Two hearts beating out of time: Mapping and ablation of concurrent atrial fibrillation and macroreentrant left atrial flutter in a transplanted heart

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Introduction

Various arrhythmias have been reported following orthotopic heart transplantation (OHT). In 1 cohort of OHT patients, the prevalence of atrial fibrillation (AF) and atrial flutter (AFL) was 0.3% and 2.8%, respectively, and 1.3% for other forms of supraventricular tachycardias (SVT), including focal atrial, atrioventricular nodal reentrant, and atrioventricular reentrant tachycardias.[1] The low prevalence of AF was attributed to the surgical technique involved in OHT, which commonly anastomoses the entire posterior left atrium (LA) of the recipient heart, including the pulmonary veins, with the anterior LA of the donor heart, effectively electrically isolating the donor heart from pulmonary vein ectopy, a common trigger of AF.[2] We report on a patient who presented with an atypical AFL after OHT. He underwent an electrophysiological study with electroanatomic activation mapping. During this we visualized AFL in the transplanted heart and AF in the remnant posterior LA simultaneously. Following catheter ablation, sinus rhythm was restored in the transplanted heart, leaving the posterior LA in AF.

KEY TEACHING POINTS

- Orthotopic heart transplantation (OHT) commonly anastomoses the entire posterior left atrium (LA) of the recipient heart, including the pulmonary veins, with the anterior LA of the donor heart. However, surgical technique may vary depending on individual circumstances and lack of this information can make interpretation of 3D electroanatomical mapping challenging.
- OHT can be associated with various forms of arrhythmia, including atrial fibrillation (AF) and atrial flutter. AF is less common in transplanted hearts, since the pulmonary veins are usually electrically isolated by anastomotic scar.
- It is important to understand whether AF in the remnant posterior LA is implicated at all in the arrhythmia in the donor heart via electrical reconnections that can develop through the anastomosis or if it remains completely isolated, in order to formulate an appropriate ablation strategy.

Case report

This was a case of a 55-year-old man with a background of OHT performed in 2012 following spontaneous rupture of a congenital sinus of Valsalva aneurysm, resulting in left-to-right shunting and rapid progressive heart failure associated with dilated cardiomyopathy. He also had a history of essential hypertension, hypercholesterolemia, type 2 diabetes on insulin therapy, and gout.

He developed AFL in 2019, which was associated with symptoms of breathlessness, subsequently undergoing DC cardioversion with improvement in his symptoms. Medical therapy included carvedilol 25 mg twice a day and rivaroxaban 20 mg once a day. AFL recurred despite medical therapy, with breathlessness once again, and he was therefore referred for consideration of catheter ablation.

A 12-lead electrocardiogram (Figure 1) had shown an atypical AFL with prominent tented positive flutter waves in the right-sided precordial leads, 4:1 conduction, an atrial rate of around 300 beats per minute, and a ventricular rate of around 75 beats per minute. Echocardiography showed normal biventricular function and mild mitral regurgitation, and the LA was severely dilated.

He came forward for an electrophysiology study utilizing CARTO 3D mapping (Biosense Webster, CA). He remained in atypical AFL from the start of the procedure. This was performed under local anesthetic and conscious sedation. In keeping with our practice, we ensured uninterrupted

