

A case of ventricular septal rupture complicating acute myocardial infarction – A rare lethal entity posing big challenge to surgeon

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ABSTRACT

Rupture of the ventricular septum, a rare but potentially fatal complication of acute myocardial infarction, is usually observed in the setting of acute myocardial infarction associated with major coronary artery occlusion. It is often accompanied by severe circulatory failure. In this paper we describe a case of a 70-year-old lady who presented with cardiogenic shock 7 days after acute myocardial infarction and was found to have ventricular septal rupture.

Keywords: *Acute myocardial infarction; Ventricular septal rupture.*

INTRODUCTION

Ventricular septal rupture (VSR) is an uncommon but lethal condition complicating acute myocardial infarction (AMI). The incidence was 1-2% in the pre-thrombolytic era but has declined to 0.17-0.31% with the evolution of acute reperfusion strategy for AMI¹. VSR should be suspected clinically in patients with recent myocardial infarction who present with signs of cardiac failure, a new pan-systolic murmur over the precordium. This can be confirmed on echocardiography with color flow Doppler imaging. Despite significant improvements over the last two decades in overall mortality for patients with AMI, the outcome of patients who develop VSR remains poor.

CASE REPORT

A 70-year-old diabetic hypertensive non-smoker lady admitted in a local hospital in

Chittagong with the complaints of central chest discomfort with dyspnea for 2 days. Electrocardiogram (ECG) showed ST elevation in lead II, III, AVF and V1-V6. Serum Trop-I was also high. Transthoracic echocardiography (TTE) revealed severe left ventricular (LV) dysfunction (EF=28%) with akinetic anterior wall. So she was diagnosed as a case of AMI (Antero-septal) with acute LVF and was being treated medically with antiplatelets (Aspirin & Clopidogrel), anticoagulant (Rivaroxaban), diuretics (frusemide & spironolactone) and other supporting medicine. However, on 7th day of admission she became haemodynamically unstable, requiring high doses of inotropes to maintain BP. TTE was repeated and found dilated left ventricle (LV) with aneurysm like dilatation of apex and ventricular septal rupture with L-R shunt (Fig-1).

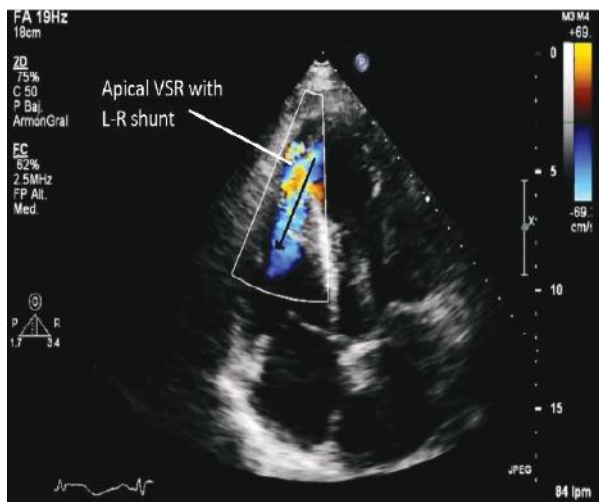


Fig. 1: TTE shows ventricular septal rupture with L-R shunt

So she was referred to a tertiary care hospital for better management. When the patient arrived in the ER of Apollo Hospitals Dhaka she was in cardiogenic shock with inotropic support. Chest auscultation revealed a pansystolic murmur loudest over the left lower sternal edge, reduced breath sounds bi-basally and inspiratory crepitations bilaterally up to the mid zone. She was shifted to CCU for further management. After 4 hours she desaturated ($SpO_2 = 82\%$) despite high flow (12 L/min) Oxygen and was put on mechanical ventilation. BP couldn't be maintained despite highest dose of inotrops. So Intra Aortic Balloon Pump (IABP) was inserted and coronary angiogram (CAG) was performed as it wasn't done before. It showed 70% narrowing in proximal left anterior descending (LAD) artery and 80% narrowing in mid LAD and tight ostial narrowing in 1st diagonal (D1) (Fig-2). Left Ventriculography showed large Ventricular Septal Rupture (VSR) (Fig-3).

