

Moderate Ischemic Mitral Regurgitation: Repair or no Intervention Concomitant with CABG?

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Ischemic Mitral Regurgitation (IMR) is a disease of myocardium.¹ While some authors believe that myocardial infarction (MI) always precedes IMR,¹ others believe that IMR is caused by coronary artery disease (CAD) and not necessarily MI.² In IMR, the leaflets and subvalvular apparatus are by definition normal; the disease must be distinguished from MR associated with CAD, in which no cause and effect relationship exists.¹ About half of the patients with ischemic heart disease and chronic mitral regurgitation (MR) have coexisting MR caused by rheumatic fever, myxomatous degenerative diseases, or other conditions.

IMR is divided into acute (within one week³ or 16 days after acute myocardial infarction [AMI]⁴) and chronic. Acute IMR may occur as a consequence of papillary muscle (PM) rupture or may be produced without PM rupture. PM rupture may be due to the complete rupture of PM, which occurs within one week after AMI or due to the rupture of one head from several heads of PM, which occurs up to 3 months after AMI.¹

Posterior PM has a single blood supply from the right coronary artery (RCA) or left circumflex artery (LCX) and is most prone to ischemia. Anterior PM has a dual supply from the left anterior descending (LAD) and LCX and is less prone to ischemia.¹ Acute IMR may be begotten by acute ischemia or MI without PM rupture, previously named PM dysfunction. In this setting in animal models, PM in the infarct zone gets 2-4 mm closer to the mitral valve (MV) annulus and the opposite PM gets away from the MV annulus. Consequently, the alteration of the normal geometry between PMs and asynchronous contraction of PMs produces MR.¹ In chronic IMR, factors contributing to incomplete mitral leaflets closure include abnormal leaflet tethering by displaced ischemic papillary muscles (type IIIb valve disease) or a dilated annulus (type I valve disease).⁵⁻⁶ Previously, there was a greater emphasis upon the role of

MV annulus dilation, as the main mechanism, in the causation of MR. There is now, however, a growing awareness of the role of leaflet restriction due to tethering and leaflet tenting in the causation of MR. Some authors believe that systolic mitral valve tenting is the main mechanism of IMR due to apical and posterior papillary muscle displacements and that the annular dilation has only an adjunct role.¹

Some authors believe that annular dilation is present with all the following 3 criteria: 1) Left ventricular (LV) dilation. 2) Annulus/anterior mitral leaflet^{>1.3/1} parasternal long axis. 3) Central MR. Others describe annular dilation in the setting of LV dilation, central MR, and size of the annulus greater than 3 cm. There are also those who maintain that left atrial (LA) dilation is present with annulus dilation. The MV annulus is smaller in late diastole and systole; that is why we measure the mitral annulus in early diastole, and it is between 2-3.4 cm in transthoracic echocardiography (TTE) and 2-3.8 cm in transesophageal echocardiography (TEE). The MV annulus is not planar and is in the shape of a hyperbolic paraboloid; it is more cephalic in midscalopes and more caudal in commissures. The maximum height is about 14±3 mm. In functional MR (FMR), this height is reduced and the distance between the high points increases.⁷

Tethering is divided into symmetrical and asymmetrical types. Symmetrical tethering is due to the apical displacement of both leaflets, whereas asymmetric tethering is caused by the posterior displacement of both leaflets. Symmetrical tethering is associated more frequently with the anterior MI, three-vessel disease (3VD), and LAD lesion and is seen more often in patients with NYHA functional class (FC) III, more LV dilation, and a low ejection fraction (EF).ⁱⁱⁱ

Asymmetrical tethering is accompanied more frequently by inferior MI, RCA lesion, and patients in NYHA FC II.⁴

MV deformation indices and local and global LV deformation indices are believed to be more impaired in symmetrical than

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they are in asymmetrical tethering.⁴

MV deformation indices include: 1) Tenting area: area enclosed between the MV leaflets and the annulus (early systole). 2) MV area: $(D_1 \times D_2 \times \pi/4)$, D_1 : apical 4-chamber, D_2 : apical 2-chamber (late systole). 3) Coaptation height: distance between the MV annulus and the coaptation point (Apical 4-chamber or parasternal long axis), early systole.

