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CASE REPORT
Congenital heart disease

Post-myocardial infarction ventricular septal rupture in a patient with large secundum atrial septal defect: a case report

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Background Ventricular septal rupture (VSR) is an uncommon but potentially fatal complication of acute myocardial infarction (AMI). The management of VSR is challenging, and its surgical correction is associated with the highest mortality among all cardiac surgery procedures.

Case summary A 57-year-old man with a history of smoking presented with AMI with a large apical VSR in addition to a large secundum atrial septal defect (ASD). His left ventricular ejection fraction was 30%, and the right ventricle was moderately dilated with normal systolic function. Cardiac catheterization revealed that the left anterior descending artery was diffusely diseased with total mid occlusion, whereas other coronary arteries had non-obstructive disease. This unique combination resulted in distinctive presentation with paradoxically better outcomes. After stabilization, the patient's interventricular septum was reconstructed, and the ASD was closed with a pericardial patch. The post-operative period was uneventful, and the patient was discharged 1 week after surgery. A follow-up echocardiography revealed no residual shunt.

Discussion Post-myocardial infarction VSR presents differently in patients with pre-existing right ventricular volume overload. In such cases, the absence of significant cardiogenic shock at presentation may result in better surgical outcomes.

Keywords Myocardial infarction • Ventricular septal rupture • Atrial septal defect • Cardiogenic shock • Case report

Learning points

- Post-myocardial ventricular septal rupture (VSR) is rare but potentially fatal complication.
- Surgical correction carries highest mortality among all cardiac surgical procedures.
- Clinical presentation predicts the outcomes.
- Elevated right heart pressures serve as protective mechanism in case of post-myocardial infarction VSR.

Introduction

Ventricular septal rupture (VSR) is an uncommon but potentially fatal complication of acute myocardial infarction (AMI). Historically the incidence of VSR was about 1–2% but recent data suggest that it complicates 0.17–0.31%.[1,2] Medical management of VSR is associated with poor outcomes, and its surgical correction carries mortality of 42.9%, which is the highest among all cardiac surgery pro cedures.[3] Predictors of poor outcomes include old age, female sex,

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haemodynamics at presentation, and the timing of surgery. Perioperative haemodynamic shock and incomplete revascularisation are the strongest predictors of poor survival.[4]

Timeline

Days	Events
Day 1	Acute ST-segment elevation, anterior wall myocardial infarction.
	Initial management with thrombolysis and anticoagulation.
Day 2	Transferred to our hospital for revascularization due to heart failure and persistent chest pain.
	Initial stabilization.
	Transthoracic echocardiography showed ventricular septal rupture (VSR) and secundum atrial septal defect (ASD).
Day 3	Coronary angiogram revealed single vessel coronary artery disease.
	Intra-aortic balloon pump inserted for better haemo dynamic support.
	Surgical reconstruction of interventricular septum and simultaneous closure of ASD.
Day 4–8	Post-operative intensive care unit stay.
Day 9	Discharge.

Case presentation

A 57-year-old man presented to a secondary care hospital following 6 h of severe central chest pain accompanied by sweating and nausea. He was a smoker (37 pack-years) but was not diabetic or hypertensive. He was diagnosed with acute ST-segment elevation anterior wall myocardial infarction and was managed with thrombolysis (alteplase 100 mL over 90 min). However, his chest pain and heart failure symptoms persisted, and he was referred to our centre for further management. On arrival, 28 h after his first medical contact, he was haemodynamically stable with a pulse rate of 92 b.p.m. and blood pressure of 107/64 mmHg. His physical examination revealed bilateral basal crepitations and a pan systolic murmur at the left sternal border, radiating to the whole precordium. His jugular venous pulse was raised; further, his oxygen saturation was 96% while on 2 L/min oxygen via a nasal cannula. Electrocardiography revealed sinus rhythm with persistent ST-segment elevation in leads V1–V4, and chest X-ray revealed bilateral pulmonary congestion. Upon questioning further, the patient described chronic, gradually deteriorating exertional shortness of breath that persisted for ∼2 years.

