Reoperation for Failure of Mitral Valve Repair in Degenerative Disease: A Single-Center Experience

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Background. The purpose of this study was to report our 19-year experience in redo surgery for failure of mitral valve repair (MVRep) in degenerative disease.

Methods. From 1987 to 2006, 43 consecutive patients (32 males) underwent either redo MVRep (n = 21) or redo mitral valve replacement (n = 22) for failure of MVRep. Age ranged from 10 to 78 years (median, 59 years). Forty-one patients (95%) had grade 3+ or greater mitral regurgitation, and 3 patients had chronic systolic anterior motion of the anterior leaflet of the mitral valve. Repair was mainly performed using Carpentier’s techniques.

Results. There was no perioperative death in the MVRep group and 2 deaths in the redo mitral valve replacement group. In univariate analysis, long-term survival was significantly superior in the MVRep group compared with redo mitral valve replacement (p = 0.011).

The long-term durability of mitral valve repair (MVRep) with Carpentier’s techniques for degenerative mitral regurgitation (MR) has been shown to be very satisfactory with a 20-year freedom from reoperation of 97% for posterior prolapse, 86% for anterior prolapse, and 83% for bileaflet prolapse [1]. Repair failures occur, however, particularly in the presence of the following risk factors: anterior leaflet prolapse, use of chordal shortening during operation, absence of remodeling annuloplasty, or residual regurgitation immediately after repair [2–6]. In addition to these procedure-related factors, the disease progression can also cause recurrence of MR.

Few surgical series dealing with reoperation for failure of MVRep have been published [7–10], so that the best surgical strategy is undetermined. In a recent surgical series, re-repair was performed in 44% of patients and was found to be an independent predictor of long-term survival [10].

Material and Methods

Study Population

The study was approved by the local institutional review board, and all patients gave their informed consent. Failure of MVRep was defined as severe valve dysfunction requiring reoperation at least 1 month after the first one. Between May 1987 and December 2006, 43 patients with degenerative mitral valve disease required a reoperation for failure of a previous MVRep. Two of these patients (5%) had their first operation performed in another center.

Cause of the primary valve disease was as follow: Barlow’s disease (n = 22; 51%), fibroelastic deficiency (n = 19; 44%), Marfan syndrome (n = 1), and Ehlers-Danlos syndrome (n = 1). Indication for surgery was severe MR owing to posterior prolapse in 14 (32%) patients, anterior prolapse in 8 (19%) patients, and bileaflet prolapse in 21 (49%) patients. Surgical details regarding the first operation are listed in Table 1. Predischarge transthoracic echocardiography revealed grade 0 to 1+ and grade 2+ mitral regurgitation in 39 (91%) and in 4 (9%) patients, respectively. Systolic anterior motion (SAM) of the mitral valve was present in 4 patients (9%). Risk factors for reoperation were present in 33 patients (77%). These included anterior leaflet prolapse (n = 29; 67%), chordal shortening (n = 20; 46%), incomplete repair,
Table 1. Repair Techniques of the First and Second Operation

<table>
<thead>
<tr>
<th>Repair Technique</th>
<th>MVRep Group (n = 43)</th>
<th>MVR Group (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadrangular resection of PL</td>
<td>35 (81%)</td>
<td>7 (33%)</td>
</tr>
<tr>
<td>Without sliding plasty</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>With sliding plasty</td>
<td>22</td>
<td>3</td>
</tr>
<tr>
<td>Triangular resection of AL</td>
<td>2 (5%)</td>
<td>5 (24%)</td>
</tr>
<tr>
<td>Chordal shortening</td>
<td>20 (47%)</td>
<td>1</td>
</tr>
<tr>
<td>Chordal transfer</td>
<td>7 (16%)</td>
<td>8 (38%)</td>
</tr>
<tr>
<td>Artificial chordae</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Papillary muscle shortening</td>
<td>9 (21%)</td>
<td>1</td>
</tr>
<tr>
<td>Papillary muscle sliding plasty</td>
<td>1 (2%)</td>
<td>1</td>
</tr>
<tr>
<td>Annuloplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>NA</td>
</tr>
<tr>
<td>Prosthetic ring</td>
<td>41 (95%)</td>
<td>NA</td>
</tr>
<tr>
<td>Pericardial posterior annuloplasty</td>
<td>1</td>
<td>NA</td>
</tr>
<tr>
<td>New prosthetic ring</td>
<td>NA</td>
<td>8 (38%)</td>
</tr>
<tr>
<td>Ring reinsertion</td>
<td>NA</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>Vegetectomy</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Pericardial leaflet extension</td>
<td>0</td>
<td>3 (14%)</td>
</tr>
<tr>
<td>Direct suture of leaflet perforation</td>
<td>0</td>
<td>2 (10%)</td>
</tr>
</tbody>
</table>

