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Complete Resolution of Pacemaker Lead Thrombus with Warfarin Following Direct Oral Anticoagulant Failure: A Case Report

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ABSTRACT/INTRODUCTION

Pacemaker lead thrombosis is a recognized complication of cardiac implantable electronic devices (CIEDs) that occurs due to endothelial trauma and increased blood turbulence caused by the foreign material of the pacemaker leads themselves [1]. The incidence of intracardiac thrombi associated with CIEDs ranges from 1.4% to 30% depending on the imaging modality and patient population studied, with the highest rates identified incidentally during lead extraction or ablation procedures [1,2]. While many cases remain clinically silent, symptomatic pacemaker lead-associated thrombosis can present with nonspecific symptoms and carries serious potential complications including pulmonary embolism and systemic thromboembolism, necessitating prompt diagnosis and treatment [1,2].

Anticoagulation therapy has emerged as the mainstay of management for most intracardiac pacemaker lead thromboses [3]. Current evidence indicates that warfarin prophylaxis lowers the incidence of venous lesions in high-risk patients, particularly those with left ventricular ejection fraction of 40% or less or multiple pacemaker leads [1,4]. Additionally, warfarin therapy has demonstrated efficacy in resolving established pacemaker lead thrombi, with resolution rates of 75 to 80% in patients initiated on anticoagulation [5]. However, there remains a notable gap in the literature regarding the management of patients with pacemaker lead thrombosis who develop thrombus while on DOAC therapy and require transition to alternative anticoagulation. This case report contributes to this body of knowledge by documenting the successful resolution of pacemaker lead thrombosis following transition from rivaroxaban to warfarin therapy.

Keywords: Pacemaker lead thrombus; Anticoagulation failure; Warfarin; Rivaroxaban; Cardiac implantable electronic device (CIED); Transesophageal echocardiography (TEE); Atrial fibrillation

CASE PRESENTATION

A 73-year-old male with a medical history notable for type 2 diabetes mellitus, hypertension, permanent atrial fibrillation, and nephrolithiasis, with a single-chamber ventricular pacemaker implanted seven months prior for symptomatic bradycardia, presented to the emergency department in June 2025 with one day of left arm pain, swelling, and erythema. He had been taking rivaroxaban for stroke prevention in the setting of atrial fibrillation but had stopped the medication on his own approximately one week before this presentation. He denied fever, chills, shortness of breath, hematuria, and neurological symptoms.

EKG on arrival showed a ventricular paced rhythm. Duplex ultrasound of the left upper extremity identified occlusive deep vein thrombosis of the subclavian, axillary, and brachial veins, with additional left basilic superficial thrombophlebitis. The left upper extremity DVT was attributed to venous outflow obstruction from the ipsilateral pacemaker lead, a well-recognized mechanical complication of transvenous device implantation. Transthoracic echocardiography was performed and identified a mobile echodensity on the pacemaker lead within the right atrium. Cardiology was consulted.

Transesophageal and transthoracic echocardiography confirmed multiple mobile echogenic structures attached to the right atrial pacemaker lead **(Figures 1,2)**. The differential was thrombus versus vegetation. Blood cultures were collected on multiple occasions and returned negative. The patient had no fever and remained hemodynamically stable throughout. While infective endocarditis could not be categorically excluded, the clinical picture favored thrombus.

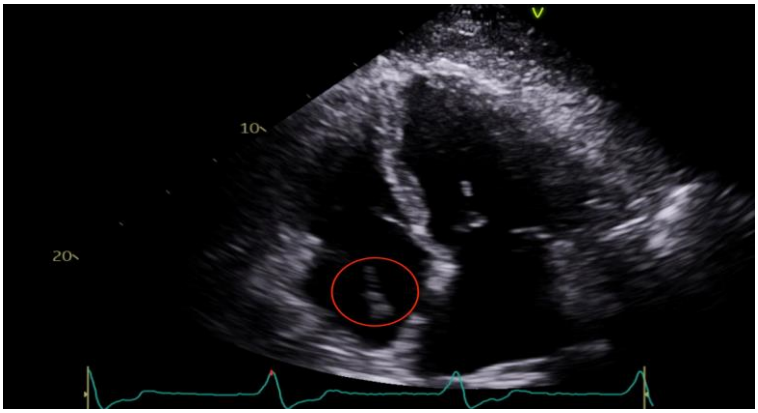

Figure 1: Transesophageal echocardiogram (TEE) in the apical four-chamber view demonstrating a mobile, echogenic mass within the right atrium (RA). The mass is adhered to the indwelling pacemaker lead, consistent with an atrial thrombus

