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*Case Report*

# Percutaneous repair of post-myocardial infarction ventricular septal rupture presenting with cardiogenic shock

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**Abstract:** Ventricular septal rupture (VSR) is an uncommon and devastating complication with a high mortality rate due to limited available interventions required by expert hands in a small window of opportunity. Most commonly seen following delayed myocardial infarctions (MI), the rate of VSR has decreased partly from protocol driven reperfusion therapy; however, cases are still present, particularly when diagnosis is delayed. We present a case of a critically ill patient in cardiogenic shock following a large anterolateral wall ST-elevation MI complicated by a large VSR whom was transferred to our academic institution for percutaneous repair. Of note, such intervention was initially performed by Lock in 1988 and a comprehensive review published in 2016 noted only 273 such cases. This review noted patient cases since that initial percutaneous closure by Lock with a majority of cases utilizing an Amplatzer system; others being Clamshell and CardioSEAL. Our patient underwent the percutaneous VSR closure utilizing an Amplatzer Occluder delivery system with successful insertion of an 18 mm muscular VSD Amplatzer closure device. Although the rarely performed procedure was successful and provided invaluable insights into the treatment and management of VSR, the patient succumbed to multiple critical disease processes in the following days post intervention. Patient consent and ethics committee approval for publication, as per Saint Louis University case publication guidelines, were confirmed and approved.

**Keywords:** Ventricular septal rupture, ST-elevation myocardial infarction, interventional cardiology, occluder device, septal surgery, intracardiac shunt, cardiogenic shock, ventricular assist device, percutaneous repair, critical care

## Introduction

The advances with reperfusion therapy have shown significant and lifesaving alterations to patients presenting with acute MI, making catastrophic adverse events, such as VSR, seen less commonly. These unfortunate events are still seen in patients whom have a delayed presentation and/or intervention leading to ischemia related myocardial tissue breakdown and rupture of the septum, free wall or papillary muscles. Interestingly, VSR are more often seen in patient's suffering their first MI and have limited co-morbidities that would "protect" these patients via an increase in collateral circulation. Commonly in these situations, the patient is critically ill necessitating advanced circulatory support in addition to inotropic and vasopressor therapies. While the traditional treatment strategy has been delayed surgical repair [2] to correct the shunt and provide adequate cardiac output, few have attempted percutaneous repair of an acute rupture, which we discuss here.

## Case

A 71-year-old man with significant medical history notable only for tobacco usage presented to an outside hospital with mid-sternal chest pain lasting over 24 hours and acutely worsening dyspnea. There he was diagnosed with an anterolateral ST-elevation MI complicated by cardiovascular collapse. He underwent primary

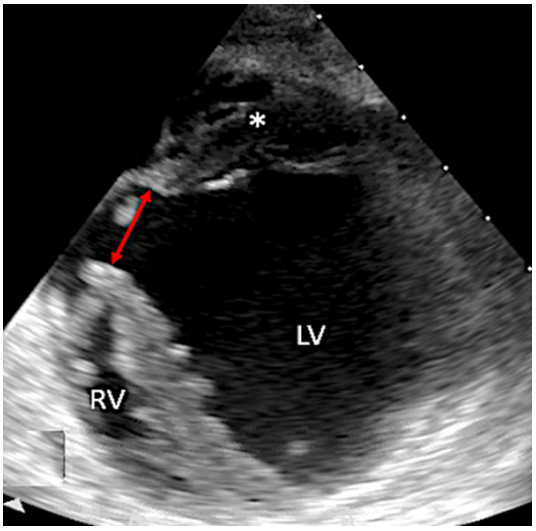


**Figure 1.** TEE visualization of VSR. Large apical muscular VSR as depicted by red arrow and LV aneurysm (asterisk) on TEE imaging via a transgastric mid SAX view. RV, right ventricle, LV, left ventricle.

