Left Ventricular Rupture Following Mitral Valve Replacement*

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During a 10-year period, we have encountered 6 patients (mean age, 61.2 years) with left ventricular rupture following mitral valve replacement, with an overall incidence of 1.8 percent. Four patients had early rupture, one had delayed rupture, and one had late rupture with a false aneurysm formation. Among four patients with early rupture, there were two patients with external repair by using a large ventricular patch and two patients with internal and the external repair by removing the prosthetic valve and patching both the inside and outside of the ventricle. In a patient with delayed rupture, bleeding from an epicardial hematoma was recognized along the atrioventricular groove in the intensive care unit. It was possible to control bleeding by packing the gauze, hemostatic cellulose [Surgicel], and fibrin glue. Late rupture was recognized as a false aneurysm; however, there were no clinical symptoms. All patients survived the surgery, but two patients with early rupture subsequently died. One of these died of renal failure and the other died of multiple organ failure. The sites of rupture in all patients were in accordance with type 1 rupture (Treasure's classification); however, an autopsy review demonstrated the initial laceration in one case was recognized in the membranous septum 5 mm below the mitral ring and extended to the posterior atrioventricular groove. These findings suggest that the injury in the anterior mitral annulus could lead to type 1 rupture, although in the posterior mitral annulus more commonly. Since 1987, we have preserved the posterior leaflet with attached chordae when the mitral valve was fragile and myxomatous. As a result, no instances of left ventricular rupture were encountered.

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Rupture of the left ventricle has been one of the major complications of mitral valve replacement. It is an infrequent but potentially lethal complication. In the review of Karlson et al.,1 the incidence averaged 1.2 percent and approximately 75 percent of the patients died of this complication. Even though many predisposing risk factors have been reported for the etiology of this complication,2,3 it is difficult to define the etiology of rupture and note the precise localization of disruption of the myocardium in most cases.5,4

We report our experience of six patients with rupture of left ventricle, with an emphasis on the surgical repair and consequences of this complication.

METHODS

Study Population

Between January 1980 and December 1990, 328 patients underwent mitral valve replacement at Osaka National Hospital. Among these patients, six patients (1.8 percent) had left ventricular rupture following mitral valve replacement. There were three male and three female patients with a mean age of 61.2 years (range, 48 to 73 years). Of these six patients, one had rheumatic mitral stenosis, three had myxomatous mitral regurgitation, and two had reoperation associated with the dysfunction of a prosthetic heart valve.

Operative Procedures of Mitral Valve Replacement

Operative techniques were standardized and performed with cardiopulmonary bypass under moderate hypothermia. In all procedures, potassium crystalloid cardioplegia was used for intraoperative myocardial protection. Interrupted mattress sutures were used to anchor the valve to the mitral annulus. Both anterior and posterior leaflets were excised with the attached chordae in the four patients who underwent the first replacement of the mitral valve for the first time. Low profile tilting disk valves were implanted in five patients, and a bioprosthetic valve in one.

Onset, Sign, and Repair of Rupture

The onset of rupture can be divided into three distinct time patterns as reported by Karlson et al.1 Early rupture is defined as an event occurring in the operating room anytime after discontinuation of cardiopulmonary bypass; delayed rupture is defined as an event in the recovery room; and late rupture occurs days to years after valve replacement and presents as false aneurysm of the left ventricle. In this regard, four patients had early rupture, one had delayed rupture, and one had late rupture with a false aneurysm formation.

In all four patients with early rupture, the predominant sign of rupture was an epicardial hematoma along the atrioventricular groove after resumption of cardiac ejection prior to discontinuation of cardiopulmonary bypass. It was difficult to make precise localization of a myocardial tear in the spreading hematoma beneath the fatty tissue in this area. Two patients had successful treatment by external repair and the other two patients had internal repair with the additional external repair. External repair was performed under cardiopulmonary bypass, and we sutured a Dacron patch over the area of hematoma on the external ventricular surface, using deep mattress sutures passed beneath the coronary vessels in the atrioventricular groove.

We combined the internal and external repairs in two patients under cardiopulmonary bypass by patching the mitral annulus and the outside of the ventricle. This procedure involved the reopening of the left atrium and correction from within the cardiac chamber with the removal of the prosthesis. Because the mitral ring was involved in the extensive hematoma, we reinforced the mitral annulus with circumferential Dacron felt. The intra-aortic balloon pump was used in all patients to unload the left ventricle and reduce tension on the repair.

In a patient with delayed rupture, the first sign of rupture was the unexpected bleeding from the chest drainage tube after the return to the intensive care unit, the origin of which could not be defined until the chest was reopened and the heart was dislocated.
An epicardial hematoma and bleeding were recognized along the atrioventricular groove under direct vision, but it was possible to control bleeding by packing the gauze, hemostatic cellulose (Surgicel, Johnson & Johnson) and fibrin glue. Despite a stormy course, this patient made a good recovery.

There were no clinical symptoms associated with the left ventricular rupture in a patient with late rupture. Left ventriculogram at the time of hospital discharge noted the oval-shaped false aneurysm in the posterior left ventricular wall (Fig 1).

