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Case Report

Pacemaker lead perforation causing hemopericardium eight years after implantation

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ABSTRACT

The number of patients with intracardiac devices, including permanent pacemakers and implantable cardioverter-defibrillators is increasing. Lead perforation is a recognized complication which most often occurs during or shortly following pacemaker implantation. Late lead perforation occurring over 30 days after device insertion is a rare, potentially life-threatening complication. We present a case of late lead perforation unmasked greater than eight years after pacemaker implantation by initiation of anticoagulation.

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1. Introduction

The number of patients with intracardiac devices, including permanent pacemakers (PPM) and implantable cardioverter-defibrillators (ICD) is increasing. Lead perforation (LPF) is a recognized complication which most often occurs during or shortly following PPM implantation. Late LPF occurring over 30 days after device insertion is a rare, potentially life-threatening complication. We present a case of late LPF unmasked greater than eight years after PPM implantation by initiation of anticoagulation.

2. Case

A 74-year-old man with multiple medical comorbidities including sinus node dysfunction, status-post dual-chamber

PPM with a bipolar active-fixation right ventricular (RV) lead (Medtronic, model 5068-58) implanted 8 years prior presented to the emergency department with a two week history of left leg swelling. Lower extremity ultrasound demonstrated extensive acute deep venous thrombosis. Treatment with warfarin was initiated after the immediate administration of low-molecular-weight heparin bridging therapy and he was discharged home. The following day he awoke with sharp, nonexertional, central chest pain prompting return to the emergency department. Vital signs were within normal limits. Electrocardiogram showed normal sinus rhythm with a ventricular rate of 69 beats/minute and low anterolateral forces. Chest X-ray (Fig. 1) revealed an enlarged cardiac silhouette and a left-sided chest wall PPM with leads that appeared to be appropriately positioned. CT pulmonary angiogram was performed which demonstrated no pulmonary embolus; however, the RV PPM lead was seen to penetrate through the RV

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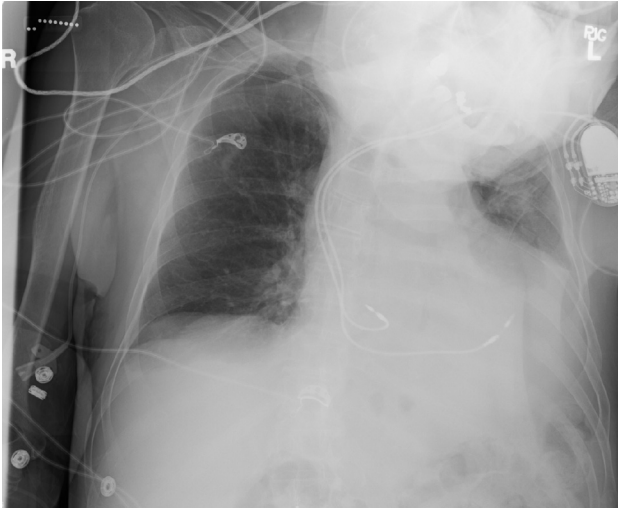


Fig. 1 – Portable chest X-ray (anterior–posterior view) showing a mildly enlarged cardiac silhouette and left pleural effusion. Left chest wall pacemaker is in place with leads in the right atrium and right ventricle.

apex with an associated moderately-sized hemopericardium (Fig. 2). Transthoracic echocardiogram revealed a moderate circumferential pericardial effusion. No extravasation of agitated saline was seen and there were no features of cardiac tamponade. Anticoagulation was discontinued and an inferior vena cava filter was inserted. Pericardiocentesis was performed draining 610 mL of bloody pericardial fluid. A pericardial catheter was left in situ for 5 days. Device interrogation showed that the device was set to DDDR mode. AV pacing and sensing delays were 150 and 120 ms, respectively. The lower pacing rate was 50 beats per minute, the upper tracking rate 90 beats per minute, and the upper sensing rate 90 beats per

