Perioperative outcome and long-term survival of surgery for acute post-infarction mitral regurgitation

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Abstract

Objective: To determine factors predictive of mortality in patients undergoing emergency mitral valve surgery in the setting of severe post-infarction regurgitation. Methods: Patients admitted for an acute myocardial infarction who required urgent mitral valve surgery for severe regurgitation were studied. Factors predictive of outcome were analysed. Results: Fifty-five consecutive patients (mean 65 ± 10 years, 37 males) were included. The infarct was inferior in 31 patients, posterior in 10, anterior in 9 and lateral in 5. Thirty-four patients (62%) were in Killip class IV. Peroperative findings confirmed total papillary muscle rupture in 25 patients (posteromedial in 21, anterolateral in 4), and partial rupture in 12 patients (posteromedial in 10, anterolateral in 2). Papillary muscle dysfunction without rupture was responsible for regurgitation in 18 patients (posteromedial in 15, anterolateral in 3). The mitral valve was replaced by a prosthesis in all but 4 patients, who had valvuloplasty. Coronary angiography was done in 32 patients, of whom 18 underwent concomitant coronary artery bypass grafting and 2 balloon angioplasty. Surgery was performed on average 7 days after infarction. Thirteen patients (24%) died during the perioperative period. Absence of coronary revascularisation was significantly associated with increased perioperative mortality (34% vs. 9%, P = 0.02). Of the 42 surviving patients, there were 5 deaths during a mean follow-up of 4.0 ± 3.7 years. Conclusion: In patients with acute post-infarction mitral regurgitation, perioperative mortality is high, but can be improved with concomitant CABG in addition to valve surgery. Long-term outcome of survivors is favourable.

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1. Introduction

Severe mitral regurgitation (MR) occurs in about 3% of patients presenting with an acute myocardial infarction (AMI) [1], and in about 10% of those with an AMI and shock [2]. This is a severe complication, and may be associated with an in-hospital mortality of up to 70% [2]. Since the first valvular replacement performed in this setting by Austen in 1965 [3], several reports have emphasised that the prognosis was greatly improved by surgical management [4,5]. However, the operative mortality remains high. One possible explanation for this finding may be the lack of concomitant revascularisation. This may be due to the poor hemodynamic condition of many of these patients, making it difficult to perform coronary angiography. The present study was undertaken to determine the factors predictive of mortality in patients undergoing emergency mitral valve surgery in the setting of severe post-infarction MR.

2. Methods

Charts of patients admitted to the coronary care unit of our institution for AMI from April 1985 to June 2002 were reviewed. Patients with acute MR (defined as occurring within 1 month of the infarction) who required emergency or urgent mitral valve surgery were thus identified. AMI was confirmed by typical ECG changes (ST segment elevation of >0.1 mV in at least two limb leads or >0.2 mV in at
least two pre-cordial leads) and a rise of serum Creatine Kinase (CK) and CK MB fraction. Significant stenosis at coronaryography was defined as > 75% reduction of vessel diameter (>50% for the left main vessel).

Patients were sorted according to Killip class. Cardiogenic shock was defined as a systolic arterial pressure <90 mmHg, urine output <30 ml/h and clinical evidence of systemic hypoperfusion.

Follow-up was done using outpatient records and by mail or phone contact.

2.1. Statistical analysis

Statistical association between perioperative deaths and the following variables were tested by Fisher’s exact test: age > 65 years, gender, infarct location, MR mechanism, and a delay between infarct diagnosis and surgical treatment of > 6 days. Perioperative survival at 30 days according to pre-operative Killip class and revascularisation status was estimated by the Kaplan-Meier method and the level of significance evaluated by the log rank test. Commercially available software packages used were STATXACT for Windows (exact tests) and SPSS 7.5 (Kaplan-Meier).

