

Salvagable Free Wall Rupture of the Left Ventricle

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ABSTRACT

Free wall rupture of the left ventricle remains a lethal complication of acute myocardial infarction. Nevertheless, where it presents subacutely with tamponade, salvage with a good outcome is still possible with timely pericardiocentesis preceding definitive repair using a sutureless technique.

Keywords: Free wall rupture, subacute, tamponade, pericardiocentesis, sutureless

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INTRODUCTION

Free wall rupture of the left ventricle is an often fatal complication⁽¹⁾ of acute myocardial infarction (MI). Hence, when suspected clinically in a patient presenting with sudden haemodynamic compromise after a recent myocardial infarction, confirmatory echocardiography followed by rapid pericardiocentesis are essential for patient survival. Relieving the tamponade allows time for arrangements to be made for emergency repair utilising methods that are chosen based on the degree and extent of infarction. A good surgical outcome can be obtained if all three prerequisites are met, namely quick recognition of rupture, timely relief of pericardial tamponade and emergent surgery for definitive repair with minimal collateral damage.

CASE REPORT

A 56-year-old Chinese male was admitted to a local hospital with worsening chest pain that started three days ago. He was a smoker with a long history of hypertension. There was no past history of myocardial infarction (MI). Investigations confirmed an acute antero-septal MI. Intravenous streptokinase was started but he became hypotensive during infusion requiring dopamine for stabilisation. As he continued to have angina with persistent ST segment elevation at V 1-V4, he was transferred immediately to our hospital for percutaneous intervention. On arrival, however, his systolic blood pressure was 60 mmHg, with a heart rate of 60/min. He was restless, agitated



Fig. 1 Free wall rupture of antero-apical portion of the left ventricle.

and extremely breathless. He was intubated and ventilated. An intra-aortic balloon pump (IABP) was inserted and inotropes started. Cardiac catheterisation revealed two significant tandem lesions in the left anterior descending artery (LAD) of 70 and 90% stenosis respectively. The large left circumflex artery was normal. The right coronary artery (RCA) had a mid 70% lesion. The ejection fraction (EF) was 45%. A right heart study revealed a mean right atrial pressure of 20 mmHg with the pulmonary wedge pressure of 28 mmHg. Cardiac tamponade from rupture was suspected in view of the recent infarct, bradycardia and hypotension. An urgent echo confirmed a large circumferential effusion. Immediate pericardiocentesis drained 400 cc of blood with improvement in haemodynamics. The pericardial sac was now empty on echo. No obvious visible wall defect was apparent. Another 250 cc of blood was collected within half an hour. Emergent surgery was arranged.

Operative Technique

Immediate transfer to operating room (OR) was achieved. Patient was cleaned and prepared with rapid induction of anaesthesia. A successful pericardiocentesis allowed an expedient but unhurried sternotomy. Cardiopulmonary bypass was established with aortic and two stage venous cannulation. The vein was harvested for bypass as

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the wall rupture was assessed. This was seen at the antero-apical area with bloody infiltration of surrounding muscle (Fig. 1). Aortic cross clamp was applied and warm cardioplegic induction followed by maintenance cold antegrade and retrograde cardioplegia given. The vein graft was anastomosed to the distal RCA and another vein graft to the LAD just proximal to the more distal obstruction (ruptured plaque) to ensure that a large diagonal branch present was perfused.

Additional cardioplegia was infused through the grafts to protect the right ventricle. The infarcted area was too extensive (involving the apex and lateral free wall of the ventricle) and tissues fragile, precluding any direct suturing method. A sutureless technique was chosen. Gelatin-resorcinol formaldehyde (GRF) glue[®] (Cardial, Saint-Etienne, France) was warmed to 50 deg C and applied only to the infarcted area. Then TachoComb[®] (Nycomed, Austria, GmbH, Linz, Austria) was applied in two layers in a criss-cross fashion to add strength; each layer consisting of two small rectangular strips, applied by finger compression. When it was firmly attached, an equine pericardium (Baxter, Switzerland) was used to cover this repaired area by sewing its circumference to normal epicardium using continuous 4/0 polypropylene suture taking care to avoid major coronary vessels. The size of the patch was kept close to the limits of the infarcted area. This is to ensure that identification of coronaries in future operations is not made more difficult. Then Tisseel[®] (BAXTER AG/IMMUNO AG Vienna) was injected beneath it to prevent blood leakage and to give added strength, reducing the chances of pseudoaneurysm formation from rerupture. This process gave a "pericardium-buttressed sutureless repair" consisting of a sandwich of GRF-TachoComb-Tisseel-Pericardium. A terminal hotshot was given and the aortic clamp released with completion of two top ends of the vein grafts. After a period of adequate reperfusion, the patient was weaned off cardiopulmonary bypass without difficulty utilising a combination of inotropes (adrenalin, milrinone and noradrenalin) and the IABP. The patient made an uneventful recovery. The IABP was removed on the 5th postoperative day to keep LV afterload and thus wall stress, low. He was kept in hospital until the 14th postoperative day as the risk of re-rupture significantly falls after two weeks. A pre-discharge echocardiogram revealed an EF of 45-50%. The basal to mid anterior septum, the anterior segment of the LV wall and the lateral free wall were hypokinetic. The apex and distal septum were akinetic. There was mild central mitral regurgitation. No pseudoaneurysm was apparent at the site of

repair. His ECG showed poor R-wave progression with T wave inversion consistent with a large anterior wall infarction.

