Rupture of a Papillary Muscle of the Heart; Report of Two Cases

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The paper presents clinical and pathological data on 33 cases of ruptured papillary muscle of the heart as revealed by review of the literature. All of the known causes of this event are listed and the characteristic clinical features are emphasized by including two additional typical case reports.

According to Stevenson and Turner, the first case of rupture of a papillary muscle of the heart was reported by Merat in 1893. Since then only 33 cases have been reported and all but 2 of these reports have been recently summarized in a review of the literature by Davison. Davison found 3 instances of papillary muscle rupture in 14,000 autopsy subjects examined at the Mount Sinai Hospital in New York City. Stevenson and Turner found 2 such cases in 6,000 autopsy subjects examined at the Johns Hopkins Hospital in Baltimore. Review of 10,500 autopsies recorded at the Institute of Pathology, University Hospitals of Cleveland, revealed one case of papillary muscle rupture. This is one of the cases reported in this article. The second case is taken from the autopsy records of the Department of Pathology of Cleveland City Hospital.

Case Reports

Case 1—The patient was an Italian laborer, 56 years old, who was well until November, 1937. At this time he experienced nonradiating severe substernal chest pain of sudden onset and pronounced shortness of breath. There were moderate cyanosis and rales throughout both lungs. The patient remained in bed for one week following the onset of symptoms. Thereafter, he complained of no symptoms and was able to do light work. On February 21, 1938, he suddenly experienced extreme shortness of breath accompanied by a sense of marked apprehension. He was put to bed and his condition did not change until the next morning when he complained of pain in the left arm. By 7:00 p.m. he had become comatose. He was admitted to University Hospitals of Cleveland at noon on February 23, 1938.

Physical examination revealed a well-developed, comatose white man who was markedly cyanotic, perspiring profusely, and breathing rapidly. The temperature was 40°C., the pulse rate 158, and the respiration rate 44. The blood pressure was 110/92. Auscultation of the chest revealed stertorous breath sounds, and fine rales were present over the posterior bases of the lungs. Percussion revealed the left border of cardiac dullness to extend 12 cm. from the mid-sternal line in the sixth intercostal space. The heart beat was rapid and regular. A harsh systolic murmur was present over the apex of the heart and was somewhat obscured by the breath sounds. The abdomen was soft and no masses were palpated. The remainder of the physical examination revealed no abnormalities.

Hematologic examination revealed 19.6 grams of hemoglobin. There were 6,700,000 erythrocytes and 18,000 leukocytes per cu. mm. of blood. Differential count of 100 cells revealed 70 segmented granulocytes, 18 band cells, 10 lymphocytes, and 2 monocytes. The urine was brown, clear, and contained a trace of albumin. The blood Wassermann reaction was negative. An electrocardiogram made on the day of admission was thought to give evidence of a recent and old infarct of the posterior wall of the left ventricle.

The patient was promptly placed in an oxygen tent but remained unconscious. By 3:00 p.m. on the day of admission the pulse and blood pressure were unobtainable. Gasping, labored respirations developed and the patient died at 2:00 a.m. on February 24, 1938.

Autopsy Report (6118): The heart weighed 500 grams. The epicardium was smooth and transparent except over the posterior surface of the left ventricle at the apex where a region measuring approximately 5 by 3 cm. was discolored gray and slightly roughened by fibrous tags. The underlying myocardium was grayish white and thin, measuring, in one region, 8 mm. in thickness. A narrow zone of muscle at the periphery of this region was soft and discolored dark purplish-red. Elsewhere the myocardium was moderately firm and light brownish red throughout. The left posterior papillary muscle arose from the center of the thin portion of the ventricle.

From the Institute of Pathology, Western Reserve University Hospitals, Cleveland, Ohio.
and was completely separated in its midportion by an irregular laceration. The surface of the segment arising from the ventricular wall was covered by a grayish-brown mural thrombus. The free portion was attached to the posterior mitral leaflet by twisted chordae tendineae which drew the muscle segment close to the free margin of the valve (fig. 1).

Branches of the right coronary artery extended down the interventricular groove and over the posterior wall of the left ventricle. Transverse sections of the right coronary artery at the acute margin of the heart revealed a large lumen occluded, except for minute central openings, for a distance of 1.5 cm., by firm gray tissue. Distal to this region the lumen was occluded for a distance of 1 cm. by a recent thrombus that was red mottled with brown. Transverse section of the left coronary artery revealed a patent lumen moderately narrowed by focal yellowish-gray intimal plaques.

The other pertinent gross diagnoses included bronchopneumonia, recent infarcts of the right and left kidneys, old infarct of the spleen, and moderate arteriolar nephrosclerosis. The gross diagnoses were confirmed by microscopic examination.

Comment: The stertorous character of the respirations was thought to obscure partially the harsh apical systolic murmur characteristic of ruptured papillary muscle.

