Atrial fibrillation originating from recipient left atrium after an orthotopic heart transplantation

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Introduction

Atrial fibrillation (AF) is rare in patients after orthotopic heart transplantation (OHT) and most commonly has been described in the setting of acute rejection or transplant vasculopathy.[1–3] The principal reason postulated for the rarity of AF after OHT is the fact that the pulmonary veins and posterior left atrial (LA) wall are isolated as a direct result of the left atrioatrial anastomosis of the remnant LA cuff (which includes the recipient pulmonary veins) to the donor left atrium. Previous reports of late supraventricular tachycardias after OHT have described arrhythmias originating in the recipient atrium and conducting to the donor atrium, but in the vast majority of these reports the arrhythmia is a regular atrial tachycardia or flutter originating from the right atrium (RA).[4,5]

Case report

A 67-year-old man presented with a recurrent irregular supraventricular tachycardia despite sotalol therapy. He had a history of ischemic cardiomyopathy and was status post OHT 2 years previously. The surgical technique used was a standard right bicaval and left atrioatrial anastomosis. At 1 year post transplant he was diagnosed with AF and was started on anticoagulation with warfarin and sotalol. He did not have hypertension, diabetes, or sleep apnea before or after heart transplantation and his pretransplant LA size by transthoracic echocardiography was normal. He had undergone evaluation by the heart transplant team and showed no signs of rejection or vasculopathy based on laboratory testing, right ventricular biopsy, and selective coronary angiography. Thyroid function was normal, and infection work-up and common cytologic testing including cytomegalovirus titers were negative. Because of progression of his tachycardia, which became incessantly repetitive despite antiarrhythmic therapy, he was referred for electrophysiological testing and possible ablation. His electrocardiograms demonstrated an irregular narrow complex tachycardia with a single dominant P-wave morphology that was positive in V_1 and negative in the inferior leads, and a relatively fixed PR interval of 130 ms (Figure 1).

A baseline electrophysiological study was performed to determine the mechanism of the tachycardia. A decapolar catheter was placed in the coronary sinus (CS), a quadripolar catheter was placed in the right ventricle, and a ThermoCool SmartTouch catheter (Biosense Webster, Inc, Diamond Bar, CA) was placed in the RA. The rhythm mechanism was determined to be an irregular atrial tachycardia, which was repetitive and nearly incessant, with only brief periods of sinus rhythm. Three-dimensional electroanatomic mapping of the RA and CS using the CARTO system (Biosense Webster, Inc, Diamond Bar, CA) demonstrated earliest and nearly equal activation times recorded from the proximal CS and low right atrial septal region, which indicated a probable LA source of the arrhythmia. LA electroanatomic mapping was performed via a transseptal approach. Mapping in the anterior portion of the LA (donor LA) demonstrated an irregular but organized atrial tachycardia (similar to the RA and CS recordings) (Figure 2A). Mapping in the posterior LA (recipient LA and pulmonary veins) demonstrated high-frequency irregular atrial activity, with variable cycle length ranging between 180 and 260 ms, consistent with AF, with the highest-frequency atrial activity recorded near the right pulmonary veins (Figure 2B). Mapping along the presumed anastomotic suture line demonstrated the earliest area of conduction from the remnant to donor LA just anterior to the right inferior pulmonary vein. Radiofrequency ablation along this region terminated the AF and restored sinus rhythm in both the remnant and donor atria. After restoration of sinus rhythm, donor-to-recipient atrial conduction was still present (Figure 3A). Mapping of the posterior LA wall was performed using a 20-pole circular catheter (Lasso NAV catheter, Biosense Webster, Inc, Diamond Bar, CA) to identify the sites of conduction breakthrough between the donor and recipient LA. The earliest sites of conduction breakthrough were along the area just inferior-posterior to the right inferior pulmonary vein (Figure 3B). Radiofrequency ablation at this region resulted in complete isolation (entrance and exit block) of the recipient LA (Figure 3C). There were no complications and the total procedure, electroanatomic mapping, and ablation

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KEY TEACHING POINTS

- Atrial fibrillation can rarely occur in postorthotopic heart transplantation in the remnant left atrium.
- Recipient-to-donor atrial conduction is necessary for clinical manifestation and maintenance of atrial fibrillation in those patients.
- Ablation of recipient-to-donor atrial conduction can be sufficient to terminate atrial fibrillation by effectively reisolating the recipient atrium.

durations were 3 hours and 20 minutes, 2 hours and 35 minutes, and 33 minutes, respectively. At discharge, sotalol was discontinued and warfarin continued.

