CME CREDIT

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An elderly woman with chest pain and shortness of breath

AN 80-YEAR-OLD WOMAN with a history of hypertension, dyslipidemia, osteoarthritis, cigarette smoking, and an unspecified thyroid problem presented to her primary care physician because of chest and back discomfort and dyspnea with exertion that began 5 days earlier. The chest and back discomfort radiated to both arms and the epigastrium, did not change with respiration, and was associated with diaphoresis. She denied associated nausea, vomiting, syncope, presyncope, or fever.

Her symptoms began about 5 days before presentation

These symptoms gradually worsened during the first few hours after onset, then improved somewhat after 1 to 2 days, but her family eventually persuaded her to seek medical attention.

Medications at the time of presentation included aspirin, atorvastatin, diltiazem, escitalopram, and celecoxib.

On initial physical examination, her heart rate was 90 beats per minute, blood pressure 104/60 mm Hg, and blood oxygen saturation 97% while breathing room air. She was breathing comfortably in no acute distress. Her lung fields were clear, and her heart had a regular rate and rhythm without murmurs or extra sounds. Her abdomen was normal. Her extremities were warm, well perfused, and without edema. Her distal pulses were strong and symmetric.

An electrocardiogram was obtained in the primary care physician's office (**FIGURE 1**).

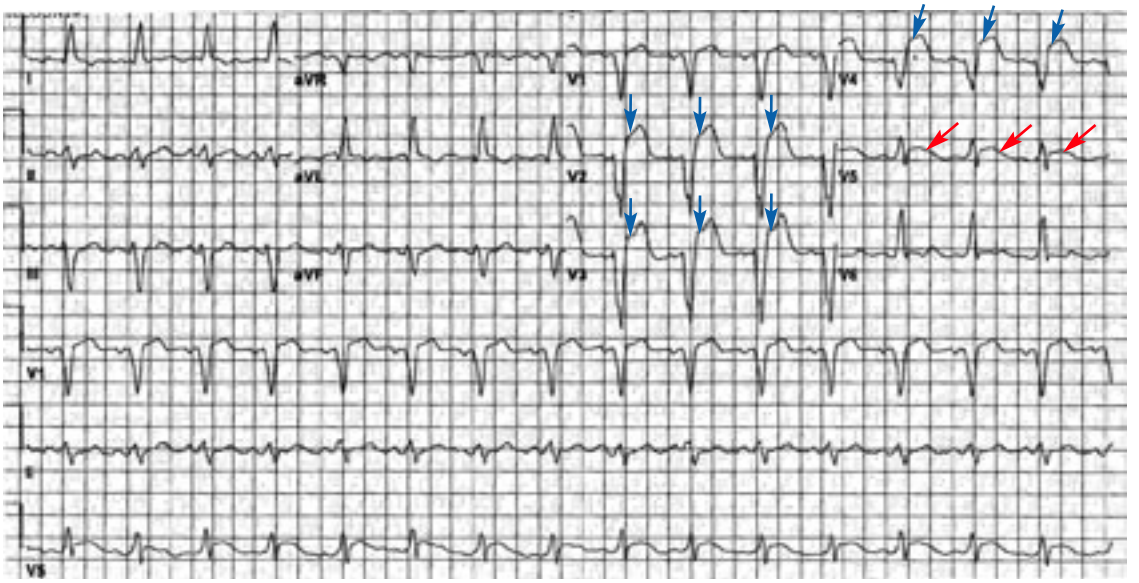

FIGURE 1. The patient's presenting electrocardiogram shows normal sinus rhythm, left bundle branch block with concordant ST-segment elevation in lead V_5 (red arrows) and discordant ST-segment elevation in leads V_2, V_3, and V_4 consistent with acute myocardial infarction (blue arrows).

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DIFFERENTIAL DIAGNOSIS

1 Which is the most likely cause of her initial symptoms and subsequent electrocardiographic findings?

- ❑ Aortic dissection
- ❑ Pericarditis
- ❑ Anterior myocardial infarction (MI)
- ❑ Proximal pulmonary embolism

Anterior MI is the most likely diagnosis, based on her symptoms, cardiac risk factors, and electrocardiographic findings, which include a left bundle branch block with concordant ST-segment elevation in lead V_5 and discordant ST-segment elevation in leads V_2 through V_4.

Although her electrocardiogram meets the criteria for acute MI with left bundle branch block (ST-segment elevation of 1 mm or more concordant with the QRS axis and ST-segment elevation of 5 mm or more discordant with the QRS axis),[1] the mere presence of a left bundle branch pattern in combination with her symptom complex suggests myocardial ischemia or MI. Additionally, based on the electrocardiographic findings, the differential diagnosis includes a post-MI anterior left ventricular aneurysm.

Aortic dissection is unlikely, as her pain gradually worsened: chest and back pain related to dissection is generally worst at the onset of the dissection. Her electrocardiographic findings are not consistent with pericarditis, and a large, proximal "saddle" pulmonary embolism would cause more hemodynamic compromise and hypoxemia.

