Anticipating the Vicious Circle of Postinfarction Mitral Regurgitation Imaging Insights

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Imaging has been key to deciphering the fundamental mechanism of postinfarction ischemic mitral regurgitation (MR) based on 2-dimensional and 3-dimensional echocardiographic visualization of leaflet tethering. Advanced magnetic resonance and radionuclide techniques combined with echocardiography have related this restrictive leaflet dysfunction to the severity and location of myocardial damage and perfusion defects. The subsequent impact of Ischemic MR on heart failure and mortality is well known. Ischemic MR—a dynamic lesion during the cardiac cycle at rest and with exercise—is also a progressive lesion, contributing to a downhill spiral in which MR begets MR through geometric changes that augment leaflet tethering and primary adverse alterations in myocardial and valvular biology. In the Valsartan in Acute Myocardial Infarction (VALIANT) study, progression of MR post infarct primarily related to mitral leaflet tethering, the strongest experimental determinant of altered leaflet force balance and closure, and the progression of MR substantially reduced survival and increased heart failure.

See Article by Kwon

The study by Kwon et al in this issue of Circulation: Cardiovascular Imaging conveys the important message that postinfarction MR is progressive during a median of 7 months in ~30% of the patients studied and that its progression further worsens survival and heart failure beyond the prognosis associated with baseline MR severity. Advanced magnetic resonance analyses indicated univariate relations of MR progression with left ventricular end-systolic volume, baseline MR, myocardial scar (especially inferior), mitral valve tethering, and incomplete surgical or percutaneous revascularization. Multivariate analysis showed progression primarily depended on baseline MR orifice area, total scar, and left ventricular end-systolic volume. This can be reconciled with the results of the VALIANT study because of the strong dependence of baseline MR on mitral valve tethering geometry, which is also related to left ventricular cavity size and scarring as determinants of tethering. Of note, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers were significantly protective, reducing death and heart transplant with a hazard ratio of 0.53 (0.31–0.90).

Future directions can address several questions posed by the results: (1) Echocardiographic follow-up can test for the probable increases in mitral valve tethering associated with MR progression. (2) The authors note that revascularization did not affect survival when ischemic MR progression was considered—perhaps because MR progression itself may relate to the adequacy of revascularization. This key point, and the potential to reverse or stabilize MR with revascularization, needs to be explored in terms of optimizing revascularization strategies and its impact on clinical practice. (3) The role of exercise-induced MR augmentation in predicting MR progression can be explored to potentially identify patients most likely to benefit from interventions. (4) Figure 3 of the article shows an unexpected decrease followed by an increase in MR orifice area over time, especially in patients with moderate-to-severe myocardial scar burden. This raises questions as to whether there are different mechanisms early versus late, including initially beneficial but potentially in the long-term maladaptive mitral valve adaptation or whether progressive left ventricular remodeling can explain the late effect. (5) The relatively small regurgitant orifice area values in part reflect the hemispherical assumption underlying the proximal isovelocity surface area calculation of MR quantification, which will underestimate MR for the elongated orifice in this condition but likely consistently among groups. The MR, however, is more important than the numbers suggest. Supplemental Figure 1 highlights the difficulties of and the need to further refine MR volume quantification because different modalities can yield different results.

Clinical Implications

This article increases the awareness that ischemic MR is a progressive lesion with poor outcome. The ability to identify risk based on cardiac imaging suggests opportunities to improve the selection of patients for early, preventive therapy, and then test for improved prognosis. Imaging plays a key role in this process, and the relation of this dynamic lesion to perfusion merits further exploration.

Sources of Funding

This work is supported in part by grant 07CVD04 of the Leducq Foundation, Paris, France, by the Leducq Transatlantic MITRAL
Network, and by the National Institutes of Health grants R01 HL109506 and HL128099. Additional support was from an American Society of Echocardiography Career Development award and an Erwin-Schrödinger Stipend (FWF Austrian Science Fund).

Disclosures

None.

References


**Keywords:** Editorials • adaptation • echocardiography • ischemia • mitral regurgitation • prognosis
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Circ Cardiovasc Imaging. 2016;9:
doi: 10.1161/CIRCIMAGING.116.005070
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-9651. Online ISSN: 1942-0080

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