Encircling All Of The Heart By A Bone Cage Due To Chronic Constrictive Idiopathic Pericarditis

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Citation


Abstract

The calcific deposits may vary in size from microscopic to massive, encircling all or most of the heart and readily visible by radiographic examination.

In this study we're presenting a massive calcific pericardial heart disease due to idiopathic ethiology and encircling all of the heart. We're reporting the pathogenesis, clinical and surgical indications for operation, and the prognosis.

Conventional open pericardiectomy via the median sternotomy, which enables a safer, wider, and more effective approach and it relieved the symptoms and altered the hemodynamic findings.

INTRODUCTION

The clinical table of chronic constrictive pericarditis (CCP) was first described in 1842 by Cheever in his Observations on the Diseases of the Orifice and Valves of the Aorta, in which he reported that dangerous symptoms arose chiefly from the compression of muscle tissue by sticky material that surrounded the heart(1,2). The evolution of a successful operation for the relief of a heart encased in and constricted by its own thickened pericardium is one of the most interesting and exciting facets of surgical history. As early as 1898, De Lorme had conceived the idea that pericardial resection for this condition might be feasible. It remained for the German surgeon, Rehn, in 1913, however, actually to resect a constricting pericardium with great improvement in his patient's condition. Subsequent successes were reported by Churchill in 1929, Becker in 1930, Harrington in 1940, and Blalock in 1941(3). Today, pericardiectomy is a safe procedure with available techniques(2). In this study we're presenting a massive calcific pericardial heart disease due to idiopathic ethiology and encircling all of the heart. We're reporting our diagnostic and successful surgical approaches under light of literature.

CASE PRESENTATION

A 43 years old woman admitted to our Cardiology Clinic for weakness, fatigue and chest pain in the last 3 months. In physical examination, auscultation revealed a pericardial friction rub (a metallic 3rd voice with high frequency, just after the 2nd voice in diastole), peripheral edema and hepatomegaly. Laboratory results showed hypoproteinemia and hypoalbuminemia and the other parameters were normal. Chest radiography was very specific for chronic constrictive pericarditis and it showed massive pericardial calcific deposits visible and encircling all of the heart(Figure 1). PPD (purified protein derivative of tuberculin) was negative. There was no tuberculosis anamnesis. Two-dimensional, color-flow Doppler echocardiography revealed massive pericardial calcification.
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Figure 1
Figure 1: Encircling all of the heart by massive pericardial calcific deposits image in our patient's chest radiography.

Figure 2
Figure 2: Image of encircling all of the heart by diffuse calcific deposits in cardiac catheterization.

Figure 3
Figure 3: Image of encircling all of the heart by diffuse calcific deposits and right coronary arteriography.

Thorax CT showed massive and diffuse pericardial calcific deposits. We performed cardiac catheterization with coronary angiography. We found, nonsignificant coronary artery disease and diffuse pericardial calcific deposits, the right ventricular end-diastolic pressure to be 25mmHg, the pulmonary arterial pressure to be 38/25mmHg and the left ventricular ejection fraction to be 0.55 and there was a hypokinetic contraction because adhesion (Figures 2 and 3).

Preoperatively, she was taking diuretics and digitalis at optimal doses ordered by Cardiology clinic. Preoperatively her functional capacity was in New York Heart Association (NYHA) functional class III. She went under operation.
SURGICAL TECHNIQUE

We approached via median sternotomy. We freed the pericardium in this order: first from the aorta and pulmonary artery, including the left ventricular outflow tract; then from the left and right ventricles and the left pulmonary vein orifices; and finally from the superior and inferior venae cavae. During these steps, we set the amplitude of the cautery under 60 mV to avoid causing diathermal dysfunction of the right ventricle during electrocauterization. To avoid damage to the phrenic nerves, we surgically resected the entire anterior pericardium, within 3 or 4 cm of the phrenic nerves, then completed the dissection of the pericardium from the diaphragm. Constricting layers of epicardium were removed if possible (Figures 4 and 5).