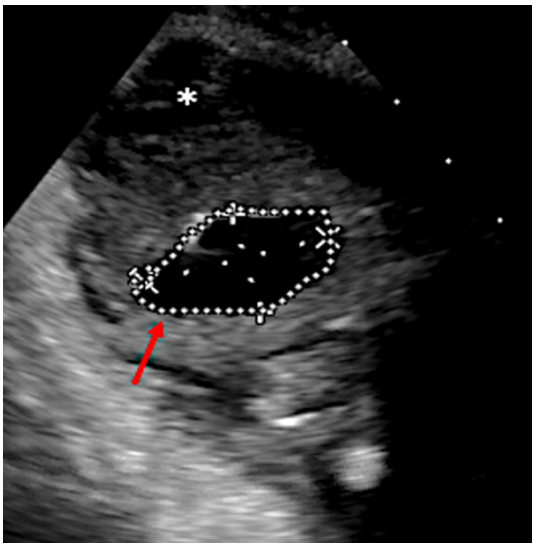


**Figure 2.** Septal wall view of VSR. TEE view of left ventricular septal wall with outlined VSR (red arrow) measuring 14 mm × 24 mm and LV aneurysm (asterisk).

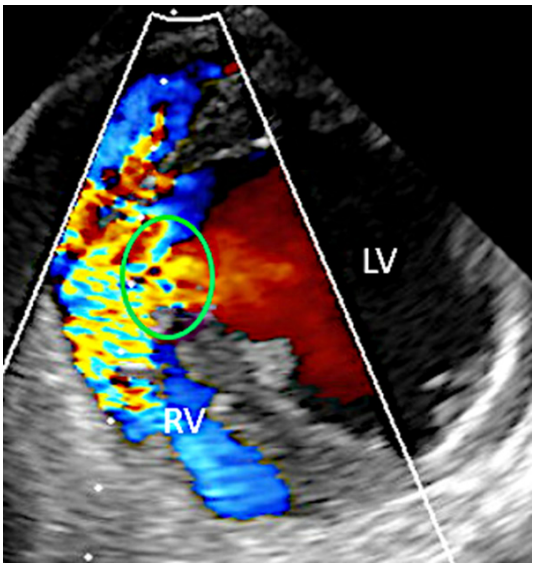


**Figure 3.** Left to right shunt. A pure left-to-right shunt across the ventricular septum, as seen on color doppler via TEE transgastric view, through the VSR (circle). LV, left ventricle, RV, right ventricle.

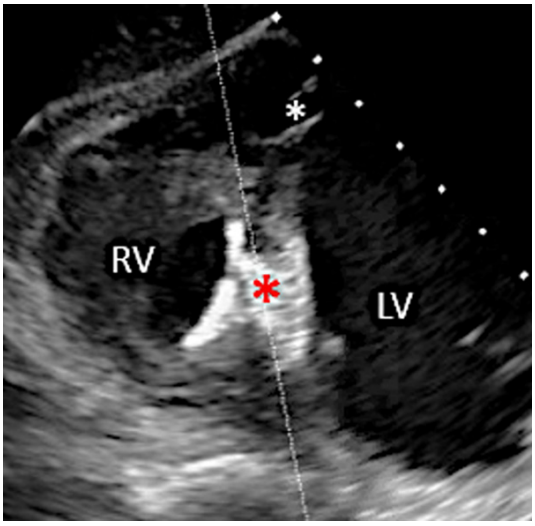


**Figure 4.** VSR post repair. Transgastric basal SAX TEE view following VSR repair with placement of 18 mm Amplatzer occluder (red asterisk). LV, left ventricle, RV, right ventricle.

percutaneous coronary intervention (PCI) for complete occlusion of mid-LAD along with placement of an Impella 5.0 for left ventricular (LV) unloading and circulatory support. Following this, transthoracic echocardiogram (TTE) revealed a large anteroseptal and apical infarction with aneurysm, LV thrombus, and large apical funnel type VSR (LV side 14 mm × 24 mm (**Figures 1** and **2**), RV side 5 mm × 10 mm) with pure left-to-right shunting (Qp/Qs >3; **Figure 3**) and severe pulmonary arteriole hypertension with a pulmonary artery systolic blood pressure (PASP) of 74 mmHg.

