnosis [14]. Despite expert agreement on the necessity of surgical repair, the timing of VSR repair and perioperative therapeutic management remains controversial [1, 2, 4, 15-18]. This study will compare the profile, risk factors, and outcomes of the various treatment options in the management of VSR with particular interest in the use of PCI, circulatory assist devices and surgical timing.

Methods

Study design and patient selection

A retrospective analysis was performed on 14 consecutive patients with VSR following AMI who presented or transferred to Montefiore Medical Center between the January 2009 and June 2015. A total of 4 patients were transferred from outside facilities with only 1 having prior diagnosis of VSR obtained at the presenting hospital. Inclusion criteria were any patient admitted for AMI who had emergent cardiac catheterization with evidence of VSR or had hemodynamic compromise with echocardiographic evidence of VSR. Patients who died on initial presentation were excluded, including those who were taken for emergent cardiac catheterization but did not survive. Diagnosis of AMI was made based on clinical symptoms and elevation of serum troponin-T > 0.1 mg/dL with or without electrocardiogram (EKG) evidence of > 2 mm ST-segment elevation in the precordial leads or > 1 mm ST-segment elevation in the limb leads. We completed a thorough chart review and analysis of the clinical profile, treatments including medical and surgical, and outcomes of all patients. The earliest recorded vital signs were used to determine the clinical hemodynamics for each case.

Cardiac catheterization

After diagnosis of AMI in the emergency department, all patients were taken for emergent left heart catheterization (LHC) with placement of IABP. A total of 13 of 14 patients received coronary angiogram with the intention to perform primary intervention. These 13 patients also had left ventriculogram with a total of 9 diagnosis of VSR prior to echocardiographic confirmation. The only patient that did not receive coronary angiogram or ventriculogram had hemodynamic compromise with prior echocardiograph-

ic evidence of VSR. Coronary artery disease (CAD) was defined based the degree of obstruction. No apparent CAD was defined as no stenosis greater than 20%. Nonobstructive CAD included at least 1 or more lesions with stenosis greater than 20% but less than 70%. Obstructive CAD was defined as any stenosis greater than 70% or left main stenosis greater than 50% with a distribution involving 1, 2 or 3 vessels. A total of 12 patients received a right heart catheterization. Only 4 patients underwent successful PCI with coronary stents.

Echocardiography

All patients underwent an echocardiogram with confirmation of VSR by transthoracic and/or transesophageal methods within an average of 4 hours and 36 minutes of hospital admission (maximum 13 hours and 51 minutes). Diagnosis of VSR was defined as disruption in the ventricular septum with evidence of left-to-right shunt by color Doppler. The location of the VSR was identified via transthoracic echocardiogram (TTE) and in surgical candidates was verified by transesophageal (TEE) prior to surgical intervention. VSR location was recorded as basal septum, mid septum, or distal septum. Left ventricular ejection fraction (LVEF) was calculated by either the Quinonez method or the biplane Simpson's method.

Outcomes

The primary outcome was all-cause mortality, defined as in-hospital death from any cause within 30 days after VSR diagnosis. Survival was defined by hemodynamic stability without any artificial support and with a return to cognitive and near-functional baseline. Additional clinical factors and outcomes were observed and analyzed including: baseline patient characteristics, clinical characteristics upon admission, location of coronary artery lesion, coronary interventions, left ventricular ejection fraction, location of ventricular defect, use of circulatory assist device, and surgical time and outcomes.

Statistical analysis

Continuous variables were summarized as mean plus/minus the standard deviation (SD). Categorical variables were expressed as percentage of the sample. Comparison between

Table 1. Baseline characteristics

Variable	No. of patients	Overall (n=14)
Age	14	69.8 (± 10.8)
Male (%)	8	57.1 (14)
DM (%)	6	46.2 (13)
HTN (%)	9	69.2 (13)
HLD (%)	5	38.5 (13)
Smoking (current or prior-%)	7	53.8 (13)
Previous CAD (%)	1	7.7 (13)
Preoperative coronary angiogram (%)		
-Nonobstructive	1	7.7 (13)
-One-vessel disease	4	30.8 (13)
-Two-vessel disease	4	30.8 (13)
-Three-vessel disease	4	30.8 (13)
Culprit vessel (%)		
-LAD	5	38.5 (13)
-RCA	7	53.9 (13)
-LCX	0	0.0 (13)
LVEF (%)	14	51.9 (14)
Location of VSR (%)		
-Basal	6	42.9 (14)
-Mid Septal	4	28.6 (14)
-Apical	4	28.6 (14)

CAD, coronary artery disease; DM, diabetes mellitus; HLD, hyperlipidemia; HTN, hypertension; LAD, left anterior descending; LCX, left circumflex; LVEF, left ventricular ejection fraction; VSR, ventricular septal rupture.

in-hospital survivors versus non-survivors was performed by Student's t-test and Fisher's exact test for continuous and categorical variables respectively.

