Original Article

Archives of Clinical & Experimental Surgery

Mid-Term Outcome of Mitral Valve Repair and Coronary Artery Bypass Grafting for Ischemic or Degenerative Mitral Regurgitation

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Received: March 12, 2012
Accepted: April 12, 2012
Arch Clin Exp Surg 2012;1:129-137
DOI:10.5455/aces.20120412025840

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Abstract

Aim of the study: To verify the impact of the etiology of mitral valve regurgitation on a 5-year outcome after repair and concomitant coronary artery bypass grafting (CABG).

Methods: One hundred and eleven consecutive patients (mean age of 69±8 years) who underwent mitral valve repair, 65 for ischemic and 46 for degenerative mitral regurgitation, and concomitant CABG, were retrospectively analyzed. The mean follow-up was 40±28 (9-104) months. Five-year survival (including operative mortality), and survival free from events (postoperative low output syndrome, progression of mitral regurgitation, onset or worsening of congestive heart failure, recurrence of myocardial infarction, and the need for mitral valve replacement) were analyzed.

Results: Compared with degenerative, ischemic mitral regurgitation was associated with a higher incidence of previous myocardial infarction (P<0.0001), left ventricular ejection fraction (LVEF) <0.45 (P<0.0001), and more diseased coronary vessels per patient (P<0.0001). Five-year all-cause mortality was 18% (20/111). Independent predictors of mortality were older age at operation (P=0.0008), LVEF<0.45 (P=0.04), and the ischemic etiology of mitral regurgitation (P=0.03). At five years, survival was 69%±7.6% for ischemic versus 87%±6.5% for degenerative etiology (P=0.03); event-free survival was 58%±8.4% versus 75%±8% (P=0.02), and freedom from late cardiac death was 85%±6.6% versus 100% (P=0.02). Freedom from mitral valve reoperation was 97±2.4%.

Conclusions: Ischemic mitral regurgitation "per se" predicted limited survival and event-free survival. Left ventricular dysfunction is frequently associated with the ischemic etiology. An early surgical indication to prevent left ventricular dysfunction could be important to improve the mid-term outcome.

Key words: *Mitral valve repair, degenerative mitral valve disease, ischemic mitral valve disease, coronary artery bypass grafting surgery*

Introduction

With current surgical techniques, most degenerative mitral valves can be repaired. Advantages of mitral valve repair, compared to replacement, include improved survival, better freedom from reoperation, and better preservation of left ventricular function [1]. Surgery for ischemic mitral regurgitation has been associated with a poor long-term outcome and survival,

compared with degenerative mitral valve regurgitation [2]. Mitral valve repair, compared with replacement, has also improved prognosis in patients affected by ischemic disease, but it has remained unclear as to whether the ischemic etiology of mitral regurgitation, compared to degenerative disease, remains an independent predictor of early and late outcomes [3-5].

The aim of the study is to analyze the impact of ischemic etiology, compared with degenerative etiology, of mitral regurgitation on survival and mid-term outcomes after mitral valve repair and concomitant coronary artery bypass grafting, and to identify independent risk factors.

Materials and Methods

One hundred and eleven consecutive patients (mean age of 69±8 years, 79 males) who underwent mitral valve repair and concomitant coronary artery bypass grafting (CABG) (65 patients for ischemic mitral regurgitation, 46 for degenerative mitral regurgitation etiology) from January 2002 to January 2010 were retrospectively analyzed.

The study was approved by our local Institutional Review Board, which waived the need for patient consent.

Preoperatively, New York Heart Association (NYHA) functional class I, II, III and IV were 18%, 30%, 37%, and 15%, respectively. Mitral insufficiency, evaluated by a 1 to 4 grade scale measured by transthoracic echocardiography, at cardiac catheterization, or both, was present as mild (2+/4) in 31 (28%), moderate-severe (3+/4) in 49 (44%), and severe (4+/4) in 31 (28%) patients. Preoperatively, 17 patients (15%) were affected by atrial fibrillation; 4 (3.6%) already had a permanent pacemaker implanted. Left ventricular ejection fraction (LVEF) was measured preoperatively by echocardiography or cardiac catheterization. The mean value of LVEF was 0.47±0.14 (range, 0.20-0.75; median value, 0.46).