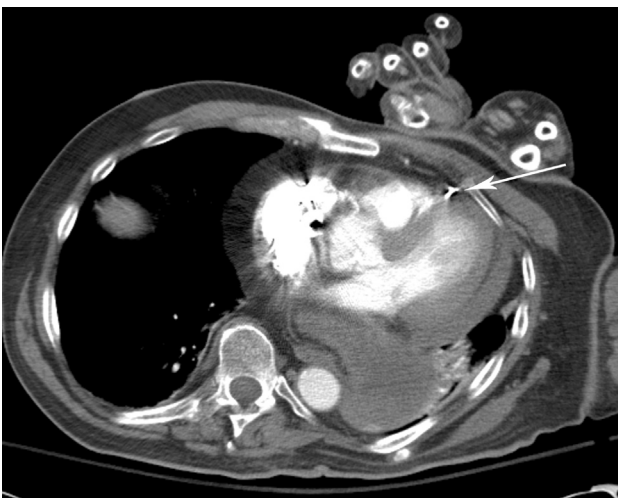


Fig. 2 – CT angiography of the chest with intravenous contrast (axial view) demonstrating a moderate amount of high density pericardial fluid consistent with hemopericardium. The tip of the right ventricular pacemaker lead can be seen penetrating the right ventricular wall at the apex (arrow).

minute. RA and RV impedance and pacing threshold were adequate. The underlying rhythm was sinus bradycardia at 55 beats per minute and the patient was not pacemaker-dependent. Given his poor functional status and the fact that there was no loss of capture or change in pacing threshold, he was managed conservatively without lead revision. He was discharged with colchicine for pain relief on day 6. He did well for the next six months until he declined elective PPM replacement due to low battery. Shortly thereafter, he entered a hospice program and expired.

3. Discussion

As the number of patients with PPMs and ICDs increases, long-term complications may become more prominent. Although late LPF can be life-threatening, it is most frequently detected incidentally in individuals undergoing imaging of the chest for other reasons. Risk factors for cardiac perforation and/or effusion immediately following PPM implantation include concomitant temporary transvenous pacing, recent oral steroid use, low body mass index, older age, longer fluoroscopy times, and the use of screw-in leads.¹

Data from the ACT and OPTIMUM Registries suggest an overall perforation rate of 0.1–0.8% with PPMs and 0.6–5.2% with ICDs.² Lead perforations are classified as acute, subacute or chronic if they occur within 7 days, between day 7–30 or greater than 30 days post-procedure, respectively. Although the vast majority of LPF occur during or shortly following device implantation, late PPM LPF has been reported in over 40 cases.³ Prior to this present case, the longest delay from implantation to diagnosis was seven years.⁴ The true incidence of asymptomatic cardiac perforation may be higher than previously suspected. Hirschl et al demonstrated a 15% perforation rate in asymptomatic patients with PPMs or ICDs based on chest CT evaluations.⁵ In their patient cohort, atrial and ICD leads were more likely to cause LPF than ventricular and PPM leads.⁵ Our patient had a PPM lead which perforated his RV, the rarest combination in their series.

Although we are uncertain exactly when our patient's LPF occurred, the 97-month delay from implantation to presentation is the longest reported to our knowledge. Most likely our patient had a long-standing asymptomatic RV perforation which was unmasked by anticoagulation. The incidence of hemopericardium in patients with cardiac devices who are anticoagulated is not well known. Although echocardiography was equivocal in visualizing the LPF in our patient, CT imaging clearly delineated the perforation and may be an effective adjunct in diagnosing LPF. It is unclear if either radiographic or echocardiographic screening should be recommended in patients with cardiac devices who are commencing anticoagulation but an awareness of the possibility of LPF in this setting is important. Furthermore, the significance and natural history of asymptomatic LPF is unclear and treatment may not be necessary. Further studies evaluating the long-term outcomes in patients with asymptomatic LPF may be of benefit, particularly in view of the need for anticoagulation in an increasing number of patients. For now, clinicians should consider lead complications such as perforation in all patients with cardiac devices who present

with chest symptoms, particularly after initiation of anticoagulation.

Conflicts of interest

All authors have none to declare.

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