Traumatic Rupture of a Papillary Muscle of the Heart

Report of a Case

WILLIAM A. FRY, M.D.** AND CLAIR E. BASINGER, M.D., F.C.C.P.

Grand Rapids, Michigan

Rupture of a papillary muscle of the heart is a rarely recorded event. Most cases are secondary to myocardial infarction. We recently encountered a case in which the rupture was secondary to trauma, and we feel that it is worthy of presentation. If the diagnosis can be made rapidly, this is a potentially correctable lesion.

Case Report

A 22-year-old white man was admitted to Blodgett Memorial Hospital emergency room at 10 a.m. on January 4, 1962. Three hours earlier, he was injured in an accident in which he was hit broadside while driving his car. He was unconscious for a brief period of time and was then taken to a local hospital for emergency care. His blood pressure there was unobtainable, and he experienced hemoptysis and hematemesis totaling 500 ml. After receiving Dextran 500 ml and hydrocortisone 100 mg., his blood pressure rose to 100/60 mmHg, and he was transferred to this institution.

*From the Department of Surgery, Blodgett Memorial Hospital.
**Presently at Department of Surgery, The University of Chicago.

When he was seen in the emergency room, his second unit of dextran 500 ml was running. His blood pressure was recorded to be 80/60 mmHg, and his pulse rate was 140. He was found to be a well-developed man complaining of pain in the left side and shoulder. He was apprehensive, but well oriented. The skin was warm and dry. The head and neck were otherwise normal. There was palpable crepitation laterally over the left hemithorax. The right hemithorax was resonant with vesicular breath sounds. There was dullness over the entire left hemithorax with diminished breath sounds. There were coarse rhonchi over both lung fields. The heart was not enlarged to physical examination and there was no thrill. The rate and rhythm was that of sinus tachycardia. The heart tones were poorly heard because of the pulmonary findings, but both the first and second heart sounds were heard, and there was no murmur audible. The abdomen was flat with no tenderness and no masses. Peristalsis was hypoactive. Neurologic and extremity examinations were normal except for pain on rotation of the left shoulder. There were small lacerations and abrasions about the face, right elbow and right leg.

FIGURE 1: Chest x-ray film on admission.

FIGURE 2: X-ray film taken five hours post-injury.
Past history revealed that he had been in good health prior to this accident. Two siblings had been treated for rheumatic fever.

Admission hematocrit was 34 per cent, hemoglobin 10.4 grams per 100 ml, and white blood cell count 23,350 per mm³. Urinalysis revealed a specific gravity of 1.027. There was a negative test for reduction, but there was a 2 plus test for albumin. The urinary sediment was loaded with red blood cells and also contained clumps of white blood cells.

Chest x-ray film on admission (Fig. 1) revealed a left hydropneumothorax with partially collapsed left lung and multiple rib fractures. The right lung appeared normal. The heart was slightly shifted to the right, but was otherwise unremarkable. There was a fracture of the left scapula. X-ray examination of the abdomen revealed no significant abnormality.

The patient was taken immediately to the intensive care unit, and a transfusion of 500 ml whole blood was started. Penicillin and streptomycin were administered. At 10:30 a.m., his blood pressure was 80/60 mmHg and pulse 136. At 11:10 a.m. his blood pressure fell to 60/26 mmHg and a second unit of 500 ml whole blood was given with little change in blood pressure. A third unit of 500 ml of whole blood was started shortly before 3 p.m. Chest x-ray film taken five hours post-injury at 3 p.m. (Fig. 2) revealed some reduction of the pneumothorax. However, the left lung was diffusely opacified, and a diffuse process had developed in the right lung. The cardiac silhouette appeared larger. A fourth unit of 500 ml of blood was started. All of the transfusions were given by gravity drip only. By 3:30 p.m. his condition was deteriorating, as his blood pressure fell to unobtainable levels. His hematocrit at that time was recorded to be 46 per cent. He became restless and began to cough up pink frothy sputum. His abdominal findings changed, as he developed generalized tenderness and rigidity, and peristalsis disappeared.

