Mitral Valve Replacement or Repair After Previous Coronary Artery Bypass Grafting

Uzi Izhar, MD; Richard C. Daly, MD; Joseph A. Dearani, MD; Thomas A. Orszulak, MD; Hartzell V. Schaff, MD; Charles J. Mullany, MB, MS

Background—Patients who have had previous CABG may subsequently develop significant mitral valve (MV) dysfunction that requires surgical intervention.

Methods and Results—We reviewed 80 consecutive patients who had had previous CABG and who underwent MV surgery between January 1972 and March 1997. Forty-seven (59%) had initial CABG elsewhere, and 5 had had previous CABG twice. The mean interval between the previous CABG and the MV surgery was 6.3 years (range, 4.4 months to 17 years). At least 15 patients had grade 1/4 to 2/4 mitral regurgitation at initial CABG. The study group included 59 men and 21 women, with a mean study group age of 65.5 years. Ninety-four percent had symptoms of congestive heart failure, 60% had angina, 96% were in NYHA class III to IV, and 37% had an ejection fraction (EF), 50%. Origin of the MV disease was ischemic in 33 patients, myxomatous in 19, combined ischemic and myxomatous in 16, rheumatic in 5, infective in 3, and unknown in 4. MV repair was performed in 46 patients (58%) and MV replacement (MVR) in 34. Concomitant repeat CABG was performed in 38 (48%) patients. In-hospital mortality was 7 of 80 (8.8%); no early death occurred among patients with myxomatous disease. EF, 50% was the only significant predictor of early mortality. Overall 1-, 5-, and 10-year survival was 83.8%, 55.6%, and 34.4% respectively. Predictors of late cardiac death were preoperative NYHA class IV (P < 0.0006), urgent or emergency operation (P < 0.0001), use of intra-aortic balloon pump (P = 0.002), and EF < 50% (P = 0.01). Seven patients had an additional reoperation: 4 received CABG, 2 MVR, and 2 MV repair.

Conclusions—Ischemic, myxomatous, rheumatic, or infective MV dysfunction may develop subsequently after CABG. MVR or MV repair after previous CABG is associated with an acceptably low operative risk and good relief of symptoms. Left ventricular function is strongly correlated with both hospital and late mortality. Surgery should be done before LV dysfunction develops. (Circulation. 1999;100[suppl II]:II-84-II-89.)

Key Words: mitral valve ▪ coronary disease ▪ bypass ▪ grafting

Patients who have both mitral valve (MV) dysfunction and atherosclerotic coronary artery disease (CAD) form a heterogeneous group in terms of origin of the valvular disease, extent of coronary atherosclerosis, left ventricular function, and hemodynamic status at operation.1 In recent years, 15% to 30% of patients undergoing MV replacement (MVR) or MV repair for nonischemic MV disease have significant CAD.2,3 In these patients, primary MV dysfunction usually is related to myxomatous changes, rheumatic disease, or infective endocarditis, with CAD as a coexisting factor or an incidental finding on the preoperative coronary arteriogram.4

Moderate-to-severe ischemic mitral regurgitation is present in 3% to 5% of the patients undergoing evaluation of CAD.5,6 The term ischemic mitral regurgitation is often used improperly to describe all cases in which mitral regurgitation and significant CAD coexist. Categorization of the patients with MV dysfunction associated with CAD according to the origin of the MV pathology may have important prognostic and technical considerations.6 Combined primary MVR and CABG continue to have relatively high in-hospital mortality compared with other commonly performed open heart procedures. The reported mortality for the combined MVR and CABG has been between 3% and 25% for nonischemic MV disease and 10% and 38% for ischemic mitral regurgitation.7-9

Studies recently showed that early mortality of patients undergoing aortic valve replacement (AVR) after previous CABG is associated with a higher mortality than is seen after repeat CABG or repeat AVR.10,11 No similar data are available for the MV. Thus, the present study was undertaken to review the outcome of patients who had had previous CABG and underwent MVR or MV repair and to suggest clinical strategies that could improve patient management.

