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Echocardiography

Mitral Valve Regurgitation: Paradoxical Behavior of Dobutamine Stress Cardiac Magnetic Resonance Imaging

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A 62-year-old woman with mitral regurgitation (MR) underwent cardiac magnetic resonance (CMR) and dobutamine stress CMR imaging, a widely used method to analyze left ventricular function and MR volumes. During dobutamine provocation at escalating doses, the left ventricular end-diastolic diameter (LVEDD) decreased, with a corresponding decrease in MR. At peak dobutamine dose, the LVEDD further decreased, with near complete relief of MR. Upon cessation of dobutamine provocation, the MR returned to predobutamine level. This case thereby demonstrates that MR may be reversible under certain conditions. (Echocardiography 2012;29:E186-E188)

Key words: mitral regurgitation, dobutamine, cardiac magnetic resonance imaging, mitral valve prolapse

Mitral valve regurgitation (MR) is a common valve abnormality, usually associated with dilation of the left atrium and left ventricle. The fluid flux becomes turbulent, and may affect blood constituents such as the platelets or the red blood cells. It is usually assessed by echocardiography, which provides measures of the cardiac chambers and also important information concerning the regurgitated blood flux.[1] On the other hand, magnetic resonance imaging (MRI) is increasingly recognized as a valuable tool for the study of the mitral valvular and subvalvular aparatus, as well as the cardiac chamber diameters. It also provides a functional assessment and measurements of the regurgitant volume and fraction.[1] Here we describe a case of an atypical response on an MR patient during a dobutamine stress cardiac MRI.

Case Report:

A 62-year-old woman presented to our hospital for surgical evaluation of MR by the cardiovascular thoracic surgery staff. She had a history of atypical chest pain and dyspnea on exertion. She underwent a transesophageal echocardiogram, which demonstrated a small torn secondary chordae tendineae believed to be complicating her known mitral valve prolapse. The estimated ejection fraction was 60%. Additionally, there was severe right atrial dilation and moderate left atrial dilation, with mild anterior and posterior leaflet mitral valve prolapse. Cardiac catheterization revealed normal coronary arteries. She underwent cardiac magnetic resonance (CMR) imaging to better characterize systolic function and mitral apparatus, as well as etiology for her atypical chest pain, during stress dobutamine CMR imaging.

Spin echo and steady state free precession images were performed on a 1.5T MRI scanner (HD EXCITE; GE, Milwaukee, WI, USA). The images revealed normal left ventricular size and systolic function. A dobutamine stress CMR protocol was performed (Tables I and II). The mitral regurgitation severity was assessed by qualitative analyses with a subjective visualization of the jet area in left atrium and the quantitative analyses was performed following the stratification: mild < 30 mL; moderate ≥ 30 mL and ≤ 59 mL; severe ≥ 59 mL.[2]

At baseline, the left ventricle was top normal size with mild mitral valve prolapse, but moderately restricted chordae tendineae. It also showed a moderate (2–3+) MR in visual analyses, and

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TABLE I

Measurements Pre- and Postdobutamine Infusions—Horizontal Long Axis (Three-Chamber View)

	ANN	AL	PL	TAR	TAN	TH
Base	35.6	26.5	10.2	78	137	4.8
20*	37.8	30.7	6.7	53	146	4.3
40*	35.4	27.9	8.5	26	141	3.8

*mcg/kg/min of dobutamine.
ANN = annulus (mm); AL = anterior leaflet length (mm); PL = posterior leaflet length; TAR = tenting area (mm^2); TA = tenting angle (°); TH = tenting high.

TABLE II

Other Data Comparing Resting and Stress Phases

	MRS	LVEDD	LVESD	EF	RV	RF
Base	+2	50.7	29	60	36	44
40*	Trace	41.5	19.6	75	8	13

*mcg/kg/min of dobutamine.
MRS = mitral regurgitation severity; LVEDD = left ventricle end-diastole diameter (mm); LVESD = left ventricle end-systole diameter (mm); EF = ejection fraction (%); RV = regurgitant volume (mL); RF = regurgitant fraction (%).

severe at phase velocity mapping. During dobutamine provocation at intermediate dose (20 mcg/kg/min), the left ventricular end-diastolic diameter (LVEDD) decreased, with a corresponding decrease in MR, while the LV became hyperdynamic. At peak dose (40 mcg/kg/min) the LVEDD diameter further decreased, with near complete relief of MR. Upon cessation of dobutamine provocation, the MR returned to the initial level (Fig. 1).

Discussion:

The evaluation, extent, and etiology of MR can be performed by CMR. Via dobutamine stress MRI, insights into the mechanism of MR can be clarified analogous to that of dobutamine stress echocardiogram. The CMR exam has been shown to be an important tool to depict the mitral valve and subvalvular apparatus,[3–5] and is widely used to analyze LV function and MR volumes.

Typically, with dobutamine stress testing the mitral regurgitation worsens as in mitral valve prolapse, due to the pressure gradient increase between the left ventricle and the left atrium. Nevertheless, in some cases with significant mitral apparatus preservation, MR can potentially improve. Reports on the literature support the idea that the decrease in MR volume is caused by a marked reduction on the orifice of regurgitation.[6] In this case, likely due to improving ventricular geometry in the setting of restricted subvalvular apparatus, the MR disappeared due to relaxation of the restricted chordae tendineae concurrent with decreasing LVEDD and left ventricular end-systolic diameter (LVESD). This resulted in an improved LV/mitral valve apparatus orientation, improved coaptation plane, and an increased tenting angle, all translating into absence of MR at peak stress. Also, as noted in Table I, tenting area and tenting angle improved during stress, with an important reduction of the tenting area and annulus diameter and increase of the tenting angle.

