Severe Ischemic Mitral Regurgitation Despite Normally Contracting Subpapillary Myocardium

Jonathan Beaudoin, MD; Robert A. Levine, MD; Chaim Yosefy, MD; Ronen Beeri, MD; Jennifer H. Neary, RDCS; Nina V. Morgan, RDCS; Jonathan J. Passeri, MD

Ischemic mitral regurgitation (MR) is common and increases heart failure and mortality after myocardial infarction.1,2 It is caused by left ventricle (LV) remodeling and subsequent papillary muscle (PM) displacement, with secondary mitral leaflet tethering. It can be seen classically in global LV dilatation and dysfunction or with more local remodeling involving the posterior PM and inferoposterior basal to mid LV wall.3 We report a case in which mitral leaflet tethering and severe MR occurred without either of these typical scenarios.

An 82-year-old woman presented to the emergency department for recent-onset progressive exertional dyspnea, orthopnea, and lower extremity edema. She had known hypertension with LV hypertrophy, normal systolic function, mitral annular calcification, and mild mitral regurgitation by echocardiogram 6 months previously. Medical history was notable for hyperlipidemia, transient ischemic attack, and family history of coronary artery disease.

Initial evaluation revealed overt heart failure with elevated NT-proBNP (> 9000 pg/mL). Chest X-ray showed bilateral pleural effusions and pulmonary vascular redistribution (Figure 1). Cardiac troponins were negative, but ECG showed new T-wave inversions suggestive of anteroseptal ischemia (Figure 2). Echocardiogram revealed new regional wall motion abnormalities with inferoapical dyskinesis and anteroapical akinesis, but preserved motion of other segments, so that LV ejection fraction was 56%. There was incomplete closure of the mitral valve with apical tenting and severe MR by color Doppler (Figure 3A). Tall E wave on mitral inflow and blunted systolic pulmonary vein wave were consistent with severe MR, and the right ventricular systolic pressure was estimated at 73 mm Hg (Figure 4). Importantly, the inferoposterior wall underlying the PM and the PM itself could be seen to thicken normally in apical and short-axis views (Figures 5, 6A, and 6B; Movies 1 and 2 in the online-only Data Supplement); in contrast, the inferior apex distal to the PM insertion was dyskinetic (Figure 5).

Stress adenosine SPECT showed a severe reversible apical defect. Cardiac catheterization showed a 90% mid-left anterior descending artery (LAD) lesion. The patient underwent successful percutaneous coronary angioplasty with drug-eluting stenting of the mid-LAD, followed by rapid resolution of heart failure. Follow-up echocardiogram after 1 month showed only mild MR with vigorous LV (LV ejection fraction 80%) and improved pulmonary hypertension with markedly decreased RV systolic pressure to 39 mm Hg (Figures 3 and 4). E wave was decreased and pulmonary vein systolic flow restored. Apical wall motion abnormalities had resolved along with mitral valve tethering (Figure 6C and 6D).

This patient is noteworthy for the absence of the 2 classical scenarios for ischemic MR. The LV was not dilated, and overall systolic function was preserved. The inferoposterior LV wall at the base and midventricle, along with the posterior PM, contracted normally in systole. On the other hand, there was dyskinesis of the inferoapical segment secondary to severe stenosis of the LAD feeding this territory. Although ischemic MR was previously reported in patients with anterior and apical infarction,2 the underlying mechanisms have only been recently explored.4

Echocardiographic observations are consistent with a mechanism in which tethering forces from apical bulging cause systolic restriction of the adjacent papillary muscle,
preventing adequate mitral valve closure and inducing MR. In this patient, LAD revascularization with subsequent resolution of the apical bulging caused dramatic improvement of MR severity, proving its ischemic origin. Understanding the exact mechanism underlying MR is of utmost importance when choosing the appropriate therapy in each patient. Of note, radionuclide testing was consistent with viability in the affected region. Surgical revascularization with mitral valve replacement could have been considered, but careful analysis of the imaging data correctly identified the underlying mechanism and suggested that percutaneous LAD revascularization alone was needed to correct the MR.

Sources of Funding
This work was funded in part by Founders Affiliate Postdoctoral Fellowship 10POST4580055 of the American Heart Association, Dallas, Texas, and Leducq Foundation Transatlantic MITRAL Network 07CVD04.

Disclosures
None.

References
Figure 4. Mitral regurgitation severity by echo-Doppler before and after revascularization. A and B, Mitral inflow pulsed Doppler: tall E wave (170 cm/s), normalized after therapy. C and D, Right superior pulmonary vein pulsed Doppler: initially blunted systolic wave with prominent diastolic wave, with systolic wave reappearance after revascularization. E and F, Tricuspid regurgitation continuous wave Doppler with initial maximal velocity of 400 cm/s, decreasing to 245 cm/s after revascularization (improved pulmonary hypertension).

Figure 5. Echocardiographic para-apical end-diastolic (A) and end-systolic (B) images showing inferoapical dyskinesis but normal contraction of the papillary muscle and underlying infero-posterior myocardium. In systole, the papillary muscle is tethered apically by the inferoapical dyskinesis (dashed arrow).
Figure 6. A and B, Apical view end-diastolic and end-systolic images showing incomplete mitral valve closure (apical leaflet tenting, dashed arrow, B) despite normal systolic contraction of the papillary muscle and underlying inferoposterior myocardium at the base and midventricle. There is inferoapical dyskinesis (B, outward arrows) in a direction that can tether the papillary muscle apically. C and D, Images 1 month after revascularization showing normal apical wall motion and resolved apical bulging and mitral tethering.
Severe Ischemic Mitral Regurgitation Despite Normally Contracting Subpapillary Myocardium
Jonathan Beaudoin, Robert A. Levine, Chaim Yosefy, Ronen Beeri, Jennifer H. Neary, Nina V. Morgan and Jonathan J. Passeri

Circulation. 2012;126:138-141
doi: 10.1161/CIRCULATIONAHA.111.064253
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2012 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/126/1/138

Data Supplement (unedited) at:
http://circ.ahajournals.org/content/suppl/2012/06/29/126.1.138.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/