AL = anterior leaflet; NA = not applicable; PL = posterior leaflet.

(n = 4; 9%), or absence of prosthetic annuloplasty (n = 1; 2%) at the first procedure.

Patients’ age ranged from 10 to 87 years (median, 59 years) at reoperation. There were 32 (75%) male patients. The patients were in New York Heart Association functional class I or II (65%) or class III or IV (35%). The preoperative cardiac rhythm was atrial fibrillation in 8 patients (19%) and sinus rhythm in the remainder. Median preoperative left ventricular ejection fraction was 0.71 (range, 0.45 to 0.80). Preoperative characteristics of each subgroup are given in Table 2.

Median time interval between the first and second operations was 36 months (range, 2 to 136 months). Reoperation was indicated for severe mitral regurgitation (grade 3+ or greater) in 41 patients (95%) or long-lasting SAM of the mitral valve in 3 patients (7%). Mitral valve endocarditis was diagnosed in 1 patient (2%). Hemolysis was present in 4 patients (9%) but did not require repeated blood transfusions.

**Table 3. Causes of Failure of the First Mitral Valve Repair**

<table>
<thead>
<tr>
<th>Lesions</th>
<th>MVRep Group (n = 21)</th>
<th>MVR Group (n = 22)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rupture of shortened or transferred chordae</td>
<td>3 (14%)</td>
<td>10 (45%)</td>
<td>0.045</td>
</tr>
<tr>
<td>Elongation/rupture of chordae from shortened papillary muscles</td>
<td>4 (19%)</td>
<td>1 (5%)</td>
<td>0.19</td>
</tr>
<tr>
<td>Chordal elongation/rupture in progressive disease</td>
<td>6 (29%)</td>
<td>8 (36%)</td>
<td>0.58</td>
</tr>
<tr>
<td>Posterior leaflet retraction</td>
<td>3 (14%)</td>
<td>1 (5%)</td>
<td>0.35</td>
</tr>
<tr>
<td>Leaflet dehiscence</td>
<td>1 (5%)</td>
<td>1 (5%)</td>
<td>0.99</td>
</tr>
<tr>
<td>Leaflet perforation</td>
<td>2 (10%)</td>
<td>0 (%)</td>
<td>0.23</td>
</tr>
<tr>
<td>Incomplete repair</td>
<td>4 (19%)</td>
<td>0 (%)</td>
<td>0.048</td>
</tr>
<tr>
<td>Ring dehiscence</td>
<td>4 (19%)</td>
<td>3 (14%)</td>
<td>0.70</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>1 (5%)</td>
<td>0 (%)</td>
<td>0.49</td>
</tr>
</tbody>
</table>

MVR = mitral valve replacement; MVRep = mitral valve repair.

**Valve Analysis and Surgery**

All patients underwent a repeat median sternotomy. Cardiopulmonary bypass was installed between the ascending aorta and the two venae cavae with mild systemic hypothermia (range, 28° to 30°C). Myocardial protection was achieved by antegrade cold-blood cardioplegia. Mitral valve exposure was obtained either through the interatrial groove approach (n = 41; 95%) or through a transseptal approach (n = 2; 5%).

During surgery, valve inspection revealed mitral valve prolapse in 32 (74%) patients owing to chordal rupture or elongation. Posterior leaflet restricted motion was found in 5 (12%) patients. Prosthetic ring dehiscence, leaflet perforation, leaflet dehiscence, and vegetations were noticed in 7 (16%), 2 (5%), 2 (5%), and 1 (2%) patients, respectively. In the 3 patients who had SAM, the major contributing factor was excess valvular tissue.

