Grand Rounds

Current Management of Ischemic Mitral Regurgitation

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Abstract

Severe coronary artery disease and myocardial infarction can be complicated by ischemic mitral regurgitation (IMR). IMR results from left ventricular remodeling after myocardial infarction and in rare instances can accompany acute ischemia. A better understanding of the pathophysiology of IMR and increased awareness of its negative impact on long-term survival explain the recent rise in the number of patients referred for surgical correction of IMR at the time of coronary revascularization. The most common mechanism of IMR is Carpentier's type IIIb dysfunction, for which an undersized remodeling annuloplasty is the treatment of choice. In this article we define ischemic mitral regurgitation and review its pathophysiology, clinical presentations, diagnosis, indication for surgery, and management.
Key Words: Mitral regurgitation, ischemic cardiomyopathy, coronary artery disease, mitral valve surgery, coronary artery bypass grafting.

Introduction

SEVERE CORONARY ARTERY DISEASE and myocardial infarction (MI) can be complicated by mitral regurgitation (MR). Commonly referred to as "ischemic MR" (IMR), in most instances it results from either left ventricular remodeling following myocardial infarction, or less frequently, from acute ischemia. This entity should be distinguished from ischemic cardiomyopathy and associated MR due to other underlying etiologies such as degenerative mitral valve disease, rheumatic disease, and endocarditis. For several years, because of a lack of understanding of the precise pathophysiology of IMR, most of the medical and surgical literature has failed to distinguish between these different clinical entities. Until recently, most clinical series have included heterogeneous groups of patients and produced contradictory results which have led to unclear conclusions and guidelines with respect to medical and surgical management of these patients. More recently, laboratory research and clinical outcome analysis have contributed significantly to improving our understanding of this complex disease and its medical and surgical management. This improved understanding of the pathophysiology of ischemic MR and the increased awareness of its negative impact on long-term survival explain the recent rise in the number of patients referred for surgical correction of IMR at the time of coronary revascularization. In this article we will review IMR with regard to its pathophysiology, clinical presentations, diagnosis, indication for surgery, and management.

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Definition of IMR and Carpentier's Functional Classification of MR

The understanding of mitral valve pathology is facilitated by the use of the "pathophysiological triad" first described by Carpentier (1). This triad is composed of etiology (cause of the disease), valve lesions (resulting from the disease) and

valve dysfunction (resulting from the lesions). These distinctions are relevant because long-term prognosis depends on etiology, whereas treatment strategy and surgical techniques depend on valve dysfunctions and lesions, respectively (2). This classification is based on the opening and closing motions of the mitral leaflets (Fig. 1). Patients with type I dysfunction have normal leaflet motion, and MR is the result of annular dilatation. There is increased leaflet motion in patients with type II dysfunction, with the free edge of at least one of the leaflets overriding the plane of the annulus during systole (leaflet prolapse). The most common lesions responsible for type II dysfunction are chordal elongation or rupture, and papillary muscle elongation or rupture. Finally, type III dysfunction results from restricted leaflet motion, with the free margins of portions of one or both leaflets pulled below the plane of the annulus, into the left ventricle, thereby reducing leaflet mobility and coaptation during systole and leading to MR. Patients with type IIIa dysfunction have restricted leaflet motion during both diastole and systole. The most common lesions are leaflet thickening/retraction, chordal thickening/shortening or fusion, and commissural fusion. In type IIIb dysfunction, there is restricted leaflet motion during systole. Left ventricular dysfunction and dilatation with apical papillary muscle displacement causes this type of valve dysfunction.

Ischemic MR can result from type I, II or IIIb dysfunction (Table). Type I dysfunction with annular dilatation occurs in basal MI. Type II dysfunction results from papillary muscle rupture (3–5). Carpentier's type IIIb dysfunction is the most common form of ischemic MR (6–11). A set of criteria is suggested in order to precisely define type IIIb ischemic MR (12): significant symptomatic multi-vessel coronary artery disease, with or without documented prior MI, *and* at least grade 2+ MR, documented on a preoperative echocardiogram or ventriculogram. In addition, the leaflet structure should be normal, there should be no leaflet prolapse (type II), in absence of any mitral stenosis (6).

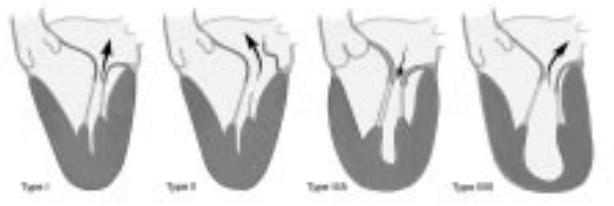

Fig. 1. Carpentier's functional classification. Type I: normal leaflet motion. Type II: increased leaflet motion (leaflet prolapse). Type III: restricted leaflet motion: A: during diastole and systole, B: during systole only.

