Atrial arrhythmia in a patient after bicaval heart transplantation: Evidence for recipient-to-donor conduction

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Introduction

Supraventricular tachycardia in orthotopic heart transplant recipients is common. Recipient-to-donor conduction has been previously reported in these patients. In this case report, we present such a case.

Case report

A 50-year-old man presented with atrial tachycardia. Twelve years earlier, owing to nonischemic dilated cardiomyopathy, he underwent an orthotopic cardiac transplant with a standard bicaval technique. His cardiac rhythm before the surgery was permanent atrial fibrillation (AF).

The patient had no history of significant rejection, tachyarrhythmia, sinus dysfunction, or atrioventricular block. During follow-up, he reported paroxysmal episodes of palpitation that interfered with his daily activities.

Twelve-lead electrocardiograms (ECGs) showed paroxysmal supraventricular tachycardia with alternation between sinus rhythm, AF, and monomorphic atrial tachycardia, with cycle length variations (Figure 1). Up to now, a stable sinus rhythm was always recorded postoperatively on surface ECG.

Transthoracic echocardiography showed no systolic dysfunction. No sign of allograft rejection was found in the myocardial biopsy. Owing to the recurrence of tachyarrhythmia, despite antiarrhythmic drug therapy with amiodarone and a beta blocker, electrophysiological study was indicated.

An activation map was performed in both right atrium and left atrium (LA) via a femoral venous puncture and a transseptal puncture. Intracardiac electrograms showed that the posterior wall of the native LA was in AF (Figure 2), and that both donor's left and right atria exhibited irregular atrial tachycardia with different cycle length. The LA activation map showed an activation pattern that began at the inferior suture line behind the left inferior pulmonary vein.

At this point, there were 2 plausible mechanistic hypotheses to explain the results: (1) a focal mechanism that originated from the atrial anastomosis, independent of the AF, and (2) a recipient-to-donor atrial conduction with an intermittent conduction block.

Radiofrequency ablation was delivered in the native LA (Figure 3A), where complex atrial fractionated electrograms were identified behind the right pulmonary veins. The immediate result was restoration of sinus rhythm in both native and donor atria.

In sinus rhythm, pacing maneuvers from the native atrium were conducted to the donor's atria. In addition, entrance conduction was assessed along the residual recipient-donor conduction channel, confirming a 2-way connection. Figure 3A confirms that the donor LA was in AF before termination. This already was evidence that the driver was AF in the recipient LA, which was in fact also evident intermittently on the surface 12-lead ECG (Figure 1B).

These findings indicate that the donor atrium was passively activated by the native LA in AF with a conduction block. Because the donor atrium was driven by the native LA,

KEY TEACHING POINTS

- Persistent recipient-to-donor connection might promote atrial arrhythmias, especially in a patient with prior history of frequent atrial arrhythmia prior to transplantation.
- Ablation of atrial fibrillation in the native left atrium is responsible for restoring sinus rhythm in the donor's heart. Ablation of the presumed junction is safe and should be preferred over medical treatment.
- Atrial fibrillation can occur years after orthotopic heart transplantation and is not always associated with a poor prognosis.

KEYWORDS Atrial fibrillation; Atrial tachycardia; Electroanatomical mapping; Orthotopic heart transplant; Radiofrequency ablation
(Heart Rhythm Case Reports 2020;6:11–14)

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https://doi.org/10.1016/j.hrcr.2019.09.011

