Rupture of a Papillary Muscle of the Tricuspid Valve Following Acute Myocardial Infarction

Report of a Case

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Rupture of the heart is a comparatively frequent complication of myocardial infarction. Maher, Mallory, and Laurents determined that rupture accounts for 9 to 11 per cent of all deaths following acute infarction, and that the most common site is through the ventricular wall. Rupture of a papillary muscle is far less common; Sanders et al., in 1957, were able to find only 56 reported cases to which they added five additional cases. Of these, however, 48 were due to myocardial infarction and all of these were present on the left side of the heart. Three additional cases have since been reported. This does not represent a true incidence, however, for many cases obviously remain unreported.

We have recently had the opportunity to observe an instance of rupture of a tricuspid papillary muscle as a result of myocardial infarction. To our knowledge, no such case has previously been reported in the English literature.

Clinical Summary

A 72-year-old white widow was admitted with progressively increasing shortness of breath of 10 days' duration.

Several months prior to admission, the patient developed dyspnea on exertion, fatigue, and angina pectoris. Ten days prior to entry, she developed palpitation and marked congestive heart failure, with pulmonary congestion; this increased in severity two nights prior to admission. Because of the progression of her symptoms, she was admitted to the hospital.

The patient had had hypertension of 200-240 systolic, 110-130 diastolic, for the past 18 years, and had been treated with Rauwolfia 6 years previously. Blood pressure readings since then had averaged 170/100.

Physical examination revealed an obese woman sitting up and exhibiting Cheyne-Stokes respiration. The fundi showed flat discs with arteriovenous compression and tortuous vessels without hemorrhages or exudates. There was distention of the cerebral veins at 60 degrees. There was dullness to percussion at the posterior bases with diminished breath sounds and rales. The point of maximal cardiac impulse was in the sixth intercostal space at the anterior axillary line. There was a grade-II harsh pansystolic murmur heard best at the lower left sternal border. A protodiastolic gallop was heard at both the apex and the left sternal border. The rate was regular, and the sounds were of good quality. The second aortic sound was louder than the second pulmonic sound. The liver edge was palpated 10 cm. below the right costal margin; no other organs or masses were felt. There was 4+ pitting edema of the feet and 2+ edema of the sacrum. The neurologic examination was normal. The blood pressure was 175/120, the pulse 108, the respiratory rate 35, and the temperature 100.8, rectally.

The admission and subsequent urinalyses, as well as hemoglobin and hematocrit, were normal. The erythrocyte sedimentation rate was 47 mm./hour. The white-cell count was 11,500, with 86 per cent neutrophils. Prothrombin, blood urea nitrogen, creatinine, total protein, bilirubin, and electrolytes were normal throughout the course. The serum glutamic oxaloacetic transaminase was 30 units on admission, rose progressively to 275 units on the fifth hospital day, and then began gradually to fall. The serum lactic dehydrogenase level was 273 units on admission, rising to

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1,385 on the sixth hospital day, and then gradually declining to 280 units. The serum glutamic pyruvic transaminase level on admission was 18 units, rising to 135 units on the eighth hospital day. The blood Hinton test was negative. Decholin circulation time was 33 seconds, and the venous pressure was 155 mm. of water on admission.

The electrocardiogram on admission revealed sinus tachycardia at a rate of 120 with a PR interval of 0.17 and QRS interval of 0.10 second. There were large S waves in V<sub>E</sub>, V<sub>1</sub>-V<sub>5</sub>; large R waves in V<sub>5</sub>; depressed ST segments in aV<sub>R</sub>, V<sub>R</sub>, V<sub>5</sub>R, V<sub>1</sub>, and V<sub>2</sub>; biphasic T waves in V<sub>3</sub>-V<sub>6</sub>. The next electrocardiogram, on the tenth hospital day, showed slurred S waves in lead I, large Q waves in lead III, Q waves in aV<sub>F</sub>, QS deflections in leads V<sub>1</sub> and V<sub>5</sub>R, depressed ST segments in leads I, II, aV<sub>L</sub>, V<sub>5</sub> and V<sub>6</sub>, scooped ST segments in V<sub>6</sub> and elevated ST segments in leads III, aV<sub>R</sub>, V<sub>1</sub>-V<sub>4</sub>.

