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Intracardiac Sterile Pacemaker Lead Thrombosis

Trombosis intracardiaca estéril asociada a electrodo de marcapasos

To the Editor,

Venous thrombosis after pacemaker (PM) implantation occurs frequently, with a reported annual incidence of 23%.¹ However, recognition of intracardiac thrombus related to permanent pacemaker lead is particularly rare. Recently, an autopsy study² reported right atrial PM lead thrombosis in 14% of the patients at 4 years after implantation. The diagnostic and therapeutic strategies are not consensual, particularly regarding the asymptomatic patient. We report a case of a large PM lead thrombus that was diagnosed using bidimensional and three-dimensional transthoracic echocardiography and surgically removed in an off-pump procedure.

A 71-year-old man with previous diagnosis of mild rheumatic mitral stenosis and permanent atrial fibrillation was seen 6 months after implantation of a single chamber pacemaker indicated because of symptomatic slow ventricular rate atrial fibrillation. The patient had been on warfarin therapy until 3 months before the visit, but treatment was discontinued by the attending urologist for macroscopic hematuria.

In a follow-up visit the patient was doing well, without dyspnea or fever, but an echocardiogram revealed a large (32 × 13 mm) right atrial mobile and echogenic mass in close proximity to the PM lead. Three-dimensional echocardiography confirmed that the mass was attached to the PM ventricular lead and entirely located in the right atrium (Figs. 1A and B). There was no right ventricular dilatation but there was increased estimated pulmonary artery peak systolic pressure of 46 mmHg.

The patient was admitted for etiologic investigation and treatment. There was no leucocytosis, and C reactive protein and consecutive blood cultures were negative. Serologic tests for indolent causes of endocarditis were also negative. The ventilation-perfusion scan showed a single small perfusion defect. Thoracic, abdominal, and pelvic computed tomography did not find any other venous thrombus or other significant findings.

We used intravenous unfractionated heparin followed by subcutaneous low molecular weight heparin for 1 month without dissolution of the thrombus. The subsequent therapeutic strategy was to remove the thrombus and the pacemaker lead by off-pump right atrial thrombectomy. The procedure consisted of implantation of a ventricular epicardial PM lead followed by a small right atriotomy inside a purse-string suture, through which the thrombus and distal lead were quickly removed. The PM lead was cut (Fig. 1C) and the atrium closed without significant hemorrhage. The proximal lead was explanted transvenously with traction. Subsequent pathology and microbiology tests confirmed that the mass was a sterile organized thrombus.

Lead thrombosis has seldom been described. In the present case possible contributing pathogenic mechanisms include atrial

fibrillation with slow blood flow in the right atrium, associated with warfarin withdrawal.

Bidimensional transthoracic and transesophageal echocardiography are routinely used in the characterization of an intracardiac mass. Intracardiac echocardiography may also be useful for detection of thrombus adherent to device leads.³ We used three-dimensional echocardiography to better define the dimension and location of the thrombus, which permitted an off-pump surgical approach.

Pulmonary embolism has been recognized to occur in association with PM lead thrombosis.⁴ Therapeutic options to avoid this complication include anticoagulation, thrombolytic therapy and surgical embolectomy. Anticoagulation failed to reduce thrombus size and thrombolytic therapy has been shown to potentially result in thrombus fragmentation and pulmonary embolization.⁵ We preferred a surgical approach that additionally permitted placement of permanent epicardial leads. Off-pump thrombectomy was demonstrated to be a feasible and a valuable option in asymptomatic, large PM lead thrombosis.

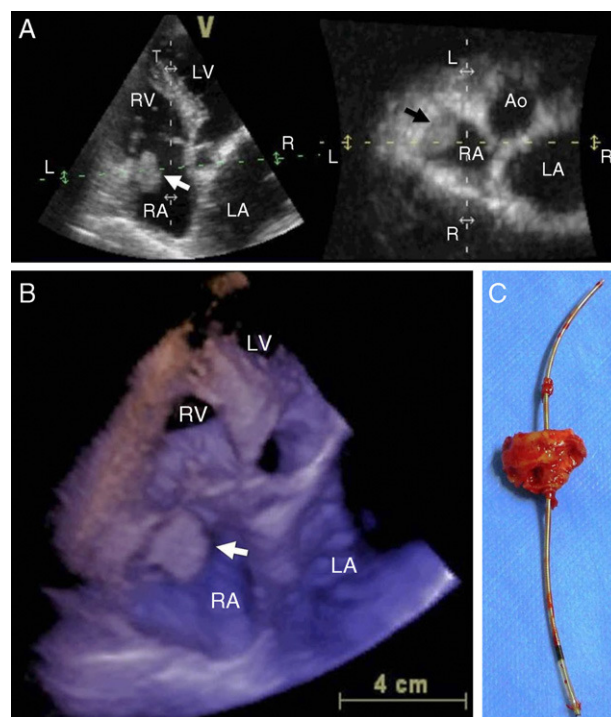


Figure 1. Pacemaker lead thrombus. A, Three-dimensional transthoracic echocardiography in apical 4-chamber view (left) and single slice method at the level of right atrium (right) showing longitudinal and transversal extension of the lead thrombus (arrows). B, Volume rendered three-dimensional image of the lead thrombus (arrow) attached to the ventricular lead of the cardiac pacemaker in the right atrium. C, Macroscopic examination of surgically removed distal pacemaker lead with attached organized thrombus. Ao, aorta; L, left; LA, left atrium; LV, left ventricle; R, right; RA, right atrium; RV, right ventricle.