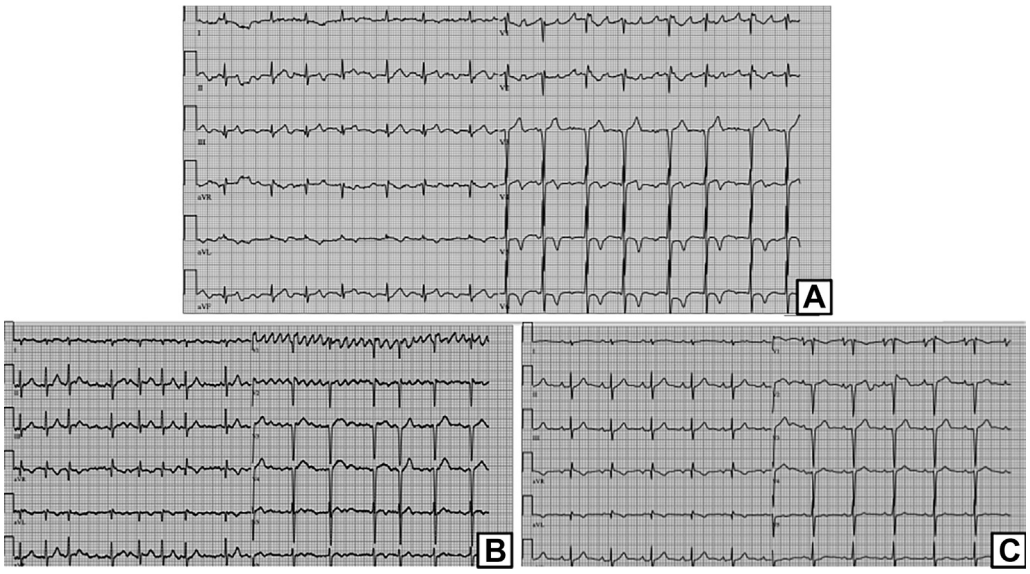

Figure 1 Twelve lead electrocardiograms. **A:** Atrial tachycardia with monomorphic P waves and cycle length variation. **B:** Coarse atrial fibrillation with normal ventricular rate. **C:** Electrocardiogram in sinus rhythm showing intra-atrial conduction delay with prolonged P-wave deflection in inferior leads.

we decided to perform not pulmonary vein isolation, but native LA isolation, and we successfully delivered radiofrequency at the presumed junction (Figure 3B).

A 3-month follow-up with 24-hour Holter monitoring showed no recurrence of AF or supraventricular arrhythmia.

Discussion

Heart transplantation is the gold-standard surgical approach for the treatment of refractory heart failure. The historical technique of cardiac transplantation is the Shumway method, owing to the technical ease in performing atrial cuffs when accomplishing venous and systemic connections. This method consists of creating anastomoses between donor and recipient atria, which creates abnormal atrial geometries. The loss of atrial geometry was previously demonstrated to cause atrial arrhythmias (44% in the first 728 days after the day of surgery).[1] The loss of atrial shape led to the development of alternative treatments, mainly the bicaval technique. This technique includes separate anastomoses in the superior and inferior vena cava, while the left atrial cuff of the donor heart is anastomosed to the pulmonary venous cuff on the recipient side, with a single suture line. The main objective is to preserve the right atrial geometry and morphology to lower the incidence of supraventricular arrhythmia.[2]