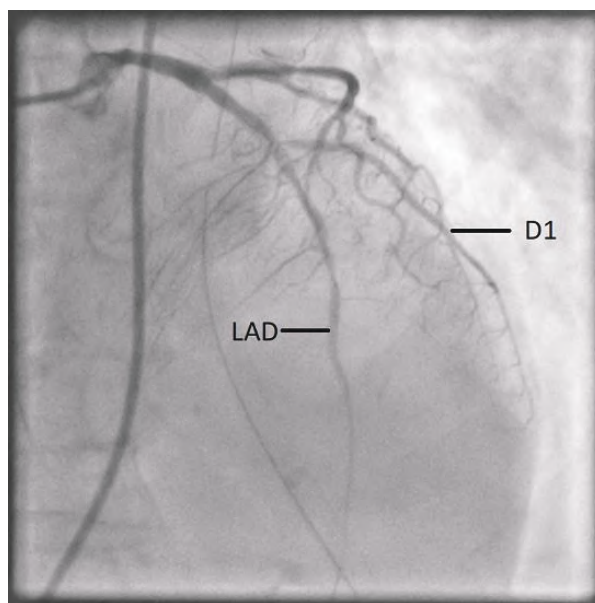


Fig. 2: CAG showed 70% narrowing in proximal left anterior descending (LAD) artery and 80% narrowing in mid LAD and tight ostial narrowing in 1st diagonal (D1)

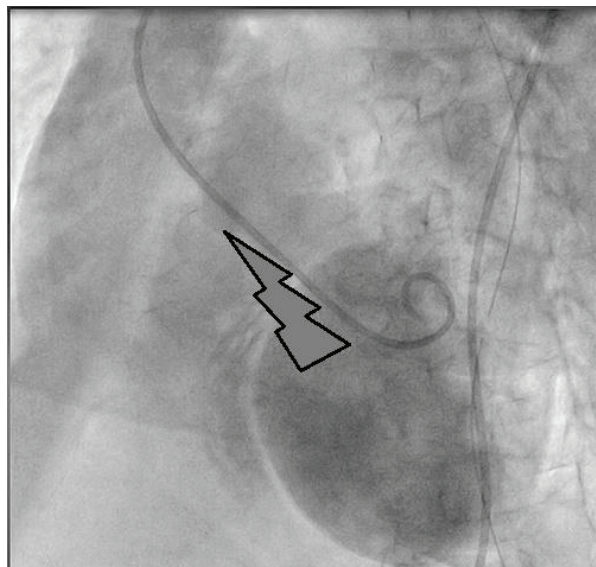


Fig. 3: Left Ventriculography showed large Ventricular Septal Rupture (VSR)

After consultation with cardiothoracic surgery team an urgent coronary artery bypass graft (CABG) and VSR repair were planned. On the day following admission she was operated and found to have dilated heart with edematous wall. LV thin walled (Aneurysm) near apex. CABG was performed, reversed saphenous vein graft (SVG) to LAD and SVG to Diagonal branch (D1). LV-tomy done through aneurysmal part. A large VSD (25 x 18 mm) was found in the mid part of the septum anteriorly. (Fig-4)

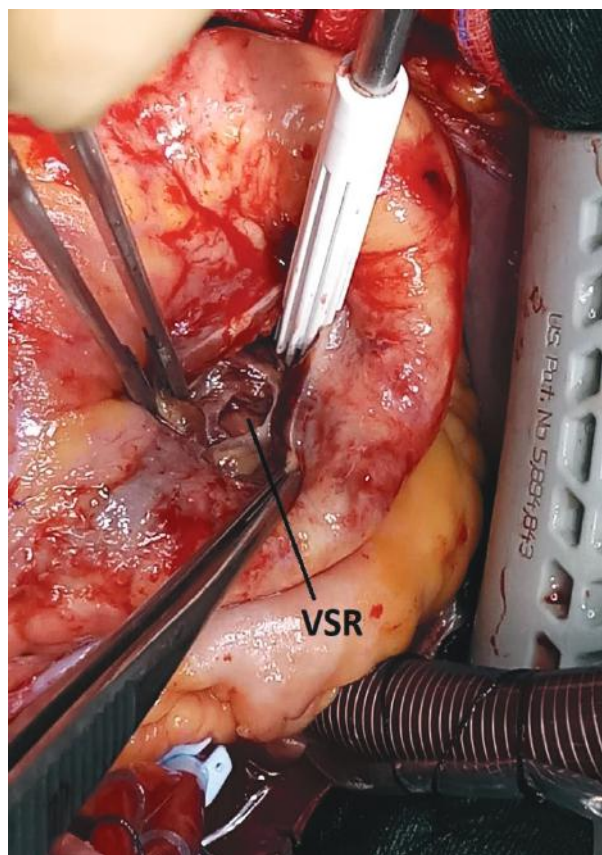


Fig. 4: A large VSD in the mid part of the septum.



Fig. 5: VSD closure by PTFE patch.

