Chronic Pericarditis with Effusion

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Pericarditis with effusion is sometimes a chronic disease, an exception to the general rule that it is acute rather than chronic. Illustrative cases are described. Features of interest are the prominent third heart sound, the large P waves, the thrombi in the right atrium, the progression to constrictive pericarditis, and the resemblance in several respects to mitral stenosis.

Pericarditis is commonly classified as acute or chronic. Pericarditis with effusion is regarded as a form of acute pericarditis and not as a form of chronic pericarditis.1,2 This is undoubtedly correct in nearly all instances. There are, however, occasional exceptions in which pericarditis with effusion follows a chronic course of many months, notable for the complete absence of the features of acute disease. The purpose of this report is to call attention to this less common type of chronic pericarditis with effusion, to describe three illustrative cases and to comment on several features of special interest.

Case Reports

Case 1.—R.Z. (U.H. 576097) was an 18 year old student when first seen on September 4, 1945. He had enjoyed good health until the age of 13 years when he had pneumonia and was told that he had rheumatic heart disease. His activities were restricted upon the advice of his physician, but he soon developed fatigue and shortness of breath upon moderate exertion which gradually increased in severity. There was no history of cardiac pain, palpitation or edema, or of previous rheumatic fever, growing pains, chorea or cyanosis, although he had frequent sore throat during childhood.

Physical examination revealed a healthy appearing youth with gross cardiac enlargement, dulness extending 6 cm. to the right and 13 cm. to the left of the mid-sternal line. The pulmonary second sound was slightly accentuated, but there was no palpable impulse, heave, shock or thrill, and no friction, murmur or gallop. The rhythm was normal. The neck veins were slightly distended. The lungs were clear. The liver was enlarged to 7 cm. below the right rib margin, but there was no ascites. Minimal edema of the ankles was present. There were no signs of thyroid disease. There was a paradoxical pulse; the blood pressure was 96/85/80.

The hemoglobin was 17.9 Gm., the blood Kahn test was negative. The electrocardiogram showed broad, notched, deformed auricular deflections (fig. 1). X-ray examination revealed moderate enlargement of the heart with a globular contour, and minimal left hydrothorax. A review of nine previous films, beginning on January 7, 1941, showed progressive increase in size of the heart, except in the films of September 18, 1943 and September 4, 1945 which showed decrease in cardiac size.

Upon limited activities, digitalis and salt restriction the patient’s course was satisfactory until the summer of 1947, when dyspnea and edema increased and ascites developed. Auricular fibrillation appeared and the course was progressively downward. On November 19, 1947 the findings were characteristic of constrictive pericarditis. There was only slight enlargement of the heart; it was definitely smaller than in 1945. In spite of the great risk, pericardiectomy seemed to be the only hope. The patient died on December 12, 1947, sixteen hours after operation. The temperature was normal throughout and the white blood cell count was normal.

The autopsy showed chronic constrictive pericarditis with partial pericardiectomy. There was polyserositis with active chronic fibrous pericarditis, pleuritis and peritonitis and “pericarditic pseudo-cirrhosis” of the liver. There was extreme atrophy of the muscle fibers of the ventricles, and hypertrophy of the atrial muscle. The mitral leaflets showed slight fibrous thickening, but no active inflammation and no vascularization and no stenosis. There was no evidence of rheumatic fever or tuberculosis.

Comment.

This patient showed clinical and roentgenographic evidence of pericarditis with effusion from 1941 through 1945, which by 1947 had progressed to constrictive pericarditis. There was no clinical evidence of active or acute disease during this time. The etiology of the pericarditis and polyserositis was not apparent; the pneumonia in 1940 might have been the cause. A feature of interest was the finding at autopsy of atrophy of the muscle fibers of the ventricles whose movements must have been greatly restricted by the diseased pericardium, and of hypertrophy of the auricular muscle.

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The broad, notched auricular deflections of the electrocardiogram made two years before death were of the type observed in mitral stenosis and in other conditions associated with auricular hypertrophy.

Case 2.—H.M. (U.H. 603646) was a 38 year old farmer, first seen on November 12, 1946, when he complained of swelling of the lower extremities. He stated that in 1943 he was kicked in the chest by a horse, following which he had dyspnea upon slight exertion and swelling of the feet, and that he was told that his heart was enlarged. He improved slowly until he was able to drive a truck, but he gave up farming. In December, 1944, he had several hemoptyses, followed by dyspnea upon slight exertion, fullness in the epigastrium and swelling of the ankles. From that time on he had recurring episodes of dyspnea and swelling of the feet and legs, improving under treatment by his physician but soon returning and becoming much more pronounced in the summer of 1946. There was no history of thoracic pain or palpitation, or of rheumatic fever, renal disease, scarlet fever or venereal disease.

Physical examination showed an obese man with massive edema of the legs, thighs and genitalia, but able to lie flat. The cardiac dulness was greatly enlarged, the sounds were distant but regular, a third sound in diastole was heard near the apex, but no friction or murmur was detected. The blood pressure was 105/80, the pulse was not paradoxical. The neck veins were distended, there were râles at the lung bases, and the liver was enlarged, but ascites was not present.