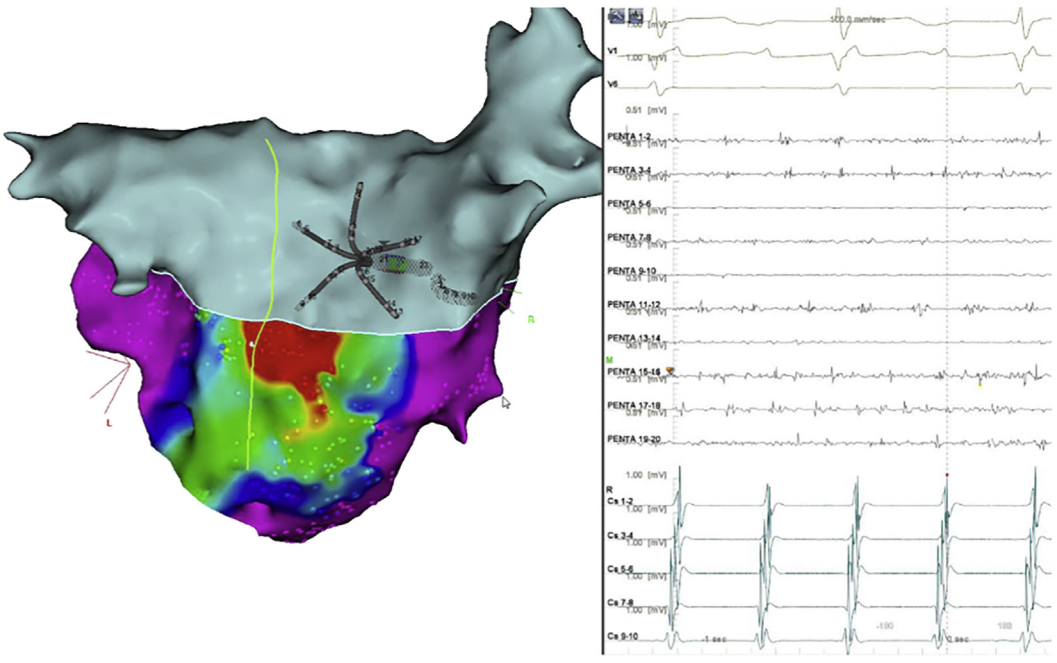

Figure 2 Activation mapping with the multipolar catheter (PentaRay, Biosense Webster, Irvine, CA) and the decapolar catheter in the coronary sinus (CS). Left atrial activation map showing that the native left atrium (LA) (*blue*) rhythm is atrial fibrillation (PENTA 1–20) while the donor LA is monomorphic atrial tachycardia (CS 1–10).

