satisfactorily after the separation of common ventricle into two chambers.

REFERENCES

Constrictive Pericarditis Associated with Echinococcus Cyst

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We present an unusual case of constrictive pericarditis caused by echinococcal cyst of the heart. Diagnosis, therapy, and unique problems caused by heart cysts are discussed.

The finding of cardiac echinococcosis is very unusual, but more unusual is the late complication of constrictive pericarditis secondary to rupture of the hydatid cyst into the pericardium. The surgical treatment of constrictive pericarditis has been standardized, but the finding of Echinococcus as an etiologic factor raises questions not encountered in causes such as tuberculosis or microbial infection. Presented here is the seventh successfully operated case of constrictive pericarditis secondary to a ruptured cardiac echinococcus cyst.

CASE REPORT

A 50-year-old man was admitted to the Christian Hospital, Mashad, Iran, August, 1969 with a history of edema of the feet and abdominal pain of one year's duration. The patient had been treated six months before with diuretics and digoxin without improvement. Significant past history was that as a child in his home, he had close contact with sheep dogs.

Physical examination showed a cyanotic man with an edematous face and mild dyspnea. His blood pressure was 120/90, pulse 130 per minute, and respiration 26 per minute. The neck veins were distended with the patient sitting upright. Examination of the chest revealed an increased A-P diameter and on auscultation, distant breath sounds were heard along with crepitant rales in both bases. The point of maximum impulse of the heart could neither be seen nor palpated. There were distant heart sounds with no murmur. The abdomen was distended and ascites was present. The liver was very tender and was enlarged 12 cm below the right midcostal margin. There was 2+- pitting edema of the legs. A pericardial knock was thought to be heard after admission.

Laboratory studies which did not give normal findings were: prothrombin time 20 seconds (control 15); BSP 18 percent retention in 45 minutes; serum bilirubin 6 mg percent total and 4 mg percent indirect; and alkaline phosphatase of 20 Bessy-Brock units. The tuberculin test was negative. A Casoni test done after operation was negative. The electrocardiogram showed low QRS voltage with diffuse ST-T wave abnormalities and absence of a Q wave in lead V4. Fluoroscopy of the chest showed little pulsation of the heart shadow and elevation of the right diaphragm.

The patient was given digitals and diuretics resulting in a slower pulse rate, but the edema, ascites, and hepatomegaly persisted. After four weeks' hospitalization, the patient was still in heart failure and not much improved. On October 23, 1969 pericardial biopsy was performed which demonstrated a thick (0.8-mm) pericardium without evidence of tuberculosis. On October 25, 1969 definitive pericardiectomy for constrictive pericarditis was performed. A collapsed hydatid cyst was found near the apex of the left ventricle which was irrigated with 30 percent saline solution. Postoperatively, the patient did well and by the eighth day most of the edema, hepatomegaly, and abdominal pain was gone. Six months after surgery the patient was well and taking only digitals.

DISCUSSION

Echinococcal disease is quite common in certain areas of the world, but echinococcus cyst of the heart is not common. Only a few of the embryos reach the heart via the coronary arteries.1,2 It is felt that the process of infestation probably occurs in childhood and takes several years for the cysts to grow and later cause symptoms.3

It has been shown that about 40 percent of all cases of cardiac cysts will rupture and most of these are fatal.4 Rupture into the pericardium will usually cause either acute hydatid pericarditis or several loculated daughter cysts which may or may not be symptomatic. Occasionally, rupture into the pericardium will later produce constrictive pericarditis.

Diagnosis is made by a high index of suspicion of echinococcus when an abnormal x-ray finding or electrocardiogram is seen in an area endemic with hydatid disease. X-ray examination may show an abnormal heart enlargement and the electrocardiogram frequently indicates ischemia of the left ventricle. Casoni intradermal test, complement fixation, or hemagglutination tests may be confirmatory of hydatid disease, but are only positive in about 60 percent of cases.

Because of the incidence of rupture of the cyst and subsequent high mortality, treatment is generally surgical removal of the cyst or evacuation of the contents and sterilization of it. The first successful operation for hydatid cyst of the heart was by Long in 1932.5 Since that time several reported cases of cysts of the heart have been successfully treated surgically.4,6 For the complication of constrictive pericarditis, as in our case, the classic pericardiectomy, as described by Holman, is adequate if
Diaphragmatic Origin of the Pacemaker Sound*

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The late onset of diaphragmatic stimulation without apparent intercostal muscle stimulation caused a pacemaker sound in a patient with a transvenous pacemaker in the absence of obvious myocardial perforation. In this situation the pacemaker sound does not invariably indicate myocardial perforation. Serial ventricular electrograms may be helpful in detecting changes in catheter position and in the diagnosis of myocardial perforation. Some manipulation of the pacing catheter is often required to eliminate uncomfortable diaphragmatic stimulation whether or not perforation has occurred.

Since Nager and co-workers² first described the pacemaker sound, several workers³-⁷ established its extracardiac origin in either intercostal or pectoral skeletal muscles, but recent reports⁸-¹⁰ have shown that diaphragmatic stimulation by percutaneous pacing catheters may also generate a pacemaker sound. This brief report describes the occurrence of a pacemaker sound caused by the relatively late onset of diaphragmatic stimulation in a patient without apparent catheter perforation of the heart.

CASE REPORT

An 81-year-old woman was admitted to Highland Hospital because of syncopal episodes, angina and congestive heart failure. The electrocardiogram showed sinus rhythm with 2:1 A-V block and a ventricular rate of 40 per minute, left axis deviation and right bundle branch block. Chest x-ray film showed slight cardiomegaly with pulmonary venous congestion. On April 29, 1970 a permanent demand (ventricular-inhibited) pacemaker delivering a 0.85 msec impulse was inserted percutaneously with a bipolar pacing catheter (electrodes 1 cm apart) wedged at the right ventricular apex. Unipolar and bipolar electrograms¹¹ were recorded (Fig 1). Pacing thresholds were measured with an external pacemaker delivering a 2 msec pulse. The threshold for unipolar pacing was 1.0 mA when the tip was the cathode and 1.2 mA when the proximal electrode was the cathode. The threshold for bipolar pacing was 0.8 mA. The postoperative course was uneventful and no pericardial friction rub or pacemaker sound was heard. In particular, no diaphragmatic or localized intercostal contractions were present with deep respiration or changes in body position. On discharge, a chest x-ray film

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