Delayed Left Ventricular Rupture Following Mitral Valve Replacement

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Abstract

Six hours after mitral valve replacement (MVR), the posterior wall of the myocardium ruptured in the ICU in a 58-year-old man. The bleeding was controlled by opening the chest and applying finger pressure over the defect, and the patient was returned to the operating room while the finger pressure was in place. After restarting the cardiopulmonary bypass (CPB) and cardioplegic arrest, the myocardial rupture site was successfully repaired externally by using a large piece of pericardium (Iranian Heart Journal 2004; 5(4): 57-60).

Key words: heart surgery, mitral valve replacement, myocardial rupture

Rupture of the left ventricular wall is an infrequent but lethal complication after mitral valve replacement. This complication is predominantly common in old patients, especially in females with small left ventricles and those affected by rheumatic mitral valve disease.1,2 It may be due to extensive resection of the valve, particularly after debridement of heavy calcified valves, excessive traction on the annulus, direct damage to the myocardium, undue displacement of the heart during de-airing and insertion of an oversized prosthesis.3-5 Treasure and Miller classified left ventricular rupture into three categories: Type I is a rupture at the posterior atrioventricular groove; type II is a rupture at the base of the papillary muscle; and type III is a rupture between the base of the papillary muscle and the atrioventricular groove.6,7 According to Karson, early rupture is defined as an event occurring in the operating room after discontinuation of cardiopulmonary bypass, and delayed rupture is defined as an event occurring in the early hours after surgery.

Late rupture may occur days to years after MVR and may present as false aneurysm of the left ventricle.8-11 The most important and challenging problem in these cases are control of bleeding and repair of the rupture. The standard method of repair in these cases is removal of the prosthetic valve, intraventricular repair of the rupture and reimplantation of the prosthetic valve.12-16 Sometimes more aggressive methods have been used for the management of this complication such as explantation, repair and reimplantation of the heart.17,18 But in any method of surgical repair, the rate of mortality is high.19-21 such that the rates of mortality have been reported in 65-100% of cases.22 In Bjork’s review in 1977, he noted a 50% mortality for early ruptures and 100% mortality in delayed ruptures.23 It is suggested that less invasive procedures be used for the management of this complication.24,25 In our case, the ventricular rupture occurred 6 hours after surgery, and we used an innovative surgical method for repair of the rupture successfully.
Case report
A 58-year-old man with a history of dyspnea on exertion, fatigue and palpitation was admitted for mitral valve replacement. In clinical examination, diastolic rumble and opening snap were noted. There was no history of hypertension and diabetes mellitus, but he had a 12-year history of cigarette smoking. Roentgenography showed a prominent pulmonary artery with a normal heart size. Two-dimensional echocardiography revealed mitral valve stenosis with a valve area of 0.98 cm². LVEDD and LVESD were 3.8 cm and 2 cm, respectively. The pressure gradient across the mitral valve was 16-30 mmHg (mean pressure, 14 mmHg). Mild tricuspid regurgitation and moderate pulmonary hypertension were also reported in echocardiography. Angiography indicated a dome-shaped mitral valve, pulmonary artery pressure of 60 mmHg with normal coronary vessels.

Surgical procedure: The patient was operated on by standard median sternotomy and cardiopulmonary bypass with moderate hypothermia. Myocardial protection was achieved by cold blood cardioplegia, administered antegrade and supplemented with topical cooling. There was a severely fibrotic mitral valve in a small-sized left ventricle with short and fused papillary muscles. Because of the small size of the annulus, the anterior mitral valve and some parts of the posterior leaflet were removed. As the papillary muscles were short, it is assumed that the papillary muscles were cut deeply. After prosthetic valve implantation and left atrial closure, cardiopulmonary bypass was discontinued uneventfully, and the patient was transported to the ICU in normal hemodynamics. Six hours later in the intensive care unit during a short crisis of hypertension, massive bleeding was encountered, such that in a few minutes, the bleeding was more than 2 liters. The sternotomy was opened immediately in the ICU, and administration of balanced electrolyte solution and blood was started. As the suture lines were intact, evaluation of the posterior left ventricle showed a small rupture at the posterior wall of the left ventricle. The bleeding was controlled by finger pressure and the patient was returned to the operating room. After cannulation of the aorta, IVC and SVC, the CPB was started. Control of bleeding was achieved by finger pressure at all times, and infusion of 6 liters of balanced salt solution, 1500 cc whole blood, and 600 cc FFP were necessary to maintain the blood pressure at 100 to 110 mmHg during this period.

By cardioplegic arrest of the heart and exact evaluation of the rupture, it was repaired externally using a large patch of pericardium. All the stitches were interrupted and the sutures were supported by large pieces of pericardium as felts. Cardiopulmonary bypass was discontinued without any complication. After 3 days’ stay in the ICU and 10 days in the hospital, the patient was discharged with normal clinical and echocardiographic findings. After eight years of follow-up, the patient is presently in good condition.

Discussion
It is possible to control bleeding and repair the left ventricular rupture after MVR by using an external patch. Different methods have been proposed for repair of this complication. It is generally recommended to remove the valve and repair the rupture intra-ventricularly, but the rate of mortality in this method is high (65% to 100%). Spencer combined both external and internal techniques by removing the prosthesis and patching the surfaces of the left ventricle, but only one patient in his series of eight survived. External repair is a simple and less aggressive method for management of this lethal complication;
however, it is suggested that this method may probably not be satisfactory in type I rupture. Even the anatomy of the LCX artery is important for the selection of this method. It seems that the more suitable case is a diminutive LCX artery and in cases in which the OM artery is far away enough from the rupture. On the other hand, any attempt at repair of the rupture must be made under cardioplegic arrest, and the stitches must be put in the normal myocardium.

It was assumed that detachment of the papillary muscle below its base and weakness of the posterior LV wall was the cause of delayed rupture since the rupture was induced after a crisis of hypertension. Finally, it is recommended that prevention be given more emphasis than the management of this serious complication.

References


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