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Case Report
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A Case of Acute Tuberculous Pericarditis Evolving into Pericardial Constriction after 32 Years : A Case Report

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Abstract

Background: *Chronic constrictive pericarditis (CCP) is a rare condition. Pathophysiologically, it is characterized by inflammation and morphological changes of the pericardium leading to a presentation of adiastole. Etiologies are multiple, and tuberculosis remains a frequent cause in developing countries. Multimodal imaging has revolutionized the diagnosis of chronic constrictive pericarditis, and effective treatment remains pericardial decortication followed by etiological treatment whenever possible.*

Case Présentation: *We report the case of a 55-year-old female patient who presented with dyspnea and signs of right heart failure evolving in a context of asthenia. Transthoracic echocardiography and thoracic CT scan helped to diagnose chronic constrictive pericarditis, with tuberculosis as the suspected etiology based on the patient's history. The patient was initially treated medically, but due to persistent signs of systemic congestion, a diagnostic and therapeutic pericardial decortication was performed. Histopathological examination of the operative specimen confirmed Mycobacterium tuberculosis infection.*

Conclusion: *Chronic constrictive pericarditis is a rare but serious condition. Clinical presentation can vary from chronic fatigue to a refractory right-sided heart failure. Tuberculosis remains a relevant pathology in developing countries and should therefore always be suspected even in the context of previously treated tuberculosis. Pericardiectomy remains the gold standard in the effective management of this condition.*

Keywords: Chronic Constrictive Pericarditis, Tuberculosis, Pericardial Decortication.

Introduction

Chronic constrictive pericarditis (CCP) is a rare condition with a challenging diagnosis. In the literature, reported prevalence of constrictive pericarditis is around 0.02% of all cardiac diseases [1,2]. Pathophysiologically, it is characterized by inflammation of the pericardium and its transformation into a rigid, fibrocalcific, inextensible shell, thus forming a true casing enveloping both ventricles, leading to a hemodynamic picture of adiastole [2, 3]. In the Western world, the main causes of chronic pericardial constriction are idiopathic or viral, followed by irritation after sternotomy and mediastinal radiotherapy. However, in developing countries and among immunocompromised patients, tuberculosis remains a major cause [4].

Early and clinical diagnosis remains difficult, hence the importance of multimodal imaging. Confirming the diagnosis of con-

strictive pericarditis is crucial as surgical intervention may be the only way to relieve symptoms. A normal pericardium has a thickness of less than 2 mm, and a thickness greater than 6 mm is specific for constriction [5].

Case Report

We report the case of a 55-year-old woman with no modifiable cardiovascular risk factors who presented to our emergency department with New York Heart Association (NYHA) stage III dyspnea evolving for six months, associated with fatigue and presyncope episodes. As past medical history, the patient was treated for acute tuberculous pericarditis in 1992.

On admission, clinical examination revealed normal blood pressure (BP) and heart rate (HR) (BP= 117/70 mmHg, HR=81 beats/min). Cardiovascular examination showed signs of right heart failure (jugular venous distention, hepatojugular reflux, ascites with bilateral lower limb edema), and a split second heart sound at the pulmonary area. Additionally, there were no signs of left heart failure.

The electrocardiogram [Fig1] showed atrial fibrillation rhythm with an average ventricular rate of 83 beats per minute.