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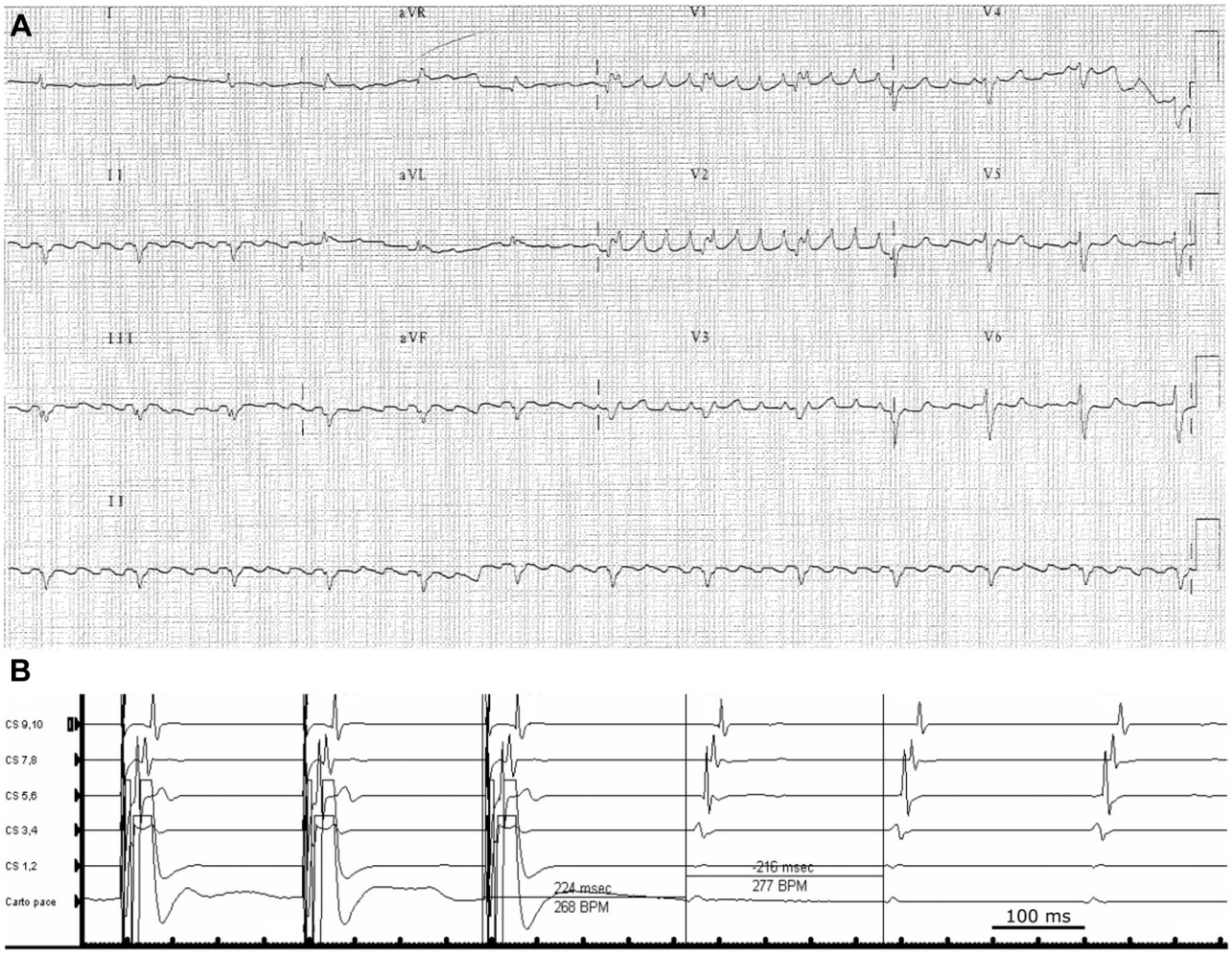

Figure 1 **A:** Preprocedure 12-lead electrocardiogram showing atrial flutter with 4:1 ventricular conduction and atypical atrial flutter waves seen maximally in leads V_1 and V_2. **B:** Intracardiac signal recording in atrial flutter with a distal-to-proximal coronary sinus (CS) activation pattern and cycle length of 216 ms (right-hand side of panel). We entrained the tachycardia by pacing from distal CS at 200 ms and demonstrated that we were driving the atrium with 1:1 conduction. CS activation was similar during pacing and tachycardia. The postpacing interval minus tachycardia cycle length was 8 ms. Similar findings were seen from the proximal CS, suggesting a macroreentrant circuit.

