Ruptured Papillary Muscle

Report of a Case with Successful Mitral Valve Replacement

By W. Gerald Austen, M.D., Charles A. Sanders, M.D., James H. Averill, M.D., and Allan L. Friedlich, M.D.

Rupture of a papillary muscle is a rare complication of myocardial infarction.\(^1\)\(^2\) The combination of coronary artery disease with severe mitral regurgitation usually results in a rapid, deteriorating course. Not until 1948 was this diagnosis correctly made ante mortem.\(^3\) Sanders et al., in 1957, discussed 61 cases of rupture of a papillary muscle and pointed out the important clinical features of this diagnosis.\(^4\) A number of other reports have appeared.\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\)

With presently available open-heart techniques, mitral regurgitation has become a correctable lesion. The present report concerns a patient with massive mitral regurgitation due to rupture of a papillary muscle in whom successful mitral valve replacement was accomplished.

Case Report

MGH no. 131-58-79. This 51-year-old plumber had had no cardiac difficulty until 2 1/2 months prior to admission, when he developed anterior chest pain and was admitted to another hospital with the diagnosis of myocardial infarction. At that time, the electrocardiogram suggested an anterolateral myocardial infarct of uncertain age. Physical examination revealed a faint apical systolic murmur. The patient did well and was discharged home. However, 3 weeks after his initial attack, he was again hospitalized, critically ill, with acute pulmonary edema. Treatment with oxygen, phlebotomy, sodium restriction, digitalis, and diuretics resulted in considerable temporary improvement, but subsequently he again developed progressively increasing dyspnea and physical findings of severe congestive failure. He was referred to the Massachusetts General Hospital by Drs. James Averill and Leo Moreau because of intractable congestive failure and the suspicion that rupture of a papillary muscle was the cause.

On admission the patient was an extremely ill man, pale, slightly cyanotic, and almost fainting when he sat up in bed. The blood pressure was 90/70 mm. Hg, although on some occasions it was not obtainable. There were findings of obvious congestive failure with pulsating neck veins to the angle of the mandible when he was in an upright position, mild sacral edema, and fine rales at the left lung base. A heaving left ventricular impulse was palpable in the midaxillary line. The sound of pulmonic valve closure was increased. There was a slightly musical grade-IV pansystolic murmur at the apex transmitted to the back. Both right and left ventricular gallops were present. The left lobe of the liver was enlarged to the level of the umbilicus.

Pertinent laboratory studies included white blood-cell count of 13,700. The hemoglobin was 10 Gm. The LDH was 378 units and the SGOT 270 units. Chest films and fluoroscopy revealed marked cardiac enlargement, particularly of the left ventricle (fig. 1). The left atrium was not significantly enlarged. There was marked pulmonary congestion and a little pleural fluid in each costophrenic angle. The electrocardiogram (fig. 2) demonstrated predominant R waves over the right precordium which, because of the clinical history and previous tracings, were thought to be due to lateral myocardial infarction rather than to right ventricular hypertrophy. Inversion of the T waves in the right precordial leads, however, was attributed to right ventricular strain.

Initially the patient was vigorously treated for congestive failure but failed to improve. The urgency of the primary problem prevented investigation of low-grade fever and anemia. Four blood cultures were negative and there were no other indications of bacterial endocarditis as evidence favoring ruptured chordae tendineae. The question of pulmonary embolus as an additional complication was raised. Diagnostically, consideration was given to the possibilities of

From the Departments of Surgery and Medicine, Harvard Medical School and the General Surgical and Medical Services, Massachusetts General Hospital, Boston, Massachusetts.

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lateral myocardial infarction with (1) marked left ventricular dilatation and functional mitral regurgitation, (2) malfunction of a papillary muscle, and (3) rupture of the anterolateral papillary muscle. The last seemed most likely.

A right heart catheterization was performed on the fifth hospital day (fig. 3). This revealed a mean pulmonary wedge pressure of 28 mm. Hg with “V” waves rising to 56 mm. Hg. The pulmonary artery pressure was 64/25 mm. Hg, with a mean of 43 mm. Hg. The right ventricular pressure was 64/4-15 mm. Hg, and right atrial mean pressure was 15 mm. Hg indicating right ventricular failure. The cardiac index was markedly reduced to 1.3 L./min./M.² and the pulmonary arteriolar resistance was calculated to be 13 units (normal, 1 to 4 units). A right ventricular angiocardiogram revealed no evidence of pulmonary embolus. The left atrium was only slightly enlarged but there was marked enlargement of the left ventricular cavity.

