

Figure 2. Lateral chest radiograph, with distal ventricular electrode in the right ventricular outflow tract.

inferior myocardial infarction), chronic obstructive pulmonary disease, and multiple myeloma. The patient was admitted for repetitive syncope after the ECG showed sinus rhythm with bifascicular block (RBBB + LAFB). Interrupted episodes of complete paroxysmal AV block were detected during electrocardiographic monitoring on the ward. Echocardiography showed mild left ventricular systolic dysfunction with inferior akinesis.

During implantation of a two-chamber permanent pacemaker via the left axillary vein, we observed LSVC persistence, the characteristics of which were similar to those described in the previous case. Two active fixation leads with distal electrodes were implanted in RA and RVOT, using manually preformed curved stylets identical to those described in case 1.

No dislocations or changes in the stimulation parameters were observed during follow-up.

Persistent LSVC can make implantation of a permanent pacemaker difficult. The fact that the coronary sinus ostium is not aligned with the tricuspid annulus means that the main technical problem consists of inserting the lead in the RV. The RVA has been the site of choice for cardiac stimulation, but reaching it in these cases usually means that the probe electrode forms a big loop in the RA.

Stimulation in the RVOT has been proposed as an alternative to RVA stimulation. In terms of lead stability, it has shown similar long-term results to those achieved with stimulation at the apex,⁶

and has also been shown to produce less dyssynchrony. Ventricular dyssynchrony promotes remodeling and in patients with left ventricular systolic dysfunction may have a deleterious effect on cardiac function. However, to date the benefits of RVOT stimulation have not been demonstrated in a randomized clinical trial. We have proposed a technique to reach this stimulation site in patients with persistent LSVC which only requires a manually preformed stylet. This can be similar to those used in normal conditions to reach the RVA, but without the need to form a loop in the RA.

Javier Jiménez-Díaz,^{a,*} Juan Benezet-Peñaranda,^b
and Arantzazu González Marín^c

^aUnidad de Arritmias, Servicio de Cardiología, Hospital General de Ciudad Real, Ciudad Real, Spain

^bUnidad de Arritmias, Unidad de Cuidados Intensivos, Hospital General de Ciudad Real, Ciudad Real, Spain

^cDepartamento de Cardiología Pediátrica, Servicio de Pediatría, Hospital General de Ciudad Real, Ciudad Real, Spain

*Corresponding author:

E-mail address: javierjimenezdiaz@wanadoo.es (J. Jiménez-Díaz).

Available online 23 July 2011

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doi: 10.1016/j.rec.2011.04.010

Prinzmetal Angina in the Migraine's Aura Resolving With Headache

Angina de Prinzmetal en el aura de la migraña que se resuelve con la cefalea

To the Editor,

We report the case of a 28-year-old woman referred to our hospital for constrictive chest pain with a left hemiparesis. She reported using oral contraceptive but had no cardiovascular risk factors. She had a history of unusual migraine with aura (left hemiparesis) since the age of 17 but had never been medicated and specifically not by ergotamine tartrate or triptan. Clinical examination showed left hemiparesis with a normal blood pressure of 132/76 mmHg. The electrocardiogram (ECG) revealed a sinus rhythm with ST-segment elevation in the inferior leads

with mirror image in the anterior leads suggestive of an acute inferior myocardial infarction (Fig. 1A). Per-critical acute trans-thoracic echocardiography revealed a mild hypokinesis of the inferior wall. The ascending aorta was normal without obvious criteria for aortic dissection. The neurologic signs led us to perform a computed tomography (Fig. 1B) that ruled out an intracerebral hemorrhage. The patient received treatment for acute coronary syndrome: sublingual nitrates, intravenous acetylsalicylic acid, loading dose of clopidogrel, and curative dose of enoxaparin. She was transferred without delay to the catheterization laboratory for primary percutaneous intervention. On her way to the catheterization laboratory, the chest pain and the hemiparesis disappeared but she complained of an intense headache. The coronary angiography demonstrated smooth coronary arteries with normal blood flow and no obstructive lesion (Fig. 1C). The postangiography ECG was normal (Fig. 1D). The 6-h troponin T rose to 11.6 ng/ml. The migraine headaches were treated by analgesics and her acute

