Etiological Profile, Clinical Features and Medical Management of Acute Pericarditis in Burkina Faso


Cardiology department, Teaching hospital Yalgado Ouedraogo, France

Abstract

Diagnosis of acute pericarditis is sometimes difficult but the first challenge to the clinician is to establish an etiologic diagnosis. This study aimed to describe etiologies of this pathology and its clinical pictures, and expose the medical treatment and outcome, at the time of the success of antiretroviral treatment.

Through a prospective study, we follow up a sample of patients presenting acute pericarditis from January 2010 to December 2011. There were 43 cases composed of 20 men and 23 women. The average age was 41.4 ± 16.1 years. The leading clinical signs were infectious syndrome (100%), dyspnea (86%), and chest pain (79.1%). Right heart failure was found in 27.9% of cases, hypotension in 23.2% of cases and pulsus paradoxus in 20.9% of cases. Echocardiography found pericardial effusion in 88.4% of cases. This effusion was of great amount in 26.3% of cases of pericardial effusion. Cardiac tamponade was encountered in six cases. We identified an etiology in 88.4% of cases. These etiologies were tuberculosis in 47.4% of cases. As treatment, anti-tuberculous drugs associated to corticosteroids were systematic in tuberculous forms. In other cases, we used aspirin associated to corticosteroids or colchicine as appropriate. Six cases of recurrence were observed in exclusively tuberculous forms. Two cases of deaths were occurred.

Keywords: Acute Pericarditis; HIV; Tuberculosis; Antiretroviral Treatment

Introduction

Acute pericarditis is a common finding in everyday clinical practice [1]. The diagnosis is sometimes difficult but the first challenge to the clinician is to establish an etiologic diagnosis. In sub-Saharan Africa, tuberculosis and HIV are often indexed rightly or wrongly as etiology [2-4]. Indeed, since the apparition of HIV infection, the viral and tuberculous pericarditis has increased in our continent [5]. But many efforts have been made to treat this disease across the continent. The etiological profile of pericarditis would it has changed so far? We wanted through this study, to describe acute pericarditis etiologies and its clinical pictures, and expose the medical treatment and outcome, at the time of the success of antiretroviral treatment.

Patients and Method

From January 2010 to December 2011, we conducted a prospective study in two medical centers in the city of Ouagadougou (Yalgado Ouedraogo teaching hospital and Saint Camille medical center). We consecutively included all patients admitted for acute pericarditis. All patients underwent medical examination. We looked for infectious syndrome, chest pain, dyspnea or nonspecific chest discomfort, cough, infection of the upper airways, signs of systemic or connective disease and emaciation. In the background, we sought episodes of pericarditis, systemic and connective disease and comorbidities. During examination, we have researched jugular venous distension, pulsus paradoxus, arterial hypotension and shock. A blood sample was taken for laboratory examinations composed of blood count, creatinine, glucose, uric acid, electrolytes, uTSH, sedimentation rate, CRP, fibrinogen, HIV serology, CCP2 antibodies, anti dsDNA. In patients suffering from cough, we searched for acid-fast bacilli-resistant. The tuberculin skin test was also performed. We also conducted a cytological, histological, bacteriological and biochemical study of pericardial fluid in patients who underwent pericardiocectomy.

In all patients we performed a 12-lead ECG and echocardiography. At ECG, we calculated heart rate, sought repolarisation disorders, alternating electric and possible arrhythmia. In echocardiography, we measured the thickness of the pericardium (next to the anterior wall of the right ventricle at subcostal 4 chambers) and the size of the cavities. When pericardial effusion was present, we quantified the amount of effusion and appreciated hemodynamic compromise. We have made a list of all met etiologies and appreciated the outcome under treatment. Patients were followed for at least 6 months. We searched recurrence and pericardial constriction.

