Rupture of the Ventricular Myocardium and Perforation of the Interventricular Septum Complicating Acute Myocardial Infarction

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One of the fatal complications of acute myocardial infarction is rupture of the affected ventricular myocardium. The free wall of the left ventricle is the region usually involved, and sudden death occurs due to acute cardiac tamponade. Occasionally the interventricular septum is perforated, and the clinical signs are characteristic. Recognition of septal perforation may permit utilization of surgical correction of the defect in suitable cases. The following case is presented and discussed to emphasize the clinical signs of rupture of the myocardium and interventricular septum.

Case History

A 56-year-old dock worker was admitted to the hospital on April 28, 1957, because of pain in the anterior portion of the neck and left shoulder. He had been well previously. On the day of admission he went to sleep at 2 p.m. but was awakened 2 hours later by severe pain in the neck and left shoulder and mild upper substernal discomfort. On admission, the temperature was 97 F., the pulse rate was 48 per minute, and the blood pressure was 104/70 mm. Hg. No significant abnormalities were found on complete physical examination. The urinalysis, blood counts, erythrocyte sedimentation rate, blood sugar value, cholesterol content of the plasma, and serologic reactions were normal. Subsequently, the serum glutamic oxalacetic transaminase content rose as high as 140 units per ml. An electrocardiogram made on admission showed sinus bradycardia and changes in the Q wave and S-T segment consistent with acute posterior myocardial infarction.

At 11:30 p.m. on the day of admission the patient noticed a mild aching in the left shoulder. His pulse rate was 60 per minute and his blood pressure was 90/70 mm. Hg. The following afternoon he developed pain in the neck and his blood pressure fell to 80/60. Sympathomimetic drugs administered intramuscularly and intravenously were required to maintain the arterial blood pressure. At 11:45 p.m., 2 days after admission, he had severe pain in the neck and upper substernal area. For the first time a loud systolic murmur was heard along the left border of the sternum over the fourth intercostal space and the murmur was accompanied by a thrill; the pulse rate was 120 per minute. The following morning he had mild pain in the neck and substernal area. The murmur was unchanged, the neck veins were distended, and the lungs were clear. At noon he suddenly developed gasping respirations, followed by a generalized tonic convulsion and death.

Discussion

Dr. William L. Proudfit: It is apparent that this man had an acute myocardial infarction. The localization of the pain was unusual in that the severe pain was in the anterior neck and left shoulder and only minor discomfort was experienced in the upper substernal area. The other feature of interest in the early portion of his illness was

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the bradycardia. Tachycardia is regarded as an unfavorable prognostic sign in acute myocardial infarction, especially if it persists after the first 24 hours. On the other hand, sinus bradycardia is not necessarily a favorable sign; it may be encountered early in the course of extensive myocardial infarction.

Except for difficulty in maintaining the arterial blood pressure, the patient progressed fairly well until the evening of the second day, when he had severe pain and a loud precordial systolic murmur was heard for the first time. Few conditions could account for the sudden development of a murmur and thrill in such a case. Rupture of a papillary muscle, secondary to myocardial infarction, would result in a loud murmur. However, the murmur is usually loudest at the cardiac apex rather than along the left border of the sternum. Furthermore, pulmonary symptoms and signs appear rapidly in this condition, but this patient's lungs were said to be clear and no mention of dyspnea was made. Perforation of the interventricular septum is another serious complication of acute myocardial infarction. The murmur and thrill usually are maximal along the left border of the sternum in the fourth intercostal space and rapid rise in the venous pressure occurs, as was described in this case. The murmur resembles that of congenital interventricular septal defect. A rupture of the aortic valve would result in a diastolic murmur and it would not be expected to occur in the course of acute myocardial infarction. A systolic or diastolic murmur might occur suddenly in dissecting aneurysm, but the described localization of the systolic murmur would not be expected in dissecting aneurysm, and the course of the disease does not seem to be consistent with that condition.