Chronic renal dysfunction, as defined when the preoperative serum creatinine level was higher than 1.4 mg/100 mL, was present in 16 patients (14.4%); chronic obstructive pulmonary disease was present in 21 (19%).

Surgery for mitral valve repair, independently from coronary artery disease, was indicated for severe or moderate mitral regurgitation and evidence of an increase in left ventricular diameters at serial echocardiograms, or paroxysmal atrial fibrillation. Concomitant CABG was performed in the presence of left main stem stenosis >50%, or single-, double- or triple-vessel coronary artery disease with stenosis >50% evaluated by means of selective coronary angiography.

Surgery for CABG, independently from mitral valve regurgitation, was given in the presence of left main stem stenosis >50%, or triple-vessel coronary artery disease or double-vessel coronary artery disease with proximal left descending artery stenosis >70%. Concomitant mitral valve repair was performed for a grade of regurgitation equal or greater than mild (2+/4).

A left atrial tissue ablation procedure, by means of mono- or bipolar radiofrequency, was added in order to treat paroxysmal or persistent atrial fibrillation.

Patients requiring emergency surgery for acute ischemic mitral regurgitation, surgery for isolated degenerative mitral regurgitation, redo surgery or concomitant procedures on an aortic valve or ascending aorta, or left ventricular aneurysmectomy were excluded from the study.

Assessment of Etiology of Mitral Regurgitation

Mitral regurgitation was defined as ischemic according to clinical history, presence of one or more previous myocardial infarctions, normal valve leaflets and chordae at transthoracic and transesophageal echocardiography, the mechanism of regurgitation demonstrated to be depending on annular dilation due to left ventricular remodeling (Carpentier type I classification), or on restricted leaflet motion (Carpentier type IIIb classification).

Mitral regurgitation was defined as degenerative when at echocardiographic or surgical inspection leaflets prolapse, chordal elongation or a rupture with or without annular dilation was found (Carpentier type II classification).

Surgical Management and Techniques of Valve Repair

Mitral valve repair and CABG were performed in all cases on cardiopulmonary bypass and intermittent antegrade tepid blood cardioplegia (600 mL as the first dose, followed by a 400 mL dose administered every 20–25 minutes) and mild hypothermia (33–34°C).

Surgical repair of a mitral valve was performed with standardized techniques in accordance with the type of regurgitation mechanism, as defined by the Carpentier classification [6,7]: triangular or quadrangular leaflet resection for the treatment of the prolapse of posterior leaflet; A2-P2 edge-to-edge technique for the correction of prolapse of the anterior leaflet or tethering of the leaflets; reductive annuloplasty was performed either using a commissural annuloplasty technique than with the use of a prosthetic ring.

Intraoperative transesophageal echocardiography was used in order to better evaluate the mechanism of mitral regurgitation before starting cardiopulmonary bypass, and immediately after performing the repair technique, to assess the effective result. The result of the repair was considered satisfactory when residual regurgitation was less than mild.

The internal thoracic artery was always used for revascularization of the left descending anterior artery when required.

Data Collection

Operative mortality included death in hospital after operation at anytime or within 30 days after discharge. Postoperative low output syndrome was defined when the cardiac index value was equal to or less than 2.0 L per min-1 per m-2.

Transthoracic echocardiography was performed in all patients preoperatively, postoperatively and at follow-up. The mean follow-up (100% complete) was 40±28 (range 9-104) months. The status of every patient was ascertained during a 2-month period (September-October, 2010). The five-year outcomes considered were survival (including operative mortality), freedom from late cardiac mortality and survival free from events occurred during hospitalization (postoperative low output syndrome requiring intra-aortic balloon pumping or infusion of inotropic drugs, and death) or during follow-up (death, progression of mitral regurgitation, onset or worsening of congestive heart failure, recurrence of myocardial infarction, and the need for mitral valve replacement). Congestive heart failure was diagnosed if patients were affected by orthopnea or bilateral basal crepitation on auscultation, bilateral edema of the legs, congested neck veins, and hepatomegaly. All causes of death, data of echocardiographic examinations, cardiac events, and the functional status of the patients were recorded at the outpatient clinic visit or by a telephone interview.

