

Real-Time Embolization of a Massive Lead-Associated Thrombus Visualized by Transesophageal Echocardiography



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INTRODUCTION

Lead-associated thrombus is commonly observed in patients with transvenous cardiac implantable electronic devices (CIEDs). Rates of clinically apparent pulmonary embolism in the presence of lead-associated thrombus are generally low, but pulmonary embolism is an underappreciated potential cause of morbidity.

We present a case of a massive lead-associated thrombus that was observed on a transesophageal echocardiogram obtained in a patient who had undergone resuscitation from cardiac arrest. The images we present are remarkable in that they capture the embolization and transit, in real time, of the thrombus from the pacemaker lead. This case highlights the thrombogenicity of transvenous CIEDs as well as the potential for associated morbidity and mortality.

CASE PRESENTATION

An 80-year-old man with a history of coronary artery disease who underwent coronary artery bypass graft surgery 10 years prior, ischemic cardiomyopathy with an ejection fraction of 25%, and placement of a Biotronik dual-chamber implantable cardioverter-defibrillator 3 months prior presented to the emergency department after a witnessed cardiac arrest at home. Bystander cardiopulmonary resuscitation was performed, and emergency medical personnel found the patient to be in a shockable rhythm upon their arrival. Return of spontaneous circulation was achieved in the field after internal shocks by the patient's device and by external defibrillation, with a total "down time" of 5 min. On initial arrival to the hospital, the patient was obtunded, and he was intubated for airway protection. The patient was afebrile, with a blood pressure of 115/88 mm Hg and a heart rate of 84 beats/min. Electrocardiography showed a sequential atrial-ventricular paced rhythm with a nonspecific ST-T abnormality. Initial laboratory studies were notable for creatinine of 2.0 mg/dL (unknown baseline), blood urea nitrogen of 33 mg/dL, sodium of 135 mmol/L, potassium of 5.0 mmol/L, chloride of 102 mmol/L, bicarbonate of 20 mmol/L, lactate of 2.8 mmol/L, aspartate transaminase of 596 U/L, alanine transaminase of 580 U/L, total bilirubin of 0.5 mg/dL, alkaline phosphatase of 78 U/L, a white blood cell count of 3,100/ μ L, hemoglobin of 10.3 g/dL, and a platelet count

of 79,000/ μ L. Initial serum troponin I was 0.08 ng/mL and peaked at 0.53 ng/mL.

Cooling measures were initiated, and the patient was transported to the intensive care unit. Interrogation of the defibrillator confirmed a primary ventricular arrhythmia at the time of the initial arrest. There was no observed or history of atrial fibrillation. Transthoracic echocardiography revealed severe global left ventricular systolic dysfunction, new-onset severe right ventricular dysfunction, and a mobile echodensity in the right atrium (Figure 1). The right ventricular-to-right atrial systolic pressure gradient was estimated at 31 mm Hg. Transesophageal echocardiography was subsequently performed and revealed a large multilobulated thrombus in the right atrium measuring 4 cm in largest diameter and adherent to the right atrial pacemaker lead (Figure 2). The thrombus was highly mobile, with intermittent prolapse through the tricuspid valve (Videos 1–3). During the acquisition of transesophageal echocardiographic images, the thrombus detached and embolized into the pulmonary circulation (Video 4). Immediate hemodynamic deterioration was not observed. Residual thrombus was seen attached to the right atrial lead measuring >2 cm in greatest dimension (Figure 3). Systemic anticoagulation, which had not been prescribed in the outpatient setting, was then initiated. The patient subsequently underwent urgent catheter thrombectomy using the AngioVac catheter extraction system (AngioDynamics, Latham, NY) under venovenous extracorporeal membrane oxygenation support. A large amount of thrombus adherent to the right atrial lead was successfully removed and was confirmed by pathologic examination. Specimen and peripheral blood cultures showed no bacterial growth. Pulmonary angiography revealed only distal defects in pulmonary circulation (Figure 4), and embolectomy was not performed. The patient returned to the intensive care unit in critical condition, but ultimately his condition did not improve. Despite maximal supportive medical care, gradual hemodynamic deterioration was observed, and the patient could not be resuscitated.