Discussion

The most common atrial arrhythmias after heart transplantation are cavotricuspid isthmus–dependent flutter and focal atrial tachycardia originating in the donor atria.[1,3,4,6] AF after heart transplantation has been described mainly in the early postoperative period.[7] Late-onset AF in stable OHT patients is very rare and has usually been described in the setting of rejection or vasculopathy.[1,2] The lack of AF in OHT patients has been primarily attributed to the fact that these patients effectively have a posterior wall and pulmonary vein isolation owing to the surgical technique used, which anastomoses the cuff of LA tissue surrounding the pulmonary veins of the recipient to the donor LA.[8] In addition, there is a complete autonomic denervation of the donor heart, which may mitigate the autonomic factors that are thought to play a role in pathogenesis of AF.[9]

Prior reports of late-onset AF after OHT not associated with rejection or vasculopathy were difficult to find. In the 1 case report we did locate, no electrophysiological evaluation was performed to determine the origin of the fibrillatory activity.[10] In our case the patient had a diagnosis of AF and presented back with a repetitive irregular tachycardia, which appeared initially to be owing to an organized irregular focal atrial tachycardia. It was not until more extensive LA mapping was performed that we demonstrated that the arrhythmia was attributable to AF from the recipient LA with conduction into donor atria. To our knowledge, this is the first reported case of a late post-OHT AF originating from the recipient LA. The relatively organized atrial activity in the donor atrium and the uniform P-wave morphology can best be explained by a limited area of conduction breakthrough from the recipient to donor atrium. The irregularity of the rhythm can be explained by both the irregularity of the AF and the variable conduction across the anastomosis from the recipient to donor atrium. Atrioatrial conduction late after heart transplantation is well described and is estimated to be present in 10% of patients based on follow-up electrocardiogram analysis.[11] The proposed mechanisms of electrical reconnection are thought to be attributable to either electrotonic transmission, owing to ionic currents inducing depolarization in nearby excitable tissue across an intervening region of impaired conductivity without actual propagation of an action potential,[12] or the growth of excitable myocardium across the anastomosis line, allowing direct conduction of cardiac action potentials. We speculate that the second mechanism is more likely in our case, given the evidence of