The arrow shows the direction of the jet in types I, II and IIIB. It shows the association of some degrees of mitral stenosis in type IIIA.

TABLE 1
Pathophysiological Triad in Ischemic MR

Dysfunction	Lesions	Chronic / Acute
Type I	Annular dilatation	Chronic
Type II	Papillary muscle rupture	Acute
	Chordal rupture	Acute
	Papillary muscle elongation	Chronic
Type IIIb	Papillary muscle displacement	Acute or chronic
	Leaflet tethering	

Type IIIb Ischemic MR

Pathophysiology

Normal mitral valve function involves a complex three-dimensional interaction between the leaflets, annulus, subvalvular apparatus and left ventricular wall. Several anatomic and pathophysiological changes are associated with the pathogenesis of ischemic MR. They include ventricular changes (wall motion abnormalities, dilatation with increased sphericity), sub-valvular changes (papillary muscle infarction, displacement or tethering) and annular changes (distortion, dilatation). The initiating insult in IMR is ventricular, specifically left ventricular remodeling following myocardial ischemia or infarction. This remodeling converts the shape of the left ventricle from ellipsoidal to spherical (increase in sphericity index), which may subsequently lead to regional annular and subvalvular distortion and ultimately to poor leaflet coaptation.

Papillary muscle displacement plays a critical role in the pathophysiology of IMR. The tethering distance has been correlated to the severity of IMR (13–15) . Papillary muscle tethering leads to "apical tenting" of the leaflets (restriction of the motion of the free margins of the leaflets), which prevents them from rising to the plane of the annulus to coapt with one another. This has been well documented using quantitative echocardiographic methods in a clinical setting (12). Tethering on the secondary chordae can result in a "sea-gull" deformation of the body of the leaflet, further impairing coaptation. Finally, annular dilatation is a common finding in clinical cases of chronic IMR. However, the degree of dilatation can vary and does not necessarily correlate with the degree of MR. The

above changes in left ventricular geometry, papillary muscle position and annular dimensions interact to produce poor leaflet coaptation during systole, which is the final common pathway for Type IIIb ischemic MR.

Clinical Presentation

Type IIIb IMR can occur in an acute or chronic setting. "Acute post-infarction ischemic MR," without papillary muscle rupture, can be documented for many patients by physical examination, ventriculography or echocardiography. Moderate-to-severe MR is present in up to 13% of these patients (16, 17) . Acute severe type IIIb ischemic MR often presents with a sudden onset of shortness of breath and/or chest pain. In most patients this incident may be preceded by an acute episode of MI; however, in others (particularly diabetic patients) it may be silent. A minority of patients will present with symptoms of heart failure and/or low cardiac output.

Although the MR will resolve over time in some patients, it will persist in others leading to "chronic post-infarction ischemic" MR. In some patients, chronic IMR will first appear up to six weeks after the MI, as the infarcted left ventricle remodels. Risk factors for post-infarction IMR include advanced age, female gender, prior acute MI, large infarct size, recurrent ischemia, multi-vessel coronary artery disease and congestive heart failure (18). In patients with chronic IMR, two clinical scenarios are commonly encountered. Patients may present with moderate-to-severe MR, symptoms of congestive heart failure or worsening left ventricular function, and they are referred primarily for mitral valve surgery. The preoperative coronary angiogram shows significant multi-vessel coronary artery disease that may or may not have been symptomatic. These patients often have clear evidence of prior MI and at least moderate left ventricular dysfunction. Other patients present with symptomatic multi-vessel coronary artery disease and are referred for coronary artery bypass grafting (CABG) with a varying degree of MR on preoperative ventriculography or echocardiography. Acute coronary syndromes, or chronic stable angina is often the dominant presenting symptom in these patients. In addition they may present with symptoms of shortness of breath and/or congestive heart failure.

Diagnosis

Electrocardiogram. Patients often present with changes on their electrocardiogram (ECG). However, changes in favor of acute or remote MI are not observed in all patients (only half present with a diagnosis of acute MI, and in most series of chronic ischemic MR, a prior MI is noted in > 80% of patients). When the ECG changes become diagnostic, inferior wall infarctions are more common than anterior or lateral infarcts. Most patients are in sinus rhythm; however, following atrial enlargement, p-wave abnormalities and atrial fibrillation may occur in the chronic setting. Conduction abnormalities are uncommon in these patients.

Chest X-ray. In the acute setting, the chest X-ray may show evidence of pulmonary interstitial edema. With disease progression, enlargement of the cardiac silhouette (left atrial enlargement and ventricular dilatation) is a common finding.