The decision to re-repair a failed mitral valve was left to the surgeon, depending on type of lesions, his experience, and the patient’s wish. Each mitral valve repair was assessed intraoperatively by transesophageal echocardiography and at discharge by transthoracic echocardiography.

**Follow-Up**

Long-term follow-up data were obtained through questionnaires and telephone contacts with patients or relatives, physicians, and cardiologists. Information regarding the cause of death was collected from physicians and
cardiologists. Long-term results were assessed on the basis of clinical evaluation and echocardiography. Cardiac rhythm, thromboembolic events, endocarditis, and reoperations were systematically recorded.

Statistical Analysis
Data were expressed as median (range) for continuous variables and as percentage for categorical variables. A 95% confidence interval (CI) was given for most clinically relevant variables. Comparisons between groups were made using the Mann-Whitney U test for quantitative variables or the Fisher’s exact test for qualitative variables.

Calculation of cumulative survival and freedom from events was performed by the Kaplan–Meier method. Comparisons of survival and freedom from events curves were performed with the log-rank test. Significance was defined as a probability value of less than 0.05.

Results
Mechanisms of Late Failure
Mitral valve dysfunction was caused by either procedure- (n = 24; 56%) or valve-related factors (n = 19; 44%). Procedure-related factors included one or more of the following: 13 ruptures of previously shortened or transferred chordae, 5 elongations or ruptures of chordae from previously shortened papillary muscles, 7 ring dehiscences, 4 leaflet dehiscences or perforations, and 4 incomplete repairs. Valve-related factors consisted of 14 recurrences of valvular prolapse in an area not involved by the previous repair, 4 valvular retractions, and 1 endocarditis.

Time interval before reoperation was 20.5 months (range, 2 to 136 months) for patients with procedure-related factors and 46 months (range, 4 to 92 months) for patients with valve-related factors (p = 0.065).

Feasibility of Redo Mitral Valve Repair
During the 19-year study, 43 consecutive patients with degenerative mitral valve disease were admitted to our department for failure of MVRep. Among them, 21 (48%) patients underwent a second MVRep and the others had an MVR with partial preservation of the subvalvular apparatus using either mechanical prosthesis (n = 8), bioprosthesis (n = 13), or mitral homograft (n = 1). When performed, valve reconstruction was mainly performed according to Carpentier’s techniques [11]. Previous annuloplasty rings were left in place in 13 patients and replaced in 7 others. New rings consisted of Carpentier-Edwards Classical (3 cases) or Physio (n = 5; 1 patient had no ring implantation at first operation). Re-repair techniques are reported in Table 1.

The 4 patients with incomplete repair (3 patients with SAM and 1 patient with no ring annuloplasty) underwent re-repair. In cases of rupture of previously shortened chordae, patients underwent MVR most often. In other circumstances, there was no statistically significant difference between the two surgical groups regarding the type of lesions responsible for the repair failure (Table 3).

Operative Mortality and Morbidity
There was no death in the MVRep group. Two patients died 9 and 19 days after MVR, of refractory septic shock (1 patient) and multiple organ failure after mediastinitis (1 patient). Overall operative mortality was 5% (95% CI, 0% to 12%).

Major postoperative morbidity mainly included low cardiac output syndrome, bleeding, and mediastinitis in 12 (28%), 1 (2%), and 1 (2%) patient, respectively. Median intensive care unit stay and hospital stay were 2 days (range, 1 to 19 days) and 11 days (range, 5 to 28 days), respectively.

Valve repair was assessed by transthoracic echocardiography in all patients before hospital discharge. Residual mitral valve regurgitation was absent in 18 (86%) patients, 1+ in 2 (9%), and 2+ in 1 (5%). There was no perivalvular leak in the MVR group.