X-ray examination showed marked cardiac enlargement, pulsations were present but diminished, and the shape of the cardiac silhouette changed with change in position of the patient. The electrocardiogram showed large, broad P waves. The total serum proteins were 6.9 per cent, albumin 4.1 per cent and globulin 2.8 per cent.

The patient improved upon rest, digitalis and a diuretic regimen. It was thought at first that he might have rheumatic heart disease with mitral stenosis, but it later became apparent that he had pericarditis with effusion.

The patient got along fairly well during 1947 on digitalis, ammonium chloride intermittently, restriction of sodium intake and an occasional mercury diuretic, and although slight to moderate edema persisted he was able to work as a taxi cab driver. Early in 1948, however, auricular fibrillation appeared and dyspnea and edema became pronounced. In June, 1948 the edema was massive, orthopnea was present and there were râles at the lung bases. The cardiac findings were as before, with greatly enlarged cardiac dulness, auricular fibrillation, a third sound in diastole, and no murmur or friction. The serum albumin had declined to 3.0 per cent. In July and August, 1948, pericardial paracentesis was performed on five occasions; the needle encountered thickened pericardium (fig. 2). The amounts of fluid withdrawn varied from 325 cc. to 1650 cc.; it was clear and...
straw colored at first, but later it was somewhat bloody. The fluid was negative for acid fast bacilli upon smears of concentrated preparations and cultures for pyogenic organisms were sterile.

Partial pericardectomy was performed by Dr. John Alexander on August 27, 1948. Thickened, inflamed, parietal pericardium was removed from the anterior and left lateral aspects of the heart. All chambers of the heart appeared somewhat enlarged and especially the right atrium. The tissue removed showed evidence of acute fibrinous exacerbation of chronic fibrous pericarditis.

Improvement was transient. Edema soon became more pronounced and paradoxical pulse and ascites appeared. Auricular fibrillation, cardiac enlargement, and the diastolic third sound persisted. On December 8, 1949, Dr. Cameron Haight removed thickened epicardium from the left ventricle and much of the right ventricle, after which the amplitude of ventricular contractions was good. The right atrium was distended, motionless and quite firm; it was thought to contain an organized thrombus. The left auricle seemed normal. The tissue removed showed evidence of chronic fibrous pericarditis. There was only slight improvement following the second operation.

Throughout the period of observation from November, 1946 to December, 1948 the clinical course was that of a chronic disease. The temperature was normal on all occasions except transient elevations following the operations. The white blood cell count was 13,800 on November 15, 1946, but was normal a few days later and upon all subsequent determinations. The acute fibrinous change observed in the tissue removed at the first operation is attributed to the repeated tappings which preceded the operation.

Comment.

This patient was known to have had pericarditis with effusion continuously from No-
November, 1946, to August, 1948, and it probably began in June of 1943. The cause of the pericarditis was not determined, but it might have been caused by the injury to the chest sustained in June, 1943. The clinical course was chronic leading finally to constrictive pericarditis. The failure to improve satisfactorily after partial pericardiectomy on two occasions may be attributed in part to the presence of a mass, probably a thrombus, in the right atrium. It is of interest that the angiocardiogram revealed dilatation and prolonged filling of the superior vena cava and slow, irregular filling of the right atrium and right ventricle, suggesting a mass in the right atrium (fig. 3).

Case 3.—N.H. (U.H. 666733), a 33 year old taxi cab driver, was first seen on August 29, 1949, complaining of swelling of the abdomen. He stated that he was not accepted for military service in 1940 because of a “leaky heart.” There had been no symptoms referable to the heart until May, 1949. Then he developed shortness of breath upon exertion which was followed in two weeks by swelling of the abdomen and ankles. The dyspnea became more pronounced and orthopnea developed. There was no pain. The patient was hospitalized elsewhere, digitals was given and abdominal paracentesis was performed on three occasions, but improvement was only transient. There was no history of rheumatic fever, growing pains, chorea or venereal disease.

Physical examination showed a well developed man with pronounced enlargement of the area of cardiac dulness, auricular fibrillation, a systolic murmur over the entire precordium, and a diastolic third sound near the apex. There was no friction rub. The blood pressure was 125/70. The neck veins were distended. The lungs were clear. The liver was enlarged and ascites was present. There was no edema of the legs.

The electrocardiogram showed auricular fibrillation and deflections of small amplitude. X-ray examination showed marked, generalized, cardiac enlargement with somewhat diminished pulsation of the left border. The blood Kahn test was negative. The blood count was normal. The urine was normal. The total serum proteins were 7.7 per cent, albumin 4.8 per cent and globulin 2.9 per cent.

It was thought at first that the patient might have rheumatic heart disease with mitral stenosis, but it soon became apparent that he had pericarditis with effusion. He improved somewhat upon rest, digitals, ammonium chloride intermittently, and restriction of sodium intake, but the cardiac enlargement, the diastolic third sound, the distension of the neck veins, and enlargement of the liver persisted and the ascites soon returned. The pulse was paradoxical at times.

