CASE REPORT

Pericardial effusion and right-sided pneumothorax resulting from an atrial active-fixation lead

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We report on a patient in whom an active-fixation pre-shaped atrial lead caused perforation of the right atrial wall, pericardium and pleura, resulting in pericardial effusion and right-sided pneumothorax. Chest X-ray did not demonstrate protrusion of the atrial lead outside the cardiac silhouette but computed tomography visualized the tip of the helix of the atrial screw-in electrode outside the contours of the right atrial appendage touching the right upper lobe of the lung. The lead was repositioned with resolution of pericardial effusion and pneumothorax. Due to their proximity to the right lung, high anterolaterally positioned atrial screw-in leads carry a small but definite risk for right-sided pneumothorax.

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Case report

A 51-year-old man with idiopathic congestive cardiomyopathy underwent implantation of a dual chamber ICD (Guidant Ventak PRIZM DR 2 1861) because of syncope due to non-sustained ventricular tachycardia. The patient had a low body weight and height (56 kg and 168 cm, respectively). He did not have any pre-existing bronchopulmonary disease. The procedure was performed under general anaesthesia. The device was implanted in the left prepectoral area. The right ventricular (RV) bipolar screw-in silicone lead (Medtronic 6943-65) was inserted via the cephalic vein and positioned in the RV apex. The right atrial bipolar screw-in silicone pre-shaped J-lead (Medtronic 5568-53) was inserted via puncture of the left subclavian vein and positioned in the right atrial appendage. There was no puncture or attempt to puncture the right subclavian or jugular veins either before or during the implantation procedure. Atrial and ventricular sensing (3 and 9.3 mV) and pacing thresholds (0.1 ms at 3 V for both) were satisfactory. Lead impedance measurements were 580 and 570 Ω, respectively. There was no diaphragmatic stimulation either with atrial or with ventricular pacing at high output. Two therapy zones were programmed: 'ventricular fibrillation' for rates >181 beats per minute (bpm) and 'ventricular tachycardia' for rates of 160–181 bpm.

Due to high defibrillation threshold (DFT) of >24 J, an additional subcutaneous lead (Medtronic 6996-58) was tunnelled over the left lateral thorax. This resulted in DFT ≤20 J. Chest X-ray after the procedure showed an enlarged cardiac silhouette with normal positions of defibrillator and the three leads. Twenty-four hours after the procedure low molecular weight heparin (LMWH) was restarted and the patient was discharged home 1 day later.

On day 3 after the implantation the patient developed pericarditic chest pain. He was admitted to a referring hospital where clinical examination demonstrated presence of a pericardial friction rub and echocardiography visualized a pericardial effusion of 0.5–1 cm without any signs of haemodynamic compromise. Chest X-ray was...
unchanged with identical position of the leads. A conservative approach was chosen with initial stabilization of the pericardial effusion with cessation of anticoagulant therapy. On day 4, chest X-ray revealed a right-sided pneumothorax not previously present. A pleural drain was inserted with resolution of the pneumothorax over the ensuing 2 days. However, since echocardiography showed progressive increase in the amount of pericardial effusion (to maximum 3 cm without any influence on right or left ventricular filling), the patient was referred to our hospital.

Interrogation of the ICD revealed slightly decreased sensing and slightly increased pacing thresholds in the atrium (1.1 mV and 0.3 ms at 3 V). The ventricular values were 6.9 mV and 0.06 ms at 3 V. Lead impedance measurements were, respectively, 361 and 541 Ω. The morphology of the atrial and ventricular electrograms was similar to that obtained at implantation, with small far-field ventricular signals on the atrial electrogram. Chest X-ray was unchanged regarding the positions of the leads (Fig. 1). Computed tomography (CT) of the chest confirmed the presence of significant pericardial effusion and a residual anterior pneumothorax (Fig. 2). The atrial lead was visualized just above the right atrial appendage with its helix perforating the right atrial wall, pericardium and pleura, reaching the right upper lobe.

On day 14 the patient underwent reoperation. First, a surgical pericardiotomy and drainage of 700 ml serosanguinous fluid were performed, followed by placement of a pericardial drain. Subsequently, the atrial lead was repositioned. Pericardial and pleural drains could be removed 2 days postoperatively and the patient was discharged on day 23 (9 days after surgery) after a further uneventful observation. One month after the intervention the patient was seen at the outpatient clinic and was asymptomatic.

**Discussion**

Complications have been reported in up to 9% of atrial lead placements[1]. They are most often related to obtaining venous access (haemorrhage, pneumothorax: 2%), lead dislodgement (4.2%), inadequate pacing and sensing (3.5%) and acute pericarditis (5% in patients receiving active-fixation atrial leads)[2-3]. Subclavian vein puncture may result in pneumothorax at the side ipsilateral to the puncture. Myocardial perforation resulting in pericardial effusion or tamponade is rare and may require percutaneous drainage or open heart surgery.

We are aware of only four reports of atrial leads perforating both pericardium and pleura, resulting in right-sided pneumothorax (Table 1)[4-7]. In all cases the pacing or ICD system was inserted in the left prepectoral area. Two reported patients as well as our patient were of small body stature.

