

# Pericardial Constriction Revealed Early in Multifocal Tuberculosis in About 01 Case

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## Abstract

We report the case of a 46-year-old patient with a history of multifocal tuberculosis (peritoneal and pericardial) treated with anti-tuberculosis drugs and corticosteroid therapy for 12 months. He presented with progressive onset right heart failure syndrome during the course of his treatment related to chronic constrictive pericarditis documented by transthoracic ultrasound and right heart catheterization. The treatment was surgical by decortication with favorable operative consequences. This observation aims to question the rapid evolution of tuberculous pericarditis to chronic constrictive pericarditis usually described in the literature as a rare complication and occurring very late after several years.

**Keywords:** chronic constrictive pericarditis, pericardial effusion, early.

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## 1. INTRODUCTION

Chronic constrictive pericarditis (CCP) is a rare and usually late condition. Tuberculosis is the most common cause in developing countries, which corresponds to postoperative and post-radiation pericarditis, which is still more common in Western countries. It is manifested by the signs of adiasstole which have great clinical diagnostic value. Although the definitive diagnosis is histological, imaging, in particular transthoracic ultrasound, cardiac CT scan, cardiac MRI and right cardiac catheterization provide elements that point to the diagnosis of pericardial constriction [1, 2]

Few data in the literature describe early-onset pericardial constriction in tuberculosis. We report to you the case of a PCC diagnosed a few months after the start of treatment for multifocal tuberculosis (peritoneal and pericardial).

## 2. OBSERVATION

Mr X, 46 years old, hypertensive and well monitored, reported in his pathological history tuberculous pericarditis complicated by a moderate abundance of effusion which was treated with anti-tuberculosis drugs and corticosteroid therapy. He presented with NYHA stage II exertional dyspnea associated with bilateral lower limb edema throughout treatment.

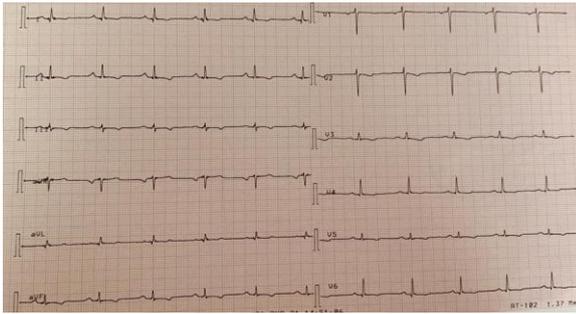
The ECG (fig. 1) showed a regular sinus rhythm at 67 / min, a 1st degree AVB, a diffuse microvoltage, repolarization disorders with negative T waves in the extended circumferential.

The lateral view of the chest X-ray (Fig. 2) showed calcifications circumscribing the cardiac shadow. Transthoracic ultrasound showed in 2D mode calcified and fibrinous thickening of the pericardium (Fig. 3).

The flow analysis (Figs. 4 and 5) revealed a variation in the mitral profile, restrictive on inspiration and an absence of pulmonary insufficiency flow with equalization of the pressures between the right ventricle and the pulmonary artery.

Thoracic computed tomography (CT) revealed constrictive pericardial thickening measuring 6mm with dilation of the inferior vena cava and necrotic mediastinal lymphadenopathy (Figures 8 and 9).

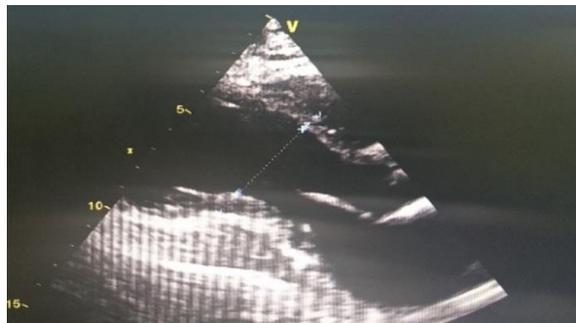
The right catheterization objectified an equalization of the intracavitary pressures and an aspect of "DIP PLATEAU".



