Organized intrapericardial haematoma: a rare cause of heart failure after 2 years of trauma

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Intrapericardial organized haematoma secondary to blunt chest trauma is an extremely rare cause of constrictive pericarditis. We report a 30-year-old male who presented with heart failure for 12 months and was found to have an organized intrapericardial haematoma secondary to blunt chest trauma in a road traffic accident 2 years prior. The use of multiple imaging modalities including two-dimensional (transthoracic and transoesophageal) echocardiogram and cardiac magnetic resonance imaging established the diagnosis. Surgical excision of the haematoma and removal of the constricting pericardium relieved his symptoms.

Keywords
Constrictive pericarditis • Trauma • Cardiac failure • Pericardial haematoma

Introduction
Constrictive pericarditis (CP) is an uncommon disorder characterized by rigid pericardium due to fibrosed and fused pericardial layers. Although commonly idiopathic, pericarditis, cardiac surgery, and mediastinal irradiation are the leading identifiable causes.1 Bleeding into the pericardium can occur following chest trauma, with resultant adhesions between the pericardial layers. Post-traumatic formation of a large, organized, calcified haematoma is very rare.2,3 We present a patient who had a blunt chest trauma, complicated by calcific CP. The patient remained asymptomatic for 2 years then he started to have symptoms and signs of heart failure (HF).

Case report
A 30-year-old male presented with progressive shortness of breath (NYHA class III) and generalized oedema for the last 12 months. He sought medical advice at another hospital where the transthoracic two-dimensional echocardiography (2DE) at that stage showed normal left ventricular (LV) function and mild to moderate pericardial effusion. He was treated as HF.

On physical examination, he was tachypneic with a respiratory rate of 30 bpm without signs of respiratory distress. His pulse was 95 bpm with a blood pressure of 110/75 mmHg. Pertinent findings included a jugular venous pressure of 8 cm, muffled heart sounds with no audible murmur, rub, or gallop and marked bilateral lower limb pitting oedema.

Chest examination revealed a stony dullness with reduced air entry to the lower zone. His abdomen was symmetrically enlarged with soft and tender hepatomegaly and positive shifting dullness. Routine laboratory tests showed normal electrolyte levels, liver and renal function with a serum brain natriuretic peptide level of 380 pg/dL, and a negative troponin I level (<0.02 mg/dL). The electrocardiogram showed normal sinus rhythm and diffuse low-voltage QRS, and his chest X-ray demonstrated mild cardiomegaly and left pleural effusion.

Transthoracic two-dimensional echocardiography showed a pronounced ventricular diastolic septal bounce with normal LV dimensions and an ejection fraction of 60%. There was a large intrapericardial mass surrounding the heart more pronounced posteriorly measuring (1.7 cm). The mass appeared to be organized, with soft-tissue, proteinaceous-fluid components, and calcification (Figure 1A and B, Supplementary data online, Movie 1). Both atria were mildly dilated with exaggerated respiratory variation of mitral and tricuspid flows and dilated (28 mm) non-collapsing inferior vena cava (IVC). A restrictive LV filling pattern was also noted.

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Cardiac magnetic resonance (CMR) showed pericardial thickness of 1.7 cm mainly at the inferoposterolateral walls and anterior to the right atrium and right ventricle. There was an organized hematoma sandwich between the visceral and parietal pericardial layers (Figure 1C and D, Supplementary data online, Movie 2). The septum was bouncing, and both atria and vena cavae were dilated (superior vena cava 25 mm, IVC 28 mm).

In the absence of history of tuberculosis, cardiac surgery, or mediastinal irradiation, it was thought that his clinical findings were most likely secondary to the blunt chest trauma he sustained 2 years earlier.

The patient underwent total pericardiectomy. Intraoperative transesophageal 2DE confirmed the transthoracic 2DE and CMR findings (Figure 2A and B, Supplementary data online, Movie 3). Intraoperative haemodynamic measurements showed mildly elevated pulmonary artery pressure (46/19 mmHg) and pulmonary capillary wedge pressure (20 mmHg). Cardiac index (CI) was 2 L/m².

Surgical inspection revealed a calcified thickened pericardium and an organized hematoma between the pericardial layers, which was excised and sent to pathology (Figure 2C, Supplementary data online, Movie 4). Microscopy revealed fibrosis with focal granulation tissue, areas of ossification, and necrosis.

Immediate post-op cardiac parameters showed improvement of CI (3.7 L/m²) and reduction of pulmonary capillary wedge pressure to 10 mmHg.

The patient was kept on oral diuretics post-operatively and had remarkable improvement of his symptoms. After 3 months, transthoracic 2DE demonstrated normal cardiac chambers. Remnants of calcified mass were present but without any significant haemodynamic effect (Figure 2D).

**Discussion**

Constrictive pericarditis is a chronic fibrous thickening and/or calcification of the pericardial sac. The most common identifiable causes include prior surgery and irradiation, but many cases are idiopathic in origin. Chest-wall trauma can produce hemopericardium, which ultimately leads to CP that can produce HF, often with a preserved LV systolic function. Delayed hemopericardium with CP after blunt thoracic trauma was previously described. Manhas et al. reported a patient who experienced chest trauma at the age of 16 years, and developed HF 40 years later secondary to a large and calcified pericardial haematoma.

Advances in cardiac imaging allow the diagnosis of CP to be made non-invasively in nearly all patients. No one method is completely reliable. Data from more than one imaging method should be considered to provide an integrative assessment of anatomical and physiological function. Characteristic transthoracic 2DE features of CP include pericardial thickening, myocardial tethering, a
septal bounce, significant respiratory variation in transmitral, pulmonary vein, and tricuspid inflows, and preserved indices of myocardial relaxation (velocity of propagation and early mitral annular velocity). Computed tomography (CT) scan has a high sensitivity in detecting pericardial calcification, whereas CMR is superior to CT in differentiating pericardial thickening from small effusions.

Our patient had a large organized pericardial haematoma due to chest trauma. However, he had no symptoms for the first 2 years after the trauma. He then developed intractable symptoms of HF for the last 12 months and yet no follow-up echocardiography was made to pick up the early signs of CP.

Although the standard imaging methods in CP should be CT scan and CMR, in our patient, transthoracic 2DE was capable of detecting pericardial thickening, and precisely identified the focal organized haematoma. He had a complex lesion manifested mainly with constrictive and little restrictive features. The constrictive features included equalization of diastolic pressures in all chambers, and the pronounced septal bounce. Restrictive features included mild bialtrial enlargement, elevated systolic pulmonary artery pressure, and a restrictive mitral inflow pattern. Intraoperative transesophageal 2DE was extremely helpful in guiding the surgeons towards the extent of the organized haematoma and ensuring relief of constriction features.

Surgical pericardiectomy, which should be as complete as possible, is the definite therapy, which offers the possibility of cure, with a low mortality rate, if performed in patients in good condition, and without advanced disease. In the majority of cases, improved or normalized cardiac function is achieved after pericardiectomy.

This case report emphasizes the importance of recognizing this uncommon, yet potentially curable cause of HF. Due to its insidious onset and progressive nature, it would be prudent to follow patients periodically with serial echocardiography after blunt chest trauma to make an early diagnosis of CP.

**Supplementary data**

Supplementary data are available at European Journal of Echocardiography online.

**Conflict of interest:** none declared.

**References**