On September 30 about 600 cc. of clear, straw-colored fluid were removed by pericardial paracentesis, after which the patient felt better without showing objective improvement. On October 31 the pericardium was tapped again and 400 cc. of fluid were removed. On November 8 a third tap yielded 320 cc. The fluid was clear and straw colored with a specific gravity of 1.017, and the spun sediment showed 10 to 12 cells per high power field, mostly lymphocytes; no acid fast bacilli were found in smears of the concentrated preparations and cultures for pyogenic organisms were sterile.

On December 7, 1949, Dr. Herbert E. Sloan, Jr., performed a partial pericardiectomy. The distended pericardium was incised and several hundred cc. of fluid were aspirated. The heart appeared greatly enlarged, chiefly the right ventricle and right atrium. The right atrium was a bulging mass 10 by 5 by 4 cm., motionless and firm, thought to contain a thrombus. The left ventricle and left atrium seemed normal. The epicardium was smooth and glistening. The parietal pericardium was only slightly thicker than normal; it was widely resected. The tissue showed evidence of chronic fibrous pericarditis.

The patient was somewhat improved following operation, but his symptoms were not entirely relieved. The cardiac enlargement, auricular fibrillation, systolic murmur and diastolic third sound persisted. The venous pressure was lower, but soon rose again and the ascites returned. The temperature was normal throughout except during the four days following operation, and the white blood cell count was normal repeatedly.

Comment.

This patient had pericarditis with effusion from May to December, 1949. The disease ran a chronic course without pain, fever or leukocytosis, but had not progressed to the stage of constrictive pericarditis by the time of operation in December. The etiology of the pericarditis was not apparent.

Discussion

These three cases are clearly examples of pericarditis with effusion in which the disease was chronic. The painless course of seven months to four years or longer, without fever or leukocytosis, and the histological evidence of chronic fibrous pericarditis, point to chronic disease. They cannot properly be classified as acute pericarditis. Although they are exceptions to the general rule that pericarditis with effusion is acute rather than chronic, it is thought that cases of this type are not rare. They present several features of interest.
In none of these cases was the etiology of the pericarditis apparent. The clinical and histological findings and study of the fluid yielded no evidence of tuberculous or rheumatic etiology. In the first patient the disease followed pneumonia the type of which is not known, while in the second case the symptoms followed an injury to the chest. In the third instance there was no preceding illness. The fluid resembled a transudate, but the tissue removed showed evidence of inflammatory disease. All three patients were relatively young, white males.

The symptoms were dyspnea, edema and swelling of the abdomen, compatible with cardiac tamponade but not differing greatly from those commonly encountered in mitral stenosis with congestive failure. The physical signs were those of pericardial effusion with cardiac tamponade, although at times paradoxical pulse was not detected.

A feature of special interest was the prominent third sound in early diastole in two of the patients, best heard near the apex. Attention has been called to a similar sound occurring in chronic constrictive pericarditis, and to its superficial resemblance to the murmur of mitral stenosis, but its occurrence in pericarditis with effusion has not been emphasized.

The electrocardiograms obtained in two of the patients during normal rhythm, before the onset of auricular fibrillation, showed large, broad or notched auricular deflections like those commonly observed in mitral stenosis. All eventually developed auricular fibrillation. The x-ray examination revealed the cardiac enlargement, but was of little help in establishing the diagnosis of pericardial effusion. It could be decisive if angiocardiography were employed, and in distinguishing pericardial from pleural effusion if films were made after the withdrawal of fluid and its replacement by air.

The diagnosis of pericarditis with effusion was not at first apparent in any of these cases. The two patients with diastolic third heart sounds were thought at first to have mitral stenosis. The absence of the other signs commonly present in mitral stenosis, such as an accentuated apical first sound or pulmonic second sound, and a more careful evaluation of all of the findings soon led to the correct diagnosis. The tendency for auricular fibrillation to occur, and for the disease to progress to chronic constrictive pericarditis is perhaps to be expected.

The finding in two of the patients at operation of evidence of a large, organized thrombus in the right atrium was of interest. It is probable that the mass partly filling the right atrium offered an impediment to the return of blood to the right heart and thus contributed materially to the elevation of the venous pressure and to the formation of ascites and edema. Indeed, in the second case, the angiocardiograms supported this view, showing dilatation and prolonged filling of the superior vena cava and slow, irregular filling of the right atrium and right ventricle. This may explain this patient’s failure to improve satisfactorily after wide resection of thickened pericardium on two occasions sufficient to permit good ventricular contractions. It is thought that the thrombus formation in the atrium was secondary to the pericardial disease and the auricular fibrillation, and not primary.

**Summary**

1. Pericarditis with effusion is sometimes a chronic disease. Although this is an exception to the general rule that pericarditis with effusion is acute rather than chronic, such cases are probably not rare. Three illustrative cases of unknown etiology are described.

2. Features of special interest are the occurrence of a prominent third heart sound in early diastole near the apex, the observation of large, broad or notched P-waves in the electrocardiograms, the presence of large, organized thrombi in the right atrium, and the tendency to progress to chronic constrictive pericarditis.

3. The diagnosis is often not at first apparent, the condition being most readily confused with mitral stenosis.

**REFERENCES**


