Case study

A Caucasian male aged 56 years underwent successful bilateral orthotopic sequential lung transplant for idiopathic pulmonary fibrosis. In the postoperative period, he experienced brief episodes of atrial arrhythmia that settled with β-adrenergic blockade. Subsequently, he described non-sustained episodes of palpitation without documentation of any sustained arrhythmias. A year following surgery, he began to experience sustained episodes lasting hours. Echocardiography revealed no evidence of structural heart disease and left atrial diameter of 3.8 cm. Flecainide was commenced with relief of symptoms. However, 6 months later, recurrent atrial arrhythmias were documented on several electrocardiograms (Figure 1) at different hospitalizations. Because of persisting symptoms despite antiarrhythmic drugs, he underwent electrophysiological (EP) studies and ablation.

Baseline intervals were normal. Programmed stimulation induced sustained right atrial flutter at a cycle length (CL) of 258 ms that was successfully terminated with ablation to create conduction block in the cavo-tricuspid isthmus (Figure 2a). Programmed stimulation on isoproterenol induced a rapid sustained atrial tachycardia (AT) at a CL of 190 ms. An electroanatomic map of the left atrium was created using a Carto mapping system (Biosense Webster, Inc., Diamond Bar, CA). Activation mapping revealed earliest activation anterior to the right superior pulmonary vein (PV) in the recipient cuff of the right venous antrum where continuous low-amplitude high-frequency signals were evident (Figure 2b). Efforts to entrain the tachycardia in the area failed to elicit atrial capture. Within the right upper PV, a circular catheter recorded slow isolated potentials. Ablation in the anterior antrum of the right upper vein proximal to the anastomotic site, based on the electrogram morphology, terminated the tachycardia. No electrical activity was recorded within the other three PVs. Following the above ablation, isoproterenol stimulation induced a third AT at a CL of 375 ms (Figure 2c). Earliest activation was recorded in the His catheter. Pacing at progressively shorter CLs failed to demonstrate progressive fusion. Left and right atrial activation was contained within an 80-ms window, suggesting a focal tachycardia. Ablation in the region of the medial mitral annulus based on early activation failed to influence tachycardia. Finally, the aortic root was mapped and registered earliest activation in the region of the non-coronary sinus where a single radiofrequency lesion terminated tachycardia (Figure 3). No further arrhythmias were inducible with isoproterenol infusion to 20 μg/min and programmed atrial stimulation. The patient has remained arrhythmia-free off anti-arrhythmic medications for 3 months.

Discussion

In the case reported, multiple atrial arrhythmias due to varying mechanisms were documented in the same patient, a year following bilateral lung transplantation.
Figure 1: Twelve-lead electrocardiograms of documented atrial arrhythmias. (a) Common right atrial flutter; (b) shows a disorganized atrial arrhythmia. (c) The atrial arrhythmia appears more regular and is likely an atrial tachycardia.
Figure 2: Intracardiac recordings during electrophysiological study and ablation. Traces from top to bottom show surface electrocardiogram leads I, II, III, V1, V5, and bipolar electrograms from a duodecapolar catheter with proximal 10 poles in the lateral right atrium (RA 9,10 is proximal in the high RA) and the distal 10 poles in the coronary sinus (CS 9,10 is proximal). The initial arrhythmia induced was common right atrial flutter that was entrained by pacing from the ablation catheter positioned in the cavotricuspid isthmus with a post-pacing interval that closely approximated the tachycardia cycle length (a). Ablation in the cavotricuspid isthmus to create conduction block in the isthmus terminated atrial flutter. (b) A rapid atrial tachycardia with earliest activation in the anterior antrum of the right superior pulmonary vein proximal to the area of anastomosis. Distal to the anastomosis, slow isolated pulmonary vein potential was evident (not shown). The recording on the ablation catheter
have been described and can lead to PV-mediated ATs. In addition, partial reconnections between the recipient LA and donor PVs can allow for re-entry around them. In the present case, all PVs had entrance block into them at the anastomotic level with isolated independent discharges from the musculature in the right upper PV.

Of the three separate arrhythmias identified in this case, none can be confirmed as a direct consequence of the patient’s lung transplant surgery. There was clinical documentation of possibly all three arrhythmias (Figure 1) and hence they were unlikely to be a laboratory finding resulting from programmed stimulation on isoproterenol. Common right atrial flutter, which was clinically documented (Figure 1a) and the first arrhythmia induced at EP study in this patient, is often triggered by premature atrial beats, bursts of AT or fibrillation, and is a well-recognized finding soon after cardiac surgery and lung transplant in children. In the series by See et al, one of four patients who underwent EP studies for post-lung transplant atrial arrhythmia had right atrial flutter dependent on the cavitricuspid isthmus. The other three involved the left atrium with perimtrial re-entry in one patient, re-entry involving the left atrial roof between the right and left venous anter anastomoses in a second patient, and one case of focal AT due to vein-mediated tachycardia with conduction across the anastomosis.

The second arrhythmia induced in our patient was a rapid AT localized by activation mapping to the anterior aspect of the recipient atrium proximal to the anastomotic site of the right veins. The mechanism of this arrhythmia was probably focal micro-re-entry, although this could not be confirmed by entrainment pacing or resetting responses because of an inability to capture the site by pacing. Continuous low-amplitude signals spanning the tachycardia cycle length, as documented in our case (Figure 2b), are typical of scar-related focal micro-re-entry. In addition, areas of patchy scarring adjacent to the anastomotic sites of the venous antrum following lung transplant surgery can provide the typical substrate for focal micro-re-entry.

The third atrial arrhythmia induced was clearly a focal AT. Although ATs are common in patients with lung disease, multifocal atrial tachycardia is the arrhythmia frequently encountered. The finding of a focal AT in close proximity to the central fibrous body has not been specifically described in association with chronic lung disease or lung transplantation. Nevertheless, the occurrence of ectopic AT with earliest activation in the His bundle region should prompt mapping of the aortic root for AT focus that can be accessed from the non-coronary sinus.

This case exemplifies the range of atrial arrhythmias that can manifest in patients late after lung transplantation. Ablation strategies should anticipate macro- and micro-re-entry as well as focal arrhythmias in such patients.

References