Pacemaker Dysfunction due to a Large Thrombus on Ventricular Lead

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ABSTRACT

Background: Pacemaker lead–related thrombosis is a rare but severe complication in patients with pacing lead implantation in the right ventricle. We present a case with recurrent syncope after single-chamber implantable cardioverter defibrillator (ICD) implantation. Pacing lead–related thrombosis was observed during open-heart surgery. This induced intermittent pacemaker dysfunction and recurrent syncope.

Case Presentation: A 67-year-old male patient presented with frequent episodes of syncope and a history of dilated cardiomyopathy and paroxysmal ventricular tachycardia. Normal coronary angiography was found, and therefore a single-chamber ICD was implanted into the right ventricle to prevent cardiac events in 2013. However, he was referred to our hospital because of recurrent syncope 3 to 4 years after ICD implantation. A comprehensive investigation was performed to find the etiology for the recurrent syncope. Pacing lead thrombosis was finally observed during open-heart surgery, which can introduce intermittent pacemaker dysfunction. After the thrombus was removed and the lead was separated from the posterior leaflet of the tricuspid valve, the ICD functioned normally after reprogramming. Oral anticoagulant was prescribed after discharging. During the 1-year follow-up period, this patient was free of syncope.

Conclusions: This case illustrated that pacemaker lead–related thrombosis should be considered when the cardiac implantable electronic device fails to prevent patients from having cardiac events. Oral anticoagulant might be important for preventing thrombosis among patients with ICD implantation into the right ventricle.

BACKGROUND

The implantable cardioverter defibrillator (ICD) has been reported to carry a significant risk of complications, including lead displacement, pneumothorax, hematoma, and infection. [Ezzat 2015] Pacemaker lead–related thrombosis is a rare but severe complication. Implanting pacing and defibrillator leads is usually straightforward and is generally associated with a low risk of thromboembolic events. In addition, there is no need for routine anticoagulant therapy. Therefore, it has been widely performed in single-chamber ICD implantation.

We report a rare case of lead-related thrombosis after ICD implantation, which induced intermittent pacemaker dysfunction and recurrent syncope. After removal of the thrombi during open-heart surgery, the pacemaker function returned to normal when it was fully programmed.
**CASE PRESENTATION**

A 67-year-old male patient presented with frequent episodes of syncope and a history of dilated cardiomyopathy and paroxysmal ventricular tachycardia. Normal coronary angiography was found, and therefore a single-chamber ICD was implanted into the right ventricle to prevent sudden cardiac death in 2013. However, he was referred to our hospital because of recurrent syncpe 3 to 4 years after ICD implantation.

Sinus bradycardia, poor cardiac pacing, and intermittent long intervals of 4.36 seconds were observed in the ambulatory electrocardiogram monitoring (Figure, A). When we programmed the pacemaker parameters, they were 5.0 V for threshold, 1.6 mV for sensing, 48 Ω for defibrillation coil impedance. The battery capacity of ICD was still sufficient. There was no episode of ventricular tachycardia and ventricular fibrillation in previous records. Transesophageal echocardiography at admission revealed a slightly echogenic mass swinging through the tricuspid valve between the right atrium and ventricle during the cardiac cycle, which indicated a probability of thrombus (Figure, B). No abnormality was found in detection of inflammatory markers, including white blood cell counts, C-reactive protein level. D-dimer and coagulation parameters were in the normal range. No fever, diarrhea, cough, or expectoration was present.

Clinical decision was made upon a comprehensive examination. A complication of lead-related thrombosis but not infective endocarditis was considered. Anticoagulation therapy using low-molecular-weight heparin and bridging warfarin was carried out for 15 days. However, subsequent echocardiography showed similar to the previous observation at admission.

On the basis of the episode of syncope, the high risk for pulmonary embolism, and the pacemaker’s dysfunction with high pacing threshold and low perception, open-heart surgery was recommended to extract the pacing electrode. After obtaining signed informed consent, pacing electrode extraction and thrombectomy were performed under general anesthesia. The extraction was done during cardiopulmonary bypass with aortic cross-clamping. On visual inspection during the procedure, the pacemaker lead appeared encapsulated with adhesion of an old thrombus at the superior vena cava–right atrium junction. Thrombi were detached and removed from the adherent posterior leaflet. Histopathologic examination confirmed the aseptic thrombus. The ICD was left in place because the detection of parameters was identified to be appropriate during the procedure (Table). Warfarin treatment was continued after discharge. During a 3-month follow-up period, the patient was free of syncope episode, and the parameters of the programmed-controlled pacemaker remained appropriate.

**DISCUSSION AND CONCLUSIONS**

Pacemaker lead–related thrombosis is a rare complication of pacemaker implantation, occurring in 0.6%–3.5% of cases in previous studies [Barakat 2000; D’Aloia 2013]. No anticoagulant therapy is recommended for patients with ICD implantation. The thrombi were potentially secondary to the endothelial injury by long-term pacemaker presence [Do Carmo Da Costa 2002; D’Aloia 2013]. Platelet aggregation after pacemaker implantation might be another explanation for the formation of thrombosis [Palatianos 1994]. Additionally, hypercoagulable states should also be taken into account, including heart failure, neoplasia, pregnancy, etc.

Buttigieg et al and Raut et al previously reported successful thrombectomy and pacemaker lead extraction in patients with atrial thrombosis documented by echocardiography [Buttigieg 2015; Raut 2015]. However, there are rare reports on pacing dysfunction caused by the thrombus on the ventricular lead. In the present case, adhesion of the thrombus to the tricuspid valve may have contributed to the changes of pacemaker parameters, ie, high threshold and low perception. During cardiac systole the thrombus will flow to the atrial side, and the pacemaker electrode might be pulled, which can cause a slight dislocation of the electrode. Therefore, thrombus removal might be the best treatment.

In conclusion, this case illustrated that the pacemaker lead–associated thrombosis should be considered when the cardiac implantable electronic device fails to prevent patients from having cardiac events. Oral anticoagulant might be important for preventing thrombosis among patients with ICD implantation in the right ventricle.

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REFERENCES