DISCUSSION

Lead-associated thrombus is commonly observed in patients with transvenous CIEDs. Although adherent thrombi are seen by transthoracic echocardiography in only 1.4% of patients with CIEDs,¹ thrombi are seen by intracardiac echocardiography in 30% of patients with CIEDs presenting for ablation procedures, in only 4% of whom is the thrombus also seen on transthoracic echocardiography.² Incidental lead-associated masses have been reported in 14% of patients undergoing transesophageal echocardiography, 72% of which are ultimately deemed noninfectious.³ The average size of these thrombi has been reported to be 1.8 \times 0.4 cm.² The most significant known risk factor for lead-associated thrombus is atrial fibrillation, which is associated with an eightfold increased incidence,¹ but low-flow states, cardiomyopathy, and underlying thrombophilic disorders may also contribute. Silicone-based pacemaker lead insulation, as

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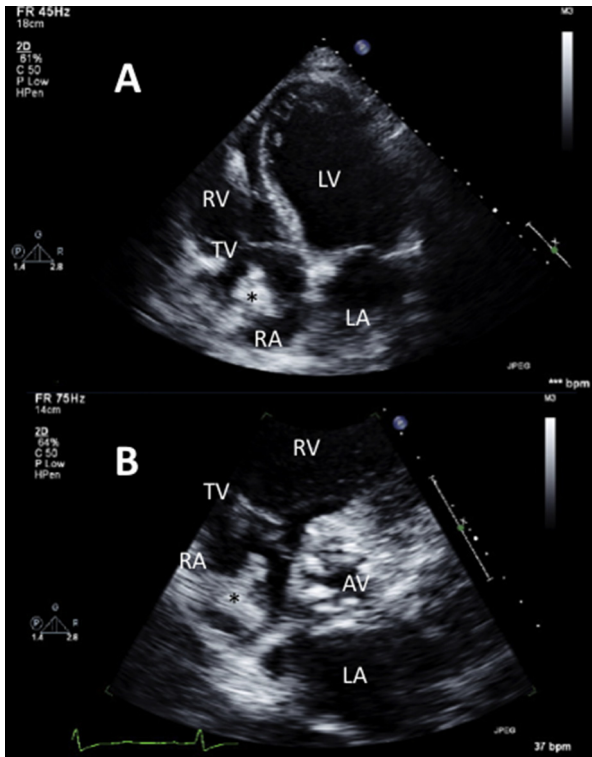


Figure 1 Transthoracic echocardiography revealed a large echodensity consistent with thrombus (asterisk) in the right atrium (RA), seen in the apical four-chamber view (**A**) and basal short-axis view (**B**). AV, Aortic valve; LA, left atrium; LV, left ventricle; RV, right ventricle; TV, tricuspid valve.

present in this case, may have some degree of intrinsic thrombogenicity; however, modern pacemaker leads commonly also have a non-thrombophilic polymer coating. Rates of clinically apparent pulmonary embolism in the presence of lead-associated thrombus are low, with an incidence of 3.32 per 1,000 person-years and an overall rate of 1.6%, and the majority of such patients (84%) demonstrate another established risk factor for pulmonary embolism.⁴ However, it is likely that microthrombi may occur, as the presence of lead-associated thrombus is associated with elevated pulmonary artery systolic pressures.²

In this case, there was no history of atrial fibrillation, but the thrombus was much larger than typically reported. It is possible that the patient's electromechanical dissociation could have contributed to generation of thrombus associated with the lead and that chest compressions could have disrupted the thrombus and increased the risk for embolization. However, more likely, the large thrombus attached to the lead was present before the arrest, spontaneously embolized, and led to massive pulmonary embolism and cardiac arrest.

CONCLUSION

This case highlights the potential for morbidity and mortality associated with transvenous CIEDs. Although clinical embolic phenomena are infrequently observed from known lead-associated thrombi, this case demonstrates that massive pulmonary embolism can occur. Continued development and expanding use of leadless CIEDs will reduce this underappreciated complication of transvenous systems. Finally, to our knowledge, this is the first reported case in which transesophageal echocardiography captured in real time the embolization of a large thrombus associated with a pacemaker lead.

