REPAIR OF CHORDAE TENDINEAE FOR RHEUMATIC MITRAL VALVE DISEASE

A twenty-year experience

Sixty-two patients with rheumatic mitral valve disease (mean age 42.2 ± 10.2 years) underwent repair of chordae tendineae between June 1974 and May 1994. Chordal shortening was done in 38 patients, fenestration in 17, resection of secondary chordae in 3, replacement in 2, and transposition in 2. In 41 patients, mitral commissurotomy was also done. Ring annuloplasty was done in all patients. The mean follow-up was 10.2 years (range 2 months to 20 years). The completeness of follow-up during the closing interval (January to July 1994) was 100%. Hospital mortality occurred in four patients (6.5%) and nine patients died during the late follow-up. The actuarial survival curve at 20 years was $65.8\% \pm 10\%$. Six patients with mitral valve dysfunction (restenosis 4, insufficiency 2) and one with aortic valve dysfunction (structural deterioration of bioprosthesis) underwent reoperation. The actuarial curve of freedom from reoperation at 20 years for mitral valve dysfunction was $73.1\% \pm 10.5\%$. In the 49 surviving patients, a Doppler echographic study during the closing interval showed a mean mitral valve area of $1.9 \pm 0.3 \text{ cm}^2$. In the 43 patients with a repaired native valve, absent or trivial mitral regurgitation was documented in 35 and mild or moderate regurgitation in 8. In conclusion, repair of chordae tendineae in rheumatic mitral valve disease when feasible is a stable and safe procedure with a low prevalence of reoperation. However, the type of reconstructive operation and experience of the surgical team are major considerations in successful repair of the mitral valve. (*J THORAC CARDIOVASC SURG* 1996;111:211-7)

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Although mitral valve reconstruction has been shown to provide better results than valve replacement,¹⁻³ there is a paucity of proper comparisons as to the long-term stability of the results obtained with different reparative operations. Technical difficulties in the repair of the subvalvular apparatus and the limited experience with this type of complex operation have been major impediments to the widespread repair, rather than replacement, of the incompetent mitral valve. On the other hand,

most of the reports tend to describe the results of repair in a mixed population with different causes of the valve disease.^{4,5} This report retrospectively analyzes the results obtained in a 20-year period in patients with rheumatic valvular disease who underwent repair of chordae tendineae.

Patients and methods

Between June 1974 and May 1994, 354 patients with rheumatic mitral valve disease, excluding those with pure mitral stenosis treated by isolated open mitral commissurotomy, papillary muscle split, or both, underwent an operation for mitral valve reconstruction with a ring annuloplasty at our institution. Repair of chordae tendineae in association with mitral valve annuloplasty was done in 62 (17.5%) of 354 patients. The patient selection criteria were based exclusively on intraoperative findings and feasibility of repairing the subvalvular apparatus. Data in these 62 patients constitute the basis for this report.

There were 14 men and 48 women with a mean age of 42.2 ± 10.2 years (range 18 to 73 years). At the time of operation, 19 (30.6%) patients were in New York Heart

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Table I. Mitral valve lesions in 62 patients undergoing repair of chordae tendineae

Anatomic lesion	No. of patients	Percent
Annular dilatation	26	41.9
Commissural fusion	30	48.4
Leaflet calcification	4	6.5
Leaflet retraction	4	6.5
Chordal elongation	38	61.3
Subvalvular apparatus fusion	19	30.6
Chordal rupture	5	8.1

Association functional class II, 34 (54.8%) in class III, and 9 (14.5%) in class IV. Before operation, 14 (22.6%) patients were in sinus rhythm and 48 (77.4%) in atrial fibrillation. Forty-two (67.7%) patients had an isolated mitral valve lesion, 17 (27.4%) an associated tricuspid valve lesion, 1 (1.6%) an associated aortic valve lesion, and 2 (3.2%) triple valve disease.

The mitral valve disease consisted of combined stenosis and insufficiency in 25 (40.3%) patients and mixed lesions with predominant insufficiency in 37 (59.7%). Preoperative evaluation included cardiac catheterization in 30 (48.4%) patients, echocardiography in 15 (24.2%), or both in 17 (27.4%). In 47 patients mean systolic pulmonary artery pressure was 41.4 ± 12.1 mm Hg and mean mitral transvalvular gradient was 8.4 ± 5.3 mm Hg.

Intraoperative testing of mitral valve competence and surgical procedures used for valve repair have already been described,⁶⁻⁹ and recently the use of transesophageal Doppler echocardiographic study has been reported. Mitral valve findings are described in Table I. The mitral annulus was found to be dilated in 41.9% of patients, the mitral leaflets were affected in 61.3% of patients, and the commissures were fused in 48.4% of patients. Rheumatic lesions of chordae tendineae were found in all patients. These involved marginal chordae in 59 (95.2%) patients and medial (secondary) chordae in the remaining 3 (4.8%). Chordal elongation was present in 61.3% of cases, severe fusion in 30.6%, and rupture in 8.1%.

