

Survival following Ventricular Free Wall Rupture: A Case Series

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ABSTRACT

Ventricular rupture occurs in 10% of acute myocardial infarctions and is associated with significant mortality. We describe two cases of ventricular rupture post myocardial infarction who survived. We highlight salient points in their diagnosis and management and review the existing literature to determine what constitutes optimal management in these difficult cases. (Critical Care and Resuscitation 2006; 8: 43-45)

Key words: Cardiogenic shock, ventricular rupture, myocardial infarction, echocardiography

Ventricular rupture complicates approximately 10% of acute myocardial infarction (AMI) in the USA, yet accounts for 15% of AMI hospital deaths.¹ Following cardiogenic shock, it is the most common cause for death associated with AMI. We present two cases of acute ventricular rupture which were treated successfully and discuss salient points which contributed to their survival in this often fatal condition.

CASE REPORTS

Patient 1

A 68 year old (diet-controlled) diabetic man, on no medications, presented in cardiogenic shock, following a two day history of chest pain. Electrocardiography (ECG) revealed an inferolateral myocardial infarction (MI), and angiography showed a completely occluded circumflex artery. Successful angioplasty was carried out, however there was little clinical improvement. An intra-aortic balloon pump (IABP) was inserted. He was transferred to the intensive care unit (ICU). Treatment included abciximab, aspirin and clopidogrel. He had a blood pressure of 60/30 mmHg, pulse rate of 120 beats per minute (bpm), serum lactate 9 mmol/L (reference range 0.7 - 2.5) and base excess -16 mmol/L (-3 to +3). He was anuric and extremely agitated. A transthoracic echocardiogram (TTE) showed a 1.6cm pericardial effusion, with diastolic compression of the right ventricle. He received fluid resuscitation, was intubated and

ventilated, and given appropriate factor correction (12 units packed red cells, 10 units fresh frozen plasma, 12 units platelets), whilst an urgent transfer to the operating room (OR) was organised.

Once on cardiopulmonary bypass, a left ventricular rupture was found and repaired with BioGlue™ (CryoLife Inc, Kennesaw, USA) and bovine pericardium. His immediate post operative course was complicated by severe coagulopathy and a high inotropic requirement. He required two further operations to achieve haemostasis.

He slowly improved during a one month stay in the ICU. After three weeks of dialysis, following acute tubular necrosis, his renal function normalised. He subsequently made a full recovery.

Patient 2

A 46 year old overweight Post Office manager, with no past medical history, was transferred from a regional hospital in shock following a late presenting lateral MI. Pericardial tamponade was evident clinically, with the classical triad of distended neck veins, pulsus paradoxus and muffled heart sounds. This was confirmed by TTE (Figure 1). He was anuric.

Following insertion of an IABP he was transferred to the OR, where a left ventricular free wall rupture was repaired with horizontal mattress sutures, with three Teflon™ (DuPont, Delaware, USA) pledgets for reinfo-

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rcement.

Post-operatively he developed multi-organ failure, which had resolved by discharge from ICU on day 11. Sixteen days after the initial surgery, he experienced a pre-syncope episode. The following day he became hypotensive and was found to have a further pericardial effusion with cardiac tamponade. The defect was again repaired with sutures and Teflon pledgets. A number of hours later he required a further operation for haemostasis, and again was found to have cardiac tamponade.

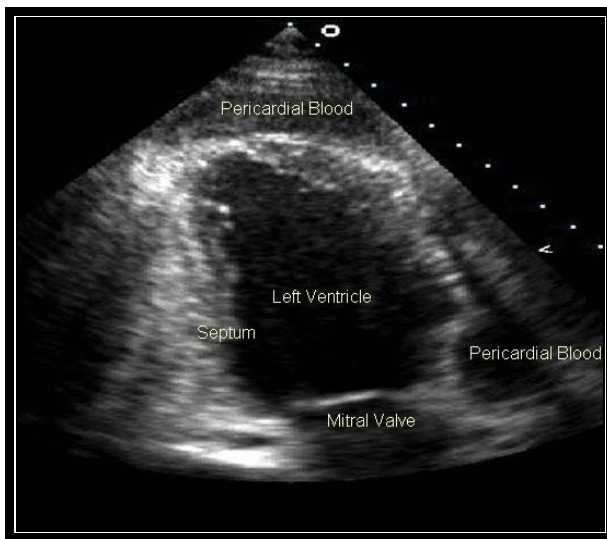


Figure 1. A trans thoracic echocardiogram on presentation to hospital

His recovery was complicated by a homonymous quadrantanopia. Computed tomography of his head revealed bi-occipital and left frontal ischaemic lesions.

