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A 74-year-old woman with hemodynamic complications of acute MI

A 74-YEAR-OLD WOMAN presents to the emergency department with chest pain lasting 12 hours. She has asthma, idiopathic pulmonary fibrosis (for which she has taken maintenance steroids for the past 3 years), hypertension, and diabetes mellitus. Ten years ago, she underwent left heart catheterization, which was reported to be negative.

The patient is admitted to the coronary care unit with a diagnosis of non-ST-segment elevation myocardial infarction (MI). Her peak creatine kinase MB level is 223 ng/mL (normal range 0.0–8.8), and troponin T 1.18 ng/mL (normal range 0.00–0.10).

She is given aspirin, a beta-blocker, intravenous heparin, nitrates, and eptifibatide, and the chest pain resolves. However, about 12 hours after admission her chest pain returns and she rapidly becomes hypotensive and goes into shock.

DIFFERENTIAL DIAGNOSIS

1 What should be considered in the differential diagnosis of sudden onset of hypotension and shock following acute MI?

- ❑ Ventricular septal defect
- ❑ Mitral regurgitation due to papillary muscle rupture
- ❑ Ventricular free wall rupture
- ❑ Right ventricular infarction
- ❑ All of the above

All of these complications should be considered in any patient with a sudden rapid hemodynamic deterioration in the hours and days following an acute MI.

Ventricular septal defects develop acutely in up to 4% of patients after an acute MI,[1–3] usually 3 to 7 days after the acute event, although they may be seen in the first 24 hours.[1] They occur with equal frequency following anterior, inferior, or posterior infarctions.[4] Patients at risk have large infarcts or single-vessel disease with poor collateral circulation.

A pansystolic murmur is usually heard in the left lower sternal border. Absence of the murmur, however, does not rule out the diagnosis, especially in patients with low blood pressure. Several features distinguish this murmur from that of mitral regurgitation due to papillary muscle rupture (TABLE 1).

Mitral regurgitation due to papillary muscle rupture. Mitral regurgitation is relatively common after an acute MI and varies in its clinical severity. Post-infarct mitral regurgitation, irrespective of the hemodynamic burden, is associated with a worse prognosis.[5]

Papillary muscle rupture complicates 1% of all acute MIs and accounts for fewer than 5% of cases of ischemic mitral regurgitation. It

TABLE 1

Murmurs of a ventricular septal defect vs papillary muscle rupture

FEATURE	VENTRICULAR SEPTAL DEFECT	MITRAL REGURGITATION DUE TO PAPILLARY MUSCLE RUPTURE
Timing	Pansystolic	Pansystolic
Location	Left lower sternal border	Apex
Intensity	Loud	Variable*
Thrills	Sometimes	Rare

*Depending on cardiac output and left atrial pressure; with low cardiac output and high left atrial pressures, the murmur may be inaudible

causes severe mitral regurgitation but has become rarer in the era of reperfusion therapy. It most often occurs 2 to 7 days after the MI.[6]

Papillary muscle rupture is more common with inferior MIs, and the posteromedial papillary muscle is more prone to rupture than the anterolateral papillary muscle. The reason: the posteromedial papillary muscle is supplied by a single artery, the posterior descending artery, which is supplied by the right coronary artery in 85% of people.[4,7,8] In contrast, the anterolateral papillary muscle is supplied by both the left anterior descending and the left circumflex coronary arteries.

A new pansystolic murmur may be audible (TABLE 1). However, the murmur may be soft or inaudible in severe cases due to rapid equalization of left atrial and left ventricular pressures. The character of the murmur may be unreliable for assessing the severity of the mitral regurgitation or for guiding the selection of an appropriate diagnostic test.

Ventricular free wall rupture, the most catastrophic complication of an acute MI, occurs in 3% of patients. It can occur up to 2 weeks after an acute MI. Thrombolytic use has decreased the overall incidence of ventricular free wall rupture, but now it tends to occur sooner than in the past.

The murmur of mitral regurgitation may not be reliable

Risk of this complication is higher in the elderly, women, patients with hypertension, and those with poor coronary collateral blood supply.[4]

With acute rupture, there is electromechanical dissociation and sudden cardiac death. With subacute rupture (eg, a left ventricular pseudoaneurysm), patients may have jugular venous distension, pulsus paradoxus, a pericardial rub, and a new to-and-fro murmur.

Right ventricular infarction. The right ventricle is involved in almost 50% of inferior MIs, but isolated right ventricular infarction occurs in fewer than 3% of all MIs.

