Massive upper extremity deep venous thrombosis after a transvenous lead extraction successfully treated by an anti-thrombotic regimen

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Introduction

The implantation of cardiac implantable electronic devices (CIEDs) is increasing to improve the survival and quality of life.[1] This also has increased the number of infections and dysfunctions related to CIEDs that require the removal of the devices.[2] Complications of transvenous lead extractions (TLEs) include vascular lacerations, pericardial effusions, hemothoraces, and venous thromboses. Among them, the incidence, risk factors, and long-term impact of venous thromboses are still unclear.[3]

We present a case of an upper extremity deep venous thrombosis after a TLE that was successfully treated with conservative treatment and careful monitoring. The clinical course, treatment decision-making process, and the images of the thrombus regression in response to the treatment are presented.

KEY TEACHING POINTS

- The incidence of an upper extremity deep venous thrombosis after a transvenous lead extraction (TLE) is reported to be 2.5%.
- Risk factors include implantable cardioverter defibrillator leads, older leads, the use of laser and powered mechanical sheaths, infections, and a left anterior chest implantation.
- Symptoms of an upper extremity deep venous thrombosis are pain, swelling, and fatigue. Functional impairment of the affected upper extremity can occur.
- Possible treatments include anticoagulation, thrombolysis, thrombus aspiration, and balloon and stent angioplasty.
- This was a case of an upper extremity deep venous thrombosis after a TLE that was successfully treated with anticoagulation.

Case report

An 82-year-old man developed a CIED pocket infection and was referred to our hospital for an extraction of the whole CIED system. Sixteen years before, the patient underwent a dual-chamber implantable cardioverter defibrillator (ICD) implantation in the left anterior chest for the primary prevention of sudden cardiac death associated with hypertrophic cardiomyopathy (ELLIPSE DR with IsoFLEXS 1642T atrial tined lead and Riata 1570 dual coil ventricular tined lead, St Jude Medical, Minnesota, MN). The ICD generator was replaced for the first time 10 years before and for the second time 1 year before. The patient had persistent atrial fibrillation, hypertension, and diabetes mellitus and took rivaroxaban 15 mg, candesartan 8 mg, azelnidipine 16 mg, and sitagliptin 50 mg. On admission, his blood pressure was 118/46 mm Hg, heart rate 70 beats/min, respiratory rate 16 breaths/min, body temperature 35.9 °C, and oxygen saturation 98% on room air. There was redness, swelling, and tenderness in the skin surrounding the generator. A blood test showed mildly elevated C-reactive protein (0.28 mg/dL) and brain natriuretic peptide (150 pg/dL) levels, and the coagulation-fibrinolytic system was normal. The blood cultures were negative. A 12-lead electrocardiogram showed ventricular pacing with atrial fibrillation. Chest radiography indicated cardiomegaly without pulmonary congestion or a pleural effusion (Figure 1A). A 3-dimensional (3D)

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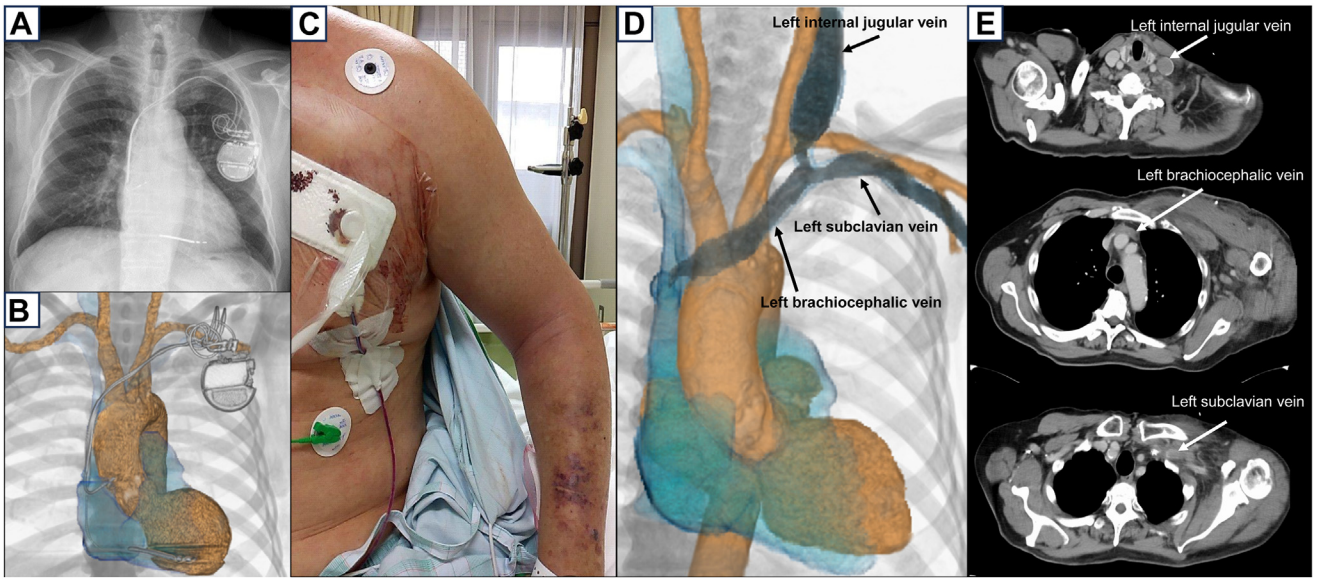

