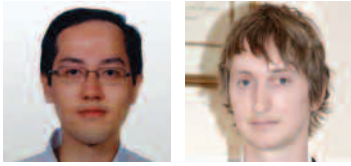


Post-device Chest Pain: A Cause for Concern

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About the Authors

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Case Presentation

A 73-year-old woman with a longstanding history of anthracycline-related dilated cardiomyopathy (left ventricular ejection fraction of 20%) sustained an out-of-hospital cardiac arrest. She was successfully resuscitated and underwent subsequent implantation of an implantable cardioverter-defibrillator (ICD) for secondary prevention.

One week following ICD implantation, she returned to the hospital with pleuritic chest pain. An electrocardiogram (EKG) tracing showed diffuse ST elevation. Her serum troponin was not elevated. A provisional diagnosis of pericarditis was made, and after 24 hours of in-hospital monitoring, she was discharged home on high-dose acetylsalicylic acid therapy.

She returned 3 days later after experiencing progressive shortness of breath and fatigue, culminating in a syncopal episode after taking nitroglycerin for transient neck pain. She denied any chest pain, palpitations, orthopnea, or ankle swelling. There had been no changes to her chronic beta-blocker, angiotensin-converting enzyme inhibitor,

or statin therapy. She was on a tapering course of prednisone for a recent exacerbation of chronic obstructive pulmonary disease.

In the emergency room, she had a heart rate of 70 beats/minute, a blood pressure of 66/31 mm Hg (without pulsus paradoxus), and a normal temperature. She was tachypneic at 18 breaths/min, and her oxygen saturation on 4 L/min of nasal prongs oxygen was 100%. The internal jugular waveform was not visible, but the external jugular vein was distended. Heart sounds were clear, and there were no murmurs present. Widespread crackles were heard on auscultation of the left chest, consistent with her known radiation-induced lung disease. There was no pedal edema or calf asymmetry. Abdominal examination was normal.

Her EKG (Figure 1) showed preserved QRS voltages and evidence of left ventricular hypertrophy with ST-segment depressions in the inferior and lateral leads, unchanged from previously. A chest radiograph showed caudal migration of the right ventricular AICD lead when compared with her discharge film (Figure 2). There was no

