Concise Review for Primary-Care Physicians

Identification and Treatment of Complications of Myocardial Infarction

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Mechanical complications of acute myocardial infarction include papillary muscle rupture with severe mitral regurgitation, ventricular septal rupture with acute ventricular septal defect, acute and subacute free-wall rupture, and hemodynamically significant right ventricular infarction. Although such complications are infrequent, their importance is underscored because of the potential ability to correct them with early diagnosis and appropriate treatment. The diagnosis necessitates a high degree of suspicion based on clinical clues and rapid diagnostic testing. Bedside two-dimensional echocardiography, sometimes with transesophageal echocardiography, is most commonly used to diagnose or exclude these complications. Patients suspected of having a mechanical complication of myocardial infarction should be urgently transferred to a medical center experienced in the management of these problems. For deteriorating patients without identifiable mechanical complications, coronary angiography and reperfusion with direct angioplasty should be considered.

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In the current era, in-hospital mortality because of acute myocardial infarction is due almost exclusively to circulatory failure resulting from either severe left ventricular dysfunction or one of the mechanical complications of myocardial infarction. Such complications include severe mitral regurgitation due to papillary muscle rupture, acute ventricular septal defect, rupture of the left ventricular free wall, and hemodynamically predominant right ventricular infarction. Unlike patients with pump failure due to severe left ventricular dysfunction, patients with mechanical infarct complications generally have well-preserved left ventricular function, and prompt diagnosis and appropriate therapy can be lifesaving.

Clinical clues (see subsequent discussion) are helpful in establishing suspicion of a particular mechanical complication; unifying features of most of the mechanical complications include sudden deterioration of clinical status with accompanying hypotension or shock. Traditional investigations, including electrocardiography, chest roentgenography, Swan-Ganz catheterization, and left coronary catheterization with arteriography, are often helpful in specific situations but are no longer the first-line approach to diagnosis. Severe left ventricular dysfunction can be distinguished from one of the mechanical complications most easily by bedside two-dimensional echocardiography and color flow Doppler imaging, occasionally with use of transesophageal echocardiography.[1] After an initial diagnosis has been made, patients are then selected for further testing and medical or surgical therapy as needed. Specific features of the echocardiographic examination should include measurement of left ventricular systolic function, search for free-wall or ventricular septal rupture, color flow imaging to detect intracardiac shunting, thorough assessment of the mitral valve and support apparatus for flail leaflet and disruption of the papillary muscle-chordal mechanism, color flow imaging for determination of presence and severity of mitral regurgitation, and evaluation for partial free-wall rupture or cardiac tamponade. Before corrective surgical procedures, most patients will undergo brief coronary arteriography without left ventriculography if the anatomic diagnosis is clear after echocardiographic examination.

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CARDIOGENIC SHOCK DUE TO SEVERE LEFT VENTRICULAR DYSFUNCTION

Although not strictly a mechanical complication of infarction, severe left ventricular dysfunction in the absence of a mechanical complication is presented herein because it portends an especially poor prognosis. The clinical manifestations can include florid pulmonary edema or severe hypotension in conjunction with cool underperfused skin, oliguria, and evidence of cerebral and visceral hypoperfusion.

Gallop rhythms, pulmonary congestion on chest roentgenography, and extensive ST-segment elevation, especially in the anterior distribution, are common. The prevalence of shock is approximately 7% in patients with myocardial infarction and has an associated historical mortality rate of approximately 80%.[2] For patients who seek medical treatment early (within 12 hours after onset of infarction or reinfarction), urgent cardiac catheterization and revascularization, usually by angioplasty, are commonly attempted. Although no controlled trials exist, observational studies have suggested an approximately 50% decrease in historical mortality with successful angioplasty.[3] This topic has been reviewed in detail.[4] For patients who are assessed at medical centers that do not have angioplasty capabilities, stabilization with inotropic agents, balloon pumping, and, possibly, intubation is warranted with plans for urgent transfer to a tertiary center capable of performing prompt revascularization. Of importance, the application of reperfusion therapy (thrombolysis or primary angioplasty) has decreased the frequency of subsequent shock due to left ventricular dysfunction.

