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CASE REPORT

CLINICAL CASE

Recurrent Pericardial Effusion Resulting From Right Ventricular Free Wall Injury Caused by Leadless Pacemaker Tines

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ABSTRACT

An 87-year-old man developed delayed cardiac tamponade 55 min after leadless pacemaker implantation and recurrent pericardial effusion 20 days later. Electrocardiogram-gated enhanced cardiac computed tomography revealed that the leadless pacemaker tines on the lateral side had penetrated the right ventricular free wall. He underwent off-pump hemostatic surgery. (J Am Coll Cardiol Case Rep 2024;29:102378) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

An 87-year-old man (weight: 45 kg, height: 166 cm, body mass index: 16.3 kg/m^2) developed atrial fibrillation and symptomatic sick sinus syndrome (Rubenstein type III). Transthoracic echocardiography (TTE) revealed normal left ventricular ejection fraction and slight pericardial effusion (PE) (Video 1). He refused catheter ablation. He reported transient pre-syncope. Furthermore, he was considered susceptible to bacterial infection because he had a history of multiple admissions because of infectious disease within the prior year. Therefore, we selected leadless pacemaker (LP) (Micra, Medtronic) implantation.

LEARNING OBJECTIVES

- To recognize the risk of delayed cardiac tamponade and late-onset PE caused by RVFW injury by lateral LP tines.
- To discuss the safe fixation site, the risk stratification for cardiac perforation, and OAC therapy interruption before LP implantation.

On day 1, LP implantation was performed without interruption of apixaban. After right atrioventriculography (**Figure 1A**, Video 2), the delivery catheter tip was connected to the intraventricular septum (IVS). At the first deployment (**Figure 1B**, Video 3), the pacing threshold was 1.0 V at 0.24 ms of pulse width, but the impedance was relatively low (400 Ω). We redeployed the LP at the lower IVS (**Figure 1C**, Video 4). The pacing threshold was 0.75 V at 0.24 ms of pulse width, and the impedance was 440 Ω. The 3 tines seemed engaged, and we fixed the LP. His vital signs remained stable throughout the procedure, and the amount of PE was unchanged.

Fifty-five minutes after returning to his room, he suddenly developed shock. He was diagnosed with cardiac tamponade and underwent epicardial drainage. We suctioned 500 mL of bloody PE. After

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ABBREVIATIONS AND ACRONYMS

CT = computed tomography

ECG = electrocardiogram

IVS = intraventricular septum

LP = leadless pacemaker

OAC = oral anticoagulation

PE = pericardial effusion

RVFW = right ventricular free wall

TTE = transthoracic echocardiography

andexanet alfa administration, the bleeding was controlled. TTE and plain cardiac computed tomography (CT) revealed minimal residual PE (**Figure 2A**). On day 2, apixaban was restarted. On day 3, epicardial drainage was removed. On day 11, he was discharged. However, on day 20, he visited our clinic complaining of general fatigue without any chest symptoms. His consciousness level was clear, blood pressure was 116/47 mm Hg, heart rate was 54 beats/min, body temperature was 36.4 °C, and SpO_2 was 98% in room air. The amount of PE had increased (**Figure 2B**, Video 5). The 12-lead electrocardiogram (ECG) showed no significant ST-T change (**Figure 2C**). He was emergently admitted.

PAST MEDICAL HISTORY

The patient had a history of hypertension, diabetes mellitus, chronic kidney disease (estimated glomerular filtration rate: 41.1 mL/min/1.73 m^2), moderate aortic valve stenosis, and abdominal aortic stenosis. Apixaban (2.5 mg, 2 times daily) was prescribed.

DIFFERENTIAL DIAGNOSIS

The etiology of recurrent PE was considered as oozing from a residual injury of the myocardium or epicardial vessel or hemorrhagic pericarditis by the irritation of LP.

