CASE REPORT

Massive Pericardial Effusion with Cardiac Tamponade Secondary to Cholesterol Pericarditis

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SUMMARY

The presence of cholesterol crystals in the pericardial fluid is a very rare finding of unknown pathogenesis with no more than 100 reported cases in literature. Patients with cholesterol pericarditis usually have large volume spills of slow development that are well tolerated, rarely causing cardiac tamponade or constrictive pericarditis. We report a case of cholesterol pericarditis with a severe pericardial effusion and cardiac tamponade in a patient with an uncertain diagnosis of tuberculosis.


KEY WORDS

Cholesterol pericarditis, pericardial effusion, cholesterol crystals

INTRODUCTION

Cholesterol pericarditis is a rare case of chronic pericardial effusion due to the presence of cholesterol crystals in pericardial fluid. The first case was published in 1919 by Alexander et al [1] who described the pericardial fluid as “an opaque, dark brown fluid of a scintillating gold paint appearance”. To date, there have been less than 100 cases reported [2]. The pathophysiological mechanism responsible for high levels of cholesterol and the formation of crystals in the pericardial fluid is unclear and probably varies depending on the etiology.

In the case of tuberculosis, possible mechanisms suggested include the release of cholesterol from pericardial cell membranes or from tubercular granulomas, red blood cell lysis, reduced pericardial absorption capacity or lymphatic obstruction [3].

We report a case of cholesterol pericarditis associated with tuberculosis in a 59 year old male with severe pericardial effusion and cardiac tamponade.

CASE REPORT

A 59 year old male was referred to our hospital for worsening symptoms after three weeks of evolution of progressive shortness of breath and a syncopal episode. He had no cough or sputum production or fever at any time. No hemoptysis, constitutional syndrome (asthenia, anorexia, or weigh loss) or alteration of bowel habits.

Past history included hospitalization for subarachnoid hemorrhage at 40 years age that required a ventriculoperitoneal shunt for hydrocephalus without sequelae, hypertension, smoker of 20 - 30 cigarettes per day, moderate drinker, and currently in treatment with doxazosin and losartan.

There was no history of tuberculosis but he reported having contact with carriers.

The initial laboratory test results showed a deterioration of renal function, hypoxemia, and hypcapnia. Chest X-ray revealed an enlarged cardiac silhouette, a chest CT scan and transthoracic echocardiography was performed, revealing a massive pericardial effusion with signs of cardiac tamponade and small bilateral pleural effusions.

Considering the clinical setting, the patient was admitted to the ICU to perform a pericardiocentesis to drain the pericardial effusion which proceeded without complications, draining a total of 2800 mL of brownish, cloudy, and serosanguineous pericardial fluid.
Table 1. Biochemical analysis of the pericardial fluid and blood.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reference Range</th>
<th>Blood</th>
<th>Pericardial Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12 - 18</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>40 - 54</td>
<td>33.3</td>
<td></td>
</tr>
<tr>
<td>Red Blood Cells (RBC)</td>
<td>4.60 - 5.70 x 10^6/µL</td>
<td>3.68</td>
<td>380000/µL</td>
</tr>
<tr>
<td>White Blood Cells (WBC)</td>
<td>4 - 11x10^3/µL</td>
<td>9.64</td>
<td>6000/µL</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>40 - 74</td>
<td>77.3</td>
<td>22</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>16 - 45</td>
<td>12.3</td>
<td></td>
</tr>
<tr>
<td>Monocytes (%)</td>
<td>4 - 12</td>
<td>8.5</td>
<td>78</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>1 - 7</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td>Basophils (%)</td>
<td>0 - 2</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>70 - 105</td>
<td>83</td>
<td>25</td>
</tr>
<tr>
<td>Total proteins (g/dL)</td>
<td>6.6 - 8.3</td>
<td>6.5</td>
<td>4.2</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>0 - 247</td>
<td>189</td>
<td>3049</td>
</tr>
<tr>
<td>ADA (IU/L)</td>
<td>&lt;30</td>
<td>74.2</td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>50 - 220</td>
<td>149</td>
<td>151</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>50 - 200</td>
<td>121</td>
<td>33</td>
</tr>
<tr>
<td>pH</td>
<td>7.35 - 7.45</td>
<td>7.50</td>
<td>7.32</td>
</tr>
</tbody>
</table>

Microscopic analysis of the pericardial fluid showed a high cell count with numerous red blood cells (380000/µL) and leukocytes (6000/µL) with a predominance of monocytes (78%) and neutrophils (22%), highlighting the presence of abundant square and rectangular crystals of different sizes [Figure 1a,1b], which were identified with polarized light microscopy (x400 magnification) as cholesterol crystals (31 - 100 crystals/field).

