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CASE REPORT

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Delayed right ventricular perforation complicated by intracardiac thrombosis after implantable cardioverter-defibrillator implantation

Tsuyoshi Ito MD | Toshihiko Goto MD | Yu Kawada MD | Nobuyuki Ohte MD

Department of Cardio-Renal Medicine and Hypertension, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan

Correspondence
Tsuyoshi Ito, Department of Cardio-Renal Medicine and Hypertension, Nagoya City University Graduate School of Medical Science, Nagoya, Japan.
Email: tuyosiito@gmail.com

Abstract

Cardiac perforation and intracardiac thrombosis are infrequent complications after implantable cardioverter-defibrillator (ICD) implantation, and a case of lead perforation complicated by thrombosis is extremely rare. We report the case of a 66-year-old man with delayed ICD lead perforation concomitant with intracardiac lead thrombosis successfully treated by anticoagulant therapy followed by transvenous lead management.

KEYWORDS
anticoagulant therapy, implantable cardioverter-defibrillator, lead perforation, lead thrombosis, transvenous lead extraction

1 | INTRODUCTION

Cardiac perforation of implantable cardioverter-defibrillator (ICD) lead is an uncommon complication, with a reported incidence of 0.6%-5.2%.[1] In particular, delayed perforation occurring more than 1 month after implantation is infrequent and can be fatal. Treatment strategies are individualized according to the perforation type and timing, lead type, and tip position. Although the majority of perforating leads can be managed percutaneously, open heart surgery is required in high-risk cases.[2] Lead-associated intracardiac thrombosis detectable by transthoracic echocardiography (TTE) is relatively uncommon,[3] and serious embolic complications can occur.[4] Although anticoagulation therapy is the treatment option of choice, when a high risk of fatal pulmonary embolism is present due to the size and mobility of the thrombus, surgical intervention is considered.[5]

Here, we present a case of delayed ICD lead perforation concomitant with intracardiac lead thrombosis successfully treated by anticoagulant therapy followed by transvenous lead management.

2 | CASE REPORT

A 66-year-old man with no significant past medical history was referred to our emergency department for cardiogenic shock due to sustained ventricular tachycardia. Urgent cardioversion restored a sinus rhythm. After detailed examination, including coronary angiography, cardiac magnetic resonance imaging, gallium-67 scintigraphy, and endomyocardial biopsy, he was diagnosed with cardiac sarcoidosis. An ICD for secondary prevention (Iperia 7 DR-T; Biotronik, Berlin, Germany) using an atrial active fixation lead (Biotronik Solia S53) and ventricular active fixation lead (Biotronik Protego ProMRI s65) was successfully implanted through the left subclavian vein. The patient was discharged after initiation of corticosteroid therapy for cardiac sarcoidosis. Two months after ICD implantation, the patient visited our hospital due to sudden-onset stabbing chest pain with concomitant cough and shortness of breath. The chest X-ray revealed no significant change in the ventricular lead position. However, multislice computed tomography (CT) without contrast depicted the tip of the right ventricular (RV) lead located outside the myocardium, left-sided pneumothorax, pleural effusion, and pneumopericardium (Figure 1). Interrogation of the ICD showed a change in RV lead–related parameters compared to those of the previous day (pacing threshold: 0.9 mV/0.4 ms to 2.6 mV/0.4 ms; impedance: 406 ohms to over 3000 ohms; R-wave amplitude: 13.4 mV to 7.7 mV). TTE demonstrated a mobile mass possibly attached to the atrial lead within the right atrium (20 × 22 mm in diameter) prolapsing into the right ventricle (Figure 2A). The patient was afebrile with no clinical vascular and immunological phenomenon. Two sets of