He was transferred to our facility for further management where he continued to deteriorate clinically despite medical and mechanical circulatory support therapies, and was deemed

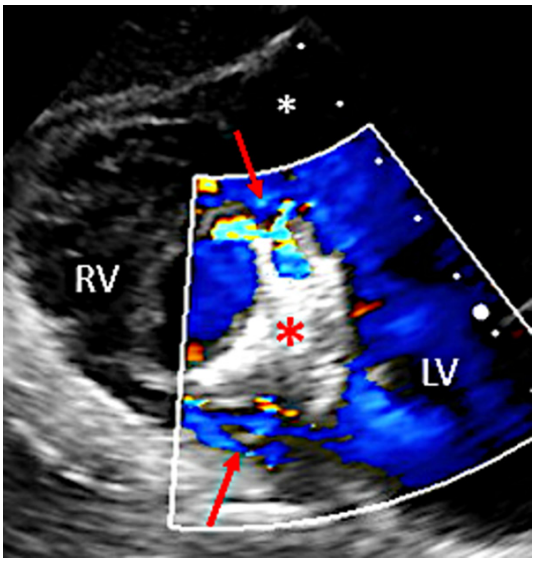


**Figure 5.** Post repair color doppler. Color doppler in transgastric basal SAX TEE window depicting near cessation of left-to-right shunt flow (arrows) following placement of Amplatzer occluder (red asterisk) in repair of VSR. LV, left ventricle, RV, right ventricle.

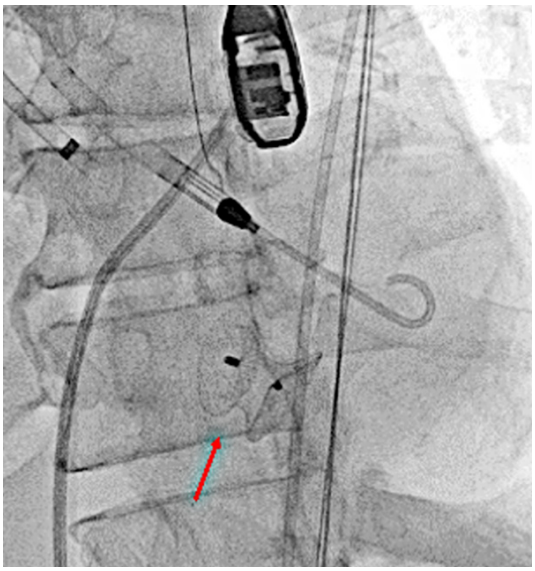

**Figure 6.** Fluoroscopy following repair. Fluoroscopic visualization of Amplatzer occluder (red arrow). Also visualized are the TEE probe, guide wire and PA catheter (not labeled).

too high risk to undergo traditional open-heart surgical VSR repair. After a multidisciplinary heart team discussion, the decision was made to perform percutaneous closure of the VSR.

The SR was approached from the LV side with left femoral artery access. A Glidewire advantage wire was used to cross the VSR into the RV cavity. Next, from the left internal jugular vein (IJV), the Amplatzer occlude delivery system was advanced into the right atrium over a J-tipped guide wire. Due to tortuous angulation from RV to PA, a balloon tipped pulmonary artery (PA) catheter was advance into the PA and then a V 18 wire was used to maintain PA position as the PA catheter was removed. A diagnostic multipurpose catheter was then used to exchange the V 18 wire for an Amplatzer Super Stiff wire. The Amplatzer occluder delivery system was then advanced over the Amplatzer Super Stiff wire into the main pulmonary artery. The Glidewire Advantage was then snared in the main PA and then externalized through the distal end of the Amplatzer Occluder delivery system from the left IJV. The Amplatzer Occluder delivery system was then advanced across the VSR and into the LV. An 18 mm muscular VSD Amplatzer closure device was then deployed across the VSR with good tissue bridge seen in all rims and near complete cessation of shunt flow (**Figures 4**-**6**) with significant improvement in PASP to 41 mmHg.

