Case Report

Left ventricular rupture postmitral valve replacement: Surviving a catastrophe

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ABSTRACT

One of the dreaded mechanical complications of mitral valve replacement (MVR) is rupture of the left ventricle (LV). This report describes the early diagnosis and successful repair of rupture of posterior wall of LV in an elderly patient who underwent MVR. We have discussed the risk factors and perioperative issues implicated in such complication. The anesthesiologist as an intra-operative echocardiographer can aid in identifying the patient at risk. Though important surgical steps are necessary to prevent the complication; nonetheless, the anesthesiologist needs to take key measures in the perioperative period.

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INTRODUCTION

Rupture of the left ventricle (LV) following mitral valve replacement (MVR) is infrequent but a highly lethal complication. It was first reported by Roberts and Morrow in 1967.[1] The reported incidence is 0.5–2%.[2-4] Rapid exsanguinating hemorrhage results in high mortality which varies between 50% and 93% with or without the use of cardiopulmonary bypass (CPB).[5] Hence, insights into its prevention and management are important. In this case report, we share and discuss our experience of a repair of posterior LV rupture following MVR for rheumatic severe calcific mitral stenosis (MS).

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CASE REPORT

We operated on a 64-year-old female patient of chronic rheumatic heart disease with severe calcific MS, mild tricuspid regurgitation, and moderate pulmonary arterial hypertension. She had undergone closed mitral valvotomy (CMV) 22 years back for rheumatic MS. She had frail built, normal LV size but with a large left atrium. The preoperative profile of the patient is given in Table 1.

The patient underwent MVR (with 31 mm, St. Jude Biocor™ stented porcine bioprosthetic valve) with standard monitoring and transesophageal echocardiography (TEE). Intra-operative TEE revealed severe calcification of the mitral valve (MV), mitral annular calcification, and severe subvalvular calcification. Surgically, dense pericardial adhesions (due to previous CMV) were also encountered and adequate adhesiolysis was done with diathermy. Posterior mitral leaflet (PML) had to be resected because of severe calcification. Tricuspid valve was inspected and found to be competent. The CPB and aortic cross-clamp (ACC) times were 82 and 56 min respectively. Eight hours after completion of surgery, while contemplating to start weaning off ventilation, an abrupt massive bleeding (1,200 ml over 10 min) was noticed from the mediastinal chest tube. The patient became hemodynamically unstable (BP = 56/30 mmHg) and was resuscitated with one liter of colloid (6%

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hydroxy-ethyl starch) and 500 ml of ringer's lactate. While resuscitation was going on, the sternum was reopened immediately. The blood in the pericardial cavity was suctioned out. Source of bleeding was found to be from the posterior aspect of LV. However, the heart was not mobilized as a large clot was noticed over the back of the LV. Surprisingly, bleeding had come down significantly by the tamponading effect of the clot covering the posterior wall of LV. With fluid resuscitation going on, the patient was shifted carefully from the ICU to the operation room (OR).

In the OR, the patient was emergently put on CPB. For the fear of disturbing the clot and aggravating bleeding, the heart was not handled until CPB was instituted. Once on CPB, the heart was lifted up, and a type III LV rupture [Table 2] was detected on the posterior wall.[6,7] On inspection of the LV rupture site, it was found that the external and the internal disruption sites were lying close to each other, and the tear did not involve the suture line of the MV prosthesis. Repair was done through an external approach using buttresses of strips of Teflon reinforced with prolene sutures. Definite damage to the circumflex coronary artery was excluded after the repair. The patient came off CPB with intravenous infusion adrenaline 0.08 μg/kg/min and dobutamine 7.5 μg/kg/min as inotropic supports. Before going on CPB, we could not insert the TEE probe as the emergency demanded subjecting the patient on CPB urgently. We used the TEE after coming off CPB. TEE revealed hypokinesia of the inferior wall of LV and moderate LV dysfunction. The CPB and ACC times were 63 and 40 min, respectively. There were impairments of liver and renal function during the postoperative course which recovered with medical management. The patient was discharged from the hospital on the fourteenth postoperative day of surgery. She has been on follow-up for the last 2 years. The recent echocardiography shows normally functioning prosthetic valve but mild LV dysfunction with hypokinetic inferior wall [Figure 1].

