Chronic Effusive Pericarditis

By S. Contro, M.D., G. De Giuli, M.D., and F. Ragazzini, M.D.

An unusual case of chronic pericarditis with effusion, lasting at least four years, in an asymptomatic child, is presented. The literature on the subject is reviewed. Attention is drawn to the fact that such cases exist, and should be considered when one is confronted with a case of cardiac enlargement of obscure etiology.

It is generally believed that effusive pericarditis is an acute ailment. Well known textbooks of cardiology,1, 2, 3 as well as the literature4 on the subject, divide pericarditis into an acute and a chronic form, and consider the latter as synonymous with the various types of constrictive pericarditis. However, a survey of the literature reveals three cases5, 6 in which the inflammation of the pericardium ran a chronic, quiet course with the presence of abundant fluid in the pericardial sac.

Recently, we had the opportunity to observe a case of chronic pericarditis with effusion of four years’ duration in a child. The patient had no symptoms referable to the cardiovascular system, and the recognition of the cardiac lesion was made almost fortuitously. The purpose of this paper is to emphasize that pericarditis with effusion may assume a chronic course without evidence of constriction.

Case Report

A seven year-old girl of Italian extraction was first seen in January, 1949. Birth and development were normal, and there was no significant past medical history. Two days prior to being seen, she developed high fever, slightly productive cough, and dyspnea. At the time of examination the pertinent physical findings were: numerous crepitant rales at the left base posteriorly and cardiomegaly, with the left heart border percussed at the left anterior axillary line in the fifth intercostal space. There was no cyanosis, cardiac murmurs, or signs of congestive failure. A diagnosis of left lower lobe pneumonia was made, and the patient was placed on penicillin therapy and bed rest in her home. After three days of this regimen, the temperature returned to normal, and the rales in the left lung disappeared. Six days later, when she was again ambulatory, x-ray films of the chest were taken, which revealed clear lung fields with sharply defined costophrenic angles and no evidence of fluid in the pleural cavities (fig. 1; a, b, c). The vascular markings were within normal limits, and the aortic contour appeared normal. There was generalized enlargement of the heart, most marked to the left. An electrocardiogram (fig. 2) taken at this time showed low voltage in the limb leads, but otherwise was within normal limits. Due to lack of symptoms and physical findings, a diagnosis was deferred, and it was decided that the patient should be observed at regular intervals to determine the evolution of the above-noted cardiac abnormality.

The patient was next seen for routine examination 10 months later. At this time she was asymptomatic, and there had been no change in the physical findings or x-ray picture.

Due to her sense of well-being and complete lack of symptomatology, she was not seen again until October, 1952, almost four years after the onset of the illness. During this period she had contracted measles which had been treated by another physician with apparent complete recovery. At this time a chest x-ray film again revealed normal lung fields, and generalized cardiac enlargement. There had been no change from films taken three years previously. Fluoroscopy showed diffuse cardiac enlargement with feeble cardiac pulsations. A roentgenkymogram (fig. 3) confirmed the impression of minimal pulsations along the heart borders.

In December, 1952, following an upper respiratory infection, the patient developed high fever, cough, and dyspnea. There was slight dullness to percussion with bronchial breathing and inspiratory rales at the left base posteriorly. Further physical examination at this time revealed no other abnormality except the previously reported cardiomegaly. A diagnosis of left lower lobe pneumonia was again made, the patient was hospitalized, and therapy with penicillin and streptomycin was instituted.

The laboratory findings at this time were as follows: The blood count showed 4,320,000 red blood cells and 13,000 white blood cells per cubic millimeter and hemoglobin of 80 per cent. Dif-
ferential count showed polymorphonuclear leucocytes 64 per cent; lymphocytes 34 per cent; eosinophiles 1 per cent; mononuclear cells 1 per cent. Urinalysis was normal, with constant absence of ketone bodies on fasting samples. Fasting blood sugar was 100 mg. per 100 cc. Blood sugar rose normally after injection of epinephrine. Blood urea nitrogen was 18 mg. per 100 cc. Total serum cholesterol was 180 mg. per 100 cc. The basal metabolic rate was plus 10. The Wasserman test was negative. The test for lupus erythematous was negative on two occasions. Agglutinations for typhoid, paratyphoid, and brucella were negative. The Congo red test was normal. Skin test and complement fixation for echinococcus was negative. The result of a Mantoux test was 1 plus at 24 hours; 2 plus at 48 hours.

The patient's symptomatology gradually diminished. Over a seven-day period, the temperature returned to normal; physical findings in the lungs disappeared; and the white blood cell count dropped to 7,000. At this time it was decided that further x-ray studies were indicated.

Artificial pneumomediastinum was induced to rule out the presence of an extracardiac mass. Films (fig. 4) taken at this time revealed the thymus to be normally located in the anterior mediastinum and to be of normal size. No extracardiac mass was demonstrable.
An angiocardiographic examination was then performed. Films (fig. 5 a, b) revealed the cardiac chambers to be centrally located and apparently of normal size. However, the distance from the inner walls of the right and left chambers to the outer limits of the cardiac shadow measured about 3 cm. 