Methods

We retrospectively reviewed the hospital records of 80 consecutive patients who had undergone previous CABG and who underwent

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interval from the last previous CABG to the MV surgery was elsewhere. Five patients had previous CABG twice, and 5 patients were known to have CAD, ischemic mitral regurgitation was determined to be ischemic in 3 patients, myxomatous in 1, rheumatic in 1, and surgically unclassifiable in 1. In recent years, intraoperative transesophageal echocardiography has been used to assess valve competence. Associated surgical report described a valve without visible lesions and annuloplasty was the only valvular procedure performed, the origin was also determined to be ischemic. Cases that involved thickened or calcified leaflets or chordae thickening were considered rheumatic. Myxomatous changes were confirmed histologically. Thirty-three patients had ischemic mitral regurgitation, 19 had myxomatous disease, 16 had both ischemic and myxomatous valvular disease, 5 had RHD, and 3 had infective endocarditis. Origin of the dysfunction could not be determined in 4 patients (Table 2).

The MV was evaluated preoperatively by echocardiography in 70 patients and by cardiac catheterization alone in 10. In all patients, MV regurgitation was the primary valve lesion, and only 1 patient (with RHD) had both mitral stenosis and regurgitation. Seventy-eight patients had moderate-to-severe (grade 3/4) or severe (grade 4/4) mitral regurgitation.

Because 59% of patients had initial surgery elsewhere, we were unable to determine the status of the MV in all patients at initial surgery. However, 15 patients had documented MV disease at the time of initial CABG. All except 1 had mitral regurgitation of mild-to-moderate severity (grade 2/4). The cause was considered ischemic in 3 patients, myxomatous in 1, rheumatic in 1, and unknown in 9. At operation, MV prolapse was found in 31 patients, ruptured chordae in 21, and annular dilatation in 44.

Operative Data
Urgent operation was defined as a procedure performed in a patient whose symptoms necessitated urgent hospital admission and who was too unstable to be discharged before surgery. Emergency operation was defined as an operation performed on a patient who was taken to the operating room from the catheterization laboratory or the intensive care unit because of instability. Forty-one patients (51%) had urgent (n = 36), or emergency (n = 5) operation.

All operations were performed using cardiopulmonary bypass, systemic hypothermia (n = 63) or normothermia (n = 17), cold-crystalloid cardioplegia (n = 29), or cold-blood cardioplegia (n = 48). In 3 patients, we did not use cardioplegia, and the operation was performed on a fibrillating heart (n = 1), with an intermittent crossclamp (n = 1), or on a perfused heart (n = 1). In 6 patients, the operation was performed through a right thoracotomy. No injuries to patent grafts occurred during sternal reentry.

Forty-six patients underwent MV repair, and in 34 patients the valve was replaced (Table 3). The type of prosthetic valve used was according to the surgeon’s preference. Multiple techniques were used to achieve valve competence in the repair group. These included posterior leaflet repair (n = 15), anterior leaflet repair (n = 6), resuspension of chordae (n = 3), and commissural plication (n = 4). Annuloplasty was performed in all cases in this group, including Kay annuloplasty, Carpentier ring, Duran ring, and posterior annuloplasty (Medtronic). In recent years, intraoperative transesophageal echocardiography has been used to assess valve competence. Associated

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**TABLE 1. Clinical Characteristics of 80 Patients Undergoing MV Surgery After Previous CABG**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total, n</th>
<th>Male (%)</th>
<th>Age (mean±SD), y</th>
<th>Preoperative symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>80</td>
<td>59 (74)</td>
<td>65.5±8.0</td>
<td>CHF 75 (94)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Angina 45 (56)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pulmonary edema 30 (37)</td>
</tr>
<tr>
<td>NYHA class</td>
<td></td>
<td></td>
<td></td>
<td>II 3 (4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>III 41 (51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IV 36 (45)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td></td>
<td></td>
<td></td>
<td>Previous CABG ×2 5 (8)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Left main disease 32 (40)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3-Vessel disease 73 (91)</td>
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<td></td>
<td></td>
<td></td>
<td>Previous myocardial infarction 62 (78)</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>CABG elsewhere 47 (59)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>EF (mean±SD), % 51±13</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>LVEDP (mean±SD), mm Hg 25±7</td>
</tr>
<tr>
<td>Values are n (%) unless otherwise indicated.</td>
<td></td>
<td></td>
<td></td>
<td>Status of operation 39 (49)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Urgent 36 (45)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Emergency 5 (6)</td>
</tr>
</tbody>
</table>

