Very-late-phase perforation of the septum and left ventricle by a passive fixation pacemaker lead: Case report

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Introduction

Ventricular perforation by pacemaker leads mainly occurs in the acute phase. Late lead perforation (LLP), defined as that which happens ≥30 days after cardiac implantable electronic device (CIED) implant, is rare.[1] The most common perforation site is the right ventricular (RV) apex and an active (rather than passive) fixation lead is considered as a predictor.[2] Here, we report a rare case of cardiac perforation from the interventricular septum to the left ventricular side by a passive fixation lead in the very late phase, more than 9 years later.

KEY TEACHING POINTS

- A case of very late lead perforation from the interventricular septum to the left ventricular side by a passive fixation lead in the very late phase is reported.
- Late lead perforation is rarer and comparatively little has been written about risk factors.
- The case is instructive for diagnosis and therapeutic strategy for very-late-phase lead perforation.

Case report

An 86-year-old Japanese woman was admitted to our hospital with general fatigue and bradycardia, which we suggest was due to pacing failure. She had been diagnosed with atrioventricular block 9 years previously and a dual-chamber pacemaker with passive fixation leads (CapSure Sense Model 4574 and 4074; Medtronic, Minneapolis, MN) had been implanted. Cardiac sarcoidosis was suspected and medication with prednisolone 5 mg/day had been started after pacemaker implantation. From 6 months previously, the bipolar RV lead threshold had been gradually increased (from 0.5 V at 0.4 ms to 1.5 V at 0.4 ms), although there was no marked change of lead impedance or sensing amplitude.

Electrocardiogram on admission showed failure of ventricular capture (Figure 1). Cardiac perforation by RV lead was suspected and pericardial effusion was confirmed by chest radiography and computed tomography (Figure 2A and 2B). Cardiac tamponade was ruled out by echocardiography. The patient's vital signs were stable on admission, except for rise in body temperature (37.4°C). She was lean, with body mass index of 19.4. Blood tests revealed an elevated white cell count (27,280/μL) and C-reactive protein (15.8 mg/dL). An obvious source of inflammation could not be identified, but blood cultures showed *Streptococcus agalactiae*, which suggested the possibility of a CIED-related infection. A temporary transvenous pacemaker was inserted, and medical therapy with antibiotics was started.

Ten days after hospitalization, progression of RV lead perforation was detected on chest radiography, chest computed tomography (Figure 2C and 2D) and fluoroscopy (Supplemental Video 1). Although the patient was hemodynamically stable, we conducted open chest surgery to extract all parts of the pacemaker system. Because the bulky tip of the passive fixation lead might damage the myocardium during removal and surgical repair of the perforation site was expected, we decided to perform thoracotomy. The RV lead was found to pass the interventricular septum, and it perforated from the left ventricle wall side and finally into the pericardium (Figure 3, Supplemental Video 2). Myocardium tissue loss was not observed at the perforation site. Therefore, the perforation site was repaired with 3-0 polypropylene and felt, and the pacemaker lead and generator were removed. The RV lead was not strongly adhered to intracardiac structures, but there was adherence with the superior vena cava that required surgical

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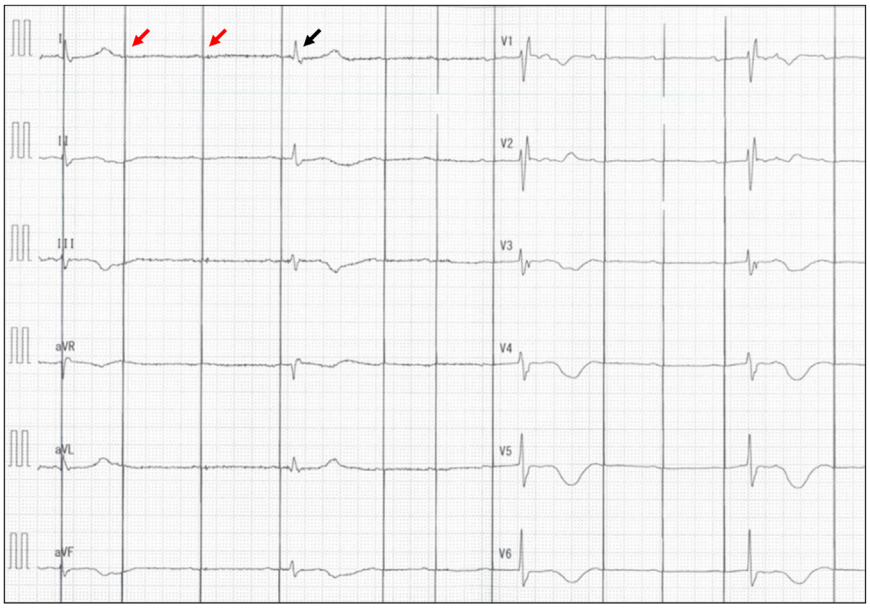