ACUTE MITRAL REGURGITATION

Papillary muscle rupture is a life-threatening and eminently correctable complication of acute myocardial infarction that accounts for approximately 5% of deaths in patients with acute infarction (Fig. 1). The setting is usually acute inferior wall myocardial infarction, and the posteromedial papillary muscle is generally involved, inasmuch as its blood supply is derived primarily from the posterior descending coronary artery. Unlike other myocardial rupture syndromes (free-wall and acute ventricular septal defect), papillary muscle rupture may occur in non-Q-wave infarction as well as in Q-wave infarction. Most patients have relatively small areas of necrosis with poor collaterals, and up to 50% may have single-vessel disease. The classic clinical manifestation is acute onset of hypotension and respiratory distress due to pulmonary edema, occurring within 2 to 7 days after Q-wave inferior wall infarction. The precordium is hyperactive, and a mitral regurgitant murmur is often not audible. In the latter case, severe regurgitation causes a rapid increase in pressure in the left atrium, and this increase coupled with a large regurgitant orifice produces insufficient turbulence of blood to create a murmur. Two-dimensional echocardiography usually demonstrates a flail segment of the mitral valve, and often a severed papillary muscle head can be seen moving freely within the heart. The left ventricular function is invariably hyperdynamic as a result of ventricular contraction against the low impedance left atrium; this finding alone, even in the absence of a discernible color flow regurgitant jet (which sometimes may be absent), should suggest the diagnosis. Successful diagnosis of this condition includes an appropriate index of suspicion, and treatment should include inotropic support and intra-aortic balloon pumping; the surgical team should be rapidly mobilized. Coronary arteriography is usually performed on the way to the operating room. Left ventriculog-raphy is usually unnecessary, particularly if mechanical disruption of the mitral valve apparatus is clearly demonstrated. As experience with

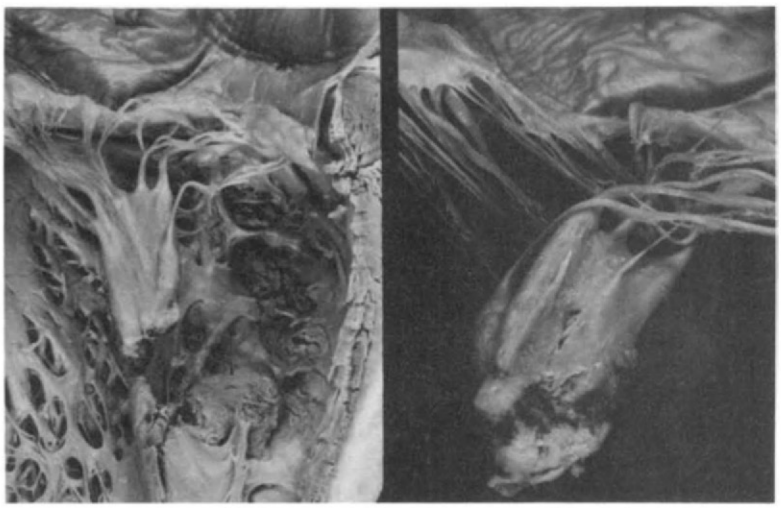

Fig. 1. Pathologic specimen, demonstrating complete transection of papillary muscle (*left*) and close-up view (*right*) caused by myocardial infarction. Severe mitral regurgitation and death occurred. (Photograph courtesy of Dr. William D. Edwards. From Reeder GS, Gersh BJ. Acute myocardial infarction. In: Stein JH, Hutton JJ, Kohler PO, O'Rourke RA, Reynolds HY, Samuels MA, et al, editors. Internal Medicine. 4th ed. St. Louis: Mosby-Year Book, 1994: 169-189. By permission.)

mitral valve repair has developed in many centers, including the Mayo Clinic, many of these valves no longer need replacement at operation. Although emergency surgical intervention has an associated operative mortality rate of approximately 20 to 25%, late results of operation show excellent survival, and the mortality of those who do not undergo surgical treatment is high.[5]

