Case Report Section

The Twenty-Year Course of Chronic Constrictive Pericarditis with Subsequent Surgery

J. RONALD POWELL, M.D. and ADDISON W. POPE, M.D.*
Nashville, Tennessee

Chronic constrictive pericarditis is a disease characterized by gradual fibrosis, thickening and contraction of the pericardium and/or epicardium leading to a reduction of cardiac function. It is most frequently a complication of tuberculous pericarditis.

White reports¹ that chronic constrictive pericarditis was first described in 1669 by Lower. Burwell states² that since that time Lancisi, Morgagni, Laennec, Hope, Chevers and Pick have recognized and added additional information to further elucidate the pathophysiology.

In 1896, Weil and Delorme suggested³ resection of the pericardium as a means of treating and perhaps curing chronic constrictive pericarditis. In 1920, Rehn⁴ reported on the first successful operation. Churchill and White in 1929 reported⁵ the first successful resection in this country.

There have only been a few reported cases of the disease followed from the initial infection to the constricting stage or to the time of operation. Furthermore some investigators report that surgical risk increases directly with the duration of the disease, the operative mortality high and the beneficial results dubious. In view of this, our paper is concerned with a patient with chronic constrictive pericarditis followed from the initial infection through 22 years of palliative management and the results obtained on this patient after pericardiectomy was performed.

M. C., a 47 year old colored woman entered the George W. Hubbard Hospital on November 30, 1956 complaining of shortness of breath, abdominal swelling and edema of the lower extremities. She had been followed in the cardiac clinic over a period of three years with similar symptoms. Numerous thoracenteses had been required to relieve her of dyspnea. Mercurial diuretics, diamox, a low sodium diet and other measures did not prevent her from becoming edematous and dyspneic to the point that she could not be managed on an outpatient basis.

A review of her history disclosed that 22 years ago on June 14, 1934 at the age of 24, she entered a local hospital complaining of chills, weakness, fatigue, muscular aches and pains. She had nausea, vomiting and severe diarrhea. These symptoms had existed for five weeks. There was weight loss of 22 pounds and cough productive of white frothy sputum. She denied hemoptysis. On examination her temperature was 103° F. She had tachycardia, and bilateral non-tender enlargement of the submaxillary and cervical lymph nodes. The area of cardiac dullness was enlarged to the left and right of the sternum. The heart sounds were distant and a pleuro-pericardial friction rub was heard along the left sternal border. The blood pressure was 130 mm. of Hg. systolic and 80 mm. of Hg. diastolic. A paradoxical pulse was present. There was dullness to percussion and diminished breath sounds in the left axilla. The remainder of the physical examination was not remarkable.

The hemogram revealed three million red blood cells and 6,450 white blood cells with a differential count of 60 per cent polymorphonuclear cells, 35 per cent lymphocytes, 3 per cent eosinophils, and 2 per cent basophils. Urine specific gravity 1.010, otherwise normal. The Wassermann and Kahn tests were reported as strongly positive. The venous pressure was 140-145 mm. of saline. X-ray film of the chest showed marked globular enlargement of the cardiac silhouette. There was no evidence of active pul-

* Meharry Medical College.
monary tuberculosis. Electrocardiogram revealed sinus tachycardia and first degree heart block. The tuberculin test was strongly positive in a 1:1000 dilution.

A diagnosis of acute pericarditis probably of tuberculous origin was made. After 34 days of non-specific therapy, she became afebrile and asymptomatic. She was discharged to be followed in the home. Approximately five months later she was readmitted to the hospital with a complaint of exertional dyspnea, headaches, and pain between the shoulders.

Physical examination on that admission revealed a moderately enlarged heart. There was dulness to percussion over the lower right lung posteriorly. The liver was palpable one and one-half fingersbreadth below the right costal margin. The venous pressure was 124 mm. of saline. A diagnosis of constrictive pericarditis was made. Surgery was advised but she refused. She was given symptomatic therapy and bedrest. After 10 days she was discharged again to the outpatient department. For a 10 year period extending from 1938 to 1948 she had no contact with the hospital and engaged in her occupation as a cook with only minimal disability.

