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Case Report

Atrial Fibrillation Revealing Chronic Constrictive Pericarditis

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Abstract

Chronic constrictive pericarditis (CCP) is rare, has multiple cause and is diagnosed late due to the absence of specific signs. It often leads to confusion with restrictive cardiomyopathy. The diagnosis can be oriented by a simple chest x-ray which shows pericardial calcifications in the form of a shell enveloping the cardiac silhouette, and confirmed by cardiac Doppler echo, right heart catheterization, and in some cases cardiac computed tomography (CT) and MRI. We report the case of a 27-year-old female patient hospitalized for suspected restrictive cardiomyopathy with atrial fibrillation, which had been developing for several years. The diagnosis of CCP was made in her. Through this case, we will discuss the diagnostic means and management of this pathology.

Keywords: ***Chronic right heart failure, atrial fibrillation, chronic pericarditis, restrictive cardiomyopathy, chest x-ray, cardiac Doppler echo, cardiac CT, cardiac MRI.***

Introduction

Chronic constrictive pericarditis is rare, characterized by fibrocalcareous pericardial thickening. Of multiple causes, tuberculosis remains the most common cause in developing countries [1-3]. The clinical presentation is a picture of right heart failure, often leading to a diagnostic delay of several years. The diagnosis is essentially based on the clinic, chest x-ray, cardiac Doppler ultrasound and right heart catheterization. In certain cases, the use of other imaging techniques such as CT or cardiac MRI is necessary. The main differential diagnosis is restrictive cardiomyopathy (RCM) [4]. Chronic constrictive pericarditis affects both ventricles, but predominates in the right ventricle. At the physiopathological level, pericardial constriction leads to a disorder of myocardial compliance and consequently to discomfort in ventricular filling which defines adiastole (During proto-diastole, the ventricular wall distends rapidly but the movement is suddenly limited and followed by complete immobility in meso and end-systole, causing the "dip-plateau" appearance [1-3]. Treatment is surgical and consists of pericardiectomy. When tuberculosis is the cause. anti-tuberculosis treatment is prescribed before surgery and continued afterwards for three months [1].

We report the case of a young girl aged 27, followed for atrial fibrillation, presents edema of both lower limbs, progressing over ten years, with a recurrent right pleural effusion, of undetermined origin (pleural puncture non-contributory). In May 2023, she was referred to us for management of her atrial fibrillation (**Figure 1**) with suspicion of restrictive cardiomyopathy (EF = 40%). The patient had signs of right heart failure refractory to diuretic treatment (injectable furosemide combined with spironolactone) with moderate right pleural effusion. It does not report the notion of tuberculosis infection or other specific antecedents. A chest x-ray was taken at home, confirming the pleural effusion with the presence of pericardial calcifications surrounding the cardiac silhouette (**Figure 2**). The biological assessment is in favor of chronic inflammation. The infectious and thyroid tests are normal and the NT proBNP level is slightly elevated. On cardiac Doppler echo, the atria are dilated, the two ventricles are slightly hypokinetic, the EF is at 50%, the mitral and tricuspid flows on pulsed Doppler are in favor of CCP; the pericardium is hyper echogenic (**Figure 3**). Chest CT then confirmed CCP (**Figure 4**).

