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Caso clínico

Transarterial permanent pacing of the left ventricle. An unusual complication

Julio A Aguilar,* Carmen Summerson*

ABSTRACT

In this report we describe a case of a 35-year-old woman with a permanent pacemaker who was evaluated for neurological symptoms consistent with amaurosis fugax and desorientation. The ECG showed a right bundle branch block that led to the suspicion of catheter misplacement. A chest radiographs and two-dimensional echocardiogram demonstrated that the pacemaker lead was not in the venous system, it was erroneously placed through the subclavian artery, across the aortic valve, and into the left ventricular chamber. Once anticoagulation was initiated, symptoms resolved. At the moment with a 6 years follow-up the patient is without symptoms.

Key words: *Pacemaker, electrode, left ventricle.*

RESUMEN

Los autores presentan el caso de una mujer de 35 años, con historia de implante de marcapasos definitivo, que fue evaluada por la presencia de amaurosis fugaz y desorientación. Un electrocardiograma mostró imagen de bloqueo de rama derecha del Haz de His, lo que hizo sospechar de una posición inadecuada del electrodo. Una radiografía PA de tórax y un ecocardiograma bidimensional mostraron que se trataba de una implantación transarterial inadvertida, pasando de la arteria subclavia a la aorta, el tracto de salida y ventrículo izquierdo. La paciente fue tratada con éxito mediante anticoagulación oral. Seis años después la paciente se ha mostrado asintomática sin evidencia de fenómenos embolígenos y con una adecuada función del marcapasos.

Palabras clave: Marcapasos, electrodo, ventrículo izquierdo.

INTRODUCTION

Since the first pacemaker implantation in 1958, cardiac pacing has continued to grow so that presently more than 500,000 patients in the United States have pacemakers. Almost 400,000 pacemakers are implanted worldwide each year. Many complications can occur as a direct result of the implantation technique or because of failure of a component of the pacing system. One of the complications associated with transvenous pacemaker implantation is the malposition of the pacing lead into the left heart. Left ventricular pacing has been reported due to the passage of the pacing lead through the interatrial septum,[1] a patent foramen ovale,[2] the interventricular septum,[3] or a sinus venous defect .[4] Infrequently it may arise from erroneous cannulation of the subclavian artery with the passage of the lead through the aorta, across the aortic valve and into the left ventricle.[5,6] The incidence and clinical course of this pacemaker complications are unknown.

In this article, we describe a case of permanent pacing of the left ventricular endocardium with a pacemaker inadvertently placed in the arterial system.

CASE REPORT

In October 1994, a 35-year-old woman was admitted for amaurosis fugax and loss of orientation lasting approximately 6 hours, later the patient was alert, restlessness and without neurologic deficit, she had a blood pressure of 110/80 mmHg and a heart rate of 62 beats/min. She had a VVI pacemaker for hypersensitive carotid sinus syndrome, placed at our institution one year prior to admission. The pacemaker lead was inserted via a right subclavian approach. An electrocardiogram with magnet showed ventricular pacing spikes; however, the ventricular morphology was consistent with right bundle-branch block. The blood biochemistry and full blood count were normal. A cranial computed tomography were without pathological findings. Posteroanterior (PA) and lateral chest films

* From Service of Cardiology and Department of Echocardiographic of Regional General Hospital No. 20 IMSS, Tijuana Baja California, Mexico.

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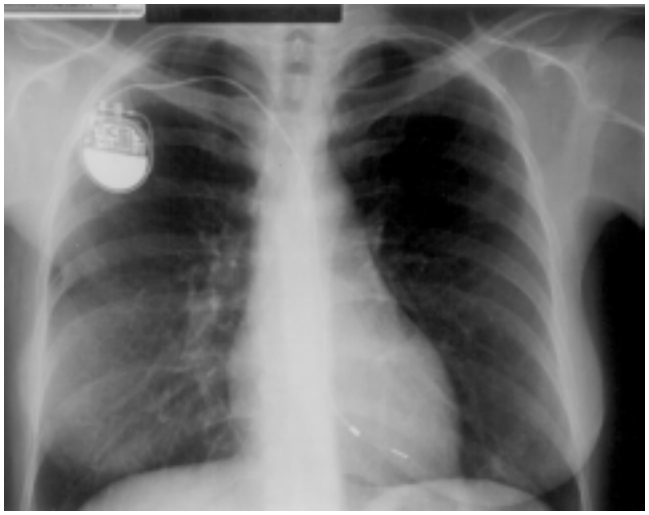

