Severe Mitral Regurgitation Secondary to Partial Papillary Muscle Rupture Following Myocardial Infarction

Brendan M. McQuillan, MB, BS, FRACP, Arthur E. Weyman, MD, FACC

Massachusetts General Hospital, Boston

The scenario is not new—elderly patient, myocardial infarction, cardiac shock, new systolic murmur, rising enzymes—but the cause may not be common. Mechanical problems are probably at the root of the complications, but transthoracic echocardiography is not pinpointing the cause. Where to turn next? This patient's diagnosis of partial papillary muscle rupture is facilitated by multiplane transesophageal echocardiography. [Rev Cardiovasc Med. *2000;1(1):57-60*]

Key words: Coronary artery bypass graft • Echocardiography • Myocardial infarction • Valvular heart disease

Mitral regurgitation (MR) following myocardial infarction (MI) may result from dysfunction of the papillary muscles or left ventricular wall secondary to ischemia or infarction,[1] from left ventricular and mitral annular dilatation,[1] or from papillary muscle or chordal rupture.[2] While papillary muscle rupture (PMR) is a rare complication of acute MI, it is an important cause of severe MR, cardiogenic shock, and death.[3] Its early recognition allows specific management with papillary muscle repair[4] and mitral valve replacement, without which outcomes are almost universally poor. Although a new systolic murmur in the setting of recent MI may suggest PMR, this condition may be difficult to distinguish from acute ventricular septal defect (VSD), dynamic left ventricular outflow tract obstruction,[5] and other causes of acute MR.

Transthoracic echocardiography (TTE) is valuable in assessing MR and PMR, but this technique may fail to detect the specific cause in some patients.[6,7] Multiplane transesophageal echocardiography (TEE) is recognized increasingly as the noninvasive diagnostic procedure of choice for PMR, especially in patients with suboptimal images from TTE and in those in whom the ruptured papillary muscle head is not directly visualized or does not prolapse into the left atrium.[8,9]

Case Report

A 77-year-old man with a history of hypertension, stroke, and type 2 diabetes mellitus presented to a Boston community hospital with a 7-hour history of chest pain and dyspnea. Physical examination revealed a pulse of 85 beats per minute and

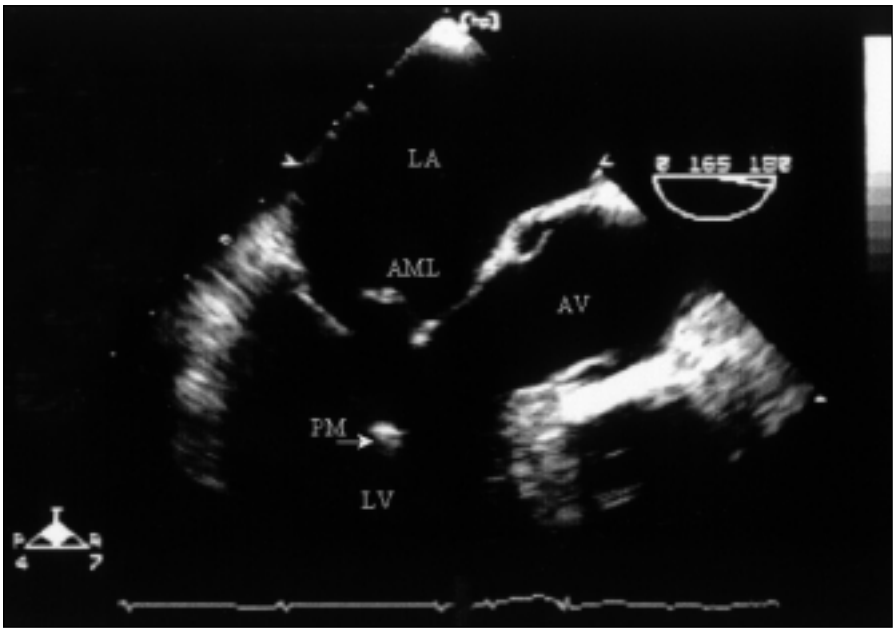

Figure 1. Transesophageal echocardiogram of ruptured papillary muscle head (PM), seen as a mobile echo mass in the left ventricle (LV) during systole. Partial flail of the anterior mitral leaflet (AML) into the left atrium (LA) is also demonstrated. (AV, aortic valve.)