Statistical Analysis

Statistical analysis was performed with Stat View 4.5 (SAS Institute Inc., Abacus Concepts, Berkeley, CA). Univariate analysis was performed using the Student's t-test for continuous data and the $\chi 2$ or Fisher's exact test for categorical data to compare preoperative and perioperative characteristics between patients with ischemic or degenerative mitral regurgitation.

Twenty preoperative variables were selected for analysis, including etiology of mitral regurgitation (ischemic vs. degenerative), age, gender, previous myocardial infarction, preoperative cardiac rhythm, smoking habit, comorbid disease (arterial hypertension, diabetes mellitus, peripheral vascular artery disease, chronic renal dysfunction, chronic pulmonary disease), Canadian Cardiovascular Society (CCS) grade of angina, New York Heart Association (NYHA) functional class for dyspnea, moderately depressed (less than 0.45) LVEF, left ventricular end-systolic and end-diastolic diameters, left atrial diameter, systolic pulmonary artery pressure, the type of mechanism of mitral regurgitation (i.e. posterior, anterior, or both leaflet prolapse, tethering, annular dilation), and the number of diseased coronary artery vessels per patient.

Operative variables, including techniques of mitral valve repair (quadrangular resection of posterior leaflet, edge-to-edge technique, annuloplasty with or without use of a prosthetic ring), cardiopulmonary bypass and aortic cross-clamp times, and the number of grafts per patient, were also analyzed.

The Cox proportional hazards method was used to evaluate the influence of variables on time to death. Overall survival (including operative mortality), event-free survival, freedom from late cardiac death, and freedom from reoperation were expressed as mean values plus or minus 1 standard error, and were computed by using the Kaplan-Meier method; the log-rank test was used to compare survival estimates among subgroups. All continuous values were expressed as mean plus or minus 1 standard deviation of the mean. All P values <0.05 were considered statistically significant.

Results

In-hospital Results

Patients with ischemic etiology, compared to those with degenerative etiology, were in a higher CCS class (P=0.008), were more frequently affected by previous myocardial infarction (P<0.0001), peripheral vascular artery disease (P=0.04), and had a lower preoperative mean value of LVEF (P<0.0001), a greater incidence of LVEF less than 0.45 (P<0.0001), a greater value of a left ventricular end-systolic diameter (P<0.0001), and a greater number of diseased coronary vessels per patient (P<0.0001). Patients with degenerative etiology had a more severe grade of mitral regurgitation (P=0.0005) and a greater left atrial diameter (P=0.002). Other variables are reported in Table 1. The mechanism of mitral regurgitation is reported in Table 2.

Annuloplasty with the use of a prosthetic ring was performed in 84 patients: in 77 (91.7%), a Cosgrove C-incomplete ring (CG Future Band Ring, Medtronic, Minneapolis, Minnesota, USA) was implanted, and in 7 (8.3%), a semi-rigid ring (Semi-Rigid Seguin Ring, St. Jude Medical, St. Paul, Minnesota, USA) was used.

A simple reductive commissural annuloplasty without the use of a ring was performed in 22 patients.

Other procedures were an edge-to-edge technique in 32 patients (29 in association with annuloplasty, 3 without) and triangular or quadrangular resection in 13 (11 in association with annuloplasty, 2 without). Associated reductive tricuspid valve annuloplasty according to a modified Kay technique was performed in 9 patients. Left atrial tissue ablation for atrial fibrillation was performed in 10 patients.

Patients with ischemic mitral regurgitation required a higher number of grafts per patient (Table 3).

The incidence of postoperative low output syndrome requiring intra-aortic balloon pumping and/or inotropic drugs was 9.2% in the ischemic group, and was absent in the degenerative group (P=0.03).