Figure 1: ECG: AF, low voltage

Figure 2 a: Chest x-ray: front Pericardial calcifications

Figure 2b: Chest x-ray: profile Pericardial fibro-calcareous shell

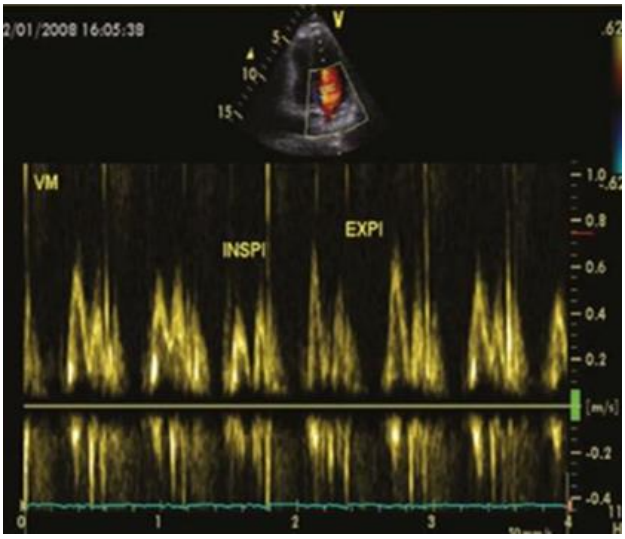


Figure 3: Cardiac Doppler ultrasound: mitral flow, inspiratory variations of 45%

Figure 4: Cardiac CT: fibro-limestone pericardial shell

Discussion

Our patient, who has lived for a long time with signs of chronic right heart failure refractory to treatment and labeled as restrictive cardiomyopathy, presents with chronic constrictive pericarditis. The diagnosis was suspected on chest x-ray which showed pericardial calcifications. It should be noted that these calcifications may be absent in one in five cases [5].

This diagnostic delay is partly due to the non-specific clinical signs of this pathology [1-4]. The transition to atrial fibrillation was the factor of decompensation with diastolic heart failure with preserved ejection fraction. Atrial fibrillation can occur in 25% of cases, caused by dilation of the atria and indicating a long-term development.

The etiologies of CCP are numerous, but the most common in developing countries remains tuberculosis. In 50% of cases the cause is undetermined [1]. CCP can mimic restrictive cardiomyopathy, hence the interest in cardiac Doppler echo. The latter allows an evaluation of the function of the left and right ventricle, analysis of the pericardium in search of pericardial thickening and calcification with the presence or absence of pericardial effusion. Other signs should be looked for on cardiac Doppler echo such as the paradoxical kinetics of the interventricular septum, variable with respiration [6] as well as respiratory variations in mitral and tricuspid flow [7].

The study of these flows makes it possible to differentiate PPC from CMR [8,9]. The transmitral and tricuspid flow are of restrictive type (type III of the Appleton classification) E/A > 2, the deceleration time is short < 150 ms, end-diastolic mitral regurgitation can be observed in severe forms, which allows 'affirm adiastole. On inspiration, tricuspid flow increases, while mitral flow decreases (by at least 25-30%). Indeed, the increase in filling of the right ventricle during inspiration leads to a reversal of the septal curvature towards the left ventricle due to ventricular interdependence in a rigid pericardial sac [11,12]. The lung failure flow shows the dip-plateau.

The flow of the pulmonary veins and the inferior vena cava is reversed. Tissue Doppler provides additional arguments. Analysis of mitral and tricuspid ring velocities shows subnormal velocities (E'> 8 cm/s, E/E'< 15, normal S wave). Differentiating them from CMR (**Figure 1-11**).

The CCP assessment is completed by right heart catheterization at the slightest doubt.

(**Figure 12**) which then shows adiastole with the appearance of dip plateau [10]. Cardiac CT is performed to confirm pericardial damage, location and extent of constriction [19,20], (**Figure 13**). A

diagnostic algorithm was developed by Boccara et al in order not to delay the diagnosis of CCP (**Figure 14**).

Treatment consists of relieving the patient by prescribing diuretics to reduce congestive signs while waiting for pericardiectomy with anti-tuberculosis treatment, when tuberculosis is the cause. It is the only effective treatment for pericardial constriction. It consists of decortication of the two layers of the pericardium by median sternotomy with or without extracorporeal circulation [13,14]. Postoperative mortality and long-term prognosis might be correlated with the etiology of chronic constrictive pericarditis [15-18].