Because these findings were considered characteristic of pericardial effusion, a pericardiocentesis was performed, and 70 cc. of clear, light-yellow fluid was aspirated. This did not clot on standing and contained 5 Gm. of protein per 100 cc. The fluid was centrifuged and microscopic examination revealed the presence of 10 to 15 lymphocytes per high power field. Gram, methylene blue, and Ziehl-Nielsen stains failed to reveal any organisms. Guinea pig inoculations and culture for acid-fast bacilli were negative.

At the time of pericardiocentesis, air was injected into the pericardial sac and was seen to move freely within an enlarged pericardial cavity at fluoroscopic examination. Moreover, the patient was rotated through 360 degrees in the horizontal plane and through 180 degrees in the vertical plane (so that the feet were in the upright position). In the latter position, the cardiac apex was outlined by air. Films were taken in upright and left lateral decubitus positions (fig. 6 a, b) and confirmed the fluoroscopic findings of an air-fluid level, normal inner cardiac silhouette, thin outer pericardial margin, and absence of loculation of fluid.

The patient was placed on streptomycin and isoniazid therapy with no change in the physical findings. She remained asymptomatic and there was no further change when last seen in June, 1953.

**DISCUSSION**

The diagnosis of effusive pericarditis was not initially entertained in the above case, owing to the belief that this condition always runs an acute course. The presence of a normal development and normal tolerance for exercise, as well as the absence of auscultatory findings, first drew our attention to the differential diagnosis of enlargement of the cardiac shadow.

The unlikely possibility of an extracardiac mass embracing the entire cardiac contour and simulating cardiac enlargement was excluded by the pneumomediastinum studies. The lack of symptoms and of progressive findings ex-
eluded the various forms of myocarditis and of tumors of the myocardium.

Tumors of the pericardium might have been considered. Jucker,7 in 1941, published two cases of primary pericardial tumor and reviewed the 22 cases previously reported. The author concluded that they can be divided into two main groups: those which produce hemorrhagic pericarditis, and those which produce infiltration of the pericardium. In both groups the symptomatology is rapidly progressive, making this an unlikely possibility in the present case. The diagnosis of chronic effusion due to myxedema was untenable in view of the normal appearance of the patient and the negative laboratory results.

A cardiac form of Von Gierke's disease was considered. The absence of ketone bodies in fasting urine and a normal rise of blood sugar after injection of epinephrine militated against it. Moreover, the survival of a patient, with this condition for four years without symptoms has never been reported. Di Sant' Agnese and coworkers,8 in a recent article, reviewed all the cases on record and showed that these patients are severely incapacitated and do not reach one year of age.

The laboratory studies also made unlikely such causes of cardiac enlargement as primary amyloidosis and echinococcosis.

The angiocardiogram was typical of pericardial effusion. A diverticulum of the pericardium was excluded by pneumopericardium.

In seeking the etiology of the effusion, the remaining possibility which can be considered seriously is tuberculosis. In favor of the diagnosis of tuberculous pericarditis is the positive Mantoux test. However, more than 50 per cent of the population in the patient's community are Mantoux positive, and, therefore, the positive reaction may have been a coincidental finding. Culture for Mycobacterium tuberculosis and guinea pig inoculations were negative and the patient showed absolutely no response to two and one-half months of antituberculous chemotherapy in adequate dosage. Furthermore, the patient was completely free of symptoms and the pneumopericardium showed no thickening of the parietal pericardium. In the absence of an anatomical examination, tuberculosis as the etiological factor in this case cannot be completely excluded. However, for the reasons cited above we believe this to be unlikely.

A search of the literature has revealed very few reports of long-standing pericardial effusion. Blumenfeld's case4 followed massive irradiation for a carcinoma of the breast and lasted about four years. Autopsy revealed remarkable fibrous thickening of the pericardium with no specific inflammatory reaction. The remaining two cases are reported in the article published by Barker and Johnston in 1950.6 Along with another case of doubtful chronicity, these cases, on microscopic examination of the pericardium, revealed only nonspecific changes. These cases are exceptions to the oft-quoted statement that the diagnosis of tuberculous

Fig. 6 A, B. Pneumopericardium in frontal upright and left lateral decubitus positions.
pericarditis should be made when no other etiology can be established. The possibility of a congenital lesion in our case has to be considered since the lesion was first discovered at the age of 7 years. This might take the form of a congenital obstruction to the lymphatic drainage of the pericardium. The protein concentration of 5 Gm. per 100 cc. in the pericardial fluid would fit this hypothesis. However, no such case has ever been recorded and, without anatomical evidence, this idea can remain only speculative.

**Summary**

A case of chronic, symptomless pericardial effusion in a child is presented. Despite adequate search, no etiological factor was discovered. A survey of the literature reveals only two or three comparable cases.

Attention is drawn to the possibility of the existence of a definite syndrome of chronic pericardial effusion.

**Summario in Interlingua**

Es presentate un caso inusual de chronic pericarditis effusive in un puera asymptotic observate durante quatro annos ab le septime al dece-prime anno de su etate. Un revista del litteratura revelava solmente duo o tres casos comparabile. Nonobstante, le occurrentia de tal casos es estabite e debe esser prendite in consideration quando on se trova confrontate con un allargamento cardiac sin etiologia clar.

**References**
