Delayed pacemaker lead perforations: Why unusual presentations should prompt an early multidisciplinary team approach

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ABSTRACT

Our first case is an 84-year-old female diagnosed with sick sinus syndrome. She underwent implantation of dual chamber permanent pacemaker without complications. On the 8th day status-postimplantation, she returned to the emergency department (ED) with moderately severe left anterior chest pain and significant ecchymosis. She was given an initial diagnosis of shingles and discharged. Two days later, she returned to the ED with increasing chest pain, dyspnea, nausea, and vomiting. Lead migration and cardiac perforation was confirmed by chest X-ray and computed tomography (CT), respectively. She was taken to the operating room (OR) for lead repositioning, and she was discharged the next day. Our second case is a 64-year-old female with a diagnosis of 2:1 high-grade third-degree atrioventricular block. A dual chamber permanent pacemaker system was implanted without initial complication. Five days after implantation, she presented to the ED following an episode of syncope due to hypotension (67/46), shortness of breath, left flank pain, and fatigue. The initial diagnosis was sepsis. A chest CT was obtained, noting lead perforation and hemothorax. The patient was taken to the OR for lead repositioning.

Key Words: Complications, lead perforation, misdiagnosis, pacemaker, skin lesion

INTRODUCTION

Cardiac pacemakers have developed as an increasingly utilized therapy since the use of the first implantable device in 1958 at the Karolinska Institute in Solna, Sweden. Over 3 million patients have implantable pacemakers worldwide, and approximately 600,000 devices are placed annually. Cardiac perforation is a rare and widely-recognized complication occurring most often during placement but occasionally delayed perforations can occur. General complications from device insertion range from 3% to 7%; lead perforation has a relatively rare complication rate of between 0.1%–0.8% for pacemaker leads and 0.6%–5.2% for implantable defibrillator leads. Delays in recognizing perforations can cause problematic and potentially fatal pericardial or pleural effusions. Retained blood, in the form of a hemothorax, can lead to infection, chest pain, or trapped lung. Predictors of postimplantation complications include: pericardial effusion, which serves as a marker of perforation, include female gender, concomitant use of another transvenous device, steroid use within 7 days, active fixation, and advanced age (>80). We present two cases of pacemaker implantation complicated by lead perforation. In both cases, the implantation procedure was performed within 10 days of readmission, and yet, each patient underwent an initial workup for a suspected medical problem unrelated to their procedure. Increased recognition of the nature of
these events is needed to prevent additional complications from a delay in diagnosis, a misdiagnosis, unnecessary diagnostic testing, and potentially invasive or harmful procedures.

CASE REPORTS

Case 1
Our first case is an obese 84-year-old female with a history of paroxysmal atrial fibrillation, left bundle branch block, deep vein thrombosis, Type II diabetes mellitus, hyperlipidemia, and hypertension who presented to our emergency department (ED) with dyspnea, dizziness, fatigue, and in sinus arrest with a junctional bradycardia (36 BPM). The patient was diagnosed with sick sinus syndrome with paroxysmal atrial fibrillation. She underwent implantation of a dual chamber permanent pacemaker (Aventio, Boston-Scientific, Marlborough, MA, USA) with right atrial (Fineline II, Boston-Scientific) and right ventricular (RV) (Dextrus, Boston-Scientific) leads without complications and discharged the following day in stable conditions with a device clinic follow-up scheduled in 2 weeks. A routine chest X-ray confirmed acceptable right atrial and RV lead placement with no initial complications [Figure 1].

On the 8th day status-postimplantation, she returned to the ED with moderately severe left anterior chest pain adjacent to her pacemaker battery pocket with radiation around and underneath her left breast. On physical examination, there is significant tenderness under the left breast, which is reproducible upon palpation. Despite the absence of skin lesions with vesicles, she was given an initial diagnosis of shingles. Routine laboratory studies, including coagulation parameters, chemistries, and complete blood count were all within normal ranges. Chest X-ray and rib films were interpreted as negative for acute cardiopulmonary process or rib fracture, leading to a general diagnosis of atypical chest pain, likely secondary to shingles and musculoskeletal etiology. The patient was discharged from the emergency room with plans for previously scheduled follow-up in device clinic in 1 week.

Two days later, she returned to the ED agitated with increasing chest pain, dyspnea, nausea, and vomiting. Nontender ecchymosis was noted over the area of the implanted pacemaker pulse generator on the left upper chest wall. No incisional erythema, purulent discharge, or other signs of infection were noted. Atypical tenderness to palpation and bruising were also noted in the left mid-axillary region [Figure 2]. A chest X-ray was obtained which demonstrated lateral lead migration. No obvious widening of the cardiac silhouette or pleural effusion was appreciated [Figure 3]. The electrophysiology and cardiac surgery teams were consulted. Device interrogation revealed RV lead malfunction consistent with lead perforation. A computed tomography (CT) scan confirmed cardiac perforation with lead erosion
into the 6th rib [Figure 4]. She was taken to the OR, the pacemaker pocket was opened, and under combined fluoroscopic and transesophageal imaging, the lead was withdrawn into the right ventricle with gentle manual traction. Hemodynamics remained stable after lead removal and imaging confirmed no evidence of pleural or pericardial effusion. The lead was then reimplemented on the RV septum. The following day, a repeat transthoracic echocardiogram (echo) was performed that showed no pericardial effusion and she was discharged.

Case 2
Our second case is 64-year-old, active and otherwise healthy, female who presented with a several month history of intermittent fatigue and shortness of breath with exercise. Previous stress testing and a 14-day event monitor demonstrated 2:1 high-grade third-degree atrioventricular (AV) block with increased sinus rates. A dual chamber permanent pacemaker system (device: AVENTIO DR, right atrial lead: Dectrus, RV lead: Dextrus, all Boston Scientific) was implanted without initial complication.