2D-echocardiography/Doppler. This is an essential tool in determining the mechanism and severity of MR (19), and of left ventricular wall motion abnormalities and function. The severity of MR can be determined by semi-quantitative measurements using jet geometry and the area in multiple views. The severity of mitral regurgitation is graded on a scale from 1+ to 4+ (1+ trace, 2+ mild, 3+ moderate and 4+ severe MR with flow reversal in the pulmonary veins). More recently, quantitative Doppler methods have been developed allowing quantitative grading of MR (7, 20). This quantitative grading is based on the calculation of regurgitant volume and effective regurgitant orifice.

Transesophageal echocardiography (TEE). TEE can be used to determine the mechanism of MR; however, several studies have shown that TEE downgrades the severity of MR in patients with type I or IIIb dysfunction. The mechanism underlying this phenomenon is almost certainly the unloading effect of general anesthesia, which results in arterial and venous dilatation, decreasing afterload and preload, respectively (6, 21).

Cardiac MRI. MRI is a diagnostic tool that provides a precise definition of the mechanisms and severity of MR, as well as left ventricular function and viability.

Cardiac catheterization. Cardiac catheterization should be performed in all patients, in order to determine the extent and severity of coronary artery disease. Left ventriculography is useful in assessing left ventricular function, and segmental wall motion in selected patients.

Negative Impact of MR in Different Clinical Scenarios

There is a growing body of evidence on the negative impact of IMR on medium-term sur-

vival in patients with coronary artery disease. The poor prognosis of patients with ischemic MR has been documented in a variety of clinical settings (e.g., after MI, percutaneous transluminal coronary angioplasty [PTCA], and CABG) with three-year survival rates ranging from 50–75%, all depending on the severity of MR and other patient characteristics.

MR after Myocardial Infarction

Several studies have documented the strong impact of IMR on early and late survival after acute MI (16–18, 22, 23). Lamas et al. (22) studied 727 patients with acute MI from the SAVE trial (survival and ventricular enlargement). In this series, left ventriculograms were used to confirm MR after MI. The presence of MR was related to the risk of developing a cardiovascular event during 3.5 years of follow-up. MR was present in 141 patients (19.4%) (Fig. 2a). Even with similar ejection fractions, patients with MR had larger end-systolic and end-diastolic volumes and more spherical ventricles than patients without MR. They were also more likely to experience cardiovascular mortality (29% vs. 12%, $p<0.001$), severe heart failure (24% vs. 16%, $p=0.0153$), and the combined end-point of cardiovascular mortality, severe heart failure, or recurrent MI (47% vs. 29%, $p<0.001$). In this study, even mild MR at the time of presentation was an independent predictor of mortality (relative risk [RR] 2.0) (4). In another series, Lehman et al. (24) did a sub-group analysis of 206 patients in the TIMI trial (thrombolysis in MI). In addition, in this trial, patients underwent a left ventriculogram within 7 hours of onset of their MI, and MR was present in 13%. In this study also MR was shown to predict cardiovascular mortality by both univariate (RR 12.2, $p<0.0001$) and multivariate analyses (RR 7.5, $p=0.0008$).

Finally, Grigioni et al. (7) conducted a study of 303 patients with a q-wave MI who underwent transthoracic echocardiography. As opposed to the 2 previous series, the authors selected patients at distance from the initial MI (> 16 days). Two groups were matched and followed. At 5 years, overall mortality was 62% for patients with IMR, compared to 50% in patients without IMR (Fig. 2b).

MR after Percutaneous Coronary Intervention

Ellis et al. (25) studied survival in patients with coronary artery disease and MR who underwent percutaneous coronary intervention (PCI). Patients were classified into different groups by MR grade (by quantitative echo). They differed significantly in regard to demographics in all groups. The three-year survival ranged from 46–76% depending on the severity of MR. This data revealed that isolated PCI without addressing MR dramatically decreases survival at 3 years (Fig. 2c).

MR after Coronary Artery Bypass Grafting

Two large studies from the mid-1980s suggested that preoperative MR is an independent risk factor for late death in patients undergoing CABG. Hickey et al. (26) found that increasing MR severity had a progressively negative impact on survival, regardless of the treatment. Adler et al. (27) reported similar results from more than 2,000 patients undergoing primary CABG. Uncorrected MR was an independent risk factor for late death, with a relative risk of 1.5 for each grade of MR. A recent study by Harris et al. (28) comparing CABG with and without mitral valve surgery in patients with 2–3+ MR showed 5-year survival rates of 60% and improved survival in NYHA Class III–IV patients who underwent concomitant mitral valve surgery.

Surgical Indications

Moderate-to-Severe Ischemic MR

As previously mentioned, most patients presenting with moderate-to-severe MR have symptoms of congestive heart failure or worsening left ventricular function. In addition they often have severe 3-vessel coronary artery disease. Medical management of these patients is associated with poor clinical outcomes. It is well established that these patients should undergo combined mitral valve surgery and myocardial revascularization, provided that the operative mortality remains acceptable (29).

