

Pleuro-Pericardial Effusions Post Dual Chamber Permanent Pacemaker Placement

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I. CASE PRESENTATION

A 61 year old female with history of hypertension, coronary artery disease and sick sinus syndrome presented to emergency room for substernal chest discomfort, dyspnea and subjective low grade fever for one week. She denied significant cough or sputum production. She had dual chamber permanent pacemaker implantation through standard subclavian approach two weeks prior to the onset of symptoms. Physical examination showed temperature 98.5° F, blood pressure 105/62, heart rate 70, respiratory rate 16 and oxygen saturation 91% on ambient air. Chest auscultation revealed diminished breath sounds in bilateral lung bases and muffled heart sounds. There was no lower extremity edema. Laboratory results on admission showed leukocytosis (WBC 11,500/ μ L) with differential counts (neutrophils 82%, lymphocytes 11%, monocytes 7%), elevated ESR (78 mm/hr) and CRP (3.35 mg/dl). BNP was 166. ECG showed normal sinus rhythm. Computed tomography (CT) of chest showed moderate left-sided pleural effusion, small right-sided pleural effusion, basilar infiltrates suggestive of atelectasis and a moderate size pericardial effusion. Pacemaker leads appeared in place, and there were no signs of atrial or ventricular free wall perforation (Figure 1). Echocardiogram confirmed moderate size pericardial effusion with no signs of tamponade physiology and normal left ventricular systolic function (Figure 2). Pacemaker interrogation showed normal thresholds and impedance. Left thoracentesis was performed and 600 ml of serosanguinous effusion was drained. The pleural fluid analysis showed elevated WBC with predominant lymphocytes, elevated lactate dehydrogenase (446 U/L) consistent with exudative nature, normal glucose (103 mg/dl) and elevated pH (>7.48). Pleural fluid gram stain did not show any organisms and cultures were negative.

What Would You Do Next?

- A. Consult cardiothoracic surgery
- B. Pericardiocentesis
- C. Diuresis
- D. Colchicine and steroids

Answer

D

Diagnosis:

Post Cardiac Injury Syndrome (PCIS)

The patient was initially started on empiric broad spectrum antibiotics for presumed infectious process based on clinical and radiological findings; however, she did not have any significant clinical improvement after 4 days of therapy. The patient continued to be symptomatic, hypoxic requiring supplemental oxygen, and repeat CT chest showed recurrence of the pleural effusion on left side. A repeat left thoracentesis performed and 350 ml of serosanguinous fluid was drained. Repeat blood work also showed worsening WBC (20,900/ μ L). Pleural fluid cultures remained negative. Infectious etiology was therefore highly unlikely, and the patient was diagnosed clinically as post cardiac injury syndrome (PCIS) based on the timing of the pacemaker placement, presence of both pleuro-pericardial effusions (with left pleural more than right), elevated inflammatory markers, and lymphocytic exudative nature of the pleural effusion. Antibiotics were discontinued, and the patient was started on colchicine and steroids. Over the next three days the patient had significant clinical improvement; she was weaned off supplemental oxygen therapy and eventually discharged home.

Non-steroidal anti-inflammatory drugs (NSAIDs) can also be used to treat PCIS but not given for this patient because of the history of GI bleed.

Pacemaker lead perforation was considered but ruled out based on the lack of convincing evidence of cardiac chamber perforation on the CT chest and echocardiogram and normal sensing parameters on pacemaker interrogation. Additionally, pleural fluid was not bloody; hence cardiothoracic consultation would not be beneficial here.

Pericardiocentesis was not indicated as there were no signs of tamponade physiology on echocardiogram. Diuretics indeed would be relatively contraindicated in the setting of moderate pericardial effusion as these patients are preload dependent and there was no evidence of fluid overload or congestive heart failure.

Discussion:

PCIS is an inflammatory process involving the pericardium and pleura in response to cardiac injury and has been described after myocardial infarction, pericardiectomy,

chest wall trauma and percutaneous coronary interventions including pacemaker insertion and ablation procedures [1,2]. Incidence of PCIS following pacemaker insertion is rare (<5%) [2]. It usually occurs within 2 months following the procedure [2]. The mechanism of pacemaker-induced PCIS is likely secondary to localized microtrauma at the site of lead insertion eliciting an inflammatory and autoimmune reaction [2,3]. Most common symptoms include chest pain, dyspnea and low-grade fever and radiological findings include pericardial effusions in >80% of patients and pleural effusions in >60% of patients [5]. PCIS effusions are usually exudative and pleural fluid can be associated with high pH [3].

