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Treatment if Pericarditis in an HIV-Infected Patient in a Regional Hospital in Thies (Senegal)

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Summary

Pericarditis is a common and serious cardiovascular disease during HIV infection. In sub-Saharan Africa, tuberculosis is the most common etiology; however, other infectious causes of pericarditis pose a problem of diagnosis and treatment. We report the case of a patient living with HIV 1 for 3 years without ARV treatment who developed a purulent pericarditis treated at regional hospital of Thiès.

We highlight the diagnostic difficulties of this condition in the decentralized zone and the means of treatment of this pathology, fatal if it is not treated early.

Keywords: Purulent pericarditis; HIV infection; Diagnostic difficulties

Introduction

HIV seroprevalence in the Thiès region is 0.3% in 2017. The treatment of PLWAs has been decentralized to the regions and health districts since 2013 with free antiretroviral treatment [1].

a) The immunodepression induced by this virus is at the origin of several affections and/or opportunistic infections responsible for morbidity and mortality, among which cardiac involvement with histological lesions in 60% of cases and clinical expression in 30% of cases.

b) The involvement of the pericardium occupies an important part among these cardiac attacks.

c) The etiological factors of pericarditis vary with the course and magnitude of HIV-induced immune deficiency.

Various causes of pericarditis have been reported in the literature but pericarditis of unidentified (idiopathic) cause can reach 45% of cases [2]. We report the case of an HIV-infected patient who developed a purulent greenish pericarditis of unknown etiology. The objective of this case presentation is to highlight the diagnostic difficulties of this condition in a decentralized environment.

Observation

Mr. Y N is a 26-year-old man, who has sex with a well-known man (MSM), single without children, from the city of Thiès. He presented himself in consultation at the hospital of Thiès October 10, 2016 for a quintessential cough with a notion of hemoptysis of low abundance not objectified. A retrosternal thoracic pain rhythmized by the breathing and a dyspnea with orthopnea type evolving since 15days and exacerbated for a few hours. He also complains of a progressive weight loss of 24kilos in 3years.

For only antecedent, this patient was diagnosed infected with HIV 1, asymptomatic, after a voluntary screening 3 years ago without treatment because He cannot be found. At admission, the patient had a fever at 38.3°C, an impairment of general condition, he was sleepy and cachectic weight 42 kg with BMI: 12.8Kg/m2.

Clinical examination revealed NYA grade 4 dyspnea, tachycardia at 108beats/min and arterial hypotension (BP 100/50mmHg). The heart sounds were muffled. Pericardial friction was not perceptible. There was no paradoxical pulse. Crackling rattles were heard on auscultation at the two pulmonary bases.

The rest of the examination found diffuse prurigo and oropharyngeal candidiasis.

The electrocardiogram revealed, in addition to sinus tachycardia, a peripheral low voltage with probable right ventricular hypertrophy, while the chest X-ray showed cardiomegaly and bilateral low-grade pleural effusions.

Complementing the exploration, transthoracic echocardiography confirmed the presence of a pericardial effusion of great abundance without fibrin network. The exploratory puncture aims to produce a purulent yellow-

greenish liquid. A percutaneous drainage under xiphoidal under local anesthesia was performed, allowing the evacuation of two liters of greenish-yellow pus free with good tolerance without complications.

The biology revealed an inflammatory syndrome with a CRP of 61.76mg/l and an accelerated sedimentation rate at the first and second hours. There was; microcytic hypochromic anemia at 5.5g/dl, 78.7% neutrophil polynucleosis. Cytobacteriological examination of the pericardial fluid identified 65% of lymphocytes, gram-negative bacilli, but the culture on solid medium was negative. GeneXpert MTB/RIF performed on sputum and pericardial fluid was negative.

As treatment, the patient received an intravenous administration of Ceftriaxone at a rate of 100mg/kg/day; Metronidazole 30mg/kg/day. It was temporarily (3 days) associated with gentamicin (3mg/kg/day) and Prednisone 20mg/day for 5 days. He had also received Furosemide Injection: 40mg/day, Captopril 25mg daily; Enoxaparin 0.4 per day; DL-lysine acetylsalicylate 100mg/day. The evolution was marked on day 6 of treatment by a disappearance of cough and dyspnea as well as apyrexia (37°4).

Cardiac echocardiography on day 14 showed a poor pericardial effusion plate insufficient for surgical drainage. The patient was put on antiretroviral therapy and on cotrimoxazole prophylaxis.

The patient was seen six months after discharge in cardiology for signs of late complication; it was stable, with a weight gain of 12kg. Moreover, the M6 ultrasonography showed a quasi-total regression of the pericardial effusion with a slight thickening of the pericardium.

Discussion

This case of purulent pericarditis is singular by its clinical presentation in a PvVIH lost sight of 3 years.