The most suitable view to measure mitral geometry is the three-chamber view. The two-chamber view is not suitable for the tenting area, tenting angle, and tenting high measurements. This view can show a blend of scallops from both leaflets: it can show the anterior and central

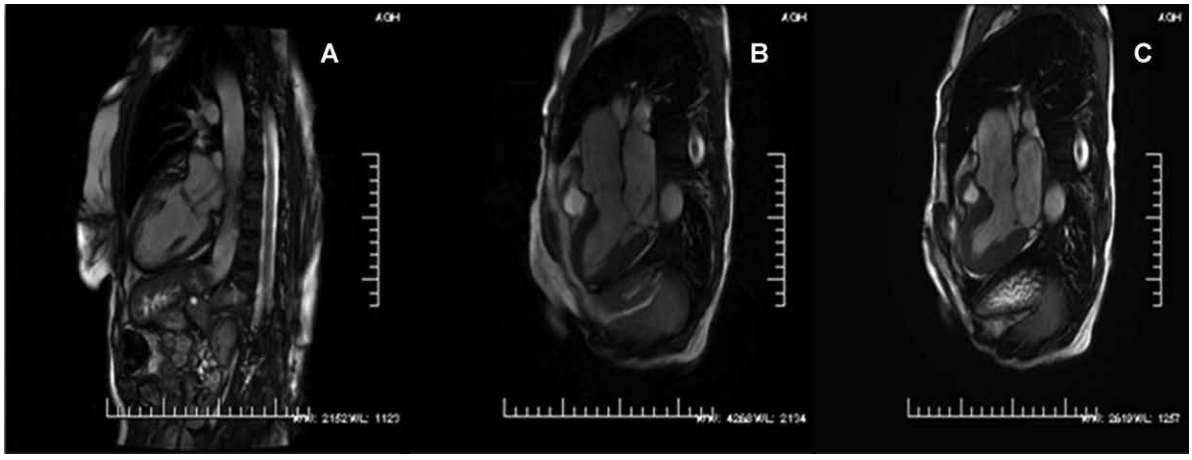

Figure 1. A. Vertical long axis (two-chamber view), note the significant mitral regurgitation; **B.** horizontal long axis (three-chamber view), note the mitral regurgitation regression meanwhile the ventricle geometry becomes shorter; **C.** horizontal long axis (three-chamber view), note the near complete relief of mitral regurgitation.

scallops of the anterior leaflet or the anterior scallop of the posterior leaflet or even the anterior, central, and posterior of one leaflet.

Heinle et al. showed improvement in MR when there is left ventricular dysfunction during echocardiogram stress test.[7] The same findings were demonstrated by Sonoda and colleagues, who noticed that dobutamine stress testing might decrease MR regurgitation flow/beat, regardless of etiology,[8] unless it was due to ischemia or infarct.

Although indications for CMR MR assessment nowadays are only for those patients without an adequate echocardiographic view, the evaluation of MR patients with cardiac CMR could be a map for individual geometry, thereby clarifying the appropriate treatment. Patients with a tenting area >1.6 mm^2, for instance, could benefit from such evaluation and avoid an unsuccessful valvuloplasty.[9]

In conclusion, in this case we hereby show that CMR imaging appears to have sensitivity in determining the underlying etiology of MR. Furthermore, we demonstrated that dobutamine provocation is an important tool delineating the behavior of the LV and valvular and subvalvular apparatus and, on occasion, providing an alternative explanation to underlying pathology of mitral regurgitation. Otherwise, the role of CMR imaging in MR patients is yet to be fully defined, including its potential contributions for diagnosis, physiopathology, and even decision making.

References

1. Dyverfeldt P, Kvitting JP, Carlhäll CJ, et al: Hemodynamic aspects of mitral regurgitation assessed by generalized phase-contrast MRI. *J Magn Reson Imaging* 2011;33(3):582–588.
2. Otto CM: *Textbook of Clinical Echocardiography*. Philadelphia: Elsevier, 2004.
3. D'Ancona G, Mamone G, Marrone GS, et al: Ischemic mitral valve regurgitation: The new challenge for magnetic resonance imaging. *Eur J Cardiothorac Surg* 2007;32:475–480.
4. Han Y, Peters DC, Salton CJ, et al: Cardiovascular magnetic resonance characterization of mitral valve prolapse. *J Am Coll Cardiol* 2008;1:294–303.
5. Keren G, Laniado S, Sonnenblick EH, et al: Dynamics of functional mitral regurgitation during dobutamine therapy in patients with severe congestive heart failure: A Doppler echocardiographic study. *Am Heart J* 1989:118:748–754.
6. Roshanali F, Mandegar MH, Yousefnia MA, et al: Low-dose dobutamine stress echocardiography to predict reversibility of mitral regurgitation with CABG. *Echocardiography* 2006;23:31–37.
7. Heinle SK, Tice FD, Kisslo J: Effect of dobutamine stress echocardiography on mitral regurgitation. *J Am Coll Cardiol* 1995;25:122–127.
8. Sonoda M, Takenaka K, Sakamoto T, et al: Effects of dobutamine infusion on mitral regurgitation. *Echocardiography* 1998;15(1):13–20.
9. Kongsaerepong V, Shiota M, Gillinov AM, et al: Echocardiographic predictors of successful versus unsuccessful mitral valve repair in ischemic mitral regurgitation. *Am J Cardiol* 2006;98(4):504–508.