Postoperative echocardiography showed no residual mitral regurgitation in 71 patients (64%), was trivial

Table 1. Baseline and Preoperative Characteristics.

Characteristic	All patients (N.=111)	IMR (N.=65)	DMR (N.=46)	P value (IMR vs. DMR)
Age, years	69±8	68.6±8	69.5±9	0.566
Male gender, N. (%)	79(71)	45(69)	34(74)	0.590
NYHA class, mean value	2.5±1.0	2.4±1.0	2.6±0.8	0.215
CCS class, mean value	1.8±1.2	2.1±1.2	1.3±0.8	0.008
Previous myocardial infarction, n (%)	46(41)	41(63)	5(11)	<0.0001
Smoking habit, N. (%)	76(68.5)	44(68)	32(70)	0.787
Diabetes mellitus, N. (%)	39(35)	27(42)	12(24)	0.053
Hypertension, N. (%)	84(76)	51(78)	33(72)	0.416
Peripheral vascular artery disease, N. (%)	30(27)	24(37)	6(13)	0.042
COPD, N. (%)	21(19)	16(26)	5(11)	0.061
CRD, N. (%)	16(14.4)	12(18.5)	4(8.7)	0.138
Atrial fibrillation, N. (%)	17(15)	7(11)	10(22)	0.259
Mitral regurgitation, mean value /4+	3.0±0.8	2.8±0.74	3.3±0.75	0.0005
Left ventricular ejection fraction, mean value	0.47±0.14	0.40±0.12	0.57±0.10	<0.0001
Left ventricular ejection fraction <0.45, n (%)	52(47)	45(69)	7(15)	<0.0001
LVEDD, mm	59.8±7.5	59.7±7	59.8±8	0.959
LVESD, mm	43±8	46±9	39±6	<0.0001
Left atrium, mm	44±8	42±6	47±9	0.002
Systolic pulmonary art. pressure, mmHg	40.5±15	41±15	40±14	0.828
Number of vessels diseased per patient	2.3±0.8	2.6±0.7	1.8±0.8	<0.0001

IMR: ischemic mitral regurgitation; DMR: degenerative mitral regurgitation; NYHA: New York Heart Association; CCS: Canadian Cardiovascular Society; COPD: chronic obstructive pulmonary disease; CRD: chronic renal dysfunction; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter.

Table 2. Mechanism of Mitral Valve Regurgitation.

Mechanism	IMR (N.=65)	DMR (N.=46)
Isolated annulus dilation, N. (%)	38(58.5)	1(2.2)
Annulus dilation with single or both leaflets prolapse, N. (%)	0	12(26.1)
Annulus dilation and tethering of leaflet, N. (%)	8(12.3)	0
Tethering of leaflets, N. (%)	19(29.2)	0
Posterior leaflet prolapse, N. (%)	0	16(34.8)
Anterior leaflet prolapse, N. (%)	0	10(21.7)
Bilateral leaflet prolapse, N. (%)	0	7(15.2)

IMR: ischemic mitral regurgitation; DMR: degenerative mitral regurgitation.

Table 3. Independent Predictors of Survival (Cox Regression Analysis).

Characteristic	IMR (N.=65)	DMR (N.=46)	P value
Cardiopulmonary bypass time, minutes	123±31	112±28	0.057
Cross-clamp time, minutes	60±13	56±13	0.135
Number of grafts per patient	2.7±0.9	1.8±0.9	<0.0001
Size of prosthetic ring, mm	30±2.1	32±2.3	0.002
Tricuspid valve annuloplasty, N. (%)	6(9.2)	3(6.5)	0.602
Left atrial ablation procedure, N. (%)	5(7.7)	5(10.8)	0.567

IMR: ischemic mitral regurgitation; DMR: degenerative mitral regurgitation.

Table 4. Operative Variables.

