Atrial lead perforation early after device implantation: A case series

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Introduction

An overall complication rate between 5.3% and 14.3% is observed for cardiac device implantations. The complication risk increases with rising number of electrodes used and is higher in the case of redo procedures. Complications can be divided by their timing into interprocedural, periprocedural (within 7 days), or late (after 7 days) and occur after access site problems using a subclavian or axillary puncture, or are related to lead placement itself owing to dislocation or perforation. Finally, infection and lead malfunction can occur.

Lead perforations in the context of permanent pacemaker or implantable cardioverter-defibrillator implantations are rare (<1%), with a broad spectrum of typical and atypical symptoms. The affected lead is more often the right ventricular lead (owing to apical or free wall perforation) than the atrial electrode and the therapy of choice is lead extraction or repositioning of the affected electrode to avoid further migration.

Atypical secondary complications include left-sided hemothorax owing to vascular access problems, or a perforation of the right ventricular lead with additional damage to lung tissue or smaller vessels within the lung, with possible lead migration into the pleural space or the lung. Right atrial (RA) perforation with damage of the right lung can result in a rare right-sided hemothorax.

The range of clinical presentation is wide: from asymptomatic (presence on heart computed tomography [CT] in 6%–15% of asymptomatic patients), to symptomatic with thoracic pain or fatigue, coughing, and dyspnea (with or without minimal pericardial effusion), to distress (with pericardial tamponade).

Since the complication spectrum is very broad, early detection and appropriate response are important to optimize management. The present case series discusses solely RA lead perforations.

Case report

Case 1: Atrial lead perforation resulting in urgent surgical repair

An 82-year-old male patient presented with symptomatic sick sinus syndrome and multiple syncope, which led to an uncomplicated implantation (35 minutes skin-to-skin time) of a dual-chamber pacemaker. The patient’s medication contained aspirin, antidiabetic therapy, and antihypertensive therapy without the need for oral anticoagulants (Table 1).

The atrial lead was fixed actively in the anterolateral right atrial appendage (RAA) and the ventricular lead in the apical right ventricle (RV) (Figure 1A). The patient was hemodynamically stable and free of any complaints during and immediately after the procedure.

Early postoperative (approximately 5 minutes after skin closure) echocardiography revealed a hemodynamically irrelevant pericardial effusion (5 mm end-diastolic). Because of that, a device check was performed subsequently after echocardiography and demonstrated varying atrial sensing values (Table 2), good pacing thresholds of both leads, and normal lead impedances (unipolar and bipolar). The patient...
continued to be closely monitored and developed a pericardial tamponade, leading to an emergency pericardiocentesis resulting in the withdrawal of 600 mL of fresh blood.

Since the bleeding did not stop by draining the pericardial effusion, the indication for thoracotomy was given. Intraoperatively it was shown that the atrial lead perforated up to 5 cm distally through the RAA (Figure 1B). After successful explantation of the RA lead the site of perforation was closed with a small patch and an epicardial atrial electrode was implanted. The patient was discharged after 7 days. At present, the patient is still alive and continues to show satisfactory pacemaker measurement values.

This case described an (acute) atrial lead perforation during dual-chamber pacemaker implantation. Changing sensing values could have suggested unstable positioning. Routine postoperative cardiac ultrasound can trigger closer or longer postoperative monitoring in patients with minimal pericardial effusion. Furthermore, the rapid management of this complication is extremely important. The acute development of a hemodynamically relevant pericardial effusion is probably the most dramatic consequence of a lead perforation.

### Case 2: Atrial lead perforation with right-sided hemothorax

An 84-year-old patient with low bleeding risk (solely low-dose aspirin and normal platelet count), was planned for dual-chamber pacemaker implantation with recurrent syncope caused by prolonged sinus arrests. The implantation was uneventful (33 minutes skin-to-skin time), with insertion of both leads via the left cephalic vein. The atrial lead was fixed actively in the lateral RA and the ventricular lead in the apical RV (Figure 2A, Table 1). The patient was asymptomatic during the procedure and intraoperative device measurements proved to be satisfactory (Table 2).