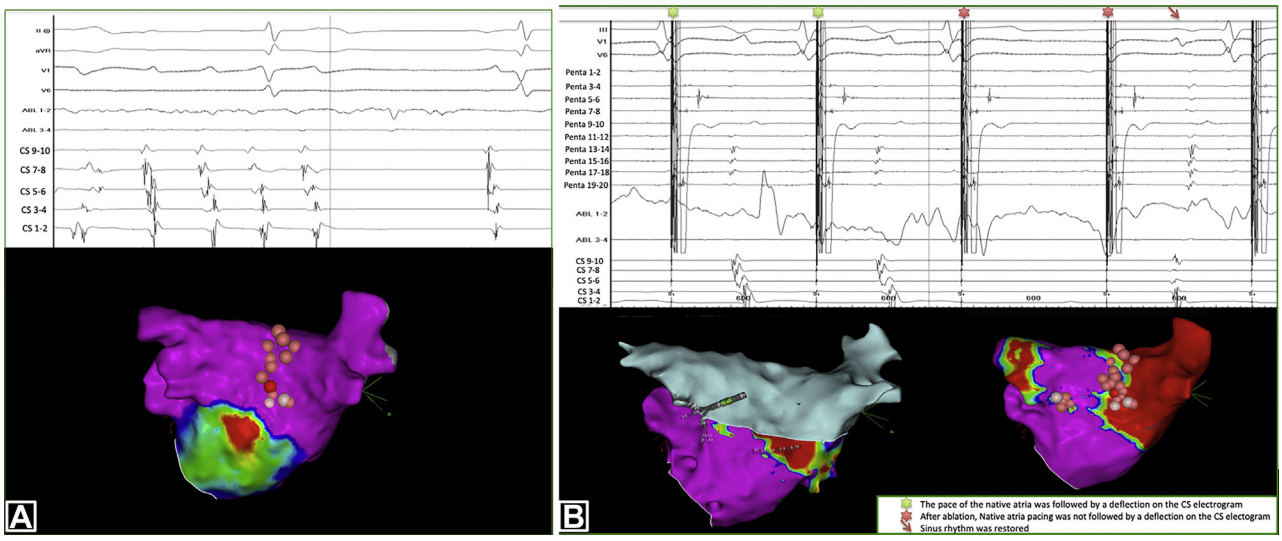

Figure 3 Electrocardiograms during the ablation procedure. **A:** Intracardiac electrogram: the native left atrium (LA) rhythm was atrial fibrillation (AF). Radiofrequency delivery consisted of targeting complex and fragmented atrial activity of the native LA behind right pulmonary veins. Termination of the AF was immediately followed by a termination of the atrial arrhythmia in the donor's heart. The donor LA also was in AF before termination. This already was evidence that the driver was AF in the recipient LA. **B:** Intracardiac electrogram: The PentaRay (Biosense Webster, Irvine, CA) catheter was positioned in the native LA. The pace of the native atria in this zone, always followed by a deflection on Penta 5–6 (native LA), Penta 11–20, and the coronary sinus (CS) electrogram (donor's LA), proved the presence of a conduction zone between the 2 atria. Radiofrequency delivery was performed on the presumed junction. Recipient atrial pacing was no longer followed by a deflection in the CS electrogram, thus confirming conduction block. Sinus rhythm was restored.

Grafted hearts mainly provide the substrate that promotes the development of supraventricular tachycardias. The cavotricuspid isthmus–dependent right atrial flutter is the most common supraventricular arrhythmia.[3] These atrial arrhythmias are associated with macroreentrant and focal mechanisms that occur in the donor heart. Rarely, they occur in the recipient heart. This latter condition can cause recipient-to-donor atrial conduction, with variable conduction blocks.[4] AF remains uncommon, owing to the pulmonary venous transection associated with the procedure.[5] Beyond the perioperative period, atrial fibrillation was only reported to be present when the allograft was rejected or vasculopathy occurred.[3]

To our knowledge, several cases described AF in the recipient atrium after transplantation, which resulted in atrial tachycardia or atrial flutter in the donor heart.[6–9] In all of them, electroanatomical mapping was used to identify and treat the atrio-atrial conduction sites, resulting in restoring sinus rhythm in the donor heart, and suggesting a recipient-to-donor conduction.

In the present case, we chose to perform AF ablation in the native LA to restore sinus rhythm in the donor heart. Atrial stimulation in the native atrium was conducted into the grafted heart, which confirmed recipient-to-donor atrial conduction. This approach was adopted, rather than targeting first the atrio-atrial conduction, in order to prove that AF was the driving source of the arrhythmia in the graft and not a mere coincidence. In addition, since a connection occurred at a site between the recipient and the donor, another connection could, as well, develop later.

Our patient's rhythm alternated between sinus rhythm, atrial tachycardia, and AF. As mentioned before, AF is uncommon after transplantation and is mainly related to allograft rejection.[5] In a meta-analysis, the prevalence of AF following bicaval heart transplantation was 11.1%. It was significantly associated with increased overall mortality.[10] As shown by Ahmari and colleagues,[11] AF is associated with decreased long-term survival. The rationale remains unknown, but left ventricle dysfunction, coronary heart disease, or diastolic dysfunction may underlie this feature. In our patient, AF was intriguing since the patient was free from any heart disease or allograft rejection. Atrial tachycardia is likely to be a non–pulmonary vein trigger for AF in the donor heart. Contrary to what is recognized, this patient with AF after heart transplantation has, therefore, a very likely better prognosis.

Several hypothesis may explain recipient-to-donor conduction. Gaudesius and colleagues[12] reported the role of fibroblasts in supporting propagation over extended distances through electrotonic interactions. Landolina and colleagues[13] suggested that electrical propagation was possible through viable myocardium bridging the surgical scar, while describing a bidirectional decremental conduction across a suture line in a grafted heart.

Conclusion

Our results suggest that a pulmonary venous reconnection could occur years after an orthotopic cardiac transplant with bicaval anastomoses. This is all the more interesting since the rhythm before the transplant was permanent AF and sinus rhythm was never restored in his native LA during surgery. Persistent connection or chronic reconnection might promote atrial arrhythmias, especially in a patient with prior history of frequent atrial arrhythmia prior to transplantation. Ablation seemed to be safe and effective and may be preferred over medical treatments.

Electrophysiological mechanism assessment of AF in transplant patients is essential and allows a better prognostic and therapeutic evaluation.

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