Review

Post-cardiac injury syndrome following transvenous pacemaker insertion: A case report and review of the literature

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Post-cardiac injury syndrome is an inflammatory process involving the pleura and pericardium secondary to cardiac injury and can develop following transvenous pacemaker insertion. We now report a patient who developed this syndrome following dual-chamber pacemaker insertion with active fixation of the atrial and ventricular leads. The pericardial fluid was bloody and had a neutrophilic predominance. The pericardial biopsy revealed fibrinous pericarditis with a mixed inflammatory cell infiltrate. The pleural effusion was exudative and had a neutrophilic predominance. Nine similar cases were identified in the English literature dating back to 1975. These patients usually present within one month after pacemaker insertion. They have exudative pericardial and pleural effusions. The pericardial effusions can cause tamponade and may require treatment with either catheter drainage or a surgical window. Some of these patients respond well to anti-inflammatory medications, including prednisone. Therefore, early identification of these patients could allow medical treatment and might help avoid the need for a surgical procedure. Cardiologists should remember that this uncommon syndrome can occur after routine endovascular procedures.

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Keywords. Pericarditis; Pleural effusion; Cardiac injury; Transvenous pacemaker

Introduction

Post-cardiac injury syndrome (PCIS) is an inflammatory process involving the pleura (pleural effusion) and pericardium (pericarditis, pericardial effusion) secondary to cardiac injury. PCIS can develop after cardiac trauma, cardiac surgery, acute myocardial infarction, and some intravascular procedures, including transvenous pacemaker lead insertion, electrophysiological studies such as AV node ablation, and percutaneous coronary interventions [1,2]. This syndrome can develop after these intravascular procedures in the absence of obvious cardiac perforation. We now report an additional case with this syndrome secondary to transvenous pacemaker insertion and review the literature on prior cases. Our case appears unique since we report the histological changes in the pericardium.

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Case

A 65-year-old African American man with hypertension presented to the hospital with chest pain. Vital signs, physical examination, and routine laboratory tests were within normal limits except mild anaemia (WBC 4.5 K/μL, Hb 12.7 g/dL, Htc 36%, Na 140 mmol/L, K 3.8 mmol/L, BUN 23 mg/dL, creatinine 1.3 mg/dL). ECG revealed normal sinus rhythm. Transthoracic echocardiogram revealed dilated cardiomyopathy with depressed left ventricular systolic function (ejection fraction—43%). Coronary angiogram performed to rule out ischaemic cardiomyopathy did not show any obstructive epicardial coronary artery disease. There was no history of arrhythmia, syncope, or cardiac arrest in the past. During the catheterisation he had severe sinus bradycardia and sustained ventricular tachycardia unrelated to catheter manipulation or contrast injection. Therefore, his clinical condition was discussed with the patient and an electrophysiologic (EP) study was recommended because of his increased risk for sudden cardiac death. Holter monitoring was not performed. The EP study demonstrated sinus node dysfunction and inducible polymorphic ventricular tachycardia. Two days later he was taken to the cardiac catheterisation lab for

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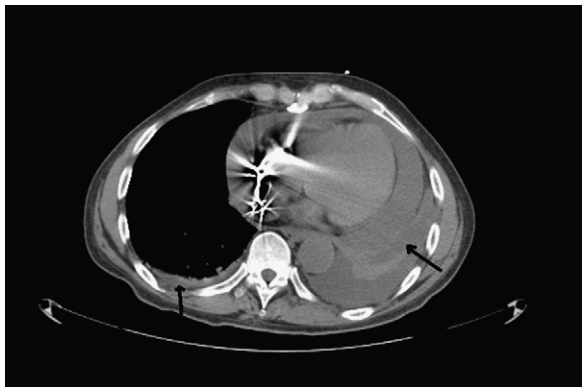

Fig. 1. *CT of the thorax reveals a large pericardial effusion and bilateral pleural effusion (arrows) (greater on the left).*

insertion of AICD (Guidant VITALITY®). Using the standard technique, the right subclavian vein was cannulated, and a 12 French sheath was introduced by the Seldinger technique. The two pacemaker leads (St. Jude Medical, 1388T, Tendril® DX) were positioned in the right ventricle near the apex (pacing threshold 0.9 V, R wave sensing 4.0 V) and in the right atrial appendage (pacing threshold 1.0 V, P wave sensing 3.0 V). The operator used active fixation for both leads. There were no procedural complications. There was no evidence or suspicion of possible lead perforation or penetration.