Local LV deformation indices include: 1) Posterior tethering (distance between the tip of PMs and the central line that separates the interventricular septum and the other LV segments). 2) lateral tethering (distance between the tip of PMs and a line perpendicular to that central line). 3) Apical tethering (distance between the tip of the posterior PM and the intervalvular fibrosa). 4) PM distances (distance between the tips of PMs). 5) Local wall motion score index (anterior basal and midportion, lateral basal and midportion, posterior basal and midportion, and inferior basal and midportion).

Global LV remodeling indices includes: end diastolic volume (EDV), end systolic volume (ESV), ejection fraction (EF), global wall motion score index (WMSI), and sphericity index ($\frac{\text{diameter}}{\text{length}}$ in apical 4-chamber, late systole).

The main determinant of systolic mitral valve tenting is the local remodeling, whereas the global LV dysfunction and enlargement are not primary causes.⁴

Tenting is described in the anterior mitral leaflet (AML), and on account of the involvement of the secondary chordae, an echocardiographic picture of the tenting of AML is called seagull sign.³ Nesta et al. reported a unique MV leaflet configuration for patients with functional MR: in a long axis view, the leaflets appear concave toward LA as opposed to their normal concavity toward the contracting LV.⁸ They measured the leaflet concavity area between the anterior leaflet and a line connecting its ends and found that patients with leaflet concavity had a significantly greater MR than did those without this finding. In the apical 4-chamber view, the tenting pattern may be subtle and variable because of the high variability of the scanning planes in this view.⁸

Three terms are commonly used to describe IMR. 1) Functional MR (FMR): Many references equate FMR with chronic IMR,³ but some use FMR for a wider range of MR including MR due to dilated cardiomyopathy or hypertrophic obstructive cardiomyopathy.⁷ 2) Transient IMR: It is MR due to reversible ischemia. In this setting, relief of ischemia causes MR to decrease or disappear, rendering a mitral valve procedure unnecessary.¹ A careful evaluation of IMR is, therefore, necessary before surgery. 3) Mitral valve prolapse (MVP) due to ischemia: Does ischemia produce MVP?

Myxomatous degenerative mitral valve is most likely congenital, but it may also be due to ischemia. There was a greater emphasis in the past upon the notion that elongated and unruptured papillary muscles due to MI may give rise to prolapse. The primary chordae, which are attached to the edge of the leaflets, prevent leaflet prolapse. As our knowledge increases about the asynchronous contraction of PMS in acute IMR and the major roles of annular dilation

and restriction of leaflets in chronic IMR, the role of prolapse due to ischemia is losing its importance. Although some surgeons describe leaflet prolapse in ischemic MR, it may be due to already existing prolapse without MR, which together with ischemia produces MR. While pathological studies have shown fibrosis and atrophy of infarcted PMS, none has demonstrated PM or chordal elongation.

Repair of moderate ischemic MR is one of the most challenging problems. Most authors believe that moderate-to-severe MR must be corrected at the time of coronary artery bypass grafting (CABG). Repair of moderate IMR is, however, controversial and several arguments have been made: First, revascularizing ischemic segments will improve regional wall motion and reduce MR.⁹⁻¹¹ Second, some studies suggest that CABG alone does not affect long-term survival or functional status.¹²⁻¹⁶

There is limited information in the literature on the outcome of patients undergoing CABG alone for moderate IMR. Mallidi et al. reported that while late survival was not affected by the presence of mild-to-moderate degrees of MR in patients undergoing CABG, these patients had poorer event-free survival and worse late functional status.¹⁷ Aklog et al. reported that 40% of patients continued to have at least moderate MR (3+ to 4+), 51% improved somewhat to mild (2+) MR, and only 9% had resolution of their MR (0 to 1+) after revascularization alone.¹⁸ Wong and coworkers found more long-term MR but no difference in survival among patients with 3+ IMR who underwent isolated CABG versus CABG plus MV repair.¹⁹ Many surgeons, however, have advocated a more liberal use of MV annuloplasty in patients with moderate MR at the time of CABG.¹⁸ They argue that CABG alone will not correct moderate IMR in many patients, especially those with scarring from MI and those with annular and ventricular dilation.²⁰