Biochemical analysis of the pericardial fluid revealed it to be an exudate with high levels of proteins, LDH, cholesterol, and adenosine deaminase (ADA). Concentrations of these parameters in blood were within normal limits [Table 1].

The cytopathological fluid study described the presence of numerous square microcrystals with isolated macrophages and was negative for neoplastic cellularity. The standard cultures for bacteria and fungi were negative. No acid-fast bacilli were seen on smear or culture. Anti-nuclear antibodies test and rheumatoid factor were within normal range, viral serology (including HIV HBV, HCV) was also negative. Tuberculin skin test (Mantoux) was positive.

Chest x-ray after pericardiocentesis showed a normal cardiac silhouette and slight to moderate pleural effusion.

EKG: sinus rhythm at 80 bpm, narrow QRS, negative T wave in the lateral asymmetric.

Echocardiography after pericardiocentesis showed slight concentric hypertrophy of LV, with preserved contractility and minimal mitral regurgitation.

After 72 hours in the ICU, the patient remained stable at all times with no recurrence of pericardial effusion, but still with slight pleural effusion. A diagnostic thoracentesis was performed obtaining a fluid of serosanguinous appearance with pH 7.56, glucose 107 mg/dL, total proteins 3.5 g/dL, LDH 170 IU/L, RBC 9600/µL, WBC 1820/µL (neutrophils 26 % and monocytes 74%). During the first days after admission the patient showed important leukocytosis 18280/µL (82% neutrophils) as well as an increase in concentration of acute phase reactants (APR), C-reactive protein (CRP) of 17.50 mg/dL and, erythrocyte sedimentation rate (ESR) of 56 mm/hour, with no fever or signs of focal infections and progressive normalization without specific treatment.

Given the clinical setting and findings in the pericardial fluid, all compatible with probable tuberculous pericarditis, the high rate of tuberculosis in our area, above 30 cases per 100000 population and the absence of data to guide other etiology, tuberculostatic treatment was started empirically with rifampicin along with isoniazid, ethambutol, and pyrazinamide until results of the microbiological cultures were available. As the patient remained stable, with a good response to treatment, no further studies were performed after microbiological results came back negative. The patient was discharged home, developing favorably and without recurrence of effusion over the last 6 months.
DISCUSSION

Cholesterol pericarditis is a rare disease of uncertain pathogenesis and many etiologies that should be considered in the course of chronic diseases that involve the pericardium. Patients with cholesterol pericarditis usually have large volume spills of slow development, so they are well tolerated and rarely cause cardiac tamponade or constrictive pericarditis [4].

Most patients present clinical symptoms of fatigue, dyspnea, and vague chest discomfort which is rarely severe. The etiology is most common to be idiopathic or associated with systemic disorders such as rheumatoid arthritis, myxedema, hypercholesterolemia or polycystic kidney disease [5]. Tuberculosis has also been suspected as a cause in some cases, due to positive tuberculin skin tests and good response on antituberculosis therapy, although no acid-fast bacilli were seen on smear or culture in any of the reported cases [6].

Figure 1a, 1b. Microscopic image of pericardial fluid with cholesterol crystals (x400 magnification).
The diagnosis is based on the casual finding of high cholesterol levels and/or cholesterol crystals in the pericardium of patients with pericardial effusion [7]. Treatment will normally relate to the underlying disease process, but in refractory cases and those who evolve to tamponade or constriction, pericardiectomy may be a good therapeutic option [8]. Our case is of particular interest due to the presentation with cardiac tamponade requiring pericardiectomy; tuberculosis was suspected as etiology but, as in other cases reported, was not proved by microbiological studies. However, the patient had a good response to therapy and no recurrence of the pericardial effusion.

References:


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