Mild-to-Moderate Ischemic MR

In this clinical scenario, patients are often referred for myocardial revascularization secondary to symptomatic coronary artery disease. The preoperative finding of mild-to-moderate type IIIb ischemic MR is often incidental.

Three recent studies have suggested that CABG alone does not completely correct IMR. Czer et al. (30) used intraoperative TEE to compare 25 patients who underwent CABG alone with

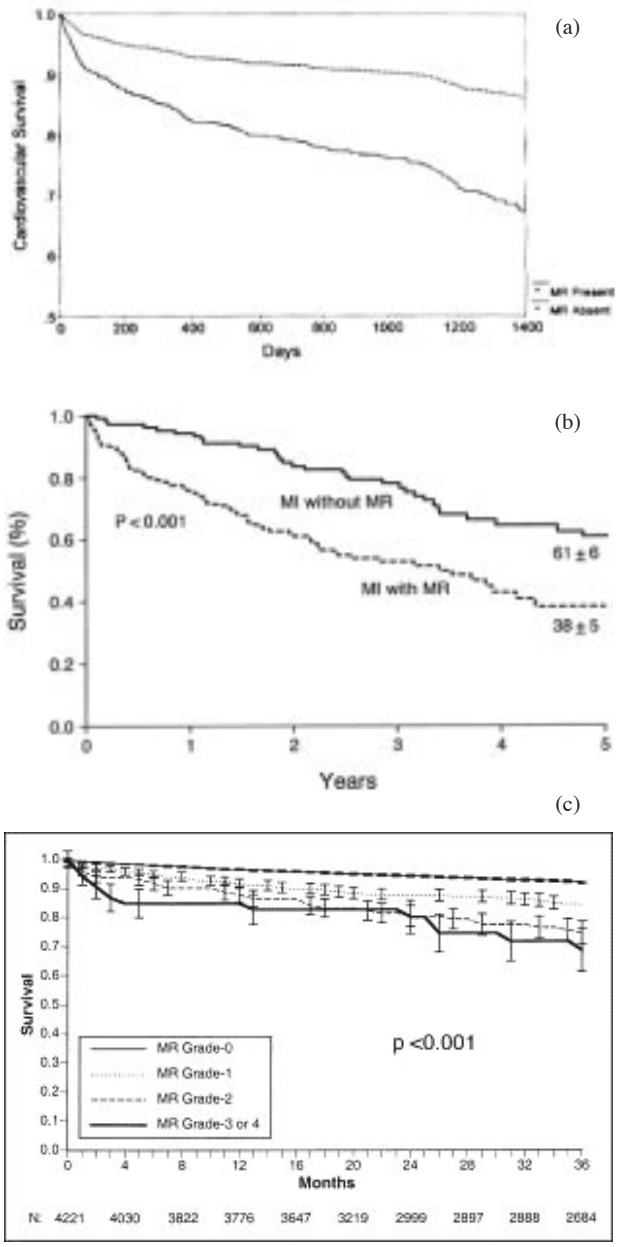

Fig 2: (a) Survival after acute myocardial infarction with early MR. Copied with permission from Lamas GA, Mitchell GF, Flaker GC, et al. Clinical significance of mitral regurgitation after acute myocardial infarction. Survival and Ventricular Enlargement Investigators. Circulation 1997; 96(3):827–833 (22). **(b)** Survival after acute myocardial infarction—MR assessed late after MI (>16 days). Copied with permission from Grigioni F, Enriquez-Sarano M, Zehr KJ, et al. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. Circulation 2001; 103:1759–1764 (7). **(c)** Survival after PCI in patients with MR. Copied with permission from Ellis SG, Whitlow PL, Raymond RE, Schneider JP. Impact of mitral regurgitation on long-term survival after percutaneous coronary intervention. Am J Cardiol 2002; 89:315–318 (25).

24 patients who also underwent suture annuloplasty. In the CABG-alone group there was no change in the annular diameter, leaflet-to-annulus ratio or mean MR grade. This study was also limited by the fact that most patients had mild or trace MR. In addition, the hazards of interpreting MR severity by TEE without controlling loading conditions have been described above. Ryden et al. (31) reviewed 89 patients with 2+ MR undergoing CABG alone and reported that the MR was unchanged or worse in 38%. A study from Aklog et al. (6) from Brigham and Women's Hospital also concluded that CABG alone was not the optimal therapy for many patients with moderate IMR. In this study, 136 patients with moderate IMR underwent CABG alone. Among the 68 patients who underwent early postoperative transthoracic echocardiography, 40% showed no improvement and were left with moderate or severe (3–4+) residual MR. Approximately 50% of the patients had some improvement and were left with mild (2+) residual MR. Only a few remaining patients (< 10%), had significant improvement, with no more than trace (0–1+) residual MR. Thus, current literature seems to suggest that although CABG alone can decrease MR severity, especially in patients with mild IMR and poor left ventricular function, it has an inconsistent and relatively weak impact on moderate IMR, leaving many patients with 2+ or greater residual MR (32). Prifti et al. reviewed 99 patients with 2–3+ IMR and ejection fractions (EF) less than 30%, split evenly between CABG alone and CABG/mitral valve surgery (nearly all repairs) (33). Although the groups were comparable with regard to preoperative characteristic, concomitant mitral valve surgery led to improved EF, decreased left ventricular dimensions and improved three-year survival.