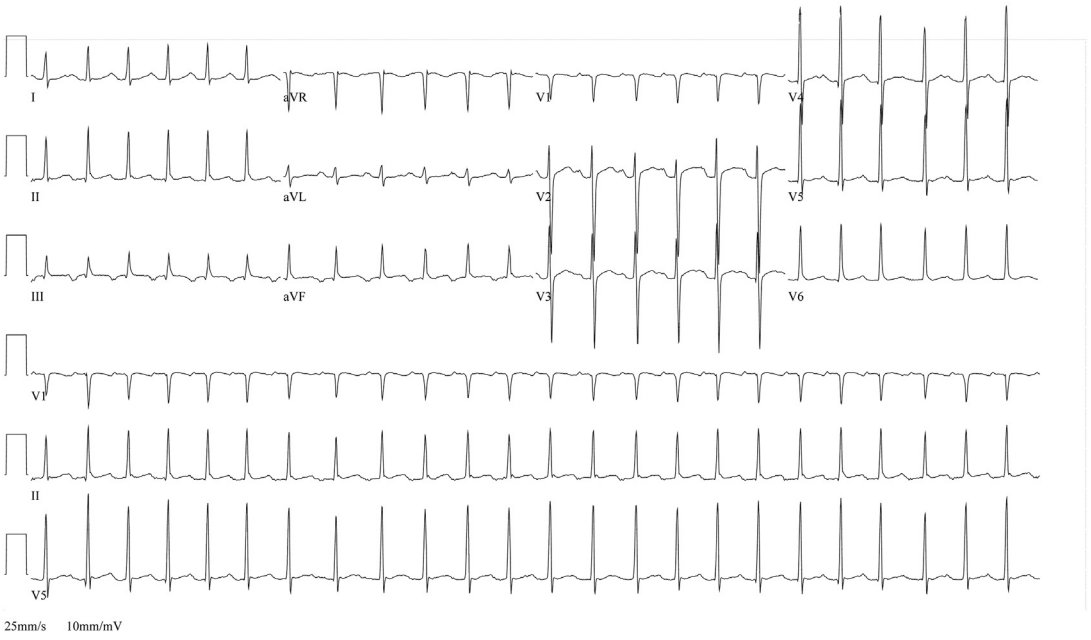

Figure 1 Electrocardiogram showing irregular narrow complex tachycardia with variable rate of 130–150 beats per minute with a single dominant P-wave morphology (positive in lead V_1 and negative in leads II, III, and aVF) with a relatively fixed PR interval of 130 ms.

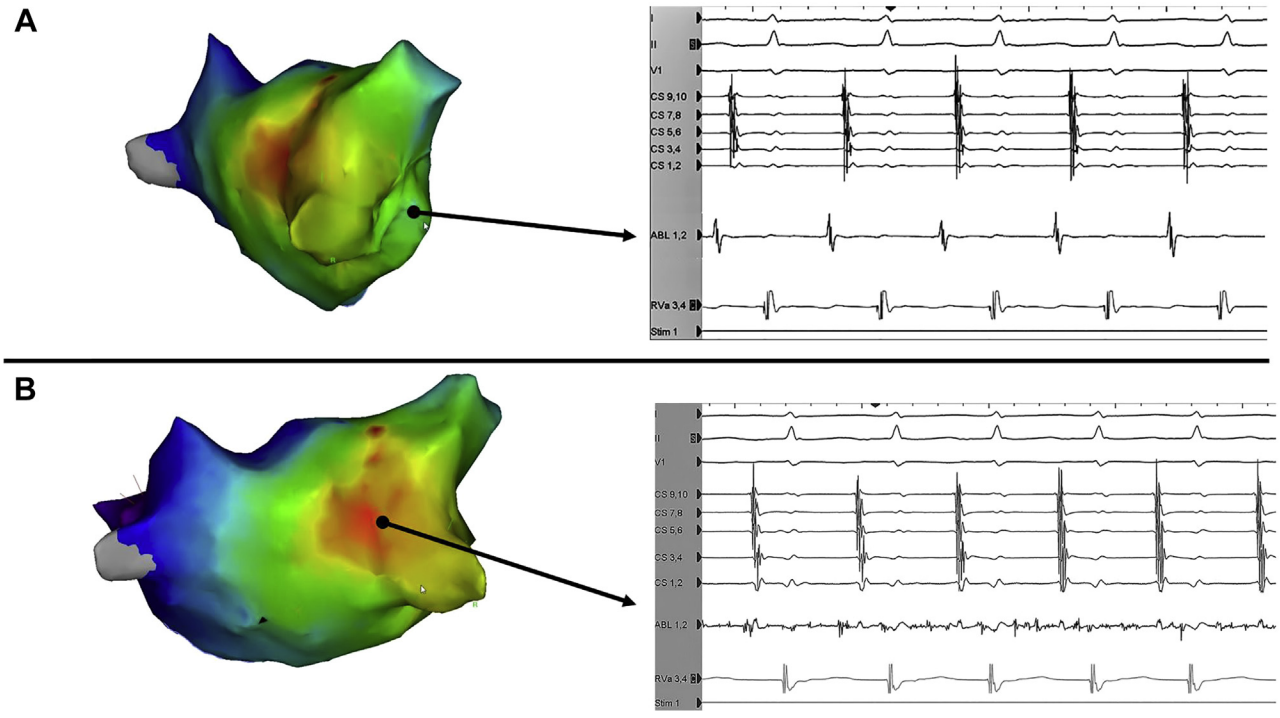

Figure 2 Electroanatomic mapping of the left atrium (LA). **A:** Right posterior oblique orientation with ablation catheter in the anterior portion of the LA (donor LA) demonstrating an irregular but organized atrial tachycardia (similar to the coronary sinus catheter recordings). **B:** Posterior-anterior orientation with ablation catheter in the recipient atrium and pulmonary veins demonstrating high-frequency irregular atrial activity, with variable cycle length ranging between 180 and 260 ms, consistent with atrial fibrillation, with the highest-frequency atrial activity recorded near the right pulmonary veins.