Case continued

The patient was taken to a local emergency department for further evaluation and management. On initial testing, her chest radiograph was normal, as were her serum electrolyte levels and renal function as reflected in her serum creatinine and blood urea nitrogen levels. Her white blood cell count was mildly elevated at 14.8 × 10^9/L (normal 4.0–11.0). Cardiac biomarkers were elevated, ie:

- Creatine kinase (CK) 306 U/L (normal 30–220)
- CK-MB fraction 23.9 ng/mL (normal 0–8.8)
- Troponin I 20.81 ng/mL (normal 0–0.1)
- B-type natriuretic peptide (BNP) 1,120 pg/mL (normal 0–100).

In view of the time course of her symptoms and her biomarker pattern (elevated CK, CK-MB, troponin I, and BNP), the patient was admitted to the cardiac intensive care unit and treated with aspirin, heparin, eptifibatide, clopidogrel, intravenous nitroglycerin, and lisinopril. Echocardiography performed upon arrival in the unit revealed a moderately dilated left ventricle with an ejection fraction of about 30% and anterior wall akinesis.

The patient was hemodynamically stable for 3 days on the above medical regimen, but on the fourth hospital day she suddenly developed hypotension and moderate respiratory distress.

CAUSE OF NEW SYMPTOMS

2 Which is the most likely cause of this patient's new hypotension and respiratory distress?

- ❑ Overaggressive medical management
- ❑ Pulmonary embolism
- ❑ Intensive care unit myopathy
- ❑ Mechanical complication of MI
- ❑ Dressler syndrome

In aortic dissection, pain is generally worst at the onset

Given the elapsed time from the onset of her symptoms to her acute decompensation, a mechanical complication of MI is most likely. Such mechanical complications include ventricular free-wall rupture, ventricular septal rupture, and acute mitral regurgitation from papillary muscle rupture. These complications usually occur 3 to 5 days after an MI but may occur as early as the first 24 hours or as late as 2 weeks after the onset of symptoms.

Ventricular free-wall rupture usually presents as either sudden death or hemopericardium, causing rapid hemodynamic deterioration from cardiac tamponade. It is characterized by acute hypotension, biventricular heart failure, and a new, harsh holosystolic murmur (often associated with a palpable thrill).

Acute mitral regurgitation is also associated with a new murmur, hemodynamic compromise, and acute pulmonary congestion.

In an emergency, transthoracic echocardiography can be used to rapidly discriminate

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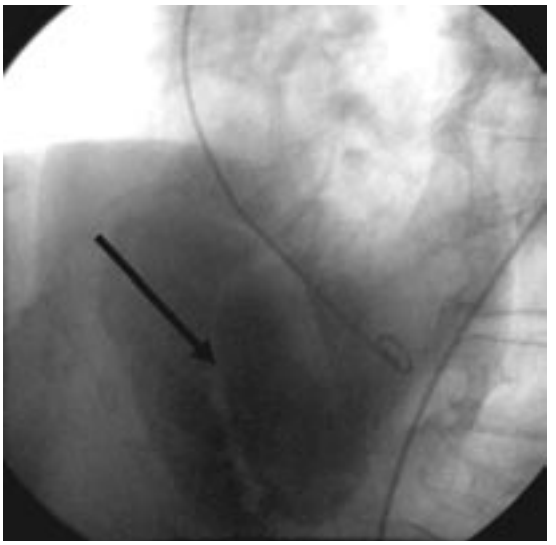

FIGURE 2. Left anterior oblique projection during left ventriculography. Note the simultaneous filling of both ventricular cavities, separated by the ventricular septum (arrow). The opacified right ventricular cavity is anterior to the septum, seen in the lower left portion of the image.

Most mechanical complications occur 3–5 days after the MI, but some are delayed up to 2 weeks

among these complications, though transesophageal echocardiography may be necessary to identify them definitively if a transthoracic study is negative but clinical suspicion is high. More invasive means of diagnosis could include left-sided and right-sided heart catheterization.

As this patient has suffered a large anterior MI, ventricular septal rupture or free-wall rupture is most likely; papillary muscle rupture tends to occur more often in inferior or posterior MIs, as the anterolateral papillary muscle receives a dual blood supply from both the left anterior descending and left circumflex coronary arteries, while the posteromedial papillary muscle receives flow from only the posterior descending coronary artery.

As for the other possibilities . . .

Pulmonary embolism should always be considered in the setting of acute hemodynamic decompensation with respiratory distress, but this patient's presentation is more clinically consistent with a mechanical complication of MI. Echocardiography would again be useful in differentiating pulmonary embolism from such a complication.

Intensive care unit myopathy (also called critical illness myopathy—acute muscle injury, most frequently associated with sepsis and multisystem organ failure) would not present in this way or after such a short time of hospitalization.

Dressler syndrome (also called post-MI syndrome—pleuritic chest pain, a pericardial friction rub, fever, and leukocytosis, occasionally with pleural effusion or pulmonary infiltrates) usually does not occur for weeks or months after MI. An elevated erythrocyte sedimentation rate is also characteristic.