DISCUSSION

Roberts and Morrow were the first to report LV rupture post-MVR on autopsy in two patients in 1976.[1] The reported incidence has varied between 0.5% and 2% and may be more as some cases may not be reported at all. However, now it is being seen less often than 15–20 years back.[2-4] None the less, high mortality (50–93%) warrants new insights into its prevention and management.[4,5]

Table 1: Preoperative profile of the patient

Variable
Sex: Female
Age: 64 years
Height: 147 cm
Weight: 34 kg
NYHA Class III
ECG: Atrial fibrillation
Chest X-ray: Cardiomegaly and bilateral pleural effusion
Two-dimensional echocardiographic evaluation:
MV area
By PHT: 0.6 cm^2
By planimetry: 0.4 cm^2
Severely calcified MV, annulus, and subvalvular structures
Trivial MR
Mild TR
Moderate PAH (RVSP=49.2+RAP)
LA diameter/BSA: 5 cm/m^2 (normal=1.5–2.3)
LV end-diastolic diameter/BSA: 2.62 cm/m^2 (normal=2.4–3.2)
No LA clot
Normal biventricular function

NYHA: New York Heart Association, PHT: Pressure half-time, MR: Mitral regurgitation, TR: Tricuspid regurgitation, PAH: Pulmonary arterial hypertension, RVSP: Right ventricular systolic pressure, LA: Left atrium, BSA: Body surface area, ECG: Electrocardiogram, LV: Left ventricle, MV: Mitral valve

Table 2: Types of left ventricular rupture post-MVR

Types	Description
I	Located at the A-V groove
	The most common type
	Results from any injury of the MV annulus, such as excessive decalcification, insertion of an oversize prosthesis, deep sutures entering the myocardium and manual cardiac compression
II	Rupture of the LV posterior wall at the base of the papillary muscle
	Primarily due to excessive resection of the posterior papillary muscle, with local hemorrhage and rupture
III	Rupture of the LV posterior wall between the base of the papillary muscle and the A-V groove

MVR: Mitral valve replacement, A-V: Atrioventricular, LV: Left ventricle

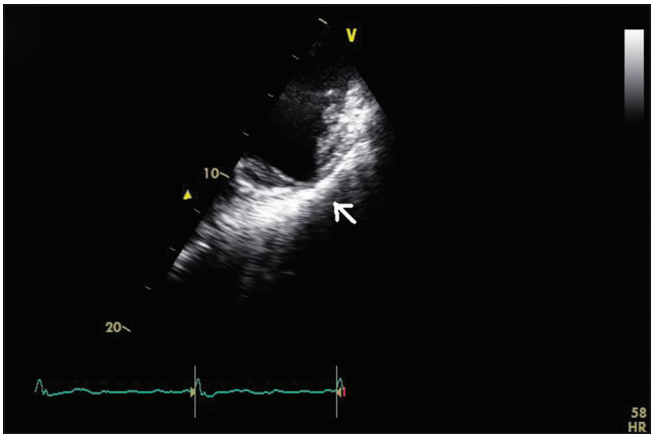

Figure 1: Transthoracic short axis view of posterolateral left ventricle (LV) during the follow-up echocardiography of the patient. The arrow mark showing the site of LV repair on the posterior wall

The LV ruptures after MVR have been classified into 3 types. In 1974, Treasure *et al*. proposed 2 types according to the location of the epicardial tear and later on in 1977 Miller *et al*. supplemented it with the type III rupture [Table 2].[8,9] Sometimes, the ventricular tear progresses and becomes a mixed type, that is, combination of any of the three types. Depending on the time of tear following MVR, LV rupture has been further categorized as immediate, delayed, and chronic. "Immediate/early" tears occur in the operating room before or after weaning from CPB; hence, early resuscitation and better survival. "Delayed" tears present hours to days after leaving the operating theatre. Clinically, these present with massive bleeding into the drainage tubes and hypotension. "Chronic" tears occur days to years after MVR and present as LV pseudoaneurysm.[10,11] The "early" rupture constitutes 2/3rd of the LV ruptures following MVR, and its resultant mortality may reach up to 50% despite immediate intervention.[12]

Patient factors implicated in the pathogenesis are chronic rheumatic heart disease, excessive calcification of the MV apparatus, old age, female sex, myocardial ischemia, infective endocarditis with MV annular abscess, myocardial disease, hemodialysis and small LV size (end-diastolic diameter <50 mm).[2,10,13]

Anesthesiologists can play an important role in identifying the patient at high-risk with the help of the intra-operative TEE. TEE can reveal the valvular and annular calcification, severe subvalvular fusion, small LV cavity and mitral annular abscess, etc. Features of LV rupture on TEE may appear in the form of air in the LV cavity (pathognomonic sign), hematoma lying adhered to the inferior wall and associated with regional wall motion abnormalities.

