

A Case of Impending Cardiac Tamponade Caused by Effusive Constrictive Pericarditis

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Abstract

Pericarditis is the most common pericardial disease worldwide, and can be recurrent in one third of the patients with viral and idiopathic pericarditis [1]. The diagnosis of pericarditis should be based on clinical criteria, history, clinical findings, electrocardiographic (ECG) changes and evidence of a pericardial effusion [2]. Elevation of the inflammatory markers and evidence of pericardial inflammation by an imaging technique such as contrast enhancement on the pericardium-CT scan or pericardial edema and pericardial late gadolinium enhancement on cardiac magnetic resonance imaging (MRI) are additional supportive criteria in the diagnosis [1].

Keywords: Cardiac Tamponade; Pericarditis; Pericardium-CT scan; Magnetic resonance imaging

1. Case Study

A 33 year old man, with history of recurrent pericarditis, presented with a new episode of chest pain, associated with EKG changes suggestive of pericarditis (Figure 1). A cardiac MRI showed normal thickness of the pericardium with trace effusion (Figure 2). There was no delayed gadolinium enhancement of the pericardium. Patient had extensive workup that was positive for Cocksackie B infection. Patient was started on aspirin and colchicine. A week later, patient presented to the emergency department with worsening symptoms of shortness of breath. On physical

examination, blood pressure 90/66 mm hg, pulse 90/min, elevated jugular venous pressure with y descent, pulsus paradoxus, pulsatile hepatomegaly, and mild peripheral edema. Laboratory testing showed ESR of 36 mm/hr, and a CRP of 156.6 mg/L. Chest X-ray showed enlargement of the cardiac silhouette (Figure 3). A high resolution CT scan of the chest showed moderate size pericardial effusion (Figure 4) that was not present before in the initial MRI. A transthoracic echocardiography (TTE) showed evidence of a large circumferential pericardial effusion (Figure 5) with inferior vena cava (IVC) plethora (Figure 6) and both right atrium (RA) and right ventricle (RV) collapse consistent with impending cardiac tamponade. An urgent pericardiocentesis was performed and drained a one liter of sero-sanguineous pericardial fluid with WBC: 1403; PMN: 44%, Lymph: 45%. Cultures were negative and cytology was negative for malignancy. A follow up TTE evidenced resolution of the pericardial effusion, and diffuse thickening of the pericardium, with persistence of a mild respiratory variation in the left ventricle (LV) and right ventricle (RV) filling concerning for pericardial constriction (Figure 7). A heart catheterization confirmed ventricular intra-dependence on simultaneous measurements of the LV and RV consistent with pericardial constriction. Patient was diagnosed with an effusive constrictive pericarditis, and was started on a trial of steroids following which he had significant clinical improvement.

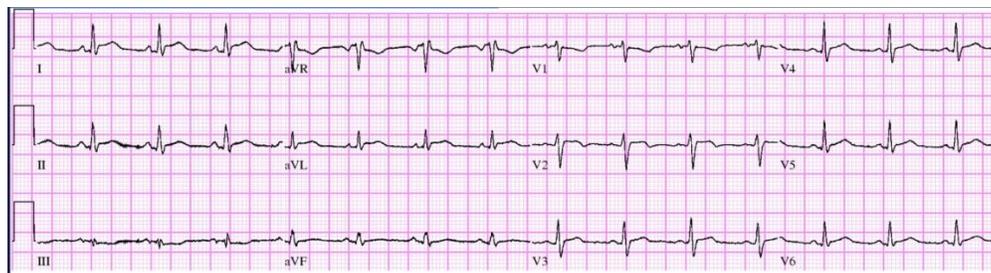


Figure 1: ECG.

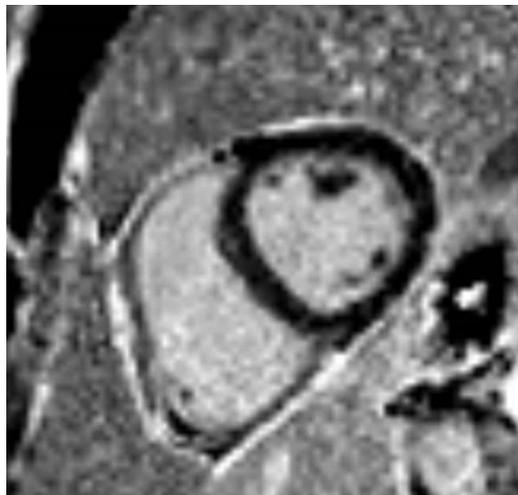


Figure 2: Cardiac MRI.