During the latter half of 1948 she was seen at another local hospital for an abscess in the upper lateral area of the right thigh. Incision and drainage was performed but no bacteriological diagnosis was made. Three months later she developed an abscess in the same area of the left thigh and the tissue removed was described by the pathologist as being granulomatous. One year later she was treated at the George W. Hubbard Hospital for a chronic draining abscess of the right thigh. Tubercle bacilli were isolated from the exudate. In 1952 she developed a right psoas abscess that required incision and drainage.

In 1954, 20 years after the initial diagnosis of acute tuberculous pericarditis, she was admitted to the George W. Hubbard Hospital because of dyspnea, abdominal distension, and edema of the lower extremities. She had generalized cardiac enlargement, distant heart sounds, auralicular fibrillation, pulmonary rales, ascites hepatomegaly, edema of the lower extremities and the venous pressure was increased. A diagnosis of constrictive pericarditis was made but she refused the suggested operation. She responded slightly to a low salt diet, digitalization and mercurial diuretics.

During the next two years she was hospitalized on numerous occasions due to the above symptoms. She developed right pleural effusion which required thoracenteses at monthly intervals. A total of (36 liters) of serous fluid was removed from the right pleural space during this period. It became more difficult to manage her and weekly thoracenteses were necessary just prior to the last admission. She was unable to perform any work and became dyspneic at bed rest. The ascites increased and edema of the lower extremities became more pronounced. Diuretics were ineffective. She appeared terminally ill and this hospitalization was recommended.

Examination at the present admission revealed her to be a well developed, poorly nourished middle aged markedly dyspneic woman. The blood pressure was 110/72, temperature 99.2° F., pulse 90 per minute and respirations were 24 per minute. There

FIGURE 1

FIGURE 2

Figure 1: Chest x-ray revealing pleural effusion prior to thoracic tap and surgery.—Figure 2: Chest x-ray two and one-half weeks post-operatively.
was marked distension of the neck veins. The area of cardiac dullness was moderately enlarged to the left. The heart sounds were faint and of poor quality. The heart rhythm was totally irregular. Tactile fremitus was decreased over the lower half of the posterior right lung and breath sounds were diminished in that area. There was dullness to percussion in the posterior aspect of the lower right lung field. Moist rales were present in the base of the left lung. The abdomen was protuberant and there was shifting dullness to percussion in the flanks. The abdominal wall was tense, shiny, and edematous. The liver was not palpable but there was percussion dullness extending to the umbilicus. There was 4+ pitting edema of the thighs, legs and feet.

Laboratory findings revealed a white blood cell count of 7,550 with a differential of 62 per cent polymorphonuclear cells, 35 per cent lymphocytes. The hemoglobin was 13.2 grams per cent and the hematocrit was 41 per cent. The pertinent urinary findings were specific gravity of 1.010 with 3-4 W.B.C. per high power field and an occasional granular cast. The blood chemistry studies were as follows: total serum protein 4 grams per cent, with albumin 2.6 grams per cent and globulin 1.4 grams per cent. The blood sugar was 94 mgm. per cent, and the N.P.N. was 25 mgm. per cent. The circulation time was 35 seconds and the venous pressure was 300 plus mm. of saline.

The pleural fluid appeared slightly serosanguineous with specific gravity of 1.012, two polymorphonuclear cells per cubic millimeter, 24,000 erythrocytes per cubic millimeter, and 0.3 grams per cent of proteins. X-ray film of the chest revealed moderate cardiac enlargement, slight enlargement of the pulmonary artery segment, slight aortic widening with elongation and minimal calcification, bilateral pulmonary congestion and pleural fluid in the right lung base. The electrocardiogram showed atrial fibrillation, low voltage on the QRS complexes, and diphasic to inverted T waves.