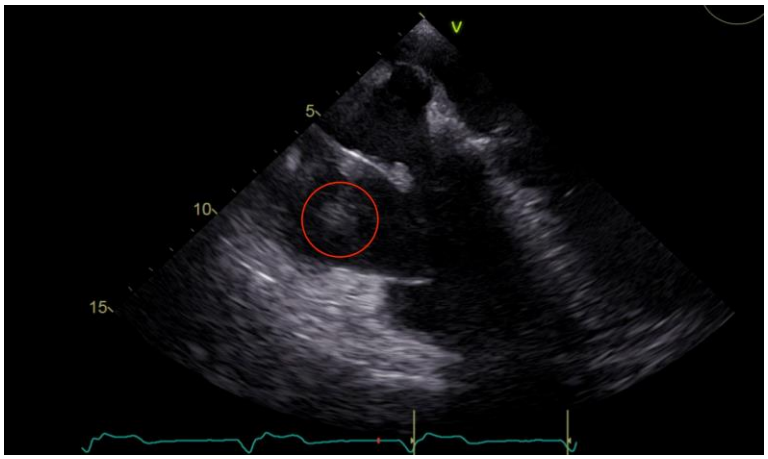

Figure 2: Transthoracic echocardiogram (TTE) in the apical four-chamber view revealing a large, well-circumscribed echogenic mass (thrombus) within the right atrium (RA). The mass is closely associated with the pacemaker lead (red circle), consistent with lead-induced thrombosis

The patient was admitted from June 16 to June 24, 2025. The thrombus was felt to have developed in the setting of recent rivaroxaban discontinuation combined with a prothrombotic milieu of atrial fibrillation and an intracardiac foreign body. Given this context, and the cardiology team's judgment that resuming rivaroxaban carried unacceptable risk of recurrence, anticoagulation was transitioned to warfarin with enoxaparin bridging. Anticoagulation was switched to warfarin with enoxaparin bridging, with a target INR of 2 to 3. He was discharged with follow-up through the anticoagulation clinic and cardiology. Repeat TEE was scheduled for four to six weeks out, and pacemaker explantation was discussed as a contingency if the masses persisted.

Management and follow-up

The patient established care in the anticoagulation clinic on August 25, 2025. His daughter, who serves as his primary caretaker, attended the visit and confirmed adherence to warfarin 5 mg daily. He had no missed doses, no dietary changes that could affect INR stability, no new medications, and no signs of bleeding or bruising. Repeat left upper extremity duplex ultrasound from two days prior showed the left brachial vein thrombus had converted from occlusive to nonocclusive, a meaningful improvement from the admission study. INR that day was 2.70.

Warfarin was continued at the same dose and a follow-up INR was scheduled for late September.

Repeat TEE was performed on August 26, 2025, roughly ten weeks after the original diagnosis. Follow-up imaging showed no evidence of continued lead-associated thrombus along its course through the right atrium and into the right ventricle. The mobile echogenic structures seen in June were gone **(Figure 3)**. Left ventricular ejection fraction was preserved at 55%. The rhythm was atrial fibrillation with ventricular pacing at 60 bpm. No thrombus or mass was identified on either lead. The findings represented complete echocardiographic resolution.

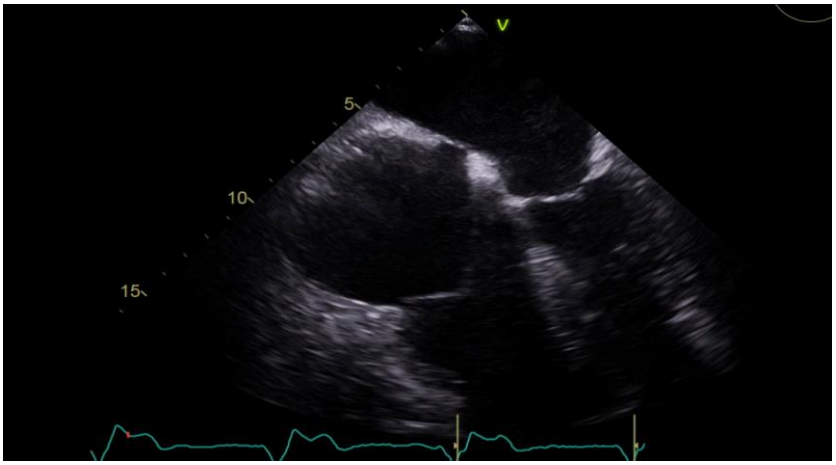

Figure 3: Follow-up transthoracic echocardiogram (TTE) in the apical four-chamber view demonstrating complete resolution of the previously visualized right atrial thrombus. The pacemaker lead is clearly visible and appears free of any echogenic masses or vegetations following ten weeks of anticoagulation therapy

At cardiology clinic follow-up on September 2, 2025, the patient reported gradual improvement in his functional capacity. He was walking around 30 minutes before needing rest. Given the full resolution on TEE, long-term warfarin was recommended. Resuming rivaroxaban was not considered, as the thrombus had formed during active therapy and repeating that approach carried obvious risk. The pacemaker itself required no procedural intervention, and routine device surveillance was continued.