Results

During the period of study, 43 cases of acute pericarditis have been admitted to the two medical centers. The sample was composed of 20 men and 23 women. The average age was 41.4 ± 16.1 years (range 19 and 90 years). The leading clinical signs were infectious syndrome (100%), dyspnea (86%), and chest pain (79.1%). Clinical signs encountered are summarized in Table 1.

Right heart failure was found in 27.9% (12 cases) of cases, hypotension in 23.2% (10 cases) of cases and pulsus paradoxus in 20.9% (9 cases) of cases. At laboratory tests, more than half of the patients (67.4%) had leukocytosis. Anemia was found in 44.1% and HIV serology was positive in 8 cases (18.6%). Table 2 summarizes biological salient results.

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Chest X-ray and electrocardiogram were contributory. Signs revealed by radiography were cardiomegaly in 79% of cases (figure 1) and pneumonia in 35.8% of cases. As for the ECG, it found tachycardia and repolarization disorder in all cases, low voltage in 53.4% (figure 2) of cases and 41.8% of alternating electric. Echocardiography found pericardial effusion in 88.4% of cases. This effusion was of great amount in 26.3% of cases of pericardial effusion. Cardiac tamponade was encountered in six cases (15.7% of pericardial effusion) (figures 3 and 4). Swinging heart was found in eight patients.

We identified an etiology in 88.4% of cases (38 patients). These etiologies were tuberculosis in 47.4% of cases (18 cases), acute benign forms (viral) in 26.3% (10 patients) of cases, HIV infection in 21% of cases (8 patients) and neoplasia in 5.3% of cases (2 patients). In other cases (five cases or 11.6%) no etiology was found. When we considered tuberculous pericarditis, acid-fast bacilli was demonstrated in 11 cases (61.1%). In other tuberculous cases, the etiology was retained before the clinical tuberculous impregnation signs, lymphocytosis, presence of fibrin in the pericardial fluid and exudative criteria of this fluid.

As far as the treatment is concerned, anti-tuberculous drugs associated to corticosteroids were systematic in tuberculous forms. In other cases, we used aspirin that was associated to corticosteroids in five cases in which the evolution was considered unsatisfactory, and colchicine in 12 cases. The pericardiocentesis was done in 26.3% of

<table>
<thead>
<tr>
<th>Sign</th>
<th>Frequency</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Fever</td>
<td>43</td>
<td>100</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>37</td>
<td>86</td>
</tr>
<tr>
<td>Chest pain</td>
<td>34</td>
<td>79.1</td>
</tr>
<tr>
<td>Cough</td>
<td>21</td>
<td>48.8</td>
</tr>
<tr>
<td>Emaciation</td>
<td>12</td>
<td>27.9</td>
</tr>
<tr>
<td>Pericardial friction rub</td>
<td>9</td>
<td>20.9</td>
</tr>
<tr>
<td>Nonspecific chest discomfort</td>
<td>7</td>
<td>16.3</td>
</tr>
<tr>
<td>Ent Throat infection</td>
<td>6</td>
<td>13.9</td>
</tr>
</tbody>
</table>

Table 1: frequency of clinical signs found in 43 patients with acute pericarditis.

<table>
<thead>
<tr>
<th>Biological sign</th>
<th>Frequency</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Increased sedimentation rate</td>
<td>43</td>
<td>100</td>
</tr>
<tr>
<td>Increased CRP</td>
<td>41</td>
<td>95.3</td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>29</td>
<td>67.4</td>
</tr>
<tr>
<td>Anemia</td>
<td>19</td>
<td>44.2</td>
</tr>
<tr>
<td>Lymphocytosis</td>
<td>12</td>
<td>27.9</td>
</tr>
<tr>
<td>Positive tuberculin skin test</td>
<td>9</td>
<td>20.9</td>
</tr>
<tr>
<td>Increased creatinine</td>
<td>4</td>
<td>9.3</td>
</tr>
<tr>
<td>Increased uric acid</td>
<td>4</td>
<td>9.3</td>
</tr>
</tbody>
</table>

Table 2: Biological salient results in 43 patients with acute pericarditis.
effusion cases (10 patients) and drainage in 28.9% of effusion cases. The outcome was favorable in 74.4% of cases. Six cases of recurrence were observed (13.9%). These recurrences occurred only in tuberculous forms as pericardial constriction that occurred in five cases. Unfortunately we recorded two deaths (4.6%).