Results

A total of 14 subjects were diagnosed with VSR after MI between the years of 2009 to 2015 (**Table 1**). The average of age was 69.8 years old with the majority being males (57.1%) and having hypertension (69.2%). Only 1 patient had a history of prior coronary artery disease (CAD). A coronary angiogram was completed in 13 of the 14 patients with equal number of patients divided into 1-, 2-, or 3-vessel disease categories. Only 1 patient had non-obstructive CAD. Right coronary artery (RCA) was the most common location for culprit lesions at 53.9%. LVEF at time of VSR diagnosis was 51.9% with the basal septum being the most common location for VSR at 42.9%.

Patient and clinical characteristics

Patients in the survivor group tended to be younger with lower BMI (**Table 2**). While there was no significant difference between the groups, survivors had a higher incidence of baseline hypertension, hyperlipidemia, diabetes and smoking history. The 1 patient with prior CAD was a non-survivor. Initial systolic blood pressure (BP) was significantly higher in the survival group (145 vs 98, p<0.01) but there was no difference in diastolic BP. High mean arterial pressure (MAP) and low heart rate (HR) was seen in the survival group (96 vs 76, p=0.03 and 75 vs 104, p=0.05). Non-survivors were more likely to have 3-vessel CAD (0% vs 44.4%, P=0.23) with a culprit lesion in the LAD (0% vs 55.6%, P=0.10), while most survivors were more likely to have culprit lesions in the RCA (100% vs 33.3%, P=0.07). A right heart catheterization (RHC) was performed on all survivors but only 88.9% of non-survivors. There was no difference in LVEF at the time of diagnosis of VSR (54.5% vs 50.9%, p=0.62). VSR occurring in the basal septum was more common in survivors (75% vs 30%, P=0.24) while VSR in the distal septum was more common in non-survivors (40% vs 0%, P=0.25).

Interventions: PCI, mechanical circulatory support, surgical repair

A total of 4 patients received PCI during coronary angiogram (30.8%), all of which were in the non-survival group (0% vs 44.9%, P=0.23, **Table 3**). No patients underwent thrombolytic therapy. All patients were placed on IABP during day 1 of hospital admission at the time of LHC. Attempted closure of VSR by percutaneous closure devices occurred in 2 patients, all occurring in the non-survival group (0% vs 20.0%, P=1.0). Overall surgical timing was 6.5 ± 3.7 days with a significant difference between survivors and non-survivors (9.8 vs 4.3, p=0.01). Use of circulatory support was common among both groups with IABP used in all patients at the time of initial LHC for 4.1 ± 3.4 days. There was a non-significant difference in days of IABP use between survivors and non-survivors (6.5 vs 3.2, p=0.36). Post-operative use of IABP was 1.0 ± 1.4 days and was similar between the survival groups (0.75 vs 1.1, p=0.63). ECMO use occurred in 28.6% of patients and more commonly used in survivors than non-survivors (50% vs 20%, P=0.52). ECMO was used for a total of 10 ± 1.7 days in all patients, similar

Table 2. Comparison of clinical characteristics

	Survivors (n=4)	Nonsurvivors (n=10)	P value
Age (years)	67.5 (4)	70.7 (10)	0.69
Male Gender (%)	50.0 (4)	60.0 (10)	0.78
BMI	25.0 (4)	28.2 (10)	0.27
Hypertension (%)	100 (4)	55.6 (9)	0.04*
Hyperlipidemia (%)	50.0 (4)	33.3 (9)	0.64
Diabetes (%)	50.0 (4)	44.4 (9)	0.88
Smoking (%)	75.0 (4)	44.4 (9)	0.35
CAD (%)	0 (4)	11.1 (9)	0.34
Systolic BP	145 (3)	98 (9)	<0.01*
Diastolic BP	71 (3)	62 (8)	0.24
Mean Arterial Pressure	96 (3)	76 (8)	0.03*
Heart Rate	75 (3)	104 (8)	0.05*
CAD Significance (%)			
Non-Obstructive	0.0 (4)	11.1 (9)	1.0
1V-CAD	50.0 (4)	22.2 (9)	0.53
2V-CAD	50.0 (4)	22.2 (9)	0.53
3V-CAD	0.0 (4)	44.4 (9)	0.23
Diseased Coronaries (%)	1.5 (4)	2.0 (9)	0.31
RCA Culprit	100 (4)	33.3 (9)	0.07‡
LAD Culprit	0.0 (4)	55.6 (9)	0.10‡
LCx Culprit	0.0 (4)	0.0 (9)	--
Right Heart Catheterization (%)	100 (3)	88.9 (9)	1.0
LVEF (%)	54.5 (4)	50.9 (10)	0.62
VSR Location (%)			
Basal septum	75.0 (4)	30.0 (10)	0.24
Mid septum	25.0 (4)	30.0 (10)	1.0
Distal septum	0.0 (4)	40.0 (10)	0.25