The type of technique used for mitral valve repair is shown in Table II. The Duran flexible ring was used in 95.2% of patients and the rigid Carpentier ring in 4.8%. A total of 66.1% of patients underwent open commissurotomy and 4.8% underwent partial leaflet resection. Chordal shortening was done in 61.3% of patients, fenestration in 27.4%, secondary chordal resection in 4.8%, replacement¹⁰ in 3.2%, and transposition in 3.2%. A total of 170 mitral valve reparative procedures were done in 62 patients, for an average of 2.7 procedures per patient. Operations on other valves included tricuspid valve repair in 19 patients (Duran flexible ring annuloplasty in 19, concomitant tricuspid commissurotomy in 7) and aortic valve operation in 2 (bioprostheses 1, valve repair 1). Myocardial revascularization was done in three patients, atrial septal defect closure in two, left atrial thrombectomy in six, left atrial appendage exclusion in eight, and other cardiac procedures in four.

Surviving patients in sinus rhythm were given antiplatelet therapy for 3 months after the operation ($n = 12$). Those patients with atrial fibrillation received crystalline

Table II. Surgical techniques in 62 patients with rheumatic mitral valve disease undergoing repair of chordae tendineae

Operation	No. of patients	Percent
Annular repair*	62	100
Commissurotomy	41	66.1
Leaflet resection	5	8.1
Chordal shortening	38	61.3
Chordal fenestration	17	27.4
Chordal resection	3	4.8
Chordal replacement	2	3.2
Chordal transposition	2	3.2

*Flexible ring annuloplasty, 59 patients; rigid ring annuloplasty, 3 patients.

warfarin sodium (Coumadin) for 3 months after operation ($n = 35$) or on a long-term basis when the patient was considered to be at especially high risk for late thromboembolic complications because of a giant left atrium or massive thrombus ($n = 11$). However, this thromboembolic prophylaxis was modified according to the patient's requirements during follow-up.

Follow-up of all surviving patients was done at regular intervals of 3, 6, and 12 months after operation and then annually. Follow-up was completed from January to July 1994. The completeness of follow-up during the closing interval was 100%. All patients were seen at our outpatient clinic and had a Doppler echographic study during the closing interval. The mean follow-up was 10.2 years, with a range from 2 months to 20 years.

The values are expressed as means of the average and standard deviation. The X^2 test was used to analyze the significance between variables. Actuarial curves were obtained by the life-table method.

Results

Hospital mortality occurred in four (6.4%) patients. The causes of death were heart failure in two patients and massive hemorrhage in two. Hospital mortality for patients undergoing isolated mitral valve operation was 4.8% (2/42) and for patients having polyvalvular operations 10% (2/20) ($p =$ not significant).

Of the 58 surviving patients, nine died during the late follow-up (15.5%). The causes of death were heart failure in three patients, thromboembolism in one, noncardiac death in one, and unknown in four. The actuarial survival curve at 20 years was $65.8\% \pm 10\%$ (Fig. 1). For the group of 42 patients who underwent isolated mitral valve repair, the actuarial survival curve at 20 years was $75.3\% \pm 13.3\%$, whereas for the 20 patients who had polyvalvular operation it was $52.1\% \pm 18.6\%$ ($p =$ not significant) (Fig. 2).

Nine patients had a thromboembolic episode

Fig. 1. Actuarial probability of survival after mitral valve repair. Number above x-axis represents total number of patients at risk.

Fig. 2. Actuarial probability of survival in patients with isolated mitral valve lesions (*squares*) and in patients with multiple valve lesions (*asterisks*). Number above x-axis represents total number of patients at risk.

during the follow-up: two peripheral and seven central (4 transient, 2 with permanent neurologic impairment, and 1 death). The actuarial survival curve for freedom from thromboembolic events at 20 years was 74.3% ± 7.7% (Fig. 3). There were no statistically significant differences in survival free

from thromboembolic events between patients with isolated mitral valve repair and patients with poly-valvular disease or between patients with or without anticoagulation or those in atrial fibrillation or sinus rhythm.

Seven patients required a valve reoperation. The

Fig. 3. Survival free from thromboembolic events. Number above x-axis represents total number of patients at risk.

Fig. 4. Survival free from reoperation. Number above x-axis represents total number of patients at risk.

reoperation was necessary because of mitral valve dysfunction in six patients (stenosis in 4 after an interval of 194 ± 33.2 months, mitral insufficiency in 2 after an interval of 51 ± 33.7 months) and because of structural deterioration of the aortic bioprosthesis in one. There was no mortality at reoperation. The actuarial curve of freedom from reoperation at 20 years for mitral valve dysfunction was $73.1\% \pm$

10.5% (Fig. 4). Other valve-related complications such as infective endocarditis or ring dehiscence did not occur. The actuarial curve of freedom from valve-related complications at 20 years was $47.1\% \pm 9.4\%$ (Fig. 5). Linear rates of valve-related complications were $4.2\% \pm 1.2\%$ per patient-year. Statistically significant differences in survival free from reoperation and survival free from valve-related

Fig. 5. Survival free from valve-related complications. Number above x-axis represents total number of patients at risk.

complications between patients with isolated mitral operation and those with polyvalvular disease were not found.

