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ACUTE PURULENT TUBERCULOUS PERICARDITIS WITH CARDIAC TAMPONADE: A CASE REPORT.

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ABSTRACT

Tuberculosis is a disease caused by Mycobacterium tuberculosis. Pulmonary localization is the most frequent. However, tuberculous pericarditis can cause fatal complications. A 37 year old man, without pathological history, presented with mid-thoracic pain associated with dyspnea, preceded, 10 days earlier, by fever and night sweats..Diagnosis of tamponade was confirmed by trans-thoracic echocardiography. Ultrasound-guided diagnostic and therapeutic pericardiocentesis were performed. Bacillus research by PCR and culture in the middle of *Lowenstein* later returned negative. Lab tests revealed an important infectious syndrome. Taking into account the epidemiological profile of the country, diagnosis of primary tuberculous tamponade was certain. Quadruple anti- tuberculosis treatment associated to corticosteroid therapy was administered, resulting in a good outcome. The patient became asymptomatic, and follow-up echocardiography found no signs suggesting chronic constrictive pericarditis

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INTRODUCTION

Tuberculosis is a disease caused by mycobacterium tuberculosis, discovered in 1882 by Robert Koch. This condition prevails in the most vulnerable populations. The treatment success exceeds 85 %, with a detection of 95 %, which has decreased incidence of mortality [1]. The number of new cases detected remains concentrated around the major cities of the Moroccan kingdom [2]. In 2012, 83 new cases per 100 000 inhabitants were detected in Morocco, according to the epidemiological services of the Moroccan Ministry of Health [1].Pulmonary localization is the most frequent [3, 4]. However, although rare, tuberculous pericarditis can cause fatal complications, and without immediate treatment often results in a poor prognosis.

CASE REPORT

A 37-year-old man, without medical history, presented to the Emergency Room with a sudden onset of mid-thoracic pain associated with dyspnea, which had been preceded, 10 days earlier, by fever, night sweats, important fatigue and slimming.

These symptoms persisted despite self-medication by Ciproxin, Acetaminophen and non-steroidal anti-inflammatory drugs.

On clinical examination, the patient was in poor general condition, restless, and suffering from dyspnea with a respiratory rate at 40 breaths per minute. He had fever at 39° to 40°. Cardiovascular examination revealed tachycardia at 125 beats per minute, with paradoxical pulse, and a blood pressure at 110/70mmHg. There was a spontaneous jugular venous distension and a tender hepatomegaly. Auscultation showed muted heart sounds without pericardial friction. The remaining physical examination was unremarkable.

Chest radiography showed cardiomegaly with symmetric edge straightness. Electrocardiogram showed sinus tachycardia at 125bpm, microvoltage, electrical alternation of QRS complexes, and a diffuse ST segment elevation. (Fig.1).

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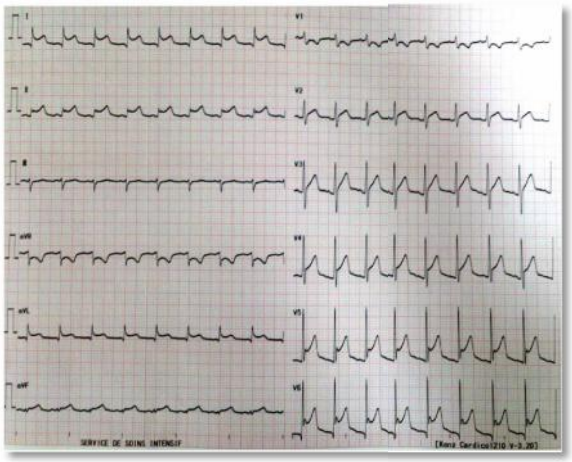

Figure 1: Electrocardiogram revealing sinus tachycardia, microvoltage and electrical alternation of QRS complexes, and a diffuse ST segment elevation

Diagnosis of tamponade was suspected, then confirmed by trans-thoracic echocardiography revealing abundant circumferential pericardial effusion measuring 40mm, with prolonged collapse of the right atrium and ventricle, paradoxical septum, (Fig. 2,3) and significant changes in the inspiration flow.

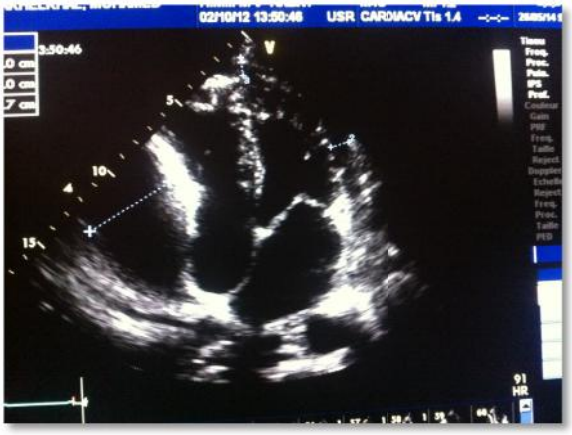

Figure 2: Transthoracic echocardiography showing abundant circumferential pericardial effusion

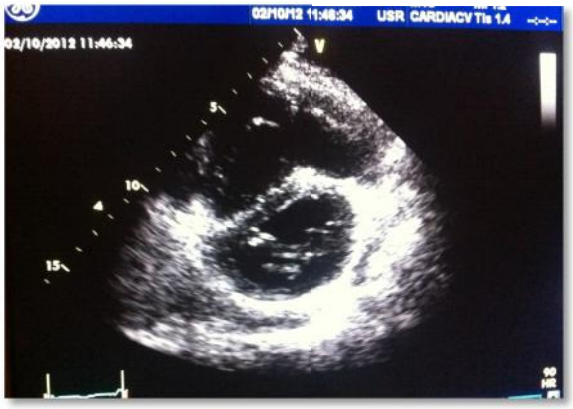

Figure 3: Trans-thoracic echocardiography showing paradoxical septum

This pericardial effusion had a dense inhomogeneous echogenicity, with some hyperechoic bands. Ultrasound-guided diagnostic and therapeutic pericardiocentesis was carried out. Two liters of a cloudy yellow and slightly viscous pericardial fluid were gradually evacuated (Fig. 4).