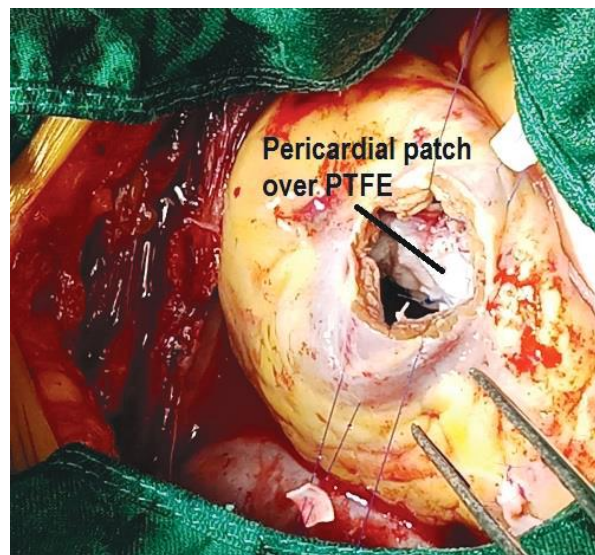


Fig. 6: VSD closure by PTFE patch with continuous suture

Glutaraldehyde treated pericardial patch was sutured over PTFE patch by continuous suture (Fig-5,6). Then LV-tomy repaired with 4/0 prolene over Teflon felt on each side (Fig-7). Post-operatively patient was shifted to ICU intubated, ventilated & keeping chest open to prevent thoracic compartment syndrome. Chest was closed in the evening on Day-1.

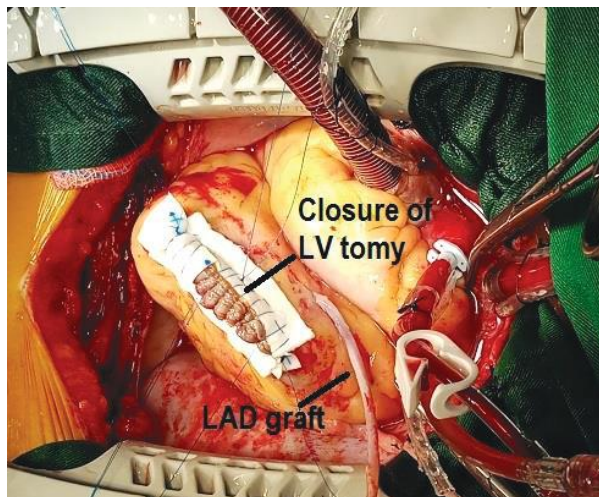


Fig. 7: Picture after repair of LV-tomy

As patient was hemodynamically stable, IABP support was withdrawn on 2nd postoperative day (POD) and patient was extubated on Day-3.

Post-operative course was devoid of significant major complication or organ failure. However it was protracted by some minor issues like dyselectrolytemia, deranged LFT, hypoalbuminaemia, hyperglycemia etc. Inotropic support was withdrawn very slowly as it took a bit longer time to improve heart failure. TTE repeated after 12 days of operation which revealed VSD patch in situ with no residual left to right shunt. (Fig-8)

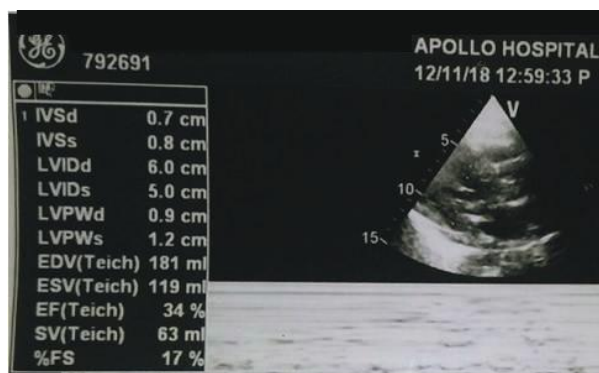


Fig. 8: TTE shows VSD patch in situ with no evidence of left to right shunt.

Patient was shifted to ward on 8th POD and discharged from hospital 19 days after operation. On subsequent visits in OPD she was found to be recovered well without any significant complication.

DISCUSSION

Rupture of ventricular septum is an uncommon but life-threatening mechanical complication of AMI. It develops after transmural infarction on inter-ventricular septum and associated with occlusion of major coronary arteries i.e. left-anterior-descending (LAD), dominant right coronary or dominant left-circumflex arteries². The underlying pathology is coagulative necrosis of ischaemic tissue causing thinning and weakening of septal myocardium. There are several independent risk factors for VSR including older age, female sex, prior stroke, chronic kidney disease, and CHF. Interestingly, patients who develop VSR are less likely to have a history of hypertension, diabetes, prior smoking, or prior MI. According to anatomical location VSR can be classified into ‘anterior or apical’ and ‘posterior or basal’ type. The former one is caused by occlusion of LAD and more common than the latter which is due to occlusion of dominant RCA. Our patient was an elderly non smoker lady with no previous history of MI but had hypertension and diabetes. In this case VSR was anterior in type and the culprit lesion was in LAD.

