Outcome following mitral valve replacement in patients with mitral stenosis and moderately reduced left ventricular ejection fraction

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Abstract

Background: Some patients with mitral stenosis (MS) have moderately reduced left ventricular (LV) ejection fraction (EF), due to either depressed myocardial contractility or alterations in loading conditions. The effect of moderately reduced LV EF on outcome after mitral valve replacement (MVR) is not known. Methods: We studied 16 consecutive patients with LV EF ≤0.50 and MS without significant mitral regurgitation or other valvular or coronary artery disease (Group I). We selected four controls with LV EF >0.50 for each patient, matched for time of surgery (Group II, n = 64). Mean EF in Groups I and II was 0.45 and 0.66, respectively. We compared short- and long-term outcome between the two groups. Results: There were no perioperative deaths. Group I patients had a higher incidence of in-hospital postoperative heart failure (25% vs. 6%, P = 0.02). Mean follow-up was 9 years in both groups. Mean New York Heart Association class improved from 2.4 to 1.7 in both groups. Group I patients had a higher incidence of heart failure deaths (13% vs. 2%, P = 0.03) and admissions (40% vs. 13%, P = 0.01). There were, however, no differences between Groups I and II in overall mortality (27% vs. 21%), rate of cardiac admissions (69% vs. 53%), or mean Specific Activity Scale Score (2.5 vs. 2.5). Conclusions: Although patients with MS and moderately reduced LV EF are at higher risk for heart failure after MVR, overall mortality is not different from that of patients with normal EF. Moderate depression of LV EF should not be a contraindication to MVR for MS. © 2002 Elsevier Science B.V. All rights reserved.

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1. Introduction

In patients with mitral stenosis (MS), heart failure results predominantly from resistance to left ventricular (LV) filling associated with a diastolic gradient across the mitral valve. In a sizable subset of patients with MS, however, LV systolic dysfunction contributes to the pathophysiology of heart failure. Low LV ejection fraction has been noted in up to half of patients with MS [1–12]. Low LV ejection fraction in the presence of MS has been variously attributed to impaired diastolic filling (i.e. inadequate preload) [6,8,12], impaired myocardial contractility, possibly resulting from rheumatic myocarditis [5,7,13], excessive LV afterload [6,9], distortion, immobility, and rigidity of the posterobasal LV myocardium [1–3], right ventricular enlargement [2], or a combination of these factors. The influence of depressed LV ejection fraction on the long-term prognosis of patients with MS undergoing mitral valve replacement (MVR) is not known. The objective of this study was therefore to analyze the outcome of patients with MS and moderately reduced LV ejection fraction undergoing MVR.

2. Methods

2.1. Patient groups

We reviewed the hospital records of all patients with MS and low (≤0.50) LV ejection fraction who underwent isolated MVR at our institution from May 1978 through June 1992. Sixteen consecutive patients with MS, moderately reduced LV ejection fraction, and no significant mitral regurgitation, aortic or tricuspid valvular disease, or coronary artery disease were identified (Group I). For each patient, we selected four controls with MS and LV ejection fraction >0.50 who underwent isolated MVR, matched for time of surgery (Group II, n = 64). LV ejection fraction was assessed from cineventriculography in 68 subjects and echocardiography in the other 12. Patients with any of the following findings were excluded from the study: (1) mitral regurgitation >1 + [14]; (2) aortic regurgitation >1 +...
[14]; (3) aortic stenosis with mean gradient > 10 mmHg; (4) coronary artery disease with ≥ 50% diameter narrowing of a major epicardial artery.

2.2. Preoperative evaluation

All patients underwent preoperative right and left heart catheterization and coronary arteriography. Cardiac output was measured by either the Fick oxygen or thermodilution technique. The mitral valve area was calculated by the Gorlin formula [15]. Left ventriculography in the right anterior oblique projection was performed in 14 patients in Group I and 54 patients in Group II, and LV ejection fraction was calculated by the area-length method. Data from preoperative echocardiograms were available in eight patients in Group I and 16 patients in Group II. In 12 patients, LV ejection fraction was calculated from echocardiograms using end-diastolic and end-systolic diameters with a correction factor for apical contraction [16].

2.3. Operative procedure

MVR was performed within 6 months of cardiac catheterization. Standard cardiopulmonary bypass and cold blood potassium cardioplegia were used. Chordal preservation was not performed in any patient in our series. Eleven different surgeons performed the surgeries.