therapeutic anticoagulation but did not perform a transoesophageal echocardiogram before the case. Three right femoral vein punctures were performed under ultrasound guidance and 3 sheaths were placed into the vein. A steerable decapolar catheter (Biosense Webster) was placed in the coronary sinus (CS), demonstrating a distal-to-proximal activation pattern suggesting an LA origin (atrial cycle length 213 ms), with variable AV conduction seen on the surface electrocardiogram (Figure 1). Entrainment from distal CS resulted in a slightly shorter postpacing interval than from proximal CS. A PentaRay mapping catheter (Biosense Webster) was used to map the right atrium (RA) initially, which demonstrated late passive activation from the LA. A single transseptal puncture was performed using a SL0 sheath and BRK1 needle (Abbott, IL), through which 2 sheaths were subsequently advanced into the LA. Intravenous heparin was used for anticoagulation and the activated clotting time was kept above 300 seconds while catheters were in the LA.

An activation and voltage map of the LA were created with the PentaRay catheter (Figure 2). This demonstrated a macroreentrant flutter circuit, which traveled around the anterior LA via the mitral isthmus (cycle length 215 ms). Simultaneously, the posterior LA demonstrated more fractionated signals with a more rapid cycle length (142 ms), in keeping with AF. AF and AFL were separated by a low-voltage line of scar. We did not see any obvious other low-voltage areas peripheral to the low-voltage line. Figure 2 shows the voltage map in flutter with a scale of 0.2–0.5 mV; the area near the anterior line is >0.5 mV. This can be contrasted with the map created during left atrial appendage (LAA) pacing following ablation (Figure 3), where ablated tissue in the same area shows considerable low voltage/scar.

We used the SmartTouch SF catheter (Biosense Webster) in a power-controlled mode at 40 W, irrigation of 30 mL/min. We targeted an impedance drop of 10 ohms. We did not use ablation index, as we do not feel there are validated measures for the mitral isthmus. Radiofrequency energy was applied to the mitral isthmus, first endocardially and finally from within the CS, and sinus rhythm was restored during ablation.