A few studies have examined whether CABG alone corrects IMR in a variety of settings. Christenson et al. reviewed 56 patients with severe left ventricular dysfunction (EF ≤ 25%) and varying degrees of MR by preoperative echocardiography who underwent CABG alone (34). They observed that 93% of patients had no more than trace (0–1+) MR and the remaining patients had mild (2+) MR on postoperative echocardiography. They concluded that "moderate co-existing MR seems to normalize after myocardial revascularization and should not be surgically corrected therefore at the primary operation." This study, however, has several limitations which make it difficult to interpret and do not justify this broad recommendation. Most important, only 7 patients (13%) in this study had moderate (3+) preoperative MR and more than 40% had trace (1+) MR. A similar study by Tolis et al. evaluated 49 patients with EF < 30% and "mild-to-moderate" MR who underwent CABG alone

(35). Mean MR grade decreased from 1.7 to 0.5, and the authors recommend CABG alone for this subgroup. These conclusions are limited by the fact that less than 10% of patients had 3+ MR and 40% had 1+.

Patients with mild-to-moderate IMR (2–3+) should undergo concomitant mitral valve repair at the same time as myocardial revascularization, unless preoperative risk factors suggest that the additional operative morbidity and mortality would be prohibitive (i.e., extensive mitral annular calcification, or strong indication for off-pump CABG in patients with heavily diseased aortas).

Surgical Management

Perioperative Considerations

Standard techniques of monitoring (e.g., arterial line, central venous access, Foley catheter) are used in patients undergoing combined mitral valve repair and CABG. A Swan-Ganz catheter should be inserted in every patient. A transesophageal echocardiogram (TEE) should be performed (36). TEE is a key element for determining the mechanism of MR, assessing LV function and quality of repair, and de-airing of the cardiac cavities at the completion of the procedure. Because afterload reduction secondary to general anesthesia downgrades the degree of MR, TEE is not used to assess MR severity. An epiaortic scan of the ascending aorta is recommended, to rule out the presence of atherosclerotic lesion prior to arterial cannulation.

Surgical Approaches, Cardiopulmonary Bypass, and Myocardial Protection

Median sternotomy is the surgical approach of choice for patients undergoing combined mitral valve repair and CABG, and should be used in the reoperative setting as well. However, in rarer cases, a right anterolateral thoracotomy may be used for selected patients referred for isolated ischemic MR with prior CABG and patent grafts, particularly internal thoracic arteries. Cardiopulmonary bypass is instituted between the ascending thoracic aorta and both venae cavae. Intermittent antegrade, or combined antegrade and retrograde cardioplegia with high-potassium-content cold blood, is used for myocardial protection. Further myocardial protection can be obtained by moderate systemic hypothermia (between 28 and 30°C) and local hypothermia with topical ice. Careful de-airing at the end of the procedure is essential.

Mitral Valve Repair

Following completion of coronary bypass grafting, the perfect exposure of the mitral valve is essential before undertaking mitral valve repair. The most commonly used approach is the interatrial approach through Sondergaard's groove. Mitral valve repair is the procedure of choice for correction of type IIIb ischemic mitral regurgitation.

The goals of valve repair include preserving leaflet mobility and restoring a large surface of coaptation by reducing the septo-lateral dimension with a remodeling annuloplasty. The valvular apparatus is examined with a nerve hook in order to assess the precise mechanism of MR (37). As previously mentioned, it is posterior leaflet restriction due to posterior papillary muscle displacement, affecting P2 and P3 with associated annular dilatation, which is most commonly present (Fig. 3).