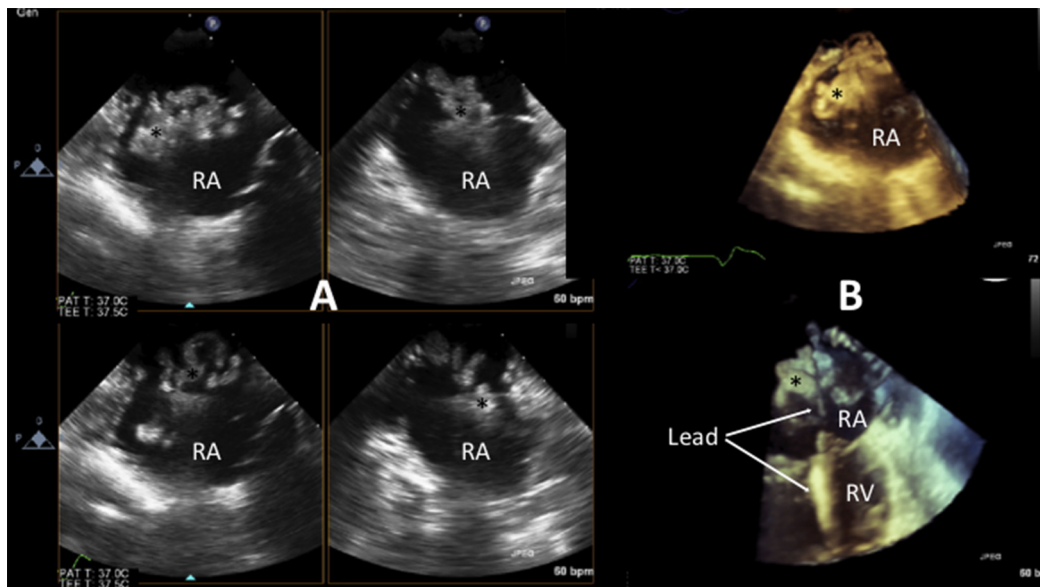


Figure 2 Transesophageal echocardiography revealed a large, mobile, multilobulated echodensity consistent with thrombus (asterisk) adherent to the right atrial pacemaker lead, seen on midesophageal two-dimensional X-plane views (**A**) and three-dimensional views (**B**) of the right atrium (RA). RV, Right ventricle.

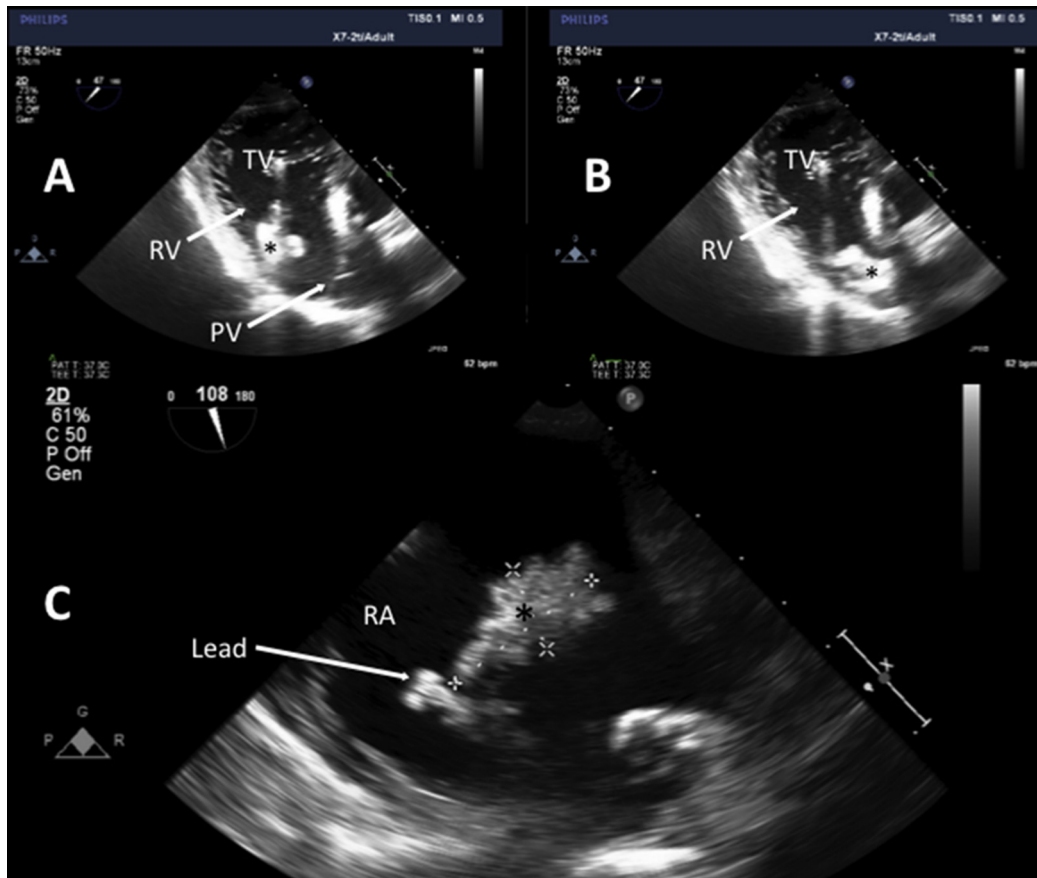


Figure 3 Transgastric views of the right ventricle (RV) on transesophageal echocardiography demonstrated real-time embolization of the large echodensity consistent with thrombus (asterisk) through the right ventricular outflow tract and across the pulmonic valve (PV) (A,B). Subsequent midesophageal view of the right atrium (RA) on transesophageal echocardiography demonstrated a large residual thrombus measuring 2.8×1.4 cm, which remained adherent to the pacemaker lead after embolization (C). TV, Tricuspid valve.



Figure 4 Pulmonary angiography revealed distal embolization of thrombus (white arrow) in the right pulmonary circulation but no proximal central thrombus.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2018.02.003>.

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