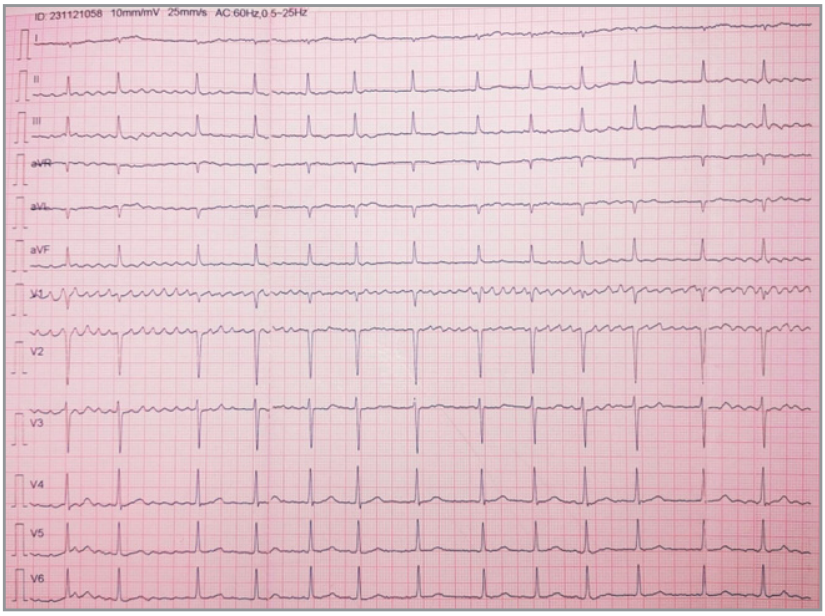

Figure 1: Resting electrocardiogram showing atrial fibrillation.

Transthoracic echocardiography [Fig2] revealed a non-dilated, non-hypertrophied left ventricle with good overall contractility and presence of septal bounce. Left ventricular function was preserved at 66% by Simpson's Biplane method. The E-wave velocity was 90 cm/s with a short E-wave deceleration time (EDT) of 24 ms. Atrial chambers were dilated (left atrium surface area at 24 cm2, right atrium surface area at 22.3 cm2) with a calcified appearance of the pericardium. The right ventricle was non-dilated. The inferior vena cava was dilated at 26 mm, with a collapsibility index of less than 50%.

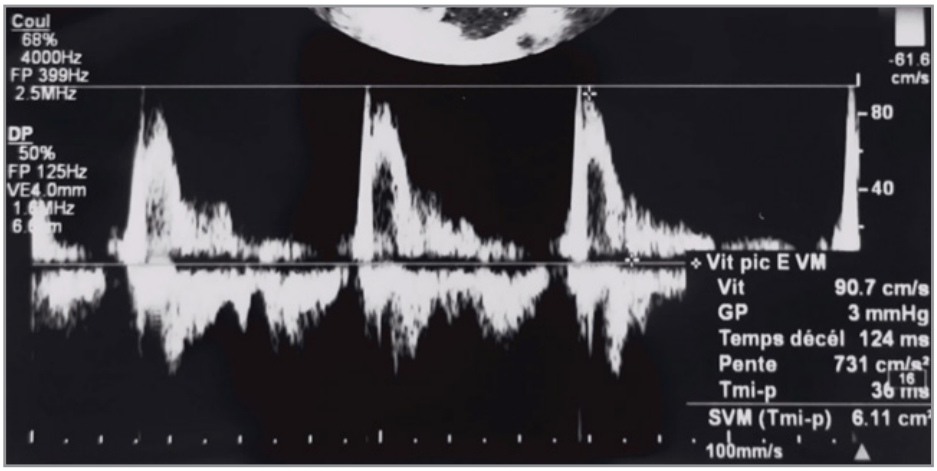

Figure 2: Doppler echocardiography revealing restrictive mitral inflow velocity.

The thoraco-abdominal CT scan revealed thoracic findings of cardiomegaly with diffuse pericardial thickening and calcifications [Fig3]. In the abdominal region, the liver was enlarged, exhibiting heterogeneous parenchymal enhancement with mosaic perfusion pattern, and there was significant ascites.