blood pressure of 91/61 mm Hg. On auscultation, a harsh grade 3/6 apical systolic murmur that radiated to the axilla was heard. The patient had tachypnea with bilateral basal and midzone inspiratory crepitations. ECG demonstrated sinus rhythm, left axis deviation, inferior Q waves, 0.5 mm ST-segment elevation in leads II and aVF, early R-wave transition in anterior leads, and anteroseptal ST-segment depression. A chest roentgenogram revealed infiltrates consistent with acute pulmonary edema. Results of initial tests of cardiac enzymes included creatine kinase (CK), 322 U/L (normal, 60 to 400 U/L); CK-MB isoenzyme, 29.9 ng/mL (normal, 0 to 5.0 ng/mL); and troponin I, 18.6 ng/mL (normal, 0 to 1.5 ng/mL). The patient was treated with aspirin, furosemide, and inhaled oxygen. Progressive respiratory failure and hypotension developed. A dopamine infusion was started, and he was intubated before transfer to Massachusetts General Hospital for urgent cardiac catheterization.

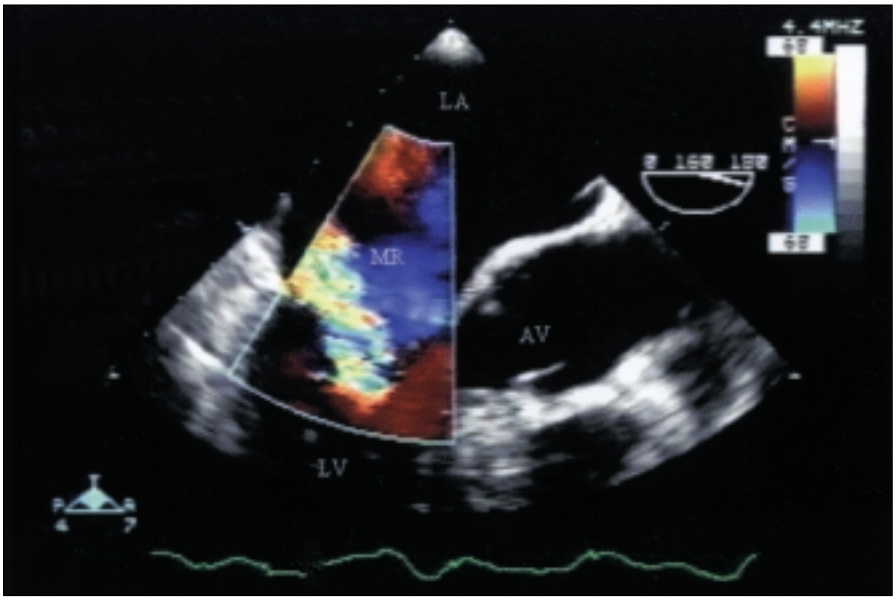

Figure 2. Transesophageal echocardiogram (color Doppler) demonstration of severe mitral regurgitation (MR) associated with the flail anterior mitral leaflet seen in Figure 1. (LV, left ventricle; LA, left atrium; AV, aortic valve.)

Coronary angiography demonstrated occlusion of the mid-left anterior descending (LAD) coronary artery and a 90% stenosis of the mid-right coronary artery (RCA); ostial stenosis was seen in the first and second obtuse marginal (OM) branches (50%, OM1; 75%, OM2) of the circumflex (CX) coronary artery. There was faint late filling via collaterals of the mid-to-distal LAD artery. The pulmonary artery systolic pressure was 50 mm Hg, and the pulmonary capillary wedge pressure was 32 mm Hg. Attempted angioplasty of the LAD artery was unsuccessful, and the patient was considered a poor candidate for urgent coronary artery bypass graft (CABG) surgery. After receiving tirofiban, he was transferred to the ICU for continued medical therapy.

The patient's peak CK level was 943 U/L; CK-MB, 63.7 ng/mL; and troponin I, 37 ng/mL within 24 hours of initial presentation. TTE images were technically limited but showed anterior and anteroseptal hypokinesis, right ventricular hypokinesis with an ejection fraction of 45%, and mild to moderate mitral regurgitation. There was no evidence of mitral valve prolapse, and no mobile masses were seen in the

left ventricle or atrium. His subsequent course was complicated by cardiogenic shock with deteriorating renal function and by paroxysmal atrial fibrillation. He continued with medical management, including ventilation, intra-aortic balloon-pump (IABP), and inotropic support. Eleven days after admission, he experienced acute deterioration with hypotension and worsening gas exchange. No change in his previously described systolic murmur was noted. Repeated cardiac enzyme tests revealed a second rise in his CK level to 316 U/L, with a CK-MB level of 24.3 ng/mL. The patient was restabilized in the ICU, and the decision was made to proceed to CABG surgery.