Undersized Remodeling Ring Annuloplasty for Type IIIb Ischemic MR

Remodeling annuloplasty using a prosthetic undersized ring is the technique of choice in type IIIb dysfunction (29). Once sutures are placed around the annulus, standard ring sizers are used to

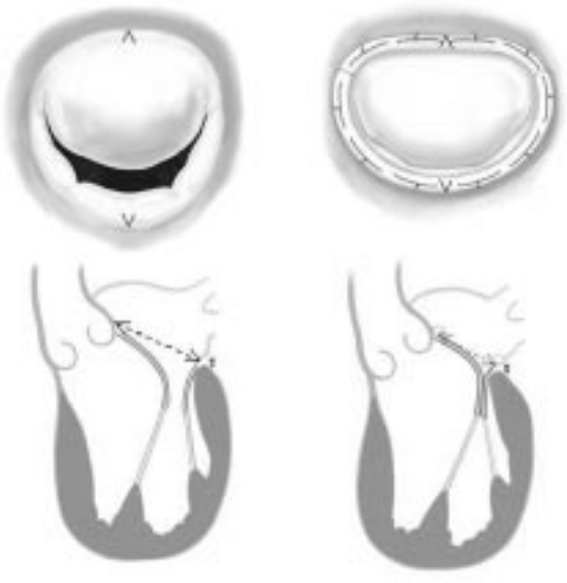

Fig. 3. Schematic diagram of undersized remodeling annuloplasty. **Left:** In patients with type IIIb MR there is a significant restriction of the posterior leaflet and an associated annular dilatation (AP diameter > transverse diameter). **Right:** Undersized remodeling prosthetic annuloplasty restores a large surface of coaptation with the physiological ratio of the mitral annulus, while preserving maximum orifice area.

select the appropriate ring, then the ring is selected and the interrupted sutures are passed through it. When completed, a saline test is used to confirm the line of coaptation along the margin of the leaflets. Nearly the entire orifice is occupied by the anterior leaflet, allowing the entire restricted posterior leaflet to increase the surface of coaptation (Fig. 4).

Surgical Results

Operative Mortality

Concomitant mitral valve surgery/CABG has a mortality rate of over 10%. Several authors have demonstrated that early and late outcomes of combined mitral valve and CABG depend strongly on the underlying etiology, to the extent that it can be determined in retrospective analyses, with significantly worse outcome in ischemic disease (8, 9, 38). Although the reported operative mortality for mitral valve replacement at the time of CABG has remained relatively high (4, 8, 39–41), the outcomes for mitral valve repair appear to be improving over time (4, 8, 40, 41), with most recent series reporting operative mortalities well under 10%. For 2002, the STS database reported an 8% operative mortality for mitral valve repair/CABG (down from 12% in 1993) and 11.5% mortality for mitral valve replacement/CABG (down from 17% in 1993) (42). Filsoufi et al. (43) reviewed early outcomes following CABG and mitral annuloplasty for moderate (3+) IMR and found that operative mortality had decreased from 14% to 3.7% during the 1990s (29). The latter rate was not dramatically different from the 2.9% mortality reported previously in an unmatched but contemporaneous group of patients undergoing CABG alone for moderate IMR (6).

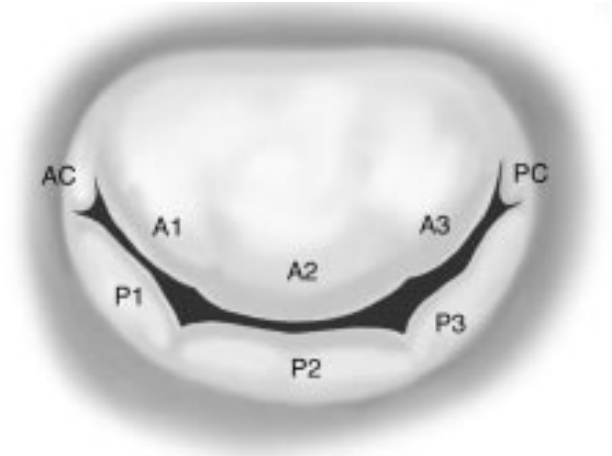

Fig. 4. Segmental description of the valvular tissue. The mitral valve is divided into 8 segments: anterolateral and posteromedial commissures (AC, PC), and posterior and anterior leaflets, which are divided into P1, P2, P3 and A1, A2, A3 segments respectively.

Late Outcomes

Multiple reports over the past twenty years suggest that late survival in patients with IMR undergoing mitral valve surgery is suboptimal and significantly worse than in those with degenerative or rheumatic mitral valve disease (12, 28, 30, 40, 43–45). Medium-term (3–5 year) survival in these studies ranged from 50–80%, depending on the risk profile. Two recent studies have sought to directly compare outcomes following CABG alone with those following CABG/mitral valve surgery.