The triad of hypotension, clear lungs, and an elevated jugular venous pressure strongly suggests right ventricular infarction,[9] although similar findings may be found in patients with pericardial tamponade, constrictive pericarditis, restrictive cardiomyopathy, or a large pulmonary embolus. Many patients also have left heart failure due to multivessel disease and left ventricular infarction.

Other considerations. Our patient was on long-term steroid therapy, and the stress of having an MI can precipitate adrenal insufficiency in such patients. Furthermore, although it would be rare to have both an acute pulmonary embolism and an acute MI, pulmonary embolism should be considered in a patient with sudden hypotension.

DIAGNOSTIC TESTING

2 What is the next diagnostic step?

- ❑ Transthoracic echocardiography
- ❑ Electrocardiography
- ❑ Chest radiography
- ❑ Left heart catheterization

Transthoracic echocardiography with color flow Doppler is the test of choice, as it is quick, is easy to perform, and provides the most information for a rapid and accurate diagnosis.[10] It can easily show the cardiac chamber size and function, valve anatomy, septal defects, and the pericardium.

In some cases, however, transesophageal echocardiography may be required. Transesophageal echocardiography provides detailed information about the mitral valve and can aid in deciding whether the valve should be repaired or replaced.[11]

Electrocardiography may reveal a recent inferior MI, although our patient has a non-ST-segment elevation MI.

Chest radiography may show evidence of pulmonary edema.

Left heart catheterization is usually done before mitral valve surgery to assess the coronary arteries, in case coronary artery bypass grafting is also required.

Case continued

On physical examination, our patient has elevated jugular venous pressure, a new pansystolic murmur, and lung rales.

Electrocardiography shows no new changes. Chest radiography reveals pulmonary edema. Transthoracic echocardiography shows rupture of the anterolateral papillary muscle with severe mitral regurgitation (FIGURE 1).

Left heart catheterization shows severe stenosis of both the left anterior descending

and left circumflex coronary arteries, resulting in compromised blood flow to the anterolateral papillary muscle (FIGURE 2).

TREATMENT OPTIONS

3 What are the treatment options for papillary muscle rupture?

- ❑ Medical management
- ❑ Mitral valve repair
- ❑ Mitral valve replacement

In acute severe mitral regurgitation due to papillary muscle rupture, initial aggressive medical therapy is a bridge to definitive, emergency surgical management.

Medical therapy includes intravenous arterial vasodilators such as sodium nitroprusside, which helps unload the left ventricle and thus decreases left ventricular filling pressures and the degree of mitral regurgitation.

In hypotensive patients who cannot tolerate vasodilators, an intraaortic balloon pump (IABP) should be inserted. An IABP reduces the load on the left ventricle by decreasing the afterload and also improves coronary perfusion.

The definitive management, however, is surgery. The decision to repair or replace the mitral valve is complex and is guided by the anatomy of the mitral apparatus. Transesophageal echocardiography is valuable to assess the feasibility of repair.[11,12] Surgery for a ruptured papillary muscle carries a high perioperative death rate. Among survivors, short-term and long-term postoperative death rates are influenced by the presence and severity of left ventricular dysfunction.[13]

Case continued

In our patient, a surgical consultation is obtained promptly and an IABP is inserted for hemodynamic support. Emergency left heart catheterization shows severe atherosclerosis in three coronary arteries (FIGURE 2). Mitral valve replacement with a #27 Carpentier-Edwards valve and coronary artery bypass grafting to the right coronary, left anterior descending, and left circumflex coronary arteries are successful, and the patient is discharged in good condition on the 10th postoperative day.