Long-Term Outcomes
Follow-up was complete in 41 patients (95%). One patient in each group was lost to follow-up. Median follow-up was 74 months (range, 10 to 189 months) in the MVRep group and 107 months (range, 9 days to 170 months) in the MVR group. There were 1 (5%) late death in the MVRep group and 7 (32%) in the MVR group. The cause of death was as follows: cancer (n = 2), pulmonary edema (n = 2), sudden death (n = 1), prosthetic endocarditis (n = 1), stroke (n = 1), and acute alcoholism (n = 1). Kaplan–Meier survival rates (including operative death) at 7 years were 95% in the MVRep group (95% CI, 85% to 99%) and 69% in the MVR group (95% CI, 48% to 90%; Fig 1).

Three reoperations were performed in 1 patient (5%) in the MVR group for prosthetic valve endocarditis. Severe MR as a result of structural valve deterioration of a bioprosthesis was present in 1 (5%) patient from the MVR group. Owing to a high operative risk, surgery was
Ventricular ejection fraction was 0.62 (range, 0.43 to 0.77).

and 2 (12%) patients, respectively. Mean transmitral grade 2

previously shortened chordae [8–10]. Valve-related fac-
ture or prosthetic ring dehiscence, and rupture of

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two categories. Procedure-related factors with a 35% to

degenerative MR. In the last 5 years, redo surgery accounts for 3.5% of cases of

our series. It is important to clearly identify the mechanism of MR in this context. One of the reasons is that in types I and IIIa, there is no need to dissect all the heart because access to the subvalvular apparatus is not required for repair. This is not the case in types II and IIIb MR. Exposure of the mitral valve was usually achieved through the interatrial groove (95% of cases). Once exposed, segmental analysis of the mitral valve is performed and compared with the intraoperative transesophageal echocardiographic analysis. Examination of the prosthetic ring is also important at this stage. If the size of the ring has been well matched to the height of the anterior leaflet at the primary operation, there is no need to remove it. When necessary (incorrectly sized ring, large dehiscence, poor intraventricular access), ring removal is then performed.

Valve reconstruction was mainly performed according to Carpentier’s techniques [11]. Valvular prolapse was the most frequent mechanism of MR (74%) in the present series. Both posterior and anterior prolapse were usually corrected by quadrangular or triangular leaflet resection or chordal transfer. Although we rarely have recourse to artificial chordae (1 case in our series), this might be a valuable alternative, especially in patients with thin chor-
dae. Leaflet extension using an autologous pericardial patch pretreated with glutaraldehyde is a useful tech-
nique to treat posterior leaflet retraction. The patch, usually ovoid, may be asymmetric if valvular retraction predominates on one segment.

Systolic anterior motion has been a rare cause of repair failure (3 cases; 7%). It was always related to an excess of valvular tissue relative to the mitral annulus area. The surgical cure of SAM relied on several principles that have been previously proposed [14]. First, the height of the posterior leaflet is easily reduced to no more than 1.5 cm by using the sliding plasty with triangular resection of the mural leaflet. Second, the choice of the ring annulo-
plasty is critical. One should make sure that the vertical diameter of the ring is the same as the height of the anterior leaflet. Third, in some instances of significant excess tissue, reduction of the height of the anterior leaflet becomes a good solution [15].

Comment

In the present series, rupture of previously shortened chordae accounted for 35% (15 patients) of MVRep failure. Chordal shortening has been shown to be associated with a 5 to 10 times higher risk of MVRep failure in comparison with chordal transfer or chordal replacement with artificial chordae [6, 13]. Based on our experience and on these reports, we no longer recommend the use of chordal shortening to treat valvular prolapse, in particular in degenerative mitral valve disease.

Mitral valve repair failure in degenerative disease mainly consists of recurrence of MR. Basically, the pre-

operative analysis of the mechanism of MR relies on transthoracic or transesophageal echocardiography. The three functional types can easily be recognized, with type I corresponding to leaflet dehiscence or perforation, type II corresponding to leaflet prolapse, and type III corresponding to leaflet restriction. If present, the mechanism of SAM can also be analyzed.

