

CONSTRICTIVE EPICARDITIS: A CASE REPORT

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We describe a patient in whom only visceral pericardial (epicardial) scarring and calcification have caused myocardial constriction symptoms. The parietal pericardium was normal and pericardial space was completely free from adhesions. The etiology was not identified from the history, laboratory work-up and pathologic examination. The constrictive features were relieved by complete resection of the visceral pericardium. In the currently available literature in English, to our knowledge, similar pathology has not been published. It should be kept in mind that the inflammatory process may merely affect the visceral pericardium which can cause restriction of myocardial function.

Key words: Epicarditis, constructive pericarditis, restrictive cardiomyopathy

We report an unusual case of myocardial constriction caused by fibrous, calcified epicardium with normal parietal pericardium.

What is called, the "pericardium" is actually the parietal pericardium which is a fibrous sac surrounding the heart. The visceral pericardium is a serosa layer comprising the epicardium of the cardiac surface (1).

Pericardial constriction may be a consequence of any of the identified causes of pericardial inflammation (2,3). The accompanying scarring usually effects the parietal and visceral pericardium (epicardium), obliterating the pericardial space. The pericardial space may be completely or partially obliterated with dense adhesions from fibrous or fibrocalcific pericardium and epicardium that limits diastolic expansion and seriously restrict cardiac output.

CASE REPORT

A 32-year-old woman was admitted to the hospital with a history of dyspnea, ascites and peripheral edema for six months of duration. History was not supportive for tuberculosis, collagen-vascular diseases, malignancy, irradiation, renal failure, metabolic disorders or chest trauma.

On examination, blood pressure was 110/70 mm Hg with an irregular pulse of 92/min and a respiratory rate of 22/min. The neck veins were distended to the angle of the jaw at 90° while sitting.

There was pleural effusion bilaterally. The heart sounds were somewhat decreased but audible; no third heart sound was heard. No

rubs but an apical 1-2/6 holosystolic murmur was audible. No pulsus paradoxus was present. Moderate ascites was demonstrated and liver was palpable 5 cm below the right costal margin. Umbilical hernia was noted on the abdominal wall. There was 2+ edema over the ankles.

Laboratory data

Hematocrit was 32.8%, WBC $2.92 \times 10^3/\text{mm}^3$, sedimentation rate 65mm/h (Westergren), C Reactive Protein 28.1 mg/L, rheumatoid factor 20.0 IU/ml. Kidney and liver function tests were in normal limits. PPD was negative. Chest x-ray showed bilateral pleural effusion, right-sided pleural thickening and pericardial calcification, especially in the lateral view. The heart was not enlarged. EKG revealed low voltage QRS complexes and atrial fibrillation. Echocardiographic findings were consistent with pericardial constriction, 1+ mitral regurgitation and 2+ tricuspid regurgitation, but the pericardium was only mildly thickened and calcified, and there was no pericardial effusion.

Hospital course

Right heart catheterization was performed. Mean right atrial pressure was 18 mm Hg, right ventricular pressure was 48/5 mm Hg, pulmonary artery pressure was 48/20 (36) mm Hg, and pulmonary capillary wedge pressure was 20 mm Hg. Chest CT scan revealed thick and calcified pericardium particularly at the anterior surfaces of the heart, bilateral pleural effusion and bilateral irregular pleural thickening. There was no mass or lymph node enlargement. The patient was operated on. A midline sternotomy was performed. The parietal pericardium appeared slightly thick but otherwise normal and shiny. There were no adhesions between the two layers of the pericardium. The pericardial space was completely free from adhesions and there was a minimal amount of serous pericardial fluid. The visceral pericardium (epicardium) was white-yellowish in color all over the heart, thickened, almost 1 cm thick in most areas and heavily calcified especially at the anterior surfaces of the heart around the right atrioventricular groove. Epicardium was

meticulously freed from the myocardium all around the heart. Cultures of pericardial fluid and resected epicardium were negative for *M. tuberculosis* or the other pathogens. Microscopic examination, revealed extensive scarring, hyalinization, fibrosis and calcareous deposits, no specific microscopic diagnosis was made.

The postoperative course was uneventful and the patient was discharged at the 5th postoperative day.

DISCUSSION

Constrictive pericarditis is a rare disease. Pericardial constriction may follow almost any of the identified causes of pericarditis. Sometimes, a specific etiologic diagnosis is obtained but idiopathic cases have a large contribution in the published series (2,3). In constrictive pericarditis, usually the pericardial space is not only obliterated by adhesions between the parietal and visceral pericardium, but is transformed into a dense layer of scar or calcification (4).

The case reported here is atypical in that only the visceral pericardium was involved. In the currently available literature in English, to our knowledge, similar pathology has not been published.

It should be kept in mind that inflammatory process may merely affect the visceral pericardium and restrict myocardial function.

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