Figure 1 Physical and examination findings of the patient before and after the cardiac implantable electronic device extraction. **A:** Chest radiography on admission. **B:** This shows the 3-dimensional (3D) constructed computed tomography (CT) image on admission. The orange depicts the arterial vessels, and the light blue depicts the venous vessels. **C:** Significant swelling of the upper extremity deep venous thrombosis the day after the cardiac implantable electronic device extraction. **D:** This shows the 3D-constructed image of a thrombus by contrast-enhanced CT. Orange depicts the arterial vessels, light blue depicts the venous vessels, and dark gray depicts the thrombus in the veins. A thrombus is seen from the left brachiocephalic vein to the internal jugular and subclavian vein. **E:** Thrombosis detected by contrast-enhanced CT in the left internal jugular, brachiocephalic, and subclavian veins (*arrow*).

constructed image by computed tomography (CT) showed the leads' trajectories and tip location (Figure 1B). Transthoracic echocardiography showed concentric left ventricular hypertrophy with a normal left ventricular function and dilated left atrium, and transesophageal echocardiography showed no obvious vegetations.

On the 7th day of hospitalization, a CIED extraction was performed.[3] Renal function was preserved with a creatinine clearance of 62.7 mL/min, so rivaroxaban was stopped 24 hours before the procedure. The procedure was performed in a hybrid operating room under general anesthesia with continuous electrocardiographic and arterial blood pressure monitoring, using transesophageal echocardiography guidance. Standby cardiac surgery including the availability of extracorporeal circulation and a perfusionist for the treatment of emergency complications was always available. A stiff guidewire from the right femoral vein to the right internal jugular vein was available for the potential use of a bridge occlusion balloon (Philips-Spectranetics, Colorado Springs, CO) in cases of vascular lacerations. A locking stylet (Liberator® Beacon® Tip Locking Stylet, Cook Medical, Bloomington, IN) was inserted into the inner lumen of the atrial lead and a normal stylet into the inner lumen of the ventricular lead because the locking stylet could not be inserted into the tip of the lead. An 11-F Evolution Shortie RL sheath (Cook Medical) was advanced to the midclavicular line of each atrial and ventricular lead, and the adhesions were dissected. Dissection of the atrial lead proceeded using an 11-F Evolution RL sheath with a steady sheath (Cook Medical), and the atrial lead could be extracted. Next, a 14-F excimer laser sheath (GlideLight™, 80 Hz, Spectranetics Corporation) was used to dissect the adhesions of the ventricular lead from the entry to the superior portion of the superior vena cava. Tight fibrous adhesions surrounding the superior vena cava coil were dissected using the 13-F Evolution RL rotational sheaths with a steady sheath, and the ventricular shock lead was completely extracted. Compression, electrocautery, and suture ligation of the left subclavian vein were required for active bleeding. The total bleeding volume was 1450 mL. He was then extubated and returned to the intensive care unit. The operation and anesthesia time were 3.25 and 5.25 hours, respectively. A blood transfusion was not indicated because the hemoglobin level was 9.7 g/dL after the surgery.