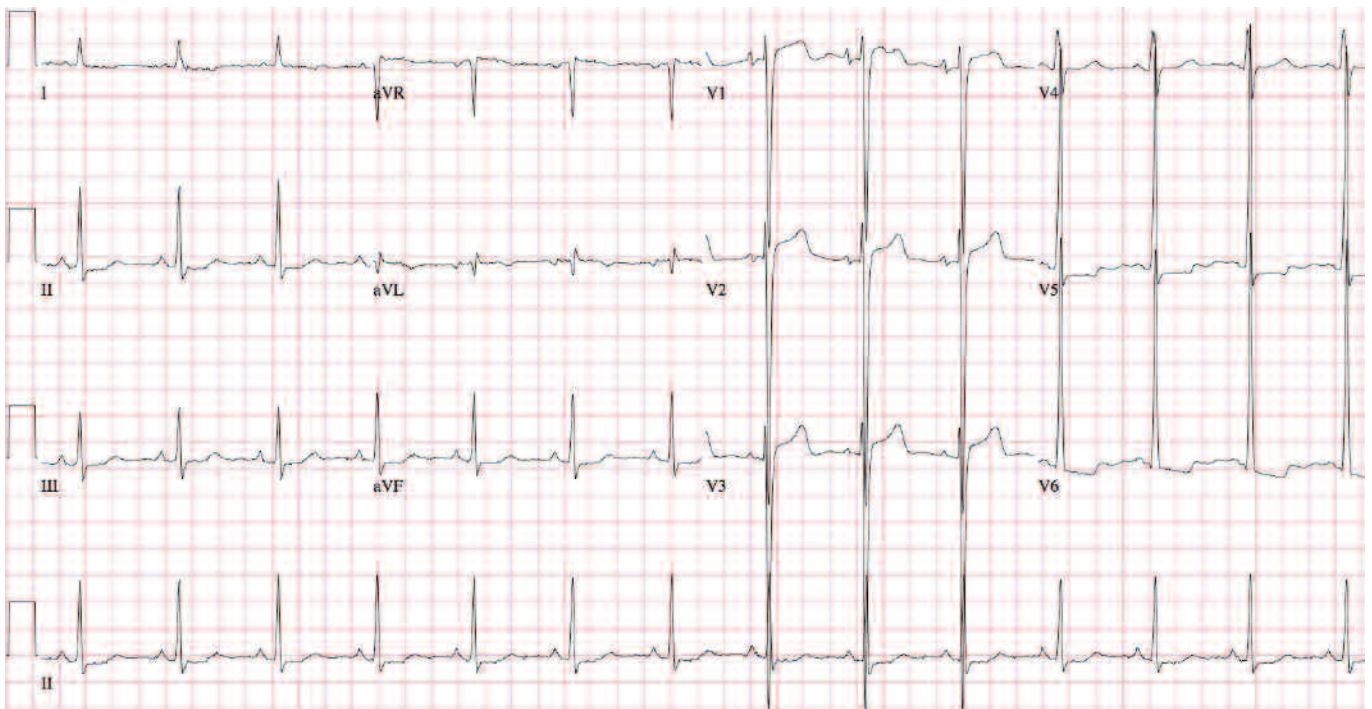


Figure 1. Electrocardiogram demonstrating preserved QRS voltages and evidence of left ventricular hypertrophy with ST-segment depressions in the inferior and lateral leads – unchanged from previously.

evidence of pulmonary edema, and the cardiac silhouette and mediastinum appeared unchanged.

The patient remained hypotensive despite fluid resuscitation, and progressive hemodynamic instability required the use of vasopressors. An emergency bedside echocardiographic study revealed 2 cm of circumferential pericardial fluid with right atrial diastolic collapse suggesting cardiac tamponade. Urgent pericardiocentesis via a sub-xiphoid approach was performed. Aspiration of approximately 120 cc of non-clotting, sanguineous pericardial fluid resulted in an immediate improvement and the return of hemodynamic stability.

Interrogation of her AICD showed the right ventricular lead was not sensing appropriately, suggesting a migration of the lead. A computed tomography (CT) scan of the chest confirmed a perforation of the right ventricular AICD lead into the pericardium (Figure 3). She returned to the operating room where a new right ventricular pacing lead was successfully implanted.

Discussion

The implantation of transvenous endocardial leads has become increasingly commonplace in the context of both permanent pacemakers (PPM) and implantable cardioverter defibrillators (ICD). Despite modern techniques and increased familiarity with PPM/ICD insertion, complications of implantation are not uncommon.¹ Case series and reviews have suggested that the incidence of complications associated with PPM implantation ranges between 3 and 9%, and is roughly twice that rate with ICDs.^{2,3} Complications can be divided into those associated with the implant procedure itself, those related to leads and venous access, and problems associated with device function.¹ Early (<2 weeks postimplantation) and late complications occur with equal frequency.³

Myocardial perforation by transvenous leads is a rare but well-recognized complication of PPM-ICD insertion.¹ With the use of thinner, more flexible pacing leads, the rate of acute PPM-related lead perforations has fallen to <1%. ICD leads have remained relatively thick and stiff to allow for the delivery of high-energy currents.^{2,3} As such, although the incidence of ICD-related lead perforation was initially quoted at <1%, this rate may be as high as 3–5% due to the aforementioned lead characteristics.^{4,5}

The clinical presentation of transvenous lead perforation is variable. While the majority of cases present within 2 weeks, this can occur as late as 16–23 months postimplantation.^{3,5–10}