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Chronic Infective Endarteritis Due to *Propionibacterium Acnes* on Aortic Prosthetic Graft

Endarteritis infecciosa crónica por *Propionibacterium acnes* sobre tubo prótesico aórtico

To the Editor,

A 62-year-old man was admitted to his reference hospital with right hemiparesis, dysphasia, and fever. He reported experiencing general malaise and shivers in the preceding weeks.

In his cardiovascular history, the patient had undergone a type A dissection at age 46. This required ascending aortic and aortic arch replacement surgery by means of the Cabrol technique with 2 woven Dacron[®] grafts. Two years earlier, he had been admitted to hospital for an episode of abdominal pain; computed tomography revealed splenic infarction and ascites. *Propionibacterium acnes* was isolated from the ascitic fluid. He was discharged in an asymptomatic state with diagnosis of primary neutrophilic

peritonitis. Subsequently, the symptoms recurred and he was readmitted 1 month after onset of symptoms. Splenic abscess was suspected; he received 4 months empiric treatment with imipenem and underwent splenectomy. The pathology report revealed chronic splenic abscess and *P. acnes* was isolated from the samples.

For the next 2 years, he suffered intermittent episodes of fever and shivering. In the evaluation in his reference hospital to rule out infectious endocarditis, blood cultures and transesophageal echocardiography were performed, with negative results, and it was decided not to initiate antibiotic treatment.

During a subsequent hospital admission, he had a fever (39 °C) and was hemodynamically stable. Physical examination revealed mixed dysphasia and right hemiparesis. Sinus rhythm was found in the electrocardiogram and the chest X-ray was normal. The blood work-up showed leukocytosis (26 700/μL) and the brain computed tomography without contrast enhancement did not reveal any signs of hemorrhage or medial displacement. In the brain magnetic resonance imaging, an ischemic lesion was detected in the left

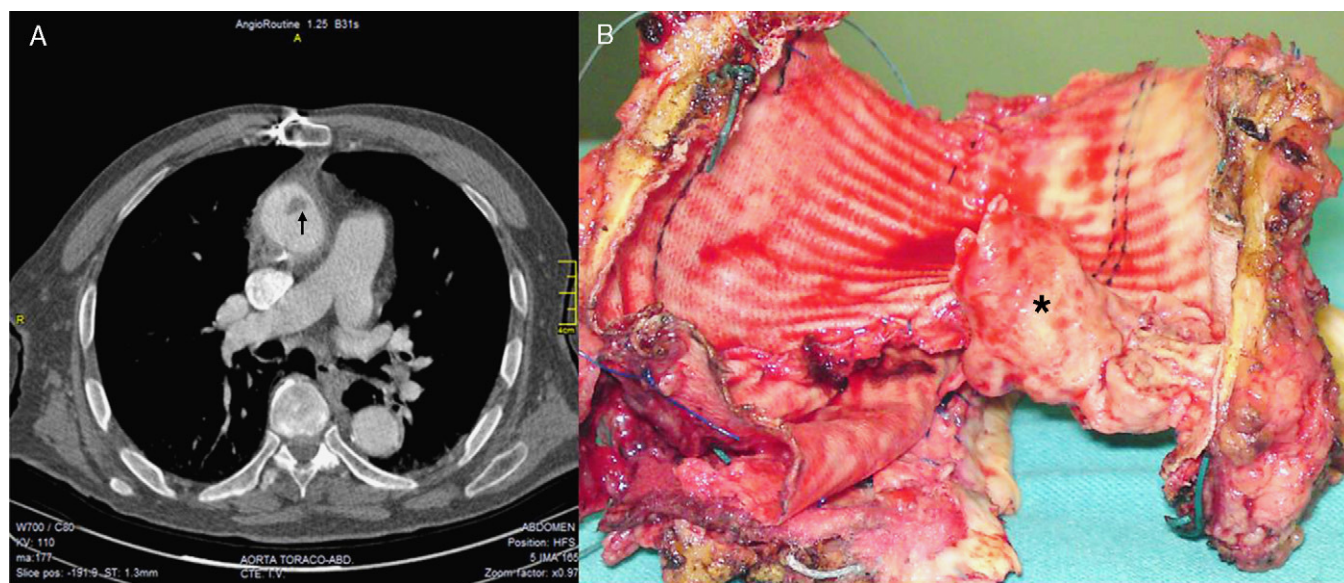


Figure 1. A, Contrast-enhanced chest computed tomography. A mass (arrow) can be seen as a filling defect in the aortic lumen. B, Surgical piece; a large vegetation (asterisk) can be seen where the two prosthetic grafts join.