Six hours after the surgery, swelling of the left upper extremity was noted (Figure 1C). The day after the surgery, the D-dimer level was elevated to 6.9 μg/mL, and contrast-enhanced CT showed a thrombosis from the left brachiocephalic to the internal jugular and subclavian vein (Figure 1D and 1E). The CT showed a hematoma in the CIED pocket but no active bleeding and no apparent bleeding in the mediastinum or pericardial sac. Echography also showed nonmobile thrombosis filling the left internal jugular, brachiocephalic, and subclavian veins (Figure 2A1, B1, and C1).

Treatment Strategy

Possible treatments for an upper extremity deep vein thrombosis after the TLE include anticoagulation, thrombolysis, thrombus aspiration, and balloon and stent angioplasty.[4,5] Anticoagulation can be initiated early after the thrombus onset if active bleeding is absent. However, anticoagulation alone may not prevent thrombus development in the acute

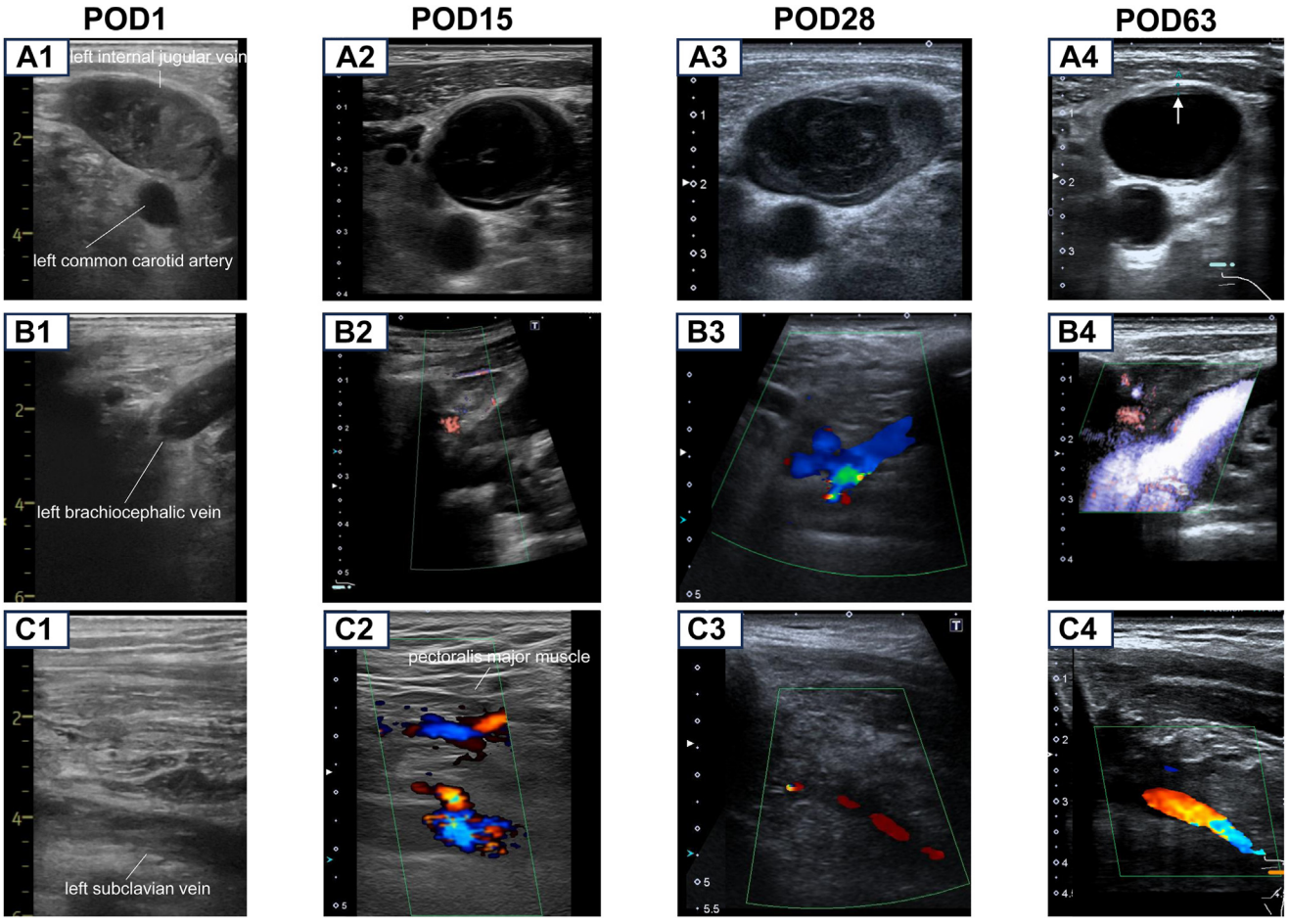

Figure 2 Echography images of a thrombus and its regression in the left internal jugular (**A**), brachiocephalic (**B**), and subclavian veins (**C**). Echography images of a thrombus and its regression in the left internal jugular (**A**), brachiocephalic (**B**), and subclavian veins (**C**) are shown. On postoperative day (POD) 1, the thrombus filled these veins (A1, B1, and C1), and decreased gradually on PODs 15 and 28 (A2/3, B2/3, and C2/3). Thereafter, the thrombus had almost disappeared on POD 63 (A4, B4, and C4), and only a mural thrombus was present in the left internal jugular vein (A4, *arrow*). POD = postoperative day.

phase. Catheter-directed thrombolysis would be recommended rather than systemic thrombolysis to reduce the risk of bleeding.[6] Thrombus aspiration is expected to reduce the risk of bleeding because it can be performed without thrombolytics.[5] Conversely, major bleeding has been reported in 4% of patients undergoing percutaneous mechanical thrombectomy.