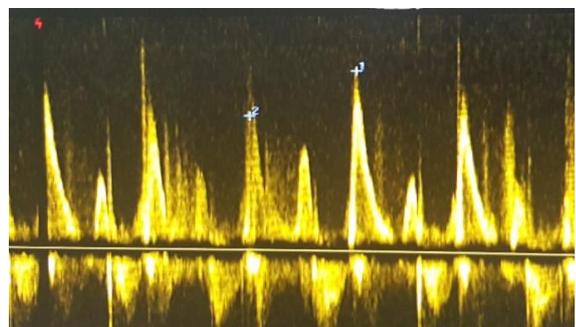
**Fig-1: Electrocardiogram showing microvoltage and AVB 1**



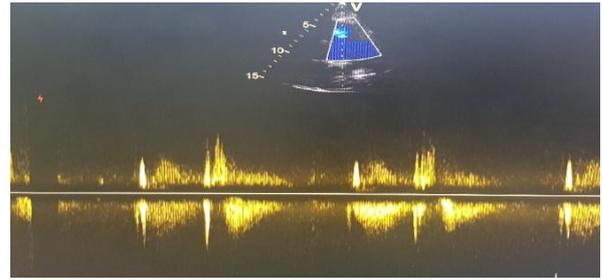
**Fig-2: Chest x-ray, lateral view showing calcifications (arrows)**



**Fig-3: 2D transthoracic ultrasound showing calcified and fibrous thickening of the pericardium**



**Fig-4: Mitral flow in pulsed mode showing a respiratory variation of the mitral profile, restrictive in inspiration**



**Fig-5: Transthoracic ultrasound, showing an absence of pulmonary insufficiency flow**



**Fig-2: Cardiac CT showing thickening of the pericardium**

### 3. DISCUSSION

Pericardial constriction is a rare complication of tuberculosis and usually occurs beyond several years. Its pathogenesis is related to a fibrous transformation of the pericardium which becomes rigid and inextensible interfering with the diastolic expansion of the cardiac chambers which defines adiasole. Pericardial effusion of tuberculous origin increases the risk of pericardial constriction in an immunocompetent subject despite optimal antibacillary treatment combined with corticosteroid therapy. It usually takes a long time to appear, up to several years after tuberculous pericarditis [3].

Dyspnea on exertion is the most common symptom (85% of cases). Right heart failure syndrome is almost constant and nonspecific. Blood pressure is often lowered. During inspiration, blood pressure may decrease by 10 mmHg, defining Kussmaul's paradoxical pulse. Auscultation can reveal a third protodiastolic noise in more than half of the cases. The physical examination may in some cases be normal [4].

In most cases, the EKG shows diffuse T wave abnormalities and diffuse microvoltage. On a standard chest x-ray, the heart shape is most often normal, with in a third of cases much more visible calcifications on the lateral views [5].

Objective two-dimensional (2D) transthoracic ultrasound, pericardial thickening in association with moderate dilation of the atria and inferior vena cava. The TM mode shows an aspect of plateau DIP at the

level of the septum, a paradoxical anterior movement of the septum with a sudden protodiastolic retreat and a straightness of the posterior wall with disappearance of the enddiastolic retreat. Premature opening of the pulmonary sigmoid can be observed by equalization of pressures in the chambers of the heart which is a sign rarely seen in CCP. [6] Doppler analysis is an essential step in the diagnosis of pericardial constriction. It allows the analysis of mitral flow, tricuspid flow and flow in pulmonary and hepatic veins. This study makes it possible to look for the signs of adiastrale and especially the respiratory variations of the flows which make it possible to differentiate CCP from restrictive cardiomyopathy. The study of the mitral flow reveals at the time of inspiration a large E wave, a small A wave and a short TDE <150 ms and a return to the normal profile on expiration, representing an important sign in the ultrasound diagnosis of pericardial constriction. On tissue Doppler, the rate of displacement of the mitral ring (E wave) is normal (> 8 cm / s). The study of pulmonary venous flow shows a decrease in the S wave, an increase in the speed and duration of the A wave in inspiration. There is a respiratory variation of the D wave which increases during expiration and decreases during inspiration. A decrease in the speed of the D wave of more than 40% in inspiration is in favor of pericardial constriction. Conversely, there is an increase of more than 25% of the E wave in inspiration. The flow of pulmonary insufficiency shows an equalization of the pressures between the right ventricle and the pulmonary artery. Analysis of the hepatic venous flow reveals a decrease in the S wave and an increase in the A wave [7].