3. Results

The population was made up of 55 patients (37 men and 18 women, age 65 ± 10 years, range: 37–82). Fifteen patients had a history of angina pectoris or a prior AMI. Infarct location, defined by ECG criteria, was inferior in 31 patients, posterior in 10, anterior in 9 and lateral in 5. MR was clinically suspected by a systolic murmur associated with pulmonary oedema and/or cardiogenic shock in all but 3 cases. Transthoracic echocardiography demonstrated entire rupture of a papillary muscle in 8 patients, and mitral valve prolapse in another 11 patients. Transoesophageal echocardiography was also done in 29 patients, showing total papillary muscle rupture in 6 patients, partial rupture in 7 patients (of whom 1 was found to have total rupture peroperatively), and valvar dysfunction in 15 patients. Right-sided catheterisation was done in 23 patients. The mean capillary wedge pressure was 19.6 ± 9.8 mmHg (range: 3–36 mmHg) and a prominent V wave was noted in 7 cases (38.6 ± 14.2 mmHg, range: 21–59 mmHg). Left ventriculography revealed severe MR in all 16 patients in whom it was performed. Coronary angiography was done in 32 patients and revealed significant stenosis of the left anterior descending artery (LAD) or its branches in 19 cases, of the left circumflex in 20 cases, of the right coronary artery in 18 cases, and of the left main in 1 case. Twelve patients had single-vessel disease, 13 had two-vessel disease (including the patient with left main stenosis), and 7 three-vessel disease. Preoperative left ventricular function was reported in 37 patients, and was 51 ± 14%.

The mean delay between AMI and mitral valve surgery was 7.3 ± 7.4 days (range 1–33 days). Surgery took place within the first 24 h of diagnosis of MR in 24 patients, between the second and the fourteenth day in 27 cases, and after the second week in 4 cases. Twenty-eight patients received inotropic agents while awaiting surgery and 10 required mechanical ventilation. Eleven patients had an intra-aortic balloon pump inserted for hemodynamic stabilisation prior the surgical intervention.

Peroperative findings confirmed total papillary muscle rupture in 25 patients (posteromedial in 21, anterolateral in 4), and partial rupture in 12 patients (posteromedial in 10, anterolateral in 2). MR due to dysfunction of the subvalvular apparatus without rupture of the papillary muscle was found in the remaining 18 cases (posteromedial in 15, anterolateral in 3). A mechanical prosthetic valve was inserted in 47 cases (Saint Jude Medical in 31, Duromedics in 6, CarboMedics in 1, Lillehei-Kaster in 7, Wessex in 1, ATS in 1). Bio-prostheses were inserted in 4 cases (Carpentier-Edward in 2, Liotta in 1, Mosaic in 1). Four patients underwent mitral valvuloplasty (ring annuloplasty in all 4 with reinsertion of a ruptured anterior papillary muscle in 1). Amongst the 32 patients in whom coronary angiography was performed, 18 had coronary artery bypass grafting (CABG), 17 with venous grafts (sequential bypass to the left anterior descending artery and the left circumflex artery in 8 patients, to the left anterior descending artery in 4, and to an obtuse marginal branch of the left circumflex artery in 5). One patient had an internal mammary artery graft to the LAD. Two patients with single-vessel disease had balloon angioplasty with stenting (of the LAD and of the circumflex). In addition to valvular surgery, left ventricular aneurysmectomy was performed in one patient. In another patient, repair of a ventricular septal defect was attempted.

Thirteen patients (24%) died during the perioperative period (within one month of the intervention). Two patients died during surgery, due to unsuccessful repair of a ventricular septal defect in one and refractory cardiogenic shock in the other. Four patients died during the first 24 h following surgery (left ventricular free-wall rupture in 2 patients and intractable shock in the others). Four patients died of heart failure on days 3, 5, 10 and 16. One patient died of sepsis and renal failure on day 3. One patient died on day 9 after acute thrombosis of the prosthetic valve, and there was one death on day 18 due to staphylococcal mediastinitis. Thirty-day survival curves according to revascularisation status are depicted in Fig. 1.

The mean follow-up was 4.0 ± 3.7 years (range 1 month to 11 years). Among the 42 surviving patients, one was lost to follow-up. Five patients died of unidentified causes at 3, 3, 33, 37, and 97 months after surgery. Long-term survival is depicted in Fig. 2. All patients were in NYHA class I or II during follow-up.

Statistical analysis (Table 1) revealed that only the absence of coronary revascularisation was significantly predictive of increased perioperative mortality, with a trend
of increased mortality in patients with Killip class IV prior to surgery.

4. Discussion

This is the largest report of patients undergoing surgery for severe MR shortly after an AMI. The main finding of our study is that these patients benefit from concomitant CABG. Our results are comparable to those of a series of 22 patients with post-infarction papillary muscle rupture, in which concomitant CABG was the only factor that improved both immediate and long-term survival [6].