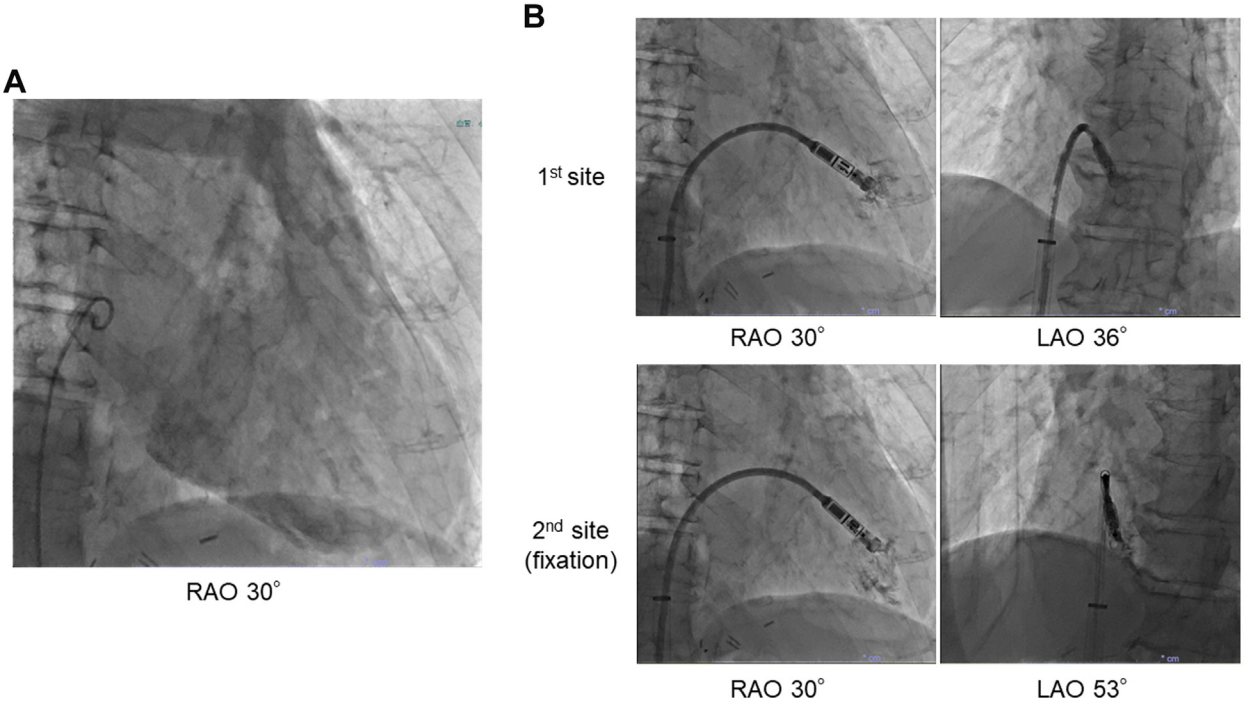
INVESTIGATIONS

On ECG-gated enhanced cardiac CT, the LP tip was connected to the IVS, but 2 lateral tines had penetrated the right ventricular free wall (RVFW) (**Figure 3**, Video 6), suggesting that the mechanism of increased PE was related to RVFW injury.

