Atypical Atrial Flutter and Ablation of Recipient-to-Donor Atrioatrial Conduction After Orthotopic Heart Transplant

Aleteo auricular atípico y ablación de la conducción transauricular receptor-donante tras trasplante cardíaco ortotópico

To the Editor,

In orthotopic heart transplant, the bialtral anastomosis technique introduced by Lower and Shumway is widely used. In this technique distorts the right atrial geometry and hemodynamics, leading to increased mean atrial pressure, tricuspid and mitral regurgitation, electrical asynchrony, and an increased incidence of atrial flutter. This sequence, the most commonly described type is cavotricuspid isthmus-dependent flutter, in which the posterior barrier of the circuit is the atrioatrial suture line rather than the crista terminalis. However, when there is electrical conduction between the recipient atrium (A<sub>R</sub>) and the donor atrium (A<sub>D</sub>) across the atrial anastomosis, either by electrotonic transmission mechanisms or by direct conduction, the A<sub>R</sub> can contribute to the development of arrhythmias in the donor heart.

We present 4 patients who underwent orthotopic heart transplant with the bialtral anastomosis technique and developed atypical atrial flutter long after surgery (median, 5.5 years [range, 3–10 years]). All reported palpitations, with no signs or symptoms of heart failure, and electrocardiogram showed atrial flutter with variable atioventricular conduction (Figure 1). Once rejection had been excluded as an acute cause, a guided ablation procedure was performed with endomyocardial biopsy using a 3-dimensional navigation system (CARTO: ThermoCool SmartTouch ablation catheter, Biosense Webster).

In all patients, endocardial mapping showed a macroreentrant tachycardia with a variable relationship between the A<sub>R</sub> and A<sub>D</sub>. There was a frequency gradient from the A<sub>R</sub> which had a shorter, regular cycle length, to the A<sub>D</sub>, which had a longer cycle length (Figure 2A). The A<sub>D</sub> cycle length was also variable in 2 patients, with a Wenckebach–type periodicity. Guided by the presence of double potentials, we proceeded to anatomical delineation of the atrial suture (A<sub>R</sub>-A<sub>D</sub>) and performed activation mapping of the A<sub>D</sub> to determine early activation sites around the suture (Figure 2B). In 3 patients, an earliest activation site was found in the lateral wall of the right atrium (9–10 o’clock position of the suture line in an anteroposterior view), and in 1 patient, 2 earliest activation sites were found (at the 10 o’clock and 12 o’clock positions). A<sub>R</sub> entrainment maneuvers demonstrated an increased variability and prolonged mean cycle length in the A<sub>D</sub> in all patients and short return cycles in the right A<sub>R</sub> in 1 patient. Electrocardiograms of the earliest activation sites in the right A<sub>D</sub> showed features of atrioatrial continuity; radiofrequency ablation of these areas achieved A<sub>R</sub>-A<sub>D</sub> electrical dissociation. Thereafter, the A<sub>R</sub> remained in atrial flutter, and the A<sub>D</sub> in sinus

![Figure 1. 12-lead electrocardiogram of atypical atrial flutter in the 4 patients.](image-url)
rhythm, and the palpitations resolved. One patient underwent ablation of a macroreentrant tachycardia in the right AR; in the other 3 patients, long return cycles were confirmed and, with the suspicion of a left AR origin, sinus rhythm was restored by overstimulation.

As a general rule, atrial fibrillation is common in the immediate posttransplant period, whereas macroreentrant tachycardia develops in the longer term and is predominantly cavotricuspid isthmus dependent.5 However, some series have described a similar incidence of atypical flutter with an AR origin and transmission to the AO via atrioatrial connections.5 Electrocardiographic diagnosis is often complicated: distorted electrical propagation in the atria means that F waves are not generated, even in the common types of atrial flutter.5 Therefore, in electrophysiologic studies, electrical conduction between the AR and the AO should be included as part of a systemized study that also includes: a) analysis of frequency gradients; b) entrainment maneuvers in the cavotricuspid isthmus, and c) entrainment maneuvers in the right AR.

The optimum treatment strategy for atypical forms of atrial flutter is not fully established. As our series demonstrates, ablation of atrioatrial connections at the surgical suture line can be enough to terminate macroreentrant tachycardia in the AO and its associated symptoms, namely palpitations in these patients. However, there remain some unknowns regarding persistence of the tachycardia in the AR, including the embolic risk conferred to patients. The ideal strategy would be ablation of the slow conduction isthmus of the macroreentrant tachycardia, located in the AR. This was only possible in 1 (25%) of our patients, who had a right AR origin. The other patients had signs of a left AR origin, which would have required a transseptal puncture; these patients were treated conservatively. No patients received anticoagulant therapy during follow-up (54, 35, 5, and 4 months, respectively), and they made good clinical progress free from symptoms and complications, with sinus rhythm on electrocardiogram.

Diego Pérez Díez,* David Calvo Cuervo, José Manuel Rubín, Beatriz Díaz Molina, José Luis Lambert, and César Morís de la Tassa

Unidad de Arritmias, Servicio de Cardiología, Área del Corazón, Hospital Central de Asturias, Oviedo, Asturias, Spain

* Corresponding author:
E-mail address: dpdcardio@gmail.com (D. Pérez Díez).

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