Extremely late asymptomatic atrial lead dislodgement from the right atrial appendage to superior vena cava with autopsy findings

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Introduction

The complication rate of pacemaker insertion is 3.8%–13.2%, and the most frequent lead-related one is dislodgement, with 0.79% (procedure-year)[1–4]; this complication mostly occurs within 2 months after the procedure. Atrial lead migration >5 years after implantation is extremely rare, and little has been reported on further observation of the causes, such as pathologic anatomy or patient characteristics. Here, we describe a case in which an atrial lead with passive fixation dislodged 9 years after the implantation without an obvious trigger and in which an autopsy was performed for the pursuit of causes.

Case report

A 77-year-old woman underwent insertion of a pacemaker (generator: Fidelity ADx XL DR 5388; atrial lead: IsoFlex S 1642T-46; ventricular lead: IsoFlex S 1646T-52; St. Jude Medical, Sylmar, CA) for complete atrioventricular block at another hospital. Her cardiac function was normal on echocardiography. The atrial lead was placed at the right atrial appendage (RAA), and the ventricular lead was placed at the apex (Figure 1A and B). The atrial lead was placed with slight upward tension. Both leads were passive fixation type. The pacing parameters of both leads were within normal levels (atrial lead: P wave, 4.9 mV; pacing threshold, 1.125 V/0.4 ms; lead impedance 469 ohms; ventricular lead: R wave, 9.4 mV; pacing threshold, 0.35 V/0.4 ms; lead impedance, 707 ohms). Annual pacemaker check did not detect any lead problems. She also had rheumatoid arthritis and had been receiving azulfidine for more than a year. Surgical procedures around the generator and leads, including pacemaker exchange for battery exhaustion, were not performed. In addition, the generator and the lead positions had not been shifted for years.

Nine years after pacemaker implantation, she was admitted to our hospital to undergo surgery for ankle osteoarthritis. Chest radiography preoperatively did not show changes compared to that at the time of pacemaker implantation (Figure 1C). The surgery was performed under local anesthesia in the supine position and her upper body did not move during the procedure. She underwent walking rehabilitation with crutches starting the following day. One week postoperatively, she had dyspnea, and chest radiography was performed, which showed slight pleural effusion. Moreover, the tip of the atrial lead seemed to be located much more above the RAA (Figure 1C, D). The following day, we performed chest radiography again, and the atrial lead was dislocated, as expected. We checked the pacemaker and lead parameters; the percentage of atrial pacing was >99%, and the threshold of the lead and impedance were not changed before and after the surgery (0.75 V/0.4 ms and 294 ohms preoperatively and 1.0 V/0.4 ms and 292 ohms at atrial lead dislodgement). These data were not changed in the supine or sitting position. We also confirmed the lead movement by fluoroscopic imaging, and the tip of the atrial lead was anchored to some tissue of the superior vena cava (SVC) and not floating. Therefore, we did not immediately replace the atrial lead. After 1 month, the patient died owing to systemic capillary syndrome, which was also the cause of pleural effusion. The consent for autopsy was obtained from her family.

The atrial lead was already removed, so we did not find the exact location where it was finally anchored. The macroscopic findings outside the heart showed that the 2 RAA spots had turned black (Figure 2A). The lower spots were covered with fibrous tissue. Inside the upper black spots, some clots were attached (Figure 2B). Hematoxylin-eosin staining showed that some artificial coating was found inside

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KEY TEACHING POINTS

- This case showed the late dislodgement of an atrial lead with passive fixation 9 years after the implantation. The tip of the atrial lead was moved from right atrial appendage to superior vena cava (SVC). The tip was caught in the SVC and was not moved by fluoroscopic imaging; in addition, estimated parameters were within normal range.
- We were able to perform an autopsy for further investigation. The cause of lead dislodgement on chronic phase was supposed to be a rough adhesion between the atrial lead and right atrial appendage owing to the past microperforation, the long-time administration of an immunosuppressive drugs, and the stretched lead position upwardly.
- Although the exact trigger of the lead dislodgement was unclear, the walking rehabilitation using crutches after the ankle surgery might have partly contributed to the lead movement.

the lower cavity, which was thought to be part of the tip of the atrial lead. The top of the lower cavity was covered with thin fibrous tissue but had no myocardial tissue (Figure 3A, C, and D). This indicated that the lead was perforated once but did not penetrate the epicardium, and fibrous tissue covered over the tip. The upper spot showed a thin myocardial layer and scattering blood cells inside but no fibrous tissue (Figure 3B). This suggested that there was some mechanical contact, but both were not stuck for a long time. These findings indicated that the atrial lead was originally located at the lower RAA, and asymptomatic microperforation developed. The adhesion between the tip of the atrial lead and atrial tissue was not firm enough, and after 9 years, the lead was temporarily moved by some kind of trigger, such as crutches, to the upper RAA and finally migrated to the SVC.