BMI, body mass index; BP, blood pressure; CAD, coronary artery disease; LAD, left anterior descending; LCX, left circumflex; LVEF, left ventricular ejection fraction; VSR, ventricular septal rupture. *Statistically significant. ‡Trend towards significance.

between the survival groups (11 vs 9, p=0.42). There was a non-significant difference in postoperative ECMO use between survival groups occurring 4.5 versus 2 days (p=0.35).

Survival

The overall 30-day mortality after VSD was 71.4% with surgical mortality of 60% (6 of 10 patients). Of the 4 patients who did not undergoing surgical intervention initially, 1 had percutaneous closure device insertion with an overall mortality of 100% in the non-surgical group. One patient had both percutaneous closure followed by surgical repair but did not survive.

Discussion

Our study confirmed previous observations that delayed surgical intervention, specifically occurring 9 or more days after VSR diagnosis,

was associated with a significant survival benefit. In addition, we demonstrated that use of mechanical circulatory support with both IABP and ECMO was more common and for a longer duration in the survival group but provided no significant survival benefit. Our study also shows that culprit lesions in the RCA demonstrated a near-significant survival benefit while early reperfusion with coronary intervention provided no survival benefit. High systolic BP, high MAP and low heart rate at the time of VSR diagnosis was similarly associated with improved survival benefit.

Patient and clinical characteristics

Younger age, hypertension, hyperlipidemia, diabetes and prior smoking history were all more common in the survival group but none were statistically significant. There were no significant differences in baseline characteristics between the survival groups. A left heart catheterization (LHC) with diagnostic coronary angiogram was performed on 13 of the 14 patients with a total of 4 patients receiving PCI (1 survivor and 3 non-survivors). Patients were more likely to have coronary disease at the time of VSD diagnosis with equal distribution between 1, 2, or 3-vessel CAD. A culprit lesion in the LAD was more common in the non-survival group while culprit RCA lesions more common in survivors showing a trend toward survival benefit. The location of coronary lesions and corresponding area of infarction contradicted many prior investigations including the SHOCK and GUSTO-I trial which showed LAD lesions and anterior infarctions as having better outcomes [6, 10].

Percutaneous coronary intervention

Early reperfusion with PCI was found only in the non-survival group indicating a possible increased mortality association; this difference however was not statistically significant. A worse outcome with early reperfusion contradicts both the GUSTO-I and GRACE trials which showed lower incidence for VSR with early reperfusion by thrombolysis or PCI [6, 8]. However, patients who were treated with PCI

Table 3. Interventions between survival groups

	All (n=14)	Survivors (n=5)	Nonsurvivors (n=9)	P value
Coronary Intervention (%)	30.8 (13)	0.0 (4)	44.4 (9)	0.23
Percutaneous Closure Device (%)	14.3 (14)	0 (4)	20.0 (10)	1.0
Days to Surgery	6.5 ± 3.7	9.8 (4)	4.3 (4)	0.01*
IABP Use (%)	100 (14)	100 (4)	100 (10)	–
Total Days	4.1 ± 3.4	6.5 (4)	3.2 (10)	0.35
Post-OP Days	1.0 ± 1.4	0.75 (4)	1.1 (10)	0.63
ECMO Use (%)	28.6 (14)	50.0 (4)	20.0 (10)	0.52
Total Days	10 ± 1.7	11.0 (2)	9.0 (2)	0.42
Post-OP Days	3.3 ± 1.9	4.5 (2)	2.0 (2)	0.35

IABP, intra-aortic balloon pump; ECMO, extracorporal membrane oxygenation; OP, operative. *Statistically significant. ‡Trend towards significance.

after MI similarly had a higher mortality in both the SHOCK and APEX-AMI trials [7, 10]. This possibly infers that while early reperfusion decreases the incidence of VSR it increases the overall mortality if VSR complications do occur.