Transthoracic echocardiography and transoesophageal echocardiography revealed a large (27 mm), serpiginous antero-apical VSR (*Figure 1* and Supplementary material online, *Video S1*) and a large (29 mm) secundum atrial septal defect (ASD) (*Figures 2* and *3*; Supplementary material online, *Videos S2* and *S3*) with left-to-right shunts. Left ventricular systolic function was severely depressed with an ejection fraction of ∼30%. The left ventricular apex, anterior wall and intraventricular septum were akinetic. The right ventricle was moderately dilated with a base diameter of 51 mm; however, systolic function was normal with a tricuspid annular plane systolic excursion of 18 mm. The right atrium was moderately dilated (volume index 38 mL/m^2), whereas the left atrium was normal (27 mL/m^2). There was moderate tricuspid regurgitation with severe pulmonary hypertension, with a pulmonary artery systolic pressure of 72 mmHg. Coronary angiography revealed the left anterior descending artery was diffusely diseased with total mid occlusion. The other arteries showed non-obstructive disease.

The patient was initially stabilized with intravenous diuretics (frusemide infusion 10–15 mg/h), inotropes (dobutamine 5–10 µg/kg/min), anticoagulant [unfractionated heparin infusion targeted to maintain activated partial thromboplastin time (aPTT) at 50–70 s], and an intra-aortic balloon pump. Next morning, 14 h after presentation at our hospital, the patient underwent surgery for the simultaneous repair of VSR and ASD. The ventricular septum was approached through a transinfarct incision parallel to the left anterior descending artery. Ventricular septal rupture was repaired with a pericardial patch using an infarct exclusion technique (*Figures 4* and *5*). The ventriculotomy was closed with 3-0 proline over-and-over sutures buttressed with Teflon felt. Atrial septal defect was approached through the right atrium and closed with a pericardial patch (*Figure 6*). The left anterior descending artery could not be grafted towing to its small size and the distal diffuse disease.

Post-operatively, the patient remained stable and was extubated the next day. Post-operative echocardiography revealed no residual intracardiac shunt. However, there was moderate left ventricular systolic dysfunction, and right ventricular function was moderately impaired. The patient was discharged 1 week after surgery.

At follow-up conducted at 2 weeks, he was haemodynamically stable, albeit he reported mild exertional dyspnoea (New York Heart Association functional classification I/II). His medication was adjusted. Two months after discharge, he was asymptomatic and could perform routine daily activities. Clinical follow-up and echocardiography have been scheduled for 6 months post-operatively.

Discussion

Ventricular septal rupture is a rare but devastating complication of AMI. With the use of modern reperfusion modalities, such as thrombolysis and primary percutaneous interventions, only 0.17–0.31% of patients with AMI experience VSR.[2] Ventricular septal rupture usually occurs within the first week post-AMI (mean time interval 3–5 days). The factors most associated with VSR complicating AMI include anterior infarction, female sex, advanced age, and no smoking history.[5] In addition, cardiogenic shock at the time of surgery and incomplete revascularisation were found to be independent, strong predictors of poor 30-day, and long-term survival.[4]

The clinical presentation of VSR varies widely from an asymptomatic cardiac murmur to advanced cardiogenic shock. Right ventricular systolic dysfunction is a predictor of early death.[6] Angiographic data have shown that patients who develop VSR after AMI are likely to experience total occlusion of the infarcted artery, causing acute severe ischaemia, and myocardial necrosis.[7]

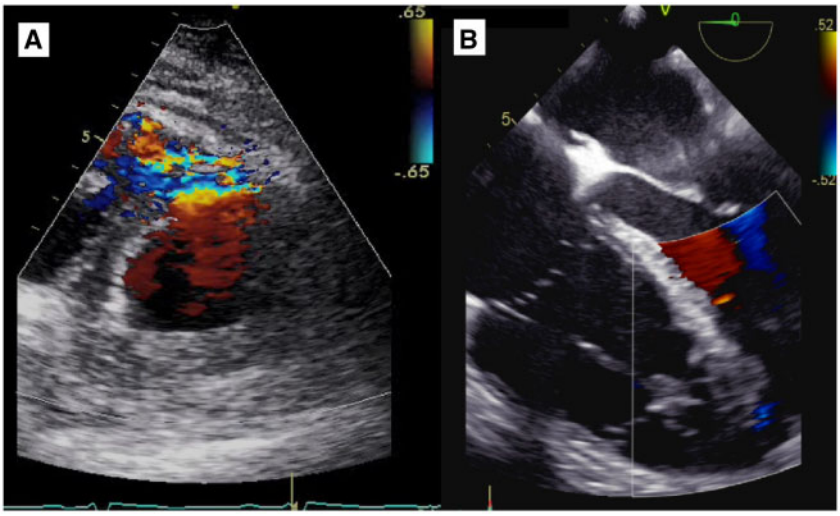

Figure 1 (*A*) Transthoracic echocardiography in the parasternal short-axis view showing the serpiginous antero-apical ventricular septal rupture with a left-to-right shunt. (*B*) Transoesophageal echocardiography showing the apical septal defect.