Characteristic	OR	95% CI	P value
Age[a]	1.3	1.115–1.517	0.0008
Preoperative LVEF <0.45	3.9	1.027–14.68	0.045
Etiology (ischemic)	0.3	0.119–0.928	0.035
Gender (female)			NS
Chronic obstructive pulmonary disease			NS
Aortic cross-clamp timea			NS
Cardiopulmonary bypass timea			NS
Number of vessels diseased per patienta			NS
Number of grafts per patienta			NS

LVEF: left ventricular ejection fraction. CI: confidence interval; NS: not significant. [a]greater values

in 35 (31.5%), mild in 4 (3.6%), and mild-to-moderate in 1 (0.9%). Mitral valve stenosis was never observed, also after performing a double-orifice technique.

Five-Year Results

Five-year all-cause mortality, including operative mortality, was 18% (20/111): 15 deaths in the ischemic group, 5 deaths in the degenerative group of patients, respectively. Cardiac mortality was 9% (10/111).

Operative mortality was 7.7% (5/65) in the ischemic group. Causes of operative death included low output syndrome in 3 cases, myocardial infarction in 1, and respiratory failure in 1. There was no mortality in the degenerative group (P=0.054).

During follow-up, late all-cause mortality was 14.2% (15/106 patients). Six patients died from cardiac causes (congestive heart failure, n=3; acute myocardial infarction, n=1; sudden death, n=2), 3 for malignancy, 2 for renal insufficiency, 2 for respiratory insufficiency, 1 for complications related to diabetes mellitus, and 1 for unknown reasons.

Five-year survival for all patients was 77%±5%. On multivariate Cox analysis, independent predictors of survival were older age at operation (75±7 versus 68±8 years, P=0.0008), an LVEF value less than 0.45 (P=0.04), and the ischemic etiology of mitral regurgitation (P=0.03) (Table 4).

Five-year survival was 87%±6.5% in patients affected by degenerative etiology, compared with 69%±7.6% in those affected by ischemic etiology (P=0.03) (Figure 1).

Five-year survival was 83%±6.4% for patients with an LVEF value equal or greater than 0.45 versus

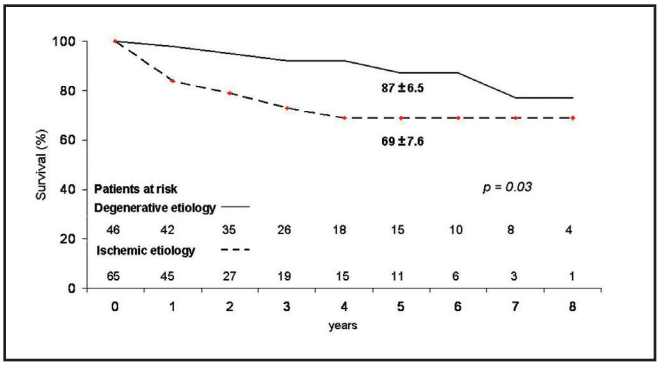

Figure 1. Survival after MVR and CABG during follow-up (40±28 months) in patients with ischemic and degenerative etiology (log-rank test). MVR: mitral valve repair; CABG: coronary artery bypass grafting.

70%±8.1% for patients with an LVEF value less than 0.45 (P=0.04) (Figure 2).

Ischemic vs. degenerative etiology and LVEF less than 0.45

Five-year survival was 90%±5.4% for patients (n=39) with degenerative etiology and LVEF equal or greater than 0.45, 75%±20% for patients (n=7) with degenerative etiology and LVEF less than 0.45, 66%±16% for patients (n=20) with ischemic etiology and LVEF equal or greater than 0.45, and 70%±8% for patients (n=45) with ischemic etiology and LVEF less than 0.45 (P=0.046).

Five-year event-free survival was 75%±8% in patients affected by degenerative etiology, compared with 58%±8.4% in those affected by ischemic etiology (P=0.018) (Figure 3).

Five-year event-free survival was 77%±7.3% for an LVEF value equal or greater than 0.45 versus 60%±9% for an LVEF value less than 0.45 (P=0.007) (Figure 4).

Five-year freedom from late cardiac death was 100% in patients affected by degenerative mitral valve regurgitation, compared with 85%±6.6% in those affected by ischemic mitral valve regurgitation (P=0.02) (Figure 5).