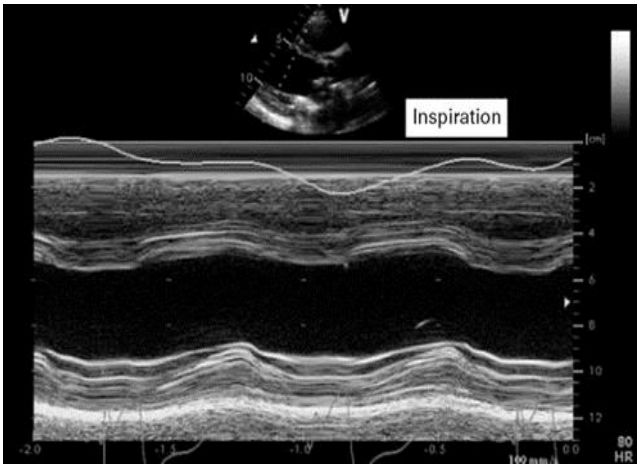


Figure 6: TM (VG), paradoxical SIV [20]

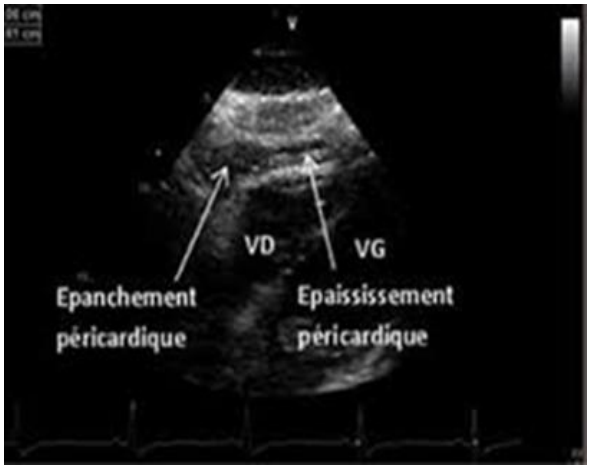


Figure 7: Pericardial thickening [20]

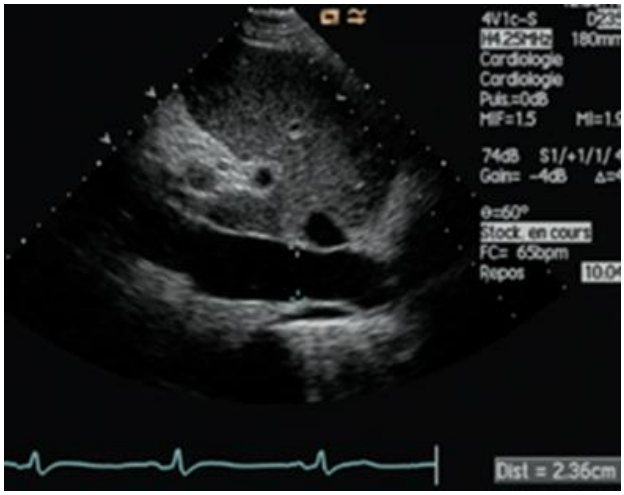


Figure 8: Dilated, non-compliant IVC [20]

