Surgical Management of Acute Septic Pericarditis

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Four patients with acute septic pericarditis were treated by emergency pericardiectomy through a bilateral anterior thoracotomy. The findings of exudative loculated fluid, extensive granulation tissue and septate adhesions, not only indicated the limitations of antibiotic therapy, but portended the development of subsequent chronic constrictive pericarditis. There was immediate relief of cardiac tamponade and dramatic interruption of the septic course. All have been long-term survivors who have exhibited no postoperative complications.

Pericardiectomy is a well established procedure in the treatment of chronic pericarditis, often used in the management of recurrent pericarditis, but less often advocated for acute pericarditis.

The current medical management of acute non-specific or idiopathic pericarditis is successful in most of the cases, although, occasionally, pericardiectomy may be required for recurrent episodes. Many patients with acute septic pericarditis succumb due to sepsis and/or cardiac tamponade. Survivors then usually progress to a state of severe chronic constrictive pericarditis. The pericardiectomy in the chronic state is technically more difficult and hazardous, compared to surgery during the acute phase of the disease.

Four patients with acute septic pericarditis had evidence of low cardiac output, poor tissue perfusion, and impending death and, therefore, were treated by emergency pericardiectomy. Following pericardiectomy, all survived. Adequate cardiac function returned promptly, and the septic course was dramatically interrupted. Follow-up studies showed no evidence of sepsis, effusion, or development of constrictive pericarditis.

Case Reports

Case 1

This 70-year-old dentist was admitted on Sept. 14, 1965, with a five-day history of chest pain, fever and malaise. He was known to have had asthma for a long time.

Physical examination revealed a well developed, well nourished, elderly patient in no acute distress. The blood pressure was 140/80 mm Hg, pulse rate, 104 per minute and regular, temperature 102.2°F, and respiratory rate 15. The breath sounds were distant and rales were present at the bases. The heart sounds were also distant and a grade 2/6 holosystolic murmur was heard at the apex.

Laboratory studies showed: hematocrit, 40 percent; white blood cell count 13,600 cu/mm, with 86 percent polymorphonuclear cells. Results of blood chemistry studies were normal. Moderate cardiomegaly was seen on the chest x-ray film and the electrocardiogram showed first degree atrioventricular (AV) block.

Hospital Course

Initial treatment consisted of bed rest and antibiotics, but his condition continued to worsen, and on Sept. 19, 1965, he developed engorged neck veins, increasing chest pain, fever, hepatomegaly and tachyarrhythmias. A complete blood cell count showed leukocytosis of 21,600 cu/mm, with 92 percent polymorphonuclear cells. The cardiac silhouette size had markedly increased since the last examination (Fig 1), and the ECG revealed QRS complexes, with decreased voltage. Staphylococcus albus coagulase-negative organisms were isolated from the blood culture. He was treated with intravenously administered penicillin, sodium methicillin (Staphcillin), digitalis and diuretics. In spite of intensive medical management for a week, his condition did not improve. On Sept. 28, 1965, a pericardiocentesis yielded 200 ml of bloody fluid, which contained multiple gram-positive cocci.

Twelve hours after pericardiocentesis was performed he developed severe chest pain, a temperature of 104°F and hypotension. An emergency pericardiectomy was performed through a bilateral anterior thoracotomy approach. The pericardium was acutely inflamed and thickened. There was exuberant granulation tissue on the visceral, as well as on the parietal surfaces, of the pericardium. Three hundred...
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milliliters of bloody fluid was aspirated, which grew Staph albus coagulase-negative organisms. The pathologic report of the specimen was "acute and subacute pericarditis." His convalescence was uneventful, and follow-up showed no evidence of chronic constrictive pericarditis. He is now asymptomatic seven years later.

CASE 2

This 59-year-old nurse was admitted with left chest pain, low grade fever, and dyspnea on exertion of five days' duration. Physical examination revealed an obese woman in slight distress. The blood pressure was 90/60 mm Hg, pulse rate 85 per minute, temperature 99.2°F, and respiratory rate 15 per minute. The heart tones were distant, but no friction rub or murmurs were heard.

Laboratory studies revealed: hematocrit, 35 percent; white blood cell count 23,400 cu/mm with 83 percent polymorphonuclear cells. Results of the blood chemistry studies were normal. Chest x-ray films showed an enlarged cardiac silhouette and left pleural effusion. Inverted T waves and depressed ST segments were seen on the electrocardiogram.

