Received: 29 December 2022 | Revised: 3 April 2023 | Accepted: 25 May 2023

DOI: 10.1002/ccr3.7525

CASE REPORT

Clinical Case Reports Open Access WILEY

Delayed perforation of an atrial pacemaker electrode: Lifelong risk for a rare but serious complication

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Key Clinical Message

Nonspecific symptoms such as pleuritic or pericardial chest pain in cardiovascular implantable electronic devices patients, even with unremarkable ECG or device parameters, should always raise suspicion of electrode perforation, regardless of how long ago the implantation was performed.

Abstract

We report the successful percutaneous management of a 77-year-old woman who had a dual-chamber pacemaker implanted more than 1 year ago and presented with pericarditis pain and compensated pericardial hemorrhagic tamponade. The symptoms were due to very late acute perforation of the atrial lead. This report is intended to raise awareness of procedure-related complications in the large group of cardiovascular implantable electronic device patients. Pleuritic or pericardial pain in these patients should raise suspicion of electrode perforation, as the risk of perforation is not restricted to the period immediately after implantation and a lifelong risk cannot apparently be excluded.

KEYWORDS

atrial lead, cardiac perforation, CIED, pacemaker, pericardial effusion, tamponade

1 | INTRODUCTION

Cardiovascular implantable electronic devices (CIED) with transvenous leads have become a central and indispensable part of modern cardiac therapy. Not least because of a constantly expanding range of indications and the now relatively simple and remarkably safe procedure, the number of CIED implantations has risen steadily in developed countries over the past few decades.[1] Among the generally low rate of procedure-associated complications, lead perforation represents one of the most dangerous. According to a large meta-analysis including over 60,000 patients, this complication is very rare, with a median prevalence of 0.4%. It occurs predominantly acutely during implantation or within the first 24 h post-procedure.[2] Symptomatic delayed lead perforations occurring within days or month after procedure are extremely rare, and only very few cases have been reported.[3] Here, we report a case in which an active-fixed atrial electrode was perforated more than a year after implantation, accompanied by pericardial chest pain and hemodynamically compensated pericardial tamponade which was successfully managed.

2 | CASE REPORT

A 77-year-old female patient was admitted to the emergency department because of thoracic complaints that had been present for 3 days and fluctuated in intensity and character. The symptoms started with pain in the back and radiated to the neck and jaw, which then spread retrosternally and to the epigastrium, whereupon an emergency presentation was made on suspicion of an acute coronary syndrome. On inquiry, the respiratory- and movement-dependent character of the pain and a significant decrease in exercise capacity with dyspnea NYHA III were reported. The presence of dizziness or syncope was denied.

The patient's relevant medical history included arterial hypertension, coronary artery disease with PCI of the right coronary artery 12 years ago, paroxysmal atrial fibrillation on oral anticoagulation and symptomatic sick sinus syndrome, with implantation of a dual-chamber pacemaker 13 month ago in order to prevent intermittent dizziness.

On initial admission, the normal-weight (1.65 m, 66 kg, BMI 24.2 kg/m^2) patient presented hemodynamically stable (blood pressure 127/82 mmHg, heart rate 70 bpm, SpO2 96% breathing ambient air, 16 breaths per minute, 36.3°C body temperature). The ECG showed sinus rhythm with intrinsic AV conduction and a previously known complete right bundle branch block without changes suspicious for ischemia or pericarditis. Laboratory chemistry revealed a markedly elevated NT-proBNP level (1644 pg/mL, reference value <738 pg/mL), a borderline elevation of high-sensitivity troponin I (hs-TNI, 24.7 ng/L, reference value <15.8 ng/L), a significant elevation of C-reactive protein (CRP, 211.4 mg/L, reference value <5 mg/L) with marginal leukocytosis (11.5 G/L, reference range 3.9–10.2 G/L), and acute renal failure with a glomerular filtration rate (GFR) of 27 mL/min.

Physical examination, urine status, and chest X-ray showed, except for a moderate global cardiomegaly no abnormalities or provided evidence of an infectious focus or abnormalities related to the implanted pacemaker device. Transthoracic echocardiography revealed normal right and left ventricular function with, however, a circular pericardial effusion measuring 35 mm and the impression of perforation of the right atrial pacemaker lead, which was confirmed by CT scan (Figure 1). Interrogation of the pacemaker showed regular aggregate function with unremarkable parameters (RA sensing 1.6 mV bipolar, RA pacing threshold 0.9 V at 0.4 ms bipolar, RA impedance 292 Ohm). However, only knowledge of the last interrogation performed 6 months earlier in our clinic indicated a decrease in RA sensing (4.3 mV bipolar to 1.6 mV bipolar) as well as a subtle decrease in RA stimulation impedance (370 to 292 Ohm). The trend of electrode impedance provided by the device (Enticos 4 DR, Biotronik) revealed a slight decrease in atrial impedance that had occurred acutely within the last few days and coincided with the onset of symptoms (Figure 2).

