An unusual case of epicardial lead migration presenting with hemoptysis

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Introduction
Permanent epicardial pacer lead placement via surgical incisions is generally reserved for patients with difficult transvenous access. As such, permanent epicardial leads may be left in place during cardiac surgery and tunneled into the abdominal or chest wall soft tissue in anticipation of permanent postoperative pacing requirement. We present a case of a patient presenting with hemoptysis and empyema, associated with chest wall abscess from migrating epicardial pacemaker leads.

Case report
A 76-year-old man underwent aortic valve replacement with a pericardial bioprosthetic valve through a median sternotomy in February 2015, at an outside institution, for symptomatic aortic insufficiency with left ventricular (LV) dysfunction. Preoperatively, the patient was noted to have 12-lead electrocardiogram evidence of left bundle branch block exceeding 150 ms. Given the degree of his preoperative cardiomyopathy and the possible risk of postoperative heart block in the setting of recognized conduction system disease, a decision was made to place epicardial LV leads at the time of the index procedure to facilitate cardiac resynchronization therapy (CRT), if eventually indicated. To improve the likelihood of maintaining durable pacing parameters, 2 permanent epicardial active-fixation leads (Guidant 4046 and 4047; Boston Scientific, Marlborough, MA) were placed on the LV epicardium and tunneled to the left chest wall infraclavicular space and placed in the soft tissue.

After surgical recovery and upward titration of goal-directed medical therapy, in June 2015, his LV ejection fraction remained severely depressed (≤30%), there was chronic systolic heart failure with New York Heart Association class II symptoms of congestive heart failure, and he remained with a left bundle branch block, QRS duration >150 ms. He underwent implantation of a CRT defibrillator system, with placement of the generator in the standard left pectoral position, anterior to the prepectoral fascia. Transvenous right atrial (RA) and right ventricular (RV) leads were placed via the axillary vein via direct puncture. The previously placed epicardial lead was retrieved via a tunnel made from the inferior aspect of the newly constructed pocket. The newly placed RA and RV leads and 1 of the previously placed LV leads were serially connected to the CRT implantable cardioverter-defibrillator (ICD) generator. The previously implanted LV epicardial lead was utilized for LV pacing. He had a small pocket hematoma post procedure, which ultimately resolved with conservative management.

KEY TEACHING POINTS
- In patients with a history of epicardial lead placement, there should be a high index of suspicion for infection, when patients present with atypical symptoms of systemic illness, including reports of pulmonary complaints of cough and hemoptysis.
- When patients have a history of epicardial lead implantation, suspected device-associated infection requires the cooperation of a multidisciplinary team of cardiac electrophysiologists, imaging specialists, infectious disease specialists, and cardiothoracic surgeons. Attempts at partial extraction may result in inadequate “source control.”
- When complete system extraction is infeasible or contraindicated owing to inability to remove epicardial leads, the free ends should be “capped” in an atraumatic manner and, ideally, tethered, to prevent migration.
In May 2017 he presented with a small amount of pain, swelling, and erythema at the site of his ICD suspected to be secondary to physical trauma to the site. There were no constitutional signs of illness. The patient was prescribed cephalexin for a course of 7 days. There was essentially complete resolution of surface cellulitis. One month later, however, the patient was noted to have near erosion of the ICD generator. The patient was brought to the operating room for system extraction. The RV ICD and RA leads were extracted transvenously and the 2 epicardial leads were cut flush at the left anterior chest wall in situ, and allowed to retract into the chest wall and pleural cavity. The tips of the RA and RV leads, as well as the generator from the pocket, grew coagulase-negative *Staphylococcus aureus* cultures. After a 7-day course of vancomycin, with negative blood cultures and absence of constitutional symptoms, the patient ultimately underwent right-sided, prepectoral, transvenous CRT defibrillator implantation.

Eighteen months later, the patient presented with subacute-onset cough, purulent sputum production, and intermittent hemoptysis. Physical examination was notable for left chest wall swelling and pain with palpation. Chest radiography demonstrated migration of a cut and retracted epicardial lead from the chest wall to the pleural cavity, which was different from his prior chest radiograph (Figure 1). Contrasted computed tomography scan demonstrated migration of this lead to the level of the chest wall intercostal space, surrounded by an inflammatory mass suspicious for hematoma or an abscess (Figure 2). The second epicardial lead had migrated and retracted back into the pleural cavity directly into the left anterior lung parenchyma, with associated inflammation and consolidation (Figure 2). The patient was started on amoxicillin/clavulanic acid for a pneumonic process, and bronchoscopy was performed, which demonstrated purulent discharge from the left lobe, with no blood present. Given his recurrent hemoptysis and pneumonia caused by erosion from the migrating epicardial lead tip, the patient was taken to the operating room for removal of the epicardial leads via a left thoracotomy.