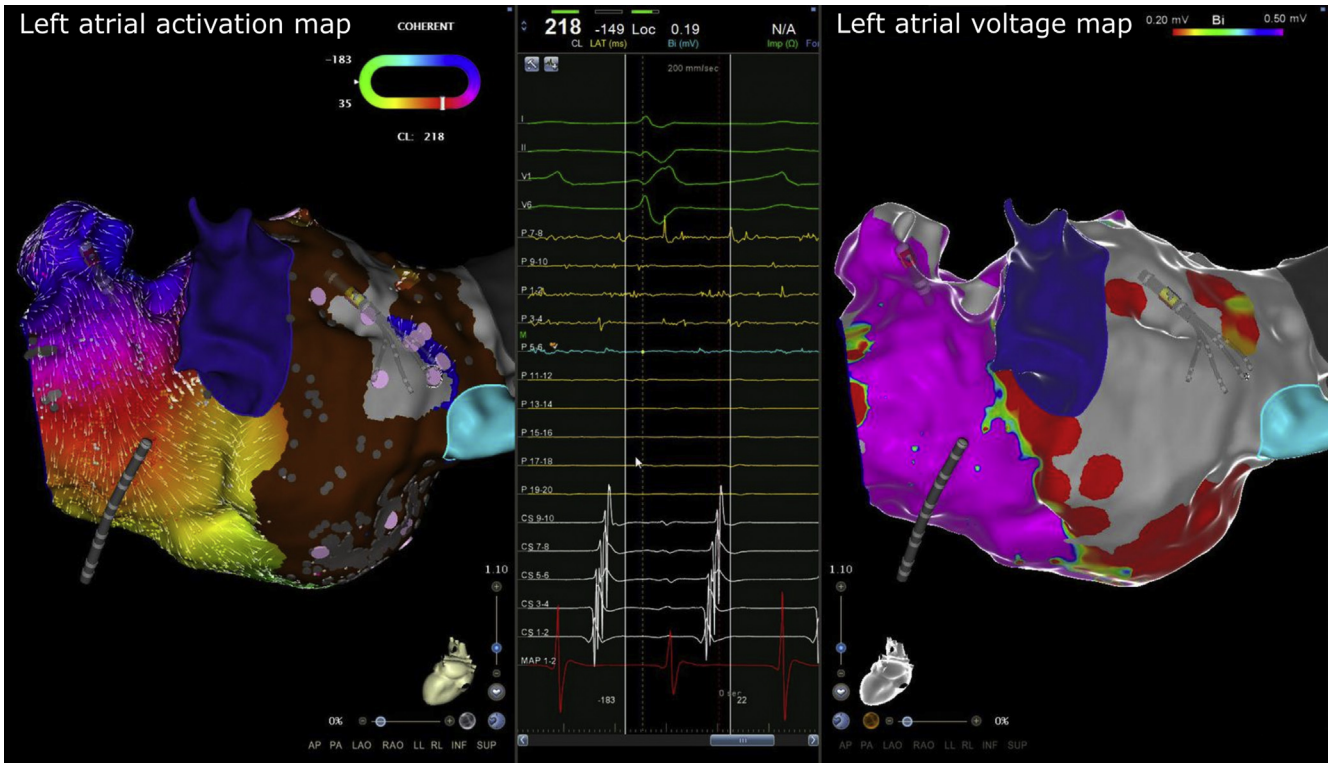

Figure 2 A 3D map of the left atrium (LA) in a left lateral orientation showing activation and voltage maps. The activation map shows a clockwise mitral isthmus–dependent atrial flutter in the anterior LA and disorganized signals in the posterior LA, representing atrial fibrillation. The dark blue area represents the left upper and lower pulmonary veins. The voltage map reveals a clear distinction in signal anterior to the pulmonary veins, representing the anastomosis between donor and recipient portions of the LA. Signals from the surface electrocardiogram, coronary sinus catheter, PentaRay catheter (Biosense Webster, CA) positioned in the posterior LA, and ablation catheter (positioned in the LA appendage) are shown.

Repeat activation mapping demonstrated block across the mitral isthmus, while the posterior LA remained in AF (Figure 3). We paced in the LAA demonstrating 100 ms before CS activation was seen and a proximal-to-distal conduction in the CS catheter. We then paced CS proximal and demonstrated a similar time to activation in the map catheter, which was still in the LAA. We did not attempt to reinduce arrhythmia.

Total ablation time was 488 seconds and total procedure time was 2 hours 36 minutes. There were no acute complications from the procedure. He was discharged from hospital with no medication changes. At follow-up in clinic 3 months later he had remained in sinus rhythm; a 24-hour tape in the meantime had shown sinus rhythm throughout, and he reported feeling less breathless, with no further palpitations.

Discussion

Surgical techniques used for heart transplantation vary, and a clear understanding of previous interventions is of great importance in interpreting subsequent clinic arrhythmias. Bicaval OHT is the most common technique used today, which anastomoses the donor heart to the recipient at the inferior and superior vena cavae, pulmonary artery, aorta, and LA. When removing the recipient's heart, the LA is dissected anteriorly to the pulmonary veins, while on the donor heart an LA cuff is prepared by joining the pulmonary vein orifices.[3] More classically, an anastomosis may be made within the RA rather than at the vena cavae, while other adjustments may need to be made to account for distortions present as a result of previous cardiac surgery or congenital abnormalities.[4] It can therefore be a challenge to interpret the initial information being gained during mapping if the full details of the surgical technique previously used are not known.