Five-year freedom from late cardiac death was 95%±4.6% for patients with a preoperative LVEF value equal or greater than 0.45 versus 88%±5% in other patients (P=0.04) (Figure 6).

Freedom from Redo Operation and Clinical Results

Five-year freedom from mitral valve reoperation was 97%±2.4%. One male and one female patient, both affected by degenerative regurgitation 15 and 24 months after mitral valve repair, required mitral valve replacement with a mechanical prosthesis, one for progression of posterior and anterior leaflet prolapse at level of A3 and P3 scallops, and the other for progression of posterior leaflet prolapse in P2 scallop. No patient required reoperation during follow-up for bypass graft failure.

At 40±28 months of follow-up, echocardiography showed significant improvement of the mitral regurgitation grade in all patients: 0.8±0.6 /4 versus 3.0±0.8 /4 preoperatively (P<0.00001); the left ventricular end-diastolic diameter decreased from 59.8±7.5 mm preoperatively to 55±10 mm (P=0.001); the end-systolic

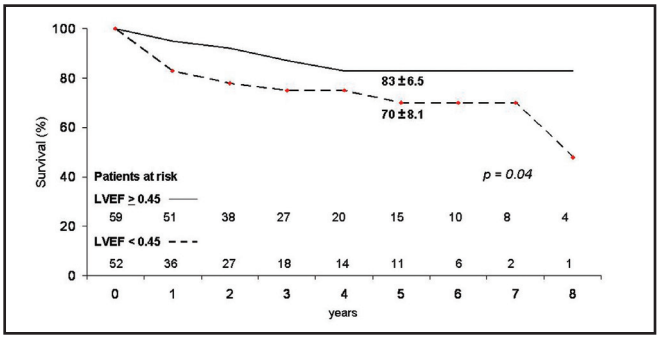

Figure 2. Survival after MVR and CABG during follow-up (40±28 months) in patients with and without LVEF less than 0.45 (log-rank test). MVR: mitral valve repair; CABG: coronary artery bypass grafting.

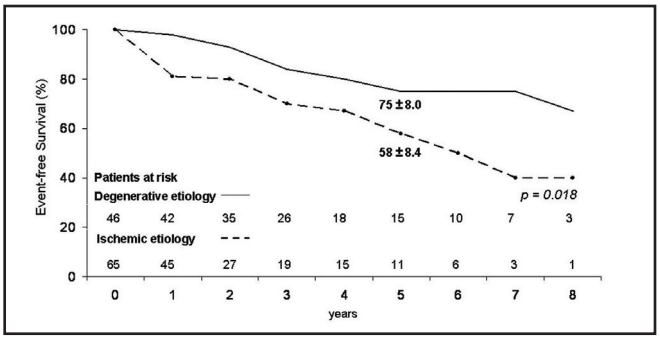

Figure 3. Event-Free Survival after MVR and CABG in patients with ischemic and degenerative etiology (log-rank test).
MVR: mitral valve repair; CABG: coronary artery bypass grafting.

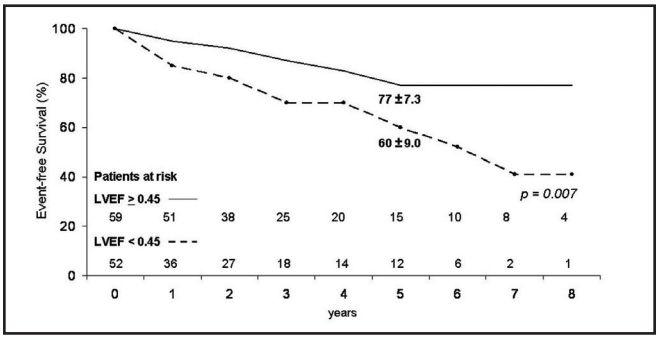

Figure 4. Event-Free Survival after MVR and CABG in patients with and without LVEF <0.45 (log-rank test). MVR: mitral valve repair; CABG: coronary artery bypass grafting; LVEF: left ventricular ejection fraction.

diameter decreased from 43±8 mm preoperatively to 41±10 mm, although with minor statistical relevance (P=0.05); systolic pulmonary artery pressure decreased from 40±16 mmHg preoperatively to 34±10 mmHg (P=0.001).