Doctor Tapia, you have been interested in auscultation of the heart. Did you listen to this man's heart or record his sounds?

DR. FERNANDO A. TAPIA: There were no heart murmurs at the time of admission or during the first 48 hours of hospitalization. He then developed a grade-IV, rough holosystolic murmur, which could be heard over the entire precordium but which was distinctly loudest over the fourth intercostal space at the left sternal border; it was associated with a palpable thrill at this level. These findings were detected when the patient was examined because of severe pain in the upper subternal area and neck. A phonocardiogram recorded on magnetic tape the following morning confirmed the auscultatory findings; the murmur had large vibrations of high frequency and there was some midsystolic increase in intensity.

While the phonocardiogram was being made, the patient suddenly developed gasping respirations and transient generalized tonic convulsions. The simultaneous and continuous electrocardiographic and phonocardiographic recording (fig. 1) showed rapid decrease in the intensity of the heart sounds and systolic murmur until they disappeared completely with cessation of the vital signs. However, the electrocardiogram remained unchanged and a normal sinus rhythm continued for several minutes after the patient's clinical death. In the attempt to restore his vital functions by artificial respiration and rapid intravenous infusion of drugs, the microphone was removed temporarily from the chest wall. Two minutes later the microphone was replaced; unchanged electrocardiographic complexes were recorded for 2 or 3 more minutes, but cardiac sounds could not be heard nor could they be recorded graphically. The QRS complexes then gradually became bizarre and broad, and the ventricular rate became slower and irregular until there was complete cessation of ventricular activity.

DR. PROUDFIT: From Dr. Tapia's discussion, it seems necessary to conclude that this man had a perforation of the interventricular septum secondary to acute myocardial infarction. The findings at the time of death are important. It is apparent that the patient did not have a "mechanism death." I believe that actual "mechanism deaths" in the course of acute myocardial infarction are infrequent. It is true that ventricular fibrillation is a terminal event frequently, but usually it is
obvious that death is imminent prior to the development of the arrhythmia. In many other cases the heart simply stops. In this instance neither situation was encountered. The electrocardiogram remained unchanged after clinical death. The fact that the cardiac sounds rapidly became distant just prior to death suggests that something was interfering with the transmission of the sounds or was severely and progressively limiting the diastolic filling or systolic contraction of the heart. The occurrence of a convulsion is consistent with a severe reduction in cardiac output. Rupture of the free wall of the left ventricle with resultant acute cardiac tamponade could be responsible for the findings described.

Fig. 1. Electrocardiogram and phonocardiogram taken immediately before death (paper speed 75 mm. per second in transcription from magnetic tape recording).

Dr. Lawrence J. McCormack: Although this necropsy was limited to the heart, it demonstrates once again how much information can be obtained when even a limited examination is granted. The pericardial sac contained about 300 ml. of partially clotted blood. The heart weighed 410 Gm., the region of greatest enlargement appearing to be the left ventricle. A red, soft, granular area 5 by 5 cm., straddling the septal region, was seen on the posterior surface of the heart. At the inferior margin of this area was a 1-cm. rent (fig. 2). Usually the site of rupture is anterior or lateral. There was variation from the usual pattern of coronary circulation: the posterior circulation of this heart was derived from the left circumflex coronary artery. This artery (the "Achilles' heel" of this heart) had become atheromatous and a superimposed recent thrombus was located 3 cm. from the origin of the artery. The remaining dissectable coronary vessels showed minimal atherosclerosis. When the left ventricle was opened, a laceration, 2.5 cm. in diameter, was found posteriorly 4 cm. from the apex at the junction of the septum and the posterior myocardium (fig. 3). A probe was passed with ease through it and through the externally visible point of rupture. The origin of the internal rent and this dissection channel formed one limb of a Y. Another limb was formed by a channel that passed into the right ventricle with a slit-like orifice located near the base of the medial papillary muscle; this rent was 1.0 cm. in length. The area of destruction of myocardium, represented by softened muscle, seen on a cross section occupied the posterior half of the septum and most of the posterior surface of the left ventricle, and appeared to involve the entire thickness of the myocardium. Microscopic examination of the damaged area disclosed muscle fibers with loss of cross striations, eosinophilia, and swelling with considerable intermingled hemorrhage. There was also a profuse infiltrate of polymorphonuclear leukocytes, far in excess of that usually seen in myocardial infarction but of the degree commonly encountered when rupture of the heart has occurred. No other
anatomic abnormalities were found within the heart. The anatomic diagnoses are (1) atherosclerosis of the left circumflex coronary artery with superimposed recent thrombus formation; (2) anatomic variation of arterial pattern with the left circumflex coronary artery supplying the posterior surface of the heart; (3) myocardial infarction, recent, transmural, posterior; (4) posterior rupture of the myocardial infarct into the pericardial sac with hemopericardium (300 ml.); (5) rupture of the interventricular septum, small, recent; and (6) cardiomegaly (410 Gm.).