Unexpectedly, 1-day later, he developed worsening hypoxia with elevated ventilator inspiratory and plateau pressures, and PASP increasing to 81 mmHg. He was found to have pulmonary hemorrhage from his right lung possibly related to wire injury during the procedure. Urgent bronchoscopy revealed heavy clot burden despite aggressive suctioning. He was not able to be oxygenated despite high ventilator pressure, with decision being made to manually ventilate with an ambu bag which ultimately led to pulmonary rupture. He quickly developed refractory ventricular fibrillation and soon expired.

## Discussion

Ischemic ventricular wall rupture is a rare but lethal complication, as seen in **Table 1**, occurring in <0.5% of MIs [2, 3]. With timely reperfusion therapy such as fibrinolytics and PCI, the incidence of VSR has been reduced from 1%-3% down to 0.2%-0.5% [4]. A large study by Thiele et al. investigating patients presenting with post MI VSR and cardiogenic shock, found the 30-day mortality rate was 65% with long term mortality rate of 93%. Patients who pre-

**Table 1.** Literature Review

| References (Manuscript titles) | Key findings |
|---|---|
| Interventional post-myocardial infarction ventricular septal defect closure: a systematic review of current evidence (Schlotter F, et al.) [1] | ● Reviewed 13 series, 273 patients who underwent percutaneous device closures of post-infarction VSD<br>● 89% rate of successful device implantation<br>● >75% technical success rate<br>● 32% overall in-hospital/30 day mortality with a range of 36 to 81%<br>● 16.5% of patients underwent secondary percutaneous approach following primary surgical VSD closure<br>● Complications included arrhythmias (atrioventricular blocks, ventricular tachycardia/fibrillations), embolization of device into pulmonary artery leading to death, ventricular rupture and hemolysis following repair |
| Percutaneous repair of post-myocardial infarction ventricular septal defect: current approaches and future perspectives (Baldasare MD, et al.) [2] | ● 2 cases of both primary and secondary percutaneous closure of post-infarction VSD<br>● 1st patient presenting with chest pain and dyspnea, found to have multi-vessel disease and VSD underwent successful coronary artery bypass grafting (CABG) and VSD repair. One week later found to have residual VSD requiring successful percutaneous closure with a 28-mm CardioSEAL device. On 1 year follow up had worsening heart failure requiring heart transplant<br>● 2nd patient presenting 3 weeks post anterior MI found to have VSD in cardiogenic shock requiring percutaneous closure with a 18-mm Amplatzer Septal Occluder. Repeat imaging showed residual VSD and patient underwent surgical closure 2 weeks following |
| Percutaneous repair of ventricular ruptures (Bing R, et al.) [3] | ● Three cases of percutaneous repair of ventricular ruptures<br>● 1st case of a patient presenting 6 months post inferior MI found to have large inferobasal LV pseudoaneurysm for which a 11-mm Amplatzer Septal Occluder was successfully placed<br>● 2nd case two months post mitral valve implantation presenting with heart failure and enlarging, pulsatile chest wall mass found to have a bidirectional flow into a large pseudoaneurysm at the LV cannulation site; closure obtained with a 16-mm Amplatzer PI Muscular VSD Occluder<br>● 3rd case patient with inferior MI and inferior wall LV wall defect with left to right flow underwent closure with a 14-mm Amplatzer VSD occluder |
| Primary transcatheter closure of post-myocardial infarction ventricular septal rupture using amplatzer atrial septal occlusion device: A study from tertiary care in South India (Aggarwal M, et al.) [4] | ● Retrospective, observational, single center review of 21 patients undergoing percutaneous post-MI VSD closure (20 with Amplatzer, 1 with Cera occluder devices)<br>● 13 patients (62%) had residual defect<br>● All cause mortality at 30 day follow up was 42.9%<br>● Time to VSR closure, diameter of VSR and creatinine levels were found to be significantly related to 30 day mortality |
| Immediate primary transcatheter closure of postinfarction ventricular septal defects (Thiele H, et al.) [5] | ● Reviewed 29 patients undergoing primary percutaneous VSD closure<br>● Initial procedural success rate of 86%<br>● 30 day survival of 35%<br>● Patients with cardiogenic shock had significantly higher mortality vs non-shock (88 vs 38%) |
| Ventricular Septal Rupture (Mubarik A) [6] | ● In depth overview of VSD including etiology, epidemiology, pathophysiology, history and physical findings, evaluation, treatments with complications and prognosis |
| Posterior ventricular septal defect in presence of cardiogenic shock: early implantation of the Impella recover LP 5.0 as a bridge to surgery (La Torre MW, et al.) [7] | ● Evaluation of 5 patients between 2004 and 2007 with cardiogenic shock and VSD undergoing surgical repair and early Impella Recover LP 5.0 Support System utilization<br>● 1 patient required transplantation due to inability in surgical repair<br>● Mean duration of Impella support 14.4 days with no deaths during<br>● 30 day mortality 40% |