Residual mitral regurgitation at echographic follow-up was not significantly different in patients affected by degenerative (0.9±0.7 /4) or ischemic (0.7±0.6

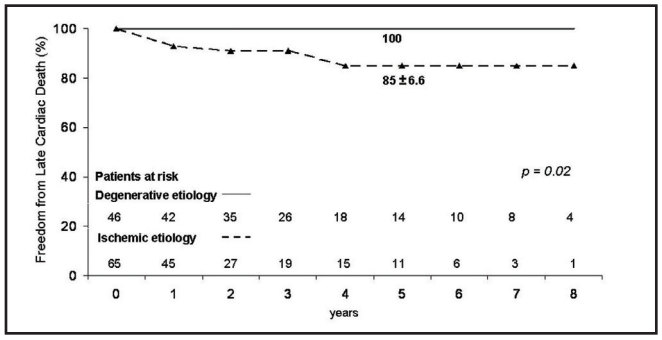

Figure 5. Freedom from late Cardiac Death after MVR and CABG in patients with ischemic and degenerative etiology (log-rank test). MVR: mitral valve repair; CABG: coronary artery bypass grafting.

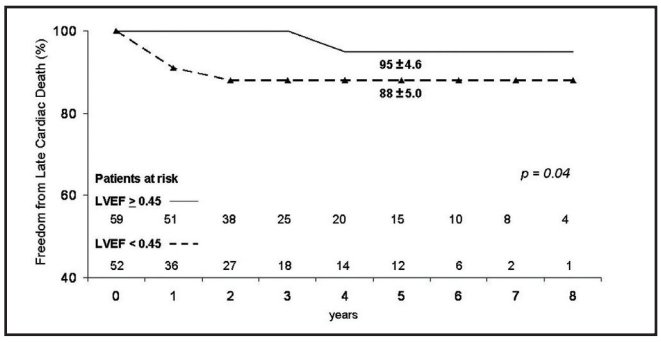

Figure 6. Freedom from late Cardiac Death after MVR and CABG in patients with and without LVEF <0.45 (log-rank test).
MVR: mitral valve repair; CABG: coronary artery bypass grafting; LVEF: left ventricular ejection fraction.

/4) etiology.

In all patients, the NYHA class improved from 2.5±0.9 preoperatively to 1.6±0.7 at follow-up (P<0.0001), and the CCS class from 1.8±1.1 preoperatively to 1.0±0.2 (P<0.0001). Freedom from non-fatal myocardial infarction was 100%.

Discussion

Mid-term Outcome after Mitral Valve Repair and Concomitant CABG

The operative mortality rate observed in our series (4.5%) as well as the 77% 5-year survival rate is comparable to that reported in most recent series [8-13]. The reduced mortality observed in this subset of patients during this decade could be related to a better perioperative management for the treatment of cardiac, pulmonary, renal, and gastrointestinal complications. The most important determinants of mortality in this subset of patients, as previously reported, include advanced age, left ventricular dysfunction, extent of coronary artery disease, and a higher NYHA functional class [4,8,14-16].

Moreover, mitral valve repair, compared to replacement, can better provide the maintenance of left ventricular-valvular continuity and the ventricular recovery from volume overload, in addition to the beneficial effect of CABG on left ventricular function.

Impact of Etiology on Survival and Event-free Survival

It is well known that treatment of ischemic mitral regurgitation shows worse in-hospital and late results, compared to treatment of degenerative mitral regurgitation. Seipelt and coworkers [17] reported a significantly higher operative mortality (19.5%) for patients with ischemic disease, compared to that (6.7%) of patients with non-ischemic mitral regurgitation. There are few data comparing late outcomes in these two groups of patients with both mitral regurgitation and coronary artery disease. In a review from the Mayo Clinic, Dahlberg and coworkers [18], on 302 patients subjected to mitral valve repair or replacement and CABG, reported that patients with ischemic mitral regurgitation had more left ventricular dysfunction, more extensive coronary artery disease, and higher NYHA. Ten-year survival was 33% in the ischemic group and 52% in the degenerative group.