**TABLE 2. MV Pathology in Relation to Surgical Procedure and Early Mortality in 80 Patients Undergoing MV Surgery After Previous CABG**

<table>
<thead>
<tr>
<th>Valve Pathology</th>
<th>MV Repair, n</th>
<th>Hospital Mortality, n</th>
<th>MVR, n</th>
<th>Hospital Mortality, n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic</td>
<td>21</td>
<td>2</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Myxomatous</td>
<td>12</td>
<td>0</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Ischemic and myxomatous</td>
<td>10</td>
<td>1</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Rheumatic</td>
<td>5</td>
<td>5</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Infective</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>46</td>
<td>3</td>
<td>34</td>
<td>4</td>
</tr>
</tbody>
</table>

MVR or MV repair between January 1972 and March 1996. All patients who had previous MV procedures were excluded.

Preoperative Data

The study included 59 men (74%) and 31 women, with a mean study group age of 65.5 years (Table 1). Twenty-six patients were >70 years of age. Forty-seven patients (59%) had previous CABG elsewhere. Five patients had previous CABG twice, and 5 patients had left ventricular aneurysmectomy at the time of CABG. The mean interval from the last previous CABG to the MV surgery was 6.3±4.3 years (range, 4.4 months to 17 years).

All patients had preoperative coronary arteriography; 32 (40%) had left main coronary artery stenosis with >50% luminal narrowing and 73 (91%) had three-vessel disease. In 26 patients, left (n = 23) or right (n = 3) internal mammary arteries had been used, and all were patent. Forty-four of 77 grafts to the left anterior descending artery were patent at the time of the mitral surgery. Mean ejection fraction (EF), measured during coronary arteriography, was 51±12% (n = 58); 30 patients had an EF <50%. Seventy-five patients (94%) had symptoms of congestive heart failure before the MV surgery, and 65% had angina. Seventy-seven patients were in NYHA class III or IV.

Valve Pathology

Specific attention was directed at the preoperative echocardiographic study and the operative and pathologic description of the MV to determine the origin of the MV dysfunction. Because all patients were known to have CAD, ischemic mitral regurgitation was restricted to the following criteria: (1) signs or symptoms of myocardial ischemia diagnosed before mitral regurgitation, (2) no history of rheumatic heart disease (RHD), (3) no evidence of degenerative changes or congenital defects of the MV on macroscopic appearance or pathologic examination. In cases in which the
procedures were performed in 48 patients (Table 3), including repeat CABG in 38 patients, with a mean of 1.8 grafts per patient. Most patients were anticoagulated postoperatively for 2 to 3 months, at which time the anticoagulation was stopped if the patient was in sinus rhythm and did not have a prosthetic valve or another medical condition that required anticoagulation.

Follow-Up
Follow-up data were obtained from the patient’s most recent clinical visit, a patient survey, and a telephone interview. All patients who were alive were contacted within the year preceding publication of the present study. Total follow-up was 367.6 patient-years, with a mean of 5.0 years.

Statistical Methods
The association of discrete risk factors with hospital mortality was evaluated by χ² or Fisher’s Exact test when appropriate. The relationships of continuous risk factors such as age to hospital mortality were evaluated with 2-sample t tests or Wilcoxon rank-sum tests. Survival and survival free of other events, such as reoperation, was estimated by the Kaplan-Meier method. Survival curves were compared with log-rank tests.

Results
Mortality (30-Day)
Seven 7 (8.8%) deaths occurred in-hospital or at 30 days after surgery. For valve repair, the mortality was 3 of 46 patients (8.3%) and for replacement, 4 of 34 (11.8%) [P=0.67]. Two patients died in the operating room of myocardial failure, 3 with low cardiac output or ventricular arrhythmia, and 1 each from sepsis and stroke. When univariate analysis was applied, the only factor that significantly affected early mortality was EF <50% [P=0.01]. Factors that were not significantly associated with increased operative risk were age, left main CAD, origin of the valve dysfunction, preoperative NYHA class, surgical procedure (replacement or repair), use of an intra-aortic balloon pump, concomitant repeat CABG, and year of surgery.