2.4. Clinical data

Preoperative and in-hospital postoperative data were obtained from hospital records. Preoperative variables recorded included items regarding clinical history, physical findings (as documented in the examination of the attending cardiologist), laboratory data, electrocardiogram, chest radiograph, echocardiogram, and cardiac catheterization. Operative variables recorded included timing of surgery (elective, urgent, or emergency), prosthesis type, aortic cross-clamp and cardiopulmonary bypass times, and requirements for pressor therapy or intraaortic balloon counterpulsation during separation from bypass. Postoperative variables recorded included the number of days spent in surgical intensive care and regular hospital care units and conditions such as diabetes (6% in Group I vs. 9% in Group II), hypertension (0% vs. 8%), hypercholesterolemia (31% vs. 45%), renal disease (0% vs. 2%), chronic obstructive pulmonary disease (19% vs. 8%), thyroid disease (6% vs. 9%), and cancer (0% vs. 3%) was similar in the two groups. Thirteen percent of patients in Group I and 6% of patients in Group II had a pacemaker before undergoing MVR (P = NS). There was a higher prevalence of a right ventricular heave in Group I (62 vs. 34%, P < 0.05). Other physical exam findings were similar in the two groups. LV ejection fraction was 0.45 ± 0.01 (range 0.35–0.50) in Group I and 0.66 ± 0.01 (range 0.51–0.88) in Group II.

Other preoperative hemodynamic and echocardiographic data are shown in Table 2. Hemodynamic parameters, including mean systemic arterial pressure, cardiac index, pulmonary vascular resistance, and mitral valve gradient and area, were similar for the two groups. Echocardiographic LV end-diastolic diameter was higher in Group I (52 ± 2 vs. 44 ± 1 mm, P < 0.05).

MVR was performed electively in all patients in Group I and in 58 patients in Group II (100% vs. 91%, P = NS), while the other six patients in Group II underwent urgent

Table 1
Preoperative clinical characteristics of patients with reduced EF (Group I) and patients with normal EF (Group II)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (n = 16)</th>
<th>Group II (n = 64)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61 ± 4</td>
<td>60 ± 1</td>
</tr>
<tr>
<td>Gender (% female)</td>
<td>88%</td>
<td>88%</td>
</tr>
<tr>
<td>Duration of symptoms (years)</td>
<td>14 ± 2</td>
<td>15 ± 2</td>
</tr>
<tr>
<td>NYHA class</td>
<td>2.4 ± 0.2</td>
<td>2.4 ± 0.2</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>75%</td>
<td>70%</td>
</tr>
<tr>
<td>Prior commissurotomy</td>
<td>44%</td>
<td>45%</td>
</tr>
<tr>
<td>Prior stroke</td>
<td>19%</td>
<td>17%</td>
</tr>
<tr>
<td>Alcohol</td>
<td>38%</td>
<td>36%</td>
</tr>
<tr>
<td>Tobacco</td>
<td>38%</td>
<td>47%</td>
</tr>
</tbody>
</table>

* EF, ejection fraction; NYHA, New York Heart Association.
surgery. Histopathological examination of the stenotic mitral valve specimen revealed similar prevalence of calcification (81% vs. 83%), commissural fusion (81% vs. 77%), and chordal tethering (56% vs. 53%) in Group I and Group II. In Group I, porcine heterografts (one Carpentier–Edwards and three Hancock) were placed in four and mechanical prostheses (five Björk–Shiley, one Starr–Edwards, and six St. Jude Medical) in 12, while in Group II porcine heterografts (14 Carpentier–Edwards and four Hancock) were placed in 18 patients and mechanical prostheses (11 Björk–Shiley, ten Starr–Edwards, 20 St. Jude Medical, and five Medronic–Hall) in 46. Aortic cross-clamp (42 ± 3 vs. 51 ± 3 min) and bypass (79 ± 6 vs. 90 ± 5 min) times were similar in the two groups.

3.2. Clinical outcome

There were no perioperative or other in-hospital deaths. Thirty-one percent of patients in Group I and 16% of patients in Group II required intraaortic balloon counterpulsation after MVR (P = NS for both). One patient in Group I suffered a postoperative stroke, and one patient in Group II had a myocardial infarction. Renal failure occurred in one patient in Group II and liver failure occurred in one patient in each group. Patients in Group I had a higher incidence of heart failure before discharge (25% vs. 6%, P = 0.02). The length of hospitalization was 22 ± 6 days in Group I and 20 ± 1 days in Group II (P = NS).

The mean duration of follow-up was 9 ± 1 years in both groups. Six percent of patients in Group I and 2% of patients in Group II were lost to follow-up. Mean NYHA class improved from 2.4 preoperatively to 1.7 at follow-up in both groups. There was no significant difference in overall mortality (27% in Group I vs. 21% in Group II, Fig. 1), Specific Activity Scale Score, or likelihood of cardiac admission (69% vs. 53%), stroke (20% vs. 29%), or repeat MVR (20% vs. 11%) between Groups I and II. In Group I, the cause of late death was heart failure in two patients, renal failure in one patient, and myocardial infarction in one patient; in Group II, the cause of death was cancer in four patients, stroke in four patients, myocardial infarction in one patient, endocarditis in one patient, heart failure in one patient, immune deficiency syndrome in one patient, and sepsis in one patient. Patients in Group I, however, had higher rates of heart failure mortality (13% vs. 2%, P = 0.03) and heart failure admissions (40% vs. 13%, P = 0.01).