Discussion

The present case showed an atrial lead with passive fixation located at the RAA that migrated adiabatically to the SVC 9 years after pacemaker implantation without any invasive procedure around both leads and the generator.

The most frequent complication of pacemaker leads has been reported to be dislodgement, and the FOLLOWPACE study showed that dislocation of an atrial lead within 2 months was found in 1.9% of patients with active lead fixation and 3.9% of patients with passive lead fixation ($P = .059$). For a mean of 5.8 years except the first 2 months, its occurrence was 1.3% and 1.1%, respectively.[2] The cause of lead dislocation in one-third of patients was inadequate fixation of sleeves.[5] Regarding the late complication of atrial

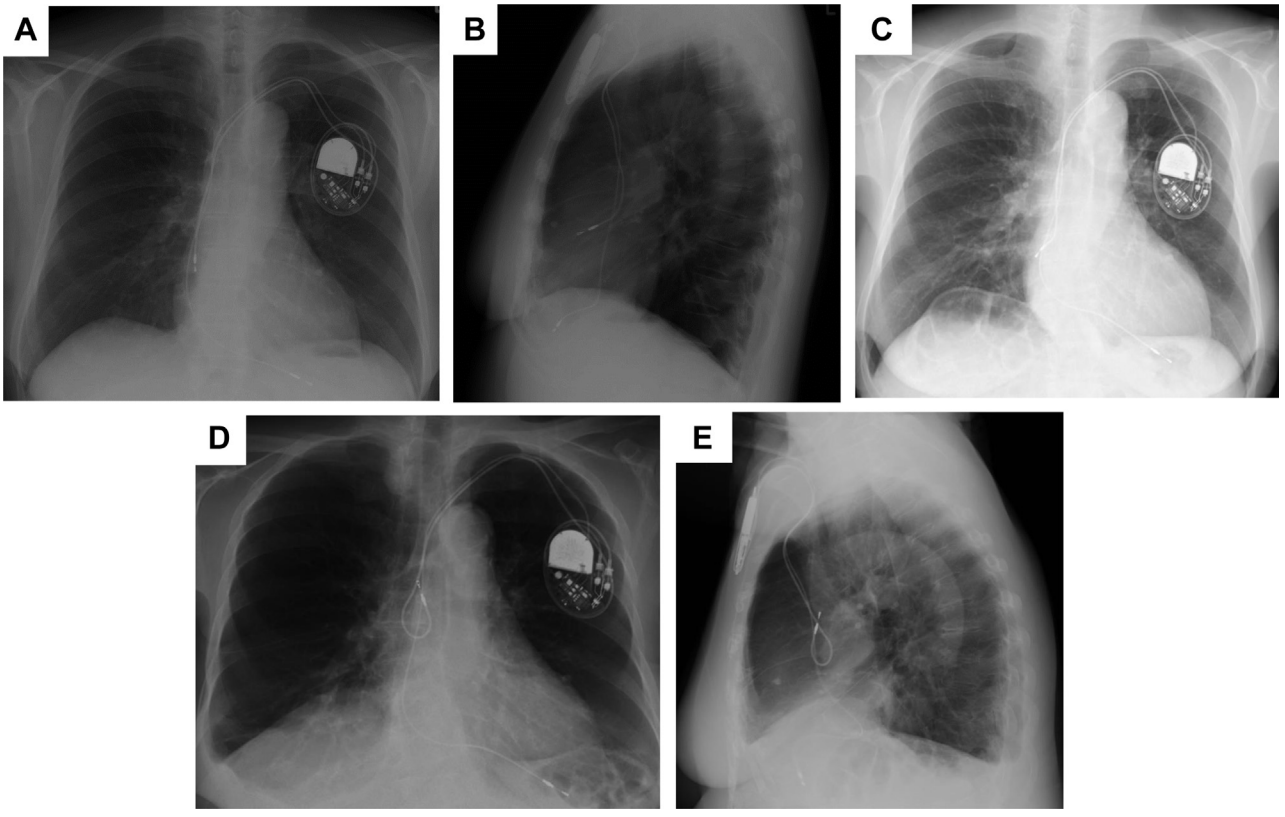

Figure 1 Chest radiographs during pacemaker insertion (**A, B**) and at hospitalization after 9 years (**C**). After the surgery, the tip of the atrial lead was moved to the superior vena cava (**D, E**).