Clinical presentation varies from complete hemodynamic stability to frank circulatory collapse depending on the size of the defect, presence of RV infarction, ongoing RV ischaemia, or stunning of the RV from volume overload. Conventional mechanism of septal rupture through coagulative necrosis is a sub-acute process and usually requires 3-5 days. However rupture may occur within 24 hours of presentation due to dissection of an intramural haematoma or haemorrhage into ischaemic myocardium which is commonly seen in posterior/basal type. Unpredictable

hemodynamic deterioration is the norm in most patients in the days and weeks following VSR and may associate with other mechanical complications such as ventricular aneurysm, free wall rupture, or papillary muscle rupture. In our case VSR was diagnosed 7 days after the presentation of AMI when patient suddenly became hemodynamically unstable and developed cardiogenic shock. There was left ventricular aneurysm near apex as well.

The diagnosis is usually made by transthoracic echocardiogram identifying drop-out of the ventricular septum in the 2D image and demonstration of flow across the septum using colour Doppler. Evidence of right-ventricular dilation and pulmonary hypertension are also important clues to the diagnosis. Colour Doppler evaluation can also be useful to assess the anatomical size of the defect. TEE should be considered when the patient has poor acoustic windows due to mechanical ventilation or body habitus. Left ventriculography performed in the left-anterior oblique (LAO) projection in a patient with VSR may demonstrate shunting of contrast from the LV to the RV.

Surgical repair is the treatment of choice for VSR as medical management is usually futile. However operative treatment is usually challenging and associated with high mortality. Ideally, VSR would be diagnosed prior to PCI. The key point is to limit the ischaemic burden in the infarct-related artery, especially if there is right-ventricular involvement, so immediate collaboration between the interventional cardiologist and cardiac surgeon is critical to develop a case-specific approach. Timing to surgery should be decided on a case to case basis taking into account the hemodynamic stability of the patient³. As reported in the Society of Thoracic Surgeons National Database, improved outcomes were seen with delayed surgery with 54.1% mortality (surgery within 7 days) vs. 18.4% (surgery after 7 days) as those requiring

early surgery are likely sicker with greater hemodynamic instability⁴. Due to hemodynamically instability our patient underwent urgent CABG with repair of VSR within short time after having a diagnostic CAG. Principles of successful repair include debridement of infarcted tissue back to healthy myocardium (even if it involves enlarging the defect), and avoiding tension on the repair by using an appropriately sized patch⁵. There are two common techniques used for repair of VSR, the Daggett and David procedures. The Daggett is a single or double patch technique which closes the VSR by placing a patch over the defect and sewing to the RV and LV⁶. In contrast, the David technique is an infarct exclusion procedure with all sutures placed in the LV⁷. We followed Daggett double patch technique putting PTFE patch at first with interrupted pledgeted stitch which was then overlaid by Glutaraldehyde treated pericardial patch by continuous suture.

CONCLUSION

VSR complicating AMI has a high mortality no matter how intensively being treated. So we should emphasize on its prevention through early reperfusion therapies in AMI. The management of VSR is complicated, and requires substantial critical care, imaging, interventional, and surgical expertise. It is therefore advisable, when clinically feasible, to transfer these patients to regional centres with adequate individual experience in the care of patients with VSR.

REFERENCES

1. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, Krishnaswamy A. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. *Eur Heart J*. 2014;35(31):2060-2068.
2. Murday A. Optimal management of acute ventricular septal rupture. *Heart* 2003;89: 1462–6.
3. Papalexopoulou N, Young CP, Attia RQ. What is the best timing of surgery in patients with post-

- infarct ventricular septal rupture? *Interact CardiovascThorac Surg.* 2013;16(2):193-196.
4. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, Mc-Carthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. *Ann Thorac Surg.* 2012;94(2):436-443; discussion 443-434.
 5. Daggett WM, Guyton RA, Mundth ED, Buckley MJ, McEnany MT, Gold HK, Leinbach RC, Austen WG. Surgery for post-myocardial infarct ventricular septal defect. *Ann Surg* 1977;186:260–271.
 6. David TE, Dale L, Sun Z. Postinfarction ventricular septal rupture: repair by endocardial patch with infarct exclusion. *J Thoracic CardiovascSurg* 1995;110:1315–1322.
 7. Daggett WM, Buckley MJ, Mundth ED, Sanders CA, Austen WG. The role of infarctectomy in the surgical treatment of myocardial infarction. *Am Heart J* 1972;84:723–726.