ABSTRACT

A 56-year-old man presented with acute anterior ST elevation myocardial infarction. Initially he was thrombolysed at a peripheral hospital and a transthoracic echocardiography revealed multiple (2-3 mm) apical muscular ventricular septal defects suggesting ventricular septal rupture (VSR), with the largest measuring 10mm with left to right shunt and max gradient was 74 mmHg. His left ventricular ejection fraction was 45%.

A coronary angiogram revealed tight proximal (95%) and mid segments (80%) stenosis in the left anterior descending artery (LAD) but diffusely diseased distally. Another significant stenosis (80%) was present at the ostium of the right posterior descending artery (r-PDA). He was in Society for Cardiovascular Angiography and Intervention (SCAI) cardiogenic shock Stage B, hence cardiac surgeons advised conservative medical treatment in order to stabilize the infarct area with view of good surgical outcome. Although, there was a dilemma between the surgeon and the cardiologist regarding timing VSR closure, classification of shock stages helped to delay surgery. Eventually, he was taken for surgery at the 18th day of admission with a graft to r-PDA rather to LAD (due to difficult visualization) and repair of VSR with Gortex patch.

In conclusion, in all patients with post MI VSR, SCAI shock stages classification has to be applied in determining the timing of surgery.

Key words: Acute myocardial infarction, echocardiography, ventricular septal rupture

INTRODUCTION

Ventricular septal rupture (VSR) is an uncommon but potentially fatal complication of acute myocardial infarction (AMI). Historically, the incidence of VSR is about 1–2%, but recent data suggest that it complicates 17–0.31%.[1,2]

Medical managements of VSR is associated with poor outcomes, and its surgical correction carries a mortality of 42.9%, which is the highest among all cardiac surgery procedures.[3] Predictors of poor outcomes include old age, female sex, hemodynamics at presentation, and the timing of surgery. Perioperative hemodynamic shock and incomplete re-vascularization are the strongest predictors of poor survival.[4]

The most common dilemma between cardiologists and surgeons is when to do surgery.

CASE PRESENTATION

A 56-year-old man presented to a peripheral hospital with a history of severe acute central chest pain accompanied by sweating. He has been suffering...
from diabetes mellitus for 3 years for which he was taking oral hypoglycemic agent (glibenclamide 5 mg, BID). However, he was not on regular follow-up. He was hypertensive for 3 years and for which he was on regular medication.

He was diagnosed with acute ST-segment elevation extensive anterior wall myocardial infarction and was given Streptokinase due to unavailability of reteplase/alteplase. Although the chest pain improved, his post lysis ECG showed poor resolution. However, next morning the ECG showed transient superior leads ST elevation with fast atrial fibrillation (AF). He was given a bolus intravenous amiodarone (300 mg). While he was on amiodarone infusion, he became hypotensive and dobutamine was started at low dose (5 mcg/kg/min). Eventually, his case was discussed and it was decided he needed a tertiary care hospital. He was shifted with 12 hours for further management.

On arrival, his radial arterial blood pressure was 106/70 mmHg, mean arterial BP was 59 mmHg. He was maintained on low dose dobutamine. The PR was 96 b.p.m, in sinus rhythm. His urine output was 75 ml/hour. On physical examination, he had bilateral fine crepitation and a pansystolic murmur at the left lower sternal area, radiating to the right precordium. Further examination showed the jugular venous pulse was mildly raised but no lower leg edema and warm touch. Oxygen saturation was 98% on 4 L/min oxygen facemask.

Electrocardiography [Figure 1] revealed sinus rhythm with persistent ST-segment elevation in the anterior (V1-V6) with established Q-waves in anterior leads. Chest X-ray [Figure 2] showed bilateral mild pulmonary congestion.

Transthoracic echocardiography revealed multiple (2–3 mm) apical muscular ventricular septal defects (VSD) with the largest VSD measuring 1 cm [Figure 3a], and a left to right shunt [Figure 3b]. Left ventricular (LV) systolic function was mildly impaired with an ejection fraction of 45%. The left ventricular apex was akinetic. There was Grade-I diastolic dysfunction with normal right side dimension and functions. There was no significant valvular regurgitation or stenosis, but there was moderate pericardial effusion measuring 1.6 cm posteriorly and 1.1 cm anteriorly [Figure 3c]. There was no sign of cardiac tamponade. The results of the biochemical Labs were unremarkable except for mild renal impairment (creatinine 105 mmol/L, eGFR-65) and raised Troponin T (364 pg/ml). Lactate level was 2.1mmol/L.

Coronary angiography (CAG) revealed the left anterior descending artery had tight lesions in the proximal (95%) and mid segment (85%) with diffusely diseased distal LAD [Figure 5a]. Right coronary had proximal 30% stenosis; the right posterior descending artery had ostial (90%) lesion [Figure 5b]. There was non-significant lesion at distal left circumflex artery. LV Angiography showed Apical VSR with LV to RV flow [Figure 4a and 4b].

His Society for Cardiovascular Angiography and Intervention (SCAI) cardiogenic shock (CS) stage was determined to be Stage B. At this time cardiac surgeons were consulted, who advised to continue medical treatment with an intention to heal infarct area spontaneously and they would consider either for coronary artery bypass graft (CABG) plus VSD repair versus Percutaneous Coronary Intervention (PCI) plus device closure.