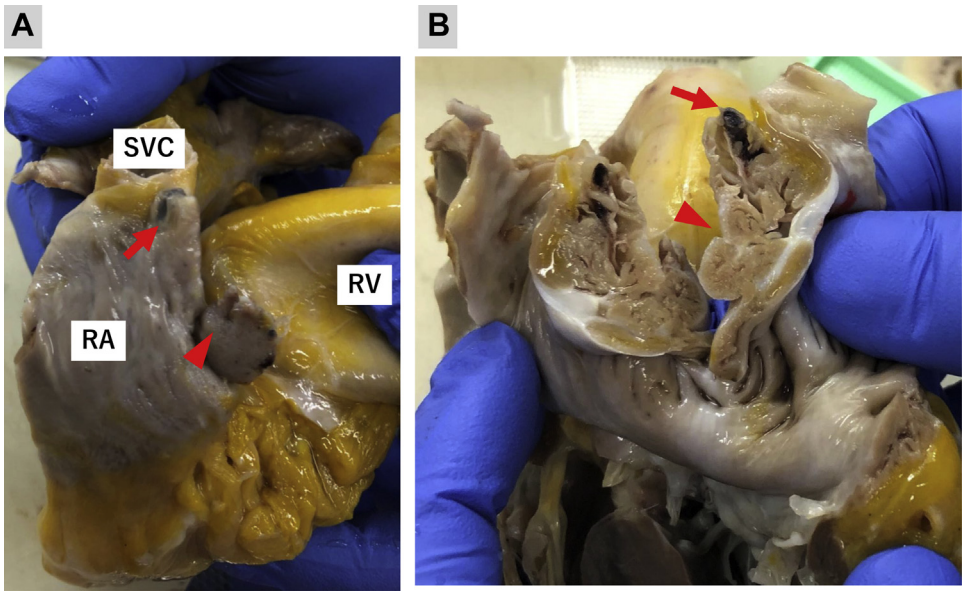

Figure 2 **A:** Macroscopic image of the outside of the heart. Two black points on the top of the right atrial appendage (RAA) were found (upper spot, *red arrow*; lower spot, *red arrowhead*). RA = right atrium; RV = right ventricle; SVC = superior vena cava. **B:** The macroscopic image inside the heart, cut on the surface of the upper black spot. The entrance of the RAA was white and thick, which indicated that the atrial lead was inserted from here to the RAA. Inside the top of the RAA, some clots adhered (*red arrow* and *arrowhead* pointed to the same objects as in **A**).

lead, Kristensen and colleagues[6] reported late dislodgement of the atrial lead with active fixation 7–8 years after lead implantation, and the mechanism of dislodgement was speculated to be that the operation for replacement of the new generator caused a change in the lead traction. The present case is the first case report of extremely long-term dislocation of the atrial lead with passive fixation and detailed observation by autopsy.