Many HIV-infected patients have cardiac involvement [3]. The prevalence of pericardial lesions is regularly reported in all African clinical series: nearly 28% in the Democratic Republic of Congo (DRC) and 35.3% in Congo Brazzaville [2]. In Senegal, already in 1984, D. SOW et al reported a prevalence of purulent pericarditis of 11% in pediatric hospitalized children [4]. I. Thiam found 2.5% of cases of tuberculosis pericarditis with microscopic discovery [5]. Ngouala G. in a study conducted in a decentralized area (Louga) found 3.7% of pediatric cases of tuberculous pericarditis [6].

Pericarditis remains however a serious and rare entity which is almost always fatal because of a late management. The presence of clinical signs and paraclinical examinations allow rapid diagnosis in order to start early treatment [7]. This patient had advanced dyspnea, febrile chest pain on a febrile deterioration of the general condition that had been evolving for 15 days, and effusions indicating delayed diagnosis [3].

The dominant functional symptomatology in the literature is dyspnea of effort and deterioration of the general state. This finding has been made in Africa by several authors [3,8]. Fever is also a reason for frequent consultation. For Cohen, the presence of fever and dyspnea is suggestive of purulent pericarditis or myocarditis [8].

The deafening of heart sounds and tachycardia were the stethoacoustic signs present in our patient and most found in the Niakara and Pio studies, which reported respectively 43.7% and 47.5% of cases [4,8].

The low rate of pericardial friction reported in the literature may be related to the abundance of pericardial effusion [8,9]. He was not present with our patient. Acute pericarditis can be caused by a wide variety of etiologies, which can be infectious or non-infectious [10].

Possible causes include connective tissue disorders, malignancies, radiation, heart lesions, uremia, and infections (including viral, bacterial, and fungal etiologies) [10,11]. In the majority of cases (45 to 80%), infectious pericarditis is of viral origin [11,12].

Bacterial pericarditis is a rare cause of acute pericarditis in the era of modern antibiotics with an incidence of less than 1% [13,14]. The most common living microorganisms involved are *Streptococcus sp, Staphylococcus sp, Haemophilus sp and Mycobacterium tuberculosis* [15,16].

Pericarditis in a seropositive person, living in Africa in addition, should first look for a tuberculous etiology [3,5,6]. It occurs most often in the early stage of infection, but can also occur in the AIDS stage [3].

No primary infectious focus was found in our patient; the etiological investigation was difficult in our working conditions in the decentralized zone because of the absence of a performing microbiological laboratory, however the favorable evolution under probabilistic antibiotherapy with broad spectrum allowed us to retain the hypothesis of a cause non-specific bacterial.

Predisposing conditions for bacterial pericarditis are immunosuppression, malignant tumors, pre-existing pericardial effusion, alcoholism, uremia, thoracic trauma, cardiac and thoracic surgery, and the insertion or use of catheters for drain the pericardial fluid [15]. This case of pericarditis occurred on HIV-related immunosuppression. The Niakara study in Ouagadougou had a seroprevalence of HIV of 47% out of 79 cases followed for 75 months from 1993 to 1999, reinforcing the work that established a correlation between HIV infection and pericarditis especially in Black Africa [3]. During HIV infection, the occurrence of pericarditis with pericardial effusion is common.

The mechanisms are multiple; pericarditis may be related to viral infection by HIV or other viruses, bacterial or fungal superinfection in an immunocompromised patient or the

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presence of Kaposi's lymphoma or sarcoma [3,10,15]. Laboratory assessment may reveal systemic inflammation with leukocytosis and elevated CRP and SV [15]. The chest X-ray usually shows cardiomegaly with an abnormal heart shape. Pulmonary infiltrates, pleural effusion and mediastinal enlargement may also be present [15,16].

The ECG often makes it possible to evoke the diagnosis, especially in its acute form by the Holtzman stages. The signs that we observed at the ECG have no specificity as demonstrated by a piece of literature [9].

In our case, none of the paraclinical examinations were directed to the diagnosis of pericarditis. Echocardiography remains the first-line imaging examination for the diagnosis of pericardial effusion by specifying abundance and location. The abundance of the effusions facilitates the ultrasonography diagnosis of these pericarditis [3,9], as was the case in our patient; he had significant pericardial effusion without cardiac tamponade.

The treatment is based on probabilistic antibiotherapy in the absence of bacteriological data of pericardial fluid and / or blood culture. Surgical drainage is often necessary [8,9]; it allowed our patient to evacuate two liters of greenish yellow pus without complications. Even under treatment, the rate of complications and deaths remains high, with a lethality rate close to 40% secondary to tamponade, pericardial constriction or sepsis [15-17].

Conclusion

Although rare, purulent pericarditis usually responds to probabilistic treatment provided that a tuberculosis etiology is eliminated even in a health facility with limited means. Early recognition and rapid intervention are essential to the success of the treatment whose etiological research is confronted with daily technical difficulties in a decentralized environment.

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