Mitral regurgitation due to papillary muscle rupture

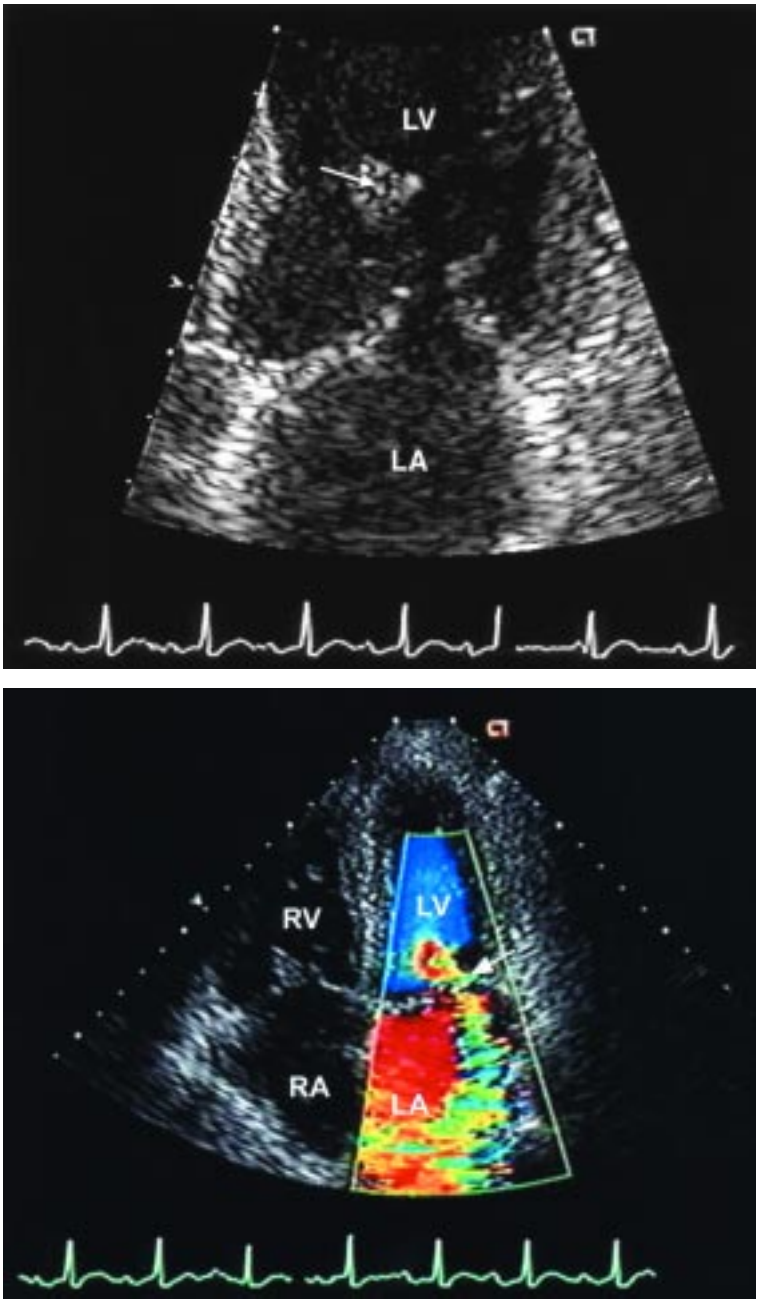

FIGURE 1. Top, transthoracic apical four-chamber view (LA = left atrium, LV = left ventricle) with a close-up of the mitral valve apparatus in diastole showing the ruptured anterolateral papillary muscle attached to the anterior mitral leaflet (arrow). **Bottom,** transthoracic apical four-chamber view with color flow imaging showing severe mitral regurgitation (arrow) (LA = left atrium, LV = left ventricle, RA = right atrium, RV = right ventricle).