Because of inadequate respiration, a tracheotomy was performed at 4:10 p.m. Shortly thereafter, spontaneous respiration and heart action ceased. In spite of vigorous attempts at resuscitation, he expired at 4:40 p.m.

Necropsy revealed multiple injuries.† There were multiple rib fractures in the left thorax. The upper lobe of the left lung was lacerated and contained a hematoma. The rest of the left lung was contused. There was a perforation of the left diaphragm with an associated small subcapsular rupture of the spleen. The heart weighed 350 grams and was dilated. There was a ruptured papillary muscle attached to the mural leaflet of the mitral valve with acute mitral insufficiency (Fig. 3). The disrupted papillary muscle was twisted through the chordae tendineae (Fig. 4). In addition, severe bilateral pulmonary edema was present.

**DISCUSSION**

The most complete résumé of a ruptured papillary muscle was by Sanders et

†Necropsy performed by Dale L. Kessler, M.D., Pathologist, Blodgett Memorial Hospital.

![Figure 3: Ruptured papillary muscle attached to mural leaflet of mitral valve with acute mitral insufficiency.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20461/ on 06/25/2017)
In 1957, Pry and Basinger reviewed all 61 cases reported up to that time. Only six of these cases were secondary to trauma. The vast majority were secondary to myocardial infarction. Other causes were bacterial endocarditis, luetic myocarditis, tuberculous myocarditis, polyarteritis nodosa, and myocardial abscess. Hansen reported three more cases secondary to myocardial infarction in 1962. Kidera et al. published a report in which a diagnosis of ruptured papillary heart muscle following myocardial infarction was made on clinical grounds with survival of three years at the time of the publication. All other cases reported have proved fatal. Our case represents the 66th reported and the 7th secondary to trauma.

Those cases secondary to trauma often have associated rib fractures. The sequence of pain or trauma followed by shock, an apical systolic murmur of mitral insufficiency, and severe pulmonary edema are the important findings. Important in differentiating a ruptured papillary muscle from an acute ventricular septal defect is the absence of palpable thrill in the former and its presence in the latter. On necropsy, twisted chordae tendineae at the site of papillary muscle rupture is the usual finding.

In this case, the deterioration of the patient's general condition with the development of acute pulmonary edema coincided with the restoration of his blood volume by transfusion and signaled that our impression of contusion of the lung did not adequately explain the patient's clinical course. It is of interest that he was able to survive the effect of his injury for almost ten hours.

We feel that this lesion is correctable, now that open heart procedures can be performed successfully and on an emergency basis. This is particularly true in those cases in which the rupture is of traumatic origin and not secondary to underlying cardiac disease. The importance of early diagnosis cannot be overemphasized.

References
TRAUMATIC RUPTURE OF A PAPILLARY MUSCLE

103


For reprints, please write: Dr. Basinger, 1816 Wealthy Street, SE, Grand Rapids, Michigan.

Announcing 1967 POSTGRADUATE COURSES

DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR AND PULMONARY DISEASES
Fontainebleau Hotel, Miami Beach, Jan.
30 - Feb. 5, 1967

CLINICAL APPLICATION OF CARDIOPULMONARY PHYSIOLOGY
Ambassador Hotel, Los Angeles, Febru-
ary 6-10, 1967

DIAGNOSIS AND TREATMENT OF DISEASES OF THE HEART AND LUNGS
International Inn, Washington, D. C.,
April 6-8, 1967

Tuition for each five day course is $75 to members of the American College of Chest Physicians and $100 to non-members.
Tuition for three day course is $60 to members of the College and $75 to non-members.

Application for post-graduate courses are being accepted in order received.

Additional information may be obtained by writing to Mr. Murray Kornfeld, Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago.