Chronic Constrictive Tuberculous Pericarditis *
Report of a Case with Pericardiectomy

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As its name implies, this disease is distinguished by an inflammatory and cicatricial transformation of the pericardium, trapping the heart in a compressive, inelastic envelope. The deposition of scar tissues may incarcerate the heart, fix the heart and pericardium to unyielding surrounding structures and constrict the great veins and aorta. Clinically, the predominance of any or all of these features will have its peculiar bearing on the disease. The cumulative result is circulatory failure from deficient filling of the heart. The problem seems to stand clear in its simplicity—the removal of a mechanical obstruction, a familiar task to the surgeon. Unfortunately, the location of the involvement, its etiology and the wretched condition of the patient makes this task formidable indeed.

The organism held frequently responsible for constrictive pericarditis is the tubercle bacillus. Blalock and Burwell demonstrated a tuberculous etiology in 18 of 28 cases. Three cases were due to pyogenic organisms. Harrington found tuberculosis in 5 of 21 cases. Rheumatic pericarditis rarely progresses to the chronic constrictive stage and therefore rheumatic fever cannot be incriminated. Tuberculous pericarditis usually occurs in association with tuberculosis of the lymphatics, pleura or peritoneum. The pericardium commonly becomes involved by extension of adjacent infection from caseous mediastinal or peribronchial lymph nodes. The dynamics of constrictive pericarditis follow the familiar pattern of tamponade. They were clearly described by Burwell and Blalock as: (1) High peripheral venous pressure. (2) Low peripheral arterial pressure. (3) Basal heart rate is elevated. (4) Movements of heart are diminished. (5) Total blood volume is 30–45 per cent above normal. (6) Circulation time is prolonged. (7) Per-minute output of the heart is diminished. No significant increase in stroke volume with exercise.

The patient with constrictive pericarditis of tuberculous origin

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may present the picture of heart disease with or without parallel tuberculous activity elsewhere. Generally, the imperative nature of pericardial involvement dominates the clinical scene. Impressive features are the marked venous engorgement, hepatomegaly, recurrent ascites and pleural effusion and the small paradoxical pulse. The heart is rarely enlarged, the heart sounds are faint and murmurs are absent. Blood pressure and pulse pressure are low. Mild pulmonary congestion is paled by the marked peripheral venous distention. Dyspnea at rest is rare and when present is expressive of massive pleural effusion and ascites. The patient may be febrile and this response is an important index of tuberculous activity.

The elevated venous pressure is not appreciably altered by pleural and abdominal paracentesis. Frequent venous pressure readings give reliable objective evidence of the progress of the disease. The return of venous pressure to normal is the prime criterion of successful therapy. Radiological study of the heart is an important aid in diagnosis.\textsuperscript{8,11} Roentgenoscopic signs are: limitation of cardiac shift with changes in position of the patient, decrease in amplitude of cardiac pulsation, and limitation of the elongation of the heart with descent of the diaphragm. Roentgenographic findings are: pericardial calcification, a small aortic knob and a triangular or globular shaped heart. The x-ray kymogram gives a graphic record of the reduced excursion of the heart muscle. Opinions vary as to whether the kymogram record changes

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\caption{Figure 1: Chest x-ray film preoperatively.}
\caption{Figure 2: Kymogram, eight months postoperatively.}
\end{figure}
consistent with relief secured by surgery. Heuer and Stewart\textsuperscript{11} and White\textsuperscript{12} have described electrocardiographic changes in chronic adhesive pericarditis. These are mainly distinguished by low QRS complexes and flat T waves.

Definitive therapy is the removal of the binding pericardial-epicardial scar. All efforts are bent to eliminate edema and serous effusions in preparation for surgery. A low salt, high protein diet is given. Mercupurin and ammonium chloride are effective diuretics. Large effusions may require paracentesis. Digitalization is not recommended because it has been shown to decrease the per minute output of the heart in this condition. Burwell\textsuperscript{3} feels that digitalis may be indicated where there is auricular fibrillation; when failure of the atrophic myocardium is expected after pericardiectomy; and in the rare case that has added myocardial failure. Quinidine may be used preoperatively, as in this case, to forestall ventricular fibrillation when the heart is being manipulated. Conflicting opinions exist as to the necessary limits of cardiac decortication. Most American surgeons advise liberating the right and left ventricles without going beyond the auriculo-ventricular groove. The auricles and great vessels are avoided. Some European surgeons\textsuperscript{9} are content with freeing the left ventricle. Practically, the extent of decortication is dictated by the manifold dangers of adherent coronary vessels, attenuated myocardium and obliterated cleavage planes. In addition, the surgeon must skirt the hazards of ventricular fibrillation and cardiac

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\includegraphics[width=\textwidth]{pericardial-scar.png}
\caption{Pericardial scar at operation.}
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arrest. Hence, physiological needs are frequently subordinated to surgical caution. Pericardectomy is not calculated to, nor does it affect the basic tuberculous infection. Spread and recurrence may develop after operation. Appreciation of this fact is required to explain some of the dismal results in the face of adequate surgery. Perhaps streptomycin will fill the need for controlling the element of infection.

FIGURE 4: Angiocardiogram, two and a half months postoperatively.

The determination of the optimum time for surgery requires exquisite judgment. There is general agreement that operation should be delayed until the tuberculous infection has become inactive. Certainly, morbidity statistics confirm this view. Unfortunately, the rapid march of compressive symptoms may dictate surgical intervention before infection has subsided. The surgeon must weigh the relative influence of infection and constriction, and place most emphasis on circulatory interference.

The following case is presented:

The patient is a 31 year old white barber. Familial and past history were not related to the present illness. In January, 1947 he was stricken with a pulmonary infection alleged to be pneumonia. He was treated with penicillin and remained in bed for two weeks. The patient tried to resume his job, but was forced to quit working because of increasing dyspnea and weakness. He had an unproductive cough. There was no hemoptysis. He was hospitalized at the Veterans Administration Hospital, Tuscaloosa, Alabama. At that institution, his chest was tapped and about one quart of clear yellow fluid was removed. His abdomen and ankles began to swell and he became progressively weaker. He was transferred to the Kennedy Veterans Hospital, VAMTG on March 30, 1947.

On admission the patient appeared cyanotic, dyspneic and chronically
ill. His neck veins were distended. The left hemithorax was flat to percussion. No breath sounds could be heard over the same area. The PMI of the heart could not be felt. On percussion, the heart was not enlarged. Heart sounds were distant. There was regular sinus rhythm. No murmurs could be heard. Blood pressure was 80/60. The pulse was paradoxical. Abdominal distention and shifting dullness were apparent. Hepatosplenomegaly was present. There was 2 plus pitting pretibial edema.

Laboratory Findings: Blood count, RBC 4.8 million, WBC 9,500, polymorphonuclears 78, lymphocytes 18, monocytes 4. Urinalysis: Light amber, pH 4.5, sp. gr. 1.005, albumen negative, sugar negative, many fine and coarse granular casts, occasional WBC. Blood chemistry: total protein 6.5 per cent, albumin 3.6, globulin 2.9. Serum bilirubin 2.2 mg. per cent. Malaria smear negative. No agglutination for typhoid, paratyphoid, brucella abortis, tularemia. Blood culture sterile. Urine culture, staphylococcus aureus and gamma streptococcus. Pleural fluid: protein 1.8 per cent, smear and culture negative for tuberculosis and other organisms, many WBC, no tumor cells. X-ray film of chest: moderate pleural effusion on the left. Fluoroscopy: massive left pleural effusion with displacement of the mediastinum moderately to the right. The heart was not enlarged and no pulsations of the cardiac outline were seen. Venous pressure: 32 cm. of water.

The patient was seen by Dr. Abraham Gootnick, Chief of the Cardiovascular Section who made a diagnosis of constrictive pericarditis. A tuberculous etiology was entertained, but could not be proved. The diagnosis was based on: (1) Elevated venous pressure. (2) Pleural effusion. (3) Ascites and dependent edema. (4) Low blood pressure. (5) Paradoxical pulse. (6) Absence of valvular murmurs. (7) Enlarged smooth tender liver. (8) ECG showing low voltage, and inverted T waves in all leads.

