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Constrictive pericarditis - a difficult diagnosis

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ABTRACT.

Constrictive pericarditis is an uncommon condition, often of unknown etiology. The diagnosis of constrictive pericarditis can be difficult and is often delayed, because the signs and symptoms of this disease can be falsely attributed to other causes. We report the case of a 62-year-old woman presented with a one year history of progressively worsening dyspnea, peripheral oedema, prominent jugular distension, hepatomegaly, ascites. The patient is known with a history of effusive pericarditis. Blood test showed a normal white cell count, anaemia, raised CRP. Chest X-Ray shows a normal sized heart and without calcification of the pericardium. The echocardiographic exam showed septal bounce-abrupt transient rightward movement, left, right ventricular size decreased-heart tubularin shape, mild atrial enlargement, IVC plethoric and unresponsive to respiration, hepatic veins dilated. Doppler echocardiographic findings were consistent with constrictive pericarditis. Cardiac catheterization showed elevation and equalization of diastolic filling pressures, and dips and plateau configuration of ventricular pressure during diastole (square root sign). Based on these results pericardiectomy was necessary. Constrictive pericarditis was also confirmed at the time of surgery. The pericardium was found with thickening

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of up to 30 mm in some areas. Histopathological exam showed fibrosis and calcification within the pericardium with no evidence of malignancy or tuberculosis. The patient was discharged 10 days later. At 3 months she had no significant symptoms. Diagnosis of constrictive pericarditis remains challenging. Constrictive pericarditis should be suspected in patients with clinical features of right-sides heart failure. Echocardiography and cardiac catheterisation are important investigation in diagnosis of constrictive pericarditis and avoiding unnecessary treatments.

Keywords: constrictive pericarditis, echocardiography, heart failure

Introduction

Constrictive pericarditis is an uncommon condition, represents the end stage of an inflammatory process involving the pericardium and causes myocardial compression and restricted filling of the heart. In the past tuberculosis was the most common cause of constrictive pericarditis, and it still remains important in developing countries. In the developed world the cause is most commonly idiopathic, postsurgical, or radiation injury, but also can be caused by viral, bacterial or fungal infection, autoimmune disorders, uremia, neoplasia, posttraumatic, and inflammatory reaction to a foreign body [1] The clinical presentation is usually characterized by clinical signs and symptomps of right-heart failure

due to pericardial constriction. The diagnosis of constrictive pericarditis can be difficult and is often delayed, because the signs and symptoms of this disease can be falsely attributed to other causes.

Case presentation

We report the case of a 62-year-old woman presented with a one year history of progressively worsening dyspnea, peripheral edema, prominent jugular distension, hepatomegaly, ascites. The patient is known with a history of effusive pericarditis. Blood test showed a normal white cell count, anaemia, raised CRP. ECG shows sinus rhythm, normal voltage and nonspecific T wave abnormalities (negative T wave in V1-V3). Chest X-Ray shows a normal sized heart and without calcification of the pericardium. The echocardiographic exam showed septal bounceabrupt transient rightward movement, left, right ventricular size decreased-heart tubularin shape (Figure 1), mild atrial enlargement, IVC plethoric and unresponsive to respiration, hepatic veins dilated, mild pulmonary hypertension (PAPs 40 mmHg) and no pericardial thickening.



Figure 1 - four chambers echocardiographic view with normal cavities dimensions

Doppler echocardiographic findings showed restrictive mitral inflow E/A>2, DT 116 msec, respiratory changes in the mitral E velocity (>50%) (Figure 2), reversed hepatic venous flow during expiration, high E' velocity (14 cm/sec).

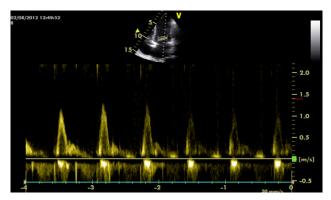


Figure 2 - restrictive mitral inflow E/A>2, DT 116 msec, respiratory changes in the mitral E velocity (>50%)

A subsequent cardiac catheterization indicated no significant coronary artery and a hemodynamic profile consistent with constrictive, showed elevation and equalization of diastolic filling pressures, and dip and plateau configuration of ventricular pressure during diastole (square root sign) (Figure 3).

