Late Right Ventricular Perforation and Hemothorax After Transvenous Defibrillator Lead Implantation

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ABSTRACT: A 53-year-old man with ischemic cardiomyopathy underwent prophylactic transvenous implantable cardioverter-defibrillator (ICD) placement. Nine days after the procedure, he had recurrent chest pain and left pleural effusion associated with a drop in hemoglobin. Hemothorax and right ventricular (RV) lead perforation were suspected on chest radiography and lead interrogation, and confirmed by thoracentesis and contrast computed tomography (CT) scanning, respectively. The CT-scan clearly demonstrated the RV lead tip projecting beyond the cardiac border into the anterior left pleural space. The perforated lead was removed in the operating room under transesophageal echocardiography guidance and a new transvenous lead was successfully placed a month later. This case highlights: 1) the importance of suspecting late RV perforation in patients with ICD implantation presenting with recurrent chest pain and/or pleural effusion; 2) the value of CT in its diagnosis; and 3) the need for a more careful management of this potentially life threatening complication. **KEY INDEXING TERMS:** Implantable cardioverter-defibrillator; Computed tomography; Cardiac perforation; Hemothorax. **[Am J Med Sci 2007;334(3):1–1.]**

Late perforation of the right ventricular (RV) free wall following transvenous implantable cardioverter-defibrillator (ICD) lead placement is an extremely rare complication with a few cases described in literature.[1–5] We report a case of late RV perforation from ICD implant involving the left pleural space with resultant large hemothorax.

Case Report

A 53-year-old man with ischemic cardiomyopathy and previous coronary bypass grafting was referred to us for prophylactic ICD placement, according to MADIT-II criteria.[6] He weighed 125 kg and was 180 cm tall. Electrocardiographic (ECG) recording showed sinus rhythm with narrow QRS complex, normal QTc interval and T-wave inversion in the anterolateral leads. Transthoracic echocardiography and Doppler flow analysis revealed left ventricular (LV) ejection fraction of 25%, LV end-diastolic diameter of 7.1 cm, global LV hypokinesis with anteroseptal, apical and distal inferoseptal akinesis, no mechanical dyssynchrony or mitral regurgitation. Coronary angiography showed patent bypass grafts; hence, no percutaneous coronary intervention was performed.

The patient was successfully implanted with a single-chamber ICD (Model V-193 Atlas with transvenous active fixation electrode 1580 Riata, St. Jude Medical system, Sylmar California). Perioperative pacing threshold was 1.0 V/0.5 ms, R wave amplitude 12 mV, defibrillation electrode impedance 720 Ohm, and defibrillation threshold 20 J. Twenty-four-hour postprocedural chest radiography showed no evidence of pneumothorax, pericardial or pleural effusion with the lead tip in the RV (Figure 1A). The lead parameters remained stable at the time of discharge. The patient was discharged with his prehospitalization medications including, carvedilol, digoxin, furosemide, and eplerenone. F1

Nine days after implantation, the patient presented to the emergency department complaining of a 1-day history of sudden onset left lateral chest pain, sharp and constant in nature, worsening on deep inspiration. There were no associated symptoms. Initial physical examination was unremarkable except for mild tenderness along the left lateral chest wall. There were no significant ECG changes as well as no change in the position of the RV lead on chest radiography. Over the next 48 hours, laboratory values, serial cardiac enzymes, telemetry and chest radiographs showed no significant abnormalities. The patient was diagnosed with persistent musculoskeletal pain and discharged with anti-inflammatory/analgesic drugs. The following day, he returned to the emergency department complaining of worsening chest pain, now associated with diaphoresis. Initial vital signs were stable. On physical examination, poor inspiratory effort secondary to the chest pain was noted. Serial cardiac enzymes were negative and the ECG remained unchanged. The chest x-ray film demonstrated silhouetting of the left hemidiaphragm and opacification of the retrocardiac and left lower lung fields with the ICD and its lead in place. Laboratory results revealed an elevated white count of 15.8 × 109/L. These findings were attributed to pneumonia and treatment was initiated with oral levofloxacin. Overnight, although the patient's chest pain improved, he became febrile with productive cough of yellow green sputum. On hospital day 4, the patient's hemoglobin fell from 11.9 g/dL on