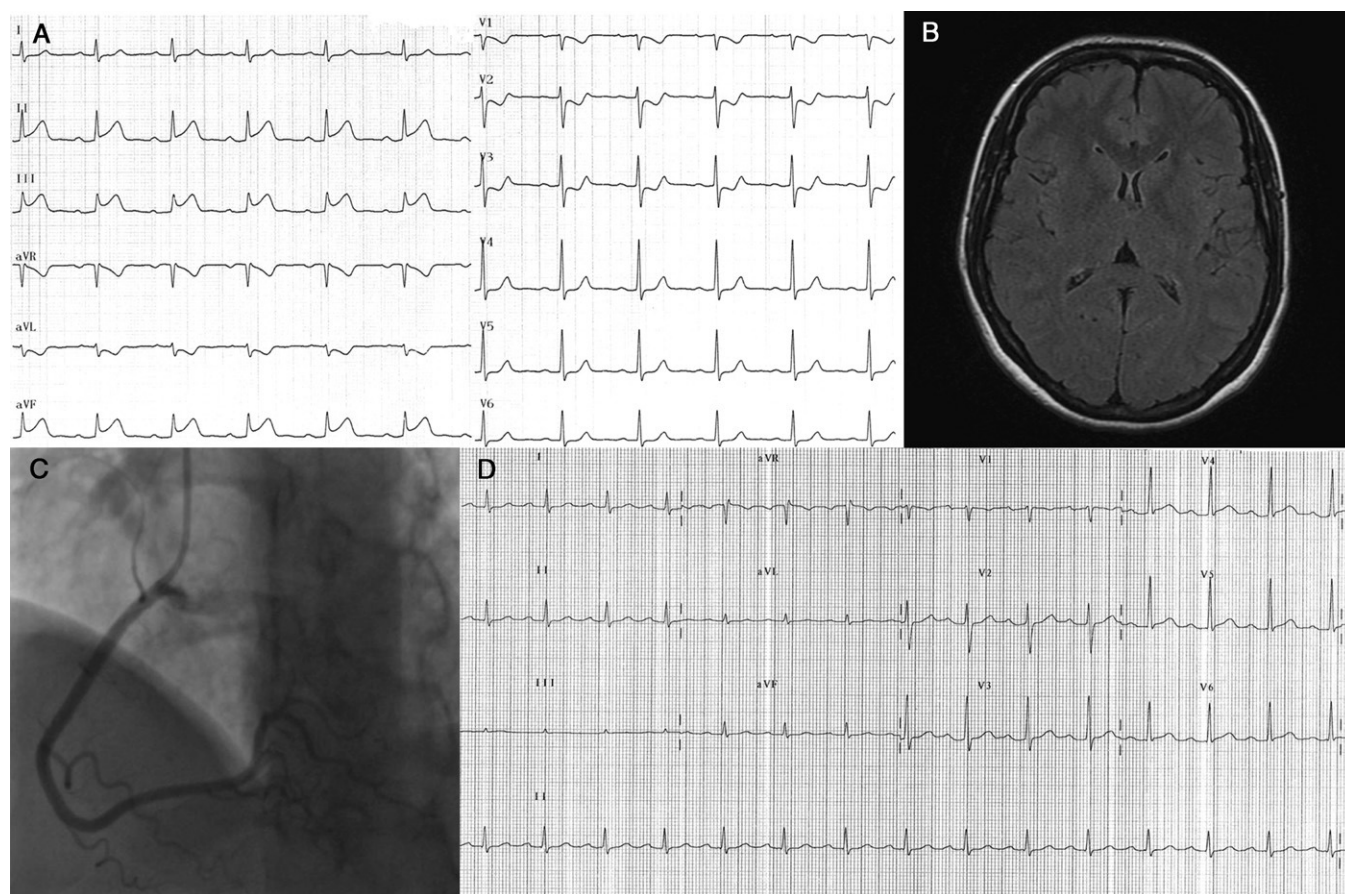


Figure 1. A, Per-critical ST-segment elevation in the electrocardiogram inferior leads. B, Normal cerebral computed tomography. C, Normal coronary angiogram with a smooth right coronary artery. D, Post-critical normal electrocardiogram.

coronary syndrome was treated by acetylsalicylic acid and verapamil. The echocardiogram at day 5 was normal and the patient was discharged. She remained event-free at 1 year.