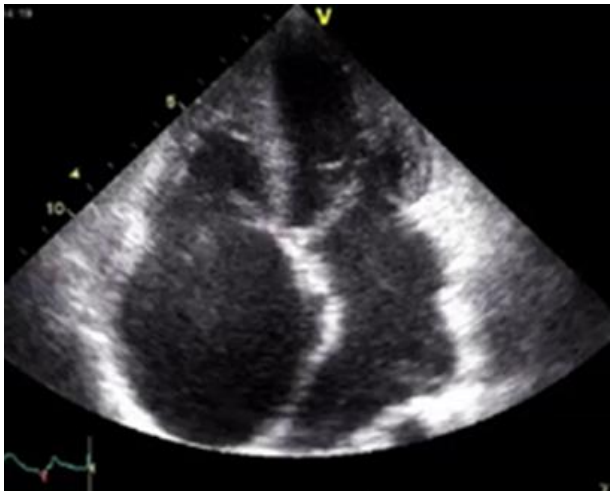
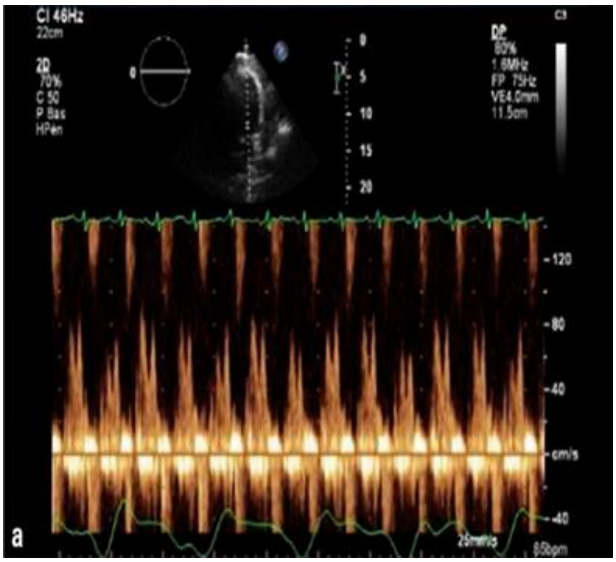
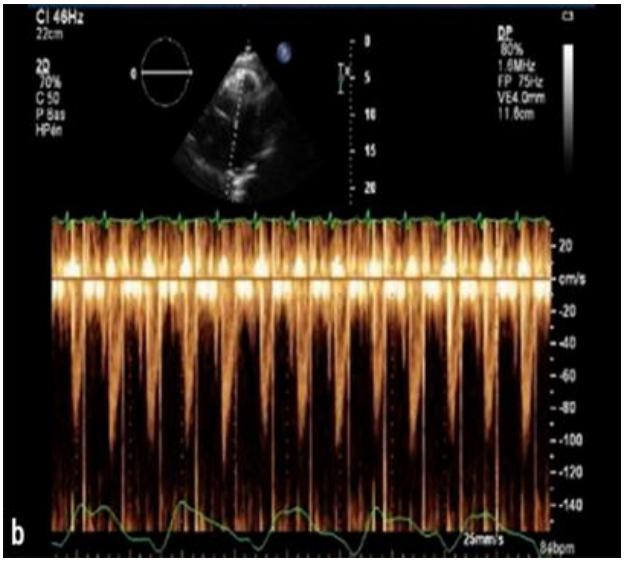


Figure 9: Dilatation of the auricles [20]





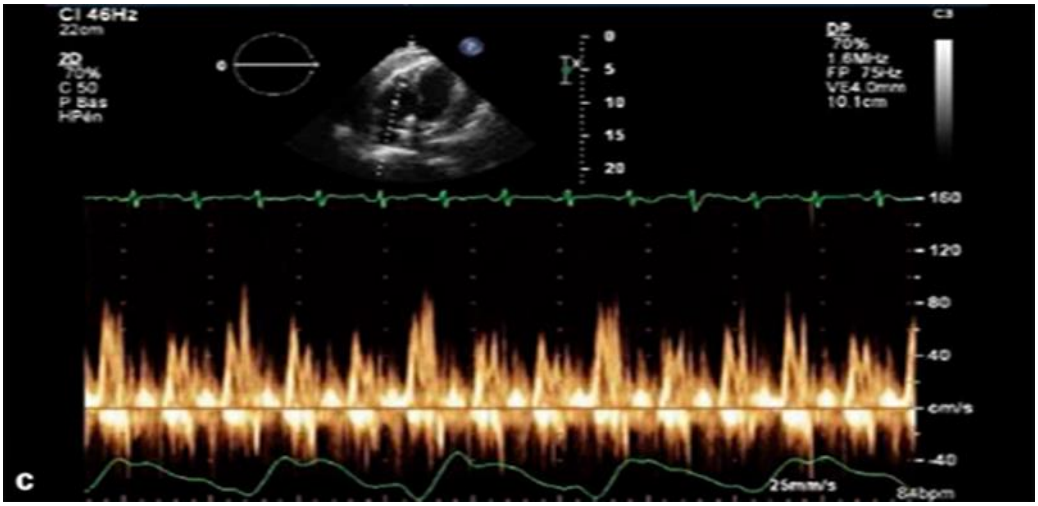


Figure 10: Variation of anterograde flows in pulsed Doppler in 4-chamber incidence: 30% on the mitral flow (a) and on the aortic flow (b), 40% on the tricuspid flow (c) [20].