Case 2.—The patient, a white laborer, 58 years old, was well until February 6, 1948, when he first experienced pain in the left side of his chest. The pain was of abrupt onset and radiated to the left shoulder, arm, and hand. It was intermittent and of about thirty minutes' duration with recurrence every three to four hours for the following three days. At 11:00 p.m. on February 9, 1948, the patient suddenly experienced a constant and severe substernal squeezing sensation associated with aching and numbness of the left arm and hand. He perspired freely, vomited several times, and became cold and apprehensive. He was admitted to Cleveland City Hospital at 2:00 a.m. on February 10, 1948, in a state of collapse.
Fig. 2.—The heart of Patient 2 opened for injection study of the coronary arteries shows rupture of the posterior papillary muscle of the left ventricle.

Fig. 3.—Roentgenogram of the heart of Patient 2 after injection of the coronary arteries with radio-opaque latex. An extensive thrombus occludes the lumen of the right coronary artery. (Appreciation is extended to Dr. Arthur Young for preparation of figure 3 and permission for its publication in this report.)
The pulse rate was 120, respiration rate 28, temperature 37.2°C, and blood pressure 80/50. Physical examination revealed an acutely ill white man who was apprehensive, pale, and slightly cyanotic. Examination of the heart revealed no enlargement. Although the cardiac sounds were barely audible, the rhythm was regular and a soft systolic murmur was heard over the apex of the heart. The lungs were clear to auscultation and percussion. The abdomen was soft and nontender. The remainder of the physical examination revealed no abnormality.

Hematologic examination revealed 12 grams of hemoglobin. There were 5,200,000 erythrocytes and 21,000 leukocytes per cu. mm. of blood. Differential count of 100 cells disclosed 84 segmented granulocytes, 14 lymphocytes, and 2 monocytes. Urinalysis was not remarkable.

Soon after admission the blood pressure dropped to 60/30 and then became unobtainable. The patient continued to vomit and died approximately one hour after entering the hospital.

Autopsy Record (17386): The heart weighed 420 grams. Palpation revealed a region of softening over the posterior wall of the left ventricle extending from the apex at its junction with the interventricular septum to within 4 cm. of the base. Section disclosed a light brownish-red, moderately firm myocardium except in the region of softening where the cut surface was dark brownish-red and dull. The posterior papillary muscle of the left ventricle arose from the center of the region of softening and was completely separated in its midportion by a ragged oblique laceration (fig. 2). The free segment was held to the mural endocardium by a short tendinous cord. A branch of the right coronary artery formed the posterior descending vessel. Transverse sections of the right coronary artery disclosed a large lumen completely occluded 4 cm. from its orifice by a firm, mottled, dark-red and gray thrombus that extended for 6 cm. along the course of the vessel (fig. 3). Transverse sections of the anterior descending and circumflex branches of the left coronary artery revealed the lumens of the first portion of each to be markedly stenotic because of yellowish-gray intramural deposits that cut with calcific hardness.

Histologic examination revealed a recent thrombus of the right coronary artery, marked arteriosclerosis with stenosis of the left coronary artery, recent infarcts of the posterior wall of the left ventricle, focal fibrosis of the anterior wall of the left ventricle, and marked passive hyperemia of the lungs.

Comment: The clinical manifestations of papillary muscle rupture were characteristic in that there was an abrupt increase in the severity of the angina and sudden onset of profound shock three days after the onset of the illness.

**Discussion**

Of the 33 reported cases of rupture of a papillary muscle, 18 were the result of thrombosis of a coronary artery with infarction of the myocardium. The posterior papillary muscle of the left ventricle was ruptured in 13 of the subjects, in 11 of whom there was a thrombus of the right coronary artery or circumflex branch of the left coronary artery. In 2 patients with rupture of the left posterior papillary muscle the thrombus was in the anterior descending branch of the left coronary artery. Rupture of the anterior papillary muscle of the left ventricle resulted from coronary artery thrombosis in 5 patients, in 3 of whom the thrombus occluded the circumflex or anterior descending branch of the left coronary artery. In 2 patients with rupture of the left anterior papillary muscle the thrombus was in the right coronary artery.

In two cases, reported in the years 1824 and 1865, there was rupture of a papillary muscle of the right ventricle. The first occurred in a white woman, 22 years old, with advanced pulmonary tuberculosis. Autopsy examination disclosed vegetations on the tricuspid valve leaflets, attached chordae tendineae, and ruptured papillary muscle. The second occurred in a white woman, 23 years old, who died seven weeks post partum with puerperal sepsis, peritonitis, and pneumonia. Autopsy disclosed vegetations on the leaflets of the tricuspid valve, attached chordae tendineae, and ruptured papillary muscle of the right ventricle.

