Late ventricular pacemaker lead perforation after electrical cardioversion—A case report

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Introduction
Cardiac implantable electronic devices (CIED) have a wide range of indications and implant numbers have been increasing in the past decades, with a recently reported age- and sex-adjusted annual incidence of 89.0 per 100 000.1 Inevitably, there is an associated risk of complications with these procedures.2 Further, both atrial and ventricular pacing are associated with an increased risk of atrial fibrillation (AF) and AF burden.3 Electrical cardioversion (ECV) is a commonly used and effective method to restore sinus rhythm in patients with persistent atrial arrhythmias.4

We present a case report of a patient presenting with pleuritic chest pain 2 weeks after ECV owing to ventricular lead perforation of a dual-chamber pacemaker implanted 3.5 years prior.

Case report
An 81-year-old male patient with persistent AF, gastroesophageal reflux disease, and arterial hypertension presented on January 4, 2022, with a 3-day history of pleuritic, breathing-dependent, left-sided chest pain with a visual analog scale score of 4 out of 10. The pain started in the afternoon while resting after a day with regular physical activity. The patient denied symptoms compatible with any acute infection or inflammatory disease.

The patient had been implanted with a left-sided dual-chamber pacemaker (Medtronic Adapta; Medtronic, Minneapolis, MN) for symptomatic Mobitz type II atrioventricular block in February 2018. Both the atrial and ventricular pacing leads were Medtronic 5076 CapSureFix Novus MRI SureScan active fixation leads and the generator was Medtronic Adapta (Medtronic, Minneapolis, MN). The atrial lead was positioned in the right atrial appendage, whereas the ventricular lead was implanted in the right ventricular apex, and both had excellent bipolar lead values at implant (Figures 1 and 2A). During his follow-up, he was diagnosed with AF, initially paroxysmal but evolving to persistent AF, for which he was started on apixaban 5 mg twice daily and the pacemaker was programmed to VVIR mode. On December 21, 2021, he underwent an elective ECV for persistent AF after being started on sotalol 40 mg twice daily a few days prior. The ECV was successful in restoring sinus rhythm.
with 1 synchronized 200 joule shock with the defibrillation pads in anteroposterior electrode position. Pacemaker interrogation before and after the ECV (Figure 1) did not show any significant changes in lead parameters.

The patient presented on January 4, 2022, to our CIED clinic with these symptoms. On device interrogation a significant increase in bipolar right ventricular threshold was noted from 1.0 V at 0.4 ms to 3.75 V at 0.4 ms, while bipolar lead impedance and atrial lead measurements remained unchanged (Figure 1). Chest radiograph showed unchanged lead position compared to the postimplant radiograph in 2018 (Figure 2A). The output on the ventricular lead was increased to 6.0 V at 1.0 ms with bipolar programming and the patient was booked for a semi-urgent ventricular lead revision as an outpatient.

Three days later the patient presented again with progressive symptoms of pleuritic chest pain and new dyspnea upon minimal exercise. Upon device interrogation the bipolar ventricular lead threshold increased further to 5.0 V at 0.4 ms. A transthoracic echocardiogram showed a moderate-sized pericardial effusion without echocardiographic signs of tamponade, which was not present on a routine transthoracic echocardiogram in August 2021 (Figure 2B). Further, on echocardiogram a high suspicion of ventricular lead perforation was noted as the lead projected in the epicardial fat (Figure 3A). This diagnosis was confirmed on computed tomography (CT) scan, where the ventricular lead clearly projected through the myocardium in the pericardial space (Figure 3B). Apixaban was held and the patient was admitted for lead extraction with implantation of a new ventricular lead.

On January 14, 2022, the patient underwent a complex laser lead extraction, as during the procedure the ventricular lead was found to have significant adhesions to the superior vena cava. A new ventricular lead (Medtronic 5076 CapSureFix Novus MRI SureScan; Medtronic, Minneapolis, MN) was implanted using axillary vein access with a septal position. The patient tolerated the procedure hemodynamically well and transesophageal echocardiogram showed a stable pericardial effusion throughout the procedure. The patient was discharged the next day.