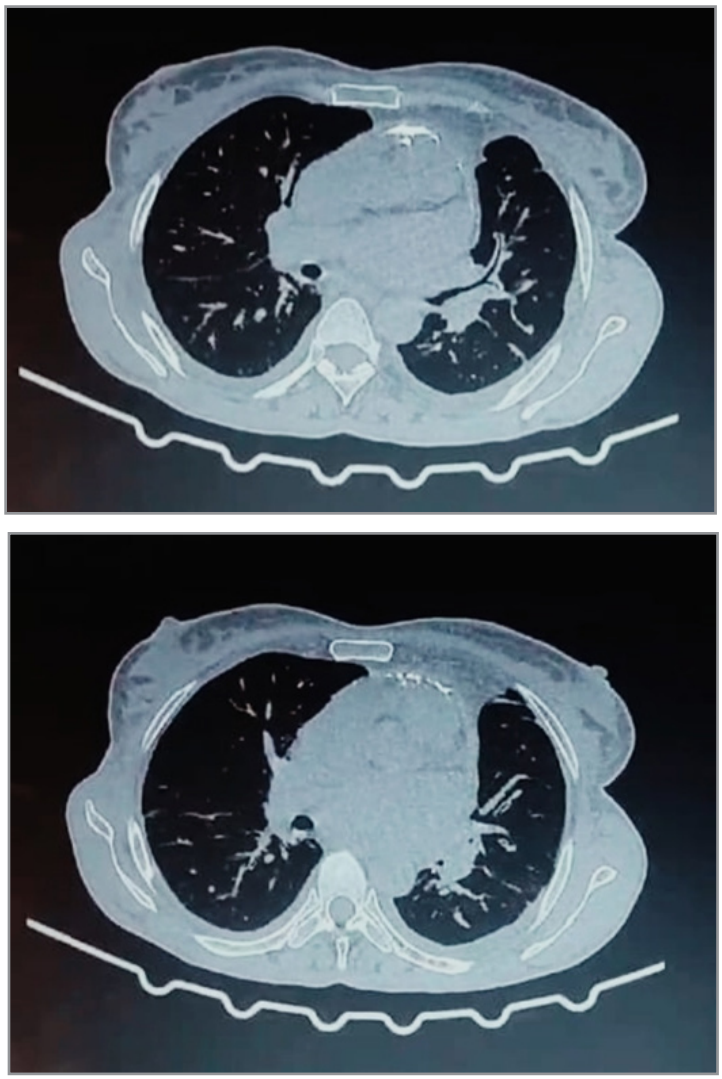

Figure 3: Chest CT revealing cardiomegaly with diffuse pericardial thickening and calcifications.

In light of these signs, the diagnosis of chronic constrictive pericarditis was made. On the therapeutic front, the patient was started on Furosemide 250 mg/day, Spironolactone 75 mg/day, and therapeutic dose of Enoxaparin for her atrial fibrillation. Given the persistence of systemic congestion signs and significant functional impairment, the decision was made to proceed with surgical pericardial decortication.

The patient underwent a beating-heart pericardiectomy. The intraoperative diagnosis supported chronic constrictive pericarditis, evidenced by the presence of thickened, calcified pericardium encasing the heart, which was rigid. The histopathological examination of the operative specimen revealed an infection with Mycobacterium tuberculosis, for which the patient was started on anti-tuberculous treatment with good clinical progress.

Discussion

Chronic constrictive pericarditis results from chronic inflammation of the pericardium secondary to insults that can be associated with various etiologies, summarized non-exhaustively in Table 1 [6]. The prevalence of tuberculous origin chronic constrictive pericarditis is poorly understood but remains a common cause, particularly in developing countries [7].

Table 1: Causes of constrictive pericarditis

Idiopatic or viral
Postinfectious
Tuberculosis or purulent pericarditis
Bacterial, fungal, or parasitic
Radiation exposure and neoplastic disease
Postcardiac injury syndrome
After cardiac surgery
Post myocardical injury (Dressler's syndrome)
Postpericardiotomy syndrome
Connective tissue disease

The main hemodynamic abnormality in constrictive pericarditis is characterized by the loss of pericardial compliance, leading to a dependence on elevated ventricular pressures to maintain adequate ventricular filling and cardiac output. These mechanisms collectively result in primary diastolic dysfunction [7].

The majority of patients present with a clinical picture of refractory right heart failure, notably jugular venous distention, which is always present due to venous hypertension, as well as hepatomegaly, lower limb edema, and ascites [8, 9].

On the electrocardiogram, findings may include microvoltage, negative T waves, and atrial fibrillation (AF) [7,10], témoignant de l'augmentation de la pression dans les ventricules qui se transmet aux oreillettes à l'origine d'une dilatation atriale qui constitue le substrat anatomique de la FA.