There have been 2 cases reported, neither of which is included in the review by Davison, in which the papillary muscle rupture was caused by trauma. The patient reported by Glendy and White, a white seaman, 24 years old, died twenty-six hours after being run over by a truck. Autopsy examination revealed a purplish-red contusion on the anterior surface of the left ventricle. There was no penetrating wound of the external surface of the heart. A hemorrhagic laceration separated the base of the left anterior papillary muscle from its ventricular attachment. The coronary arteries revealed no thromboses or extensive ruptures. There were also a fracture of the left eleventh rib, bilateral bronchopneumonia, and extensive hemorrhagic contusions of the thorax and abdomen. The patient of Payne and Hardy, a white man 51 years old, was found
unconscious beside a moving conveyor belt which he had attempted to repair with a heavy stick. Death occurred approximately one hour later and autopsy examination revealed a fracture of the sternum and left third and fourth ribs and a contusion of the anterior wall of the left ventricle. The base of the left posterior papillary muscle was partially separated from the ventricular wall. The coronary arteries were intact and patent throughout.

One case, reported by Spaulding and Von Glahn,\(^5\) was thought to be due to syphilis. The patient was a Negro, 31 years old, who died suddenly during a recurrent episode of congestive heart failure. The blood Wassermann reaction was four plus. Autopsy revealed the posterior papillary muscle of the left ventricle to be ruptured and microscopic examination of the stump is reported to have revealed a central focus of coagulative necrosis surrounded by a thin subendocardial zone of fibrosis in which a Levaditi preparation revealed spirochetes. The coronary arteries were patent and the myocardium of the ventricle was of normal consistency and color. In addition, there was syphilitic aortic valvulitis with aortic insufficiency.

One case referred to by Davison, report of which has not yet been published,\(^2\) was thought to be due to polyarteritis nodosa. The patient was a white man, 57 years old, who died twenty-four hours after suddenly developing pulmonary edema. Examination disclosed a harsh systolic murmur. Autopsy examination revealed polyarteritis nodosa involving the heart, liver, and urinary bladder. There were multiple small hemorrhagic infarcts of the myocardium including that of the anterior papillary muscle of the left ventricle which was ruptured. The coronary arteries revealed slight arteriosclerosis but were patent throughout.

Of the 31 cases reported in which trauma was not the etiologic factor, a significant change in the patient's clinical condition indicating the probable time of papillary muscle rupture was described in 17.\(^2\) In 10 of these patients, death occurred in less than nine hours after this clinical change and none lived more than thirty-six hours thereafter. Death occurred in less than fourteen days in all but 2 of the remaining patients in whom the time of papillary muscle rupture was not ascertained. One patient lived twenty-one days after the onset of symptoms and the patient of Merat, according to Stevenson and Turner,\(^1\) lived twenty months after what was clinically thought to be the time of the papillary muscle rupture.

The clinical signs and symptoms preceding rupture of a papillary muscle are usually those of recent myocardial infarction. The rapidity with which death follows this event often precludes the clinical diagnosis. However, sudden increase in the severity of the angina, the appearance of a harsh apical systolic murmur, and profound shock suggest the diagnosis. Murmurs present before the rupture of the papillary muscle usually change in character and increase in intensity.\(^2\) The antemortem diagnosis of papillary muscle rupture was considered in only one of the reported cases.\(^2\)

The differential diagnosis includes rupture of the following structures: an aortic cusp, the interventricular septum, the ventricle, and the mitral chordae tendineae. Rupture of an aortic cusp is manifested by a high pulse pressure and the abrupt onset of a loud diastolic murmur over the aortic area.\(^6\) Rupture of the interventricular septum is distinguished by a left atrium of normal size and a thrill and systolic murmur, most pronounced over the third or fourth intercostal space, that is transmitted chiefly to the right.\(^6\) A large ventricular rupture causes sudden death. A small ventricular rupture may cause hemo-pericardium and cardiac tamponade manifested by a low pulse pressure and a small silent heart.\(^7\) Rupture of the mitral chordae tendineae is usually antedated by bacterial endocarditis and rarely causes death within a short time. It is characterized by a harsh systolic and diastolic murmur and thrill of abrupt onset that is loudest over the apex of the heart.

**Summary**

The thirty-fourth and thirty-fifth case reports of rupture of a papillary muscle of the heart are presented. The reported causes of
this event are coronary thrombosis with myocardial infarction, trauma to the chest, and syphilis. One case, not yet reported, is supposed to have been due to polyarteritis nodosa. The diagnosis is suggested in a patient, exhibiting evidence of a recent myocardial infarct, who suddenly develops a pronounced increase in the severity of the chest pain associated with profound shock and who on examination reveals a harsh apical systolic murmur which was previously absent. Death usually occurs within twenty-four hours following rupture of a papillary muscle of the heart.

REFERENCES

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