Five weeks later the patient returned with increasing shortness of breath. The complete metabolic panel and blood cell counts were similar to prior lab. He did not have fever. His sedimentation rate was 48 mm/h. An echocardiogram and CT of the chest revealed large pericardial and left pleural effusion (Fig. 1). There were no signs of tamponade physiology. There was no sign of pacemaker dysfunction. Sensing and pacing thresholds were optimal. A pericardial window for pericardial fluid drainage was created under general anaesthesia using a subxiphoid incision. A 3 cm × 3 cm section of pericardium was sent for pathology. Pericardial fluid analysis revealed RBC 565,000/cmm, WBC 7500/cmm, neutrophils 81%, lymphocytes 18%, LDH 1557 IU/L, pH 8.0, protein 6.9 g/dL, pericardial fluid to serum protein ratio 0.8, and no malignant cells. Pleural fluid analysis revealed RBC 325/cmm, WBC 225/cmm, neutrophils 51%, lymphocytes 5%, macrophages 13%, mesothelial cells 22%, LDH 427 IU/L, glucose 89 mg/dL, pH 8.0, protein 6.2 g/dL, pleural fluid to serum protein ratio: 0.72, and no malignant cells. Cultures of pericardial and pleural fluids were negative. The pericardial biopsy revealed a fibrinous pericarditis with a mixed inflammatory cell infiltrate (mostly macrophages and lymphocytes) (Fig. 2). ANA and rheumatoid factor levels in the serum were negative. His clinical condition gradually improved. He received aspirin (325 mg daily) but no nonsteroidal anti-inflammatory drugs (NSAIDS) or corticosteroids. A chest x-ray obtained four months later revealed a normal cardiac silhouette and no pleural effusion. Transthoracic echocardiogram revealed that there was no pericardial effusion.

Methods

We searched the English literature with the PubMed data base using the MESH terms pericarditis, pacemaker, pleural effusion, and pericardial effusion. We also carefully reviewed the reference list of all identified cases.

Results

We identified nine case reports related to pacemaker implantation published since 1975 (Table 1) [3–11]. These reports included six women and three men. The age ranged from 54 to 84. Six patients had no prior diagnosis

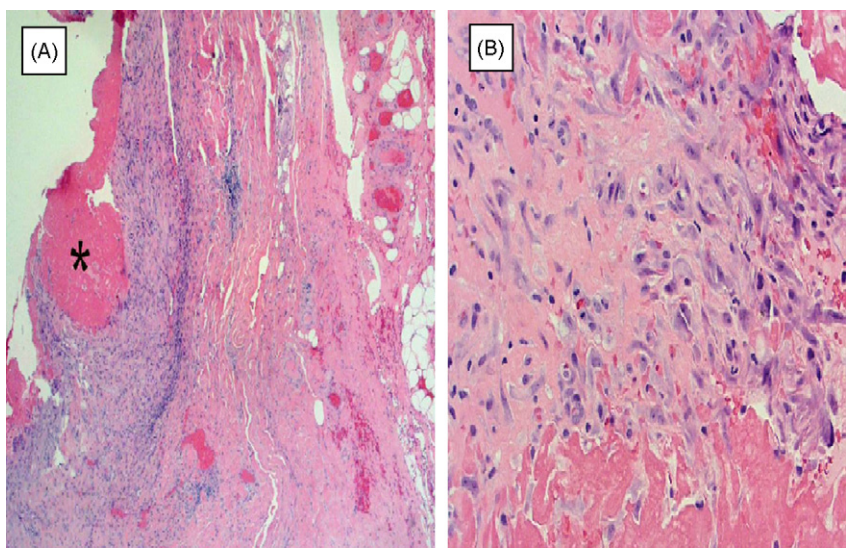

Fig. 2. *(A) A mixed inflammatory infiltrate is seen in the (zonated) pericardium which is topped with fibrin (*). (Haematoxylin–eosin stain, original magnification ×40.) (B) Area of the pericardium, which is infiltrated with macrophages and lymphocytes, and adjacent fibrin. Scant neutrophils are present. (Haematoxylin–eosin stain, original magnification ×200.)*