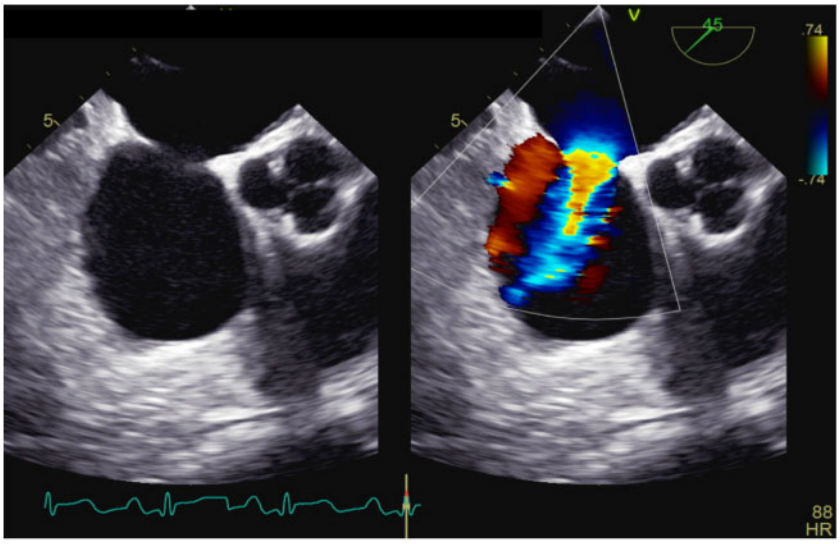

Figure 2 Transoesophageal echocardiography showing the secundum atrial septal defect with left-to-right shunt.

Vasodilators can be used in the initial treatment; however, ultimately, an intra-aortic balloon pump should be used to reduce the mechanical afterload. Other options include a left ventricular assist device as a bridge to surgery; however, evidence supporting this strategy is limited.[8] Haemodynamic stability before definitive treatment is beneficial to the patient; however, deferring surgical intervention to improve the haemodynamics usually results in poor outcomes. Medical therapy alone is associated with 90% mortality.[9] Despite modern surgical techniques, specialized cardiac anaesthesia and myocardial protection during cardiac surgery, surgical outcomes remain poor and mortality is high. Moreover, the timing for surgical intervention is controversial. The current guidelines of the American College of Cardiology and American Heart Association recommend immediate surgical correction regardless of the patient's haemodynamic status; conversely, some studies have found no association between mortality and the timing of surgery for VSR.[10,11] These inconsistent results can be attributed to the highly diverse nature of the clinical presentation, haemodynamics, and surgical techniques used.

Various techniques have been successfully used to repair post-infarction VSR. Cooley *et al.*[12] performed the first open repair of a

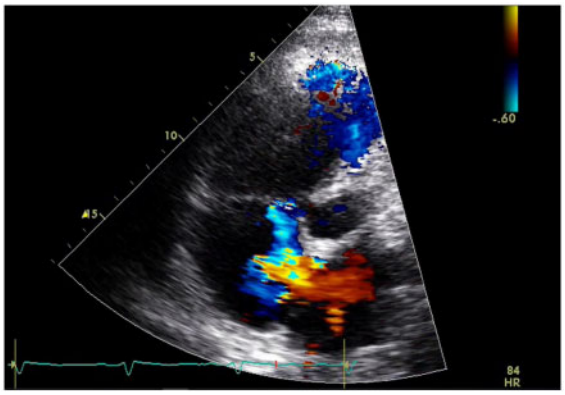

Figure 3 Transthoracic echocardiography showing both the ventricular septal rupture and the atrial septal defect in a single image, with tricuspid regurgitation and dilation of the right ventricle.