Figure 1. *Posteroanterior chest X ray suggesting atypical lead position in the left ventricle.*

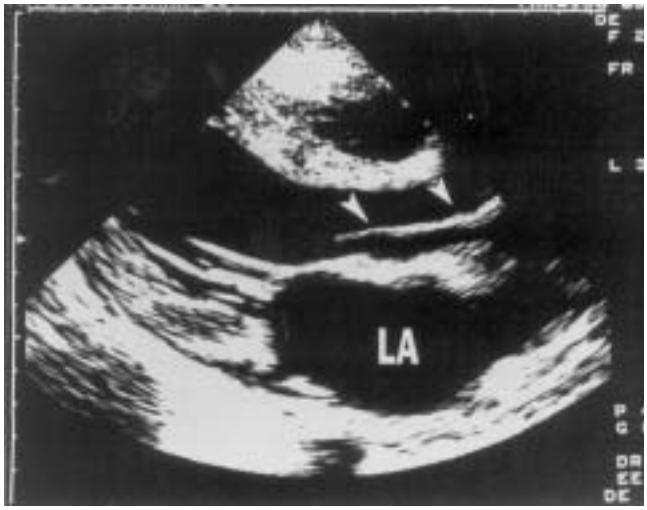

Figure 3. *Paraesternal long axis view, showing an echo-dense pacing lead into the left ventricular outflow tract (arrows). LA, left atrial.*

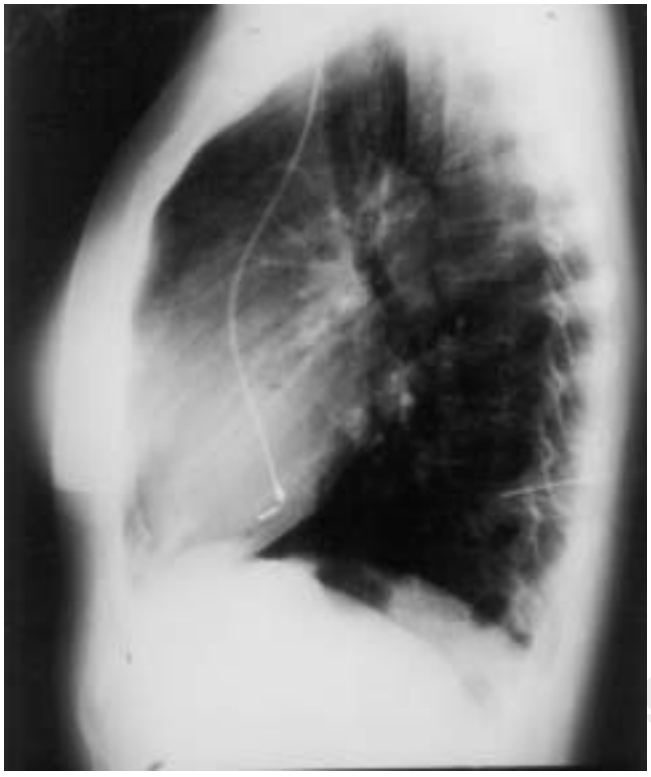

Figure 2. *Lateral chest X ray. The electrode tip takes a posterior course, this film is diagnostic of left ventricular lead placement.*

were taken. The PA film *(Figure 1)* shows the ventricular lead position to be higher and less apical than normally seen in the right ventricle. The lateral film *(Figure 2)* confirms insertion of the ventricle lead into the posterior wall of the left ventricle. An echocardiogram revealed an echo-dense pacing lead passing through the aortic artery to the left ventricular outflow tract and advancing into the left ventricle *(Figure 3).* Conventional Doppler examination did not showed any obstruction or regurgitation. Valves and chambers were normal and left ventricular contractility was normal. It was concluded that the pacemaker was implanted in the left ventricle via the right infraclavicular artery. At this time, the patient was started on warfarin, she was anticoagulated with an INR > 2.5 with resolution of symptoms.