At his December 2025 visit, the pacemaker lead remained free of any mass and anticoagulation was ongoing without complication.

DISCUSSION

Pacemaker lead thrombus is an underrecognized complication of transvenous cardiac implantable electronic device placement, with reported prevalence ranging from 1.4% to 30% across echocardiographic series, owing largely to the high proportion of asymptomatic presentations [6]. Notably, studies using TEE to screen CIED patients have found non-infectious lead-associated masses in 20% to 27.6% of cases, with oral anticoagulation consistently associated with lower odds of these findings, suggesting a protective role for therapeutic anticoagulation in this population [11,12]. The pathogenesis is attributed to endothelial disruption along the lead surface, inciting a foreign-body inflammatory response with subsequent fibrin deposition and thrombus propagation in the low-flow environment of the right atrium [1]. The pathogenesis is attributed to endothelial disruption along the lead surface, inciting a foreign-body inflammatory response with subsequent fibrin deposition and thrombus propagation in the low-flow environment of the right atrium [1].

A central diagnostic challenge in these cases is distinguishing lead thrombus from infective endocarditis, as echogenic masses on pacemaker leads are morphologically indistinguishable between the two entities on imaging alone[7]. Serial negative blood cultures and a persistently afebrile clinical course, as observed in this

patient, support a thrombotic rather than infectious etiology, though endocarditis cannot be categorically excluded on echocardiographic criteria alone [7].

The coexistence of atrial fibrillation and an intracardiac foreign body creates a high-risk thrombotic milieu, and even brief interruptions in anticoagulation may be sufficient to precipitate lead-associated thrombus formation in susceptible patients[5]. In the present case, thrombus was identified following a self-reported one-week lapse in rivaroxaban. Rather than resume the same agent, the clinical team elected to transition to warfarin given its more predictable anticoagulation profile and prior evidence supporting its efficacy in resolving lead-associated thrombus [5,8]. Complete echocardiographic resolution was subsequently achieved, consistent with this approach.

Anticoagulation is the established first-line treatment for pacemaker lead thrombus in hemodynamically stable patients without confirmed device infection [6]. Complete resolution with vitamin K antagonist therapy has been documented in prior case reports, reinforcing that conservative management can be effective and durable when the clinical picture is favorable [8,9]. In this patient, the lead-associated thrombus carried clinical significance beyond an incidental imaging finding, given its temporal association with symptomatic upper extremity DVT, the presence of multiple mobile echogenic structures confirmed on both TTE and TEE, and the cardiology team's active consideration of device explantation as a contingency. Its complete resolution with warfarin therefore carried direct management implications, sparing the patient a procedure with known procedural mortality risk [10]. Transvenous lead extraction carries non-trivial procedural risk, with reported 30-day mortality as high as 3.4% even at high-volume centers, and long-term mortality significantly elevated in elderly patients and those with comorbidities, making avoidance of extraction a meaningful clinical benefit [10].

This case contributes to the limited literature supporting conservative anticoagulation as definitive management for pacemaker lead thrombus, demonstrates complete echocardiographic resolution with warfarin following documented DOAC failure, and highlights the importance of serial TEE in guiding treatment decisions and determining when device explantation can safely be deferred.

CONCLUSION

This case demonstrates that pacemaker lead thrombus can resolve completely with vitamin K antagonist therapy alone, without the need for device explantation. The development of thrombus following brief rivaroxaban discontinuation in a patient with atrial fibrillation and an intracardiac foreign body underscores the vulnerability of this population to even short lapses in anticoagulation. Warfarin, with its more predictable anticoagulation profile, achieved full echocardiographic resolution within ten weeks and remained effective at long-term follow-up. Serial TEE proved essential in monitoring treatment response and guiding the decision to defer extraction. Clinicians should consider early transition to warfarin when pacemaker lead thrombus develops on DOAC therapy, and conservative management should be the initial approach in hemodynamically stable patients before pursuing the higher-risk option of lead extraction.

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