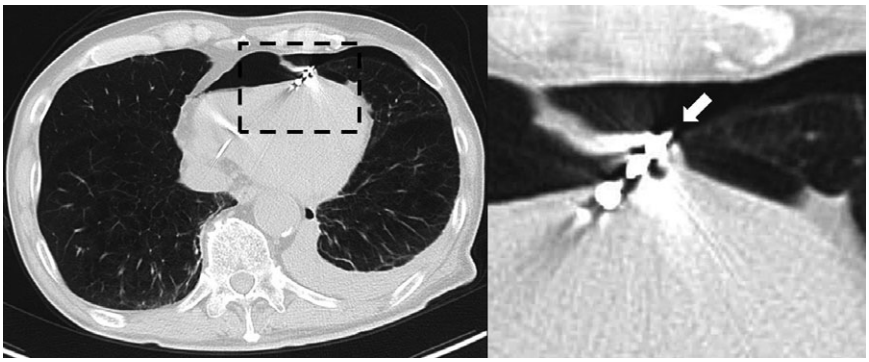

FIGURE 1 Computed tomography showing the tip of the right ventricular lead located outside the myocardium (arrow), left-sided pneumothorax, pleural effusion, and pneumopericardium

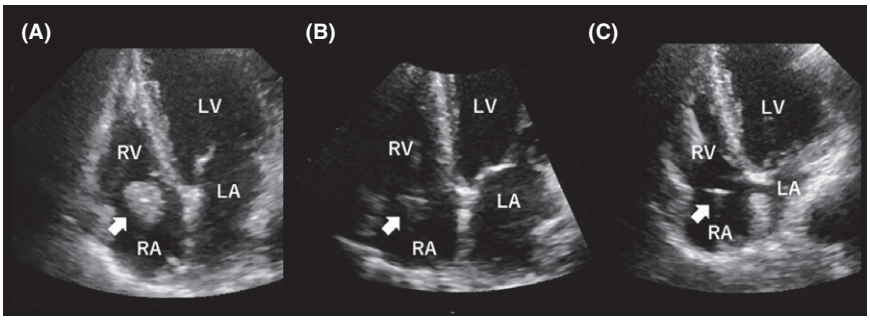

FIGURE 2 A, Transthoracic echocardiography (TTE) at admission demonstrating a mobile mass (arrow) attached to the atrial lead. B, TTE after 7 d of anticoagulation showing a decrease in the size of the mass (arrow). C, TTE after 3 mo of anticoagulation showing that a mass on the atrial lead (arrow) disappeared. LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle

blood cultures were negative, and a high level of D-dimer was observed (19.1 μg/mL). It was likely a thrombus. The systolic pulmonary artery pressure estimated from the peak tricuspid regurgitant velocity was normal (20 mm Hg), suggesting that a massive pulmonary embolism was not present. Pericardial effusion was not identified on CT or TTE. Based on these findings, we diagnosed delayed RV lead perforation with right atrial lead thrombus. A 7-day trial of intravenous heparin was performed, and the thrombus decreased in size without increasing pericardial effusion or the estimated pulmonary artery pressure (Figure 2B). Transvenous perforated lead extraction was performed under TTE guidance and surgical backup and a new RV lead implanted without any complications. The postprocedural course was uneventful and the thrombus disappeared after 3 months of oral anticoagulant treatment (Figure 2C).

3 | DISCUSSION

A case of delayed right ventricular perforation concomitant with intracardiac thrombosis has not been reported previously, and the appropriate management is uncertain. TEE findings were suggestive of infectious endocarditis, but the patient had no clinical signs of infection and vascular and immunological phenomenon with negative blood culture. Thus, the mass was thought to be a thrombus rather than vegetation. Hypercoagulable state due to steroid use and inflammatory response to cardiac perforation might be a cause of thrombus formation. In our case, surgical lead extraction and thrombus removal could be considered to control bleeding and avoid thrombus embolization, but the procedure is invasive and infectious complications associated with corticosteroid use are concerned. Considering the stable hemodynamic state with no evidence of pericardial effusion and pulmonary embolism, we decided to start less invasive treatment. Although a risk of cardiac tamponade caused by anticoagulation and transvenous lead extraction was present, we were able to successfully treat the patient under careful monitoring and surgical backup. If pericardial effusion developed in the course of treatment, surgical rescue might be better. Considering a risk of bleeding associated with anticoagulation and a low incidence of clinical pulmonary embolism related to lead-related thrombus,[3] lead extraction before anticoagulation therapy is an alternative strategy. In the case of lead perforation concomitant with intracardiac thrombus, individualized management is important. Anticoagulant therapy with careful monitoring and transvenous lead management can be a choice of treatment.

CONFLICT OF INTEREST

Authors declare no Conflict of Interests for this article.

ORCID

Tsuyoshi Ito http://orcid.org/0000-0002-1871-0954

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