Epicardite constrictive

Constrictive epicarditis

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RÉSUMÉ

La péricardite constrictive est une pathologie peu fréquente. Le plus souvent elle est silencieuse jusqu'à un stade tardif. Les phénomènes constrictifs intéressent les deux feuillets péricardiques mais exceptionnellement seul le feuillet viscéral est touché.

On rapporte l'observation d'un patient de 25ans qui présentait une épicardite constrictive.

A travers ce cas clinique on va évoquer certaines spécificités du diagnostic, du traitement chirurgical et des suites opératoires. On va aussi s'intéresser à la dysfonction ventriculaire droite fréquemment observée après péricardiectomie et qui représente une complication importante.

Mots-clés

Péricarde, constriction, dysfonction ventriculaire, péricardiectomie.

SUMMARY

Constrictive pericarditis is relatively uncommon. Constrictive phenomenon involves in the majority of cases the two layers of the pericardium namely the parietal pericardium and the visceral one. Chronic epicarditis is a distinct and very scarce form where only the visceral pericardium is interested by the pathologic process. We present herein the case of a 25 years old patient admitted in our department for surgical treatment of a chronic visceral pericarditis. We discuss along some important clinical and therapeutic points related to this specific presentation with a special interest to the right ventricular dysfunction after pericardiectomy.

Key-words

Pericardium ,constrictive pericarditis , right ventricular failure, pericardiectomie.

INTRODUCTION

Constrictive epicarditis is a particular and scarce form of pericardial diseases. We report a case of a 25 years old man and we rule out some important issues concerning surgical treatment and outcome.

Aim

In this report we raise some issues concerning the constrictive physiopathology, surgical treatment and outcomes, mainly right ventricular dysfunction. Although this is a particular presentation, it leads to some reflections on pericardial constrictive disease which is still incompletely elucidated.

REPORTED CASE

We present the case of a young patient, with no past medical history or cardiopulmonary symptoms, who had undergone an abdominal ultrasound imaging for renal lithiasis symptoms. Other than lithiasis, this exam showed an important dilatation of the supra-hepatic veins. Therefore, a thoraco-abdominal CT scan was performed showing a thickening of pericardium, an important dilatation of the supra-hepatic veins and inferior vena cava. The patient was then referred to cardiology. Physical examination was normal with no clinical signs of heart failure. Electrocardiography showed no abnormalities, and chest X rays showed a small cardiac silhouette and pericardial calcifications. Cardiac echography and Doppler were performed, featuring a thickening of the pericardium with multiple small calcifications around the ventricles, respiratory inflow velocity >30%, normal valves, normal left and right ventricle functions and a dilated non-compliant inferior vena cava. Cardiac catheterization showed typical findings of constriction. The patient was the transferred to our department for surgical pericardiectomy.

Surgery was performed through a median sternotomy. The parietal pericardium was minimally thickened and non-adherent to the heart which was surrounded by an epicardial thickening that was dense and mildly calcified (figure 1). We successfully achieved full decortication of the visceral epicardium without major incidents. Although parietal pericardium was macroscopically normal, we resected it from phrenic to phrenic nerves. Histopathology analysis showed a non-specific chronic inflammation.

Cardiac echography performed five days after the surgery showed no residual constriction-type echographic signs. The only abnormality was a reduced right ventricular (RV) systolic function with tricuspid lateral systolic velocity wave S'=7cm/sec (Figure 2). The follow-up transthoracic echocardiography during his uneventful postoperative period, three months after operation, demonstrated slightly improved RV systolic function, S'=8cm/sec. Cardiac magnetic resonance imaging (MRI) confirmed the RV dysfunction one year after surgery (Ejection Fraction 40%).

Three years after surgery, tissue doppler showed persistently reduced RV systolic function with S'=8.5cm/sec, but normal right ventricular size and thickness. A low dose of oral diuretics is still ongoing and the patient remains completely asymptomatic.

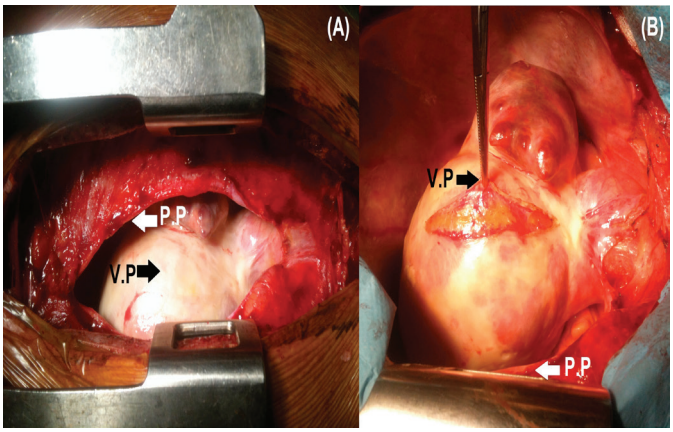

Figure 1 (A, B) : The parietal pericardium (black arrow) was minimally thickened and non-adherent to the heart which was surrounded by a dense visceral pericardium thickening (white arrow). V.P, visceral pericardium; P.P, parietal pericardium.

