Case report

A 64-year-old male with prior orthotopic heart transplant (biventricular), allograft vasculopathy, and dual-chamber pacemaker implantation for donor sinus node dysfunction presented with complaints of shortness of breath, fatigue, and exercise intolerance. A 12-lead electrocardiogram showed what appeared to be an atrial tachycardia (AT) with right bundle branch block aberrancy (Figure 1). Pacemaker interrogation showed AT with 2:1 and at times variable conduction and a ventricular rate of ~115 bpm.

During the electrophysiology study, a decapolar catheter was placed in the coronary sinus (CS), and a duodecapolar (Halo™) catheter was placed in the right atrium. The initial tracing obtained (Figure 2) showed two different ATs, one with a cycle length (CL) of 270 ms as seen in the Halo catheter and another with a CL of 340 ms with a proximal to distal CS activation. Given history of biventricular anastomosis, the AT recorded on the CS catheter was presumed to be the donor AT. Pacing at a faster rate (250 ms) from Halo bipole 17,18 accelerated the AT recorded on the Halo catheter to the pacing CL without affecting the AT recorded on the CS catheter (Figure 3). This suggested that perhaps the faster AT originated from the recipient part of the atrium and that there was conduction block between these two segments of the right atrium, resulting from an intact anastomotic suture line.

The ablation catheter was positioned at the donor tricuspid isthmus (identified by fluoroscopic and electrogram guidance), and at this location concealed entrainment was seen with post-pacing interval matching tachycardia CL (Figure 4), confirming the donor AT to be tricuspid isthmus-dependent flutter. Catheter ablation was performed from the tricuspid annulus to the anastomotic suture line between the donor and recipient atria, resulting in termination of atrial flutter. There was complete atrial asystole in the donor atrium upon termination of atrial flutter, and so the patient was atrially paced using the pacemaker.

Immediately afterwards, three different rhythms were seen in the right atrium (Figure 5). The recipient part of the atrium still appeared to be in a fast AT (now with a CL of 190 ms) with complete dissociation from the donor atrium, which showed an atrial paced rhythm at 800 ms. Most interestingly, the ablation catheter, which at this time was positioned in the low lateral region of the right atrium, recorded a third rhythm that is best described as an irregular AT. The respective positions of the Halo, ablation, and CS catheter at the time of this tracing, in the right anterior oblique fluoroscopic view are shown in Figure 6.

A longer strip at short sweep speed (Figure 7) shows no consistent relationship between the irregular AT recorded by the ablation catheter and the donor atrium or the fast AT in the recipient part of the atrium. Also, atrial pacing from the donor atrium did not seem to conduct into the segment showing the irregular AT (Figure 7). A partial voltage map of the right atrium (Ensite Velocity™, St. Jude Medical, Minneapolis, MN) showed an electrically silent suture line between the
Figure 1: Baseline 12-lead electrocardiogram showing what appears to be an atrial tachycardia with 2:1 AV block and right bundle branch block.

Figure 2: Intracardiac tracings showing two different atrial tachycardias. The coronary sinus catheter, located in the donor atrium, shows an atrial tachycardia with a cycle length of 340 ms with proximal to distal activation. The Halo catheter shows an atrial tachycardia with a cycle length of 270 ms. Shown are surface leads I, II, aVF, and V1 and intracardiac recordings from a duodecapolar (Halo), coronary sinus (CS), and right ventricular (RV) catheters.
Figure 3: Pacing at a faster rate (250 ms) from Halo 17, 18 entrains the atrial tachycardia recorded on the Halo catheter and dissociates it from the atrial tachycardia in the donor atrium, demonstrating conduction block between this region and the donor part of the atrium. Abbreviations are the same as in Figure 2.

Figure 4: Entrainment of donor atrial flutter from ablation catheter placed at the tricuspid isthmus shows concealed entrainment with post-pacing interval equal to the flutter cycle length, proving isthmus-dependency for the clinical donor atrial flutter. Note that the atrial tachycardia (cycle length 270 ms) originally seen in the Halo catheter has now changed to a different, faster atrial tachycardia (cycle length 180–190 ms) but is still dissociated from the donor atrial flutter. Shown are surface leads I, II, aVF, and V1 and intracardiac recordings from a duodecapolar (Halo), coronary sinus (CS), ablation (ABL) and right ventricular (RV) catheters.
donor and recipient atrium (Figure 8a,b). The source of the irregular AT appeared to be a blind pouch in the low lateral right atrium, likely part of the donor, and was electrically isolated from both the recipient as well as the rest of the donor right atrium. Thus, this electrically isolated segment, likely due to scarring from surgery, along with the recipient and the rest of the donor atria were responsible for the coexistence of three different ATs, all electrically dissociated from each other, in this orthotopic heart transplant patient.