Since catheter study supported the presence
of marked mitral regurgitation, the patient was operated on the following day. The mitral valve was approached through the left chest with resection of the fifth rib. Venous drainage for cardiopulmonary bypass was accomplished by cannulation of the left femoral vein and the right ventricular outflow tract. Arterial inflow was accomplished through the right femoral artery. Prior to perfusion, palpation of the valve substantiated massive regurgitation through the anterior portion of the mitral valve. It was possible to feel the ruptured anterior papillary muscle and the resultant flail anterior portions of the mural and aortic leaflets. Cardiopulmonary bypass was then instituted. Through a left atriotomy, the mitral valve was exposed and the ruptured anterior papillary muscle was observed. The mitral valve and attached papillary muscles were excised and a no.-3 Starr-Edwards mitral valve prosthesis was inserted. The atriotomy was closed in the usual fashion and the patient was taken off cardiopulmonary bypass. Cardiac action and myocardial color were considerably improved after valve replacement.

Pathologic examination revealed a normal mitral valve with infarction and rupture of the anterior papillary muscle (fig. 4).

The patient’s postoperative course was smooth. The systolic blood pressure was in the range of 100 mm. Hg for the first 4 days after surgery but subsequently rose to a level of 120 mm. Hg. On the first postoperative day he was stronger than before operation and no longer dyspneic. By the fifth postoperative day venous distention and enlargement of the liver had disappeared. A spontaneous diuresis resulted in a 23-pound weight loss. Some time was required to regulate anticoagulant therapy and he was discharged on the twenty-first day following surgery. By this time the heart had decreased 1.5 cm. in size on x-ray and the electrocardiogram no longer showed the T-wave inversions suggesting right ventricular strain. He was able to climb stairs without dyspnea or chest pain.

The patient was seen 2 and 5 months postoperatively at which time his medications included digitoxin and Coumadin and he was on a no-added-salt diet. He was normally active with no dyspnea on climbing stairs or walking up to one-half mile. There were no physical signs of congestive failure. A chest film (fig. 5) showed further decrease in heart size.

**Comments**

This case illustrates many of the clinical features previously described in rupture of a papillary muscle, although the time of myocardial infarction and of rupture of the papillary muscle is not clearly defined in this patient. Usually the latter is associated with severe pulmonary edema and the sudden appearance of an apical systolic murmur.1–4, 7, 8, 10 The murmur, however, is not an invariable accompaniment, occurring in only half the reported cases.2–4 An associated thrill is rare.5 The electrocardiogram may be helpful, since it usually fails to show evidence of
fresh myocardial infarction to explain the rapid clinical deterioration.\textsuperscript{4} Chest films demonstrate the typical features of left ventricular failure.\textsuperscript{4} In contrast, the finding of a paradoxically pulsating small left atrium by fluoroscopy or cineradiography suggests mitral regurgitation of recent onset. A tall “V” wave in the pulmonary wedge or left atrial pressure tracing, as seen in the present case, provides further evidence of mitral regurgitation which is out of proportion to that generally seen as a result of left ventricular failure alone.\textsuperscript{13}

Many patients die within a few hours

\textbf{Figure 4}

\textit{Sections of ruptured papillary muscle. Left. Hematoxylin and eosin stain × 12. Necrotic papillary muscle with origins of chordae tendineae at top of figure. Right. Hematoxylin and eosin stain × 140. Fibrous organization of necrotic papillary muscle.}

\textbf{Figure 5}

\textit{Chest roentgenograms 2 months postoperative.}
RUPTURED PAPILLARY MUSCLE

following papillary muscle rupture. The surviving patients are likely to develop congestive failure that is refractory to medical treatment, and death usually results within a few weeks or months.4,9

The posterior papillary muscle is the one most commonly ruptured and this follows posterior or inferior myocardial infarction. Rupture of the anterior papillary muscle, which occurred in the present case, is encountered infrequently and rupture of a papillary muscle in the right ventricle has been reported only rarely.1-4,7-10

Surgical correction of mitral regurgitation due to a ruptured papillary muscle has only occasionally been attempted12 and, to our knowledge, no successful repair has been reported. The present case is a notable exception. The correct diagnosis was suspected clinically and was supported, though not proved, by right heart catheterization. Mitral valve replacement with cardiopulmonary bypass was successful and has resulted in a complete clearing of what had been a moribund state of intractable congestive failure.

Summary

A case of severe mitral regurgitation secondary to myocardial infarction and rupture of the anterior papillary muscle is presented. Mitral valve replacement resulted in striking improvement.

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References


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W. GERALD AUSTEN, CHARLES A. SANDERS, JAMES H. AVERILL and ALLAN L. FRIEDLICH

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