Roentgenogram of the chest on admission revealed left ventricular dilatation, bilateral pleural effusions, and vascular engorgement of the lung. Subsequent films on the fourth and eighth days showed progression of the engorgement but were otherwise unchanged.

The patient was placed on a low-salt diet, bed rest, digitoxin 0.1 mg. daily, and oxygen by mask. Five hundred milliliters of slightly turbid, straw-colored fluid were removed from the right pleural cavity. On the second hospital day, peripheral edema had decreased and one observer heard a harsh loud, grade-II/III (IV) systolic murmur at the apex and a harsh, soft, grade-I/II (IV) systolic murmur at the aortic area. Both these murmurs had been heard in the past.

On the fourth hospital day, the patient was drowsy and less responsive. In addition to the protodiastolic gallop, an intermittent fourth heart sound was heard near the apex. A second right thoracentesis of 180 ml. was performed at this time. The next morning, the patient was disoriented and lethargic. Electrocardiogram revealed third-degree heart block, nodal beats, and low atrial and ventricular beats. Digitoxin was discontinued, and, 3 days later, there was a return to normal sinus rhythm. On the tenth hospital day, digoxin 0.25 mg. twice a day was started. Six days later, the patient was again confused and having hallucinations. The physical findings were unchanged except for a bradycardia of 50: an electrocardiogram 2 hours later revealed an irregular supraventricular rhythm without good P waves, interpreted as probable fibrillation. The patient began to cough and was febrile. Rales and irregular rhythm persisted. Chest roentgenogram at this time revealed increased engorgement and fluid accumulation. Tetracycline therapy was started to no avail and she died on the twentieth hospital day.

**Pathologic Examination**

The postmortem examination was limited to the chest organs. There were 4+ pretibial and presacral edema and marked peripheral cyanosis. Six hundred milliliters of amber turbid fluid were found in the right pleural cavity; the left pleural cavity and the pericardial cavity contained no increased amount of fluid. The coronary arteries showed severe, diffuse arteriosclerosis with multiple focal narrowings but no occlusion. The heart weighed 400 Gm. and showed generalized hypertrophy and dilatation, especially on the left side. The valves were normal. In the left ventricle there were a healed apical infarct with mural thrombus and a recent transmural infarction, 6 by 7 cm., involving the posterior basal half of the interventricular septum, the posterior wall, and part of the lateral wall of the ventricle. A small organizing infarct, about 0.5 cm. in diameter, was also found on the anterior wall of the right ventricle.

A septal papillary muscle, 1.5 by 0.4 cm. with its chordae tendineae intact, was lying free in the right ventricle. The muscle was yellowish-white and the free end was irregular and pale gray.

The right lung weighed 460 Gm. and the

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**Figure 1**

The free edge of the ruptured tricuspid papillary muscle. Mallory PTAH stain; × 20.
left 210 Gm. There was massive infarction of the right middle and lower lobes. No infarction was present on the left. The main arteries to the right middle and lower lobes, and their subdivisions, were occluded by thromboemboli. There was also focal segmental and subsegmental arterial thromboembolism in the left lung.

Microscopic examination of the heart revealed at least three different stages of infarction. The oldest one of several months’ duration was found at the apex. An acute infarct of a few days’ duration with necrosis, disintegrated neutrophils, and only slight peripheral organization was found in the center of the posteroseptal infarct. Part of the posteroseptal infarct and the isolated right ventricular infarct were characterized by rich mononuclear infiltrate, many fibroblasts, pigmented macrophages, and newly formed capillaries and collagen, estimated to be about 4 weeks old. The ruptured papillary muscle was in this area. Its free portion (fig. 1) was composed of necrotic muscle fibers and disintegrated neutrophils and was covered by endothelialized fibrin clot which contained many fibroblasts, capillaries, and newly formed collagen. The portion of the papillary muscle still attached to the septum (fig. 2) was similarly covered by partially organized fibrin.

