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MOBILE THROMBI IN CARDIAC IMPLANTABLE ELECTRONIC DEVICE IN A PATIENT WITH ANTIPHOSPHOLIPID SYNDROME AS A RISK FACTOR FOR THE DEVELOPMENT OF SYMPTOMATIC PULMONARY EMBOLISM: CASE REPORT

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BACKGROUND

Antiphospholipid syndrome (APS) is characterized by arterial and/or venous thromboses and pregnancy morbidity in the persistent presence of antiphospholipid antibodies (aPL). Cardiac implantable electronic device (CIED) represents a foreign body in contact with the bloodstream and has a prothrombotic surface that leads to activation of the coagulation system by a complex interplay between blood cells and plasma proteins. We hereby report a case of a patient with primary APS who developed a cardiac pacemaker thrombosis, complicating with clinical pulmonary embolism (PE).

CASE REPORT

A 35-year-old female patient was diagnosed with interatrial communication and underwent surgical correction. In 2007, she developed atrial fibrillation/flutter, secondary to sinus node disease, and was submitted to implantation of a VVIR pacemaker. Since then, she was put on anticoagulation with warfarin. However, in 2014, she presented with pulmonary thromboembolism diagnosed by transthoracic echocardiogram, which showed multiple filamentous, echogenic, mobile images, adhered to the pacemaker lead in the right cameras and close to the entrance of the superior vena cava, suggestive of a thrombus. These findings were confirmed by transesophageal echocardiography. At that time, pulmonary hypertension was also investigated, with catheterization of right chambers showing group II pulmonary hypertension secondary to heart disease. During the workup for hereditary and acquired thrombophilia, she tested positive for both lupus anticoagulant and for anticardiolipin twice (63 and 92 MPL), with an interval greater than 12 weeks between tests. No obstetric complications were found. As the patient had no other criteria for other autoimmune disease, she was diagnosed with primary APS, and was anticoagulated with warfarin with an INR target of 2.5–3.5.

CONCLUSION

Thrombus on cardiac device leads were described in 30% of patients submitted to intracardiac echocardiography, but were not reported on transthoracic echocardiography. The clinical impact of such findings is not very clear, because although there is an increased risk of thrombus formation and asymptomatic PE in patients with CIEDs, only a minority develops clinical PE. Some studies concluded that the majority of patients who developed PE associated with a cardiac device had another established cause for PE. Therefore, this case reflects the importance of investigating additional risk factors that may complicate a pacemaker insertion, such as the presence of aPL.