Myocardial Left Ventricular Posterior Wall Rupture After Mitral Valve Replacement (*)

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Between February 1985 and June 1988, three cases of (1.2%) left ventricular rupture occured, following 250 cases of mitral valve replacement (MVR) with bioprosthetic valves. One of the ruptures was attributed to technical maneuvers in the operation and the other two, to cardiac massage during the early postoperative period. Surgical repair was performed with the aid of cardiopulmonary bypass. In two cases external repair and in one case internal approach was performed. Surgical repair done on one patient by internal approach survived, the other two patients died.

Left ventricular posterior wall rupture following MVR is a major lethal complication. Early diagnosis and establishment of cardiopulmonary bypass are life saving measures.

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Rupture of the left ventricle is one of the major lethal complications of MVR. It has been reported with a 0. 5-2% incidence (4). Left ventricular posterior wall ruptures can be classified into three types[7,12,15]: A type I lesion is defined as a defect along the posterior atrioventricular sulcus, and tpye II designation refers to a rupture of the posterior wall of the left ventricle at the base of the papillary muscle. In type III, the rupture is on the posterior wall of the left ventricle between the base of the papillary muscle and the atrioventricular groove (Fig.1).

Materials, Methods, and Results

Between February 1985 and June 1988, three (1.2%) cases of left ventricular posterior wall rupture occured following 250 cases of MVR. In every case a bioprosthetic valve had been inserted.

Case I: A 36 years old, caucasian female patient was admitted to the hospital because of rheumatic mitral ste-

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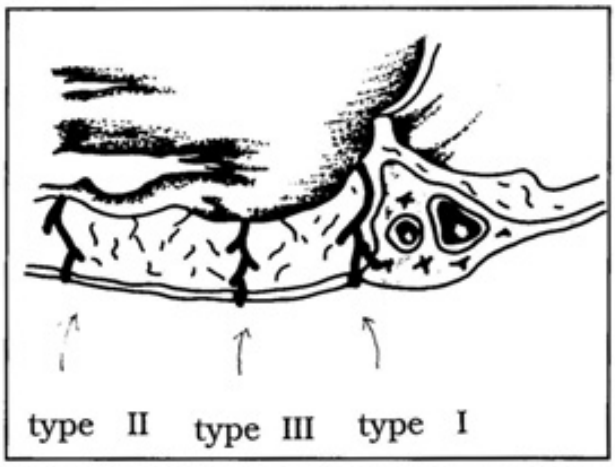


Fig. 1: Three types of left ventricular posterior wall rupture.

nosis and insufficiency. A MVR was planned and the patient was operated on by standard techniques of cardiopulmonary bypass (CPB). At the mitral location, there was a block calcification. The deformated valve was resected and calcified tissue cleaned, a 29 No. of Carpentier-Edwards bioprosthetic valve was inserted with interrupted pledgeted sutures. When the CPB was terminated massive bleeding into the pericardial sac was detected. Exploration revealed a large hematoma at the posterior left ventricular wall in the atrioventricular groove. CPB was established again and the rupture was sutured externally. The bleeding was stopped but because of prolonged cross-clamp time the patient could not be weaned from CPB. On autopsy a rupture near the posterior leaflet, probably due to decalcification was detected.

Case II: A 58 year old, caucasion female patient, had a closed mitral comissurotomy 6 years ago at another center. Mitral restenosis and insufficiency and pulmonary hypertension was present. CPB was established and MVR was performed. A 31 No. of Carpentier-Edwards bioprosthesis was inserted and CPB ended without any event. At the 10th postoperative hour, arrythmia and hypotension were detected. Massive bleeding following external cardiac massage led the patient to CPB again. A 1.5 cm long myocardial rupture was detected right under the circumflex coronary artery. The rupture was repaired with a large teflon patch and interrupted pledgeted sutures. The bleeding was stopped and the patient was weaned from CPB. Low cardiac output developed during the early postoperative period. Despite inotropic support and IABP assistance, the patient died on the first postoperative day.

Case III: A 64 year old male patient was admitted to the hospital due to mitral stenosis and insufficiency, tricuspid regurgitation and pulmonary hypertension. The patient also had a right coronary artery stenosis. The patient was taken into CPB and a 29 No. Biocor bioprosthetic valve was inserted. An aortocoronary saphenous bypass to the right coronary artery was performed concomittantly. At the postoperative 10th hour, first rhytm disturbances and hypotension was seen. Following extracardiac massage, massive bleeding occured. The patient was taken to the operating room and CPB was estabilished.

A 1.5 cm long myocardial rupture was detected under the circumflex coronary artery. The atriotomy was reopened and the bioprosthetic valve removed. The intraventricular rupture was repaired with a teflon patch and interrupted pledgeted sutures. A bioprosthetic valve was inserted by continuous suture technique. The patient was mobilized on the third postoperative day. The patient is under long-term follow up for 11 months and is symptom free.