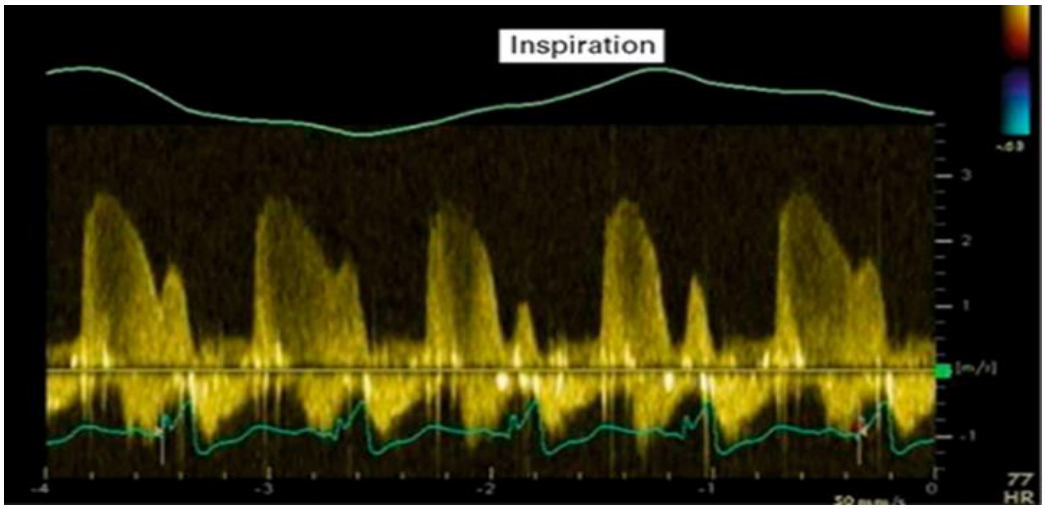


Figure 11: Plateau dip appearance of retrograde pulmonary flow [20]

Figure 12: Cardiac catheterization curves: equalization of diastolic pressures in the right cavities (left) and in the two ventricles with dip-plateau appearance (right) [20].

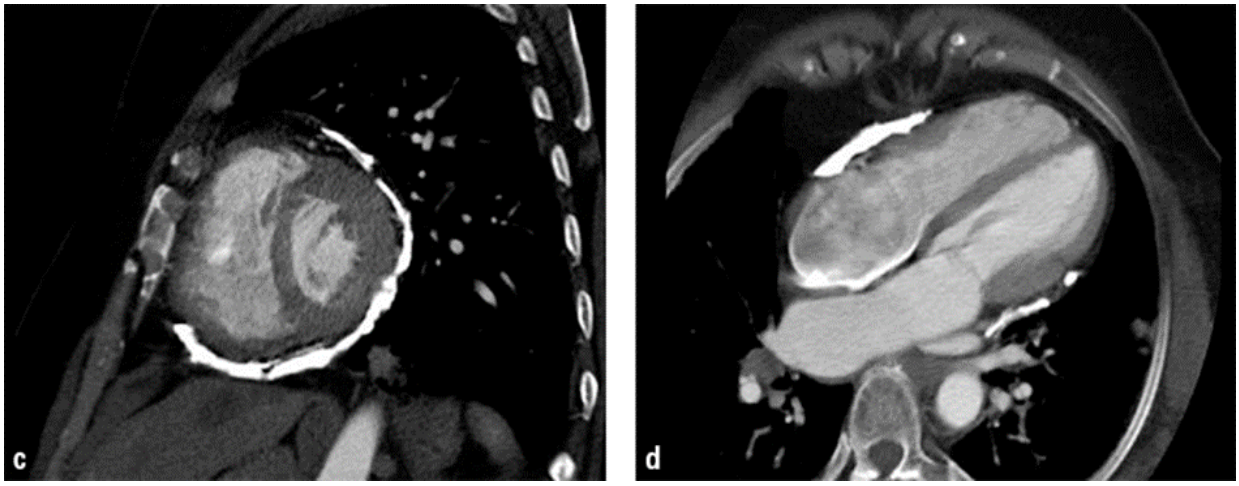


Figure 13: Cardiac CT showing extensive (a), localized (b) pericardial calcifications [20]