The patient was initially treated medically with intravenous diuretics, inotropes, and with oral aspirin and statin. Gradually, all inotropes were tapered off and he was clinically asymptomatic with stable hemodynamics without support of intra-aortic balloon pump (IABP). Later, low dose of lisinopril and carvedilol was added as vasodilators to reduce afterload.

Eventually, at the eighteenth day of admission, the patient underwent CABG (one venous graft to r-PDA) with simultaneous repair of VSD with Gortex patch.
with infarct exclusion technique. The LAD artery could not be grafted due to poor visualization hence it was decided for PCI later if needed.

Postoperatively, the patient remained stable and was extubated the next day. Follow-up echocardiography revealed no residual intra-cardiac shunt. He was discharged on the 7th post-operative day in stable condition. Due to expatriate issue, a follow-up was arranged over telephone at 2 weeks. After two months, he was asymptomatic, hemodynamically stable and could perform routine daily activities. Later a clinic follow-up was arranged with echocardiography.

**DISCUSSION**

VSR 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.\(^2\) Although, VSR usually occurs within the first of week post-AMI (mean time interval 3–5 days), in our case, it occurred on the 2\(^{nd}\) day of AMI. The factors most associated with VSR complicating AMI include anterior infarction, female sex, advanced age, and no smoking history.\(^5\) This patient had anterior wall MI. The clinical presentation of VSR varies widely from an asymptomatic cardiac murmur to advanced cardiogenic shock. Right ventricular (RV) systolic dysfunction is a predictor of early death.\(^6\) In contrast, this patient had normal RV function which resulted in good outcome.

Three important factors determine patient prognosis in post-MI VSR. Timing of surgery, cardiogenic shock at the time of presentation, and incomplete revascularization were found to be independent, strong predictors of poor 30-day, and long-term survival.\(^4\)

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 hemodynamic status; conversely, some studies have found no association between mortality and the timing of surgery for VSR.\(^7,8\) In our patient the commonest dilemma of timing of surgery was discussed by the heart team. As proposed SCAI we applied the new 5-stage cardiogenic shock (CS) classification scheme proposed (SCAI) for the purpose of risk stratification\(^9\):

(i) A or At Risk: Neither hypotension/tachycardia nor hypoperfusion

(ii) B or Beginning: Hypotension/tachycardia without hypoperfusion

(iii) C or Classic: Hypoperfusion without deterioration.

(iv) D or Deteriorating: Hypoperfusion with deterioration but not refractory shock.

(v) E or Extremis: Hypoperfusion with deterioration and refractory shock.

Figure 3: (a) Transthoracic echocardiography in the apical 4-chamber view showing the anteroapical ventricular septal rupture, (b) Showing the apical septal defect with color flow with a left-to-right shunt. (c) Showing moderate pericardial effusion (PE-1.6 cm)

Figure 4: (a) Coronary angiogram right anterior view revealing left anterior descending artery has proximal tight 95% and 85% tight lesions. (b) Coronary angiogram right anterior view: right posterior descending artery has ostial 90% tight lesion
We concluded that this patient belongs to stage B requiring small dose of a single inotrope. Hence, he was managed medically, in order to stabilize the infarct area with view of good surgical outcome since the edges of the defect would become firmer and fibrotic, and thus repair is more secure. Hemodynamic stability before definitive treatment is beneficial to the patient. However, deferring surgical intervention to improve the hemodynamics usually results in poor outcomes. Medical therapy alone is associated with 90% mortality. Hence, our approach of classifying the CS stages is beneficial in such instances. Stages B and C can be managed with inotropes for few days. If the patient goes into Stage D, then surgical option should be considered. In this patient, initial medical treatment and delayed surgery led a good outcome. Angiographic data have shown that patients who develop VSR after AMI are likely to experience total occlusion of the infarcted artery, causing acute severe ischemia, and myocardial necrosis. But, this patient’s CAG revealed 95% tight lesion in the culprit vessel (LAD). Controversy surrounds the issue of whether to perform CABG in patients undergoing emergency post infarction VSR repair. Some authors have found no benefit to CABG in this setting. In those who underwent CABG, with regard to complete revascularization received partial revascularization with non-culprit vessel grafting, but eventually, it appeared as good short & medium term outcome. The LAD was not grafted due to poor quality. Various techniques have been successfully used to repair post infarction VSR. Cooley et al. performed the first open repair of a VSR in 1957. This was followed by improved techniques with better outcomes.

CONCLUSIONS

Acute myocardial infarction (AMI) that presents with ventricular septal rupture (VSR) is an uncommon, but potentially fatal complication. Transthoracic echocardiography is a diagnostic tool for early diagnosis of VSD after AMI. The surgical correction of VSR is associated with the highest mortality among all cardiac surgery procedures when it is performed either early or late. Hence, we recommend that in all patients with post MI VSR shock’ classification has to be applied in determining timing of surgery. In our opinion, immediate surgery and revascularization to the culprit vessel are not always necessary, is management strategy should be individualized based on SCAI CS classification.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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