Rupture of the Posteromedial Papillary Muscle Leading to Partial Flail of the Anterior Mitral Leaflet

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An 86-year-old woman with a history of hypertension presented with acute onset of chest pain and evidence of shock (hypotension and tachycardia). For the preceding 2 weeks, she had been experiencing intermittent nausea and vomiting; proton pump inhibitors did not improve her symptoms. The ECG showed ST elevations in the inferior leads and ST depressions in leads V₁ and V₂, with reciprocal ST depressions in leads I and aVL, suggesting an acute infero-posterior infarction (Figure 1). Physical examination also revealed a new 2/6 holosystolic murmur at the apex and significant bilateral rales. She was taken emergently to the cardiac catheterization laboratory, where 100% thrombotic occlusion of the middle right coronary artery was revealed (Figure 2A). A left ventriculogram revealed severe (4+) mitral regurgitation with a normal-sized left atrium (Figure 2B). An intra-aortic balloon pump was placed, and she was transferred to the cardiac intensive care unit. A transthoracic echocardiogram revealed normal right and left ventricular size and function, with evidence of inferior and posterior wall hypokinesis. The estimated pulmonary artery systolic pressure was significantly elevated at 77 mm Hg. Most important, however, the echocardiogram confirmed both severe mitral regurgitation with rupture of the posteromedial papillary muscle and partial flail of the anterior mitral leaflet (Figure 3A through D; Movies I through III in the online-only Data Supplement). Unfortunately the patient’s clinical status continued to worsen. She was deemed extremely high risk for surgery, and her healthcare proxy confirmed her do-not-resuscitate/do-not-intubate status. She expired several hours later despite maximal hemodynamic support.

Acute mitral regurgitation caused by papillary muscle rupture is a rare but life-threatening complication of myocardial infarction. Patients typically present with an inferior infarction with the right coronary artery as the culprit. They also tend to be older and have a preserved ejection fraction. Rupture typically occurs within 5 days of the infarct. It is postulated that the preserved contractility exerts increased stress on an already compromised papillary muscle, eventually leading to rupture.

This case highlights the importance of understanding mitral valve anatomy. The valve itself comprises an anterior and a posterior leaflet. Valvular competence is maintained by

Figure 1. Twelve-lead surface ECG showing ST elevations in leads II, III, and aVF and ST depressions in leads V₁, V₂, I, and aVL, suggesting acute inferoposterior myocardial infarction.

Figure 2. A, Right coronary artery angiogram demonstrating midvessel occlusion (arrow). B, Left ventriculogram, right anterior oblique view, demonstrating severe mitral regurgitation. LA indicates left atrium; LV, left ventricle; and Ao, aorta.

Figure 3A through D; Movies I through III in the online-only Data Supplement.

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The online-only Data Supplement is available with this article at http://circ.ahajournals.org/cgi/content/full/123/9/1044/DC1.

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(Circulation. 2011;123:1044-1045.)

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Circulation is available at http://circ.ahajournals.org

DOI: 10.1161/CIRCULATIONAHA.110.984724
the 2 papillary muscles (anterolateral and posteromedial) that are attached to the leaflets via the chordae tendinae. Typically, the anterolateral muscle is a single large structure, whereas the posteromedial muscle can have 1 to 3 heads.3 The 2 papillary muscles send chordae tendinae to both leaflets of the mitral valve; as a result, either leaflet can be affected by rupture of either papillary muscle. Blood supply to the anterolateral muscle is supplied by both the left anterior descending and circumflex arteries, whereas the posteromedial muscle is supplied only by the right coronary artery. Rupture of the posteromedial muscle is more common because of its single blood supply.4

Echocardiography is useful in the diagnosis of papillary muscle rupture with a sensitivity of 65% to 85%.4 Although it may be easy to identify prolapse or flail of the dysfunctional leaflet and the direction of the regurgitant jet on echocardiography, these findings do not reliably predict the affected papillary muscle, and direct visualization of the rupture is necessary for definitive diagnosis.

Disclosures
None.

References
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doi: 10.1161/CIRCULATIONAHA.110.984724

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