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A Walking Thrombus

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INTRODUCTION

Transvenous pacing is a relatively safe treatment with a low complication rate.[1] However, when complications occur they carries with them significant morbidity and mortality. The incidence of serious thrombotic and embolic complications ranges between 0.6 and 3.5%.[1,2]

Intracardiac sterile clots forming on pacemaker leads are an underestimated complication of cardiac device implantation and frequently present as an incidental echocardiographic finding. Although most of them remain asymptomatic, potentially serious consequences may occur.[3]

We report the case of a large pacemaker lead thrombus complicated by acute pulmonary embolism (PE) in few hours from its diagnosis.

CASE

A 67-year-old man with history of dilated ischemic cardiomyopathy was referred for the revision of the high-voltage component of his cardiac resynchronization therapy defibrillator because of low sensing and elevated output thresholds. Patient's device was recently upgraded for worsening ejection fraction from dual-chamber pacemaker without extraction of the previous right ventricular pacing lead.

He presented to our institution asymptomatic and haemodynamically stable. Physical examination and chest X-ray showed no abnormalities. Electrocardiography revealed biventricular paced rhythm. All laboratory parameters were unremarkable, except for a slight creatinine elevation. Patient underwent to defibrillator lead revision without any procedural complication.

A transthoracic echocardiography (TTE) performed before discharge accidentally revealed a large (18 × 40 mm), highly mobile, hypoechogenic mass attached to the pacemaker lead in the right atrium and passing through the tricuspid valve to the right ventricle (Figure 1A), consistent with a thrombus. To better characterize TTE findings we performed after few hours a transoesophageal echocardiogram, but multiple views didn't success to show clear images of the previously identified intracardiac mass (Figure 1B-D). A repeated TTE failed to show the mass again. After these procedures, patient started to complain a slight dyspnoea and his blood gas showed mild hypoxaemia and hypocapnia (pH 7.47, PaO_2 71 mmHg, $PaCO_2$ 28 mmHg; oxygen saturation 92%). In light of the changed clinical scenario we performed an urgent computed tomography pulmonary angiogram, which showed extensive right sided segmental and subsegmental (to upper, middle and lower lobes) pulmonary arterial filling defects, in keeping with acute pulmonary emboli (Figure 2).

Patient was then admitted to intensive cardiologic care unit. His calculated pulmonary embolism severity index was 118 (class IV, high mortality risk) and cardiac biomarkers showed an increase in blood concentration [high sensitivity troponin T 38 pg/mL (n.v. 0-14 pg/mL), NT-proBNP 2478 pg/mL (n.v. 0-125 pg/mL)]. In consideration of the stable hemodynamic profile (blood pressure 110/70 mmHg, heart rate 75 beats per minute) and the

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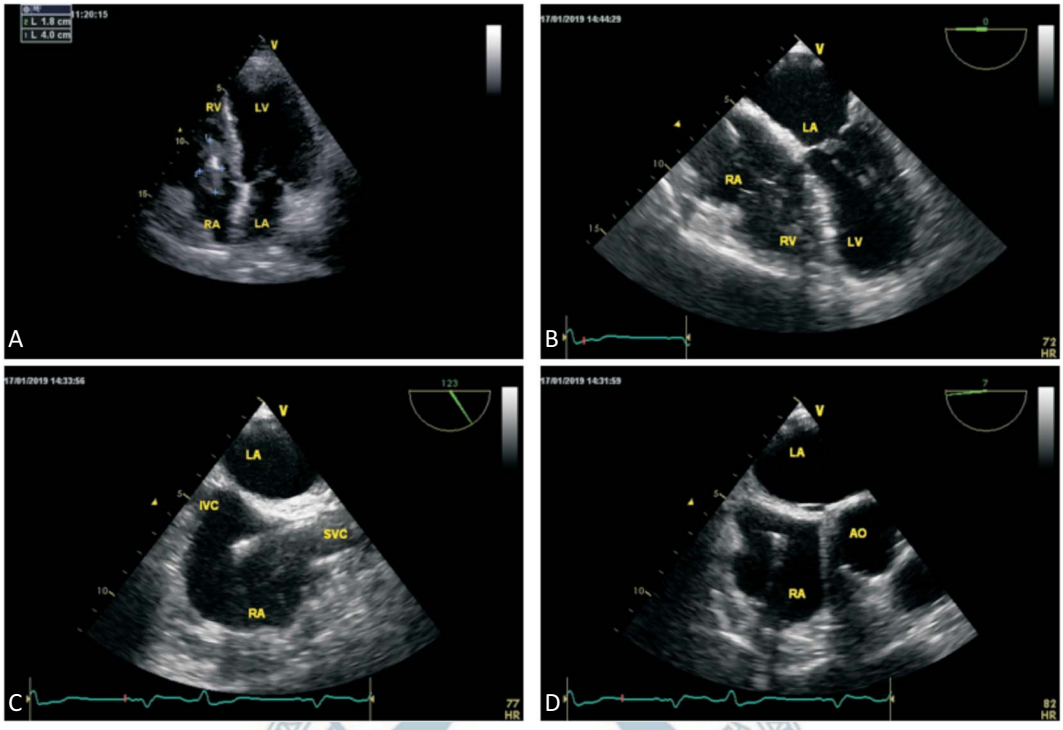