VENTRICULAR SEPTAL RUPTURE

Ventricular septal rupture occurs in approximately 2% of patients with acute infarction and causes about 5% of perinfarction mortality (Fig. 2). Unlike papillary muscle rupture, ventricular septal rupture may be noted in anterior and nonanterior infarction with similar frequency.[6] The classic anatomic substrate is a small infarction, single-vessel disease, and poor or absent collateralization of the occluded vessel. The ventricular septal defect develops at the margin of the necrotic and nonnecrotic myocardium. With anterior infarction, the defect is most commonly found in the apical septum, and with inferior infarction, it most commonly occurs at the base of the heart. The clinical manifestation differs from that of acute mitral regurgitation. Patients with postinfarct ventricular septal rupture are often comfortable while supine, and acute florid pulmonary edema does not tend to develop. In most patients, a new pansystolic murmur will develop; less commonly, a palpable thrill will be present on examination. Bedside echocardiography with color flow imaging is ideal for visualizing the ventricular septal disruption and the resultant left-to-right shunting through the defect. Occasionally, transesophageal echocardiography may be necessary to delineate the complete extent of the abnormality.

The outcome of 91 patients with ventricular septal rupture after acute myocardial infarction was recently reported, and adverse outcome correlated with advanced age, cardiogenic shock at initial examination, and a lengthy delay between septal rupture and operation.[7] In patients with cardiogenic shock, only those who underwent an operation within 48 hours survived, but the proportion surviving in this group was only 38%. In patients without cardiogenic shock, the clinical course was unpredictable, with rapid deterioration and death in approximately 50%.

In patients who are otherwise candidates for surgical treatment, the author's approach is similar to that for mitral regurgitation—define the anatomy with an echocardiographic examination, followed by coronary arteriography and prompt surgical intervention. Because of the unpredictable course and the substantial associated mortality, patients suspected of having ventricular septal rupture who are in hospitals in which experience in surgical repair of these defects is unavailable should be transferred promptly to a tertiary center for intervention.

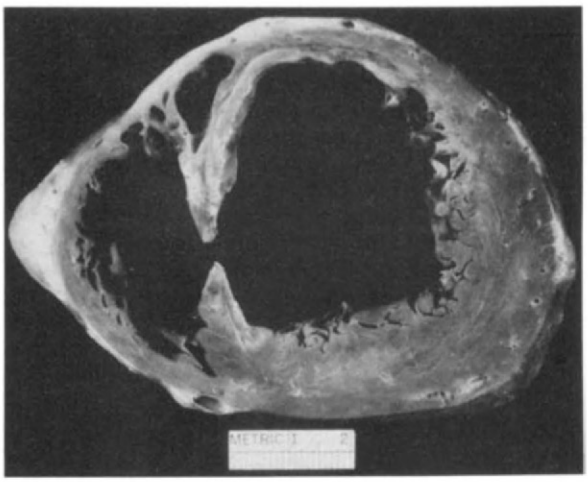

Fig. 2. Pathologic specimen, showing ventricular septal rupture. (From Edwards WD. Pathology of myocardial infarction and reperfusion. In: Gersh BJ, Rahimtoola SH, editors. Acute Myocardial Infarction. New York: Elsevier, 1991: 14-48. By permission.)

FREE-WALL MYOCARDIAL RUPTURE

Free-wall myocardial rupture (Fig. 3) accounts for approximately 10% of acute myocardial infarction-induced mortality and constitutes part of the "early hazard function" in patients treated with thrombolysis (mortality within the first 24 hours after thrombolytic treatment is actually higher in patients who receive thrombolytic therapy than in those who do not and is due, in part, to myocardial rupture). Pathologic characteristics are similar to acute ventricular septal defect, with a preponderance of small infarctions and single-vessel disease, especially in patients with hypertension who have poor collaterals. Lateral wall rupture is probably most common, although any wall may be involved. Rupture occurs within the first 5 days after infarction in about half of the cases and within 2 weeks in approximately 90%.