Mechanical circulatory support

Percutaneous circulatory assist devices were used on all patients with an intra-aortic balloon pump (IABP) placed in all 14 patients at the time of LHC. Of the surgical patients, a total of 3 of 10 patients remained on cardiopulmonary bypass (CPB) via extracorporeal membrane oxygen (ECMO) in the post-operative period. Our observations found that use of circulatory assist devices was common among both groups with IABP being the most commonly used device. Use of IABP was universal between the survival groups; however, the pre-operative duration of IABP was longer in the survival group. While there was no statistically significant association with survival benefit, ECMO use and timing was more commonly seen in the survival group.

Much of the data on the use of IABP is limited to cases studies or other small retrospective studies which show that IABP improved survival after VSR until surgical repair was performed [19, 20]. However, in a large retrospective review of 2,876 patients from The Society of Thoracic Surgeons (STS) Adult Cardiac Surgery Database (ACSD) by Arnaoutakis et al., the use of IABP both preoperative and intraoperative had significantly higher mortality outcomes [21]. The data on ECMO use in VSR has also

been limited to cases studies and to date there has not been any significant prospective or retrospective studies evaluating the mortality benefits in VSR after MI [22-25]. Use of IABP in cardiogenic shock from VSR is recommended according to the ACC/AHA guidelines [14]. Our results support the fact that both IABP and EMCO likely provide survival benefit through hemodynamic support in the pre-operative period and post-operative benefits with the use of EMCO.

Surgical repair and timing

A total of 10 patients underwent surgical repair with surgical timing ranging from day 1 to day 12 of hospital admission. Two patients underwent percutaneous closure. Emergent surgical intervention was performed on 2 patients while the remaining patients had variable timing of surgical repair. According to the guidelines by the ACCF/AHA, emergent surgical repair is required in all VSR patients regardless of hemodynamic stability given the risk for rapid expansion and hemodynamic collapse in stable patients [14]. We observed that delayed surgical intervention occurring more than 9 days post-VSR diagnosis was associated with a significant improvement in survival. In the STS-ACSD study, early repair occurring less than 7 days post-MI had much greater mortality than delayed repair occurring more than 7 days post-MI (54.1% vs 18.4%, p<0.01) [21].

Survival in the STS-ACSD study was associated with young, males, smokers with baseline hypertension, lung disease and less kidney dysfunction [21]. While not statistically significant, the observations from our study showed a similar profile in younger, female, smokers with hypertension being more common in the survival group. The overall operative mortality from our study was 60% which is much greater than the STS-ACSD study which was 42.9% and had the greatest mortality in patients who underwent emergent surgical repair after VSR (less than 24 hours).

There are two leading theories for improved survival in delayed surgical repair. The first theory suggests that delayed surgical repair gives the friable, post-MI myocardial tissue enough time to fibrose and strengthen which allows for optimal repair. The second theory is that improved survival may represent a selection bias of patients with clinically stable hemodynamics who are at lower surgical risk into the delayed repair group and unstable patients into the emergent/urgent repair group. Given that our survival group had more stable hemodynamics at the time of VSR diagnosis and ultimately resulted in a delayed surgical repair, this may indicated possible selection bias.

There are a few limitations of this study that should be mentioned. Firstly, a small population of patients with diagnosis of VSR resulted in inadequate statistical power despite obvious differences in the outcome groups. The retrospective nature of the study restricts the control of confounding factors and prevents the ability to infer that obvious associations demonstrate causality. Clinical decisions on treatment were not controlled and based solely on expert judgment. The surgical techniques varied based on performing surgeons and were not reviewed in this study. Lastly, many patients likely had VSR prior to coronary angiography therefore any intervention at the time of cardiac catheterization precludes any associations to be made between reperfusion and the development and outcomes of VSR.

In conclusion, we have shown that stable hemodynamics at the time of VSR diagnosis is a significant predictor of survival. Early reperfusion by PCI was more common in the non-survivors and suggested a possible association with increased mortality. Use of circulatory support devices such as IABP and ECMO may provide a survival benefit which we believe would be significant if a larger population was analyzed. Delayed surgical repair of VSR lesions has a significant association with improved mortality. We conclude that in patients who are able to maintain hemodynamic stability with and without medical support, a delayed surgical approach with use of circulatory support is the preferred method for VSR repair.

Disclosure of conflict of interest

None.

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