Prifti et al. reviewed 99 patients with 2–3+ ischemic MR and EF < 30, split evenly between CABG alone and CABG/mitral valve surgery (nearly all repairs) (33). In this series, concomitant mitral valve surgery led to improved ejection fraction, decreased left ventricular dimensions and improved three-year survival. Harris et al. (28) reviewed 196 patients with 2–3+ IMR and an average ejection fraction of 39% (142 CABG alone, 44 with concomitant mitral valve surgeries—mostly repairs). Those undergoing mitral valve surgery had a higher operative mortality (21% vs. 9%), but overall long-term survival was no different. In the subgroup of patients with NYHA Class III–IV heart failure, however, concomitant mitral valve surgery provided a significant survival advantage. An important secondary finding in this study was an approximately 50% incidence of 2+ or greater residual MR in patients undergoing mitral valve repair, a result which the authors acknowledge is suboptimal. Di Donato et al. (32) reviewed 60 patients with moderate IMR undergoing CABG alone (n=30) and CABG/annuloplasty (n=30) from 1998–2001. All patients underwent follow-up echocardiography at 12 and 36 months postoperative. At follow-up, it appeared that the CABG/annuloplasty patients had a lower NYHA classification, fewer signs and symptoms of congestive heart failure, and better echocardiographic parameters (smaller LV volume, increased ejection fraction, and lower pulmonary artery pressures). In this series, the preoperative annular size was seen to be a risk factor for postoperative heart failure events. The authors conclude that the combined approach for patients with moderate MR leads to a better clinical status and better hemodynamic profile.

Overall, these data suggest that concomitant mitral valve surgery might improve late outcomes for patients with IMR, especially for those with

heart failure, and if it can be performed with little additional operative risk and a low incidence of residual MR.

Residual Mitral Regurgitation

Although the routine use of a remodeling annuloplasty and more aggressive downsizing will probably decrease the incidence of residual MR after CABG/annuloplasty, there is a small group of patients for whom a classical undersized remodeling annuloplasty may not sufficiently correct MR.

Aklog et al. (6) recently reported on 288 patients with Carpentier type I or IIIB IMR who underwent CABG/annuloplasty. A complete remodeling, semi-rigid ring (Carpentier-Edwards Physio, Edwards Lifescience, Irvine, CA) was used in 92 patients and a partial, restrictive, flexible band (Cosgrove-Edwards, Edwards Lifescience, Irvine, CA) in 196. Patients who received a Carpentier ring had less residual MR ≥ 2+ (14% vs. 21%, p=0.10). The mechanism of residual MR was impaired leaflet coaptation as a result of isolated annular dilatation (type I) and/or leaflet restriction (type IIIb). One possible explanation for residual MR after CABG/annuloplasty is inadequate downsizing. The anteroposterior dimension must be aggressively reduced to bring the restricted posterior leaflet close enough to the anterior leaflet to allow adequate coaptation. Inadequate downsizing can lead to inadequate coaptation and residual MR.

Furthermore, a recent clinical study from the Cleveland Clinic has provided anatomical and morphological descriptions of type IIIb IMR, which may also be a potential explanation for residual MR after undersized remodeling annuloplasty. In this study, three-dimensional echocardiography was used to further refine analysis of leaflet restriction (type IIIb) in ischemic vs. dilated cardiomyopathy (46). The authors observed a significant difference in mitral valve deformation between the two patient groups. The pattern of mitral valve deformation from the posteromedial to the anterolateral commissure was asymmetrical in the ischemic group, while it was symmetrical in patients with dilated cardiomyopathy. These differences in mitral valve geometry underscore the fact that in patients with ischemic cardiomyopathy, the P2 and P3 segments are the most restricted segments, causing an asymmetrical mitral orifice. These asymmetrical morphological changes may explain why the implantation of a symmetrical, undersized prosthetic ring insufficiently reduces the septo-lateral dimension in some patients with type IIIb IMR, and may be the main cause of residual MR after valve repair (6, 9, 29).

New Developments: Carpentier-McCarthy-Adams Etlogix Ring

In an attempt to address the above-mentioned issues (inadequate undersizing, marked P2–P3 restriction relative to P1) and to potentially reduce the incidence of residual MR, a new remodeling annuloplasty ring was developed in collaboration with the Department of Cardiothoracic Surgery at Mount Sinai Medical Center, New York. This new prosthetic ring combines the principles of undersizing, while addressing the specific asymmetric deformation observed in type IIIb IMR (Fig. 5). This new design leads to increased leaflet coaptation due to the reduced anteroposterior (AP) dimension. In addition, the asymmetric 3-D design with reduced P2–P3 curvature allows for a better accommodation of tethered P2–P3 segments (Fig. 5).

The Carpentier-McCarthy-Adams Etlogix ring was first implanted at Mount Sinai Medical Center in December 2003, shortly after Food and Drug Administration (FDA) approval. Fifteen patients (14 male and 1 female, mean age of 66 years) presented with greater than 3+ MR. The mean ejection fraction was 31% (10–57), and all patients were in NYHA Classes III and IV. They underwent successful mitral valve repair with this new prosthetic ring (mean size 28 mm). Concomitant procedures included CABG, left-atrial maze, tricuspid valve repair and patent foramen ovale closure. Expected operative mortality calculated by Euroscore for this cohort was 19% (4–47). However, the ob-

