Chronic constrictive tuberculous pericarditis with apical pseudo-ballooning

Péricardite chronique constrictive tuberculeuse avec un pseudo-anevrysme apical

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Résumé
Nous rapportons le cas d'un homme de 64 ans, aux antécédents de péricardite tuberculeuse, admis pour une dyspnée d’aggravation progressive. L’échocardiographie a montré une dysfonction ventriculaire droite avec une dilatation de l’oreillette gauche, un épaissement du péricarde avec des variations respiratoires de plus de 30%. La fonction systolique ventriculaire gauche était conservée. La radiographie du thorax a révélé des calcifications péricardiques. Le diagnostic de péricardite constrictive chronique était fortement suspecté. Le patient a eu un cathétérisme cardiaque qui a conforté le diagnostic de péricardite constrictive devant la pression télé diastolique ventriculaire droite élevée. La coronarographie était normale. Une tomodensitométrie cardiaque a montré des calcifications diffuses et une ballonisation apicale double plus prononcée au niveau du ventricule. L’IRM cardiaque a confirmé l’anévrysme focal apical. Le patient a été opéré et a eu une péricardectomie avec des suites simples.

Summary
Here we report the case of a 64-year-old man, with a history of tuberculous pericarditis, presented with increasing dyspnea. Echocardiography showed right ventricular dysfunction with a dilated left atrium, a thickened pericardium a respiratory tricuspid flow of more than 30%. Left ventricular systolic function was preserved with an ejection fraction about 55%. Plain radiographies revealed pericardial calcifications. The diagnosis of chronic constrictive pericarditis was strongly suspected. The patient underwent cardiac catheterization that supported the diagnosis of constrictive pericarditis by measuring elevated end-diastolic right pressure. Coronarography was normal. An Electrocardiogram-gated cardiac multidetector computed tomography showed diffuse calcifications and a double apical outpouching more pronounced on the RV. The patient was referred for cardiac magnetic resonance imaging (CMR). CMR confirmed the focal ballooning of the cardiac apex. The patient was referred for pericardiectomy. No post-operative complication occurred and the patient was discharged 5 days after pericardiectomy.

Keywords
Pericardium, Computed tomography, Surgery, Calcifications

Mots-clés
Péricarde, Tomodensitométrie cardiaque, Chirurgie, Calcifications
INTRODUCTION

Constrictive pericarditis is associated with scarring and loss of elasticity of the pericardium, causing impaired cardiac filling. Computed tomography is the gold standard imaging tool for cardiac calcification, it allows a nice anatomic delineation of the pericardium and its calcifications which may be very useful in determining the optimal surgical approach for pericardial resection.

CASE REPORT

A 64-year-old man, former smoker, with a history of tuberculous pericarditis insufficiently treated in 1979, presented with increasing dyspnea (New York Heart Association class III and peripheral edema, worsening for the past two years. On physical examination he had signs of right sided heart failure: peripheral edema, ascites, hepatomegaly, distended neck veins and hepatojugular reflux.

The patient was explored in cardiology department. Electrocardiogram showed a slow atrial fibrillation. Echocardiography (EC) showed right ventricular (RV) dysfunction with a Peak systolic velocity at (8 cm/s) and Tricuspid annular plane systolic excursion (1.4 mm), a dilated left atrium (33 cm²), a thickened pericardium (3.5 mm) a respiratory tricuspid flow of more than 30%. EC also revealed an enlarged liver with dilated hepatic veins and dilated-non-collapsing inferior cava vein (IVC). Left ventricular (LV) systolic function was preserved with an ejection fraction about 55%. Trans esophageal EC did not find any cardiac thrombus.

Plain radiographies revealed pericardial calcifications. The diagnosis of chronic constrictive pericarditis (CCP) was strongly suspected, the patient underwent cardiac catheterization that supported the diagnosis of CCP by measuring elevated end-diastolic right pressure. Coronarography was normal.

There were no surgical contraindication and pericardiectomy was therefore indicated. An Electrocardiogram-gated cardiac multidetector computed tomography (MDCT) was performed to assess the extent of calcifications. MDCT showed-in addition to the morphologic results of the EC pericardial thickening >4 mm with diffuse calcifications (Figure 1) localized essentially in the inferior segments of left and right ventricle, bialtral dilation and a double apical outpouching more pronounced on the RV (Figure 2). The patient was referred for cardiac magnetic resonance imaging (CMR). CMR confirmed the focal ballooning of the cardiac apex (Figure 3). Cine-MRI sequences allowed a functional study noticing shift of the interventricular septum to the left during early diastole and a non-collapsing IVC. Both cardiac MDCT and MR showed no pericardial effusion neither intra-myocardial calcification but a pleural effusion of low abundance.

The patient was referred for pericardiectomy. Intraoperatively the pericardium was thickened and calcified massively sparing the apex where surgeons noticed the hernia of RV [1] (Figure 4). The LV hadn’t been inspected. Anti-tuberculosis quadritherapy was initiated. No post-operative complication occurred and the patient was discharged 5 days after pericardiectomy.

EC performed one month later showed the vanishing of the dip-and-plateau and a preserved LV systolic function. CMR performed months later showed persistence of the apical outpouching and a better contraction and function of both right and left ventricles.

CCP is a rare but severely disabling consequence of the chronic inflammation of the pericardium, leading to an impaired filling of the ventricles and reduced ventricular function.

Figure 1: 3 D Volume Rendering (VR) reconstruction on acquisition of iodinated contrast-enhanced MDCT showing massive pericardial calcifications (arrowhead) sparing the apex and right ventricular ballooning (arrows)
Pericardial constriction is usually the result of long-standing pericardial inflammation leading to pericardial scarring with thickening, fibrosis, and calcification. The most frequent causes are mediastinal radiation, chronic idiopathic pericarditis, after cardiac surgery, and tuberculous pericarditis.

Imaging plays a particularly important role in distinguishing CCP from other diagnosis and more particularly from restrictive cardiomyopathy which may be challenging. The differentiation of diseases is crucial because constrictive pericarditis is a surgically curable disease [2]. Advance in MDCT and CMR has revolutionized the exploration of PCC. They are presently the standard...
methods for accurate measurement of pericardial thickness [3]. MDCT and MRI help predict patients of high risk of poor surgery outcome. Cardiac mortality and morbidity at pericardiectomy is mainly caused by the pre-surgically unrecognized presence of myocardial atrophy, myocardial fibrosis or calcifications.

![Figure 4: Pericardiectomy realized without extracorporeal circulation to liberate the heart from the thickened and calcified pericardium (headarrows), right ventricular apex has a pseudo-balloonized shape (arrow). Left ventricle hasn’t been inspected. RV = right ventricle](image)

**REFERENCES**