These clinical and electrical signs are not specific to constrictive pericarditis (CP), therefore the diagnosis unquestionably requires the contribution of multimodal imaging (echocardiography, cardiac CT scan, and sometimes cardiac MRI).

The Mayo Clinic has defined echocardiographic criteria to suspect constrictive pericarditis, including abnormal septal motion related to respiration, normal or increased velocity of the medial mitral E' wave, and inversion of expiratory diastolic flow in the hepatic veins [11].

In a clinical presentation of adiastole, cardiac CT scan can reveal certain abnormalities such as pericardial thickening, presence of calcifications, atrial dilatation, dilatation of the vena cava, deformation of ventricular contours, as well as paradoxical septal motion [10, 12].

Note that our patient exhibited significant pericardial calcifications on the CT scan, along with signs of hepatic venous congestion in the abdominal region.

In patients with constrictive pericarditis (CP), MRI allows for the visualization of septal flattening, increased ventricular interdependence, and pericardial adhesions [13].

Right heart catheterization provides characteristic hemodynamic findings by demonstrating a typical pattern known as "dip-and-plateau" [Fig4]. This pattern indicates a normal pressure in the right ventricle during early diastole followed rapidly by an increase in pressure and then a meso- and telediastolic plateau [14].

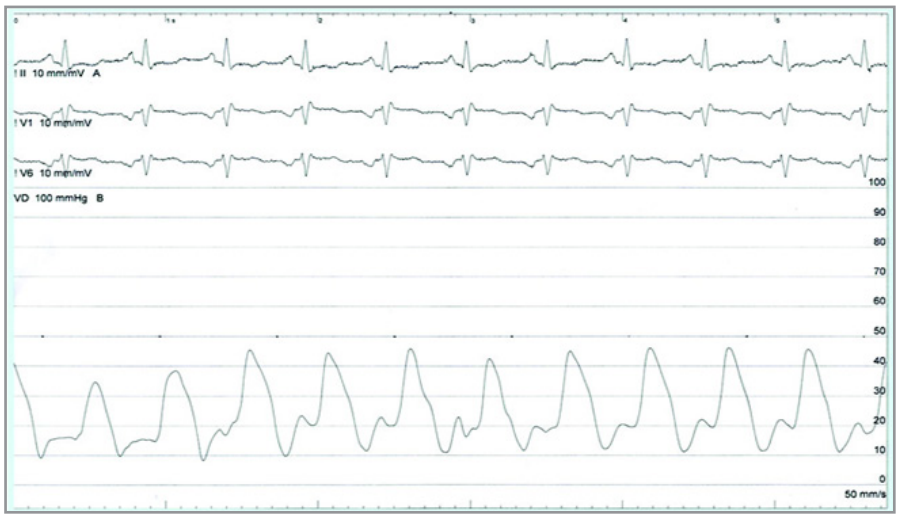

Figure 4: The "dip-and-plateau" aspect on right heart catheterization [14].

The initial treatment relies on the use of diuretics to address volume overload. However, the only effective treatment for chronic constrictive pericarditis is pericardial decortication surgery [15, 16].

Conclusion

Chronic constrictive pericarditis is a rare but serious condition, most often manifested by a presentation of right heart failure. Tuberculous origin remains common, particularly in developing countries. While medical treatment can alleviate symptoms in some patients, pericardiectomy remains the gold standard in the effective management of this condition.

Author Contributions

E.L : Monitoring the patient throughout the hospitalization+ data collection+ principal writer of the manuscript+ literature research.
D.R : Data collection, writer of the manuscript+ literature research.
D.H : Data collection, correction of the manuscript.
E.F : Data collection, correction of the manuscript.
M.F : Data collection, correction of the manuscript.
L.F : Supervision and data validation.
M.S : Supervision and data validation.
F.R : Supervision and data validation.
All authors read and approved the final manuscript.

Availability of Data and Materials

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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Non applicable

Conflict of Interest

None

Sources of Funding
None

Ethical Approval
N/a

Consent
Written informed consent was obtained from the patient for the anonymized publication of this case report.

Research Registration
N/a

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