MANAGEMENT

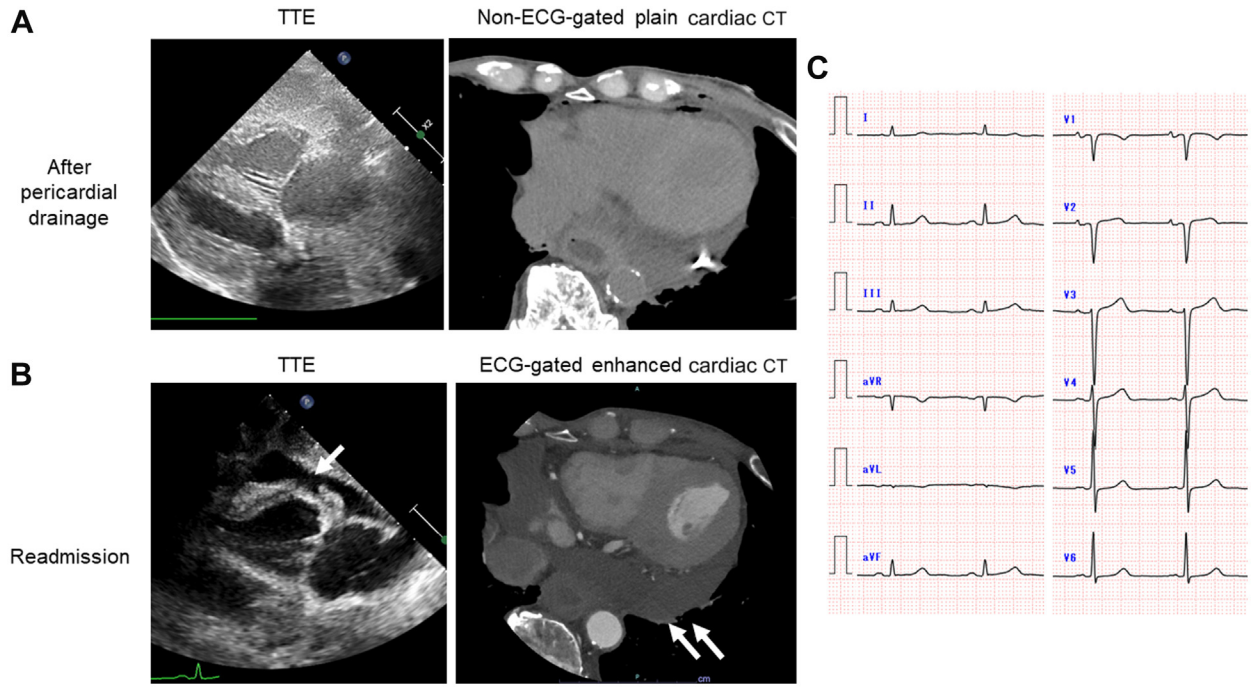
Considering the involvement of pericarditis, we first administered nonsteroidal anti-inflammatory drugs. However, the amount of PE gradually increased. On day 26, we performed open-chest hemostasis. Considering his frailty, we selected off-pump surgery via a lower hemisternotomy. In the pericardial cavity, old bloody PE had accumulated. A tine was present in

FIGURE 1 Leadless Pacemaker Implantation Procedure



(A) The right atrio-ventriculogram (right anterior oblique [RAO], 30°). (B) (Top) The first deployment; (bottom) the second deployment. LAO = left anterior oblique.

FIGURE 2 **Pericardial Effusion After Pericardial Drainage and at the Readmission**



(A) Nonelectrocardiogram (ECG)-gated plain cardiac computed tomography (CT) and transthoracic echocardiography (TTE) immediately after pericardial drainage. (B) ECG-gated enhanced cardiac CT and TTE at readmission. White arrow indicates pericardial effusion. (C) 12-lead ECG at readmission.

the visceral pericardium without active bleeding (**Figure 4**, Video 7). Because the parameters were good, we did not remove the LP. An autologous pericardial patch was attached around the tine. We repeatedly observed the amount of PE, and there was no recurrence.