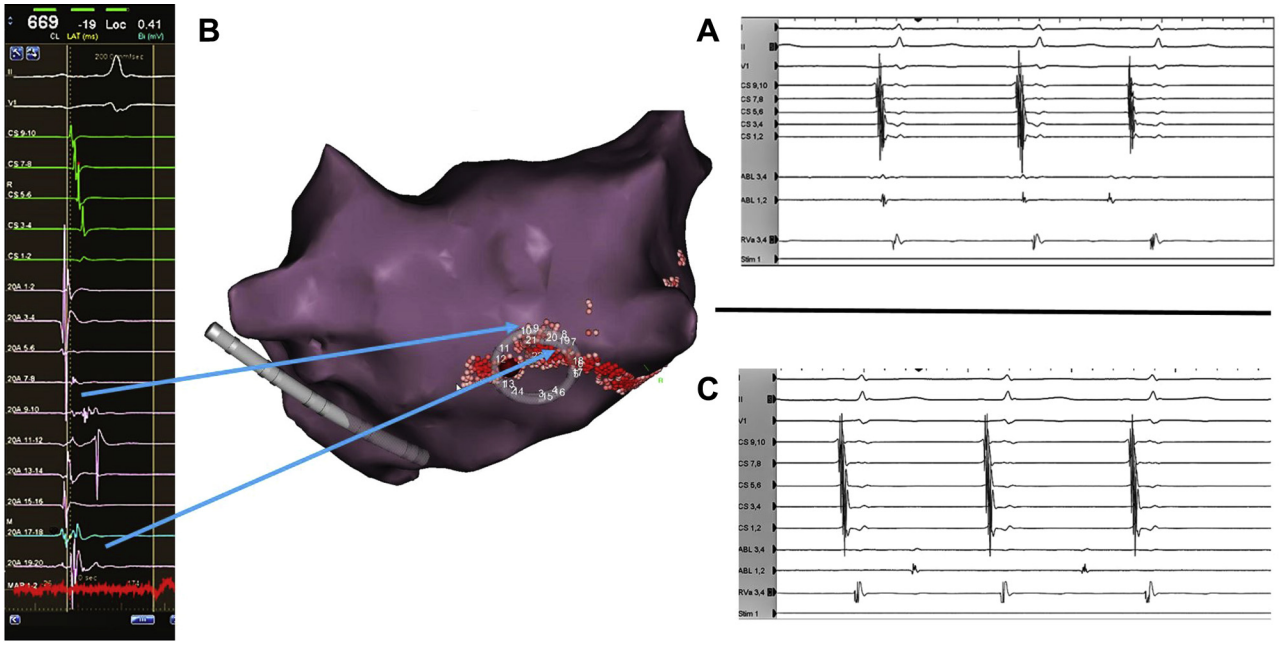

Figure 3 Electroanatomic mapping of the left atrium (LA) in posterior-anterior orientation after restoration of sinus rhythm. **A:** Ablation catheter in the posterior wall (recipient LA) and coronary sinus (CS) catheter in donor CS; notice presence of bidirectional conduction between recipient and donor. **B:** Mapping of the posterior LA wall was performed using a 20-pole circular catheter (Lasso NAV catheter, Biosense Webster, Inc, Diamond Bar, CA) to identify the sites of conduction breakthrough. The earliest sites of conduction breakthrough (poles 9–10 and 19–20) were along the area just inferior-posterior to the right inferior pulmonary vein. **C:** Radiofrequency ablation at sites (9–10 and 19–20) resulted in complete isolation (entrance and exit block) of the recipient LA.

bidirectional conduction between the recipient and donor atria, which was clearly demonstrated after restoration of sinus rhythm. Prior case reports and a prior series of supraventricular tachycardias occurring after OHT have clearly demonstrated atrial arrhythmias that resulted from focal atrial tachycardias in the recipient atrium with conduction to the donor atrium.[4,5,13] These same reports also documented the utility of electroanatomic mapping for localizing and ablating the areas of atrioatrial conduction for arrhythmia control. In these prior reports, however, the remnant atrial tissue that caused the arrhythmia was mainly right atrial in origin, except for 1 focal tachycardia originating from recipient left atrium.[11] To the best of our knowledge this is the first report of an AF originating from remnant LA tissue and the first report to demonstrate the utility of electroanatomic mapping for performing an LA posterior wall isolation for management of AF by mapping and ablating along the line of the prior LA anastomosis.