The most common clinical presentation of

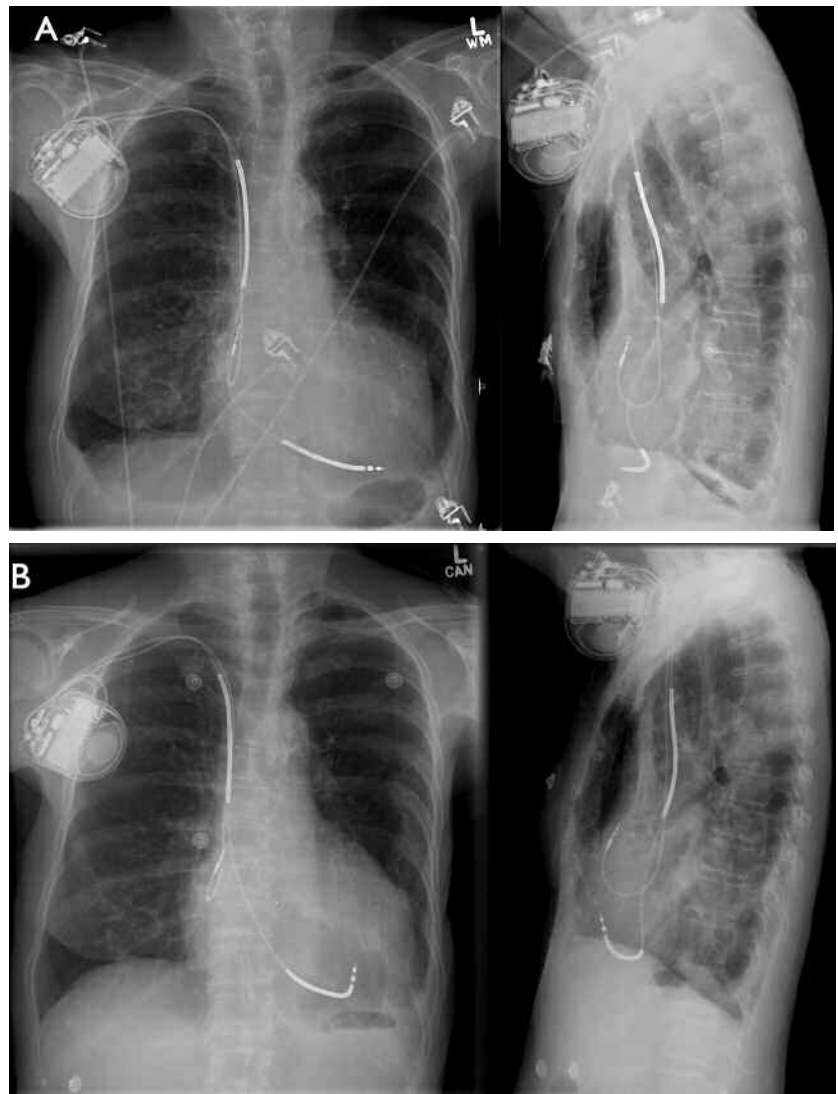


Figure 2. Chest radiograph demonstrating ventricular lead position at time of device implantation (A) and post-migration at time of presentation with tamponade (B).

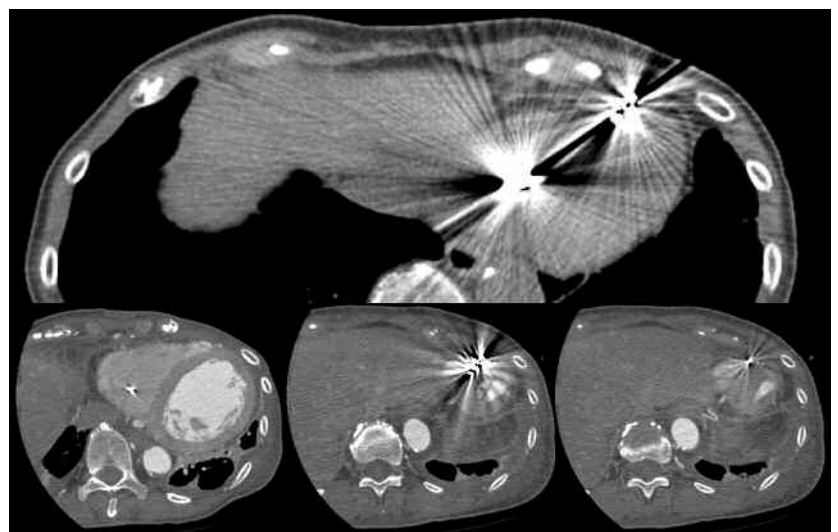


Figure 3. Computed tomography scan of the chest demonstrating perforation of the right ventricular lead through the right ventricular myocardium into the pericardial space.