Five days after implantation, she presented to the ED following an episode of syncope. The patient reported feeling well until the day of presentation when she developed progressive weakness and lightheadedness. While ambulating to the bathroom at home she had a several second loss of consciousness prompting presentation to the ED. Initially, she was noted to be hypotensive (67/46), and complained of shortness of breath, left flank pain, and fatigue. The initial presumptive diagnosis was sepsis from a possible intra-abdominal event and/or pneumonia. Chest X-ray demonstrated a large left-sided pleural effusion. Treatment with intravenous fluid and broad-spectrum antibiotics was initiated. An abdominal CT scan was performed to further evaluate flank pain and noted no abdominal pathology. However, uppercuts confirmed a large left-sided pleural effusion and the electrophysiology team was consulted. Review of telemetry over the past 6 hours in the ED demonstrated progressive bradycardia with intermittent failure to capture and a 2:1 AV block. Device interrogation demonstrated poor RV lead sensing, intermittent failure to capture at high output, and high lead impedance. A chest CT was obtained, which confirmed RV lead perforation. The cardiac surgery teams were consulted for the management. The patient was immediately taken to the OR for lead removal under general anesthesia, continuous transesophageal echo monitoring, and fluoroscopic guidance. The pacemaker pocket was opened, and the RV pacemaker lead was withdrawn into the right ventricle without hemodynamic compromise or development of pericardial effusion. The lead was then repositioned and actively fixated on the ventricular septum. A chest tube was placed to drain her hemothorax. After several uneventful days, the chest tube was removed, and she was discharged home.

Two days later, she returned with reports of low-grade fever and malaise. Her white count, procalcitonin, and cultures (urine and blood) were normal. However, a repeat chest CT demonstrated re-accumulation of the left-sided hemothorax. Hematocrit was unchanged, and device function remained normal. Repeat echo showed no signs of a pericardial effusion. To treat associated symptoms and reduce risk of additional bleeding and potential future infection, she was taken to the operating room for a left-sided video-assisted thoracoscopic evaluation. A residual bloody effusion was drained and visually, there was no obvious source of bleeding. The chest was irrigated and a chest tube placed. Over the next couple of days, her cultures remained negative, her chest X-ray was clear, her symptoms improved, and there was no further drainage from the tube. The tube was removed, and she was discharged home. She reported resolution of all symptoms and was noted to have normal functioning pacemaker during follow-up evaluation 2 weeks later at the device clinic.

DISCUSSION

Our cases illustrate the range of symptoms that patients can present with in the settling of a delayed pacemaker lead perforation. From the cases presented, it should be noted that lead perforation toward the thoracic wall can present with varying symptoms: focal chest pain, syncope secondary to hypotension, shortness of breath from a hemothorax, or reactive pleural effusion, and ecchymosis. In addition, subtle and potentially underappreciated cardiac rhythm changes might further suggest a problem with a recently implanted device. The first case demonstrates lower left chest pain and
ecchymosis that was initially diagnosed as probable musculoskeletal pain. The second case presented with symptoms that initially prompted an evaluation for an intra-abdominal source of sepsis. Early involvement of the appropriate team is crucial to limit delayed or incorrect diagnosis and to correctly focus the case workup and management.

When a patient presents poststatus implantation of pacemaker with chest discomfort, fatigue, fever, dyspnea, pain, or syncope, the differential diagnosis should include lead malfunction as a likely cause with the highest suspicion reserved for elderly female patients (>80), who are at the highest risk for perforation. Acute lead perforations are those diagnosed <1 month after implantation and occur more often than delayed lead perforations in literature. One of the distinguishing features of delayed lead perforation, when compared to acute lead perforation, is the decrease or absence of cardiac tamponade or death. Cardiac perforations are rare complications that occur within the first 3 months and account for significant comorbid complications and in-hospital mortality risk in 0.8-1% of implantations. In a study of 2200 implanted patients by Cano et al., acute and delayed perforations account for 76% and 24% of all perforations, respectively.

Since patients are typically discharged the day following the implant procedure, discharge instructions should include a thorough review of concerning symptoms and EP-device clinic follow-up. Increase awareness and proper follow-up care are critical to ensure an early response to potential early lead-related problems. Such evaluations should preferably involve health-care providers who are familiar with the nuances of cardiac device system malfunctions or postimplant complications. Whenever there is an index of suspicion, a CT scan will have greater specificity and sensitivity defining lead positioning and potential perforation, while a chest X-ray might only demonstrate lead migration when compared to immediate postimplant imaging. Lead positioning is an important factor to consider, with serious potential complications. Apical positioning is associated with a higher risk of perforation and should be avoided especially in high-risk patients. RV lead positioning on the ventricular septum reduces risk of perforation; however, sensing is often lower and lead stability inconsistent, which could result in lead dislodgement. Careful selection of lead positioning is patient specific with the ideal location along the lower septum for most patients.

Many cases of lead perforation have been reported, but none presented with signs and symptoms that prompted an initial workup unrelated to their recent device implant. With the growing use of cardiac pacemakers and lead systems, a greater awareness of these delayed complications is critical for appropriate patient care. The intent of this report is to emphasize the importance of a high index of suspicion with any subacute presentation following device implantation to allow prompt, appropriate diagnostic testing, which can best diagnose potential device complications. Delayed presentation without timely testing and treatment, especially in the elderly, could result in increased morbidity and mortality.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

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