Figure 1 Twelve-lead electrocardiogram on admission. Failure of ventricular capture (*red arrow*) and escape beat (*black arrow*) are shown.

detachment. The patient was frail and elderly, so rather than a 2-stage strategy of CIED removal and implantation, we performed simultaneous CIED removal and implantation with epicardial leads as part of the same surgery. The patient was discharged on the 54th hospital day without serious complications, and she has been uneventful for 9 months.

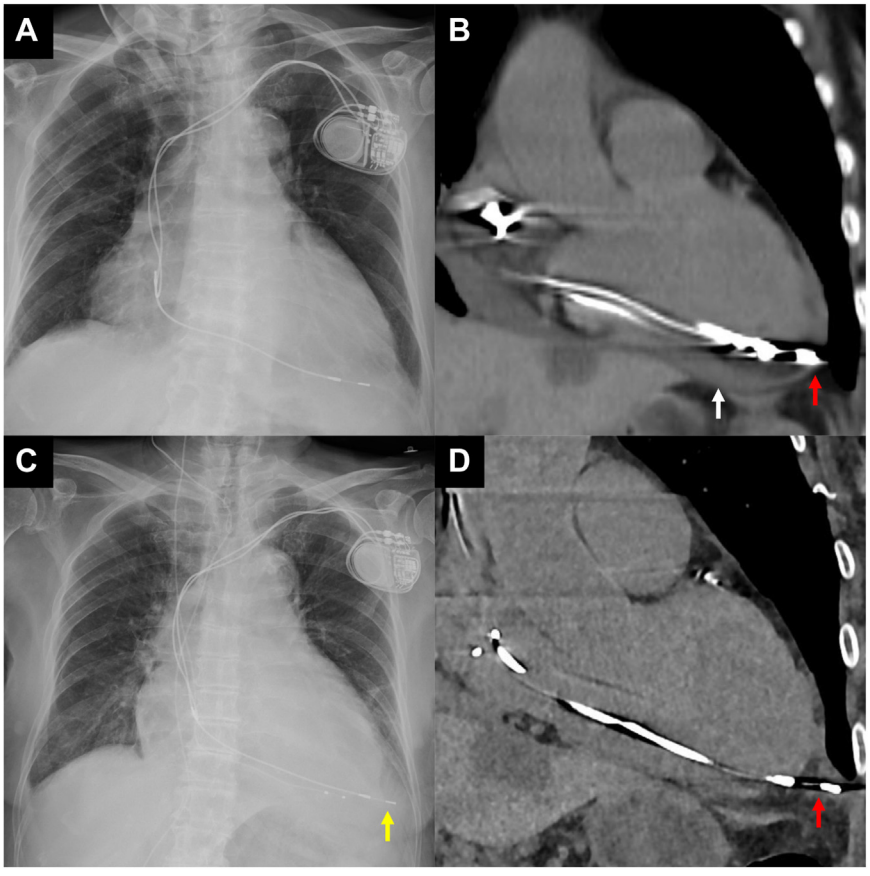

Figure 2 Chest radiography and computed tomography images. **A:** Chest radiography on admission. **B:** Computed tomography on admission. Cardiac perforation by right ventricular lead (*red arrow*) and pericardial effusion (*white arrow*) were confirmed. **C:** Chest radiography on the tenth hospitalization day. The right ventricular lead progressed (*yellow arrow*). **D:** Computed tomography on the tenth hospitalization day. The right ventricular lead progressed (*red arrow*) and pericardial effusion decreased.