The patient was transferred to the cardiovascular service for further study and preparation for surgery. A low salt, high protein diet was given. Repeated left thoracentesis and abdominal paracentesis were performed. Mercurial diuretics were administered. The effectiveness of these measures was limited, since the pleural effusion recurred rapidly. However, ascites and peripheral edema became less marked. The patient remained afebrile until April 28, 1947 when he suddenly had several bouts of fever mounting to 104 degrees F. The fever was presumed to be evidence of the activation of the underlying pericardial disease. Gradually, the fever reached a lower level. Relief of circulatory interference, by medical means, was transitory. It became evident, that despite the active infection, surgery was indicated to release the seriously compromised heart. Quinidine was administered for four days and on May 15, partial pericardiectomy was performed.

OPERATION

Findings: The pericardium varied in thickness from 3 to 7 mm. The adhesions between the epicardium and pericardium were pliable and relatively easily separated by blunt dissection. There was a fairly definite band around the great vessels at the base of the heart. The apex was freed completely, as was the diaphragmatic surface. The pleura was very adherent on the left, and could not be stripped as completely as desired. The right auricle was partially exposed. The region of the superior vena cava was freed as much as possible from the left side but
could not be safely freed entirely. Apparently the inferior vena cava was released by this separation.

Procedure: The patient was placed on his back. The usual skin preparation was done. A curved incision was made over the second rib extending downward to the sternum, and laterally along the 5th cartilage. This was extended through skin, subcutaneous tissue, and pectoralis muscle. Portions of the 2nd, 3rd, 4th and 5th cartilage, and ribs were resected subperiosteally. Internal mammary vessels were ligated, and incision was made through the intercostals, exposing the mediastinum. The left pleura was stripped to the left. Several holes were made and repaired with atraumatic catgut. Apparently no hole was made in the right pleura in stripping it back. An incision was made over the heart through the pericardium, and a line of cleavage was established. An incision was made at right angles to the original incision, and the flaps kept intact and carried in all directions. Excess pericardium was removed, and the heart was further freed by blunt finger dissection as far as possible. The anterolateral surface of the aorta was freed, releasing a band around the base of the heart. One per cent novocaine was dropped on the heart from time to time. The space was irrigated with saline and 200,000 units of penicillin was instilled. The wound was closed with interrupted catgut sutures with two rubber tissue drains in place; one into the mediastinum, and one anterior to the periosseum and perichondrium. The skin was closed with interrupted mattress and simple sutures of silk. The patient left the operating room in about the same condition as when the operation began.

PATHOLOGY REPORT

Microscopic: Section is composed of dense hyalinized connective tissue diffusely infiltrated by mononuclear cells, epithelioid cells and occasional giant cells of the Langhan's type. Tubercle formation is widespread and most of the tubercles show caseation necrosis in their centers. In many instances the caseous necrotic areas are confluent forming larger zones in which no cellular detail can be recognized. In one section there is a rather large amount of tuberculous granulation tissue and recent hemorrhage. Near one margin the surface of both sections is covered by a fibrinous exudate. Beneath the surface the tissue is of a fibroadipose type, and contains many dilated blood vessels, neutrophiles and chronic inflammatory cells.

Diagnosis: Tuberculous pericarditis.

