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CASE REPORT

Drainage of pleural effusions in the course of unrecognised constrictive pericarditis: a cause of severe haemodynamic deterioration

Drenaż wysięku opłucnowego w przebiegu nierozpoznanego zaciskającego zapalenia osierdzia: przypadek ciężkich powikłań hemodynamicznych

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Abstract

Constrictive pericarditis (CP) is a rare disease which can be easily overlooked in the absence of typical pericardial calcification. One of its most frequent manifestations is pleural effusion. We present a case report of a patient with unrecognised CP in whom plerocentesis led to haemodynamic collapse, and we speculate about the potential pathomechanism. No obvious criterion of CP or severe systolic dysfunction was found in baseline echocardiography. We consider that patients with advanced CP are remarkably prone to haemodynamic decompensation secondary to pleural drainage. Therefore every effort should be taken to recognise the disease and apply definitive treatment, i.e. pericardiectomy.

Key words: constrictive pericarditis, pleural effusion, drainage

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INTRODUCTION

Constrictive pericarditis (CP) is a rare disease which can be easily overlooked. It is believed that the main reason for CP is tuberculosis and that the disease presents as easily-detectable pericardial thickening, calcification, or both. This thesis remains true in some nonindustrialised countries [1, 2]. In developed countries, however, the most frequent causes of CP are prior cardiac surgery, radiotherapy and idiopathic pericarditis [3]. Typical calcification is observed only in 10% of patients with CP secondary to cardiac surgery, and in 55% of idiopathic forms [3].

Patients present clinically with typical symptoms of right heart failure that may also mimic liver cirrhosis. One of the most frequent manifestations of CP is pleural effusion. Expected echocardiographic findings are: thickened, sometimes calcified pericardium, unusual motion of the interventricular septum (an inspiratory septal shift to the left) and the presence of the dilated and non-collapsing inferior caval vein [4]. However, atypical forms of CP may develop with a pericardium of normal thickness [5]. On Doppler echocardiography, CP manifests with restrictive flow pattern and increased respiratory flow variation through mitral and tricuspid valve. Next, tissue Doppler reveals normal velocities. In general, echocardiography is not a definitive tool for recognising CP.

Proper diagnosis of CP is essential, while it can be treated in advanced cases with pericardiectomy [6, 7]. In many patients, however, the diagnosis of CP is tricky and congestive heart failure (CHF) remains for a long time a baseline diagnosis. Since CHF is resistant to pharmacological treatment, pleural puncture is sometimes undertaken to relieve symptoms. We found that it may have an unfavourable effect and speculate about the potential pathomechanism.

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CASE REPORT

We present the case of a 44 year-old male with a long history of liver cirrhosis of unknown origin complicated by severe CHF (New York Heart Association class IV). According to that, recurrent hydrothorax, enormous ascites, peripheral oedema and oesophageal varices (stage I) were treated as secondary to liver disease. During the last four years, he had peritoneal and pleural cavities drained several times (an overall documented 93 L of liquid was obtained). Finally, he was admitted to the intensive care unit due to an acute respiratory insufficiency in the course of pneumonia. After initial recovery, he had pleura punctured. A few hours afterwards, he developed pulmonary oedema and was transferred to our Department.

Baseline blood pressure was 100/60 mm Hg, heart rate 90/min. In our opinion, abdominal ultrasound showed no pronounced features of liver cirrhosis (contradicting previous results). Baseline echocardiography revealed no obvious criteria of CP or severe systolic dysfunction, only a small separation of visceral and parietal pericardium. No typical restrictive profile of mitral inflow was visualised (E/A = 1.3, DT = 105–120 ms); however, respiratory flow variation through the mitral valve was increased. Inferior caval vein was enlarged with limited respiratory collapse. Computed tomography showed substantial thickening of the pericardium (Fig. 1). The patient was qualified for early pericardiectomy, which confirmed the diagnosis. After the procedure, he initially improved, however, three weeks afterwards he died due to perioperative complications and severe general condition.