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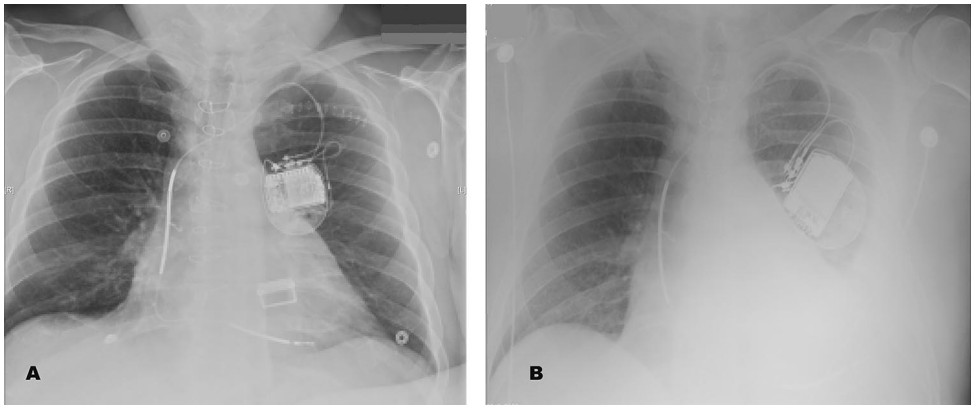

Figure 1. A, Radiograph showed development of a moderate-sized left pleural effusion, postprocedural chest radiograph showed no evidence of pneumothorax, pericardial or pleural effusion with the lead tip in the right ventricle; B, radiograph showed development of a moderate-sized left pleural effusion position with no clear evidence of right ventricular perforation.

admission to 7.6 g/dL, which stabilized at 10.9 g/dL after transfusion of 3 units of packed red blood cells. A repeat chest radiography showed development of a moderate sized left pleural effusion, which layered on lateral decubitus position (Figure 1B). Hemothorax and RV lead perforation were suspected at this time. Indeed, on diagnostic thoracentesis 60 mL of grossly bloody (hematocrit = 27%) pleural fluid was obtained and lead interrogation via the device showed poor sensing (R-wave amplitude 2 mV) and pacing threshold (6.0 V/0.5 ms). A subsequent thoracic computed tomography (CT) scanning with contrast showed left-sided pleural effusion and clearly revealed the ICD lead tip projecting beyond the cardiac border into the anterior left pleural space (Figure 2). The lead was successfully explanted in the operating room under transesophageal echocardiographic guidance. A chest tube placed for drainage of the hemothorax drained 800 mL of bloody fluid. A new ICD lead was transvenously placed uneventfully 1 month later. The patient remains asymptomatic at 9 months, with optimal ICD and lead electronic parameters.

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Discussion

Late penetration into the myocardium and/or perforation of the RV wall by ICD leads is a rare complication. It can be fatal if it is slow to be recognized. In published reports, the incidence of delayed RV perforation from transvenous ICD placement has varied from 0.66% (1/150 patients)[1] to 1.28% (1/78 patients).[2] Delayed ICD lead perforations have been documented to occur from as early as 8 days to 23 months after implantation.[2,3,7] Ellenbogen et al[8] reported a case of hemothorax from lung penetration a month after transvenous implantation of a single coil ICD. We report the second case of hemothorax from late RV transvenous ICD lead perforation. Although we cannot definitively establish the exact time of lead perforation it is unlikely that it occurred soon after the procedure because lead parameters remained stable one day after implant.