Trigano et al. identified the possible risk factors for cardiac perforation using an active-fixation atrial lead[8]. Overscrewing of the lead, distal stylet insertion, abrupt withdrawal of the lead with extended screw or inadvertent displacement of the atrial lead during positioning of the ventricular lead were associated with the complication. Overscrewing was unlikely in our case due to a specified number of clockwise turns under fluoroscopic control. Moreover, we used a pre-shaped J-lead, precluding too distal placement of the stylet since it needs to be retracted for positioning. The atrial lead

![Figure 1](https://academic.oup.com/europace/article-abstract/5/4/419/552134)
was not repositioned after initial placement. Atrial and ventricular sensing and pacing parameters as well as fluoroscopic positions were satisfactory. The patient, however, exhibited low body weight and height (56 kg and 168 cm) like in the cases described by Wan-Jing et al. and Oginosawa et al. [5,7].

It is of interest to note that in our patient, neither the sensing and pacing parameters nor chest X-ray could definitely point to lead perforation as the cause of his symptoms. In two of the four previously reported cases the diagnosis was made on the basis of a PA chest X-ray, showing the presence of the atrial lead or helix outside the cardiac silhouette, while, in a third, clear protrusion could be visualized in a left anterior oblique fluoroscopic view (indicating the usefulness of different radiographic angles in suspected cases). In our patient, however, only CT visualized the tip of the atrial screw-in electrode just outside the contours of the right atrial appendage and into the right upper pulmonary lobe. As there was no gross protrusion of the body of the lead outside the cardiac silhouette we conclude that the perforation must have been caused by the extendable screw of the atrial lead. Since this is the first case describing right-sided pneumothorax in the absence of clear protrusion radiographically, it is unknown what is the sensitivity of CT under these circumstances. Our finding, however, points to the additional diagnostic information that can be provided by CT. How far similar images may be found in other patients with atrial active-fixation leads (i.e. its specificity) has not been studied. Also, how far electrode artefacts may preclude correct lead tip positioning in similar cases is unknown.

The CT images also illustrate that the anterolateral right atrial wall, due to its thin aspect and its proximity to the overlying right lung, predisposes to atrial and pleural perforations (Fig. 2). Implantation in the lateral and anterolateral right atrial wall also has been reported to predispose to post-implant pericarditis. Moreover, there seems to be an association between pericarditis and the use of pre-shaped J atrial leads [2,3], although no prospective data are available on this topic. It can be speculated that implantation of a straight screw-in atrial lead and/or anteromedial fixation (i.e. between the right atrial appendage and septum) might decrease the risk of this complication.

In two of the four described cases the patients presented symptoms within hours of device implantation, while in the others symptoms emerged on day 3 (our patient), day 4 or after 1 month. Since the majority of device implantation procedures are uneventful after day 2, we believe that common practice of early discharge (the day after the implantation) remains justified, although a closer outpatient follow-up could be advocated in patients with small body stature.

In summary, right-sided pneumothorax due to atrial lead perforation is a very rare but potentially catastrophic
Table 1. Published case reports on atrial lead perforation resulting in right-sided pneumothorax

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient, pacing/ICD indication</th>
<th>Atrial lead</th>
<th>Symptom/signs</th>
<th>Time after implantation</th>
<th>Lead/helix outside cardiac silhouette*</th>
<th>Pericardial effusion</th>
<th>Revision and repositioning of atrial lead</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irwin et al.</td>
<td>Woman, 80 years, bradycardia</td>
<td>Passive fixation, unipolar, tined (Medtronic-4511-53)</td>
<td>Hiccoughs, loss of atrial capture</td>
<td>7 months</td>
<td>CXR: yes, CCT: yes</td>
<td>No</td>
<td>Yes: atrial repair and epicardial atrial lead</td>
</tr>
<tr>
<td>Wan-Jing et al.</td>
<td>Woman, 79 years, third degree heart block</td>
<td>Screw-in, polyurethane, bipolar, pre-shaped J (CapSureFix, Medtronic 5568-53)</td>
<td>Dyspnoea</td>
<td>4 h</td>
<td>CXR: yes, CCT: ND</td>
<td>No</td>
<td>Yes: new tined atrial lead</td>
</tr>
<tr>
<td>Tran et al.</td>
<td>Man, 33 years, hypertrophic cardiomyopathy</td>
<td>Screw-in lead (Medtronic)</td>
<td>Left-sided pleuritic chest pain, increasing right-sided pleural effusion despite drainage</td>
<td>4 days</td>
<td>CXR: no, CCT: ND</td>
<td>Unknown</td>
<td>1 cm × 1.5 cm pericardial defect (thoracoscopy): closed with fibrin glue and surgicel No</td>
</tr>
<tr>
<td>Oginosawa et al.</td>
<td>Man, 26 years, HOCM with transient AV block and ventricular tachycardia</td>
<td>Screw-in, bipolar, silicone lead (CapSureFix, Medtronic 6940)</td>
<td>Chest pain associated with breathing</td>
<td>Immediately</td>
<td>CXR: yes, CCT: no</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Dilling-Boer et al., this study</td>
<td>Man, 51 years, dilated cardiomyopathy, ventricular tachycardia</td>
<td>Screw-in, bipolar, silicone, pre-shaped J (Medtronic 5569-53)</td>
<td>Pericarditis-like chest pain</td>
<td>3 days</td>
<td>CXR: no, CCT: yes</td>
<td>Yes</td>
<td>Yes: reposition of original screw-in atrial lead</td>
</tr>
</tbody>
</table>

*CXR, chest X-ray; CCT, chest CT; ND, not done.
complication. Extraction of the lead is not mandatory; lead revision with repositioning was feasible in all described cases and allows maintenance of dual chamber pacing and sensing. The procedure should preferably be carried out in collaboration with a cardiac surgeon and/or after prior placement of a pericardial drain, since repositioning of the lead could potentially result in rapidly increasing pericardial effusion and tamponade.

References