DISCUSSION

Overall, cardiac rupture occurs in less than 1% of patients suffering an acute myocardial infarction⁽⁷⁾. However, up to 7% of such hospitalised patients perish from this complication. Further, those receiving thrombolytics contribute a greater proportion of these deaths than those not receiving this form of treatment (12.1% versus 6.1%)⁽¹⁾. Death also occurs earlier and usually within 24 hours of treatment initiation⁽⁷⁾. Most acute ruptures are thus fatal. However in the subacute form, some patients can be salvaged with early recognition and appropriate intervention⁽²⁻⁴⁾. Predictive factors for rupture include age older than 60 years, preexisting hypertension without left ventricular hypertrophy, no previous myocardial infarction and an acute anterior MI⁽⁵⁾. Delayed thrombolysis (Day 4 of MI in our patient) is thought to increase the risk of rupture⁽⁶⁾. With a heightened suspicion, the diagnosis can be confirmed quickly with a bedside echo demonstrating a pericardial effusion and tamponade in a patient who has suffered a recent myocardial infarction. The initial objectives of treatment are effective resuscitation with fluid and inotropic infusion with IABP support. Relieving the tamponade by percutaneous drainage is crucial to this objective and allows time for arrangements to be made for urgent percutaneous and/or surgical intervention. Cardiac catheterisation can be done but should not delay transfer to the operating room if instability persists. Different methods of repair are available. These include conventional infarctectomy with dacron patch repair⁽⁸⁾, felt strip buttress with direct mattress suture⁽⁹⁾, sutured patch cover and sutureless techniques involving surgical glue⁽¹⁰⁾. The choice is controversial. However factors to consider include the following: The extent of infarction, the expected residual cavity with any infarctectomy, the quality of infarcted tissue and the type of rupture. The latter can vary from a direct blow-out to multiple serpiginous tracks that make their way through infarcted tissue⁽¹¹⁾. The amount of "collateral" damage to normal tissue is greatest with infarctectomy and direct suture repair as adjacent normal non-necrotic muscle is incorporated in the suture line. This is best avoided where extensive damage and extremely fragile tissues are present. The initial haemodynamics compromise is contributed heavily by the tamponade as well as the amount of infarction. Smaller infarctions with tamponade would have a good early outcome if initial resuscitative efforts are successful. Also, in the

current era of reperfusion therapy with thrombolytics, sudden myocardial haemorrhage into a weakened area of non-transmural damage can predispose to rupture⁽¹²⁾. In this group of patients, the “salvaged” myocardium may have reasonably well preserved function when reperfusion is early and techniques with minimal damage to adjacent normal muscle are chosen. This was probably the case here as the pre-discharge and a sixth week post discharge echo revealed an EF of 45-50%.

CONCLUSION

Salvage was successful because the diagnosis was suspected and confirmed quickly allowing rapid pericardiocentesis and onward surgical repair. Also, greater preservation of cardiac function was achieved because minimal collateral damage was sustained with the appropriate sutureless technique.

REFERENCES

1. Becker RC, Gore JM, Lambrew C, et al. A composite view of cardiac rupture in the United States national Registry of myocardial infarction. *J Am Coll of Cardiol* 1996; 27:1321-6.
2. Pretre R, Benedikt P, Turina M. Experience with postinfarction left ventricular free wall rupture. *Ann Thorac Surg* 2000; 69:1342-5.
3. Sutherland FWH, Guell FJ, Pathi VL, Naik SK. Postinfarction ventricular free wall rupture: Strategies for diagnosis and treatment. *Ann Thorac Surg* 1996; 61:1281-5.
4. Slater J, Brown RJ, Antonelli TA, et al. Cardiogenic shock due to free wall rupture or tamponade after acute myocardial infarction: A report from the SHOCK trial registry. *J Am Coll Cardiol* 2000; 36:1117-22.
5. Reardon MJ, Carr CL, Diamond A, et al. Ischemic left ventricular free wall rupture: Prediction, diagnosis & treatment. *Ann Thorac Surg* 1997; 64:1509-13.
6. Honan MB, Harrell FE Jr, Reimer KA, Califf RM, et al. Cardiac rupture, mortality and the timing of thrombolytic therapy: a meta-analysis. *J Am Coll Cardiol* 1990 Aug; 16(2):359-67.
7. The Fibrinolytic Therapy Trialists Collaboration. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality & major morbidity results from all randomized trials of more than 1,000 patients. *Lancet* 1994; 343:311-22.
8. Kirklin JW, Barrat-Boyes BG, eds. *Cardiac Surgery*. Churchill Livingstone, 1986; 296.
9. Chemnitz JM, Schmidt T, Wojcik J, et al. Successful surgical management of left ventricular free wall rupture in the course of myocardial infarction. *Eur J Cardiothorac Surg* 1991; 5:51-5.
10. Padro JM, Caralps JM, Montoya JD, et al. Sutureless repair or postinfarction cardiac rupture. *J Cardiac Surgery* 1988; 3:491-3.
11. Perdigao C, Andrade A, Ribeiro C. Cardiac rupture in acute myocardial infarction. Various clinico-anatomical types in 42 recent cases observed over a period of 30 months. *Arch Mal Coeur* 1987; 80:336-40.
12. Gertz SD, Kragel AH, Kalan JM, et al. Comparison of coronary and myocardial morphologic findings in patients with and without thrombolytic therapy during fatal first myocardial infarction. *Am J Cardiol* 1990; 66:904-9.