An intraoperative TEE demonstrated mobile echodensity in the left ventricular cavity consistent with a ruptured posteromedial papillary muscle head (Figure 1). The mass did not prolapse into the left atrium. Severe MR was noted, with partial flail of both mitral leaflets but predominantly of the anterior mitral leaflet (Figure 2). Inferior akinesis was observed in addition to anteroapical hypokinesis. The mitral valve was replaced with a 29-mm Hancock II prosthesis. A pedicled left internal mammary artery was anastomosed to the distal LAD artery, a saphenous vein graft (SVG) was placed to the RCA, and a further SVG was anastomosed to the OM1 and OM2 vessels. The patient made a slow postoperative recovery and was weaned from the IABP and intravenous inotropic agents during the following week. He remains under inpatient care 6 weeks postadmission.

Discussion

Acute cardiac shock following MI is usually the result of massive myocardial necrosis and is associated with a poor prognosis.[10] The finding of a new systolic murmur in the postinfarct setting should, however, raise the suspicion of mechanical complications, including PMR, that may not be amenable to reperfusion therapies or CABG surgery.[11] Murmurs arising from MR must be distinguished from acute VSD and dynamic left ventricular outflow tract obstruction.[12] While auscultatory features of the murmur may be of some value, further investigation with echocardiography is usually required to establish the diagnosis. It also should be noted that a murmur is not invariable with PMR because of the rapid equalization of left atrial and left ventricular pressures in some patients.[7]

Two-dimensional and Doppler TTE are useful in the detection of PMR either by direct visualization of the ruptured papillary muscle, as a mobile mass appearing in the left atrium during systole or in the left ventricle during diastole, or by identification of flail or partial flail of 1 or both mitral leaflets. The presence and severity of MR can be assessed, and the direction of the regurgitant jet may indicate the specific mitral leaflet involved.[13,14] Because of suboptimal images in tachypneic patients or in those requiring mechanical ventilation and circulatory support, the specific diagnosis may not be made by TTE in cases in which direct visualization of the defect is not possible. Mobile masses must be differentiated from tumor, vegetation, and thrombus; flail mitral leaflets may also result from ruptured chords. TEE is superior to TTE for imaging the left atrium and mitral apparatus. Among patients with nondiagnostic TTE images, proceeding to multiplane TEE is likely to improve diagnostic accuracy for PMR.[6,7,14] Prolapse of a mobile echo mass into the left atrium may not be seen in many patients with PMR.[15] Additional features to improve the specificity of diagnosis may include demonstrating large-amplitude and erratic motion of a large echo density in the left ventricle.[9]

The posteromedial papillary muscle is supplied solely by the RCA and is thus more susceptible to ischemic injury and rupture than is the anterolateral muscle, which is generally supplied by both diagonal branches and the CX coronary artery. For the same reason, posteromedial PMR is much more common with inferior MI than with anterior infarction.[14] Partial rupture, in which only 1 or more heads of a papillary muscle are separated from the ventricular wall, is more common

Main Points

- Papillary muscle rupture (PMR) may cause severe mitral regurgitation (MR), cardiogenic shock, and death.
- A new systolic murmur in the setting of a recent myocardial infarction may suggest PMR.
- A murmur may not be present in some patients with PMR, because left atrial and ventricular pressures equalize rapidly.
- Transthoracic echocardiography can assess MR and PMR but may not be able to detect their cause in some patients.
- Multiplane transesophageal echocardiography is used increasingly to detect PMR.

and is likely to be less catastrophic than is complete PMR. In some series, post-MI patients with PMR have been found to be older and less likely to have triple-vessel disease than are those with severe MR unrelated to PMR.[16] PMR is also common in subjects with delayed presentation (24 hours or longer) to the hospital and in those who suffer inhospital recurrent angina or infarct extension.[17]

MR in the setting of acute MI carries a significant mortality rate and unfavorably alters the prognosis for medical and surgical therapy. PMR carries a poor outlook that may be improved by prompt recognition and surgical correction.[18] Surgical intervention has demonstrated improvements in MR (reduced severity), left atrial size, left ventricular end-diastolic and end-systolic dimensions, and ejection fractions. While the majority of procedures involve mitral valve replacement, surgical repair of the mitral apparatus is feasible and has produced encouraging survival results in some centers.[4]

This case describes partial PMR resulting in severe MR and contributing to cardiac shock in an elderly man with a late (more than 6 hours) presentation of acute MI. Coronary angiography demonstrated triple-vessel disease, and attempts at early reperfusion with angioplasty were unsuccessful. This patient suffered protracted cardiac shock and, while a systolic murmur consistent with MR was present from the initial stages of the admission, an early TTE with technically limited views demonstrated only mild to moderate MR and did not display any other features suggestive of PMR. Extension of the initial MI was suspected as the cause of the patient's late decompensation and prompted surgical intervention. Given the delay between the initial TTE and the subsequent intraoperative TEE, it is not possible to establish the exact timing of PMR in this case. It is postulated that ischemic MR with papillary muscle dysfunction may have developed into partial PMR around the time of the second elevation in cardiac enzymes. TEE should be considered for the assessment of possible mechanical complications, including PMR, in patients with acute cardiac shock post-MI. ■