Immediately postoperative, there was a marked serous discharge from the wound. Cyanosis and neck vein distention receded considerably. Blood pressure was 90/70 mm. of mercury and venous pressure 15 cm. of water. On the following day the patient became icteric. Within two weeks the jaundice gradually subsided. Amigen was given to replace protein lost in the serous discharge. On the third postoperative day dyspnea recurred. X-ray inspection showed a large left pleural effusion. Drains were removed and left thoracentesis was performed. Repeated aspirations of the left hemithorax were instituted to remove the rapidly accumulating fluid. Low salt diet and mercurial diuretics were resumed. Evidence of liver damage was seen in the laboratory studies: BSP 15 per cent retention in 45 minutes, cephalin flocculation 3 plus in 4 hours, serum bilirubin 1.5 mg. per cent. Low grade fever persisted after healing of the wound. It was assumed to be due to active infection in
the remaining pericardium. The pleural fluid was a transudate and negative culture. On June 19, 1947 streptomycin therapy was begun. Fifty grams of the antibiotic was given as 0.2 gram every four hours for five doses daily. The fever gradually subsided and he remained afebrile after July 30, 1947. The main difficulty was the ever recurring pleural effusion and edema. There was cause for alarm when the venous pressure rose to 22 cm. of water on August 1, 1947. An angiogram showed no superior vena caval obstruction. Constriction about the caval orifice was ruled out as a cause of the venous engorgement. Kymographic studies demonstrated poor pulsations of the cardiac border. On September 26, 1947 no recurrence of pleural effusion was noted. X-ray inspection showed marked reduction in the pericardial and mediastinal thickening. The kymogram indicated increased amplitude of contractions of the left ventricle. Ascites, edema and venous distention disappeared. The enlarged spleen and liver diminished in size. Venous pressure had dropped to 7.5 cm. The patient was ambulatory and had no dyspnea on ordinary effort. On January 2, 1948 he returned from a month's leave, having gained four pounds. His liver was just barely palpable and the spleen could no longer be felt. BSP test showed no retention after 45 minutes. Cephalin flocculation was negative; prothrombin time 100 per cent; serum bilirubin 0.6 mg. per cent. The ECG exhibited a normal pattern. On fluoroscopy, increased motion of the heart was seen. The patient was considered apparently cured and transferred to a convalescent hospital February 1, 1948. Following a paranoid type of schizophrenic reaction he went home in remission July 29, 1948.

Comment

This case presented some interesting and perplexing problems. The most disturbing of these was the slow recovery of the patient. Persistent anasarca and elevated venous pressure, in the face of adequate surgery, can be a source of chagrin and impatience to the surgeon. The consensus is that complete recovery can frequently not be expected for months.\(^1,2,6\) Apparently the liberated myocardiogram is atrophied and requires time to regain its strength. This is a comfortable assumption if the surgeon can be assured that no recurrent or overlooked cicatricial bands compress the great vessels and the heart. Angiocardiography is suggested as a method of visualizing the patency of the superior vena cava. By such means we were convinced of innocence in that quarter. In view of the subsequent recovery it may be assumed that the patient was suffering from myocardial failure in his postoperative course.

Mercurial diuretics, salt free diet, bed rest and thoracentesis were of great value in successfully weathering this precarious period. The pre- and postoperative use of these measures must be assigned a role of importance, second only to pericardectomy. There is no information in the literature concerning the effect of streptomycin on tuberculous pericarditis. Streptomycin was not available, here, preoperatively. Its use postoperatively may
have curbed the activity of the disease in the remaining pericardium. Possibly, it protected the open pleural cavities from the disease. These remarks are conjectural; no unequivocal conclusions can be drawn from this case as to the value of streptomycin in active chronic productive tuberculous pericarditis. Should further study prove the specificity of streptomycin, the management of this disease will be simplified. More cases of tuberculous pericarditis with effusion may progress to healing and instances of obstructive scarring may become rare. Where decortication will be required, one can envision earlier operations on cases rendered inactive by the drug. Earlier operation will mean coping with less cicatrization and a healthier myocardium. Tuberculosis of the pericardium will not lose its terrors, but streptomycin may blunt its sting.

SUMMARY

1) The clinico-pathological aspects of constrictive pericarditis have been summarized.

2) A case of tuberculous constrictive pericarditis with partial pericardiectomy and apparent cure has been presented.

3) Freeing the heart from its fibrous encasement is the initial step in the relief of cardiac embarrassment. Postoperative supportive care of the weakened myocardium is important in the total therapy of the disease.

4) Streptomycin was used postoperatively. Its value will be determined by more extensive clinical studies.

RESUMEN

1) Se resumen los aspectos clínico-patológicos de la pericarditis constrictiva.

2) Se ha presentado un caso de pericarditis constrictiva tuberculosa con pericardiectomía parcial y aparente curación.

3) La liberación del corazón de su coraza fibrosa es el paso inicial en aliviar el embarazo cardíaco. El cuidado sustentante postoperatorio del miocardio debilitado es importante en la terapia general de la enfermedad.

4) Se usó estreptomicina después de la operación. Su valor será determinado por estudios clínicos más extensos.

REFERENCE

1 Bowers, R. F.: Personal communication, Kennedy Veterans Hospital, Memphis, Tennessee.