A left anterior thoracotomy incision was made, encountering a 5 × 5-cm collection present posterior to the pectoralis major muscle, which was evacuated and washed out. The intercostal space was next entered, and a loculated empyema was encountered with a thick inflammatory rind. After the lung was decorticated and empyema was washed out, the tip of the epicardial lead was visualized in the fourth intercostal space, in between the ribs. The inner coil was exposed from within the external insulation of the lead. This lead was then dissected and followed until it met the second epicardial lead. The second epicardial lead was then dissected through the inflammatory rind and noted to enter directly the lung parenchyma, in essence creating a bronchopleural fistula (Figure 3). The lead was then removed from the lung parenchyma, and it was noted that once again the inner coil had been exposed from within the external insulation of the lead. The 2 leads were then dissected further towards the heart, but given the extensive scarring along the epicardial surface, only 1 of the 2 leads was able to be fully

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**Figure 1**  Posteroanterior and lateral chest radiographs depicting the initial position of the epicardial leads after the transvenous lead extraction (A, B), compared to upon presentation after migration of the leads (C, D). One of the migrated epicardial leads can be seen traversing in a backward trajectory from the chest wall into the lung (D, arrow).
removed. The second lead traversed posteriorly along the left ventricle near the hilum and phrenic nerve; and so as not to cause further injury, it was cut near the epicardial surface and capped, and covered with soft tissue.

The patient ultimately recovered from his thoracotomy surgery and was discharged home. Intraoperative cultures grew *Micrococcus*. He has not had any recurrent hemoptysis or sputum production in 30 days of follow-up, remaining on long-term amoxicillin/clavulanic acid antibiotics.

**Discussion**

Permanent epicardial pacing leads can be placed in anticipation of difficult transvenous access or to avoid excessive transvalvular lead positioning. This may be accomplished via open thoracotomy or utilizing minimally invasive techniques including thoracoscopy or laparoscopy, or concomitantly during primary cardiac surgery. More frequently, temporary ventricular pacing wires are implanted in the epicardial LV surface, in patients who are thought to be at elevated risk of bradycardia after cardiac surgery.

Temporary wires are typically removed by direct traction, with gentle pressure, owing to concerns of infection and migration. However, if resistance is encountered upon pulling the temporary wires, they are often cut at the skin level and allowed to retract into the soft tissue. There have been several reports of temporary epicardial lead fragment migration into intracardiac chambers, aorta, lung, and bronchus, as well as the skin, such as along the jaw.\(^1\)–\(^4\)

Permanent epicardial lead fragments may also migrate. There are multiple suspected mechanisms of migration. With regard to the active-fixation helix, this may perforate, erode, or dislodge directly from the epicardial surface, leading to hemopericardium.\(^5\) Similarly, erosions of epicardial lead patches into adjacent structures can occur. For example, bronchopleural fistulization to the airways may result in
massive hemoptysis and pneumonia. These erosions may be attributed to inadequate anchoring of the lead or patches, whether by screws or sutures, to the epicardial surface, or by an inflammatory process directly occurring on the epicardial surface of the heart.

Migration of the actual body of the permanent pacing leads can also potentially occur. Intraperitoneal migration of permanent epicardial leads and the generator has been reported, especially if left in the preperitoneal and posterior rectus space.

This case appears to be an unusual presentation, with the body of permanent epicardial leads migrating and subsequently eroding into the lung parenchyma, causing hemoptysis and pneumonia, along with surrounding empyema and chest wall abscess. There may have been several potential mechanisms. After cutting and retraction of the lead tips, the anchoring point of the distal end of the lead, typically the generator, was removed, thus allowing the leads to freely migrate as they are affected by the natural contractile movement of the heart and ventilating lungs. Adhesions and inflammatory fibrotic changes from prior mediastinal surgeries may have placed further fixed forces on the lead, causing it to retract. In addition, disruption of the lead insulation, exposing the potentially traumatic inner metal coil, allowed the lead to penetrate the surrounding lung parenchyma more easily, and may have served as a nidus for localized infection.

Although the early recognition of the clinical symptoms stemming from migrated leads can mitigate the complications and their sequelae, further preventative measures at the time of initial epicardial lead placement or intervention should be employed. Careful attention should be paid to proper placement with firm and secure anchoring points for the lead tips and connection to the generator. The leads should be carefully positioned within the mediastinal cavity so as to limit interference from the beating heart and movement of the lungs.

If initial device removal is necessary for infection, every effort should be made for total device extraction, as opposed to partial extraction, as was performed in our case when the patient initially presented with pocket erosion and thus led to the risk of lead fistulization from the cut epicardial lead. The approach for extraction can be challenging, given the inflammation and adhesions in the soft tissue and mediastinal cavity. A tailored combination approach should be employed, with transvenous extraction of intracardiac leads along with complete surgical removal of epicardial leads. An initial strategy would be to perform pocket exploration and free up the epicardial leads within the soft tissue and from the device generator. Next, based on the trajectory of the epicardial leads, a sternotomy can be performed if the leads are coursing more anterior, as opposed to a thoracotomy if the leads are coursing laterally within the mediastinal cavity. Minimally invasive approaches, such as partial sternotomy or thoracotomy, thoracoscopy, or subxiphoid window, can be considered as well for epicardial lead removal; however, these can be limited by the inflammatory adhesions, which may hinder optimal visualization.

Conclusions
This is an unusual case of epicardial lead migration presenting with hemoptysis. Though rare, migration of permanent epicardial leads can occur, and there should be a high index of suspicion in patients who present with atypical signs or symptoms following their procedures. The absence or disruption of anchoring points, or transection and breakdown of leads without proper capping or insulation, should elevate the level of concern. Treatment typically involves complete removal of the leads, as lead migration can be associated with significant morbidity.

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