Discussion

Dated by the pathologic examination, the rupture of the papillary muscle occurred about 4 weeks prior to death, and, hence, about a week prior to the final admission. Part of the large posterolateral and the right ventricular infarcts occurred at about the same time. The sudden appearance of severe, predominantly right-sided heart failure as evidenced by peripheral edema, prolonged circulation time, venous engorgement, high venous pressure, and enlarged liver, as well as the initial murmur best heard along the left sternal border, also indicate an episode of tricuspid insufficiency. However, this picture was masked by pulmonary edema and was further complicated by the extension of the myocardial and pulmonary infarcts.

Although rupture of many other areas of the heart following acute myocardial infarction has been reported previously, the present case appears to be the first instance reported in the English literature of a rupture of a tricuspid papillary muscle due to infarction. Rupture of a papillary muscle of the tricuspid valve due to other causes has been reported on four occasions; all of these occurred in young people between the ages of 20 and 30 years, and were the result of bacterial endocarditis in three and trauma in a fourth.

The rarity with which an infarcted tricuspid papillary muscle ruptures is probably attributable to several factors. Of 160 cases of myocardial infarction studied by Wartman et al., the incidence of right ventricular infarction was only 13.8 per cent. Most of these cases represented predominantly left ventricular and septal infarctions with extension to the right ventricle, and only a few were isolated right ventricular infarcts. When one considers that rupture of a mitral papillary muscle occurs in less than 1 per cent of acute myocardial infarctions, and that the systolic pressure gradient of the right ventricle is only a fraction of that on the left, rupture of an infarcted right papillary muscle might be expected to occur with extreme rarity indeed.

Figure 2
The septal base of the ruptured papillary muscle. Mallory PTAH stain; × 20.
An additional protective factor lies in the anatomic arrangement of the tricuspid papillary muscles. The large anterior and small conus papillary muscles of the tricuspid valve are relatively constant in position, but the posterior muscle, which arises from the diaphragmatic wall of the right ventricle, is represented by a group of muscles that are inconstant in number and position. Furthermore, each of the papillary muscles attaches to two adjacent leaflets of the tricuspid valve, and, even should rupture occur, the resultant tricuspid insufficiency might not be clinically significant unless dilatation of the annulus supervened. Clinical signs of rupture of a mitral papillary muscle, with the pulmonary capillary bed behind the insufficient valve, are likely to be much more dramatic.

Summary
A case of rupture of a tricuspid papillary muscle due to myocardial infarction, apparently the first to be reported in the English literature, is presented. Anatomic, physiologic, and pathologic reasons for the rarity of such a rupture are advanced.

References

Clinical Observations
The great Book of Nature, which is alike open to all, and is "incapable of deceiving," I have hourly read, and I trust not wholly in vain. During the first twelve or fourteen years of my professional life, I recorded almost every case which occurred to me either in private practice, or in the chief conduct of an extensive charity. When afterwards the multiplication of common examples seemed to me an unnecessary waste of inestimable time, which might be much more profitably employed, I contented myself with the more useful task of recording chiefly such cases, or, on a few occasions, such particular circumstances only of cases, as led to the establishment of principles. This I have generally done on the spot, or rarely deferred beyond the day of observation, always rejecting what, on repeated and varied inquiry, I have not been able fully to verify.—Preface. Collections from the Unpublished Medical Writings of the Late Caleb Hillier Parry, M.D.F.R.S. Vol. I., London, Underwoods, Fleet-Street, 1825, p. 47.
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