Figure 4: aspect of the pericardial fluid.

Direct examination revealed the presence of 14 400/mm^3 white cells (99 % neutrophils) and gram-negative bacilli. Bacillus research by PCR and culture in the Lowenstein medium later returned negative. Lab tests revealed an important infectious syndrome with 29 700/mm^{3}of white blood cells, predominantly neutrophils, CRP 322 mg /l, Sedimentary rate42 mm, fibrinogen 7.61g /l and pro – calcitonin47.53ng /ml.

When considering the epidemiological profile of Morocco regarding tuberculosis, the diagnosis of a primary tuberculous tamponade was certain. Quadruple anti- tuberculosis chemotherapy associated to corticosteroids was administered. Consequently were noted the disappearance of dyspnea, a blood pressure increase to 120/75 mmHg, and a heart rate decrease to 95bpm. Electrocardiogram showed the disappearance of microvoltage, Trans-thoracic echocardiography

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showed a decrease of pericardial effusion abundance to less than 5mm, and lab tests revealed a pseudo- normalization of the infection markers: CRP 24mg/l, SR 12 mm, fibrinogen 3.24g /l, and pro-calcitonin 0.009ng/ml.
After 12 months, the patient was asymptomatic, with a complete remission. Follow-up echocardiography found no signs suggesting a chronic constrictive pericarditis. Epidemiological investigation in the patient's family was unremarkable.

DISCUSSION

Despite its rapidly progressive atypical presentation, and purulent character in an immune-competent subject, this observation reminds us that tuberculosis will continue to surprise us with its clinical polymorphism.
Pericardial location represents 1-2 % of extra-pulmonary tuberculosis [5]. This prevalence may reach 65 % in HIV-infected patients [6,7]. It primarily affects young patients under 40 years [7, 8]. Pericardial location is usually secondary to bacterial spread by way of hematogenous or lymphatic dissemination but rarely by contiguity [9]. In ganglion locations, mediastinal involvement is the most common after cervical involvement, 54 % versus 70% [10]. In a study of 221 patients, CT scan objectified presence in 60% of cases of lymphadenopathy in the lodge BARETY; Identification of an inflammatory lymphadenopathy should guide diagnosis to tuberculosis, especially in the presence of signs of tubercular infiltration. Tuberculous pericarditis is chronic or sub-acute. It is usually responsible for abundant but well tolerated pericardial effusion [11]. The evolution towards tamponade is rare and is seen in the context of a chronic tuberculous pericarditis or in immunodeficiency [12]. Acute onset tuberculous pericarditis, with rapid progression to tamponade, has not been reported in medical literature [13].
This atypical clinical form in our patients is also characterized by the purulent nature of the pericardial effusion. Usually, the presence of purulent pericardial fluid indicated the implication of pyogenic germs. Another unique aspect of our biological observation was the predominance of neutrophils in the pericardial fluid, which is extremely rare, and the tuberculin anergy, that is rare in adult population. It would be a limiting factor necessary for the expression of cellular immunity responsible for tuberculin anergy cooperation [14].
The diagnosis of tuberculous pericarditis is initially suspected in the presence of classic signs of tuberculosis, called impregnation, and often associated with chronic dry cough and precordial chest pain [15]. Trans-thoracic echocardiography is essential to diagnosis, especially in the case of an insidious disease with inaugural tamponade. It's typically identified as fibrous fringes appendues visceral pericardium, but non-specific to tuberculosis. Microbiological diagnosis is rarely obtained [16, 17]. Identification of the bacillus of Koch in the pericardial fluid in our patient was negative. The response to treatment was a valuable diagnosis argument in our case. It is based on a combination of quadruple antituberculosis chemotherapy (isoniazid, rifampicin, pyrazinamide and ethambutol) for two months, supported by dual therapy (isoniazid and rifampicin) for four more months [18]. Indication for oral corticosteroid therapy remains controversial. Its benefit in terms of mortality reduction and prevention of progression to constriction is uncertain [19]. Constrictive pericarditis is a possible severe complication, whose frequency increases from 31.65 % to 52.74 % when the pericardial fluid is purulent [20]

CONCLUSION

In comparison with other cases reported in the literature, our case highlights the great clinical and biological tuberculosis polymorphism. It appears that pericardial location, although rare, is unpredictable and maybe life-threatening. The diagnosis by isolation of Koch Mycobacterium remains difficult and time consuming. Every clinician is led to suspect and suggest the diagnosis of tuberculosis whenever the general signs are highly suggestive, especially given the epidemiological context of the patient's geographical location. In our patient, the rapid introduction of anti-tuberculosis treatment reduces short-term and long-term complications, such as tamponade and pericardial constriction.

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