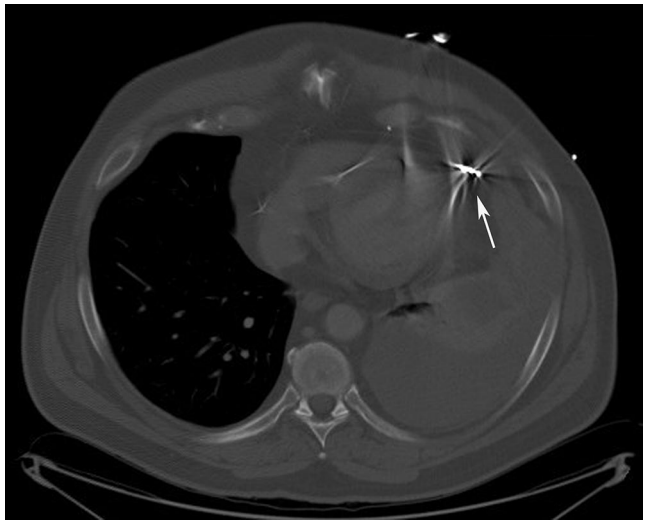

Figure 2. Contrast computed tomography scan showing the implantable cardioverter-defibrillator electrode tip (arrow) projecting beyond the wall of the right ventricle into the left pleural space all the way through the lung parenchyma. Also noted is a large pleural effusion with compressive atelectasis of the left lung.

The thickness of a normal RV wall averages 4 to 5 mm. The traditional location for placement of a ventricular electrode placement has been the RV apex for reasons of stability. But the risk of perforation is almost none when the lead is screwed onto the thicker ventricular septum. Often, ICD leads are placed in patients with dilated cardiomyopathy. In these situations myocardial walls may be thinner than anticipated due to infarction, fibrosis, and ventricular dysplasia increasing the risk of penetration by active fixation leads.[4]

In prior published series evaluating thicker and heavier ICD leads, the incidence of cardiac perforation was greater in comparison to pacemaker leads.[2] With the advent of modern leads, the incidence of ICD lead perforation is comparable to that of pacemaker leads.[3] In transvenous ICD leads, the risk for perforation may be increased with the use of active fixation,[9] overscrewing of leads or placing coils of standard leads into the ventricles of smaller built patients.[3]

Most commonly, lead perforation presents with pleuritic chest pain or with friction rubs from pericarditis.[1–3] At the onset of chest pain, small pericardial effusions are usually detected.[4] As opposed to acute lead perforation, development of large effusions causing cardiac tamponade and requiring intervention is a rare complication with delayed perforations.[3,7] Probable mechanisms causing chest

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pain include constant pericardial irritation causing intermittent leakage or constant mechanical pressure from the screw leading to abrupt late myocardial perforation.[8] Radiographically, separation of the lead tip from the radiolucent epicardial fat by less than 3 mm ("fat pad sign") or extension of the transvenous lead beyond the pericardial outline can indicate lead penetration or perforation.[3] The diagnosis of perforation can be facilitated by device interrogation, echocardiography and fluoroscopy.[3,5] However, in a recent case series of three patients all three modalities were suggestive but not conclusive of lead perforation and CT scan resulted in definitive diagnosis.[5] In addition, Ellenbogen et al[8] reported a case of hemothorax and lung penetration a month after implantation of a single coil transvenous ICD. The patient presented with hemoptysis, dyspnea, recurrent chest pain and hypotension. As in our patient, the diagnosis was confirmed only with a CT scan of the thorax.