Forty-three (87.8%) of the 49 surviving patients at the closing interval of follow-up had the repaired native mitral valve and were in functional class I or II. In these 43 patients, mean left ventricular systolic diameter was 29.5 ± 6.8 mm and mean diastolic diameter 48.4 ± 6.2 mm. Mean left ventricular ejection fraction was $61.7\% \pm 9.8\%$ and mean left atrial diameter was 43.7 ± 10.5 mm. The mean mitral valve area was 1.9 ± 0.3 cm². Absent or trivial mitral regurgitation was documented in 35 (81.4%) patients and mild or moderate regurgitation in 8 (18.6%).

Discussion

Although data from different reports have confirmed the predictability and stability of the results achieved with reconstructive procedures of the mitral valve,^{1, 11, 12} repair of chordae tendineae is done occasionally and only by some surgical teams. In most patients, valve replacement continues to be a widely accepted approach for rheumatic disease because of inadequacy of the valve anatomy to be repaired. Repair of chordae tendineae is conditioned by the limited surgical experience, the variety of operations used, and the lack of terminology to classify both subvalvular abnormalities and types of

valvular procedures, as well as by the absence of long-term results with this kind of complex operation.

On the other hand, conservative surgery of the mitral valve often implies repair of the leaflets, the annulus, and each structure of the subvalvular apparatus. In our study, an average of 2.7 procedures per patient was required to complete valve reconstruction successfully. The presence of abnormalities of the chordae is frequently a major impediment to the repair of incompetent mitral valves by means of mitral valve annuloplasty alone.

Although different attempts to repair chordae tendineae have been made in the United States as early as 1960,^{13, 14} chordal repair was eventually abandoned as a result of its unpredictable efficacy. The principles of mitral valve repair introduced by Carpentier and coworkers^{15, 16} renewed interest in mitral valve reconstruction and encouraged various European and North American groups to use conservative operations regularly.^{1, 17-21} Newer techniques were evolved and others were used with indications different from those originally intended.

Mitral valve repair in rheumatic mitral valve disease is technically more difficult and less stable than repair in ischemic and degenerative lesions because of the progressive nature of the pathologic condition and the type of patient population affected.^{4, 22, 23} This confirms the importance of re-

porting the results in patients with a homogeneous cause of the disease.²⁴⁻²⁶

Although our study, like others,^{24,25} has shown the necessity to repair the subvalvular apparatus in most patients with rheumatic heart disease, the degree and extent of chordal involvement are primary determinants of the likelihood of successful repair. Leaflet retraction is a most common finding in rheumatic mitral valve disease that frequently represents a major impediment for mitral valve repair. In our experience, only four patients with mild leaflet retraction were successfully treated by means of ring annuloplasty to increase leaflet coaptation. Chordal elongation, a less common finding in these patients, is more suitable for repair. Intraoperative two-dimensional and Doppler echocardiography done before cardiac operations provides a reliable assessment of the feasibility of valve repair versus replacement.²⁷ However, partial homograft replacement of the mitral valve is an alternative for patients with extensive rupture or elongation of marginal chordae, commissural involvement, and anterior leaflet disease that cannot be repaired by standard techniques.^{28,29}

Advanced morphologic changes in the subvalvular apparatus, progression of native valve disease, and lack of experience have been considered important risk factors for failed mitral valve repair.^{4,23,30} One of the arguments against this kind of operation is that chordal repair is time consuming and results in a much longer ischemic time and significantly higher hospital mortality than straight replacement. In the present study, however, the hospital mortality rate was lower than that for mitral valve replacement.³¹ Long-term survival at 20 years has been lower in patients with polyvalvular disease than in patients with isolated mitral valve lesions (52.1% $\pm$ 18.6% versus 75.3% $\pm$ 13.3%).

Thromboembolism has been the most common late postoperative complication in this group of patients. However, given that a substantial number of patients had polyvalvular disease with a high rate of preoperative risk factors, such as atrial fibrillation, giant left atrium, previous thromboembolism, and left atrial thrombosis, the prevalence of postoperative thromboembolic events has not been higher than that reported for bioprosthetic valve replacement.³¹

Only seven patients in this series underwent reoperation because of mitral valve dysfunction. When valve insufficiency was the cause of failure, reoperation was done early in comparison with the

timing of reoperation for restenosis (51 $\pm$ 33.7 months versus 194 $\pm$ 33.2 months). These findings may indicate that in most cases residual insufficiency is a result of incorrect indication for repair, technical errors, or incomplete repair causing early valvular incompetence. Restenosis of the repaired mitral valve is usually a result of progression of rheumatic disease. A Doppler echographic study done during the closing interval showed, in 81.4% of patients, a normal functioning mitral valve, a mean valvular area of 1.9 $\pm$ 0.3 cm², and no evidence of valve incompetence.

In conclusion, repair of chordae tendineae in unselected patients with rheumatic mitral valve disease when feasible has been shown to be a stable and safe procedure with acceptable long-term results, in particular with regard to a low prevalence of reoperation. There are, however, insufficient data about the long-term results of this procedure in young populations with rheumatic disease. The type of reconstructive operation and experience of the surgical team are major considerations in the successful repair of the mitral valve.

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