Case continued

The patient underwent left heart catheterization, which revealed that the left anterior coronary artery was occluded in its mid-portion, left ventricular systolic function was reduced (ejection fraction about 20%), and a large left-to-right shunt was present, apparently from ventricular septal rupture (FIGURE 2). Simultaneous right heart catheterization revealed elevated pulmonary filling pressures and a significant left-to-right shunt.

RISK FACTORS FOR MECHANICAL COMPLICATIONS

3 Which feature of this patient's presentation did *not* increase her risk of this complication of MI?

- ❑ Single-vessel left anterior descending coronary artery disease
- ❑ Tobacco use
- ❑ Large area of infarct and significantly depressed left ventricular ejection fraction
- ❑ First MI
- ❑ Female sex

Ventricular septal rupture complicates 0.2% of all MIs and is more common with a first MI, more often in the left anterior descending territory than in that of the left circumflex or right coronary artery.[2–5] Anterior MI results in apical ventricular septal rupture, while inferior or posterior MI results in basal ventricular septal rupture; each rupture type occurs with roughly equal incidence.[6]

The degree of myocardial necrosis also correlates with the risk of mechanical complications; the risk of rupture of either the ven-

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tricular wall or septum is higher if the peak concentration of the CK-MB fraction is greater than 150 U/L.[2] Such an association does not seem to exist between infarct size and papillary muscle rupture, as the specific location of the infarct is much more important with this complication.

The risk of ventricular septal rupture is lower with primary percutaneous revascularization than with fibrinolytic therapy.[7] Women have a higher risk than men.[3]

Somewhat paradoxically, the risk is higher in patients who did not have extensive coronary disease before the MI,[4,8,9] perhaps because they do not have as much collateral circulation in the ventricular septum. The same mechanism might explain why the risk is higher with a first MI—and seems to be lower in smokers, who tend to have more diffuse, multivessel coronary artery disease, giving rise to more extensive collateral circulation.[3,5,9]

CORRECTING SEPTAL RUPTURE

4 Which is the optimum technique and timing for correcting post-MI ventricular septal rupture?

- ❑ Surgical correction emergently
- ❑ Percutaneous correction emergently
- ❑ Surgical correction when hemodynamically stable
- ❑ Percutaneous correction when hemodynamically stable

In 2004, the American College of Cardiology and American Heart Association published the findings of a joint task force regarding mechanical complications of MI, giving emergent surgery a class I indication.[10]

Because the death rate is high and data from randomized trials are lacking, most decisions about the method of repairing post-MI ventricular septal ruptures are made on a case-by-case basis.[6]

Rather than a single, discrete defect in the septum as is usually seen in congenital ventricular septal defects, post-MI ventricular septal ruptures tend to result in serpiginous, poorly defined communications in the muscular septum that often grow over time as the damaged myocardium is gradually replaced by scar (in effect, a "Swiss cheese" septum), making them very difficult to repair completely.[6]

In the first Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) study, patients with MIs complicated by ventricular septal rupture had a significantly higher death rate: 74%, compared with 7% among patients without ventricular septal rupture. Post-MI ventricular septal rupture accounts for approximately 5% of periinfarction deaths overall.[3]

Patients who undergo surgical repair of post-MI ventricular septal rupture have lower death rates than those who do not: 40% of GUSTO-1 patients with ventricular septal rupture were taken to surgery, and the death rates at 30 days and 1 year were 47% and 53%, respectively. Long-term survival has been reported to improve with simultaneous revascularization as well.[10,11]

More recently, percutaneous methods have been developed to close post-MI ventricular septal ruptures, using various devices. The best results seem to have been achieved with the Amplatzer PI Muscular VSD Occluder (AGA Medical Corp, Golden Valley, MN): in a series of 18 post-MI patients (age range 52–86 years) with ventricular septal rupture who underwent placement of the device, the 30-day and 60-day death rates were 28% and 41%, respectively. Echocardiography was used to assess residual shunt, and 2 of 18 patients required a second procedure to repair residual shunt (usually occurring through the fabric covering of the device).[13]

Both surgical and percutaneous studies have been affected by selection bias in this high-risk population; currently, there have been no additions to the 2004 joint task force recommendations concerning percutaneous interventions for mechanical complications of MI.

Mortality in GUSTO patients with ventricular septal rupture: 74%; Without rupture: 7%

Case continued

The patient was taken for a repair procedure less than 48 hours after the diagnosis of ventricular septal rupture was confirmed. As is often the case with ventricular septal rupture, the left-to-right shunt could not be adequately repaired, and the patient died within 30 days of her initial presentation.

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PROMPT MI TREATMENT IS VITAL

This case illustrates not only a typical time course to a post-MI complication but also the need for early presentation in MI to offer early revascularization and thereby improve outcomes and prevent complications. When treating patients at high risk for mechanical complications of acute MI (and thereby at high risk of death should a complication occur), vigilance in monitoring for the development of such complications is essential.

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