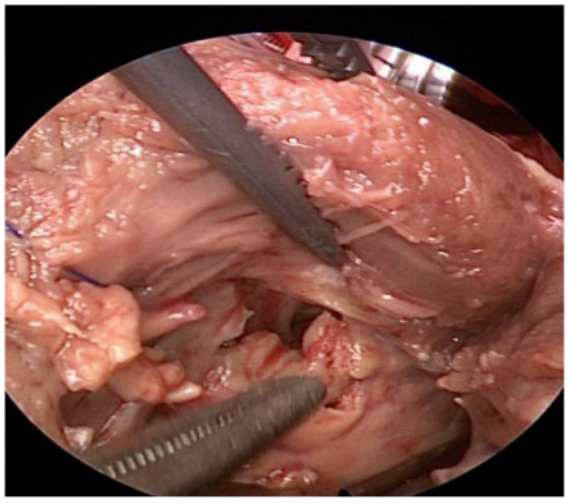

Figure 4 Ventricular septal rupture after the transinfarct incision parallel to the left anterior descending artery.

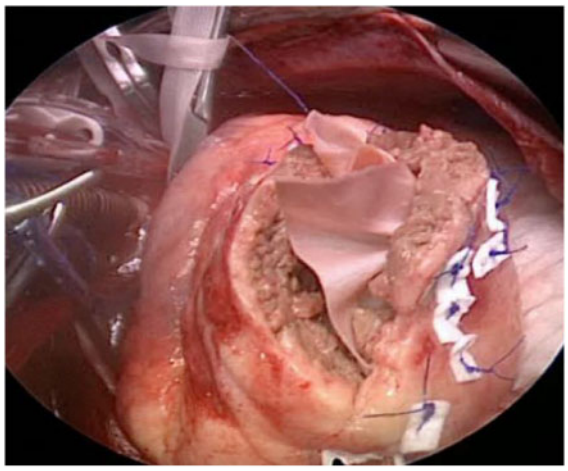

Figure 5 Repair of the ventricular septal rupture using a pericardial patch with the infarct exclusion technique. The left anterior descending artery is visible to the left of the incision.

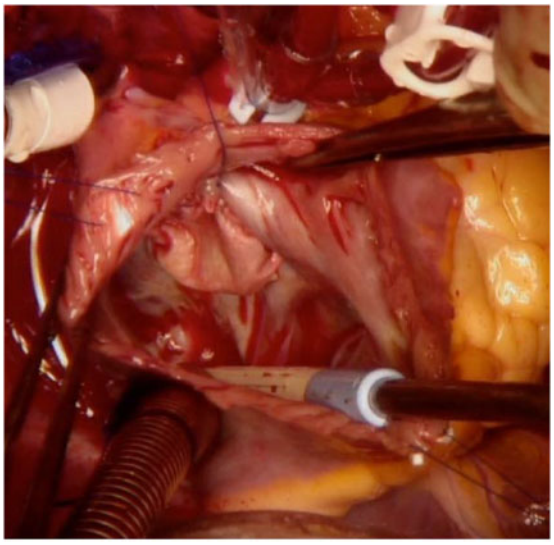

Figure 6 The atrial septal defect was closed with a pericardial patch.

VSR in 1957. This was followed by improved techniques with better outcomes.[13,14] Determining the exact border between infarcted and healthy myocardium is often difficult, making suture placement challenging. The simultaneous repair of a congenital ventricular septal defect and ASD is elective, and both can be surgically or percutaneously corrected.

Our patient exhibited a unique combination of a large secundum ASD and post-AMI VSR. However, the chronically dilated right heart and elevated right ventricular pressure secondary to the left-to-right shunt served as a protective mechanism that limited the shunt fraction at the ventricular level, explaining the relatively stable preoperative haemodynamics. Despite the high morbidity and mortality associated with the individual correction of both shunts, our patient tolerated the surgery very well. In our opinion, the preconditioned right ventricle and elevated right-sided pressures prevented cardiogenic shock by limiting the shunt through the VSR.

Conclusions

In summary, the treatment strategy should be individualized according to the patient's clinical status and haemodynamics. We inferred that the elevated right-sided pressure during ventricular rupture is a protective mechanism. The successful surgical outcome depended on the prompt recognition and simultaneous repair of both lesions.

Lead author biography

Dr Shabir Hussain Shah is a qualified Cardiovascular and Thoracic surgeon since 2005 with more than 13 years' experience (post M.Ch./post-doctorate) in operative Cardiovascular and thoracic surgery with keen interest in the areas of Research and Development. After finishing clinical fellowship in advanced adult cardiac surgery at Mazankowski Alberta Heart Institute (MAHI), University of Alberta, Canada, he joined King Fahad Medical City, Riyadh Saudi Arabia. He is a Consultant cardiovascular surgeon and head of cardiovascular intensive care unit. He is also serving as programme director of cardiac surgery residency programme at the same centre.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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