Postprocedurally, the patient suddenly complained of breath-dependent thoracic stabbing with a pain intensity of 7 on the Visual Analogue Scale (VAS). The pacemaker measurements were normal as well as the echocardiographic control immediately after implantation. The patient responded well to analgesic therapy. Regressive symptoms and continued satisfactory device measurement values (Table 2) resulted in patient discharge the following day.

Four days later, he presented to the emergency department with recurrent breath-dependent thoracic stabbing (VAS 8).

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**Table 1** Baseline and procedural data

| Case # | Antiplatelet or NOAC | Sex | BMI  | Duration (min) | X-ray time (min) | Access RA lead | Access RV lead | RA lead position | RV lead position | X-ray dose (Cgy/cm²) |
|--------|----------------------|-----|------|----------------|------------------|---------------|---------------|-----------------|-----------------|------------------|
| 1      | Aspirin 100 mg       | Male| 30.8 | 35              | 2.3              | Subclavian vein | Subclavian vein | RAA             | RV apex         | 95               |
| 2      | Aspirin 100 mg       | Male| 27.0 | 33              | 3.1              | Cephalic vein   | Cephalic vein  | RA lateral      | RV apex         | 106              |
| 3      | Aspirin 100 mg       | Female| 24.7 | 35              | 2.1              | Subclavian vein | Subclavian vein | RA lateral      | RV apex         | 43               |
| 4      | Apixaban 5 mg        | Male| 24.3 | 115             | 18               | Subclavian vein | Subclavian vein | RA lateral      | RVOT            | 909              |

BMI = body mass index; NOAC = novel oral anticoagulant; RA = right atrium; RAA = right atrial appendage; RV = right ventricle; RVOT = right ventricular outflow tract

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**Figure 1** Atrial lead perforation treated with urgent cardiac surgery. **A:** Chest radiograph immediately post implant: atrial lead is fixed into the right atrial appendage and the ventricular lead has been fixed in the right ventricle (arrows). **B:** Supine position, sternotomy. Surgical pincers holding the perforated right atrial electrode (arrow). The pericardial drain is still visible (asterisk). LV = left ventricle; RAA = right atrial appendage; RV = right ventricle.
Pericardial effusion was still absent on cardiac ultrasound and both unipolar and bipolar pacemaker measurements remained fine.

Owing to the timeline and symptom characteristics, a lead perforation was suspected and a CT of the thorax was performed. Heart CT confirmed a perforated atrial electrode (Figure 2B) with lead tip protrusion of approximately 13.7 mm from the pericardium into the right lung. The lead did not penetrating the pleura because no pneumothorax was visible. No pericardial fluid was observed. Furthermore, a right-sided hemothorax, most likely caused by vascular injury in the context of the perforation, was found. Therefore, lead revision with RA lead extraction and subsequent drainage of 700 mL of bloody effusion was performed. The patient remained hemodynamically stable periprocedurally. The thoracic drainage was removed on the second day and the patient was discharged at day 7. During follow-up, the patient remained asymptomatic and no recurrence of hemothorax occurred.

This case is illustrative because the RA lead perforated through the pericardium into the right lung, in the absence of a tamponade, resulting in an isolated right-sided hemothorax. There was neither an injury of the pleura (absence of pneumothorax) nor an injury of larger vessels close to the heart—which tend to develop left-sided hemothoraces. A clinical key fact was the atypical breath-dependent stabbing, often pathognomonic for lead perforation. This case also demonstrates the diagnostic importance of CT, as it quickly identified not only the cause of the symptoms, but also the lead involved (RA lead) and the accumulation of fluid (hemothorax) in the right lung. A CT scan should therefore be performed at a very early stage if a lead perforation is suspected or typical symptoms are described.

