Ischemic Mitral Regurgitation
Challenges in Qualification

Robert M. Savage, MD, FACC, FACC
Vice-Chair, Department of Cardiothoracic Anesthesia
Head, Sections of Perioperative Echocardiography
Cleveland Clinic Anesthesiology and Heart & Vascular Institutes
Cleveland, Ohio 44195

Objectives

Upon completion of this lecture, the participant will understand:

- Definition of ischemic MR
- Mechanisms & Classification of ischemic MR
- Challenges of Quantifying Ischemic MR
- Accurate Measurement of Ischemic MR
- Assessing Mechanisms of ischemic MR
- Severity Assessment & Outcomes

Definition of Ischemic Mitral Regurgitation (MR)

Ischemic mitral regurgitation (IMR) is defined as MR caused by chronic changes of left ventricular structure & function due to ischemic heart disease. It is not a disease of the mitral valve, but manifestation of remodeling & functional changes of a diseased ventricle. Typically these changes occur ≥ 2 weeks post-myocardial infarction (MI) in the absence of structural MV disease. This definition does not include causes of acute MR (papillary muscle rupture or deformity) classified as complications of myocardial infarction. The incidence of IMR depends on the detection technique, presence degree of revascularization, post-infarct period, and infarct size. Echo studies report a 50% incidence of MR ≤ 1 month of post-MI with 12% having moderate & severe MR. IMR is associated increased incidence of heart failure and death within 5 years. IMR patients with an ERO of ≥ 20 mmHg have double the mortality of those with no MR.

Mechanisms of Ischemic MR

Systolic retrograde flow from the LV to the LA due to inadequate leaflet coaptation and a gradient between the two cavities. The severity of IMR is dynamic throughout systole and varies according to regurgitant orifice area and factors affecting the LV-LA gradient (systolic regional or global dysfunction, diminished LA compliance) in a ventricle damaged by ischemic heart disease. Coaptation is reduced in IMR due to an imbalance between increased tethering forces (papillary muscle displacement, LV dilatation, annular dilatation) and reduced MV closing forces (contractility, LV & papillary dyssynchrony, and annular contraction). The dominant features are apical and posterior displacement of the papillary muscles which are compounded by annular dilatation and dysfunction.

1. Posterior leaflet displacement (asymmetric pattern)- Posterior displacement of the PMVL, associated with inferolateral infarcts, results in posterior displacement of the PMVL with an asymmetric AMVL override & secondary chordal tethering of the AMVL with a sea-gull deformity.
2. Left ventricular dilatation with bileaflet displacement (symmetric pattern)-
Spherical remodeling of the LV results in apical displacement of both leaflets & ventricularization of point of coaptation with symmetric tethering and a central MR jet. Annular dilatation and dysfunction further increase the regurgitant orifice area.

3. **Annular dilatation and loss of systolic saddle shape annulus**
   MV annular area decreases as it reconfigures from a planar to a saddle shaped systole as structure. The area of the MV leaflets is normally > 2x the annular area in systole and delays the development of MR until more advanced stages of ventricular remodeling.

4. **Ventricular and Papillary muscle asynchrony:** LV dysynchrony is a recognized cause of incomplete leaflet coaptation and is accentuated with conduction disturbances. Cardiac resynchronization therapy has reduces the differences in papillary muscle activation times.

5. **Closing forces:** LV contractility generate the closing forces preventing incomplete coaptation. LV dysfunction may inhibits the generation of a closing force sufficient to overcome the effects of apical tethering.

**Classification of Ischemic MR**

Classifications of ischemic MR are based on the symmetry of apical tethering, & degree of annular dilatation or dysfunction. Asymmetric tethering is commonly associated with single vessel disease, inferolateral infarcts, mild-moderate increased tenting areas, inferolateral remodeling, and a MR jet that is posteriorly directed due to an AMVL override. In contrast symmetric tethering is associated with multivessel coronary disease, large anterior or multiple infarcts, greater eccentric spherical remodeling, bileaflet apical tethering, ventricularization of leaflet coaptation, larger tenting areas, and a central jet direction. Annular dilatation increases the potential severity of MR associated with asymmetric or symmetric tethering. With multivessel disease the potential for annular dysfunction (flattening, loss of saddle shape) is increased.