4. Discussion

We describe a consecutive series of 16 patients with MS and moderately reduced LV ejection fraction undergoing MVR, and compare their short- and long-term outcome to that of 64 subjects with normal LV ejection fraction. The analysis shows that, while patients with MS and moderately reduced LV ejection fraction are at higher short- and long-term risk for heart failure, short- and long-term mortality is similar to that of patients with normal preoperative LV ejection fraction.

LV systolic dysfunction in patients with MS was definitively demonstrated in 1970 by Heller and Carleton [1]. In 25 patients with MS varying from mild to severe (valve area 0.4–2.1 cm²), mean LV ejection fraction at cardiac catheterization was 0.56, compared to 0.77 in a normal control group. Low LV ejection fraction in a sizable subset of patients with MS has been observed in a number of other studies [2–10]. Most recently, Snyder et al. [11] reported LV ejection fraction ≤0.50 in 21 of 72 patients undergoing cardiac catheterization for MS, while Choi et al. [12] found LV ejection fraction <0.45 in 18 of 36 patients by a radionuclide technique.

In all clinical conditions, LV systolic dysfunction (low ejection fraction) may be explained by depressed myocardial contractility, abnormal loading conditions (low preload and/or high afterload), or a combination of the two. Low LV
ejection fraction in MS has traditionally been attributed to a ‘myocardial factor’ associated with rheumatic carditis. Hemodynamic studies have not, however, demonstrated impairment of myocardial contractility in patients with MS [6,9]. Goto et al. [8] reported that mean LV ejection fraction increased from 0.61 to 0.67 as a result of percutaneous mitral valvuloplasty in a group of 15 patients with MS, attributing the improvement to higher preload after the procedure. Similarly, Liu et al. [18] reported an increase in LV ejection fraction, associated with both increased end-diastolic volume and improved LV compliance, 3 months after percutaneous mitral valvuloplasty.

An alternative explanation for LV systolic dysfunction has been abnormal function of the myocardium in the region of the mitral valve. The posterior wall or inflow tract of the LV is markedly shortened in many patients with MS [19,20]. Grant [21] concluded that the shortening was due to atrophy of the myocardium adjacent to the posterior rim of the mitral annulus. He hypothesized that thickening of the mitral valve leaflets and fibrosis of the chordae tendineae convert the valve apparatus into a rigid cylinder of dense scar tissue, immobilizing the posterior wall of the left ventricle and causing muscular atrophy. In the study of Heller and Carleton [1], 20 of 25 patients with MS had abnormal regional LV contraction, with distortion and rigidity of the posterobasal myocardium. Posterobasal wall motion abnormalities were also observed by other investigators [3]. Sunamori et al. [5] found fibrosis in the myocardium at the base of papillary muscles removed at the time of MVR.

Right ventricular enlargement has also been blamed for regional abnormalities of LV contraction in patients with MS. Curry et al. [2] reported that mean LV ejection fraction was 0.51 in 12 patients with MS, compared to 0.66 in four patients with right ventricular enlargement due to atrial septal defect or cor pulmonale and 0.79 in eight normal controls. The patients with MS had both anterolateral and posterobasal wall motion abnormalities, whereas those with right ventricular enlargement of other causes had anterolateral but not posterobasal abnormalities.

4.1. Effect of reduced LV ejection fraction on outcome after MVR

Little information has been available regarding clinical outcome following MVR in patients with MS and reduced LV ejection fraction. Sunamori et al. [5] reported that LV ejection fraction was unchanged after MVR in patients with marked myocardial fibrosis, but improved in those with lesser degrees of fibrosis. Snyder et al. [11] found that preoperative LV ejection fraction did not influence perioperative mortality and short-term symptomatic response in patients undergoing surgical commissurotomy or MVR for MS. The data in our study do not demonstrate a deleterious effect of moderate preoperative LV systolic dysfunction in patients with MS on mortality after MVR. This finding is in contrast to the adverse effect of low preoperative ejection fraction on postoperative mortality in patients undergoing surgery for mitral or aortic regurgitation [22,23].

4.2. Limitations of the study

Group I patients had only moderate impairment of LV systolic function. It is possible that the presence of a lower ejection fraction would increase the risk of mortality after MVR. The analysis carries the limitations of a retrospective study, in that patients did not undergo systematic postoperative evaluation of hemodynamics or ventricular function.

4.3. Clinical implications

Patients with MS and moderately reduced LV ejection fraction have a mortality rate after MVR similar to that of patients with MS and normal LV ejection fraction. Postoperative heart failure is more common in patients with reduced LV ejection fraction. Moderately reduced LV ejection fraction should not be a contraindication to mitral valve surgery in patients with severe MS.

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References