Figure 3: Chest X-ray.



Figure 4: Chest CT scan.

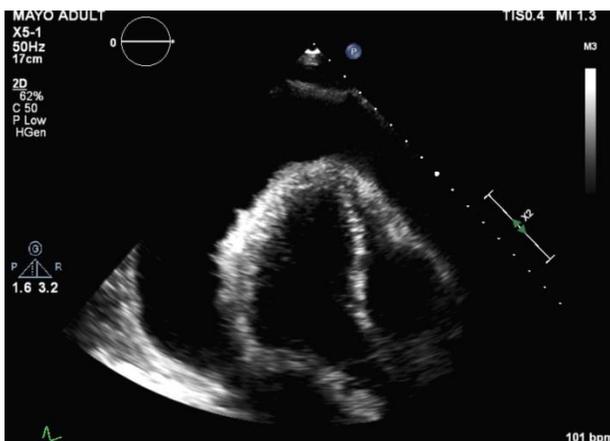


Figure 5: Transthoracic echocardiography.

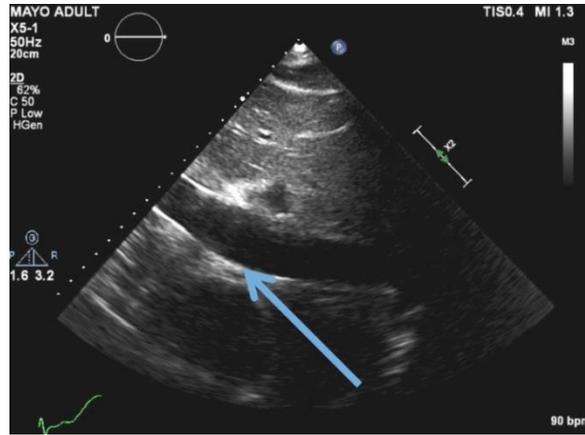


Figure 6: Inferior Vena cava.

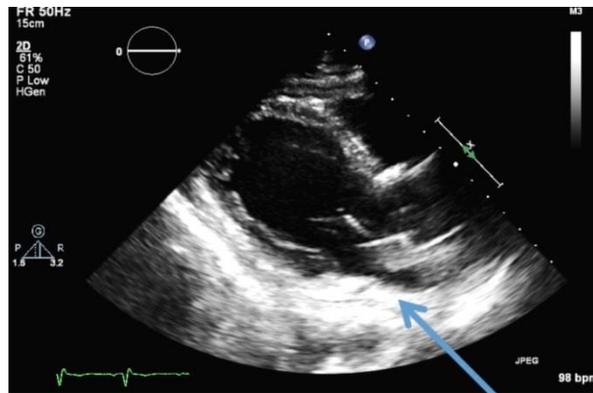


Figure 7: Transthoracic echocardiography post pericardiocentesis.

2. Discussion

Cardiac imaging is frequently used in the diagnosis of pericardial disease. The multimodal integration of different imaging techniques is needed in certain cases [3, 4]. TTE should be used in all the cases of acute pericarditis to identify effusions, and to evaluate for hemodynamic compromise as a reflection of cardiac tamponade [5, 6]. Other diagnostic approaches: hemodynamic cardiac catheterization may be necessary in establishing the diagnosis of constrictive pericarditis when the echocardiogram findings are not conclusive [5]. MRI has become the gold standard for the evaluation of the pericardium. The advantages include: lack of ionizing radiation, better anatomic evaluation and providing physiologic information regarding the cardiac function and wall motion in a non-invasive manner [7]. The sensitivity for late gadolinium enhancement has been reported to range from 94-100% [8, 9]. Even highly sensitive modalities may initially be noncontributory in the initial phase of the disease. Particularly in our case the evidence of a normal MRI could have been due to an early stage of the disorder. And sometimes multiple imaging techniques are used in association with clinical findings to provide a confident diagnosis [8]. Although an important number of patients will require pericardiectomy, some cases have a predominantly inflammatory and reversible pericardial reaction, so a trial of anti-inflammatory agents should be considered before advocating pericardiectomy [10].

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