He was discharged and was well two months later, apart from a residual mild visual deficit.

DISCUSSION

Myocardial rupture post-AMI is frequently fatal unless the diagnosis and surgical management of the shock occurs rapidly.² Emergency presentation and small sample size combine to make either study or evidence-based guidelines for treatment very difficult.

Rupture may occur hours to weeks post-MI, with approximately 50% occurring within the first 24 hours.³ Unfortunately the diagnosis can be difficult to determine clinically in patients with a recent AMI. This is well illustrated by the first patient who underwent angioplasty, without diagnosis of ventricular rupture. However, the lack of clinical improvement following successful angioplasty of the culprit lesion led to the suspicion of further pathology and subsequent diagnosis of the rupture.

Neither patient had a history of ischaemic heart disease and both AMI's were due to occlusion of the circumflex artery, affecting the lateral left ventricle wall, which is the most common site for myocardial rupture.^{1,4} Usually the circumflex or left anterior descending arteries are most commonly involved in rupture and tamponade.⁵ Other factors such as female sex, age > 65 years, raised C reactive protein, and lower body weight (BMI < 23) have all been proposed to be independent risk factors.^{1,6} Previous MI may reduce the likelihood of myocardial rupture, possibly due to scar formation.⁵

Early echocardiography is essential to establish the diagnosis. Unfortunately, this modality is not always readily available in the majority of hospitals. If the patient is in cardiogenic shock with clinical tamponade, pericardiocentesis may be diagnostic and sometimes life saving if no cardiac surgery facilities are available on site. However, the success of this procedure may be limited in the setting of rupture by continued extravasation of blood into the pericardial space through the myocardial defect.

An IABP was inserted in both cases prior to surgery, with the aim of improving the pre- and post-operative coronary circulation, along with inotropic support. Although no studies have examined IABP use specifically in cardiac rupture, it would appear to be beneficial in improving cardiogenic shock and coronary perfusion peri-operatively. It acts as a temporising measure whilst surgery is being organised, but does not offer an alternative to surgery.

Surgical treatment of myocardial rupture is aimed primarily at relieving the tamponade and repairing the ventricular defect. It has also been proposed that grafting the major vessels empirically may be prudent to avoid repeat infarction,² though this is not standard practise at our hospital.

Due to the small numbers of patients surviving until operation, there is no fully accepted gold standard repair technique. Classically, an infarctectomy and replacement of the resected segment with prosthetic patch on cardio-pulmonary bypass is performed. More recently, patch and glue techniques are becoming more popular, and have the advantage of being able to be performed without cardio-pulmonary bypass. Operative mortality rates have been reported to be around 24-35%.²

CONCLUSIONS

To summarise, a number of suggestions for the investigation and management of patients with myocardial rupture are proffered. Cardiac tamponade secondary to ventricular wall rupture must always be suspected in patients with shock post-MI, and early

echocardiography should be sought as soon as the diagnosis is considered.

Once diagnosed, early transfer to a centre with cardiac surgical facilities should be arranged and the circulation supported as able, with either inotropes and/or IABP (if available in a timely fashion) prior to surgery. If possible, the patient should be anaesthetised and intubated in the OR, immediately prior to sternotomy, the rationale being that anaesthetic induction and positive pressure ventilation causes reduction in preload, afterload, and myocardial contractility which can all precipitate cardiac arrest in the unstable patient.

Both the cases described illustrate that ventricular wall rupture is a survivable condition with expedient transfer for surgical intervention, despite variability in clinical presentation.

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REFERENCES

1. Wehrens XH, Doevendans PA. Cardiac rupture complicating myocardial infarction. *Int J Cardiol.* 2004;9:285-292
2. Malek G, Massas MD, Alexander SG. Surgical Repair of Mechanical Complications of Myocardial Infarction. *World J Surg* 2004;28:847-856
3. Becker AE, Anderson RH. Cardiac pathology. An integrated text and colour atlas. London: Gower Medical Publishing; 1983.
4. Pohjola-Sintonen S, Muller JE, Stone PH et al. Ventricular septal and free wall rupture complicating acute myocardial infarction: experience in the multicenter investigation of limitation of infarct size. *Am Heart J* 1989;117:809-818.
5. Slater J, Brown RJ, Antonelli TA, et al. Cardiogenic shock due to cardiac free-wall rupture or tamponade after acute myocardial infarction: A report from the SHOCK trial registry. *J Am Coll Cardiology* 2000;36:1117-1122
6. Ueda S, Ikeda U, Yamamoto K et al. C-reactive protein as a predictor of cardiac rupture after acute myocardial infarction. *Am Heart J* 1996; 131:857-860.