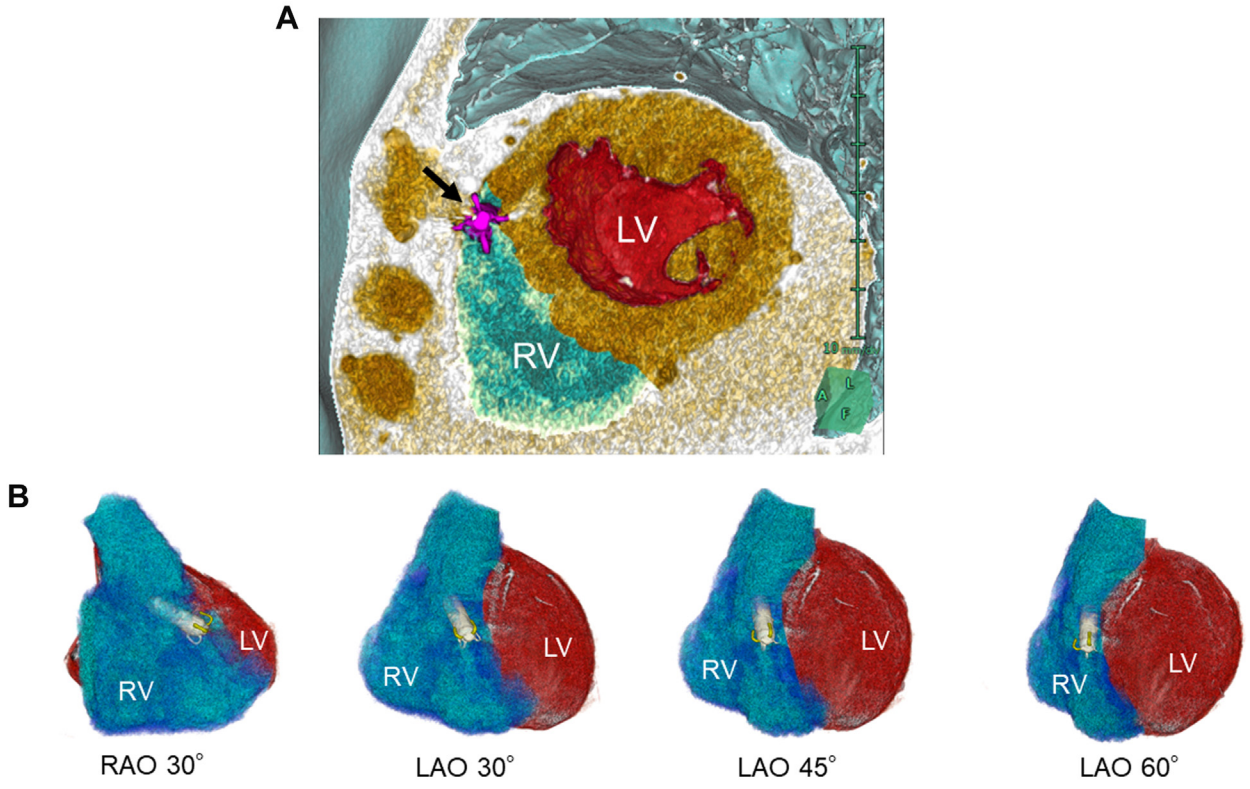
DISCUSSION

We presented a case of delayed cardiac tamponade and recurrent PE after LP implantation. RVFW injury by the LP tines caused these events. The patient underwent off-pump hemostatic surgery. Cardiac perforation, the major complication during LP implantation,[1] often results in catastrophic outcomes.[2] The present case highlights the residual injury post-LP implantation.

SAFE LP IMPLANTATION. We reviewed our implantation strategy. First, IVS should be selected as the fixation site, avoiding the apex and the hinge area.[3] On the left anterior oblique view, we confirmed that the delivery catheter had not advanced into the RVFW. However, given his narrow and tapered RV morphology, the tip tended to be parallel to the IVS, resulting in its displacement toward an apical site near the hinge area. Eventually, the lateral tines reached the RVFW. Screw-fixed LP might be better for him. Second, risk factors for cardiac perforation should be discussed.[4,5] He was classified as high-risk (2 points, age ≥85 years [+1], body mass index ≤20 kg/m^2 [+1], heart failure [+1], atrial fibrillation [−1]).[5] The perforation rate was estimated to be 9.3% after 2 deployments. We selected LP to avoid the risk of infections. In such high-risk cases, however, maximum caution and minimal deployments should be exercised. Third, oral anticoagulation (OAC) therapy should be discontinued. Because OAC therapy continuation has not been associated with cardiac tamponade,[6] we did not routinely discontinue OAC therapy, which could cause oozing from minimal tissue injuries in high-risk cases.

MANAGEMENT OF PE. We routinely checked the PE via TTE but failed to notice the increased PE post-implantation. Short procedural times and small injuries might mask PE development. After cardiac drainage and OAC therapy reversal, the bleeding seemed controllable. Because we assumed catheter manipulations or the recapture maneuver injured the

FIGURE 3 Diagnosis of Free Wall Injury Caused by Tines



(A) 3-dimensional reconstruction short-axis image of electrocardiogram-gated enhanced cardiac computed tomography. Black arrow indicates leadless pacemaker. (B) The volume-rendered image of the leadless pacemaker position (RAO 30°, LAO 30°, 45°, 60°). LV = left ventricle; RV = right ventricle; other abbreviations as in Figure 1.