Figure 1. *(A) Transthoracic echocardiography image in apical four-chamber view showing pacemaker lead thrombus. Transoesophageal echocardiography image in: (B) four-chamber view, (C) bicaval view and (D) short axis view showing the absence of any mass on the pacemaker lead. AO, aortic root; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; SVC, superior vena cava.*

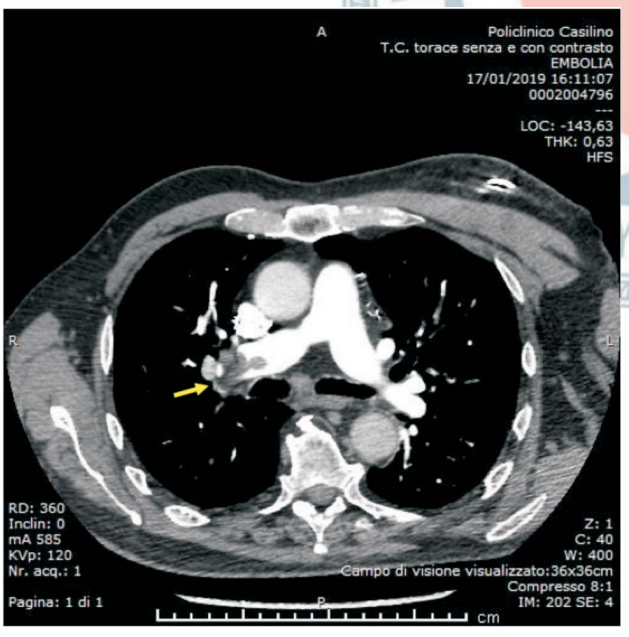

Figure 2. *Computed tomography pulmonary angiogram showing right pulmonary artery embolism (arrow).*

absence of echocardiographic signs of right ventricular dysfunction (intermediate-low risk of early mortality),[4] patient was directly treated with oral anticoagulation therapy (Rivaroxaban 15 mg twice daily for 3 weeks, followed by 20 mg once daily) according to current guidelines recommendation.[4] Duplex ultrasonography didn't show deep venous thrombosis in the upper body vessels. No clear risk factors were identified as cause of thrombosis, including negative thrombophilic and malignancy screening. The subsequent hospital's course was uneventful and the patient was discharged home in good conditions after one week with indication to long term anticoagulation.

DISCUSSION

Thrombi on cardiac implantable electronic device leads are usually asymptomatic and remain mostly undetected during clinical follow-up. Nevertheless, their incidence is not negligible and depends on the investigation method. Rahbar et al.[5] found an incidence of mobile thrombi on implantable device leads of only 1.4% in a TTE study on 1.086 patients, while a recent

autopsy series of patients with implantable cardiac devices identified thrombi on ventricular and atrial leads in 33% and 48%, respectively, and pulmonary emboli in 21% of patients.[6] Despite this relative frequency, the pathogenesis of thrombosis related to implanted devices remains controversial, as well as its clinical significance. Several factors have been postulated to be part of the pathogenesis of pacemaker lead induced thrombosis, such as congestive heart failure (CHF), atrial fibrillation, hypercoagulable states, the increased number of leads and possibly pacemaker lead material itself.[7] In general, transvenous pacemaker leads, like any foreign intravascular body, alter venous flow and increase turbulence, which may induce platelet aggregation and fibrin deposition and in ultimate analysis, together with patient's predisposing factors, lead to thrombus formation.

The treatment of asymptomatic pacemaker lead-associated thrombosis remains controversial. In small size thrombus anticoagulation is usually considered as the most reasonable option, meanwhile larger clots may require additional therapeutic options, such as thrombolysis or a surgical approach. In absence of specific guidelines, the optimal duration of anticoagulation is difficult to define and should be discussed case by case. In our patient we chose long term anticoagulation in consideration of the presence of multiple and non-modifiable risk factors for lead thrombosis (CHF, multiple leads) that could, in turn, increase the odds of further episodes of PE.

LEARNING POINTS

- Thrombi on intracardiac leads are an underestimated complication of cardiac device implantation
- Although they rarely cause clinical manifestations, large mobile thrombi may have an unpredictable fate and can be rapidly cause of serious and potentially life-threatening complications.
- In case of pulmonary embolism physicians should keep a high index of suspicion of cardiac implantable electronic device associated thrombosis, especially in high-risk patients and when the cause of embolism is not straight forward.

CONFLICT OF INTEREST

All the authors declare no conflict of interest.

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