Triple-vessel coronary artery disease

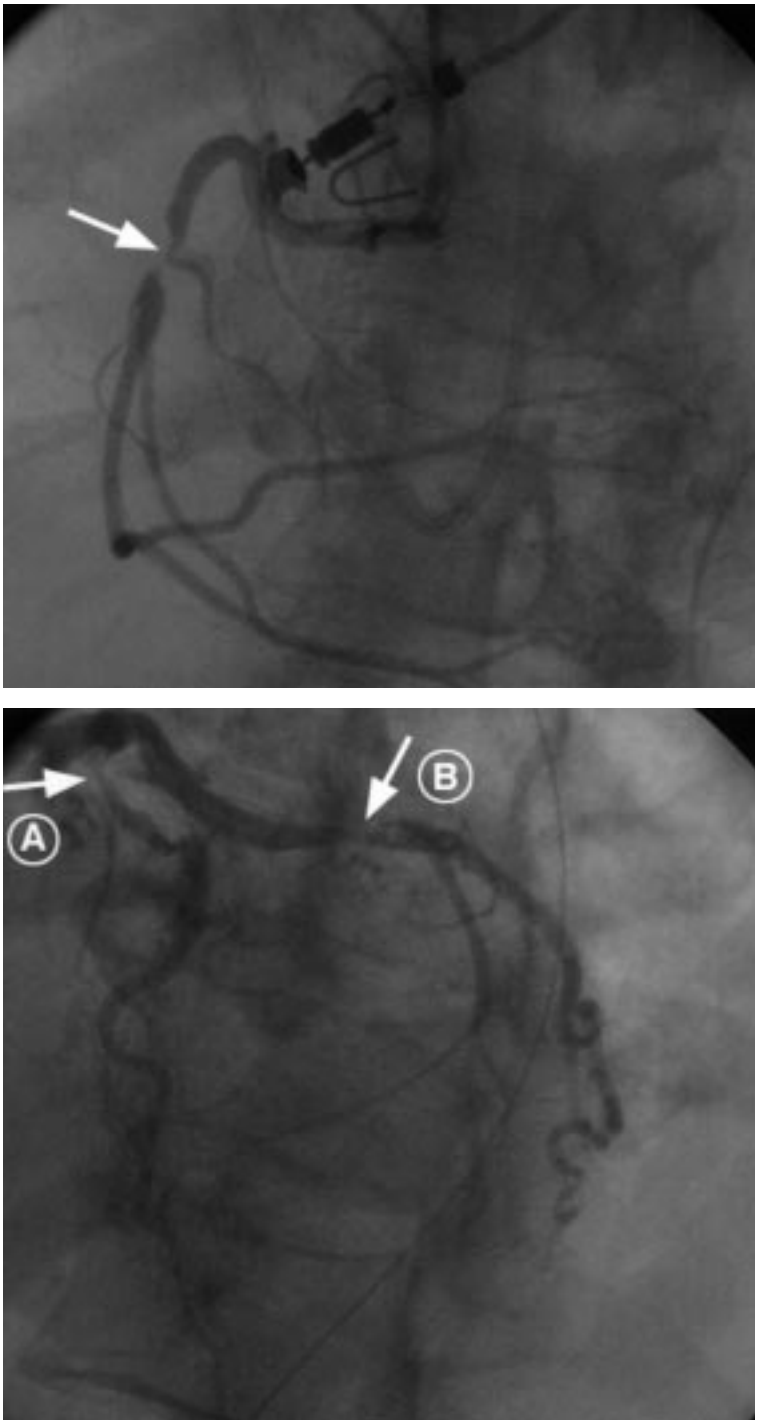

FIGURE 2. Top, coronary angiography shows severe narrowing of the proximal portion of the right coronary artery in the left anterior oblique projection (arrow). **Bottom,** severe stenosis of the proximal portions (arrows) of the left anterior descending (A) and circumflex (B) coronary arteries in the left anterior oblique projection.

Comments

A high index of suspicion is the key to early diagnosis and prompt management of mechanical complications in a hemodynamically compromised patient with an acute MI. Advances in echocardiography have made it the key diagnostic test in such patients.

REFERENCES

1. **Crenshaw BS, Granger CB, Birnbaum Y, et al.** Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation 2000; 101:27–32.
2. **Hutchins GM.** Rupture of the interventricular septum complicating myocardial infarction: pathological analysis of 10 patients with clinically diagnosed perforations. Am Heart J 1979; 97:165–173.
3. **Menon V, Webb JG, Hillis LD, et al.** Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK? J Am Coll Cardiol 2000; 36:1110–1116.
4. **Mukherjee D.** Complications of myocardial infarction. In: Marso SP, Griffin BP, Topol EJ, editors. Manual of Cardiovascular Medicine. 1st ed. Philadelphia: Lippincott Williams and Wilkins; 2000:39–55.
5. **Maisel AS, Gilpin EA, Klein L, et al.** The murmur of papillary muscle dysfunction in acute myocardial infarction: clinical features and prognostic implications. Am Heart J 1986; 112:705–711.
6. **Lehmann KG, Francis CK, Dodge HT.** Mitral regurgitation in early myocardial infarction. Incidence, clinical detection, and prognostic implications. TIMI Study Group [published erratum appears in Ann Intern Med 1992 Aug 15;117(4):349]. Ann Intern Med 1992; 117:10–17.
7. **Reeder GS.** Identification and treatment of complications of myocardial infarction. Mayo Clin Proc 1995; 70:880–884.
8. **Manning WJ, Waksmonski CA, Boyle NG.** Papillary muscle rupture complicating inferior myocardial infarction: identification with transesophageal echocardiography. Am Heart J 1995; 129:191–193.
9. **Kinch JW, Ryan TJ.** Right ventricular infarction. N Engl J Med 1994; 330:1211–1217.
10. **Roelandt JR, Smyllie JH, Sutherland GR.** The surgical complications of acute myocardial infarction: color Doppler evaluation. Int J Card Imaging 1989; 4:45–47.
11. **Lawrie GM.** Mitral valve repair vs replacement. Current recommendations and long-term results. Cardiol Clin 1998; 16:437–448.
12. **Gillinov AM, Wierup PN, Blackstone EH, et al.** Is repair preferable to replacement for ischemic mitral regurgitation? J Thorac Cardiovasc Surg 2001; 122:1125–1141.
13. **Kishon Y, Oh JK, Schaff HV, et al.** Mitral valve operation in postinfarction rupture of a papillary muscle: immediate results and long-term follow-up of 22 patients. Mayo Clin Proc 1992; 67:1023–1030.

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