sented with post MI VSR without cardiogenic shock had a long term mortality of 36% [5].

The ventricular septum is made primarily of two distinct parts, the lower more muscular portion and the upper thinner membranous portion. Any part of the ventricular septum can rupture with subsequent hemodynamic consequences dependent on location, size, and/or impact on cardiac mechanics, often occurring within one week following MI [6]. Apical VSR is usually seen with left anterior descending (LAD) infarctions as it supplies most of the anterior portion of the interventricular septum, while basal VSR from either right coronary (RCA) or dominant circumflex artery (LCx) infarction as they supply the most inferior portion of the interventricular septum. Post MI VSR usually presents with hemodynamic decline leading to cardiogenic shock due to bi-ventricular volume overload [6] as shunted blood to the right ventricle (RV) then returns to the LV increasing end diastolic volume. This is coupled with reduced forward cardiac output across the aortic valve into systemic circulation due to shunting across the VSR. The goal of treatment is expeditious correction of left-to-right shunting to minimize systemic hypoperfusion, multi-organ failure, and death [4].

Large VSRs typically necessitate earlier corrective therapies. Traditionally, surgical intervention has been a class 1 recommendation for post MI VSR [2, 4]. However, in the acutely ill presentation, this approach may not be optimal due to soft and friable tissue surrounding the VSR not amendable to suturing, along with severe hemodynamic instability that may be prohibitive to operative interventions. Additionally, residual shunts occur in 10%-37% of surgically closed VSRs with 11% needing repeated surgical intervention [2]. Better outcomes have been observed in patients who were more hemodynamically stable and who had time for tissue scaring around VSR.

A relatively new treatment approach has been percutaneous transcatheter VSR repair using an occluder device. While this approach has been reported in a handful of case reports, it has shown promising results in less critically ill patients as a bridge to eventual surgical correction. With concurrent comorbid conditions, including cardiogenic shock, the mortality rate remains high.

In certain patients, the use of circulatory support can minimize left-to-right shunting and improve forward cardiac output and tissue perfusion. La Torres et al. studied five patients with post MI posteriorly located VSR who presented with cardiogenic shock and who were supported with Impella Recover LP 5.0. All patients had hemodynamic recovery after mean 6-days of ventricular support with minimal complications [7]. Similarly, Aggarwal et al. studied 21 patients who had anterior wall MIs with VSR size ranging from 10 mm to 30 mm treated with primary transcatheter VSR closure. 9 patients died within 30-days due to systemic hypoperfusion followed by cardiogenic shock. They found an overall 42.9% 30-day mortality rate.

Our patient presented to our facility 3 days after having a large anterolateral wall MI with a large apical VSR complicated by cardiogenic shock status post PCI, multiple vasopressor therapies, and Impella placement. He was critically ill, but it was thought that correcting his large VSR may stabilize his overall clinical picture with hopes for recovery. Unfortunately, he developed significant hypoxic respiratory failure secondary to an assumed intra-procedural complication causing pulmonary hemorrhage leading to ventricular fibrillation and ultimately death.

## Conclusion

Patients presenting with post-MI VSR and cardiogenic shock are critically ill with limited treatment options and high mortality rates. Traditionally, open-heart surgery has been recommended; however, few cases have resulted in long term success following a successfully repaired percutaneously. This case highlights the critical nature of post-MI VSR, the potential complications, and various therapeutic options.

## Disclosure of conflict of interest

None.

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