Review of cases of SVT following OHT suggests that arrhythmias can occur anytime from months to years after surgery and may be related to acute rejection episodes or transplant-related coronary disease.[5] The electrophysiological properties of remnant atrial tissue have previously been studied, demonstrating with intracardiac recordings that the remnant RA of 10 out of 50 transplant patients exhibited AF or AFL.[6] These arrhythmias have been shown to be clinically relevant in some patients, with case reports of conduction from the remnant atrial tissue to the donor heart. This can occur from both the remnant RA and LA/pulmonary veins. Although the remnant atrial tissue can be the origin of the arrhythmia, there are a wide variety of other mechanisms of tachycardia that have also been seen, including macroreentrant circuits within the donor heart, macroreentrant circuits utilizing lines of conduction block created by surgical anastomosis, focal reentrant arrhythmias in the donor hearts, and atrioventricular nodal reentrant tachycardias within the donor heart. Successful catheter ablation of various SVT post-transplant have been described for all the above arrhythmia mechanisms.[7]

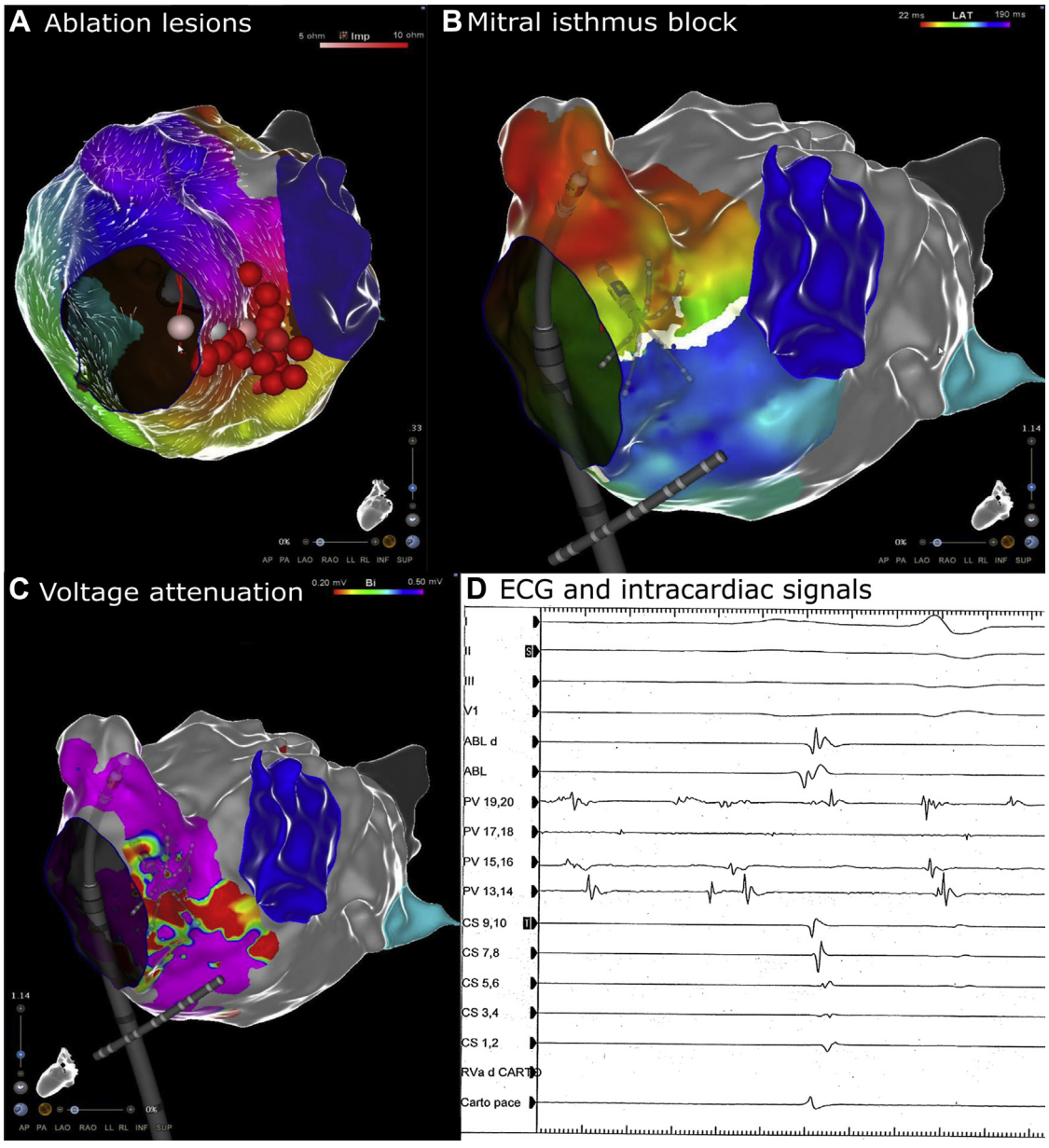

Figure 3 **A:** A 3D map of the left atrium (LA) in left anterior oblique orientation showing a mitral isthmus–dependent flutter activation pattern displaying a mitral isthmus ablation lesion set (*red dots* marking impedance drop with range of 5–10 ohms). **B:** Activation map while pacing from left atrial appendage via the ablation catheter demonstrating mitral isthmus block at the site of ablation. **C:** Voltage map demonstrating attenuated signals following ablation (*red*) previously seen as normal (*pink*) in Figure 2 before ablation (range 0.2–0.5 mV). **D:** Intracardiac signals after ablation showing sinus rhythm restored in the donor heart (P wave visible on surface electrocardiogram, coronary sinus showing proximal-to-distal activation). PentaRay signals from the posterior native LA demonstrate that this area remained in atrial fibrillation in isolation from the donor heart.