Conclusion

This report adds to the body of literature demonstrating that recipient-to-donor atrial conduction can play a role in supraventricular tachycardias occurring late after OHT. This case is unique in that it demonstrates that the LA remnant can be arrhythmogenic and in this case appeared to be able to sustain fibrillatory activity. This arrhythmia mechanism should be considered in OHT patients, and with our current state of advanced mapping and ablation techniques consideration of an ablation therapy should be undertaken early for this group, especially given the potential toxicity of antiarrhythmic agents in this patient population.

References

1. Ahmari SA, Bunch TJ, Chandra A, Chandra V, Ujino K, Daly RC, Kushwaha SS, Edwards BS, Maalouf YF, Seward JB, McGregor CG, Chandrasekaran K. Prevalence, pathophysiology, and clinical significance of post-heart transplant atrial fibrillation and atrial flutter. J Heart Lung Transplant 2006;25:53–60.
2. Khan M, Kalahasti V, Rajagopal V, et al. Incidence of atrial fibrillation in heart transplant patients: long-term follow-up. J Cardiovasc Electrophysiol 2006; 17:827–831.
3. Vaseghi M, Boyle NG, Kedia R, Patel JK, Cesario DA, Wiener I, Kobashigawa JA, Shivkumar K. Supraventricular tachycardia after orthotopic cardiac transplantation. J Am Coll Cardiol 2008;51:2241–2249.
4. Elsik M, Teh A, Ling LH, Virdee M, Parameshwar J, Fynn SP, Kistler PM. Supraventricular arrhythmias late after orthotopic cardiac transplantation: electrocardiographic and electrophysiological characterization and radiofrequency ablation. Europace 2012;14:1498–1505.
5. Magnano AR, Garan H. Catheter ablation of supraventricular tachycardia in the transplanted heart: a case series and literature review. Pacing Clin Electrophysiol 2003;26:1878–1886.
6. Li YG, Gronefeld G, Israel C, Lu SB, Wang QS, Hohnloser SH. Radiofrequency catheter ablation in patients with symptomatic atrial flutter/tachycardia after orthotopic heart transplantation. Chin Med J (Engl) 2006;119:2036–2041.
7. Cohn WE, Gregoric ID, Radovancevic B, Wolf RK, Frazier OH. Atrial fibrillation after cardiac transplantation: experience in 498 consecutive cases. Ann Thorac Surg 2008;85:56–58.
8. Shumway NE, Lower RR, Stofer RC. Transplantation of the heart. Adv Surg 1966;2:265–284.
9. Edgerton JR, Jackman WM, Mack MJ. Minimally invasive pulmonary vein isolation and partial autonomic denervation for surgical treatment of atrial fibrillation. J Interv Card Electrophysiol 2007;20:89–93.
10. Woo GW, Schofield RS, Klodell CT, Pauly DF, Hill JA, Aranda JM Jr. Atrial fibrillation as a cause of left ventricular dysfunction after cardiac transplantation. J Heart Lung Transplant 2006;25:131–133.
11. Lefroy DC, Fang JC, Stevenson LW, Hartley LH, Friedman PL, Stevenson WG. Recipient-to-donor atrioatrial conduction after orthotopic heart transplantation: surface electrocardiographic features and estimated prevalence. Am J Cardiol 1998;82:444–450.
12. Anselme F, Saoudi N, Redonnet M, Letac B. Atrioatrial conduction after orthotopic heart transplantation. J Am Coll Cardiol 1994;24:185–189.
13. Strohmer B, Chen PS, Hwang C. Radiofrequency ablation of focal atrial tachycardia and atrioatrial conduction from recipient to donor after orthotopic heart transplantation. J Cardiovasc Electrophysiol 2000;11:1165–1169.