[7] Furthermore, thrombus aspiration can also cause pulmonary embolisms. Stenting of an acute upper extremity deep vein thrombosis is not usually used because of the high risk of a stent fracture.[8] The combination of balloon angioplasty and catheter-directed thrombolysis is reported to have better outcomes compared with catheter-directed thrombolysis alone.[9] Considering all the advantages and disadvantages of these treatments by cardiac surgeons and interventionalists, we decided to perform anticoagulation in this case.

Continuous intravenous heparin was started 24 hours after the surgery, and its dosage was adjusted to maintain the activated partial thromboplastin time at 1.5 to 2.0 times the control (Figure 3). Echography was performed on postoperative days (PODs) 1, 15, and 28, and the thrombosis had gradually decreased (Figure 2 and Supplementary Video). On POD 25, the D-dimer level had decreased to 2.8 μg/mL, the heparin was stopped, and rivaroxaban 15 mg was started. Because he did not have any ventricular tachyarrhythmia events during the ICD dwelling period, he was discharged on POD 30 without the re-implantation of the ICD.

At the follow-up visit on POD 63, the swelling in the left upper extremity had disappeared. The D-dimer level had decreased to 1.8 μg/mL. The thrombosis in the internal jugular vein had almost disappeared, and the thrombosis in the brachiocephalic and subclavian vein had disappeared (Figure 2A4, 2B4, 2C4, and Supplementary Video). There have been no recurrences of the thrombosis.

Discussion

The incidence of an upper extremity deep venous thrombosis after a TLE is reported to be 2.5%.[10] Each treatment approach has advantages and disadvantages regarding the safety, therapeutic intensity, and complications. Our patient was successfully treated with anticoagulation alone. However, if the thrombus formation or symptoms had worsened, thrombolytic therapy, thrombus aspiration, and angioplasty would