Hospital Morbidity
Inotropic support for >48 hours occurred in 25 patients, and an intra-aortic balloon pump was placed in 13. Thirty-seven patients required intubation for 24 hours, and 2 needed tracheostomy. Two patients had new Q-wave myocardial infarction, 5 required reoperation for bleeding, and 2 had deep sternal wound infection (1 died). Stroke occurred in 2 patients (1 died), and 3 had transient ischemic attacks. One patient needed a permanent pacemaker.

Survival
Follow-up was completed in all patients by a mean of 5.0 years (range, 0.2 to 18 years). At the time of publication of the present article, 34 patients were alive (mean of 5.4 years; range, 1 to 14 years) and 26 (77%) are considered to be in NYHA class I or II. Of the 39 late deaths, 30 were cardiac related, 3 were not cardiac related, and 6 were of unknown causes.

The 1-, 5-, and 10-year overall survivals (±SE) were 84±4%, 56±6%, and 34±7%, respectively (Figure 1). For operative survivors, the 1-, 5-, and 10-year survivals (±SE) were 90±3%, 61±6%, and 38±7%, respectively. Predictors of late death included preoperative NYHA class IV (P<0.001, Figure 2), urgent or emergency operation (P<0.001, Figure 3), placement of intra-aortic balloon pump during operation (P=0.002), and EF <50% (P=0.01, Figure 4). Age, origin of the MV dysfunction, the surgical procedure (replacement or repair), and associated repeat CABG did not significantly affect late outcome.

Late Events
Seven patients underwent additional reoperation. Three had repeat CABG, 2 had MVR (recurrent mitral regurgitation and degeneration of porcine valve) and 1 each had CAbG and repeat MV repair and repair of perivalvular leak. All except 1 survived the third operation and are still alive at the time of publication of the present study. The 1-, 5-, and 10-year
risks of additional reoperation (CABG, MVR, or MV repair) were 1.61%, 3.62%, and 26.19%, respectively (Figure 5).

Discussion

The combination of MVR and MV repair with CABG is generally considered to have a greater early and late mortality than either procedure alone. CAD is often associated with MV disease but may not be the cause of the valve dysfunction. Furthermore, the mechanism of ischemic MV regurgitation has been a source of controversy. Acute or chronic myocardial ischemia may cause left ventricular dysfunction and lead to structural changes in the MV, in the form of posterior annular dilatation, restricted leaflet motion, or papillary muscle dysfunction or rupture. Ruptured chordae are more likely to be secondary to primary myxomatous changes, which may proceed the development of ischemic disease. In these cases, the CAD and the MV disease are etiologically not related. Most patients in this study developed MV dysfunction after previous CABG. However, in at least 15 patients in this study, detectable mitral dysfunction had been reported during initial CABG, and in 10 of these patients, the origin of the dysfunction could not be determined. In no patients was the regurgitation thought to be severe enough to warrant a surgical procedure on the MV. In general, of all patients who undergo primary CABG, 3% to 5% have ischemic mitral regurgitation with no structural abnormalities of the valve. In most patients, the regurgitation is not significant, and, therefore, surgical intervention is not indicated. In this series, most of the patients (60%) subsequently developed ischemic mitral regurgitation with the progression of CAD. Few studies have addressed this high-risk group with combined CAD, previous CABG, and severe mitral regurgitation. Our in-hospital mortality of 8.8% is comparable with reported operative mortality for primary CABG and MVR and MV repair. Current data from the Society of Thoracic Surgery National Cardiac Surgery Database indicate an operative mortality of 13% for primary CABG and MVR and 8% for primary CABG and MV repair, and reoperation would be expected to increase operative risk. Conflicting data have been reported considering risk factors for early and late mortality after combined MVR and CABG that probably reflect the heterogeneity of this group and the different surgical approaches that have been performed. In the present report, EF ≤ 50% was the only significant risk factor for early death. Indices of preoperative left ventricular function have been found to adversely influence early and late mortality in patients undergoing CABG and mitral valve surgery. Patients at risk for each 2-year period and SEM are included.
most studies of isolated MVR, CABG, MV repair, and CABG.1,16–19,21

Although origin of MV disease was not a statistically significant risk factor for early mortality, a strong trend was apparent; no early death occurred in patients with myxomatous disease ($P=0.18$). Previous studies of primary MVR and MV repair with CABG have not shown a consistent correlation between origin and early mortality.1,9,22,23 On the other hand, others have found that ischemic mitral regurgitation was a predictor for early death after combined MVR and MV repair and CABG.14,24 Our failure to find this may reflect the small numbers in each group in the present study.