Clinical Management

Vlay et al[4] stated that in patients that undergo early resolution of the clinical presentation, no treatment is warranted, especially if their sensing and pacing parameters are acceptable. Ellenbogen et al reported 2 patients with symptomatic large pericardial effusions, whose symptoms resolved after therapeutic pericardiocentesis. Therefore lead repositioning or removal was not performed. Molina et al[23] described the case of a 57-year-old man who had post-implantation pericardial effusion and underwent lead repositioning. Since he was clinically stable, pericardiocentesis was not performed, but the effusion was monitored with serial transthoracic echocardiograms. Lead repositioning has also been used to treat acute, severe, and chronic recurrent pericarditis with or without effusions, with good resolution of symptoms.[3,8] In patients that are hemodynamically unstable, perforated leads can be explanted under transesophageal echocardiographic guidance with surgical backup in the operating room.[7] Lung perforation, uncontrollable bleeding, and chronic pericarditis may necessitate lead explantation and/or repositioning.[4] Oginosawa[10] described the case of a 26-year-old man who presented with a right pneumothorax after atrial screw-in ICD lead implantation; the patient was treated conservatively with gradual resolution of the pneumothorax. Firm adhesion of a lead to the ventricular wall may even require surgical removal.[3] In another case of hemothorax and lung perforation with hemodynamic instability, surgical withdrawal and repositioning of the lead into the RV outflow tract was performed.[8] Since our patient developed a large hemothorax from ventricular perforation and was at risk for cardiovascular collapse, he underwent emergent lead extraction in the operating room under transesophageal echocardiography guidance.

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Conclusions

Physicians must be aware of the probability of cardiac perforation when an active fixation electrode is used, especially if the RV is thinned by previous infarction or cardiomyopathy. To prevent adverse outcomes from undersensing of ventricular arrhythmias or from cardiac tamponade or hemothorax, early diagnosis and treatment of perforation with removal and replacement of nonthoracotomy defibrillator leads is essential.

Routine radiography may not be sensitive in diagnosing this potentially life-threatening complication, whereas CT scanning can be a helpful adjunct to standard radiography, providing a definitive diagnosis and visualizing the precise location and path of the perforated lead in anticipation of surgery.[8]

There is no clear consensus in the management of ICD lead perforation, depending primarily on the patient's clinical condition and expertise of the treating physician and the institution. Even though the management of this complication is not well standardized, lead repositioning/explantation should always be performed in the operating room with surgical backup under transesophageal echocardiography guidance.

References

1. **Fahy GJ, Kleman JM, Wilkoff BL, et al.** Low incidence of lead related complications associated with nonthoracotomy implantable cardioverter defibrillator systems. Pacing Clin Electrophysiol 1995;18:172–8.
2. **Molina JE.** Perforation of the right ventricle by transvenous defibrillator leads: prevention and treatment. Pacing Clin Electrophysiol 1996;19:288–92.
3. **Kautzner J, Bytesnik J.** Recurrent pericardial chest pain: a case of late right ventricular perforation after implantation of a transvenous active-fixation ICD lead. Pacing Clin Electrophysiol 2001;24:116–8.
4. **Vlay SC.** Complications of active-fixation electrodes. Pacing Clin Electrophysiol 2002;25:1153–4.
5. **Henrikson CA, Leng CT, Yuh DD, et al.** Computed tomography to assess possible cardiac lead perforation. Pacing Clin Electrophysiol 2006;29:509–11.
6. **Moss AJ, Zareba W, Hall WJ, et al.** Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877–83.
7. **Khan MN, Joseph G, Khaykin Y, et al.** Delayed lead perforation: a disturbing trend. Pacing Clin Electrophysiol 2005;28:251–3.
8. **Ellenbogen KA, Wood MA, Shepard RK.** Delayed complications following pacemaker implantation. Pacing Clin Electrophysiol 2002;25:1155–8.
9. **Shohat-Zabarski R, Kusniec J, Strasberg B.** Perforation of the right ventricular free wall by an active fixation transvenous cardioverter defibrillator lead. Pacing Clin Electrophysiol 1999;22:1118–9.
10. **Oginosawa Y, Abe H, Nakashima Y.** Right pneumothorax resulting from an endocardial screw-in atrial lead in an implantable cardioverter defibrillator system. Pacing Clin Electrophysiol 2002;25:1278–9.

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