Cardiac magnetic resonance imaging for ischemic mitral regurgitation: A guide through complex surgical terrain

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Ischemic mitral regurgitation (MR) increases risk of heart failure, arrhythmia, and death with valve pathology attributed to papillary muscle displacement, leaflet tethering, and annular dilation. Treatment is challenging because ischemic MR recurs in up to one-third of patients undergoing repair.1 Traditional imaging modalities lack sufficient prognostic metrics to direct whether ischemic MR should be treated with valve repair or replacement. Cardiac magnetic resonance (CMR) imaging may be emerging as the best single study to address this complex clinical problem.

To explore the use of CMR in ischemic MR, Lancaster and colleagues2 used 3-dimensional parametric strain mapping to understand regional mechanisms of injury. They previously validated this technique with quantification and localization of regional left ventricle contractile injury in patients with coronary artery disease3 and contractile recovery after aortic valve replacement.4 The current study applies 3-dimensional topographic mapping to visually demonstrate that global and regional parameters of multiparametric strain deformation were worse in patients with ischemic MR and localized around the papillary muscle wall segments. This contractile injury is most notable in the posteromedial papillary muscle but is evident in the anterolateral papillary muscle, as well. Coronary artery lesions and echocardiographic evaluation regarding wall motion dysfunction were similar between coronary artery disease groups with and without MR. It was only the 3-dimensional CMR imaging that highlighted the regional differences that resulted in MR. The results would have been even more compelling had the protocol included gadolinium contrast to confirm nonviability with delayed enhancement5,6 and a comparison with faster, higher-resolution strain-encoded techniques.7 In addition, the small cohort size limited the ability to answer clinically compelling questions regarding recovery after surgery or progression of ischemic MR over time.

Despite these limitations, the authors contribute to the growing awareness that CMR provides a highly quantitative metric of regional contractile function compared with current imaging modalities. Kalra and colleagues8 used CMR to demonstrate that it is the impairment of the lateral shortening between the papillary muscles that adversely affects mitral valve geometry and determines the severity of ischemic MR, not passive ventricle size. CMR has also demonstrated that tethering height and interpapillary muscle distance are the strongest independent predictors of ischemic MR—not the location of infarction.7 Through improved imaging that better defines the mechanism of ischemic MR, innovative repair techniques and improved application of repair is possible.

CMR imaging is currently underutilized for surgical planning addressing ischemic MR. CMR provides topographic mapping of regional myocardial viability as well as a quantitative metric to localize contractile reserve. In patients with evidence of left ventricular aneurysm or dyskinesia causing MR, valve replacement is required. Alternatively, if CMR demonstrates that there is viable myocardium in the dysfunctional papillary muscle, repair would likely be successful. Mitral valve repair in an appropriate patient would avoid long-term anticoagulation, sacrifice of the native valve apparatus, and potential deterioration of the prosthetic valve. As clinicians recognize the comprehensive information provided by CMR, there is greater potential for patient-specific surgical treatment of ischemic MR.
References