The Echocardiographic Diagnosis of Rupture of a Papillary Muscle*

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The echocardiographic diagnosis of acute rupture of a papillary muscle is described. The pertinent findings included (1) decreased systolic motion of the posterior wall, (2) exaggerated septal motion, (3) left ventricular enlargement and pattern suggesting left ventricular diastolic overload, and (4) bizarre fluttering of posterior leaflet of the mitral valve in diastole, suggesting an unhealing of the mitral valvular apparatus. The echocardiogram is a useful noninvasive tool in the diagnosis of this often fatal complication of myocardial infarction.

Rupture of a papillary muscle is a catastrophic complication of myocardial infarction. Differentiation between dyskinesia and necrotic rupture of the papillary muscle is important, since the latter condition requires urgent surgical correction. The echocardiographic features of this entity will be outlined in a patient seen at our institution.

CASE REPORT

A 70-year-old white man entered the Jewish Hospital of St. Louis in November 1975, with an extensive myocardial infarction involving the inferior, posterior, and lateral walls. At that time a grade 1-2/6 apical systolic murmur was noted and was attributed to papillary muscular dysfunction. During his stay in the hospital, the patient had recurrent angina, intermittent atrial flutter with a rapid ventricular response, and left ventricular failure. His condition stabilized initially with therapy with digitalis and diuretic drugs; however, five days later, the apical systolic murmur increased in intensity and became pansystolic. Extrarenal azotemia developed, with the blood urea nitrogen level (BUN) rising from 28 mg/100 ml to 59 mg/100 ml. Thereafter, urinary output diminished, and left ventricular failure developed and became persistent.

Twelve days following admission, a chest x-ray film revealed severe pulmonary edema. The BUN at this point was 129 mg/100 ml. An echocardiogram (Fig 1) demonstrated marked diastolic flitting of the posterior leaflet of the mitral valve and exaggerated systolic septal motion. The calculated stroke volume was 61 ml, the ejection fraction was 34 percent, and the cardiac output was 6.3 L/min. The findings were considered diagnostic of rupture of a papillary muscle. An intra-aortic balloon was inserted, and emergency cardiac catheterization and coronary angiographic studies were performed by the Judkin's technique from the right femoral artery. The study confirmed the diagnosis of massive mitral insufficiency and demonstrated proximal occlusion of a large circumflex coronary artery, with insignificant disease in the left anterior descending and right coronary vessels.

At surgery, the posteromedial papillary muscle was ruptured. The mitral valve was excised and was replaced with a 29-mm Björk-Shiley prosthesis inserted in the subannular position. At the completion of the procedure, the patient could not be weaned from cardiopulmonary bypass, despite massive support with vasopressor drugs and the intra-aortic balloon. Permission for autopsy was not granted.

DISCUSSION

Acute papillary muscular rupture has been the subject of several recent reviews.1-3 It is estimated that less than 1 percent of acute myocardial infarctions are associated with this complication. Prompt surgical correction is recommended, since the associated massive volume overloading of the left ventricle is usually fatal. On the other hand, papillary muscular dysfunction is often associated with less severe degrees of mitral insufficiency, permitting conservative medical management in many cases. It is often difficult to distinguish between severe dyskinesia and rupture of a papillary muscle. Rarely, the two may coexist. Given the usually fatal outcome in patients managed medically, the results of replacement or reconstruction of the mitral apparatus are acceptable, with a reported operative mortality in the range of 50 percent. Early intervention and prevention of irreversible left ventricular failure may further improve these statistics.4

To achieve this, a rapid means of diagnosis is crucial. Ventricular angiography is a definitive diagnostic procedure, but selection of patients requires care and discrimination, due to the considerable risk of invasive procedures in this setting. Pulmonary capillary wedge pressure obtained at the bedside with the Swan-Ganz catheter has been very helpful in the diagnosis of papillary muscular rupture but also has certain limitations.5 First, measurement of pulmonary capillary wedge pressure may be inaccurate in states of low output and is occasionally impossible to obtain. Second, it is often difficult to tell the difference between pulmonary arterial pressure and markedly elevated veins due to acute mitral regurgitation. Finally, pulmonary capillary wedge pressure may not always, for a variety of reasons, reflect true left atrial pressure.

A noninvasive technique that will accurately diagnose papillary muscular rupture would be of immense clinical benefit. The echocardiogram in this case helped to make a precise anatomic diagnosis at no risk to the patient. The findings which confirmed papillary muscular rupture included (1) decreased motion of the posterior wall, reflecting the site of the infarct, the usual location for papillary muscular rupture, (2) left ventricular dilation, with exaggerated septal motion and a high calculated cardiac output compatible with left ventricular diastolic overload, and, finally, (3) biz-
arre fluttering of the posterior leaflet of the mitral valve in diastole, suggesting an unhinging of the mitral valvular apparatus. These findings are in some ways similar to those reported in rupture of the chordae tendineae. A flail posterior leaflet of the mitral valve is characterized by increased excursion of the mitral valve, a relatively anterior or middle position of the posterior leaflet of the mitral valve in diastole, and a marked systolic prolapse of this same posterior leaflet. These findings could be a result of rupture of either the chordae tendineae or the posteromedial papillary muscle following myocardial infarction; however, the two conditions differ in the pattern of motion of the posterior wall, which is exaggerated in the former condition, but is decreased, absent, or even paradoxical in the latter. The expected marked systolic prolapse of the mitral valvular leaflet in systole was not seen in this case.