Discussion

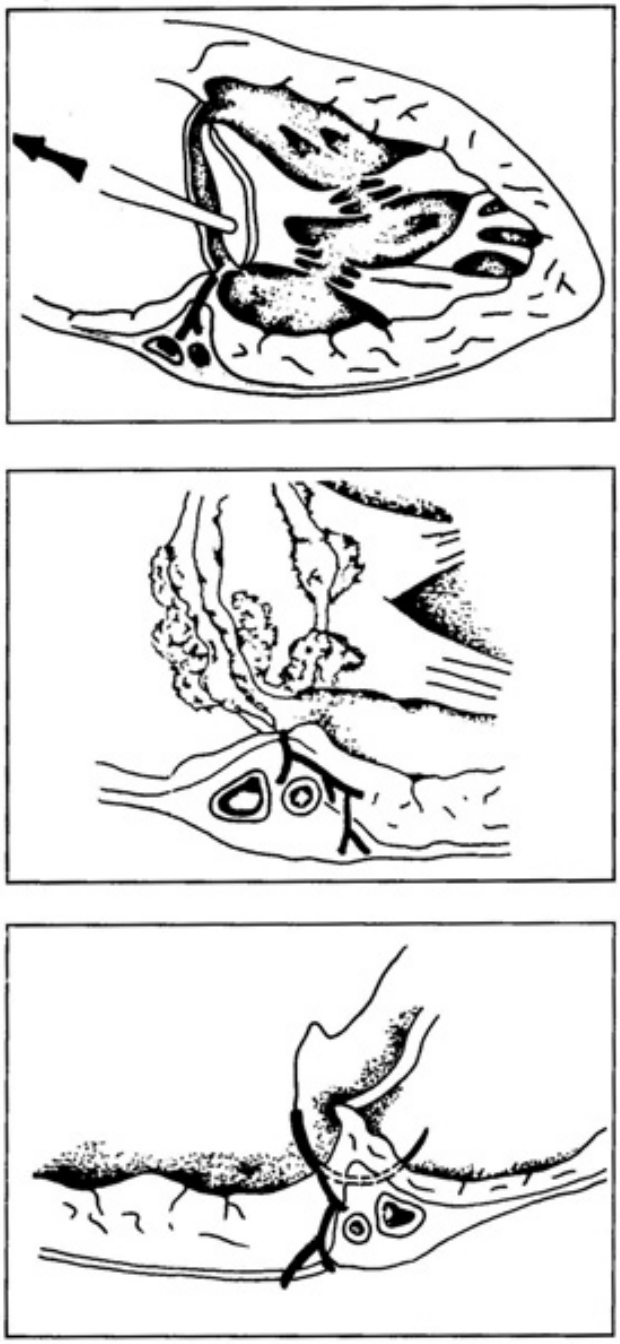
Left ventricular posterior wall ruptures, following MVR are not encountered frequently. The entity was described by Robert and Morrow in 1967[10]. In literature, 60 % mortality is reported for left ventricular posterior wall ruptures[6].

Although advanced age and myocardial fibrosis, relaxation of myocardium because of cardioplegia may be an etiologic factor in left ventricular posterior wall rupture, the main cause being surgical technique.

Retraction of the ventricle when left atrium is fixed by adhesions from a previous operation, can avulse the annulus.

Resection of excessive tissue during removal of diseased valve can result in an injury to the annulus and is probably the most common cause of type I lesion. This hazard is greatest during debridement of heavily calcified valves, especially in the region of the mural leaflet. The same injury can also be produced during removal of a noncalcified valve by forceful traction and inadvertent incision of the annulus, during dissection of an adherent left atrial thrombus, and as a consequence of inadequate exposure through a small left atrium (Figs. 2,3,4)[4,5,9,11,13].

Vigorous traction on the subvalvular structures during removal of the valve can invaginate the left ventricular wall and lead to inadvertent detachment of the papillary muscle below its base. The thinning of the left ventricular endomyocardium can progress to rupture after the heart resumes function. This mechanism is responsible for most but not all type II ruptures (Fig. 5.)[8,9,11]. Mechanical injury to the left ventricle between the base of the papillary muscle and the mitral annulus is cited as a major cause of type III ruptures. Perforation of the left ventricle posterior wall can be attributed to metal vents, scissors used during valve excision, rigid cardiotomy suckers, and curved retractors used to expose chordae tendineae.



Figs.2,3,4: Different couses of type I rupture.