Given the hemodynamic compensated status and close intensive care monitoring, revision was delayed until the rivaroxaban effect had worn off. Thus, while a cardiac surgical team was on standby, a percutaneous approach was performed in a hybrid operating room 2 days after admission. After drainage of 700 mL of bloody effusion (hemoglobin concentration 8.8 mg/dL) by subxyphoidal pericardial insertion of a pigtail catheter under echocardiographic and fluoroscopic guidance, the atrial pacemaker lead was revised. Using a J-shaped mandrin, the electrode (Solia S53, Biotronik; helix length 1.8 mm, tip surface 4.5 mm^2) was reinforced and the screw retracted. Subsequently, the electrode could then be freely mobilized, easily removed and a new electrode of the same type with a more septal orientation inserted. With persistent pericardial dryness, the pigtail drainage could be removed on the second postoperative day and oral anticoagulation with rivaroxaban (20 mg/day, appropriate dose for body weight and renal function) resumed. Finally, the patient could be discharged on the fourth postoperative day after complete resolution of the pericardial effusion and complete recovery, with device function fully intact.

3 | DISCUSSION

Symptomatic perforation of a pacemaker electrode is a rare but serious complication, with a rate of occurrence of between 0.1% and 0.8%, occurring primarily intraoperatively or within the first few hours to after surgery.[4–7] According to recently published data from a systematic analysis of CT scans performed for other reasons, subclinical and possibly delayed CIED lead perforation appears to be more common, with a reported rate of 5.6%.[8] However, due to the methodology of this study, there are uncertainties regarding the time of occurrence of the perforation, as it was asymptomatic and thus detected incidentally. Delayed (>30 days after implantation) perforations that become symptomatic and not conspicuous by changes in electrical parameters alone are a rarity and have been described only in individual case reports over the decades of CIED therapy. The case reported here is therefore remarkable because it shows a very late (>1 year) and highly symptomatic perforation of an atrial electrode while the device parameters were within the normal range. Should delayed lead perforation become symptomatic, pleuritic or pericarditis chest pain has been reported in most cases.[3] However, the clinical presentation can be very manifold, ranging from hiccups, diaphragmatic pacing, angina with ST-segment