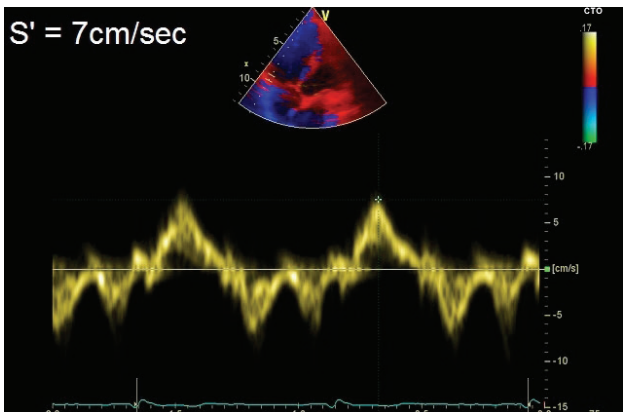

Figure 2: reduced right ventricular systolic function with tricuspid lateral systolic velocity wave S'=7cm/sec.

DISCUSSION

The distinctive feature of chronic epicarditis is that the constriction process resides only in the visceral epicardium, the parietal pericardium remains intact and independent. However, the physio-pathologic consequences are the same as constrictive pericarditis.

To our knowledge, the first case of epicardic constriction was reported by Nissen R and Schweiger W in 1968 (1), since this report, only a few recent data has been published (2,3).

Specific concerns have to be raised concerning this uncommon presentation of the pericardial disease: The scar may be very dense and infiltrates from the epicardium into the underlying sub-epicardial tissues or myocardium. Removal of the scar over the atria and venae cavae seems more challenging regarding the high risk of entering these chambers in this particular case of epicarditis when compared with pericarditis. In our experience, from an unpublished data of 25 chronic pericarditis patients, we usually avoid to perform any epicardial relaxing incisions namely knowns as cross-hatching incisions, in front of ventricular surfaces and auricular massif. We elect performing the last technique only on the surface of diaphragm facing the heart after removing the basal aspect of pericardial constriction. The cross-hatching in regard of auricular surfaces and venae cavae, in this particular case of epicarditis can be a very good option.

The biopsy of the pericardium didn't give any valuable informations in the majority of cases. Expanding the biopsy to the RV myocardium as invoked by Homsi (4) may be interesting to identify structural changes of the underlying myocardium.

In our case, although the parietal pericardium appeared perfectly normal macroscopically, it was removed to ensure that no recurrent constrictive process will involve the last one. Our decision to remove the normal and perfectly compliant parietal pericardium is based only on presumption, because of the lack of any consensus or evidence due to the rarity of this pathology. This therapeutic decision has been adopted by other authors (2,3). However, the preservation of the parietal pericardium may be, at least theoritically, a good option if we consider its hemodynamic role, even though small, on a weakened RV myocardium and also its role in ventricular interaction (5).

The persistent RV dysfunction and its slow recovery after surgery, while described as normal preoperatively, in general and in our case specifically, is still insufficiently understood and raised with great concern in only few reports. Several hypothesis have been formulated to explain the physio-pathologic mechanisms. Homsi (4) in a recent study of 4 patients have argued that reduced right ventricular systolic function in constrictive pericarditis indicates a myocardial involvement essentially by the inflammatory process that caused pericarditis.

This myocardial involvement leads to some assumptions concerning whether the onset of inflammation is myocardial or epicardial, and if this particular case of epicardial constriction is the first step of a total pericardial constriction of a distinct presentation.

Other authors (6) pointed out changes in cardiac architecture and remodeling of ventricles caused by long standing constriction. In our opinion, the particular vulnerability to the constriction of the right ventricle when compared with the left one is a complex interplay of functional and structural factors closely linked to physiologic and anatomic features of the RV.

The specific anatomic architecture of the RV and its myocardial fibers composition and orientation can have an important role to explain the issue of RV dysfunction after surgery. The occurrence of fatal and severe dysfunction of the RV have been reported in multiple reports when decompressing an acute tamponade (7), this fact supports that functional factors can take strongly the lead over structural ones or induce them: The main physiopathologic driver, after treatment, in both scenario (tamponade or constriction) are acute changes in preload and afterload of the RV . The increased veinous return in both cases may allow a muscular injury of the most distensible chamber of the heart namely the RV(7).

The critical aspect of persistent impaired RV function after pericardiectomy stresses the requirement of a systematic accurate assessment of the RV function preoperatively as it is generally overlooked. We advocate the usefulness of non-invasive techniques including echography with multiple measurement methods (to avoid potential bias caused by constriction), combined with cardiac MRI as an integrated approach. A recent study (8) using integrative approach has identified tricuspid annular plane systolic excursion as an independent predictor of cardiac events after surgery.

Finally, we emphasize the importance of considering any pericardial constrictive disease as a potential myocardial pericardial one.

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