After 6-years follow-up the patient remained asymptomatic, the pacemaker was stimulating normally and no complication was detected. She has declined consideration of removal and repositioning of the pacemaker lead.

DISCUSSION

The inadvertent placement of a permanent pacemaker lead through the arterial system is a rare complication of the implantation technique. Errors of implantation may be attributed in part to the experience of the implanter. One study demonstrated a significantly higher incidence of complications when implanters performed

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fewer than 12 implantations per year.[7] Subclavian artery puncture is usually easily recognized by the pulsating nature of the blood return and, or the aspirating syringe being filled under pressure. If this is recognized promptly and the needle removed, it is unlikely that a problem will result. During the surgical procedure the veins are clearly differentiated from the arteries. This distinction not obvious in a few cases, probably due to an undetermined vascular abnormality, or loss of pulsatile arterial flow because of hypotension or diminution of the arterial pulse pressure.

Another important cause of left ventricular pacing is failure of timely diagnosis once lead malposition occurs. The time to diagnosis varied from 1 day to 5 years. Possible vascular complications developed consisted of bleeding with hematoma formation, loss of brachial and radial pulses, and arterial thrombosis. Neurologic complications varied from desorientation and debilitating stroke with permanent aphasia.[8,9]

Arterial entry can usually be recognized fluoroscopically as passage into the left ventricle, viewed in the antero-posterior (AP) projection, and will be significantly medial to entry into the right ventricle. If a lateral view is used then a right ventricular lead will be anterior, that is, posterior to the sternum, while a left ventricular lead will be in the posterior aspect of the heart. Occasionally, the cardiac silhouette may not be clearly demarcated and consequently an intra-arterial lead placement may not be recognized. In addition, it may be difficult to differentiate the left ventricular apex on a frontal view from various positions of the right ventricular apex.[10,11]

Early activation of the left ventricle by the malpositioned lead creates a right bundle branch block pattern[12] which is a very helpful clue in the diagnosis, and was present in all patients. The sensitivity of this test for lead malposition in the left ventricle is 100%; however, a similar pattern may be seen when the lead is in the coronary sinusal or has penetrated into the ventricular septum. If lead misplacement in the left ventricle is suspected, two-dimensional echocardiography can confirm the diagnosis. The course of the catheter, site of crossing to the left heart, and exact location of the electrode tip can be demonstrated by echocardiography in order to determine the strategy for surgical intervention.[8,9] A definitive diagnosis of malposition can be established with these tests. While left ventricular pacing may be permanently secure, it has been associated with thrombus formation, embolization, and can have devastating neurological consequences. Development of any neurologic symptoms should be attributed to the malpositioned lead until proved otherwise. In such patients, serious consideration should be given to transcatheter or surgical lead extraction after a period of anticoagulation. If this not possible, chronic anticoagulation with warfarin must be initiated since antiplatelet therapy alone does not confer adequate protection against cerebral events. Reports of long-term uncomplicated left ventricular endocardial pacing exist, but the risk of embolization continues. In conclusion, that left heart pacing is not an infrequent complication of transvenous lead placement. And that a pacemaker stimulating the left ventricle through the arterial tree can be well-tolerated without complications during the time period observed here. Patients who have remained completely asymptomatic may be followed carefully with therapy.

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Reprint requests:

Julio A Aguilar MD
Service of Cardiology, Calle 2da 7567-301
Zona Centro. Tijuana, Baja California.
México. Código postal 22000.
E-mail: julioalb@att.net.mx