In the present study, we have shown that redo MVRep could be performed in nearly half of patients with late failure of a previous MVRep for severe degenerative MR. Redo MVRep was associated with a null operative mort-
tality and an improved long-term survival in comparison with MVR in univariate analysis. Mid-term freedom from reoperation rate was identical (95% at 7 years) in both groups. Both posterior and anterior prolapse were usually corrected by quadrangular or triangular leaflet resection or chordal transfer. Although we rarely have recourse to artificial chordae (1 case in our series), this might be a valuable alternative, especially in patients with thin chor-
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Freedom from reoperation after MVRep has been shown to be at least equivalent to that of mechanical MVR and superior to that of bioprosthetic MVR [3, 12]. A recent study from our group has reported that the very long-term durability of MVRep was still excellent with a

20-year freedom from reoperation rate of 92% [1]. In the last 5 years, redo surgery accounts for 3.5% of cases of degenerative MR.

The causes of failed MVRep have been classified into two categories. Procedure-related factors with a 35% to

58% relative frequency make up incomplete initial repair, suture or prosthetic ring dehiscence, and rupture of previously shortened chordae [8–10]. Valve-related fac-
tors include progression of the degenerative process or endocarditis. Procedure-related factors are dependent on the surgeon’s experience and tend to occur earlier in comparison with the valve-related factors as observed in our series.
The feasibility of MVRep after failure of a previous one has been reported to vary between 10% and 44% in degenerative disease [5, 9, 10, 16]. In our experience, the feasibility neared 50%. The amount of pliable mitral valve tissue available and particularly of the anterior leaflet is a critical determinant of repair feasibility (Carpentier’s golden rules). In degenerative disease, repair failure is usually not associated with fibrosis, calcification, or destruction (only 1 case of endocarditis in this series) of the anterior leaflet. Furthermore, from a purely technical aspect, the two main mechanisms of MR recurrence, valve prolapse and leaflet retraction, are easily corrected by either valvular resection, chordal transfer, use of artificial chordae, or pericardial valvular extension. Therefore, our re-repair rate should have been higher than 50% (probably more than 80%). However, re-repair was not performed in many cases because the surgeons or the patients were reluctant to confront the hazards of a second valve repair.

The increased long-term survival after MVRep has been attributed to better preservation of left ventricular function and reduced valve-related complications [17]. From a theoretical point of view, these beneficial effects should also persist after redo MVRep because the subvalvular apparatus (a major determinant of left ventricular systolic function) has been preserved and chronic oral anticoagulation avoided. In the present series, univariate study revealed that survival during follow-up was significantly increased after redo MVRep in comparison with redo MVR (Fig 1). This was also the case in a multivariate analysis as reported by Suri and colleagues [10]. Although our attitude is to favor MVRep, the long-term benefit of mitral valve re-repair over mitral valve replacement in cases of repair failure has to be firmly established by larger prospective studies. Therefore, there is undoubtedly a place for mitral valve replacement in cases of repair failure. Complex mitral valve repair in the elderly and limited experience in mitral valve repair are reasonable indications of mitral valve replacement in this setting.

Durability of re-repair is a critical issue. In the largest experience to date, freedom from reoperation was 83% at 5-year follow-up [10]. In the present study, 7-year freedom from reoperation was 95%, and the reoperation rate was 14% with a median follow-up of 100 months. Furthermore, our echocardiographic data revealed satisfying function of the re-repaired mitral valves (88% of no or grade 1+ residual MR). Our reoperation rate is perfectly recurrence of MR was caused by posterior leaflet retraction in 2 patients of 3. We will now not hesitate to enlarge at reoperation any retracted posterior leaflet with an autologous pericardial patch [18].

The present study has two main limitations that should be taken into account. First, only 43 patients were included in the study. This small sample size might have prevented a more precise characterization of the real impact of redo MVRep on patient outcomes. This limited number of patients despite a 19-year study period reflects mainly the excellent long-term durability of MVRep in degenerative disease. Finally, our results also reflect the current experience of a single center with a high level of expertise in mitral valve reconstruction.

In conclusion, redo MVRep was possible in nearly half the cases of late failure of previous MVRep for severe degenerative MR. Redo MVRep was associated with a null operative mortality and satisfying mid-term results (95% 7-year survival or freedom from reoperation rate).

References


