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CASE REPORT

Recurrent constrictive pericarditis: A diagnostic and therapeutic challenge☆

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KEYWORDS
Recurrent constrictive pericarditis;
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Abstract Recurrent right-sided heart failure after pericardiectomy may be caused by incomplete pericardiectomy, recurrent constriction, diastolic dysfunction or myocardial involvement. Identifying recurrent constrictive pericarditis (CP) in patients who have recurring symptoms after pericardiectomy is challenging, since the characteristic Doppler echocardiographic features may not be present if a portion of the ventricles are free of constricting pericardium, and there are no diagnostic or treatment guidelines for management of recurrent CP.

The authors report the case of a 59-year-old man with a history of pericardiectomy for tuberculous CP in 1984, admitted to our hospital with signs and symptoms of right heart failure. After a complete diagnostic workup, recurrent CP was diagnosed. Given the scarcity of cases reported on this disease, three possible therapeutic approaches are discussed: a second pericardiectomy, heart transplantation and medical therapy.

PALAVRAS-CHAVE
Pericardite constritiva recorrente;
Pericardectomia

Pericardite constritiva recorrente – um desafio diagnóstico e terapêutico

Resumo Insuficiência cardíaca direita recorrente após pericardectomia pode ser causada por pericardectomia incompleta, constrição recorrente, disfunção diastólica ou atingimento miocárdico. A identificação de pericardite constritiva (PC) recorrente após pericardectomia é desafiante, uma vez que, muitas das características da constrição podem estar ausentes e não existem ainda *guidelines* de diagnóstico ou terapêutica para a abordagem desta patologia dada a sua extrema raridade.

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Os autores descrevem o caso de um homem, 59 anos de idade, com antecedentes de pericardectomia após PC tuberculosa em 1984, internado para esclarecimento de um quadro clínico dominado por sinais e sintomas de insuficiência cardíaca direita. Após estudo complementar foi diagnosticado PC recorrente. Dada a escassez de casos reportados sobre esta patologia, foram discutidas possíveis abordagens terapêuticas nomeadamente uma segunda pericardectomia, transplante cardíaco e terapêutica médica.

Case report

The authors report the case of a 59-year-old man with a history of tuberculosis at age 12, who subsequently developed tuberculous constrictive pericarditis (CP) and underwent pericardiectomy in 1984. The surgical report refers to pericardiectomy by median sternotomy but gives no further details. He also had a history of permanent atrial fibrillation (AF), type 2 diabetes, hypothyroidism and multiple emergency department (ED) admissions for clinical settings interpreted as liver failure; he was medicated with warfarin, bisoprolol 2.5 mg daily, metformin 1000 mg twice daily and levothyroxine 0.1 mg daily. He was admitted to the ED again in September 2013 with abdominal discomfort, peripheral edema and ascites. On physical examination, he presented jugular distension, weak apical pulse, and audible and arrhythmic S1 and S2, with no cardiac murmurs; diminished breath sounds in the lower half of the right hemothorax; palpable hepatomegaly and hepatojugular reflux; and ascites and lower limb edema. He was admitted to the internal medicine department. During hospitalization, he suffered an episode of tight chest pain radiating to both arms, associated with profuse sweating, and he was accordingly transferred to the cardiology department for diagnostic investigation.

The electrocardiogram revealed AF with a pattern of right bundle branch block, right axis deviation and increased QTc interval (470 ms). Laboratory tests showed hemoglobin 13.0 g/dl, normal renal function and electrolytes, negative serial troponin I, pro-brain natriuretic peptide 313 pg/ml, negative C-reactive protein and procalcitonin, increased alkaline phosphatase (178 U/l) and gamma-glutamyl transpeptidase (109 U/l), normal aspartate aminotransferase, alanine aminotransferase and total bilirubin, reduced total protein (4.6 g/dl) and albumin (1.83 g/dl), normal protein electrophoresis, negative HIV, HBV and HCV, normal anti-neutrophil cytoplasmic antibody, immunoglobulin and complement, and normal thyroid function. Abdominal ultrasound showed an enlarged liver with a diffuse heterogeneous structure, suggesting chronic liver disease, and dilatation of the inferior vena cava and suprahepatic veins. The chest X-ray revealed calcification of the cardiac silhouette and right pleural effusion (Figure 1). Echocardiography showed severe left atrial dilatation (area 44 cm^2) and mild right atrial dilatation (25 cm^2), while the other chambers and the roots of the great vessels were of normal size; normal left ventricular thickness; altered interventricular septal motion; preserved left ventricular systolic function (ejection fraction 61%); no wall motion abnormalities; diastolic mitral flow compatible with a restrictive pattern (mean E/E′ 19); preserved right ventricular systolic function (tricuspid annular plane systolic excursion 17 mm); no significant valve abnormalities; pulmonary artery systolic pressure (PASP) estimated at 45 mmHg; thickening of the diaphragmatic pericardium due to fibrosis; and dilatation of the inferior vena cava and suprahepatic veins. Coronary angiography revealed no coronary artery disease (Figure 2), but hemodynamic data on catheterization were suggestive of constrictive physiology: dip-and- plateau pattern of the ventricular curve; equalization of ventricular end-diastolic pressures; right curve with prominent y descent; and mild pulmonary hypertension (PASP 45 mmHg) (Figure 3). Magnetic resonance imaging showed foci of hypointense signal in the region of the inferior pericardium due to residual calcifications and an associated ventricular restrictive component,

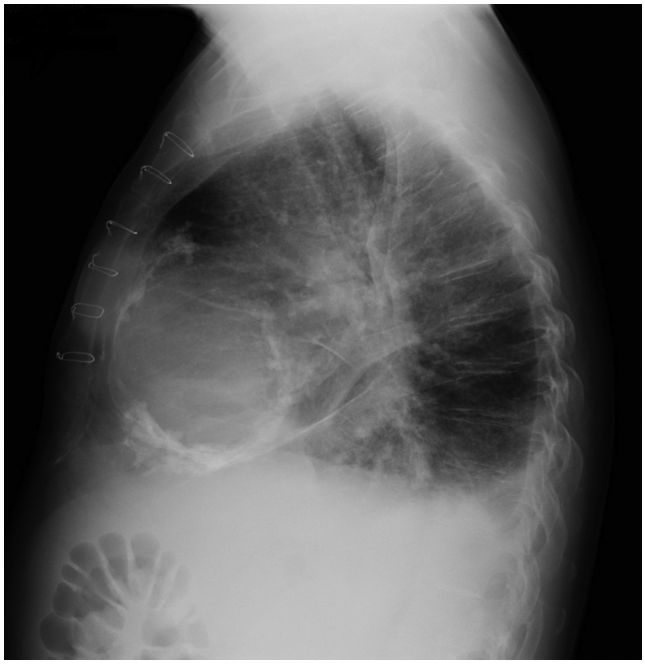

Figure 1 Chest X-ray, left lateral view, showing calcification of the cardiac silhouette.