Macroreentrant arrhythmias seem to account for the majority of arrhythmias in heart transplant patients, and the commonest circuit reported is "typical" counterclockwise flutter in the donor heart. Even in cases in which lines of block created by surgical anastomosis are critical in the arrhythmia, they are predominantly located in the right atrium. The importance of the surgical technique used is demonstrated by a finding of less AFL and atrial tachycardia in patients for whom the bicaval surgical technique was used, partly owing to fewer arrhythmias arising from an interatrial suture line.[8]

In our case we were presented with remnant tissue in AF but a more organized rhythm in the donor heart. The fact that CS distal was leading CS proximal and an activation map of the RA showing passive activation demonstrated that we were looking at an LA arrhythmia. As described above, this is uncommon, although it has been seen in cases with residual electrical conduction from remnant LA tissue to the donor heart. In our case the regular cycle length in the donor heart pointed away from conduction from the remnant heart into the LA. In addition to this, we were able to map the entire

tachycardia cycle length in the LA with the activation pattern clearly supporting a mitral isthmus–dependent flutter.

Once we had restored sinus rhythm with a mitral isthmus line, the repeat electroanatomic maps were able to prove that the anterior and posterior portions of the LA were completely isolated at the surgical anastomosis, with AF persisting in the posterior remnant, electrically independent of sinus rhythm in the donor heart, providing an elegant demonstration of the aftereffects of OHT and how the surgical technique can affect subsequent arrhythmia generation.

Conclusion

We report on a patient with a previous OHT who went on to develop AFL. This was mapped to a reentrant circuit within the donor LA conducting via the mitral isthmus. A clear suture line was visualized around the posterior wall of the LA encompassing all 4 pulmonary veins, beyond which the recipient remnant of the LA exhibited AF that was completely isolated from the donor heart. Sinus rhythm was restored in the donor heart after a line of catheter ablation from the mitral valve annulus to the suture line area closest to the left lower pulmonary vein, while the remnant posterior LA remained in AF.

Acknowledgment

All patient data have been kept confidential and appropriate consent has been obtained from the patient.

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