Pericardial tamponade can result from large pericardial effusions requiring pericardiocentesis [2,4]. Pacemaker lead perforation should be ruled out in all of these cases with the help of CT scan of chest, echocardiogram and pacemaker interrogation. It can occur acutely within 24hr or as late as more than one month [4]. Treatment of PCIS includes NSAIDs, colchicine and steroids. Nearly two-thirds of PCIS cases have spontaneous recovery but early treatment with NSAIDs improves the remission rate and reduce the recurrence rate by half. Colchicine and steroids are usually used when there is a contraindication to NSAIDs [5].



Fig. 1. Computed tomography of chest axial view

- A. Pleural effusion
- B. Pericardial effusion

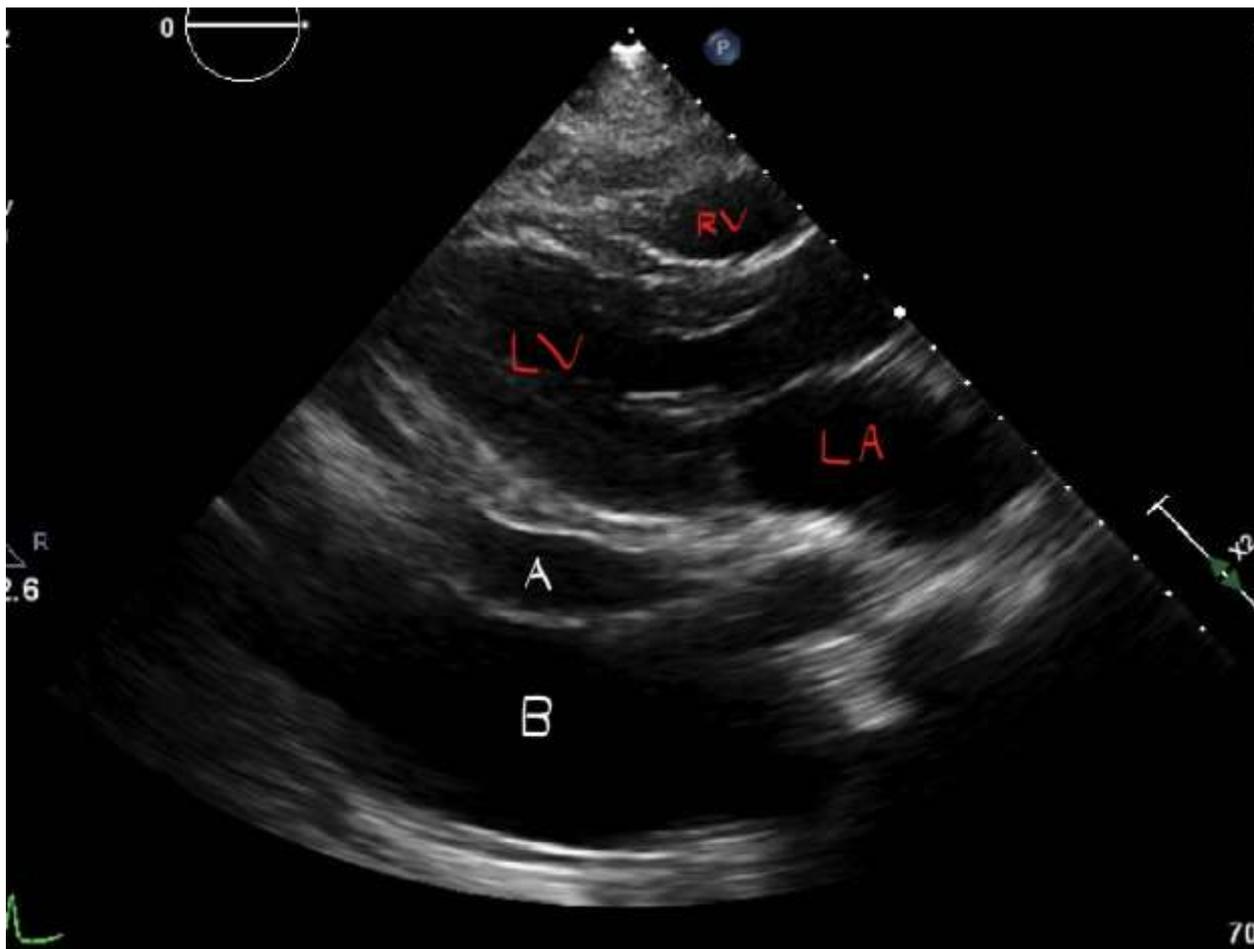


Fig. 2. Transthoracic echocardiogram parasternal long axis view

- A. Pericardial effusion
- B. Left pleural effusion
- LV – Left Ventricle
- RV – Right Ventricle
- LA – Left Atrium

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