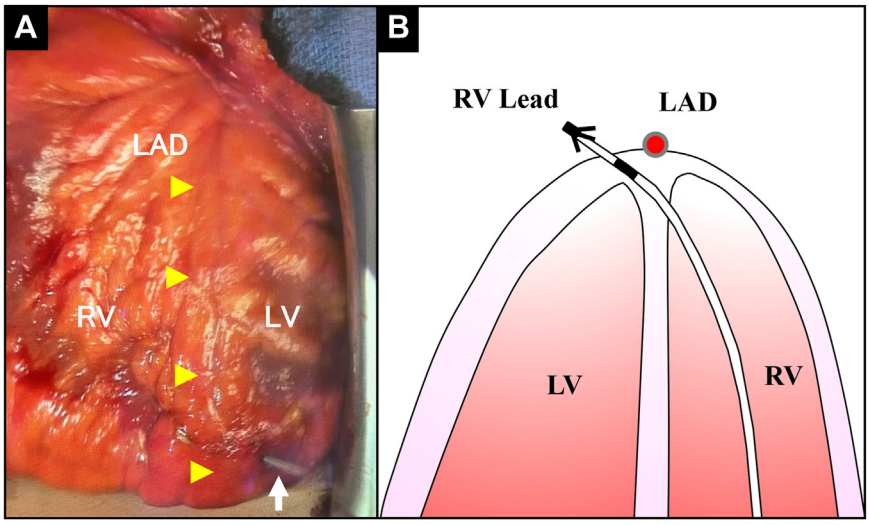

Figure 3 Operation image and schema of the lead penetrating through the left ventricular side. **A:** Intraoperative image. The right ventricular lead (RV lead; *white arrow*) penetrated from the left ventricular side close to the left anterior descending artery (LAD). **B:** Schema of lead perforation. LV = left ventricle; RV = right ventricle.

Discussion

Lead perforation after CIED implantation occurs in approximately 1.0% of cases.[1] It usually occurs within the perioperative period, and hemodynamic disruption owing to cardiac tamponade and pacing failure often occur. Reported risk factors for lead perforation in the acute phase are high age, low body mass index (<20), use of steroids, active (rather than passive) fixation leads for permanent pacemaker implantation, and the use of temporary pacing leads.[3] However, LLP is rarer and comparatively little has been written about risk factors.

Jessel and colleagues[1] reported 11 cases with LLP; the median time from lead implantation to device extraction was 246 days (longest time was 6 years), although there are only two cases with passive fixation pacemaker leads. LLP is usually associated with active fixation leads rather than with passive fixation leads.[4] Demo and colleagues[5] reviewed 8 cases of LLP with passive fixation leads; the longest time from implantation to extraction was 36 months. To date, the longest reported period of LLP with a passive fixation lead was 8 years.[6] At 9 years, our case is the longest known period from implantation to perforation, and it is notably unusual in that it was with passive fixation lead type.

In addition to the usage of a passive fixation lead and the very-late-phase event, the RV lead passing through the septum, left ventricular wall, and pericardium is notable. Typically, the perforation site of the RV lead is around the RV apex. Four cases of pacemaker lead perforation from the left ventricular wall side have been reported,[7–10] but active fixation or lumen-less pacemaker leads were applied in these cases.

Open heart surgery in the current case confirmed that the RV lead had strong adherence with the superior vena cava, although with fewer adhesions than intracardiac structures. The force of the bent portion of the pacemaker lead might therefore have been concentrated at the tip of lead, which was why the passive fixation lead could perforate even in the very late phase. We speculate that long-term oral administration of steroids might reduce intramyocardial adhesion with RV leads and cause fragility of the myocardial wall.

Conclusion

In a rare case, the left side of the ventricular wall was perforated by a passive fixation pacemaker lead that was implanted more than 9 years previously. Cardiac perforation may occur even after significant delay if there are risk factors such as steroid prescription. When abnormal measurement parameters are observed, careful observation is required.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.07.019.

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