At the 1-month follow-up clinic visit the patient was free of symptoms and functioned New York Heart Association class I. Device interrogation showed sinus rhythm without recurrence of AF, as well as normal lead measurements (Figure 1). The atrial and ventricular pacing percentages were 87.7% and 99.8%, respectively. Repeat transthoracic echocardiogram showed regression of the pericardial effusion to within normal range.

**Discussion**

This case presents a sequence of events resulting in a severe complication that, to our knowledge, has not been described
before. The patient presented with a clinically relevant ventricular lead perforation 3 years and 10 months after the initial device implant. This diagnosis was confirmed on transthoracic echocardiogram and CT scan. In a recent study the accuracy of chest radiography, transthoracic echocardiogram, and cardiac CT scan to diagnose a cardiac lead perforation was 73.1%, 82.7%, and 98.1%, respectively. The incidence of lead perforation has been described to range between 0.09% and 1.5%. Typically, lead perforation presents as an acute (<24 hours) or subacute (<1 month) complication; late presentation up to 8 years after initial implant has been described in case reports. However, in a large prospective observational cohort 10% of patients had a new pericardial effusion after CIED implant or upgrade, indicating that

![Figure 2](image1.png)

**Figure 2** Comparison of imaging studies. A: Anteroposterior chest radiograph post implant and at admission. B: Transthoracic echocardiogram. *: New pericardial effusion on parasternal short-axis view.

![Figure 3](image2.png)

**Figure 3** Cardiac imaging visualizing perforated lead with pericardial effusion. A: Right ventricular view on transthoracic echocardiogram. B: Chest computed tomography scan. White arrows: right ventricular lead projecting through the ventricular wall with pericardial effusion. *: Pericardial effusion.
subclinical lead perforation might be more common. This was confirmed in a retrospective analysis of CIED patients who underwent CT scans for other indications than the suspicion of lead perforation. This study detected asymptomatic lead perforation in 15% of patients. Unfortunately, unipolar lead measurements were not available in this case, but these can be particularly helpful in the work-up for a potential lead perforation or to detect subclinical lead perforation. These data emphasize the importance of verifying a septal position of the right ventricular lead in the left anterior oblique view.

The temporal relation between the ECV and lead perforation is striking. The ECV was performed according to the most recent guidelines with anteroposterior electrode placement. Device malfunction after ECV has been described but is mostly restricted to old reports in the era of monophasic defibrillation, anterolateral electrode placement, and rightsided implants with unipolar leads. Overall, there is a paucity of data on the safety of external ECV in CIED patients. In contemporary reports CIED complications after ECV are rare (<1%) and limited to transient increases in pacing threshold. In a retrospective, multicenter registry, data on 16,554 patients undergoing ECV were collected, of which 1809 (10.9%) patients had a CIED. Complications were reported in 11 (0.6%) patients with a CIED, all of which were transient elevations in pacing thresholds. Of note, 38% of the included centers performed ECV with an anterolateral electrode configuration.

We hypothesize that our patient had an asymptomatic, subclinical right ventricular lead perforation that deteriorated after the ECV, most likely owing to the simultaneous mechanical contraction of the atria and ventricles, which resulted in a forward pressure at the lead tip given the significant adhesions of the right ventricular lead in the superior vena cava, followed by slow migration of the lead tip into the pericardial fat. A direct cauterization effect of the ventricular lead by the electrical current of the ECV cannot be excluded. However, the latter has not been reported in bipolar leads, and one would expect immediate changes in lead measurements after the ECV. Unfortunately, as the lead required complex laser extraction, the connector pin was cut off and the lead integrity could not be tested afterwards. Therefore, this hypothesis could not be verified, and the exact chain of events remains unclear.

**Conclusion**

Late CIED lead perforations are rare but can present at any time after implant. The diagnosis should be suspected in case of pleuritic symptoms, a new pericardial effusion, or acute changes in lead measurements upon device interrogation, including unipolar lead measurements. Both transthoracic echocardiogram and cardiac CT scan can be helpful to confirm the diagnosis of lead perforation. Cardiac CT scan has the highest diagnostic accuracy; however, in the presence of a pericardial effusion echocardiogram is indicated to assess its clinical relevance. Increased awareness in CIED patients after ECV remains warranted, even though the causal relation between the ECV and lead perforation in this case could not be proven.

**Acknowledgment**

Written informed consent was obtained from the patient for their anonymized information.

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