Doctor Effler, you have had experience with the surgical treatment of perforation of the interventricular septum. Would you discuss this complication? Do you think you could have offered effective surgical repair in this case?

DR. DONALD B. EFFLER: It would be correct to say that I have had an experience with surgical closure of an interventricular septum following myocardial infarction. The patient, a 54-year-old man, was referred to me approximately 6 months after the infarction had occurred. His initial recovery from the acute myocardial infarction apparently had been fairly satisfactory until the perforation occurred. A systolic murmur which could be heard over the precordium suddenly developed, and at that time, he developed congestive heart failure with a significant increase in the size of the heart. The diagnosis of interventricular septal defect was suspected and was confirmed by cardiac catheterization.

The operation for closure of the interventricular septal defect was undertaken almost 2 years ago. A pump oxygenator was employed for total bypass; in addition, we utilized elective cardiac arrest (Melrose technic). Although there was considerable concern over the patient's coronary artery disease, it did not prove to be a factor in his recovery, and his progress has been gratifying. Subsequent studies done by means of cardiac catheterization almost a year after the operation showed the persistence of the shunt but considerable reduction in its magnitude as compared to that found at the initial study.

The septal defect was located low on the posterior aspect of the septum. It was roughly the size of a nickel; the edges were smooth and the defect was roughly circular. Surgical closure was effected by direct suturing. No prosthetic patch was employed. Perfusion of the coronary circulation must have been adequate, because the arrested heart started
promptly without arrhythmia, and there were no problems related to coronary insufficiency during the postoperative period. Although the defect was created by local ischemia, healing did take place to such an extent that most of the shunt has been abolished.

My conclusion, based on this 1 successful case and the review of 2 autopsy specimens is that successful closure of an acquired ventricular septal defect of the post-infarction variety will probably be limited to those who survive the initial insult and the acquired shunt for a significant period of time. It is quite unlikely that the patient with acute perforation and its attendant complications will tolerate successful surgical closure.


Secondary hypertrophic osteoarthropathy refers to the syndrome of clubbing of the digits, periosteal proliferation with pericortical deposition of bone, and arthritis incidental to a major visceral disease. It occurred most commonly in relationship to pulmonary, pleural, or mediastinal disease. It did not seem to occur most frequently incidental to pulmonary malignancy, but in that association it appeared to develop more rapidly and was more painful. Various theories in etiology have been suggested. It was claimed that pulmonary malignancy produced a factor responsible for clubbing. Cyanosis has long been considered in the pathogenesis of secondary hypertrophic osteoarthropathy. In the clubbing associated with cirrhosis it has been suggested that the phenomenon was due to circulating estrogens or related substances. Sectioning the vagus nerve in the chest was followed by regression of the secondary disease even when the pulmonary tumor could not be removed. Since each of these factors cannot be implicated in every patient with secondary pulmonary osteoarthropathy, the pathogenesis must still be considered unknown. The directly inciting factor may be tissue hypoxia, which may be the result of a number of different mechanisms.

Krause
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