Table 1
Factors predictive of perioperative mortality

<table>
<thead>
<tr>
<th></th>
<th>Perioperative deaths/total (%)</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 65 years</td>
<td>9/31 (29)</td>
<td>0.35</td>
</tr>
<tr>
<td>&lt; 65 years</td>
<td>4/24 (17)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
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<td></td>
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<tr>
<td>Female</td>
<td>4/18 (22)</td>
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<tr>
<td>Male</td>
<td>9/37 (24)</td>
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<tr>
<td>Infarct location</td>
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</tr>
<tr>
<td>Inferior</td>
<td>10/31 (32)</td>
<td>0.45</td>
</tr>
<tr>
<td>Posterior</td>
<td>1/10 (10)</td>
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<tr>
<td>Anterior</td>
<td>1/9 (11)</td>
<td></td>
</tr>
<tr>
<td>Lateral</td>
<td>1/5 (20)</td>
<td></td>
</tr>
<tr>
<td>Etiology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete rupture</td>
<td>6/25 (24)</td>
<td>0.92</td>
</tr>
<tr>
<td>Partial rupture</td>
<td>2/12 (17)</td>
<td></td>
</tr>
<tr>
<td>Dysfunction</td>
<td>5/18 (28)</td>
<td></td>
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<tr>
<td>Killip class</td>
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<td></td>
</tr>
<tr>
<td>III</td>
<td>2/21 (10)</td>
<td>0.06</td>
</tr>
<tr>
<td>IV</td>
<td>11/34 (32)</td>
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<tr>
<td>Revascularisation</td>
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<td>Yes</td>
<td>2/23 (9)</td>
<td>0.02</td>
</tr>
<tr>
<td>No</td>
<td>11/32 (34)</td>
<td></td>
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<tr>
<td>Delay to surgery</td>
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</tr>
<tr>
<td>&gt; 6 days</td>
<td>9/32 (28)</td>
<td>0.52</td>
</tr>
<tr>
<td>&lt; 6 days</td>
<td>4/23 (17)</td>
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<tr>
<td>LVEF</td>
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<td>&lt; 0.5</td>
<td>2/17 (12)</td>
<td>0.67</td>
</tr>
<tr>
<td>&gt; 0.5</td>
<td>4/20 (20)</td>
<td></td>
</tr>
</tbody>
</table>

4.1. Mechanism of MR

Over half of our patients had papillary muscle rupture. Because of its single blood supply from distal branches of the posterior descending artery (derived from either the right or left circumflex coronary artery), the posteromedial papillary muscle is most frequently involved. The anterolateral papillary muscle has a dual blood supply, from the left anterior descending and left circumflex coronary arteries, making it less vulnerable to ischaemia. Other causes of post-infarction MR are leaflet prolapse due to reduced tethering by the infarcted papillary muscle, or elongation of the chordae with possible rupture. MR may also result from an apical displacement of the papillary muscle during the remodelling process, as well as from generalised ventricular dysfunction with annular dilatation. Patients with papillary muscle rupture have been reported to differ from those without rupture in that they usually have a more
limited infarct with a better-preserved left ventricular function [7]. Preserved contractility may thus facilitate this complication as it is does for left ventricular free-wall rupture [8].

4.2. Perioperative mortality and long-term survival

Perioperative mortality was 24%, and is comparable to that of some series on surgery for post-infarction papillary muscle rupture [6,9,10]. Other series on post-infarction MR in patients with hemodynamic compromise report a perioperative mortality rate of about 40–50% [2, 5,7]. We and others [5,7] have not observed any difference in perioperative survival between papillary muscle dysfunction and rupture. Our results and those of others [6] suggest that the long-term prognosis of patients surviving the operation is favourable.

4.3. Mitral valve replacement or repair

In our series, only 4 of 55 patients (7%) underwent mitral valve repair. This is, however, comparable to other series that recruited patients since the 1980’s: 2 of 33 patients (6%) in one report [9] and 2 of 21 (10%) in another [10]. Experience with mitral valve repair is more recent, and may explain why the incidence is so low. The more recent SHOCK series [2] dating since 1993, reports a higher incidence of repair: 6 of 43 patients (13%).

Several series have shown favourable results with repair of ischaemic MR [11–13]. Some observational reports suggest that perioperative survival is superior with repair as compared to replacement [14,15]. This may be related to a shorter duration of surgery, avoiding complications associated with prosthetic valves, and better preservation of left ventricular function. However, it may also be due to a selection bias with repair being more often done in patients with a more limited infarction.