Successful ablation of tricuspid isthmus-dependent atrial flutter, the culprit arrhythmia in this case, resulted in resolution of patient symptoms. Given electrical dissociation of the ATs in the recipient and in the electrically isolated area in the donor from the rest of the donor atria, they were not ablated and the patient was continued on anticoagulation. During a 6-month follow-up period, the patient has remained free of ATs in the donor atria.

Discussion

Atrial fibrillation is rarely seen in stable, chronic cardiac allografts, except in acute rejection or allograft vasculopathy and is likely due to the denervated status of the transplanted heart.1,2 Macreoreentrant atrial arrhythmias, however, are not uncommon after orthotopic heart transplant, occurring in anywhere from 9% to 15% of transplant recipients.1,3 Surgical technique has been implicated in the development of atrial flutter, with more studies showing a higher incidence in patients with biatrial anastomosis.3,4 The most common mechanism for atrial flutter post-heart transplant is cavotricuspid isthmus-dependent macroreentry,1,5 and catheter ablation of the isthmus between the donor tricuspid annulus to the posterior anastomotic suture line between the donor and recipient atrium has been demonstrated to be curative.1,6,7

An intact anastomotic suture line electrically disconnects the recipient from the donor atrium. It is not uncommon to see separate sinus activity in the donor and recipient atrium. Less commonly, atrial arrhythmias can be seen in the recipient atrium with sinus rhythm in the donor or vice versa.8 Two case reports have described different ATs coexisting in the transplanted heart that were electrically dissociated from each other.9,10 This can create confusion on the surface electrocardiogram regarding the underlying rhythm and whether there is need for anticoagulation.

We describe, for the first time to our knowledge, a case with three different atrial rhythms coexisting simultaneously in the right atrium of a transplanted heart. The recipient part of the atrium demonstrated a fast AT with a CL of 190 ms, and the donor atrium had typical, isthmus-dependent atrial flutter, which was the patient’s clinical tachycardia. The third rhythm was an irregular
AT noted in a segment of the low lateral right atrium, which by electroanatomic mapping was found to be anterior to the atrial anastomotic suture line. All three rhythms were dissociated from each other. Long strips did not show any specific pattern of conduction block or variable entrance into this segment in the low lateral atrium, either from the donor or the recipient atria. Moreover, atrial pacing from the donor atrium did not show conduction into this area. Other maneuvers to confirm electrical isolation, which were not done in this case, include 1) activation mapping of each segment using its own reference, and 2) cardioversion and pacing from each chamber (donor, recipient, and the isolated segment in the low lateral right atrium) to confirm entrance and exit block.

Based on our evaluation, we believe that the part of the atrium that supported the third AT likely was a part of the donor atrium that became electrically isolated from the rest of the donor atria. This was supported by the fact that no convincing anatomic abnormality, different from what is normally seen in a biatrial anastomosis, was seen on transesophageal echocardiography. A combination of surgical technique and atrial myopathy that happened over the duration of the allograft likely contributed to electrical isolation of this segment.

Although the exact mechanism of the irregular AT remains unclear, it is possible that this irregular rhythm could represent a form of organized atrial fibrillation. Inhomogeneous and extensive scarring in this segment of the right atrium in all likelihood contributed to the AT.
age of the allograft (15 years post transplant) could have resulted in the slowness and irregularity of the rhythm. Whether denervation or late reinnervation plays a role requires further study.

The anatomical and electrical substrate abnormalities encountered as part of a biatrial anastomosis, especially when more than one AT is present, poses unique challenges in catheter positioning and interpretation of diagnostic data. Close attention should be paid to CLs and subtle changes in rhythm. Diagnosing the arrhythmia, identifying the mechanism(s) of the culprit rhythms, and determining the correct chamber to ablate becomes critical. Careful electrogram analysis and use of voltage, activation, and entrainment mapping to define boundaries of the respective segments of the donor and recipient atria and to confirm electrical dissociation of one rhythm to the other becomes essential to safely and successfully ablate the culprit arrhythmia.

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References