Therapy consisted of a low salt diet and maintenance digitalis. Thoracenteses were performed weekly. Diamox and mercuhydrin were used for diuresis. Even with this rigid therapy the pleural fluid accumulated rapidly. The ascites also increased. She could not tolerate even minimal activity and was confined to bed. At this time she consented to surgical therapy.

The operation was performed on January 8, 1957, 22 years after the initial diagnosis was made. The heart was found encased in a thick fibrous pericardium which severely restricted its movements. The fibrous pericardium was removed from both ventricles. The myocardium appeared slightly atrophic, but a marked increase in cardiac pulsations could be seen after the removal of the constrictive tissue. She tolerated the procedure well. Five days postoperatively the venous pressure had fallen to 220 mm. of saline and the circulation time was 13.4 seconds. There was a marked increase in the urinary output up to 2,000 ml. a day without diuretics. The edema and ascites subsided. She improved progressively and one month after operation the venous pressure was 90 mm. of water and the circulation time was 15.8 seconds. The total serum proteins increased to 5.8 grams per cent. A liver profile revealed a negative cephalin flocculation test, a serum bilirubin of 0.7 mgm. per cent, a prothrombin time of 17.8 seconds with 69 per cent activity. The total cholesterol was 270 mgm. per cent and the cholesterol esters 171 mgm. per cent. She was up and about the ward without dyspnea and was discharged on February 9, 1957 to be followed in the cardiac clinic.

She has been followed in the clinic for 12 weeks postoperatively and has not required a thoracentesis since the immediate postoperative withdrawal of fluid. She shows gradual improvement and is well maintained with a low salt diet and mercurial diuretics. She is able to do minimal house work at this time and states she never realized how well she could feel.

DISCUSSION

The importance of making a correct diagnosis cannot be overstated. The recent development of antituberculous agents renders constrictive pericarditis, in a great many instances, among the preventable diseases. The rapid development in thoracic surgery has rendered the condition among the curable and, if not curable, symptoms can be greatly ameliorated and life prolonged.

The diagnosis may not always be as clear as in our patient. It may mimic heart disease with predominant right ventricle failure. The persistence of ascites, hepatomegaly and marked elevation of venous pressure with lack of response to therapy should give a clue to the possibility of constrictive pericarditis. The inability of the patient to increase her cardiac output upon activity is a significant clue. X-ray and E.K.G. changes are also of value in establishing the diagnosis.
The specific treatment of chronic constrictive pericarditis is an operation consisting of removal of the thickened pericardium. Burwell\(^6\) states that the operation is in general not a curative procedure but a modifying one. This is due to the fact that the majority of patients still have some residual objective signs although they may have marked subjective improvement.

The primary purpose of an operation is the release of the two ventricles from the constricting pericardium. The improvement after operation is slow and may be incomplete. However, this fact should not deter physicians from recommending an operation in patients with constrictive pericarditis.

In the case presented, it was felt that after 22 years with constrictive pericarditis, the patient had become a poor surgical risk for the following reasons: 1) The possibility of myocardial fibrosis and atrophy suggested there may be little improvement after operation. 2) Chronic hepatic congestion may have led to irreparable liver damage such as cardiac cirrhosis. 3) The possibility of acute dilatation of the atrophic ventricular myocardium when the constricting scar was removed. However, the patient tolerated the operation well. After the pericardial scar was removed from the ventricles, the underlying myocardium did not appear too atrophic and an increase in contractions could be seen immediately.

After operation, the circulation time and venous pressure fell to near normal range. In spite of continued improvement, the patient still develops pleural fluid, some ascites and dependent edema. This however can be well controlled with diuretic drugs, and thoracenteses have not been necessary 12 weeks post-operatively. This would indicate that in spite of the duration of the disease, if a diagnosis of constrictive pericarditis is made, the patient should be given the benefit of surgery.

Special acknowledgment is given to Dr. John Thomas for his kind assistance in the preparation of this paper.

BIBLIOGRAPHY