References

1. Izumi S, Miyatake K, Beppu S, et al. Mechanism of mitral regurgitation in patients with myocardial infarction: a study using real-time two-dimensional Doppler flow imaging and echocardiography. *Circulation*. 1987;76:777-785.
2. Lavie CJ, Gersh BJ. Mechanical and electrical complications of acute myocardial infarction. *Mayo Clin Proc*. 1990;65:709-730.
3. Feneley MP, Chang VP, O'Rourke MF. Myocardial rupture after acute myocardial infarction: ten-year review. *Br Heart J*. 1983;49:550-556.
4. Fasol R, Lakew F, Wetter S. Mitral repair in patients with a ruptured papillary muscle. *Am Heart J*. 2000;139:549-554.
5. Haley JH, Sinak LJ, Tajik AJ, et al. Dynamic left ventricular outflow tract obstruction in acute coronary syndromes: an important cause of new systolic murmur and cardiogenic shock. *Mayo Clin Proc*. 1999;74:901-906.
6. Christ G, Siostrzonek P, Maurer G, Baumgartner H. Partial papillary muscle rupture complicating acute myocardial infarction: diagnosis by multiplane transesophageal echocardiography. *Eur Heart J*. 1995;16:1736-1738.
7. Manning WJ, Waksmonski CA, Boyle NG. Papillary muscle rupture complicating inferior myocardial infarction: identification with transesophageal echocardiography. *Am Heart J*. 1995; 129:191-193.
8. Patel AM, Miller FAJ, Khandheria BK, et al. Role of transesophageal echocardiography in the diagnosis of papillary muscle rupture secondary to myocardial infarction. *Am Heart J*. 1989;118:1330-1333.
9. Moursi MH, Bhatnagar SK, Vilacosta I, et al. Transesophageal echocardiographic assessment of papillary muscle rupture. *Circulation*. 1996;94:1003-1009.
10. Hochman JS, Boland J, Sleeper LA, et al. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an Inter national Registry. SHOCK Registry Investiga tors. *Circulation*. 1995;91:873-881.
11. Hickey MS, Smith LR, Muhlbaier LH, et al. Current prognosis of ischemic mitral regurgitation: implications for future management. *Circulation*. 1988; 78:I51-159.
12. Bartunek J, Vanderheyden M, de Bruyne B. Dynamic left ventricular outflow tract obstruction after anterior myocardial infarction: a potential mechanism of myocardial rupture. *Eur Heart J*. 1995;16:1439-1442.
13. Mintz GS, Victor MF, Kotler MN, et al. Two-dimensional echocardiographic identification of surgically correctable complications of acute myocardial infarction. *Circulation*. 1981;64:91-96.
14. Kishon Y, Iqbal A, Oh JK, et al. Evolution of echocardiographic modalities in detection of post-myocardial infarction ventricular septal defect and papillary muscle rupture: study of 62 patients. *Am Heart J*. 1993;126:667-675.
15. Clements SDJ, Story WE, Hurst JW, et al. Ruptured papillary muscle, a complication of myocardial infarction: clinical presentation, diagnosis, and treatment. *Clin Cardiol*. 1985;8:93-103.
16. Calvo FE, Figueras J, Cortadellas J, Soler-Soler J. Severe mitral regurgitation complicating acute myocardial infarction: clinical and angiographic differences between patients with and without papillary muscle rupture. *Eur Heart J*. 1997;18:1606-1610.
17. Figueras J, Cortadellas J, Calvo F, Soler-Soler J. Relevance of delayed hospital admission on development of cardiac rupture during acute myocardial infarction: study in 225 patients with free wall, septal or papillary muscle rupture. *J Am Coll Cardiol*. 1998;32:135-139.
18. Choudhary SK, Bhan A, Sharma R, et al. Post-infarction ischaemic mitral regurgitation: what determines the outcome. *Indian Heart J*. 1999;51: 508-514.

Other Contributors

In addition to the Medical and Contributing Editors, the following authors contributed to this issue:

R. Michael Benitez, MD
Assistant Professor of Medicine
Director of Cardiac Graphics
Division of Cardiology
University of Maryland School of Medicine
Baltimore

Brendan M. McQuillan, MB, BS, FRACP
Clinical and Research Fellow
Department of Cardiac Ultrasound
Massachusetts General Hospital
Boston