COMPLEX CASE STUDY

Dislodgement of an Active-Fixation Right Atrial Lead Causing Subacute Right Ventricular Perforation and Pericardial Effusion

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ABSTRACT. The management of dislodged cardiac pacing leads typically involves reoperation to reposition the lead. Yet, several recent studies have identified reoperation as a major predictor of device-related infection. Knowing this, some physicians elect not to reposition dislodged atrial leads. However, the presence of a freely mobile pacing lead with a deployed active-fixation helix within the heart raises concerns about possible cardiac injury. Although subacute perforation of active-fixation pacing leads is a well-described clinical entity, we believe this to be the first report of a dislodged active-fixation atrial lead causing perforation of the right ventricular free wall. This case illustrates an uncommon but potential hazard of failing to reposition a dislodged active-fixation lead. The case also illustrates the clinical presentation of this complication, the utility of computed tomography scanning in its diagnosis, and strategies for dealing with the complication.

KEYWORDS. pacing, complication, CT scanning, tamponade, perforation.

Case summary

A 60-year-old man with dilated cardiomyopathy experienced monomorphic ventricular tachycardia and received a left-sided dual-chamber implantable cardioverter-defibrillator (ICD) in 2004. In 2010, progressive increase in his right ventricular pacing thresholds was noted, and since his ICD was also nearing the elective replacement indicator (ERI), it was decided to implant a new right ventricular ICD lead at the time of his ICD generator replacement. However, at the time of surgery, it was discovered that his innominate vein was occluded and thus a right-sided dual-chamber ICD (Current 2207-36, St. Jude Medical, St. Paul, MN) was implanted. Active-fixation leads were used, and the lead tips of the right atrial lead (St. Jude Medical 7120) and right ventricular lead (St. Jude Medical 1688TC) were placed in the right atrial appendage and right ventricular apex, respectively. His chronic left-sided right atrial and right ventricular leads were capped, and the old device was removed. The patient was discharged home the following day and restarted on his chronic oral anticoagulation.

At the 2-week post-implant visit, the patient was asymptomatic and his wound was healing well. However, there was no atrial sensing and no atrial capture at maximum device output. A chest X-ray confirmed that the right atrial lead had dislodged (Figure 1a). The right ventricular sensing and pacing thresholds were unchanged from implant, and the patient had not experienced any episodes of ventricular tachycardia or fibrillation. His device was reprogrammed to VVI, and a decision was taken not to reposition the atrial lead because of concerns about the higher risk of device-related infection associated with repeat procedures.1,2

The patient presented 1 month later with a 2-week history of progressive leg edema and increasing dyspnea. He had an elevated jugular venous pressure but a pulse of 90 bpm and blood pressure of 150/92 mmHg. A chest X-ray demonstrated massive cardiomegaly with left lower lobe lung collapse. It was noticed that the tip of the right atrial lead had migrated towards the apex compared with a previous chest X-ray (Figure 1b). A transthoracic echocardiogram confirmed a large pericardial effusion. A computed tomography (CT) scan of the heart confirmed that the tip of the atrial lead had indeed perforated the right ventricular wall (Figure 2). The patient’s bloodwork was notable for an international


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normalized ratio of 6.5 and acute renal insufficiency with a serum creatinine of 239 μmol/l. Serum hemoglobin had decreased from a preoperative value of 153 g/l to 109 g/l. Urgent pericardiocentesis was performed, and 3.2 l of blood-stained fluid was aspirated from the pericardial space and a pig-tail catheter was left in place. The patient subsequently had his atrial lead removed with gentle manual traction, with the pericardial drain still in place and without the need for surgical intervention. The pig-tail catheter was removed, and the patient has done well with no subsequent reaccumulation of pericardial fluid at a 3-month follow-up echocardiogram. Pericardial fluid cultures and cytology were negative for Mycobacterium tuberculosis and malignancy.

**Discussion**

This case highlights several important issues. First, although delayed cardiac perforation is a well-described but uncommon complication of both atrial and ventricular leads, this is the first reported case of a dislodged atrial lead causing cardiac perforation of the right ventricle and development of a pericardial effusion. In this case, we are convinced that the right atrial lead was responsible for the perforation, as the newly implanted right ventricular lead remained stable in its position and continued to have excellent pacing and sensing parameters. Although it is possible that a perforation of the right atrial appendage may have developed at the time of atrial lead dislodgement, we believe that the right ventricle was the site of perforation, as the CT scan clearly demonstrated the atrial lead in the pericardial space. This case highlights the possibility that a dislodged active-fixation lead, with a deployed helix, is able to generate enough force to perforate the heart. Clinicians should be aware of this potential complication when weighing the risks and benefits of repositioning a dislodged atrial lead, particularly if the lead is abutting an atrial or ventricular wall. As previously described, the risk of perforation may also be higher with smaller diameter leads, as in this case.

Second, this case highlights the most common presentation of a delayed cardiac perforation, namely increasing dyspnea with signs of heart failure as a result of an enlarging pericardial effusion. The fact that over 3 l of pericardial fluid had accumulated in this case highlights how slow and prolonged bleeding may be in this setting. Although delayed perforation is rare compared with acute cardiac perforation, clinicians need to have a high index of suspicion of delayed perforation in a patient who develops pleuritic chest pain and/or signs of heart failure weeks to months after the initial implant.

Third, this case also demonstrates the utility of CT imaging in confirming lead perforation. CT imaging is particularly useful in patients with multiple pacing leads, to define which lead has caused the perforation so that revision of the appropriate lead can be performed.

**Figure 1:** Position of intracardiac pacing leads 2 weeks post implant (a) and at the time of presentation with a large pericardial effusion 1 month later (b).

**Figure 2:** Computerized tomography scan of thorax showing perforation of the right ventricle by the right atrial lead (large arrow) and large pericardial effusion.
Finally, this case demonstrates that leads causing cardiac perforation can be safely removed under fluoroscopic guidance without the need for surgical intervention. However, such cases should be performed with echocardiography and surgical backup immediately available and preferably with the pericardial drain still in place.

References