The indications for surgery in the present study were mainly congestive heart failure and severe mitral regurgitation. However, 50% of the patients had progression of CAD or developed significant graft disease requiring repeat CABG. The extent of the CAD, the existence of left main disease, and concomitant repeat CABG did not increase the operative risk. This is consistent with a recent report by Cohn et al.25 that demonstrated decreased operative risk of primary surgical treatment of mitral regurgitation with or without CABG. We believe the basic principles of safe resternotomy, minimal dissection of existing grafts, and good myocardial protection remain fundamental to a successful procedure. For patients who do not need repeat CABG, a right thoracotomy approach to the MV may be applied, as was undertaken in 6 patients.

Forty-six late deaths occurred, 35 of which were cardiac related. Five- and 10-year survival of 61% and 38% in this group are rates comparable with those of other reports of primary MVR and CABG.1,18,22 EF $<50\%$, NYHA class IV, use of the intra-aortic balloon pump, and urgent or emergency operation were significant predictors of late death. Left ventricular dysfunction has been found to be a significant predictor of adverse long-term survival in many studies of MVR or MV repair and CABG.1,16–19,21 In a recent analysis of long-term survival in 409 patients with myxomatous mitral regurgitation, we have demonstrated that preoperative EF was the best predictor of long-term mortality, congestive heart failure, and postoperative left ventricular function, even when other important predictors such as age and presence of CAD are included.26 We also found that after adjustment for age at operation and all other determinants of outcome, severe preoperative symptoms (such as NYHA class IV) were associated with a worse long-term survival and excess incidence of heart failure.26,27 In the present study, patients with preoperative NYHA class IV had a 5-year survival of 45% compared with 75% for those with NYHA functional class I, II, or III ($P=0.0006$). In recent years, studies have shown that the strategy of earlier operation for mitral regurgitation, even after previous surgery, may improve late outcome.28 We could not show a correlation between origin of the MV disease and late mortality, although a trend was noted toward increased mortality in the ischemic group. Previous studies of primary MVR and CABG demonstrated significant adverse late outcome for patients with ischemic mitral regurgitation.24

Numerous studies on mitral valve–conserving operation have been published in the past decade.29 Most studies have demonstrated that MV repair or preservation of the subvalvular apparatus when the valve is replaced decreases operative mortality and prolongs survival.23 Others have not found such a relationship; instead, clinical variables such as age, presence of CAD, and left ventricular function have been more important predictors of outcome.19 In the present study, 58% of the patients underwent MV repair. Although we could not demonstrate a statistically significant difference in early mortality between valve repair (3 of 46 patients; 8.3%) or replacement (4 of 34 patients; 11.8%) or in late survival, we continue to believe that valve repair should be considered if possible in cases of myxomatous or ischemic origin. The relatively high percentage of patients with ischemic or myxomatous mitral regurgitation in whom the valve was replaced may reflect the complexity of MV surgery after previous CABG. In patients with patent grafts, particularly a patent mammary artery to left anterior descending artery, and who do not require repeat CABG, the surgeon may minimize dissection of the heart to prevent potential complications, thus compromising exposure of the mitral valve, which is a key to a successful repair. In these cases, if the pathology of the valve or the mechanisms of regurgitation are uncertain, replacement of the valve is more likely. In addition, if the repair looks less than optimum, then replacement should be performed.

Limitations of the Study

This is a retrospective review of a relatively small number of patients who have various origins and pathophysiologies of MV disease in addition to CAD. Moreover, $\sim 60\%$ of the patients had initial surgery elsewhere, and we were unable to obtain all data regarding MV status at the time of initial CABG.

Conclusions

Ischemic, myxomatous, rheumatic, or infective MV disease may subsequently progress or develop after CABG. In most patients, mitral regurgitation is the principal pathophysiology, and presenting symptoms of congestive heart failure and angina are common. The operative risk is low, particularly in patients with myxomatous disease. Repeat myocardial revascularization at the time of MV surgery does not increase this risk. Repair or replacement of the valve should be undertaken before deterioration of the left ventricle occurs, to improve early and late outcome.

References

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