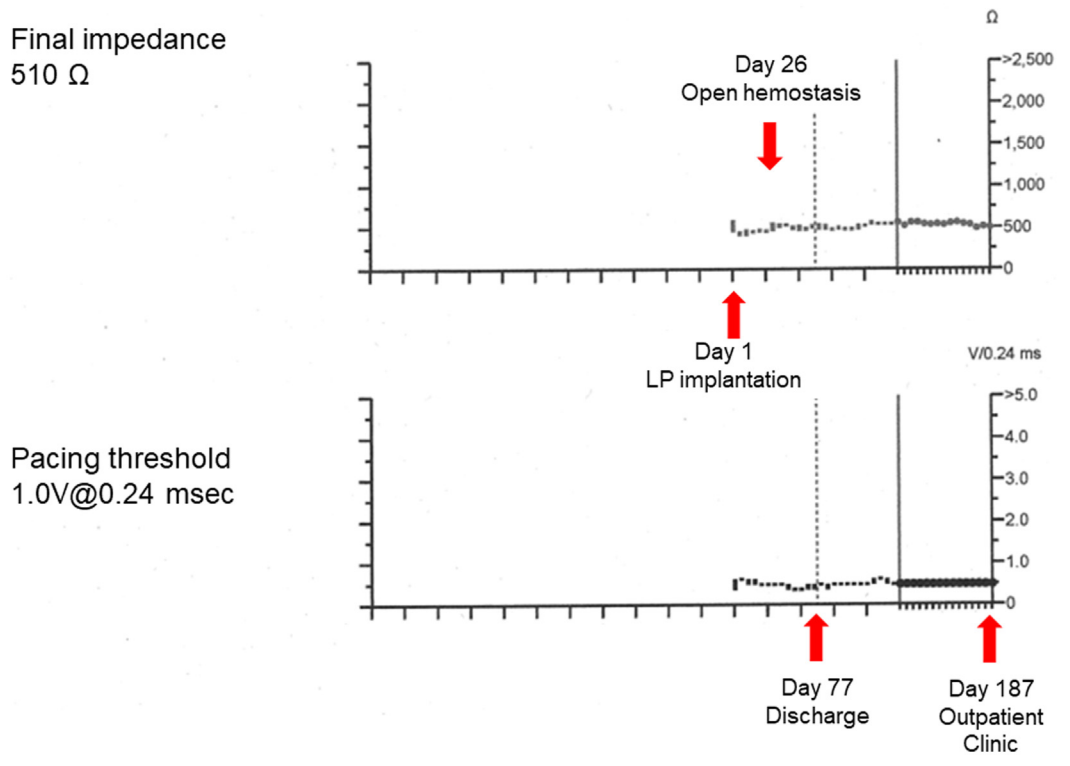
tissue,[7] we were late in detecting the RVFW injury. Immediate evaluations should be conducted to determine if the remaining LP tines injured the myocardium by ECG-gated enhanced cardiac CT.

We finally performed hemostatic surgery. On-pump LP removal may be the most reliable strategy, but it seemed extremely invasive considering his frailty. Therefore, we selected off-pump surgery with a hemisternotomy. After drainage of old bloody PE, the exposed tine seemed covered with healed tissue without active bleeding. In the first cardiac tamponade, the PE mechanism was considered to involve direct leakage from the RV cavity or epicardial vessels. In the later period, particularly around the time of the hemostatic surgery, the main mechanism of recurrent PE might involve hemorrhagic pericarditis. Because the LP parameters were sufficient for reuse, we achieved the hemostasis without on-pump LP removal. The surgical strategy should be discussed, considering the patient's general condition and the status of bleeding lesions.

FIGURE 4 Surgical View After Resecting the Pericardium

White arrow indicates leadless pacemaker tine.

FIGURE 5 The Trend of Impedance and Pacing Threshold



Upper: trend of lead impedance, Lower: trend of pacing threshold.

FOLLOW-UP

Although the patient developed urinary sepsis caused by cystitis after the surgery, he was discharged on day 77. The pacing parameters did not fluctuate post-implantation (Figure 5).

CONCLUSIONS

We present a case of delayed cardiac tamponade and recurrent PE caused by RVFW injury by LP tines. We reconsidered the management for safe LP implantation and the treatment for residual injury post-LP implantation.

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KEY WORDS cardiac tamponade, complication, fixation, leadless pacemaker, tines

APPENDIX For supplemental videos, please see the online version of this paper.