Cardiac catheterization remains the reference examination which allows to confirm adiastrale by objectifying an equalization of ventricular diastolic pressures with a dip-plateau appearance. It is characterized by a rapid and deep drop in ventricular pressure at the onset of diastole followed by a rapid rise in protodiastole to a plateau forming a square root shape. This aspect is also found in restrictive heart disease, but the study of respiratory variations in ventricular pressures (increase in right ventricular pressures and decrease in left ventricular pressures in inspiration) is essential to confirm the diagnosis of CCP [8].

MRI and cardiac CT have great sensitivity in diagnosing CCP. They provide additional data on pericardial thickening, pericardial calcifications, the morphology of the heart chambers and the condition of the mediastinum and lungs for associated lesions [9].

The cure for CCP is surgical. Medical treatment before surgery is mainly symptomatic (diuretics). It consists of a decortication of the two layers of the pericardium by median sternotomy with or

without cardiopulmonary bypass. Decortication should first be performed in the left ventricle to avoid the occurrence of pulmonary edema after release of the right ventricle (by pulmonary overload).

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#### 4. CONCLUSION

Chronic constrictive pericarditis, although described as rare and late, should always be considered in the presence of any right heart failure syndrome in the context of suspected or confirmed tuberculosis. Cardiac MRI and right catheterization have great sensitivity in the positive diagnosis. The prevention of complications depends on the speed of diagnosis and treatment, which must be surgical.

#### REFERENCE

1. Rev Med Suisse. (2015). 11; 1166-71
2. Ng, A. S., Dorosti, K., & Sheldon, W. C. (1984). Constrictive pericarditis following cardiac surgery--Cleveland Clinic experience: report of 12 cases and review. *Cleveland Clinic Quarterly*, 51(1), 39-45.
3. Kim, M. S., Chang, S. A., Kim, E. K., Choi, J. O., Park, S. J., Lee, S. C., ... & Oh, J. K. (2020). The clinical course of tuberculous pericarditis in immunocompetent hosts based on serial echocardiography. *Korean circulation journal*, 50(7), 599-609.
4. El Khorb, N., El Ouali, L., Lahlou, I., Ouaha, L., & Akoudad, H. (2012). La pericardite chronique constrictive. *MorJCardiol*, 7, 16-20.
5. De La Roche, E., Duperret, S., Arpin, D., Perol, M., Depagne, C., Nesme, P., & Guerin, J. C. (2008). Idiopathic constrictive pericarditis revealed by a massive left pleural effusion. *Revue des maladies respiratoires*, 25(5), 591-595.
6. Oh, J. K., Hatle, L. K., Seward, J. B., Danielson, G. K., Schaff, H. V., Reeder, G. S., & Tajik, A. J. (1994). Diagnostic role of Doppler echocardiography in constrictive pericarditis. *Journal of the American College of Cardiology*, 23(1), 154-162.
7. Garcia, M.J., Rodriguez, L., Ares, M. (2010). Differentiation of constrictive pericarditis from restrictive cardiomyopathy: assesment of left ventricular diastolic velocities in longitudinal axis by D
8. Hurell, D. G., Nishimura, R. A., & Tajik, A. J. (1994). Can analysis of dynamic changes during cardiac catheterizaion diagnose constrictive pericarditis? A prospective comparative hemodynamic study using high fidelity catheters in human. *Circulation*, 90, 1-121.
9. Appleton, C. P., Hatle, L. K., & Popp, R. L. (1988). Demonstration of restrictive ventricular physiology by Doppler echocardiography. *Journal of the American College of Cardiology*, 11(4), 757-768.