Different techniques of repair have been described [16] and consist of annuloplasty, suture of the ruptured papillary muscle head, or use of other techniques such as chordal transfer. When there is extensive necrosis of the papillary muscle and ventricular wall, repair of the friable tissue may be hazardous, and it is safer to perform valve replacement with preservation of the chordae tendenae [16]. A rare but often lethal complication of mitral valve replacement is ventricular free wall rupture, which was the cause of death of two of our patients, and has previously been described in the setting of posterior infarction [17]. This complication has never to our knowledge been described with valve repair.

4.4. Angiographic evaluation and role of reperfusion therapy

In the present study, statistical analysis revealed that patients with no coronary revascularisation and those with pre-operative Killip class IV had significantly higher perioperative mortality. Revascularisation may improve outcome by preventing recurrent ischaemia as well as favouring remodelling of infarcted tissue. Coronarography is usually a pre-requisite for CABG. In these acutely ill patients, however, this procedure may be associated with hemodynamic compromise, worsening of heart failure, ventricular arrhythmia, and impairment of renal function. Administering non-ionic contrast agents and avoiding ventriculography may limit these complications. Finally, coronarography may allow revascularisation by percutaneous balloon angioplasty, and avoid the need for surgery in selected patients. Data on the impact of balloon angioplasty in reducing ischaemic MR are sparse and inconsistent. A few cases of dramatic improvement with angioplasty have been reported [18–21], whereas in another series [1] valvular competence was not restored.

4.5. Timing of surgery

We attempted to analyse effect of timing by comparing outcome in patients who underwent surgery <6 days from diagnosis of MI as compared to after a week of the index infarction, and found no difference. This may indicate that there is nothing to be gained by deferring surgery. Even though the fragility of the infarcted myocardium may prompt some surgeons to wait, most authors recommend early intervention [4,9,10,22–24], even though data supporting this approach are lacking. The timing of intervention for ischemic MR not related to papillary muscle rupture remains controversial, and is less clearly defined than for papillary muscle rupture. In the present report, the mean interval from myocardial infarction to operation was a week (and was as early as 1 day). Previous series have reported surgery within a week of infarction in about half of the study population [6,10]. Intra-aortic balloon pumping may result in significant salvage in this population of patients with hemodynamic compromise. However, even if this allows temporary stability, surgery should nevertheless be urgently carried out, as clinical deterioration usually recurs. Furthermore, it is known that patients who are initially stable on medical treatment may deteriorate rapidly. We and others [2,5,7,22,25] have shown that hemodynamic compromise portends a dismal prognosis. Therefore, it would seem wise to program surgery as soon as the necessary investigations have been completed.

4.6. Limitations of the study

The main weaknesses of our study are that data were collected retrospectively (with obvious limitations in data
precision), and that management was not randomised. Despite our series being the largest reported to date, the number of perioperative deaths was too low to make multivariate analysis meaningful. Coronarography leading to coronary artery bypass grafting (CABG) may have been done only in those patients stable enough to undergo this procedure. Thus the favourable effect of revascularisation on outcome may purely be artefactual due to a selection bias. Post-operative hemodynamic instability is a major cause of mortality, and myocardial protection strategies may contribute significantly to outcome. However, our report does not specifically look at post-operative management, and this issue cannot be properly addressed. Furthermore, patients were treated by different physicians, leading to different management strategies. Patients were admitted across a 17-year period, and availability of coronaryography has obviously evolved over these years. This may explain why many patients did not undergo coronaryography. Furthermore, it was difficult, based on patient files, to ascertain why patients with coronary artery disease on angiography did not undergo revascularization. Because only four of our patients had valvuoplasty, our data will not help in resolving the issue of whether valve repair is better than replacement.

5. Conclusion

In light of our experience and that previously reported, we propose an optimal management of initial hemodynamic stabilisation (if necessary with intra-aortic balloon pumping) followed by transthoracic and/or transoesophageal echocardiography to assess the mechanism of MR and ventricular function. Coronarography should be done whenever possible, and surgery performed without delay, whenever possible, and surgery performed without delay, if the infarct is limited, or if the regurgitation is due to mechanical complications after acute myocardial infarction. J Am Med Assoc 1986;256:47–50.


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References