However, in several studies [17-19], the ischemic etiology of mitral regurgitation was not recognized as a predictor of operative mortality. In our study, we found etiology "per se" (ischemic versus degenerative) as a predictor of reduced survival in a medium-term follow-up. Ischemic etiology and LVEF less than 0.45 were also associated with a lower event-free survival and with a lower freedom from late cardiac death (Figures 3-6).

In our series, operative mortality observed in the ischemic group of patients was higher, compared with the degenerative group, as well as the incidence of postoperative low output syndrome. Similarly, at 5-year follow-up, the survival rate, event-free survival and freedom from cardiac death were significantly different between the ischemic and the degenerative group of patients, with a difference in the outcome of 15-18 percent in favor of the degenerative group (Figures 1, 3, 5). These findings can be likely related, at least in part, to a worse preoperative clinical presentation of patients with ischemic etiology, represented by a significantly

higher incidence of comorbidity (diabetes mellitus, peripheral vascular disease), a greater number of coronary vessels diseased, and left ventricular dysfunction. However, among the above-mentioned variables, at the multivariable analysis only the LVEF less than 0.45 predicted worse survival, in association with ischemic etiology and older age. Of note, the incidence of LVEF less than 0.45 was very high (about three quarters of patients) in the ischemic group. This finding can be related to the higher incidence of previous myocardial infarction, to a more extended coronary artery disease, and therefore to a more advanced ischemic heart disease. However, we did not find that the severity of coronary artery disease was a predictor of an impaired outcome at multivariable regression analysis. This fact may be explained by our attempt to perform complete revascularization in all cases, in order to improve late freedom from ischemic events. The efficacy of CABG was well documented by the low rate of fatal or non-fatal myocardial infarction (1%) and the return of angina (1%) [16-20].

The strict correlation between reduced LVEF and ischemic etiology may explain, at least in part, the impact of etiology as a predictor of a worse outcome.

Conversely, the higher value of LVEF documented in the degenerative group can likely explain both satisfactory in-hospital results and higher rates of survival as well as freedom from cardiac events, although preoperative mitral valve regurgitation was more severe in these patients. Accordingly, when survival was stratified for the LVEF value, we found that the survival rate in patients with ischemic etiology, either with reduced or preserved LVEF, was significantly lower, compared to that of degenerative disease, to confirm the ischemic etiology as an important predictor.

These findings can suggest that mitral valve repair and concomitant CABG, especially in the presence of mitral regurgitation of an ischemic origin, should be indicated before that subsequent remodeling after myocardial infarction leads to left ventricular dysfunction, and therefore to a worse outcome. For these reasons, in association with a complete myocardial revascularization performed in all cases in order to supply as much viable myocardial as possible to improve left ventricular function, our policy is to treat mitral valve insufficiency for a less severe grade of regurgitation (≥2+ /4), in order to reverse ventricular remodeling and to reduce the left ventricular volume overload [15,21].

The NYHA functional class and CCS class significantly improved at follow-up [5,10,14].

Freedom from operation of 97% at a mid-term follow-up was comparable to 90-97% reported in previous studies [8,9,15,17,18].

Limitations of the Study

The power of the statistical analysis in detecting the risk factors of a worse mid-term prognosis could have been influenced by the fairly small analyzed sample of patients.

Conclusions

Ischemic etiology, compared to degenerative etiology of mitral regurgitation, represents an independent predictor of a worse outcome in the medium term. Left ventricular dysfunction is a condition more frequently associated with ischemic mitral regurgitation, and "per se" is recognized as a risk factor. An early surgical indication to prevent left ventricular dysfunction may be important to improve the short- and mid-term outcome.

Conflict of interest statement

The authors do not declare any conflict of interest or financial support in this study.

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DOI:10.5455/aces.20120412025840