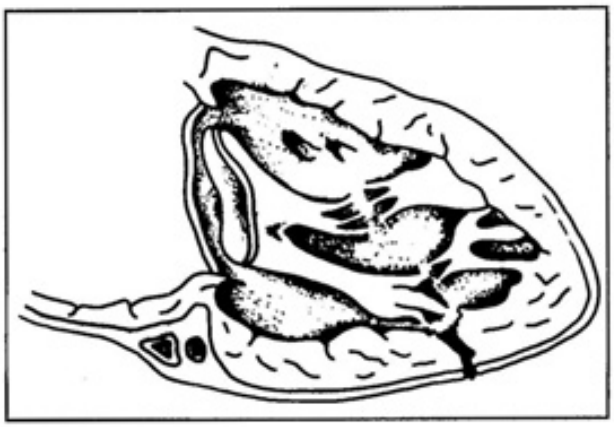
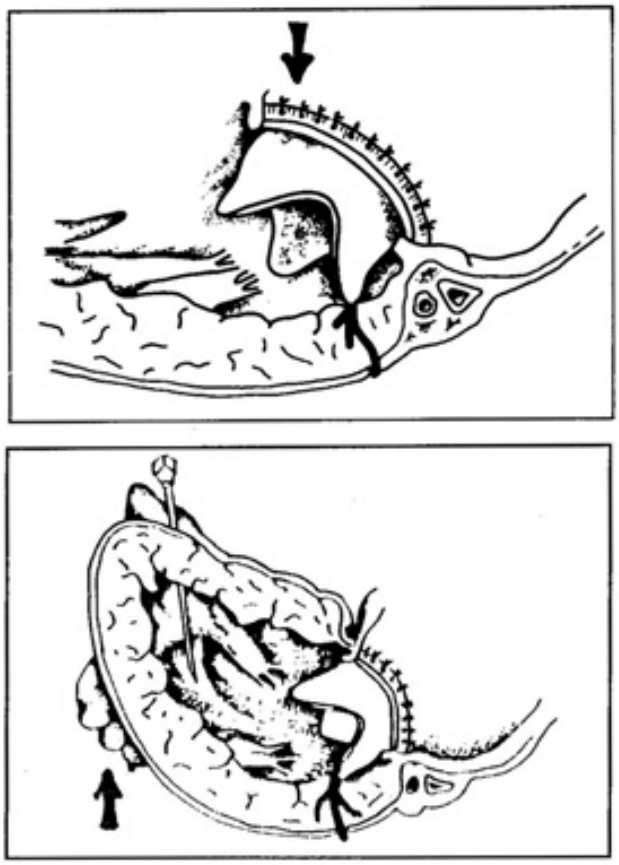


Fig. 5: Type II rupture due to vigorous traction on the subvalvular structures.



Figs. 6,7: Type III rupture due to injury of the bioprosthesis struts.

The type of mitral prosthesis employed plays a role in the production of the primary tear. Bioprostheses have been accused in many series of injuring the myocardium[1,8,11,15]. The damage is attributed to the erosion of the endomyocardial layer by the posterior strut of the bioprosthesis. Forceful manual massage of the heart against the strut of the bioprosthesis can also result in perforation of the ventricle (Figs. 6,7). Two of our patients had a rupture because of this reason.

The basic requirements of successful repair are:

Closure of the full extent of the wound, placing the sutures into the healthy myocardium, sparing the circumflex artery and its major branches. Achievement of these objections is very hard in the beating, pressure loaded heart. All the attempts to repair the wound while the heart is beating have proven to be unsuccessful. The consensus is that the repair with or without using a patch is more successful in type II and III because the defect is limited to the myocardium, and the circumflex coronary artery is less likely to be occluded by the sutures [1,3,9]. Björk and associates compressed type I tears with buttressed mattress sutures between the left ventricule and the atrium. In the atrioventricular groove the sutures were passed through the sewing ring of the prosthesis without injuring the circumflex artery[1].

Internal repair involves reopening of the left atrium and repairing intracardiacly, with or without removing the prosthetic valve. Especially type I ruptures should be repaired internally.

Only pledgeted sutures could be used, or an additional autologous pericardial patch could be put on the rupture. The surgeon must prepare himself for a CABG if circumflex artery occlusion occurs. Circumflex artery damage happens more commonly in external repair but it can happen in both techniques[5].

Left ventricle posterior wall ruptures are one of the major lethal complications of MVR. We have encountered 3 cases and lost two of them. We do believe that if we had tried internal repair instead of epicardial repair the results would have been much better. Our aim is to use the internal repair technique in our next patient if it has a type I rupture.

Conclusion

Left ventricle posterior wall ruptures are lethal complications of MVR. Surgical repair consists of internal and external techniques. Especially in type I ruptures, internal repair techniques are the safest and the adequate mode of therapy. Early diagnosis and immediate establishment of CPB are life saving measures.

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