The following considerations could help us in the decision-making process about moderate IMR: repair or no intervention:

1) When there is akinetic and scar tissue, especially in the basal and mid portions of LV, MR probably persists after revascularization alone; but when hypokinesia is responsible for MR, we expect MR to reduce after revascularization.

2) In akinesia without scar, there may be viable or non-viable tissue. Dobutamine stress echocardiography (DSE), technetium scan, or PET with defining viability can be of great help. If the segments are viable, there is a better chance of the reduction of MR after revascularization; and if the segments are non-viable, repair may be considered.

3) DSE can help us in another way: if there is a reduction in the MR severity after low-dose DSE, it signifies that with improvement in contractility, MR would reduce. But when the severity of MR has not changed or has even worsened with dobutamine, there would not be any reduction with revascularization alone. As a result, we may consider repair for these types of MR.

4) Another challenging problem in the repair of the



mitral valve is prolapse. When there is MR with persistent wall motion abnormality and MVP, MVP may be due to either ischemia or myxomatous degenerative MV disease. Often with precise echocardiography and looking at the MV leaflets, we can recognize the redundant myxomatous leaflets from the prolapse due to ischemia. If myxomatous degenerative changes are present, they will not be reduced with revascularization alone. Nonetheless, MVP due to ischemia may decrease after revascularization.

5) Do left ventricular diastolic and systolic dimensions have any influence on the persistence of MR after CABG? Although some studies have shown no relation between LV diastolic and systolic dimensions and the resolution of MR after CABG,²¹ it seems reasonable that we use MV repair concomitant with CABG when there is severe LV dilation (diastolic LV dilation > 65mm, systolic LV dilation > 45mm).

6) As regards whether there is any relationship between lower EF and persistence of MR after CABG, some authors have reported a trend for the persistence of MR after CABG in patients with LV systolic dysfunction (52.8% in EF < 50% versus 28.6% in EF ≥ 50%, p = 0.09).²¹ It, therefore, seems reasonable that for more LV systolic dysfunction, we use MV repair.

In conclusion, when approaching a patient with moderate ischemic MR, we must consider many factors such as scar tissue in LV, LV dimensions, and ejection fraction for a final decision on MR; i.e. whether to use CABG alone and no intervention on the mitral valve or to use one of the repair techniques to reduce MR. For a final decision, an agreement between the cardiac surgeon and echocardiographic cardiologist is necessary.