With acute free-wall rupture, the clinical manifestation is sudden death with electromechanical dissociation. Resuscitative measures, including pericardiocentesis, intra-aortic balloon counterpulsation, and an urgent cardiac operation, are generally futile. In some patients, however, a syndrome of subacute rupture may manifest with pericardial pain and electrocardiographic features of localized or regional pericarditis. Nausea and transient hypotension may complement these findings and should suggest the possibility of imminent rupture.[8] In patients with such clinical manifestations, the physician must have a high index of suspicion, and, again, bedside echocardiography may be helpful in demonstrating localized pericardial effusion or pseudoaneurysm (contained rupture). Unfortunately, predicting which patients may have myocardial rupture beforehand is difficult; in the few patients in whom this is possible, immediate surgical treatment

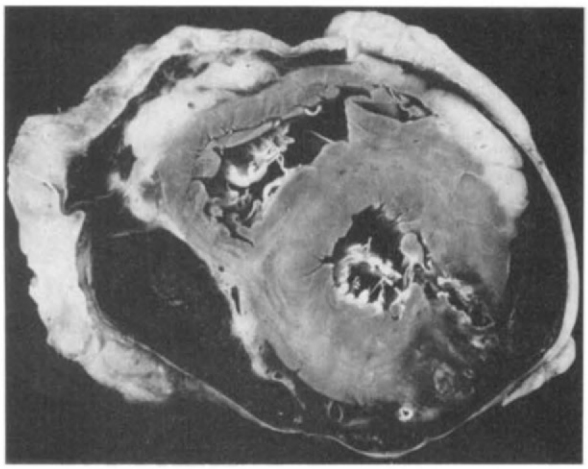

Fig. 3. Pathologic specimen, demonstrating postinfarction cardiac rupture. Free-wall rupture with hemopericardium. (From Edwards WD. Applied anatomy of the heart. In: Brandenburg RO, Fuster V, Giuliani ER, McGoon DC, editors. Cardiology: Fundamentals and Practice. Chicago: Year Book, 1987: 47-109.)

can yield gratifying results. A long-term survival rate of approximately 49% was confirmed recently in a large series of patients undergoing surgical treatment of subacute rupture.[9] These findings are extremely impressive in light of the virtually 100% fatality rate after complete rupture.

RIGHT VENTRICULAR INFARCTION

Although right ventricular dysfunction can be commonly detected in patients with inferior wall myocardial infarction, hemodynamically significant right ventricular impairment is rare, occurring in less than 10% of patients with inferior wall infarcts. In these patients, acute right ventricular failure results in underfilling of the left heart chambers. This puts the infarcted left ventricle at an undesirably low point on the Frank-Starling curve and results in a low cardiac output state with typically low pulmonary artery wedge pressure unless massive left ventricular dysfunction is also present.

The clinical manifestations of hemodynamically significant right ventricular infarction are the triad of hypotension, increased jugular venous pressure, and clear lung fields in a patient with an inferior wall infarction. A positive Kussmaul's sign (jugular venous distention on inspiration) may also be present. Patients with inferior wall infarction who initially have normal blood pressure but experience shock after small amounts of preload reducing agents, such as nitroglycerin or morphine sulfate, should also be suspected of having pronounced right ventricular involvement.

The simplest and most cost-effective screening test for right ventricular infarction is the V_{4R} electrocardiographic lead. ST-segment elevation in this lead, in the setting of suspected right ventricular infarction, has an associated predictive accuracy of approximately 80%.[10] Bedside echocardiography will demonstrate acute right ventricular dilatation (Fig. 4) and severe right ventricular dysfunction. The degree of left ventricular dysfunction varies, but typically it is not severe unless prior ischemic damage or infarction has occurred.

Most patients with right ventricular infarction have abnormally low left ventricular filling pressures, and this factor can be verified with Swan-Ganz catheterization and by response to fluid loading.[11] Typically, treatment involves volume loading with isotonic saline to achieve a pulmonary artery wedge pressure of 18 to 20 mm Hg. Occasionally, surprisingly large amounts of saline are needed—often several liters within the first hour—and this approach may be counterintuitive to many physicians who have not previously managed this complication of myocardial infarction. In contrast, administration of diuretic therapy or preload reducing drugs can be fatal in these patients. When volume loading alone is insufficient, the use of inotropic agents, such as dobutamine, has been recommended, although this strategy is not effective in all patients. Many patients with right ventricular dysfunction experience spontaneous improvement in cardiac output in 48 to 72 hours after the initial insult. Patients who do not respond to fluid loading and catecholamines, as well as those who seek medical treatment within 12 hours after onset, should be considered for reperfusion therapy, with either thrombolytic agents or, perhaps, preferably acute right coronary artery angioplasty. Those patients who remain in shock may benefit from the use of a right ventricular assist device, although this approach has not been formally tested.