Hospital Course

She was initially managed with bed rest and aspirin, but her condition continued to worsen and she developed progressively increasing chest pain, dyspnea, fever and peripheral edema, which responded poorly to digoxin and diuretic therapy. On the 10th hospital day she developed a fever of 103°F, increased venous pressure, hepatomegaly and paradoxic pulse, with narrow pulse pressure. Repeated chest x-ray films showed a significant increase in cardiac silhouette (Fig 2). Seven hundred fifty ml of serosanguineous pericardial fluid was aspirated through a subxiphoid approach. The gram-stain was negative for bacteria, and the culture of the pericardial fluid was sterile. The pathologic diagnosis was "acute serofibrinous pericarditis."

The postoperative course was uneventful, and she remains asymptomatic seven years postoperation.

CASE 3

This 18-year-old male student was admitted with intermittent attacks of diffuse left-sided chest pain, fever, chills and night sweats for one month. During the week prior to admission he had increasing chest pain and respiratory distress.

Physical examination revealed a septic and acutely ill appearing patient. The blood pressure was 120/80 mm Hg, temperature 102°F, respiratory rate 22, and pulse rate of 80. The neck veins were slightly distended. The heart tones were normal, and a pericardial friction rub was heard at the apex. The left base was dull on percussion, with decreased breath sounds. The liver was not palpable, and no peripheral edema was present.

Laboratory investigations revealed: hematocrit, 38.5 percent; white blood cell count 9,500 cu/mm, with 81 percent polymorphonuclear cells. T wave inversion was seen on the electrocardiogram. The chest x-ray films showed an enlarged cardiac silhouette and compression atelectasis of the left lower lobe. The result of a skin test for tuberculosis was negative.

Hospital Course

Over 48 hours following admission, he developed tachypnea, tachycardia, increased venous distention, disappearance of the friction rub, distant heart sounds and an enlarged liver. The chest x-ray films showed a further increase in the size of the cardiac silhouette (Fig 3) and leukocytosis of 14,000 cu/mm, with 92 percent polymorphonuclear cells. He was treated with digitalis, diuretics, and antibiotics, without significant improvement.

A cineangiocardiogram showed extensive pericardial effusion, and pericardiocentesis was attempted through the subxiphoid approach.

Because of his deteriorating condition, an emergency pericardiectomy was performed on May 17, 1970, through a bilateral anterior thoracotomy. Four hundred fifty milliliters of pericardial fluid was taken down. Results of cultures of the pericardial fluid and the pericardium were sterile. The pathologic diagnosis was "acute serofibrinous pericarditis."

The postoperative course was uneventful, and she remains asymptomatic seven years postoperation.
of serosanguineous pericardial fluid was aspirated. The pericardium was 4-6 mm thick and was loosely adherent to the epicardium. Results of the culture of pericardial fluid were sterile. The histologic diagnosis was acute fibrinous pericarditis. Two years later he is now asymptomatic.

CASE 4

This 63-year-old laborer was admitted with cough, chest pain, and fever of six weeks' duration. He was treated with antibiotics, but his condition did not improve. Ten days prior to hospitalization he developed a spiking temperature up to 105°F, dyspnea on exertion, orthopnea, malaise, and fatigue.

Physical examination revealed a critically ill man with severe respiratory distress and poor tissue perfusion. The blood pressure was 95/60 mm Hg, pulse rate 114, respiratory rate 40, and temperature 105°F. The neck veins were distended, and the heart tones were distant, but no friction rub or murmurs were heard. Moist rales were present at both bases. The liver was palpable 3 cm below the right subcostal margin. Moderate peripheral edema was present.

Laboratory investigations showed: hematocrit, 35 percent; white blood cell count, 11,700 cu/mm, with 90 percent polymorphonuclear cells. Blood gases on 40 percent oxygen and 6 liters flow were pH 7.46, Pco₂ 43, Po₂ 50 and oxygen saturation 88 percent. He had a reversed albumin globulin ratio, serum glutamic oxaloacetic transamine level of 320, and a lactic acid dehydrogenase level of 640 units. First degree AV block and flat ST segments were seen on the electrocardiogram. Chest x-ray films showed a markedly enlarged cardiac silhouette, passive pulmonary congestion and a small right pleural effusion (Fig 4). In spite of antibiotics, diuretics and digitalis therapy, his condition did not improve, and paradoxic pulse with narrowing of pulse pressure became more severe, and death appeared imminent. A cineangiocardigram showed massive pericardial effusion.