Intra-operative maneuvers blamed for precipitating the rupture are mechanical causes, such as excessive resection of the mitral leaflets, annulus and/or papillary muscle; redo MVR (adhesions from a previous operation), oversized prosthesis, deep sutures in the myocardium, forceful retraction on the mitral annulus and LV. Perforation of the ventricular wall in the region of the papillary muscles may occur during removal of the MV particularly if this is being done under conditions of ischemic arrest with a flaccid heart.[7,8,14]

The risk factors we found in our patient were advanced age, female sex, previous CMV; and dense calcification of the valvular and subvalvular apparatus.

Intra-operative precautions may, therefore, help to minimize the risk: (i) The PML in whole or part should be preserved in the high-risk patients, (ii) the integrity of the MV annulus should be maintained by avoiding excessive debridement of the calcified annulus; this supports the MV on the LV posterior wall, (iii) judicious and limited papillary muscle excision is useful, (iv) proper sizing of the MV prevents mechanical injury from an oversized prosthesis, (v) excessive traction on the ventricle should be avoided to prevent injury to the root of the papillary muscle (vii) complete adhesiolysis of adhesions from previous surgery help in preventing excess traction on the LV during surgery (vii) systemic hypertension after MVR should be prevented to reduce LV workload, (viii) in the high-risk patients, a pacemaker should be used to increase postoperative heart rate as it shortens diastole and avoids ventricular over-dilation.[10,13,15-17] The potential harmful effects of abrupt intra-operative and postoperative hypertension especially that arising with the use of bolus doses of inotropes may precipitate an incipient rupture.[15,18] Hence, the anesthesiologist role is to make judicious use of inotropes and prevent acute hypertension and vigorous LV contractions in the immediate postbypass period. Katske *et al*. opined that the elongation of the LV, which occurs when the valve is detached from the papillary muscles, might expose an area of weakness.[18] Rupture probably occurs after endocardial damage to a thin myocardium that has lost the internal buttress of the subvalvular apparatus. With the rise in intraventricular pressure at the end of bypass blood dissects into the myocardium, resulting in a large hematoma and eventual rupture. This phenomenon was termed as "untethering" of the LV by Cobbs *et al*.[6] Miller *et al*. noted that leaving part of the PML *in situ* prevents weakening of the LV wall.[9]

It may not be possible to define the actual cause of the LV rupture in every case. At our center, we avoid excessive traction on the ventricle and practice preserving the PML with its attached chordae. However, in this case due to excessive calcification of the MV with the subvalvular fusion, PML with its attached chordae had to be excised. Appropriate sizing of the valve was done while replacing the valve. Probably, the excessive resection of the calcified valve and subvalvular structures triggered the posterior wall LV rupture.

The fundamental requirements for successful repair of LV tear are closure of the full extent of the tear, placement

of sutures into the healthy myocardium avoiding the adjacent inflamed edematous tissue, and sparing the left circumflex coronary artery and its marginal branches. Repair, particularly of a tear in the atrio-ventricular groove, is likely to compromise the circumflex coronary artery circulation.[15] Involvement of the coronary artery requires bypass graft. Wherever needed the valve prosthesis may have to be removed for proper visualization of the site of the tear. Conceivably, the repair of the tear is easier under CPB.[5,11] Due to an injury to LV, the LV dysfunction often demands high inotropic support and intra-aortic balloon pump support. In view of the high-risk surgery and LV dysfunction, these patients may require prolonged postoperative elective ventilation under sedation to stabilize.

Rupture of the LV wall after MVR is a rare event which may accompany MVR. Factors resulting in a fragile myocardium clubbed with operative trauma appear to precipitate an incipient rupture. High degree of suspicion of the catastrophe, immediate resuscitation; and repair of the tear on a decompressed heart under CPB may salvage the patient.

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