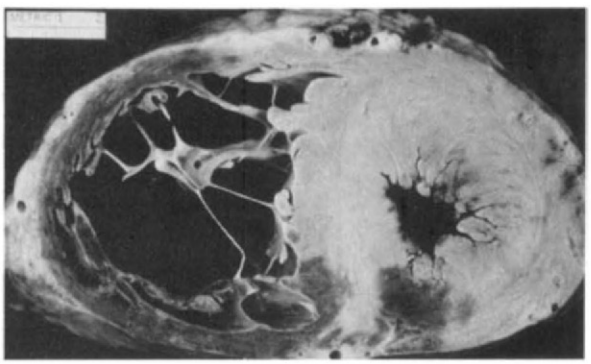

Fig. 4. Pathologic specimen, showing extensive right ventricular infarction associated with inferior wall infarction. Severe right ventricular failure caused patient's death. (Photograph courtesy of Dr. William D. Edwards. From Reeder GS, Gersh BJ. Acute myocardial infarction. In: Stein JH, Hutton JJ, Kohler PO, O'Rourke RA, Reynolds HY, Samuels MA, et al, editors. Internal Medicine. 4th ed. St. Louis: Mosby-Year Book, 1994: 169-189. By permission.)

CONCLUSION

The mechanical complications of acute myocardial infarction are important because they represent a correctable cause of acute hemodynamic deterioration and are often fatal if untreated. Sudden hemodynamic deterioration, coupled with the clinical clues as previously described, should heighten the physician's suspicion that a complication has occurred and lead to rapid diagnostic testing, usually with bedside echocardiography. In hospitals where this study is unavailable, patients should be transferred immediately to a tertiary center where appropriate testing and corrective surgical treatment can be efficiently performed.

ACKNOWLEDGMENT

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Questions About Myocardial Infarction *(See article, pages 880 to 884)*

1. Which *one* of the following is *most* responsible for in-hospital deaths due to myocardial infarction?
   a. Arrhythmias
   b. Pump failure due to extensive myocardial necrosis
   c. Myocardial rupture
   d. Acute mitral regurgitation
   e. Right ventricular infarction
2. Which *one* of the following is *true* regarding papillary muscle rupture in acute myocardial infarction?
   a. Occurs only with Q-wave myocardial infarction
   b. Occurs with equal frequency in anterior and inferior wall infarctions
   c. Involves principally the anterolateral papillary muscle
   d. May occur with either Q-wave or non-Q-wave myocardial infarction
   e. Always necessitates valve replacement
3. Which *one* of the following statements is *most* applicable to hemodynamically significant right ventricular infarction?
   a. Patients cannot lie supine because of high left-sided filling pressures
   b. Diuretic agents are the treatment of choice initially
   c. Dobutamine infusion should be given as the first step in treatment
   d. Electrocardiography with the V_{4R} lead is the most cost-effective screening test
   e. Echocardiography is the most cost-effective screening test
4. Which *one* of the following statements is *true* in patients with acute free-wall myocardial rupture?
   a. Has an associated fatality rate similar to that for right ventricular infarction
   b. Can be managed with prompt resuscitation, intra-aortic balloon pumping, and urgent operation in many patients
   c. May manifest as a subacute variant with pericardial pain and an area of loculated pericardial effusion on echocardiographic examination
   d. Occurs equally in non-Q-wave and Q-wave myocardial infarction
   e. Frequently occurs 2 to 4 weeks after acute myocardial infarction
5. Which *one* of the following statements is *false* regarding shock in the setting of acute myocardial infarction?
   a. Has an associated historical mortality of approximately 80%
   b. Demands a search for a correctable mechanical complication
   c. Occurs in 20% of acute myocardial infarctions
   d. Is probably an indication for immediate angioplasty, if within 12 hours after onset and no mechanical complication is found
   e. Is more common with anterior than inferior myocardial wall infarction

Correct answers:

1. *b*, 2. *d*, 3. *d*, 4. *c*, 5. *c*