Results

Operative Results

All patients with early rupture survived a repair, but two patients subsequently died (Table 1). One patient died of renal failure at 14 days, secondary to retrograde aortic dissection. The aortic dissection was induced by an intra-aortic balloon catheter and extended from the left common iliac artery, all the way past the renal artery, and ended just below the left subclavian artery. The other died of multiple organ failure at 27 days, secondary to postoperative low cardiac output syndrome. The patient with delayed rupture and the one with late rupture had no serious complications following the rupture of left ventricle at the time of hospital discharge.

Consequences of Rupture of Left Ventricle

Among four survivors (two early rupture, one delayed rupture, and one late rupture), two patients were in New York Heart Association (NYHA) functional class I, and the other two patients were in NYHA class II. Successful repair of a myocardial tear in the atrioventricular groove was not likely to compromise the circumflex coronary artery and there were no myocardial infarctions in circumflex artery regions in patients with early rupture. The patient with delayed rupture has not been evaluated by the angiographic studies, but he is remaining in NYHA class I and active in his profession in a 10-year follow-up. Two patients (one with early rupture, and one with late rupture) had a false aneurysm of the posterior left ventricular wall. An aneurysm was detected by the left ventriculogram in both patients at the time of hospital discharge. In a follow-up of 7 years and 3 years, respectively, both patients remain in NYHA class II.

Histopathologic Examinations

Postmortem examinations were performed in two patients. In one patient, a slight laceration and a hematoma were observed in the membranous septum from the left ventricular view, and the bleeding around the coronary sinus was identified from the right atrial view. The dissection reached the epicardial surface in the atrioventricular groove and posterior ventricular wall. Histologic examination demonstrated that the extensive hematoma and erythrocyte infiltration were recognized in the membranous septum, the atrioventricular groove, and in the posterior ventricular wall (Fig 2). At the autopsy review of the other patient, the spreading hematoma could be seen just below the posterior mitral annulus and extended to the posterior atrioventricular groove.

Table 1—Clinical Results in Six Patients With Ventricular Rupture Following Mitral Valve Replacement

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Onset</th>
<th>Repair</th>
<th>Site of Hematoma*</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Early</td>
<td>External</td>
<td>AV groove</td>
<td>Survive</td>
</tr>
<tr>
<td>2</td>
<td>Early</td>
<td>External</td>
<td>AV groove</td>
<td>Survive, false aneurysm</td>
</tr>
<tr>
<td>3</td>
<td>Early</td>
<td>Internal</td>
<td>AV groove</td>
<td>Death</td>
</tr>
<tr>
<td>4</td>
<td>Early</td>
<td>Internal</td>
<td>AV groove</td>
<td>Death</td>
</tr>
<tr>
<td>5</td>
<td>Delayed</td>
<td>External†</td>
<td>AV groove</td>
<td>Survive</td>
</tr>
<tr>
<td>6</td>
<td>Late</td>
<td>External</td>
<td>AV groove</td>
<td>Survive, false aneurysm</td>
</tr>
</tbody>
</table>

*AV = atrioventricular.
†Packing gauze and hemostatic cellulose (Surgicel) in intensive care unit.
DISCUSSION

Rupture of the left ventricle following mitral valve replacement, although infrequent, may be a highly lethal complication. Many reports that described the incidence of this complication and mortality have followed. Björk et al reported that 8 of 394 patients undergoing mitral valve replacement (2 percent) had left ventricular rupture and 3 subsequently died (38 percent). Whbain described 18 patients with left ventricular rupture in 1,221 mitral valve replacements (1.47 percent) and 16 deaths (89 percent). Azariades and Lennox described 10 patients with left ventricular rupture in 710 mitral valve replacements (1.4 percent) and 4 deaths (40 percent). In the review of Karlson et al, the incidence averaged 1.2 percent and approximately 75 percent of the patients died of this complication. Most patients can not survive this catastrophic complication, which is manifested by exsanguinating hemorrhage.

According to the general classification, type 1 is defined as a tear along the posterior atrioventricular groove, type 2 is a tear in the midportion of the left ventricle overlying the papillary muscle, and type 3 is a tear on the posterior wall of the left ventricle between the base of the papillary muscle and the atrioventricular groove. In many cases, it is difficult to define the site of rupture by the spreading hematoma beneath the epicardial tissue. In our experience of early and delayed rupture, an epicardial hematoma was noted along the atrioventricular groove on the posterior wall, which was in accordance with type 1 rupture.

However, it is interesting to note that the autopsy review in a patient with type 1 rupture has demonstrated the initial injury in the myocardium in the membranous septum 5 mm below the mitral annulus, an area likely to have been injured at operation, and extended to the posterior atrioventricular groove. This phenomenon suggests that type 1 rupture could be initiated by laceration or injury in the anterior mitral annulus, although in the posterior mitral annulus more commonly. In a patient with late rupture, left ventriculogram revealed a false aneurysm along the atrioventricular groove, which site was accordant to type 1 incomplete rupture.

