Cardiac transplantation carries a known risk of atrial arrhythmias. We present an unusual case and discuss the mechanism of an atrial arrhythmia arising from a donor pulmonary vein in a lung transplant recipient. A 59-year-old white woman with type 2 diabetes mellitus, obstructive sleep apnea, and hypertension underwent bilateral lung transplantation in 2005 because of end stage cystic fibrosis. She had no prior history of arrhythmias or structural heart disease. At the time of bilateral lung transplantation, the donor’s pulmonary veins along with the surrounding left atrial cuff were sewn to the corresponding area of the recipient’s left atrium. Subsequently, in 2006, she developed episodes of symptomatic palpitations. ECG and Holter monitor revealed paroxysmal atrial tachycardia with cycle length 400 ms and 2:1 conduction to the ventricle. P waves were broad and positive in V1 and isoelectric in I and aVL (Figure 1).

Initial medical therapy did not suppress the tachycardia. Electrophysiology study including 3-dimensional electroanatomical mapping revealed a focal atrial tachycardia, cycle length 420 ms, originating from the donor left superior pulmonary vein ostium. The activation wave front crossed the anastomosis line between the donor pulmonary veins and the recipient left atrium (Figure 2). The left-sided pulmonary veins were successfully isolated with circumferential radiofrequency ablation, which restored sinus rhythm. She had no recurrent palpitations or documented arrhythmias over 4 years of follow-up.

Conduction across suture lines has been reported in cardiac surgeries like Maze procedures and Fontan surgery for congenital heart disease, as well as in orthoptic heart transplantation. However, this phenomenon of an atrial arrhythmia originating from a donor pulmonary vein ostium and crossing the left atrial suture line is rare. Potential mechanisms of an atrial tachycardia crossing suture lines include bridging myocardium growing across suture lines, excitatory tissue inducing depolarization in nearby areas, and mechanical coupling causing generation of action potentials. Animal studies done in the last century revealed that mechanical excitation of 2 juxtaposed tissues can mutually stimulate each other because of their intrinsic pacemaker capacity.

Atrial tachycardias can arise anywhere in the atria, pulmonary veins, or the coronary sinus. Focal atrial tachycardias can be treated by direct ablation at the origin of the tachycardia or by isolating the area where the tachycardia is originating, as was done in this case with pulmonary vein isolation.

Our case demonstrates two interesting points: (1) Atrial tachycardia can originate from donor pulmonary veins after lung transplantation, and (2) the electric activation wave front can cross the anastomosis line between the donor pulmonary vein antrum and the recipient left atrium.

**Figure 1.** ECG showing atrial tachycardia with 2:1 conduction and voltage criteria for left ventricular hypertrophy with reciprocal ST-T wave changes.
Disclosures

None.

References


Figure 2. Left. A 3-dimensional electroanatomical bipolar voltage map of the left atrium (LA), pulmonary veins (PV), and coronary sinus from a posteroanterior view. The anastomosis line between the donor left PV antrum and recipient LA is noted by the line of low voltage (red and green color). Right, Local activation time map with the earliest activation shown in red. The atrial tachycardia (AT) focus originates from the donor left PV antrum and crosses the anastomosis line to activate the recipient LA.
Atrial Tachycardia Originating From a Donor Pulmonary Vein in a Lung Transplant Recipient
Mohammed Nazmul, Thomas M. Munger and Brian D. Powell

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