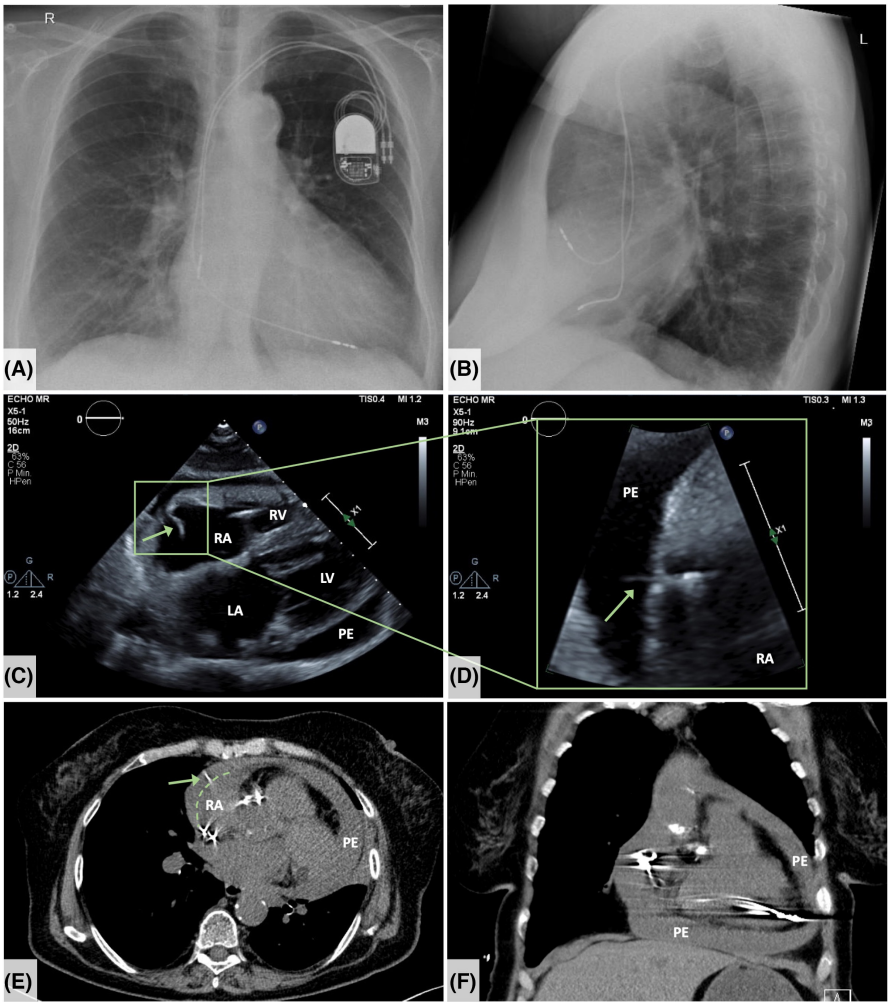

FIGURE 1 Chest X-ray in posterior–anterior (A) and lateral (B) projection showing global cardiomegaly, but without obvious dislocated or migrated pacemaker leads. Transthoracic echocardiogram of the subxiphoid view (C, D) showing the large pericardial effusion and the perforated atrial lead highlighted with an arrow. CT scans (E, F) showing an axial slice (C) with the perforated atrial lead highlighted with an arrow. The contour of the lateral wall of the right atrium is indicated by the dashed green line. The coronary CT slice (D) shows the large extent of pericardial effusion (PE). LA, left atrium; LV, left ventricle; PE, pericardial effusion; RA, right atrium; RV, right ventricle.

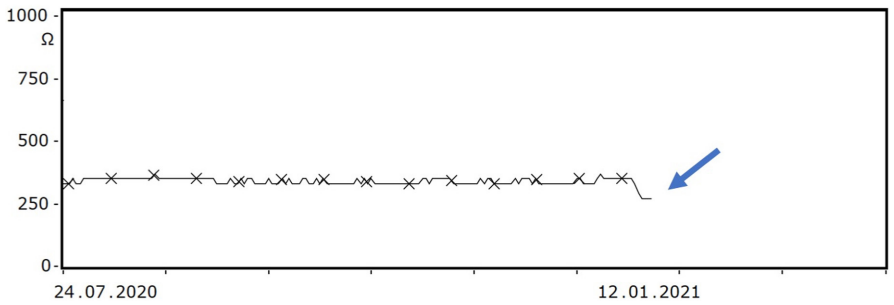

FIGURE 2 Extract from the device interrogation showing the trend in atrial electrode impedance since the last check 6 months ago. The blue arrow indicates the subtle decrease in atrial stimulation impedance, which correlates with the onset of symptoms. Dates are shown in the day.month.year format.

elevations to hemodynamic instability.[9–12] Regarding the electrical performance of the device, loss of capture or drastic increase in stimulus threshold usually associated with undersensing are the main malfunctions in this context.[3,7,13] While risk factors for symptomatic lead perforation such as steroid and anticoagulant use, screw-in leads, older age, and BMI <20 kg/m^2 have been identified,[14] an understanding of the specific mechanism of perforation remains subject to speculation. An unfavorable relationship between low profile and smaller diameter electrodes is often suspected, resulting in a higher force per unit area.[4] This could possibly explain why perforations can appear, even when very flexible and looped electrodes with minimal linear force at the tip are implanted, as is the case with atrial electrodes in the standard position in the atrial appendage.

As in the case reported here, most perforations are manageable percutaneously, but are nonetheless dependent on various factors such as perforation characteristics and damage to adjacent organs.[7]

With our report, we intend to raise the medical community's awareness of procedure-related complications, even those occurring after a considerable period of time, in the large group of CIED patients. Even nonspecific symptoms, like pleuritic or pericardial chest pain in CIED patients should raise suspicion of electrode perforation. In addition, since a lifelong risk apparently cannot be excluded, it does not matter how long ago the implantation was performed.

AUTHOR CONTRIBUTIONS

Christian Waechter: Conceptualization; data curation; investigation; project administration; visualization; writing – original draft. **Alexander M. Koenig:** Visualization. **Georgios Chatzis:** Visualization; writing – review and editing. **Julian Mueller:** Writing – review and editing. **Bernhard Schieffer:** Supervision; writing – review and editing. **Ulrich Luesebrink:** Data curation; investigation; supervision; writing – review and editing.

ACKNOWLEDGMENT

Open Access funding enabled and organized by Projekt DEAL. The graphical abstract was created using BioRender.com.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data supporting the findings of the present report are available upon request from the corresponding author.

CONSENT

The authors acknowledge that written informed consent was obtained from the patient for submission and publication of this report, including images, in accordance with COPE guidelines.

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How to cite this article: Waechter C, Koenig AM, Chatzis G, Mueller J, Schieffer B, Luesebrink U. Delayed perforation of an atrial pacemaker electrode: Lifelong risk for a rare but serious complication. *Clin Case Rep*. 2023;11:e7525. doi:10.1002/ccr3.7525