Discussion

Acute pericarditis is frequent in Africa. In two years of study, we recruited 43 patients, compared to Khoueir et al. who have recruited 73 patients in 4 years [6] and Cohen et al. who have recruited 55 patients in 3 years [7].

In the opinion of several authors, the etiologies of pericarditis in Africa are often tuberculous [3,8-11]. The incidence of tuberculous pericarditis in sub-Saharan Africa is increasing as a result of the Human Immunodeficiency Virus (HIV) epidemic, and this trend is likely to appear in other parts of the world where the spread of HIV is leading to a resurgence of TB [10,11]. This seems still today, despite the efforts made in the fight against HIV. In our study indeed, tuberculous etiology was the most frequent. It was found in 47.4% of cases. In the Western Cape, one half of the patients presenting with large tuberculous pericardial effusions are infected with HIV [3]. In one series from the Western Cape Province of South Africa, tuberculous pericarditis accounted for 69.5% (162 of 233) of cases referred for diagnostic pericardiocentesis [3]. By contrast, tuberculous pericarditis accounts for only 4% of cases in developed countries [9]. Recent reviews on the diagnosis and management of pericardial disease have paid scant attention to tuberculous pericarditis, which is arguably the leading cause of pericarditis in the world [12,13].

The most frequent etiologies of pericardial effusion were neoplastic (36%) in the series by Colombo et al. [14]. In his study, 44% of patients presented with cardiac tamponade whereas in ours it represented 15.7% of pericardial effusion. In a small proportion etiology is not found. This is the case in our study with 5 cases. In Corey et al. [15] investigation, no etiology was found in 4 patients. The usual clinical manifestations and ECG anomalies of acute pericarditis are well known [13,16], but the original clinical picture can be very atypical. In our study, most of the patients had symptoms suggestive. Classically, the diagnosis is primarily clinical and electricity, based on at least two of the following three criteria: chest pain suggestive, pericardial friction rub, ECG abnormalities compatible [13,16-18].

We have used anti-TB drugs, corticosteroids, aspirin and colchicines as appropriate. Aspirin and non-steroidal anti-inflammatory drugs are mainstay of therapy with the possible adjunct of colchicine [19]. Corticosteroids are a second choice for difficult cases requiring multi-drug therapies and specific medical conditions. Non-steroidal anti-inflammatory drugs remain first-line therapy for uncomplicated acute pericarditis, although colchicine can be used concomitantly with non-steroidal anti-inflammatory drugs as the first-line approach. Systemic corticosteroids can be used in refractory cases or in those with immune-mediated etiologies, although generally should be avoided due to a higher risk of recurrence [20].

Anti-tuberculosis chemotherapy increases survival dramatically in tuberculous pericarditis [21]. In our study, no tuberculous pericarditis died. The effectiveness of treatment with corticosteroids in tuberculous pericarditis remains controversial [13,22,23]. Constrictive pericarditis is one of the most serious sequelae of tuberculous pericarditis [9,24]. TB is said to be the most frequent cause of constrictive pericarditis in Africa and Asia [8]. Constrictive pericarditis occurred in 5 cases and all were tuberculous.

Conclusion

Acute pericarditis is common in everyday cardiologic practice. The lead clinical manifestations are infectious syndrome and chest pain. Etiologies are always dominated by tuberculosis. The prognosis is guarded given the large number of recurrence and pericardial constriction. Much work remains to be done in the fight against tuberculosis.

Authors’ contribution

All authors participated in the writing of this article and gave their consent to its submission.

References