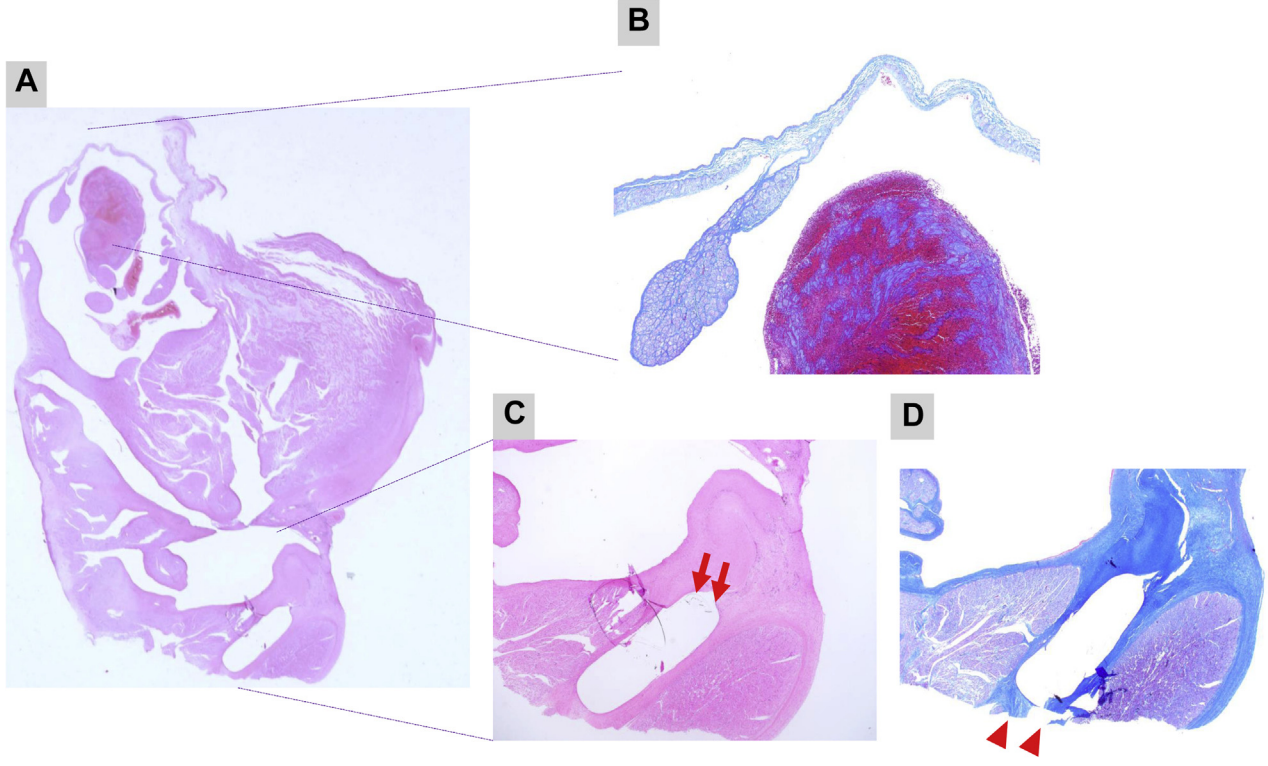

Figure 3 **A:** Hematoxylin-eosin stain at the slice shown in Figure 2B. **B:** Zoom of the upper cavity showed a thin myocardial layer and blood cells inside the tissue. There was no fibrous sheath inside the myocardial tissue. **C:** Zoom of the lower cavity. There was an artificial coating inside (*double red arrow*). **D:** Masson stain version of **C**. **C** and **D** were slices at the same part of RA. The cavity was coated only with fibrous tissue, and no myocardium was noted outside the top of the cavity (*double red arrowhead*).

In this case, the atrial lead was a typical passive-fixation tined lead. The tip electrode was made of platinum-iridium and the surface area of that was 3.5 mm^2. The outside except the tip was covered with silicone rubber. From the product performance report (2020, first edition), IsoFlex S 1642T showed that among 27,139 implanted leads, lead dislodgement occurred within 30 days of implantation was reported in 49 (0.18%) and subsequently in an additional 44 leads (0.16%), respectively. The further details of these complications were unknown.

The location at the atrial lead after dislodgement was in the SVC, and the sleeve of excitable tissue was long enough to capture during atrial pacing. In addition, the connection between the lead and atrial tissue/SVC was not strongly adhered before and after the event. That was thought to be the reason for the unchanged parameters of the lead. From the macroscopic view, there was a bleeding spot inside the RAA other than where the tip of the atrial lead was placed, which indicated that the lead penetrated another wall of the RAA after the lead had been removed from the original place. From a microscopic view, the first place where the lead was located was covered with fibrotic tissue without myocardial cell outside. This suggests that silent lead perforation occurred at some point, but there was no protrusion through the pericardium, and fibrous tissue proliferated over it. Moreover, this case showed that the atrial lead had upward tension from the beginning, which indicated that the pulling force was constantly applied on the lead. Regarding immunosuppressive agents, there was no report about the relationship between immunosuppressive drugs and lead dislodgement. However, it is known that immunosuppressive agents influence fibrinolytic process by the inhibition of some chemokines and cytokines. In an animal experiment, the use of immunosuppressive drugs prevented postoperative peritoneal adhesions. Therefore, the fibrotic adhesion between atrial tissue and a pacemaker lead could be inhibited under long-time administration of an immunosuppressive agent through the similar process.[7]

The microperforation, pulling force, and immunosuppressor administration for a long period involved insufficient and vulnerable fibrous tissue construction. Consequently, these complicated factors were thought to contribute to long-term lead dislocation. Although the exact trigger of the lead movement was uncertain, stretching up of shoulders using crutches may have contributed to the dislodgement.

Van Gelder and colleagues[8] reported a review of late atrial lead perforation that had occurred in 26 patients an average of 45.7 months after lead implantation. Four leads were passive fixation type, 9 patients were asymptomatic, and perforations were unintentionally detected by lead extraction for device infection, chest radiography, angiography performed for other reasons, or pacing and/or sensing failure.[8] Even though there is no pericardial effusion or abnormal parameter of the pacemaker, lead perforation could occur asymptomatically.

From these findings, we have to consider that there could be much more unnoticed lead perforation or dislodgement even in the extremely late phase and pay extra attention to the relaxed shapes of atrial leads, especially in patients receiving immunosuppressive agents.

Conclusions

We report a case of atrial lead dislodgement 9 years after pacemaker implantation. Microperforation, lead instability, and the administration of an immunosuppressive agent were thought to affect extremely late lead dislocation.

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