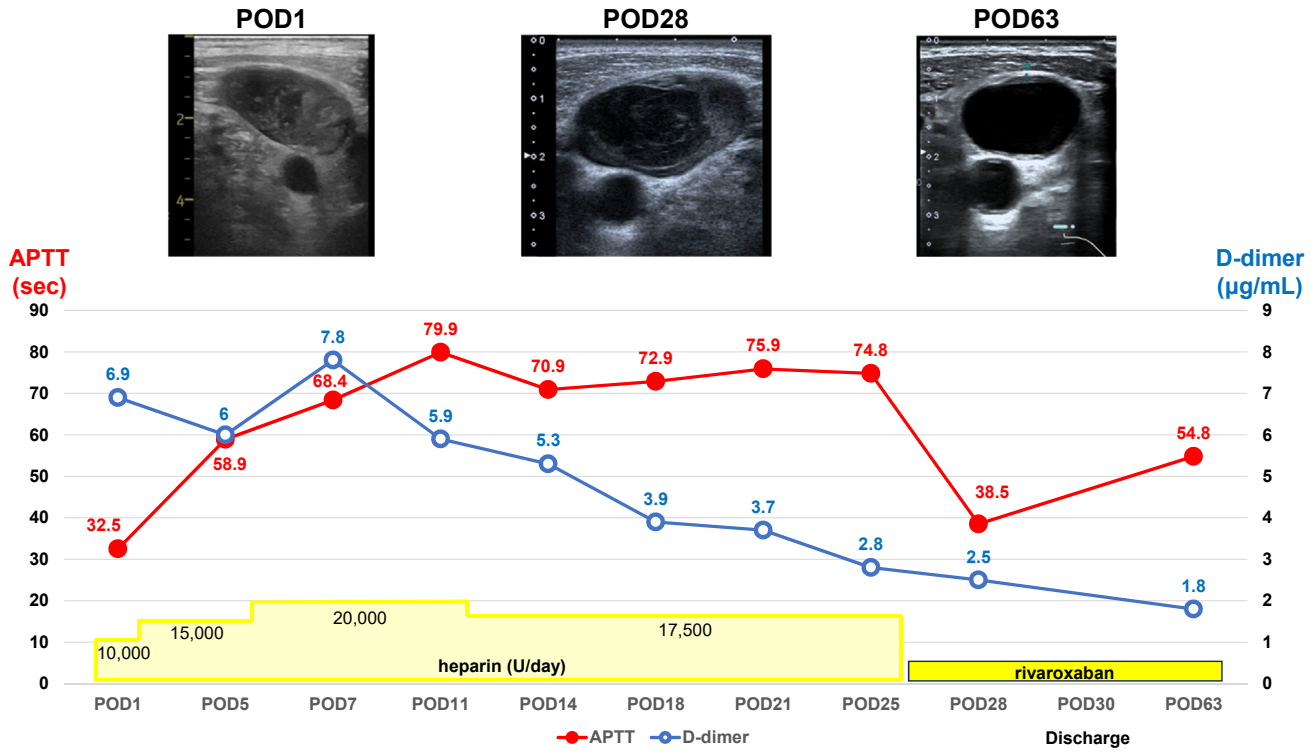

Figure 3 Overview of the clinical course after the cardiac implantable electronic device extraction. The upper figures show the thrombus on echography of the left internal jugular vein on postoperative day (POD) 1, 28, and 63, which depicts a gradual thrombus regression. The lower graph shows the patient's treatment course and trend of the activated partial thromboplastin time (APTT) value on the left Y-axis and D-dimer on the right Y-axis. On POD 25, the intravenous heparin administration was stopped, and oral rivaroxaban 15 mg was initiated. Thereafter, the patient was discharged on POD 30. APTT = activated partial thromboplastin time; POD = postoperative day.

be indicated, considering the risk of bleeding and a pulmonary embolism.

Regarding the pathogenesis of a venous thrombosis after a TLE, the remnants of the vascular and myocardial tissue in the extracted lead have been reported to injure the vascular system and form abrasive surfaces, and the exposure of these surfaces to blood flow causes thrombosis.[11] Venous injury tended to be observed in cases with older leads, ICD leads, and the use of larger-diameter-powered sheaths. Both systemic and local infections were associated with an increased risk of an access vein obstruction.[4] Furthermore, a left-sided lead placement is associated with a higher incidence and severity of a lead-related venous stenosis/obstruction.[12] A pocket infection occurred over 15 years after left-sided ICD implantation, requiring a larger-diameter-powered sheath for the TLE, and all those issues might have contributed to the postoperative thrombosis formation in this patient. The pocket hematoma also might have caused an impaired blood flow leading to thrombus formation. Anticoagulation during surgery would be a possible option to prevent a postoperative thrombosis; however, we believe it is impractical because TLE can be associated with fatal bleeding complications. Additionally, prophylactic antithrombotic therapy after a TLE in patients with a higher risk of a postoperative thrombus should be carefully discussed, considering the bleeding complications. A just and minimum-sized equipment selection can prevent collateral vessel injury, decrease vascular endothelial dysfunction, and reduce the risk of thrombus formation. An upper extremity deep venous thrombosis is a complication of a TLE that should be known.

Conclusions

We presented a case of an upper extremity deep venous thrombosis after a TLE for a CIED infection successfully treated with anticoagulation. Conservative treatment with anticoagulation would be a viable option that could reduce major bleeding and the occurrence of a pulmonary embolism.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.10.014.

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