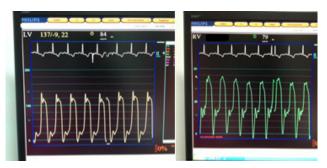


Figure 3 - showed elevation and equalization of diastolic filling pressures, and dip and plateau configuration of ventricular pressure during diastole (square root sign)

Based on these results the final diagnosis was constrictive pericarditis and pericardiectomy was considered necessary. Constrictive pericarditis was also confirmed at the time of surgery. The pericardium was found with thickening of up to 30

mm in some areas. Histopathological exam showed fibrosis and calcification within the pericardium with no evidence of malignancy or tuberculosis. Based on histopathological exam, laboratory exams and no significant medical history the diagnosis of idiopathic constrictive pericarditis was made. The patient was discharged 10 days later. At 3 months she had no significant symptoms.

Discussion

The diagnosis of constrictive pericarditis is still difficult and requires a high degree of clinical suspicion. Constrictive pericarditis is characterized by the thickening of the pericardium by chronic fibrosis resulting in severe diastolic dysfunction with abnormal diastolic filling of the ventricles due to constriction within the pericardial sac. The characteristic hemodynamic pattern consists of rapid ventricular filling in the early stages of diastole that ends when the stiff pericardium limits the filling. At this point all diastolic pressures are elevated and equalized ("diastolic equalization") [2]. Functionally this cause systemic venous congestion and failure of outflow. There are many causes of venous congestion, and due to the difficulty in clinical diagnosis, investigation are vital in making the diagnosis. A prior history of pericarditis, trauma or cardiac surgery makes the diagnosis of CP more likely [3].

Constrictive pericarditis and restrictive cardiomyopathy have some similarities in clinical presentation but their pathophysiology, clinical features, and more importantly therapeutic approach is different, so that the correct diagnosis should be made before taking the therapeutic decision [4]. Echocardiography, cardiac catheterization, CT and MRI are important investigation to establish the correct diagnosis.

There are a number of echocardiographic differences between constrictive pericarditis and restrictive cardiomyopathy, but because they also

share important haemodynamic characteristics, they have a number of Doppler characteristics in common. The most important is a restrictive mitral flow or ventricular filling pattern with striking E dominance and a short deceleration time, due to early rapid filling that is seen in both entities. In establishing the correct differential diagnosis between CP and RCMP the most important are the chances with respiration of the mitral flow velocity (early diastolic left ventricular filling increases with expiration and reciprocal changes in right-sided Doppler flows) [3,5]. The respiratory variation in ventricular filling velocity in RCMP is usually minimal, less than 10%, and in patients with CP may have variations as high as 30-40%. There are both false positive and negative results when examining the respiratory variation of mitral flow velocity to differentiate CP from RCMP. The ventricular filling velocity is highly influenced by preload, when LAP is greatly elevated respiratory variation in this parameter may not be seen in patients with CP, and in case of atrial fibrillation the presence of highly variable RR-interval makes the diagnosis of CP difficult [3,5].

Computed tomographic scanning of the heart in case of CP shows increased pericardial thickness and calcification, but a normal appearance or non-visualization of the pericardium does not exclude the diagnosis of CP, since pericardial stiffening can occur without calcification.

In some cases invasive cardiac catheterization is required to establish the diagnosis. Elevation and equalization of diastolic filling pressures occur in patients with CP, the dip and plateau configuration of ventricular pressure (square root sign) during diastole corresponds to a rapid early filling aided by augmented suction followed by a hampered further filling caused by rapidly increasing pressures.

In the reported case we did not considered necessary CT exam, because no pericardial thickening and calcification were seen in echocardiographic exam and chest x ray, and cardiac catheterization was necessary for the diagnosis of constrictive pericarditis.

Conclusion

Diagnosis of constrictive pericarditis remains challenging. Constrictive pericarditis should be suspected in patients with clinical features of right-sides heart failure. Echocardiography and cardiac catheterisation are important investigation in diagnosis of constrictive pericarditis and avoiding unnecessary treatments.

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