References

- Gorman RC, Gorman III JH, Edmunds H. Ischemic mitral regurgitation. In: Cohn LH, Edmunds LH, eds. *Cardiac Surgery in the Adult*. 2nd ed. New York: McGraw-Hill; 2003. p. 751-769.
- Gillinov AM, Cosgrove DM. Mitral valve repair. In: Cohn LH, Edmunds LH, eds. *Cardiac Surgery in the Adult*. 2nd ed. New York: McGraw-Hill; 2003. p. 933-949.
- Borger MA, Alam A, Murphy PM, Doenst T, David TE. Chronic ischemic MR: repair, replace or rethink. *Ann Thorac Surg* 2006;81:1153-1161.
- Agricola E, Oppizzi M, Maisano F, Schinkel L, Torracca L, Margonato A, Melisurgo G, Alfieri O. Echocardiographic classification of chronic ischemic mitral regurgitation caused by restricted motion according to tethering pattern. *Eur J echo* 2004; 5:326-334.
- Carpentier A, Lessaana A, Ralland J, Belli E, Mihaileanu S, Berbebi AJ. The "physio-ring." An advanced concept in mitral valve annuloplasty. *Ann Thorac Surg* 1999;60:1177-1186.
- Timek TA, Lai DT, Tibayan F, Liang D, Rodriguez F, Daughters GT. Annular versus subvalvular approaches to acute ischemic mitral regurgitation. *Circulation* 2002;106:127-132.
- Valocik G, Kamp O, Visser CA. Three dimensional echocardiography in mitral valve disease. *Eur J echo* 2005;6:443-454.
- Nesta F, Otsuji Y, Handschumacher MD, Messas E, Leavitt M, Carpentier A, Levine RA, Hung J. Leaflet concavity: a rapid visual clue to the presence mechanism of functional mitral regurgitation. *J AM Soc Echocardiogr* 2003;16:1301-1308.
- Christenson JT, Simonet F, Bloch A, Maurice J, Velebit V, Schmuziger M. Should a mild to moderate ischemic mitral valve regurgitation in patients with poor left ventricular function be repaired or not? *J Heart Valve Dis* 1995;4:484-488.
- Ogus NT, Hulusi Us M, Ogus H, Islik O. Coronary artery bypass grafting alone for advanced ischemic left ventricular dysfunction with significant mitral regurgitation: early and midterm outcomes in a small series. *Tex Heart Inst J* 2004;1:143-148.
- Tolis GA, Korkolis DM, Kopf GS, Elefterades JA. Revascularization alone (without mitral valve repair) suffices in patients with advanced ischemic cardiomyopathy and mild-to-moderate mitral regurgitation. *Ann Thorac Surg* 2002;74:1476-1481.
- Balu V, Hershowitz S, Zaki Masud AR, Bhayana JN, Dean DC. Mitral regurgitation in coronary artery disease. *Chest* 1982;81:550-555.
- Duarte IG, Shen Y, MacDonald MJ, Jones EL, Carver JM, Guyton RA. Treatment of moderate mitral regurgitation and coronary disease by coronary bypass alone: late results. *Ann Thorac Surg* 1999;68:426-430.
- Connolly MW, Gelbfish JS, Jacobowitz IJ, Rose DM, Mendelsohn A, Cappabianca PM. Surgical results for mitral regurgitation from coronary artery disease. *J Thorac Cardiovasc Surg* 1986;91:379-388.
- Arcidi JM Jr, Hebel RF, Craver JM, Jones EL, Hatcher CR Jr, Guyton RA. Treatment of moderate mitral regurgitation and coronary disease by coronary bypass alone. *J Thorac Cardiovasc Surg* 1988;95:951-959.
- Rydén T, Bech-Hanssen O, Brandrup-Wognsen G, Nilsson F, Svensson S, Jeppsson A. The importance of grade 2 ischemic mitral regurgitation in coronary artery bypass grafting. *Eur J Cardiothorac Surg* 2001;20:276-281.
- Mallidi HR, Pelletier MP, Lamb J, Desai N, Sever J, Christakis GT. Late outcomes in patients with uncorrected mild to moderate mitral regurgitation at the time of isolated coronary artery bypass grafting. *J thorac cardiovasc Surg* 2004;127:636-644.
- Aklog L, Filsoufi F, Flores KQ, Chen RH, Chon LH, Nathan NS, Byrne JG, Adams DH. Does coronary artery bypass grafting alone correct moderate ischemic mitral regurgitation? *Circulation* 2001;104:168-175.
- Wong DR, Agnihotri AK, Hung JW. Long-term survival after surgical revascularization. *Ann Thorac Surg* 2005;80:570-578.
- Czer LSC, Maurer G, Bolger AF, Derobertis M, Chaux RNA, Matloff JM. Revascularization alone or combined with suture annuloplasty for ischemic mitral regurgitation: evaluation by color Doppler echocardiography. *Tex Heart Inst J* 1996;23:270-278.
- Sadeghian H, Karimi AA, Ahmadi H, Salarifar M, Sadeghian S, Fallah N, Paydari N, Majd M. Moderate mitral regurgitation and coronary disease: treatment with coronary bypass alone. *J Tehran Univ Heart Ctr* 2006;1:17-22.