Case 3: Severe chest pain requiring lead repositioning

A 63-year-old female patient with a symptomatic binodal disease and normal systolic left ventricle function was assigned for elective implantation of a dual-chamber pacemaker (35 minutes skin-to-skin time, Table 1). Postprocedurally, an uneventful echocardiographic and device control was observed. Shortly before transfer back to the ward, the patient complained of severe breath-dependent left-sided pain (9 on the VAS). Radiography of the thorax immediately after implantation revealed (retrospectively) an infiltration area in the right lower lung (Figure 2C and 2D). Owing to typical respiratory complaints, lead revision of both electrodes was performed. After RA lead repositioning (RA: lateral to RAA and RV: apical to inferoseptal), the symptoms immediately disappeared and the patient remained hemodynamically stable during the entire procedure and the whole hospitalization. Device control was also inconspicuous and the patient was discharged the following day without any complaints.

The third case highlights the importance to always consider perforation if patients have postprocedural chest pain, even if all measured values are inconspicuous.
contralateral clinical or radiological pulmonary anomaly should raise suspicion for lead perforation; even though this type of complication is very rare, cases of atrial lead perforation with following right-sided pneumothorax are described in the literature. In this particular case, a CT scan would also have been very helpful, as it would have given more information regarding the lead and the damage caused (especially pulmonary damage: pneumothorax vs hemothorax).

Case 4: Atrial lead microperforation with Dressler syndrome
An 82-year-old patient was implanted with a dual-chamber pacemaker for symptomatic sick sinus syndrome. Owing to a history of paroxysmal atrial fibrillation the patient was on oral anticoagulants (apixaban 5 mg twice/day), which had been stopped 24 hours before implantation. The implantation procedure itself was difficult (115 minutes skin-to-skin time, Table 1) owing to multiple repositioning of the RV lead because of right ventricular low voltage at the entire septum. Postprocedural measurements and echocardiography were satisfactory, but the patient complained of breath-dependent thoracic stabbing immediately after implantation. On the first postoperative day, symptoms were decreasing, but the echocardiographic control showed a minimal pericardial effusion without hemodynamic relevance (Figure 3B). After 3 weeks of intermittent symptoms, a CT scan of the thorax was performed, which revealed a pericardial effusion and

![Manifestations of atrial lead perforations, right-sided hemothorax.](image)
a perforated atrial electrode tip (Figure 3C). Since the pericardial effusion was not hemodynamically relevant, the oral anticoagulation was reduced to 2 weeks of low-dose 2.5 mg apixaban (owing to low risk of thromboembolic events), which resulted in regression of the pericardial effusion. After resumption of full-dose novel oral anticoagulants, relapse of the effusion was observed, so that finally extraction of the atrial lead was performed, as well as a drainage of the stable pericardial effusion (Figure 3D). A total of 900 mL of brownish tinted transudate was drained and revealed a Dressler syndrome. The patient was free of complaints and, after 3 months of prednisolone and colchicine therapy, without recurrence of effusion.

The last case demonstrates another possible course of a lead perforation. The supposedly uncomplicated atrial lead caused a microperforation with chronic inflammation and Dressler syndrome.

**Conclusion**

All the cases presented are intended to sharpen our clinical understanding that in the case of typical complaints of the patient, early revision of the leads (ventricular and atrial) should be generously considered, as this intervention can significantly facilitate the further clinical course. RA lead placement and fixation is operator-dependent, but 3 positions are recommended: RAA, anterior RA, and lateral RA, wherein the lateral atrial wall tends to be a muscle-weak (thin) area of the RA, so that a majority of implanters prefer the RAA (also because of the easy access with pre-bent stylet). However, this case series
demonstrates that even if the RA lead is fixed in the supposedly less vulnerable RAA, pronounced complications can occur, so that appropriate caution should be exercised here as well. Furthermore, CT is a very fast, meaningful, and nowadays almost always and everywhere available diagnostic tool. Based on the cases presented, it is clear to see how accurate, fast, and sensitive this diagnosis can be performed to identify both the underlying problem and the affected pacemaker lead. Finally, every operator should be aware of almost all possible complications—even very rare ones like a right hemothorax after left-sided pacemaker implantation—based on the motto “expect the unexpected.”

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