**Figure 4**
Figure 4: Remove of calcific constricting layers on pericardium, if possible.

There was no major bleeding from the pericardial edges, nor was there evidence of phrenic nerve injury either perioperatively or postoperatively. We left large plaques that did not permit the development of a cleavage plane. We performed wedge incisions that reached the epicardium, which reduced the size of the plaques and relieved myocardial constriction. The ventilation time was 6 hours. The volume of blood transfused was 1 unit. The quantity of mediastinal drainage was 200 cc. We don’t used an inotropic drug for prophylaxis against low-output syndrome in our case. The stay in the intensive care unit was day. The hospital stay was 5 days. Postoperative diagnosis was made in a histologic section of the pericardium (Figure 6).

**Figure 6**
Figure 6: This photograph showed pericardial fibrosis and calcification (Hematoxylin and eosin stain x40).

During the 1st postoperative month, the functional capacity
of our patient improved dramatically and she was in NYHA functional class I.

**DISCUSSION**

Pericardial heart disease has been found clinically in >1% of patients admitted to one large general hospital and in about 5% of consecutive necropsies at another large general hospital. Better diagnostic tools, particularly echocardiography, and prolonged longevity in many systemic illnesses have almost certainly increased the clinical recognition of pericardial heart disease in recent years.

The occurrence of calcific deposits in pericardia constitutes calcific pericardial heart disease. The calcific deposits may vary in size from microscopic, visible only by histologic examination, to massive, encircling all or most of the heart and readily visible by radiographic examination. Calcific deposits appear to represent the end stage of organization of a pericardial process and in themselves are not indicative of any specific etiology. Histologic examination of calcified pericardia rarely provides specific diagnoses. Conditions predisposing to calcific pericardial heart disease are listed in Table 1.

Table 1: Etiologic factors of calcific pericardial heart disease.

<table>
<thead>
<tr>
<th>Condition</th>
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<tr>
<td>Idiopathy</td>
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<tr>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Infection</td>
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<tr>
<td>Rheumatic fever</td>
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<tr>
<td>Trauma</td>
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For myocardial constriction to occur, the calcific deposits must encircle both ventricles. Localized bands of calcium, however, may cause cardiac dysfunction. In chronic constrictive pericarditis, there are changes in the volume elasticity slopes of both ventricles. Secondary to constriction, there is an increase in left and right ventricular end-diastolic pressures and a decrease in outflow volume. Moreover, thickened and calcified pericardium frequently comes into direct contact with the myocardium, decreasing the heart muscle's contractility and disrupting the coordination of diastolic filling of the ventricles.

To show the calcifications radiologically is very important for diagnosis. In addition angiography is useful to determine the atrial wall, rigidity of atrium and quality of atrial contraction. It shows that atrial contraction is strong in myocardial diseases and weak in constrictive pericarditis because atrium wall is adherent to pericardium with scar. However, the association of characteristic hemodynamic changes with pericardial thickness greater than 3 mm usually confirms the diagnosis.

Fibrosis and calcification can extend very severely in those patients, and this is the primary reason to perform the operation as early as possible. Chronic constrictive pericarditis of more than a year in duration decreases the success rate of surgery and increases the mortality rate. Due to early diagnosis and surgery, severe cases have become rare.

Bilateral thoracotomy, left anterior thoracotomy, and median sternotomy are all approaches that can be used in treating CCP. The sternotomy incision enables exploration of the left ventricle and right part of the heart and direct vision of the great vessels. It is possible to perform an extensive pericardiectomy with minimal cardiac manipulation. The left ventricle can be decorticated easily via sternotomy, and better cardiac hemodynamics can be achieved through a sternotomy than through a thoracotomy. Further advantages of the median sternotomy are the ease and comfort of this approach for the surgeon, the ability to initiate CPB if necessary, and the postoperative comfort of the patient; for these reasons, we used this technique. Generally, early death risk postoperatively is more than 2% in many constrictive pericarditis cases. Postoperative survival is related with age, gender and race and ranged between 55% and 90%.

Bozbuga et al reported that advanced age, atrial fibrillation, concomitant tricuspid insufficiency, inotropic support, and low cardiac output were predictors of poor survival. However, in patients with compromise of cardiac function, surgery to remove the constricting envelope is the only effective long-term treatment.

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