myocardial lead perforation is that of increasing shortness of breath or pleuritic chest pain suggestive of pericarditis. The reported incidence is 0.3–2%.^{4,7,9} There appears to be a consistent association of pericarditis with active lead fixation in the right atrium but not with passive fixation in the atrium or active fixation in the right ventricle.⁷ Other less common presentations include new-onset atrial fibrillation, hemoptysis following migration of the lead into lung parenchyma, or sudden cardiac death.^{8–10} Likewise, symptomatic pericardial effusions resulting from lead perforation are reported to occur at an incidence of 1%.¹¹ Despite the presence of pericardial pathology, a number of patients remain asymptomatic despite lead perforation and are only diagnosed when increased sensing or pacing thresholds are noted on device interrogation.¹

In most individuals, pericarditis or pericardial effusion post-lead implantation appears to have a self-limited and benign course.⁷ A few individuals progress to cardiac tamponade and shock. In one centre, up to 5.4% of all cases of cardiac tamponade requiring intervention was associated with PPM implantation.⁶ Possible predictors of those who may develop symptomatic pericardial effusions post-PPM have been derived from analysis of the Mayo Clinic database and include the use of temporary transvenous pacemaker leads (HR 2.7), the use of helical screws for active lead fixation (HR 2.5), and recent steroid use within the past 7 days (HR 3.2). Weaker predictors include advanced age, a low body mass index, and prolonged use of fluoroscopy during lead implantation.¹¹

Pericarditic or pleuritic chest pain following lead implantation should alert you to the diagnosis. Other supportive findings may include a pericardial friction rub, an alteration in the pattern of ventricular activation by the pacemaker, loss of capture, or frank pacing of the diaphragm.¹ Various imaging modalities can be used to confirm this diagnosis. On chest radiographs, a separation of the lead tip of <3 mm from the radiolucent stripe of the epicardial fat pad (the “epicardial fat-pad sign”) is suggestive of lead perforation.¹² Echocardiography can be used to detect pericardial fluid and right atrial collapse. In addition, the location of the lead tip may sometimes be visualized by echocardiography, less commonly by CT.^{1,9,10}

Management of cases of lead perforation undoubtedly depends on the clinical presentation and, more specifically, the presence or absence of cardiac tamponade and hemodynamic compromise. In cases with mild symptoms, minimal effusion, and no clear evidence of perforation, observation may be appropriate. Small effusions should be followed with serial echocardiograms.¹ As noted above, uncomplicated cases of pericarditis generally have a benign self-limited course but have been managed in some case series with nonsteroidal anti-inflammatory drugs or steroids.⁷ It is unclear whether the clinical improvement noted in these cases occurred due to the therapy itself or merely as a consequence of the natural history of the pericarditis.

Patients with cardiac tamponade require urgent pericardiocentesis with the placement of a pericardial drain, followed by the surgical removal of the lead.¹

Summary

Lead perforation is a rare but potentially fatal complication of PPM/ICD implantation. It should be suspected in individuals presenting with pleuritic or pericarditic chest pain who have had a device recently implanted. Clinical findings of pericarditis or deterioration in the quality of pacing are supportive, as are radiological signs of pericardial effusion or frank lead perforation. It is imperative that a diagnosis of lead perforation not be delayed as urgent pericardiocentesis may be required for those with cardiac tamponade. For those with uncomplicated cases of pericarditis, observation and treatment with anti-inflammatory agents may suffice.

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