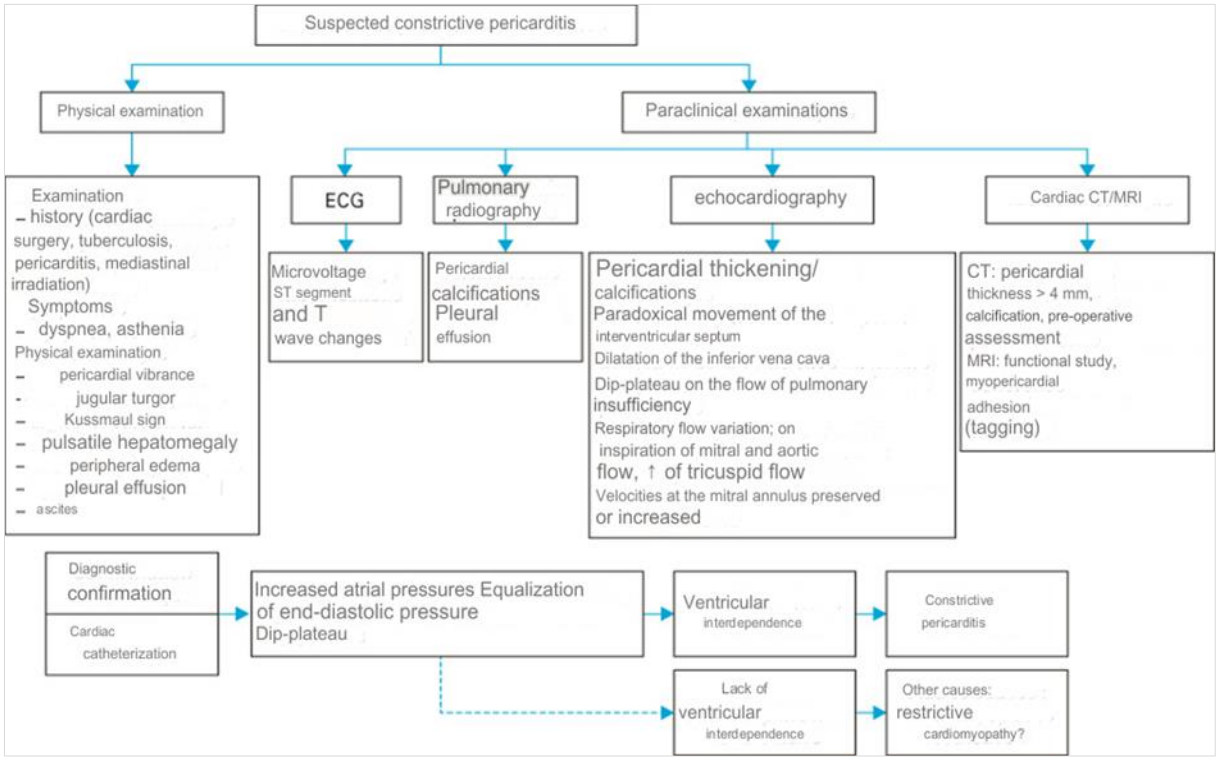


Fig 14: Diagnostic algorithm for suspected CCP [20]

Conclusion

The diagnosis of CCP is often late due to the difficulty in confirming the presence of complex and nonspecific diagnostic criteria. It is absolutely necessary to think about it when there are signs of right heart failure refractory to treatment associated with atrial fibrillation. Sometimes, a simple chest x-ray can guide this diagnosis thanks to the visualization of pericardial calcifications surrounding the heart chambers.

Declarations

Ethical Considerations

Ethical authorization was obtained from the hospital and the scientific council of the Faculty of Medicine; The patients received a written and informed consent form signed after careful explanation objectives, procedure and full involvement of participation in the study. This study was conducted in compliance with ethical rules. standards of our institution on human subjects as well as with the Declaration of Helsinki.

Informed Consent

A signed consent was obtained by the researcher and research assistants before recruitment of the participants into the study after appropriate counselling.

Conflict of Interest

There was no conflict of interest.

Data Availability

Data would be available upon reasonable request.

Funding Statement

The entire financial burdens were burn by the researchers

Author Contributions

Z. Bennoui: The principal investigator, M. Abdelbaki: was involved in the literature search and day to day conduct of the work till conclusion.

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