An echocardiogram should be obtained from all patients with myocardial infarction who develop a systolic murmur or whose condition deteriorates despite adequate medical treatment. Findings consistent with papillary muscular rupture prompt urgent cardiac catheterization with coronary angiographic studies, followed by appropriate surgical management.

**ADDENDUM**

Since writing this report, we have seen another patient with essentially similar findings. A 66-year-old white man developed rupture of the posterior papillary muscle following acute inferior myocardial infarction. The echocardiogram showed a dilated left ventricle, a hyperkinetic septum, a poorly moving posterior left ventricular wall, and a flail posterior leaflet of the mitral valve, showing a neutral position of this posterior leaflet in diastole and marked posterior prolapse in systole. A diagnosis of rupture of the posteromedial papillary muscle was made on the basis of these echocardiographic findings and was confirmed at operation.

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**Figure 1.** A (top), Echocardiogram showing hyperdynamic motion of interventricular septum (IVS), poorly moving left ventricular posterior wall (LVP), enlarged left ventricle (LV), and bizarre fluttering motion of posterior leaflet of mitral valve in diastole, as shown by arrows. B (bottom), Enlarged view, highlighting fluttering motion of posterior mitral valvular leaflet (PMVL), as indicated by arrows. Left ventricular posterior wall (LVP) is hypokinetic. RV, Right ventricle; and AMVL, anterior mitral valvular leaflet.
Intravenous Self-Administration of Metallic Mercury in Attempted Suicide*

Report of a Case with Serial Roentgenographic and Physiologic Studies over an 18-Month Period

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This report describes a 23-year-old white man who injected metallic mercury from a thermometer into his antecubital vein in an attempt at suicide. Despite the persistence of mercury throughout both pulmonary fields on chest x-ray films over an 18-month period of observation, no clinical or physiologic derangement of pulmonary function has developed. In addition, no clinical or biochemical evidence of acute or chronic mercury poisoning in any other organ has appeared during these 18 months, even though metallic mercury is seen in the abdominal viscera on roentgenographic examination. The literature on suicidal and accidental poisoning with metallic mercury is reviewed.

Poisoning with metallic mercury, acquired both accidentally and following attempted suicide, is extremely rare. Since the beginning of this century, about 28 cases (with four deaths) have been reported. Attempts at suicide with metallic mercury have been reported in three such cases. Only one had a fatal outcome. A recent case report has described the intentional intravenous injection of metallic mercury in a boxer from Latin America, where such a practice is occasionally employed in the hope of enhancing athletic prowess. Accidental poisoning with mercury has occurred in many different ways, including rubbing mercury and mercurial ointments into wounds, tattooing, rupture of the mercury-filled bag of an indwelling intestinal tube, injury from a broken thermometer, and working with this element in industry. However, of particular importance is the observation that over the past 30 years, since metallic mercury replaced mineral oil as the anaerobic seal and mixing agent in syringes, most of the cases of accidental embolization of and poisoning with metallic mercury have occurred during the sampling of blood for analysis of blood gas levels.

In contrast to poisoning with liquid metallic mercury, the toxicity of metallic mercury vapors and salts has long been recognized in a variety of occupations. Long-term intoxication has been noted in hatters and furriers, resulting in a severe chronic brain syndrome, the "mad hatters." Short-term inhalation of mercury is rare and has been reported in patients involved in industrial accidents resulting in either sudden exposure to high concentrations of mercury vapor or to small amounts of mercury vapor in closed spaces. Clinically, these patients have been reported to experience cough and pharyngeal ulcerations as the most frequent manifestations. Less commonly, fever, pain in the chest and abdomen, dyspnea, vomiting of material with a metallic taste, and central nervous system symptoms, especially headache, have been noted.

The case we are reporting is of interest in that over an 18-month period of observation and study, no symptoms, signs, or discernible biochemical or physiologic abnormalities could be demonstrated either in the lungs, liver, gastrointestinal tract, or kidneys. No comparable observations have been reported.

Case Report

The patient is a 23-year-old white man who was receiving psychiatric care for depression and was first seen a few hours after an attempt at suicide during which he broke the tip of a mercury thermometer, punctured the antecubital vein of his left arm, and injected the mercury intravenously. The patient appeared to be in no acute distress. The findings from physical examination were unremarkable, as were also his complete blood cell count, the results of urinalysis, the levels of blood urea nitrogen and creatinine, the serum levels of electrolytes, the serum levels of calcium and phosphorus, and the results of studies of hepatic chemistry. An x-ray film of the chest (Fig 1 and 2) revealed the presence of metallic mercury in both pulmonary fields, more marked at the bases. No abdominal plain x-ray film was obtained when the patient was first seen.

Tests of pulmonary function were performed a week later, including a 14-minute graded treadmill exercise tolerance test, determination of pulmonary volumes, spirometric testing, studies of intrapulmonary gas distribution, analysis of the carbon monoxide diffusing capacity employing the single-breath method and of the mechanics of ventilation (consisting of pulmonary compliance and plethysmographic airway resistance), and analysis of arterial blood gas levels measured both at rest and following exercise. All results were within


References


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