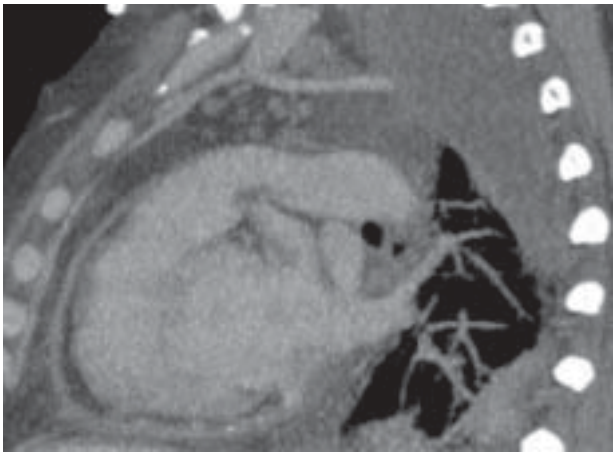

Figure 1. Computed tomography. Substantial thickening of the pericardium

DISCUSSION

We described a patient with unrecognised CP complicated by severe CHF that was resistant to standard pharmacological treatment. On the basis of the clinical course, two main problems are to be analysed: proper diagnosis and risky treatment.

There are well defined tools to recognise CP (computed tomography [CT], cardiac magnetic resonance, and cardiac catheterisation). However, reaching the diagnosis is still a challenge. There is no specific clinical manifestation. In our patient, symptoms were interpreted as CHF and, for years, CP was not even considered. Echocardiography served as a screening method. However, obtained images were not evident. This confirms again that subtle features of CP can be easily overlooked in routine examinations, if not suggested by a clinician. The first step of recognising CP was simply taking it into consideration. Anamnestic data such as history of coronary artery bypass grafting, postcardiotomy syndrome, radiotherapy and unexplained liver cirrhosis should be highly suspicious for CP. In our patient, the final diagnosis was based on CT, which is presently one of the standard methods for the accurate measurement of pericardial thickness [4, 8].

A few hours after pleural puncture, the patient developed a pulmonary oedema. Re-expansion pulmonary oedema has been described in the literature, but it concerned cases of rapid drainage of large effusions [9]. Patients with CP seem to be remarkably prone to haemodynamic decompensation following pleurocentesis and this phenomenon hasn't been previously explained. Marshall et al. [6] characterised 25 patients with CP. Because of breathlessness, some of them had pleural effusions drained, but the procedure was not described as directly leading to haemodynamic decompensation.

We speculate that a pressure drop in the chest following a drainage of pleural effusion increases venous return to the right ventricle. As a result, the right ventricle expands and its stroke volume increases. Simultaneously, due to stiffness of the pericardial sack, the septum is shifted to the left, leading to a decrease in left ventricular end-diastolic and stroke volumes. These two mechanisms together may lead to pulmonary congestion and/or blood pressure drop. On the other hand, pleural drainage leads to lung expansion and increase in pulmonary vascular bed. These should lead to decreased venous return to the left ventricle, which would prevent pulmonary congestion. As there are two potentially opposite mechanisms, we suppose that haemodynamic collapse would be a result of an imbalance between them.

We maintain that patients with CP are prone to haemodynamic decompensation following pleural drainage. Therefore, it should be used as a last resort method and always with special safety measures. The guidelines of the British Thoracic Society suggest a maximum of 1,500 mL drained at one time or a drainage speed of 500 mL per hour [9]. This concerns the general population of patients with pleural effusions, and one should consider further restrictions in patients with CP.

CONCLUSIONS

Constrictive pericarditis is in many cases a problematic diagnosis. It should be considered in patients with right heart failure that is not explained by echocardiogram findings and is resistant to standard pharmacotherapy. Ultrasound findings in CP may be easily overlooked; therefore, CT or cardiac magnetic resonance should be performed to judge the diagnosis. We maintain that patients with advanced CP are remarkably prone to haemodynamic decompensation secondary to pleural drainage. Therefore every effort should be taken to recognise the disease and apply the definitive treatment, which is pericardiectomy. In this way, one can also avoid pleural punctures that are high-risk procedures and act only symptomatically.

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