In general, a large number of intraoperative factors have been considered for the cause of this complication according to the location of the rupture: type 1—(1) retraction of the ventricle when the left atrium was fixed by adhesions from a previous operation, (2) forceful retraction and inadvertent incision of the annulus, (3) insertion of an oversized prosthesis, and (4) deeply placed sutures in the myocardium; type 2—vigorous retraction on the subvalvular structures during removal of the valve; and type 3—mechanical injury to the left ventricle between the base of the papillary muscle and the mitral annulus. However, it is difficult to define the cause of rupture in most cases. The causes of the rupture in the present series may be different and depend on the combination of the various factors.

Female sex, advanced age, mitral stenosis, small left ventricle, and small body size have been men-
tioned as factors predisposing to left ventricular rupture.\textsuperscript{5,7,8} In fact, in our series, three of the six patients with left ventricular rupture were female with small ventricle and small body size. However, these risk factors are common to cardiac surgery generally, and may not be specific to patients with left ventricular rupture following mitral valve replacement.

From a clinical perspective, late rupture has been reported much different from early or delayed rupture.\textsuperscript{1,9,10} Late rupture of the left ventricle appears days to years after valve replacement and presents as a false aneurysm of the left ventricle.\textsuperscript{1,10} Diethrich et al\textsuperscript{8} reported that if the potential mechanism for hematoma formation accompanying type 1 left ventricular rupture was appreciated, a natural sequela might well be a false aneurysm formation. Spellberg and O'Reilly\textsuperscript{10} described a false aneurysm developing in the ventricular wall at the site of previous rupture, which was retained by pericardium or extracardiac tissue. Furthermore, formation of this extracardiac hematoma was considered to prevent more extensive bleeding into the pericardium with resultant tamponade or sudden death. However, the expansion of the false aneurysm compromised the lumen of the circumflex coronary artery and produced a myocardial infarction.\textsuperscript{9} We have seen two patients with a false aneurysm formation. One patient had an aneurysm at the repair site of type 1 rupture, and the other had an aneurysm associated with late rupture. These patients had no symptoms of angina pectoris and no evidence of myocardial infarction in circumflex coronary artery regions. In follow-up of these patients, the importance of left ventriculography in the assessment of a false aneurysm, particularly in patients demonstrating clinical deterioration, should be emphasized.

With regard to the basic procedures for successful repair, two approaches of the repair have been available: the external repair and the internal repair. Chi et al\textsuperscript{11} reported external repair by using a large ventricular patch over the area of hematoma to cover pledged mattress sutures under the circumflex artery. They also noted that no instances of myocardial infarction in the circumflex artery regions were encountered among operative survivors. Björk et al\textsuperscript{8} suggested that external repair be applied to type 2 rupture, and similarly, Karlsen et al\textsuperscript{5} concluded that external repair was more applicable to type 2 and type 3 ruptures where the defect was limited to the left ventricular myocardium, and the circumflex coronary artery was unlikely to be occluded by the sutures.

Internal repair involves the reopening of the left atrium and correction from within the cardiac chamber.\textsuperscript{2,6} According to the report of Treasure et al,\textsuperscript{3} the repair was performed by placing mattress sutures from sewing ring of the mitral valve prosthesis through the ventricle below the tear. Björk et al\textsuperscript{8} also suggested that removal of the prosthetic valve facilitated better exposure and more secure repair. Celemin et al\textsuperscript{12} subscribed to removal of the prosthesis and advocated the repair of the laceration of the myocardium using an autologous internal pericardial patch.

In this series, the basic procedure of repair was based on localization of the hematoma beneath the epicardial tissue. When the hematoma was limited just to the atrioventricular groove, we first attempted to suture a Dacron patch over the area of the hematoma on the external ventricular surface. When the hematoma extended to the posterior left ventricular wall and involved the large area of the ventricle, we chose the internal repair. In our experience with internal repair, the spreading hematoma around the mitral ring made precise localization of rupture difficult even with removal of the prosthetic valve. The entire mitral annulus was reinforced with circumferential Dacron felt, since the mitral annulus was seriously damaged and could not anchor the prosthetic valve. In addition, the external repair was supplemented to the internal repair by patching the outside of the ventricle. In cases of rupture where hematoma was widely spreading, we believe the techniques combining internal and external repair by removing the prosthetic valve and patching both the inside and outside of the ventricle might be recommended for the complete repair, as previously reported by Spencer et al.\textsuperscript{8}

Following the repair, the intra-aortic balloon pump was used in all patients to unload the ventricle and reduce tension on the repair. Whbain\textsuperscript{6} similarly described that the intra-aortic balloon pump could facilitate reduction of afterload and thus would theoretically help to maintain the integrity of the repair.

In order to reduce this fatal complication, Miller et al\textsuperscript{6} suggested that the prosthetic valve should be inserted into the mitral position when the posterior mitral apparatus could be left intact. With preservation of the posterior chordae, the risk of left ventricular rupture following mitral valve replacement might be reduced. Since 1987, we have preserved the posterior leaflet with attached chordae in patients with a fragile and myxomatous mitral valve. As a result, no further instances of left ventricular rupture were encountered in our series.

References

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