The migraine headache is a common disease affecting approximately 13% of the population.¹ For 25% of these patients, migraine is associated with aura characterized by neurological symptoms and signs preceding or accompanying the headaches. In the literature, migraine with aura but not the migraine without aura was associated with an increased risk of myocardial infarction and ischemic stroke.²

The underlying pathophysiology of migraine is not fully understood. The condition is viewed as an inherited brain disorder but the vascular mechanisms are clearly implicated. Indeed, altered vascular reactivity has been found in patients with migraine.³ In our patient, we think that the aura was associated with a coronary spasm, most probably of the right coronary artery at the origin of the ST-segment elevation in the inferior leads. It remains possible that a coronary thrombosis with spontaneous thrombolysis or mediated by antiplatelet and anticoagulant therapy or a coronary microvascular dysfunction or a stress cardiac disease could explain the ECG changes. Indeed, no provocation test was done in our patient during the coronary angiogram but the ECG modifications and the early normal coronary angiogram suggested Prinzmetal angina. Then, during the migraine headache which is associated with a cerebral vessel vasodilation, the coronary vasospasm disappeared and the ECG normalized. It should be noted that our patient took no chronic therapy for her migraine such as triptan or ergotamine tartrate, both known to provoke coronary vasospasm.⁴ The mechanisms that link migraine to the vascular events remained under

discussion but are independent of many cardiovascular risk factors.² Several pathomechanisms have been proposed, such as an increase of the prothrombotic or vasoactive factors, decreased endothelium-dependent relaxation, and increased oxidative stress and inflammation of the vessel wall.⁵ Moreover, epidemiological data suggest a strong association between the migraine and acquired vascular disorders such as Raynaud phenomenon.⁶

Our case, with its 2-step evolution, lends support to the idea that the migraine with aura might be the manifestation of a general vasospastic disorder making the patients with migraine prone to a coronary artery spasm.

Vincent Roule,* Adrien Lemaitre, Ziad Dahdouh, Mathieu Bignon, and Gilles Grollier

Service de Cardiologie, Centre Hospitalier Universitaire de Caen, Caen, France

*Corresponding author:

E-mail addresses: roule-v@chu-caen.fr, v.roule@hotmail.fr (V. Roule).

Available online 23 July 2011

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doi: 10.1016/j.rec.2011.04.009

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.

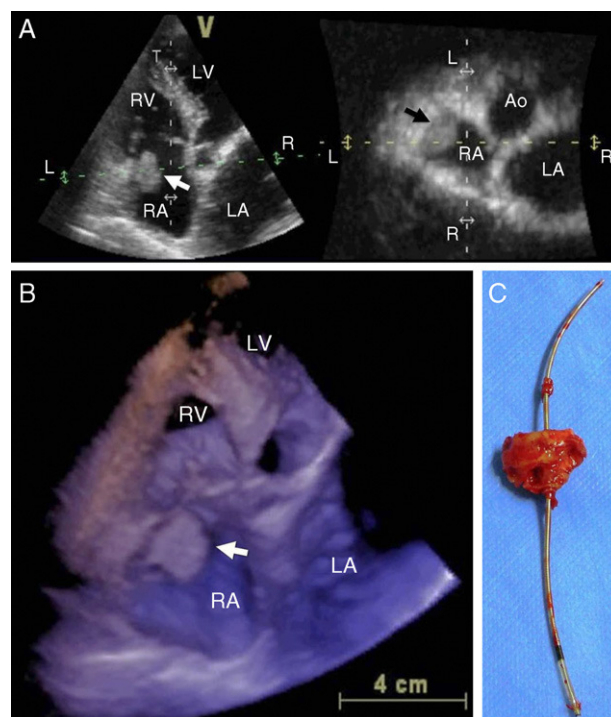


Figure 1. Pacemaker lead thrombus. A, Three-dimensional transthoracic echocardiography in apical 4-chamber view (left) and single slice method at the level of right atrium (right) showing longitudinal and transversal extension of the lead thrombus (arrows). B, Volume rendered three-dimensional image 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.