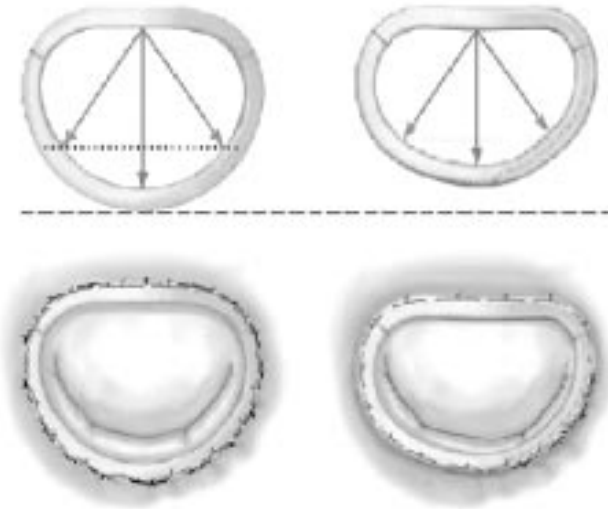

Fig. 5. Carpentier-McCarthy-Adams Etlogix Ring (Edwards Lifescience, Irvine, CA). This image shows a symmetrical Carpentier-Edwards Physio Ring (on the left), and the new asymmetrical Carpentier-McCarthy-Adams Ring (on the right). The new ring is already undersized, and has a 14% reduction in the postero-medial dimension (P2–P3 segments).

served operative mortality was 0%. Pre-discharge transthoracic echocardiography for all patients showed a significant reduction in mean MR grade (from 3.5 to 0.6, p>0.05).

Type II Ischemic MR

In this functional type of MR, the motion of one or two leaflets is excessive and the free edge of the leaflet overrides the plane of the orifice during systole. Leaflet prolapse results from partial or complete rupture of one papillary muscle or from papillary muscle elongation or, more rarely, from chordal rupture.

Papillary muscle rupture is a rare mechanical complication of MI; without early diagnosis and expeditious medical and surgical management, it has a high mortality rate. This complication occurs in 1–5% of patients who die following MI (47). Papillary muscle rupture can occur during the acute phase of MI; however, most patients present within 2–7 days after MI (48). Complete papillary muscle rupture occurs in 30% of patients and results in bi-leaflet prolapse with severe mitral regurgitation. However, partial rupture of one papillary muscle involving one or more heads remains the most common lesion in patients with type II IMR. Myocardial infarction often involves a limited area of the myocardium, which may explain the relatively well-preserved left ventricular function in most patients (3). Papillary muscle elongation results from fibrotic transformation of papillary muscle following MI. The elongation of papillary muscle causes leaflet prolapse, resulting in MR. Most patients present with chronic IMR and do not require emergency surgical treatment.

Patients with papillary muscle rupture present with sudden development of congestive heart failure and cardiogenic shock. This rapid deterioration of the clinical condition in previously stable patients should suggest the diagnosis of a mechanical complication of MI. Clinically, it remains very difficult to distinguish between these two entities, despite differences in the characteristics of the murmurs. Another common differential diagnosis is extensive MI with cardiogenic shock associated with varying degrees of ischemic mitral regurgitation without papillary muscle rupture. Other preoperative examinations, including two-dimensional transthoracic echocardiography, TEE with Doppler and cardiac catheterization, are crucial for making the accurate diagnosis of IMR due to papillary muscle rupture and evaluating left ventricular function and the severity of coronary artery disease. The principal goal of preoperative medical management is hemodynamic stabilization (preservation of cardiac output and arterial pressure to maintain peripheral organ perfusion as well as the preservation of coronary blood flow). This is best achieved with the use of inotropic agents and prompt insertion of intra-aortic balloon pump (3). However, this stabilization phase should not delay surgical intervention in these critically ill patients. Based on the preoperative investigations, the surgical procedure should combine the correction of mitral regurgitation and myocardial revascularization.

Chordal-sparing mitral valve replacement remains the procedure of choice for most patients. This technique preserves postoperative left ventricular function, contributing to improved long-term survival of these patients (49–52). However, in selected patients, mitral valve repair with papillary muscle shortening or papillary muscle reimplantation can be done using Carpentier's techniques of valve repair (53).

Operative mortality has traditionally been high. In a recent publication, Tavakoli et al. (3) reported a consecutive series of 21 patients who underwent mitral valve surgery (replacement, n=19; repair, n=2) and CABG (52% were urgent/emergent). Operative mortality was 19%; the principal postoperative complications were stroke (6%) and renal failure requiring hemodialysis (18%). In this series actuarial survival at 1, 5, and 10 years was 81, 68 and 56%, respectively. The survival seen in this series compares well to that in a similar study from the Cleveland clinic (4), which demonstrates a survival rate of 55% after 7 years. Even though early mortality is high in the acute setting, aggressive surgical treatment does improve long-term outcome, as this allows the correction of an acute hemodynamic compromise (54–56).

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