<table>
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<th>Classification of Ischemic Mitral Regurgitation</th>
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<tr>
<td><strong>Leaflet tethering</strong></td>
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<td>PMVL tethered posteriorly</td>
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<td><strong>Ventricular Coaptation Depth</strong></td>
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<td><strong>MR Jet Direction</strong></td>
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**Challenges in Quantifying Ischemic MR**

Ischemic MR is caused by a complicated combination of structural and functional factors that change throughout systole under different loading conditions. Clinical outcomes correspond to quantified degrees of MR and accurate assessment is essential to monitor, direct therapeutic interventions. The use of qualitative or semi-quantitative measurements of MR severity should be abandoned in preference to more quantitative measurements of MR (vena contracta, PISA regurgitant orifice area, and volumetric calculation). The challenges of assessing IMR severity are related to its dynamic nature and inherent difficulties in quantitative methodologies. These are related to the 3-dimensional structure of the regurgitant orifice and variations of the regurgitant flow rate during systole. The systolic flow characteristics of IMR cause inaccuracies in measurements of vena contracta (VC) & Effective Regurgitant Orifice (ERO) by PISA.

**Non-Circular Orifice:** The regurgitant orifice is frequently not circular with multiple jets in >25% of patients with IMR and creates error when using the vena contracta (narrowest diameter of a MR jet regurgitant) or PISA calculation of the ERO which assumes a circular orifice with hemispheric proximal flow convergence. The calculation of ERO Vena contract and regurgiant orifice area by 3D echo is a promising improvement.

**Systolic variation of regurgitant flow rate:** ERO and Regurgitant Volume (RVol) calculations rely on measurements of the diameter of PFC aliasing usually obtained at the time of optimal visualization and based on the following assumptions: 1) circular ERO, 2) hemispheric PFC, 3) PISA radius measured at maximal MR flow, 4) constant systolic ERO area, 5) RVol is based on average systolic regurgitant orifice reflected by ERO. Levine et al discovered the (flow rate) of ischemic-MR is bifid and greatest at the beginning and end of systole (illustration below) and flow is reduced in mid-systole because. This bifid flow rate corresponds with the bifid color M-mode PISA radius and reflects the increase early and late systolic flow rates. Using the largest PISA provides an estimation of the peak ROA and may over-estimate severity of ischemic-MR. Calculating MR RVol is a non-physiologic” calculation using an integrated velocity-time measurement (from entire systolic cycle) coupled with the maximal instantaneous regurgitant orifice area. The volumetric method of calculating MR RVol subtracts the aortic forward stroke volume from the total stroke volume through the mitral annulus. This provides an estimated “mean” ERO throughout systole which is so is independent the assumptions used with PISA ERO & RVol calculations and Vena Contract estimation of ERO.
Using the maximal PISA in early or late systole not coinciding with the mid-systolic peak regurgitant velocity, will underestimate the regurgitant flow. Color m-mode may enable an average PISA radius and provide greater reliability.

**Accurate Measurements of Ischemic MR**
Quantitative methods of measuring MR severity include the Vena Contracta, PISA proximal-flow-convergence calculation of ERO area MR RVol, and PW Doppler volumetric method (MR RVol = MV Stroke Volume – Aortic Stroke Volume). Inherent limitations of the PISA RO may be accommodated by using an PISA radius that reflects the mean throughout MR regurgitant flow and adjustments for a non-hemispheric PFC. Outcome studies validating the importance of ERO were based on the same erroneous assumptions and downplay their clinical significance. However, the time-consuming volumetric calculation of MR RVol and ERO area is the most physiologic method of quantifying IMR. The most reliable calculation of MR RVol & ERO is probably an averaging of the quantitative volumetric Doppler and PISA methods. Performing volumetric measurements accurately improves with experience and consistent comparison with other methods. Moderate IMR has an adverse prognosis making the accurate recognition of moderate lesions important in patient management decisions. This is a time-sensitive challenge in the intraoperative environment that may warrant our consideration.

**Assessing Mechanisms of Ischemic MR**

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<td>Symmetric or asymmetric restriction</td>
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<td>Tenting distance, area, &amp; volume</td>
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Severity Assessment & Outcomes

The quantified degree of MR is associated with increased morbidity and mortality. Survival curves differed markedly between patients with no IMR, moderate MR (ERO area =20 - 30mm²) or severe MR (ERO area > . 30 mm²). The dynamic aspect of IMR, as measured by exercise-induced MR, has prognostic value with a five-fold increase in risk of death in patients with exercise-induced increase of = 13 mm of the ERO area and predicts cardiovascular morbidity. The 20% of patients with improved IMR severity during exercise have better long term prognosis reflecting contractile reserve and reduced distortion of the MV apparatus.

References