An emergency pericardiectomy was performed on July 1, 1971, through a bilateral anterior thoracotomy. One thousand milliliters of cloudy pericardial fluid with flecks of fibrin was drained. There were few fibrinous adhesions between the thick, shaggy pericardium and epicardium which were easily separated, and a complete pericardiectomy was performed with relative ease. Staph albus coagulase-negative organisms were cultured from the pericardial fluid. The pathologic diagnosis was acute fibrinous pericarditis.

The postoperative course was uneventful, and at ten months' follow-up the patient is doing well.

DISCUSSION

Rehn² in 1913, reported the first successful pericardiectomy, and Churchill³ in 1929, first described this in American literature. Since then, extensive resection of the pericardium has been widely accepted for chronic constrictive pericarditis, although the role of surgery prior to the chronic constrictive phase has not been clearly defined. Holman and Willett⁴ advocated early pericardiectomy for active tuberculous pericarditis. Others support a trend toward earlier surgical intervention before the constrictive process has taken place,⁵,⁶ thereby circumventing a longer illness and the more difficult technical problems of later operations.

The diagnosis may be considered when the patient has chest pain, shortness of breath, and fever. The findings of pericardial friction rub, nonspecific ST segment changes, and an enlarged cardiac silhouette are usually present. As cardiac tamponade develops, venous distention, hypotension, paradoxic narrowing of the pulse pressure, hepatomegaly, and lowering of the amplitude of electrocardiographic complexes occur. An increase in the size of the cardiac silhouette and the fluoroscopic recognition of decreased pulsation may be helpful. Radioisotope scanning of the heart requires cooperation, which is not always possible in critically ill patients. Carbon dioxide injection in the pericardium, right atrioangiography, cineangioangiography, and coronary angiography are usually diagnostic of pericardial effusion, but often may be poorly tolerated in the patients with severe acute pericarditis. Currently, echocardiography is the
simplest, safest, quickest, and one of the most reliable diagnostic aids for pericardial effusion.\textsuperscript{7,8} It yields physiologic, as well as anatomic, information.\textsuperscript{9} Because of unavailability of this procedure in the past, it was not performed in the cases reported here. At present, echocardiography is routinely used in our institution. Bacteriologic and cytologic studies of pericardial fluid are helpful in determining the possible etiology and appropriate antibiotic therapy. Bloody effusion is usually indicative of severity of pericarditis, and the incidence of subsequent constrictive pericarditis is higher in these patients.\textsuperscript{10}

Pericardiocentesis is diagnostic and occasionally therapeutic in the patients with cardiac tamponade due to simple effusion, but seldom produces more than temporary relief\textsuperscript{11} in patients with pericarditis of the magnitude cited in the case reports referred to here. In these patients, pericardiocentesis may be hazardous and should not be undertaken unless the presence and extent of pericardial effusion is confirmed and complete facilities for resuscitation are available. It was unsuccessful in one of our patients because of loculation of the fluid. Moreover, it may cause laceration of the myocardium or coronary arteries, resulting in further intrapericardial bleeding and cardiac tamponade.\textsuperscript{12}

Pericardial window operation has been advocated by some, but generally has failed because of insufficient drainage\textsuperscript{13} and lack of protection from the development of subsequent severe constrictive pericarditis.

During acute pericarditis, there are usually only a few loose adhesions between the pericardium and epicardium, which can be easily separated with minimal blood loss and trauma to the myocardium. This makes pericardiectomy a relatively safe and easy procedure at this stage. The pericardium should be excised from one phrenic nerve to another from the diaphragm to the great vessels. A bilateral anterior thoracotomy in the fourth intercostal space with transection of the sternum provides excellent exposure.

This course of action has provided immediate relief of tamponade, dramatic interruption of the septic course, avoidance of complications of long-term drug therapy, and prevention of subsequent adhesive pericarditis. Thus, we support the concept of early pericardiectomy for patients with acute septic pericarditis.

**REFERENCES**