

Atrial Flutter in a Post-Transplant Recipient

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Ten percent of patients with heart transplantation develop symptomatic supraventricular arrhythmia following heart transplantation. We present a case of coexisting donor heart counterclockwise isthmus-dependent atrial flutter and recipient atrial tachycardia in a 72 year old woman 10 years after orthotopic heart transplantation.

PATIENT DESCRIPTION

A 72 year old woman with a past medical history of chronic renal failure and ulcerative colitis underwent orthotopic heart transplantation with atrio-atrial anastomosis for idiopathic dilated cardiomyopathy 10 years previously. She was referred to our center due to recurrent episodes

of atrial flutter and repeated electrical cardioversions and failure to maintain normal sinus rhythm with amiodarone and sotalol. Thyroid function tests were normal. A cardiac biopsy excluded transplant rejection.

Echocardiogram showed good left ventricular function and an enlarged left atrium. The patient was referred for ablation. She arrived at the electrophysiology laboratory in atrial flutter [Figure A]. It was first thought to be a typical flutter because of P wave configuration (negative P wave in II, III, AVF leads and positive in V1), the presence of only one dominant P wave, and absence of two different P wave morphologies. A 10-pole catheter was placed in the coronary sinus (Dynamic-XT, Bard, Lowell, MA, USA) and a 24-pole catheter was placed around the antero-lateral tricuspid annulus (Bard, Milwaukee). The atrial activation sequence around the tricuspid valve was counterclockwise. An entrainment map was attempted several times from several locations in the cava-tricuspid isthmus with inconclusive results because of failure to capture in several locations despite

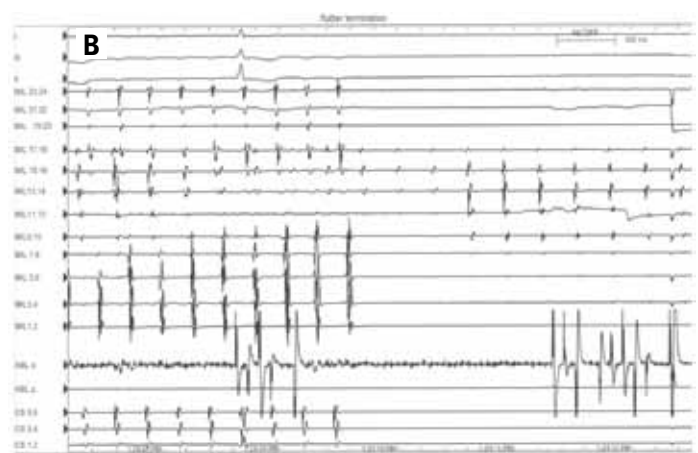
the high output and due to variable post-pacing intervals on the few occasions that capture did occur. We therefore decided to perform a three-dimensional electro-anatomical (NAV-X, St. Jude Medical, St. Paul, MN, USA) activation map in order to elucidate the arrhythmia mechanism. The activation map confirmed the diagnosis of counterclockwise typical atrial flutter. Occasionally during the study the 24-pole catheter moved to the posterior part of the right atrium, showing another tachycardia in that chamber that was dissociated from the atrial flutter in the anterior part of the right atrium (donor heart).

A successful TV-IVC isthmus linear ablation was performed from the valve annulus to the inferior vena cava. Atrial flutter converted during ablation to sinus rhythm in the donor heart, while the recipient heart remained in atrial tachycardia [Figure B]. We did not perform ablation in the recipient heart because there was evidence of electrical conduction block between the donor right atrium

TV-IVC = tricuspid valve-inferior vena cava isthmus

[A] 12-Lead ECG of the patient in atrial flutter

[B] Termination of atrial flutter during ablation, with ongoing atrial tachycardia in the recipient heart



and residual recipient RA tissue in order to minimize procedure time. Also, clinically the patient was in normal sinus rhythm and therefore would probably not benefit from any further mapping and ablation.

The patient was discharged the next day, after the procedure, and has maintained sinus rhythm for a year.

COMMENT

The transplanted heart is exposed to a number of anatomic and physiological changes that may lead to cardiac arrhythmias. Ten percent of patients with heart transplantation develop symptomatic supraventricular arrhythmia following heart transplantation [1]. The presence of a residue of native right atrium introduces the equivalent of a large region of scar in the donor atrium with a barrier-suture

line and may provide the substrate for cardiac arrhythmias [2]. Radiofrequency catheter ablation in patients with atrial flutter/tachycardia is feasible and relatively safe after heart transplantation [3,4].

We present a case of coexisting donor heart counterclockwise isthmus-dependent atrial flutter and recipient atrial tachycardia that occurred 10 years after heart transplantation. Three-dimensional electroanatomic mapping of the right atrium was used to facilitate the ablation [5]. Ablation in the recipient part of the heart was not performed because there was no evidence of conduction between the donor RA and residual recipient RA tissue as the surgical suture line provided a line of block between them. The patient remained free of atrial flutter and other supraventricular arrhythmias during the 1 year follow-up after ablation. In conclusion, typical atrial flutter in a donor heart can be cured with a standard approach (cavotricuspid isthmus line).

RA = right atrium

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Capsule

AMPK regulates NADPH homeostasis to promote tumor cell survival during energy stress

Overcoming metabolic stress is a critical step for solid tumor growth. However, the underlying mechanisms of cell death and survival under metabolic stress are not well understood. A key signalling pathway involved in metabolic adaptation is the liver kinase B1 (LKB1) AMP-activated protein kinase (AMPK) pathway. Energy stress conditions that decrease intracellular ATP levels below a certain level promote AMPK activation by LKB1. Previous studies showed that LKB1-deficient or AMPK-deficient cells are resistant to oncogenic transformation and tumorigenesis, possibly because of the function of AMPK in metabolic adaptation. However, the mechanisms by which AMPK promotes metabolic adaptation in tumor cells are not fully understood. Jeon et al. show that AMPK activation, during energy stress, prolongs cell survival by redox regulation. Under these conditions, NADPH generation by the pentose phosphate pathway is

impaired, but AMPK induces alternative routes to maintain NADPH and inhibit cell death. The inhibition of the acetyl-CoA carboxylases ACC1 and ACC2 by AMPK maintains NADPH levels by decreasing NADPH consumption in fatty acid synthesis and increasing NADPH generation by means of fatty acid oxidation. Knockdown of either ACC1 or ACC2 compensates for AMPK activation and facilitates anchorage-independent growth and solid tumor formation in vivo, whereas the activation of ACC1 or ACC2 attenuates these processes. Thus AMPK, in addition to its function in ATP homeostasis, has a key function in NADPH maintenance, which is critical for cancer cell survival under energy stress conditions, such as glucose limitations, anchorage-independent growth and solid tumor formation in vivo.

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Eitan Israeli

Without the freedom to criticize, there is no true praise

Pierre Beaumarchais (1732-1799), French playwright, watchmaker, inventor, musician, diplomat, fugitive, spy, publisher, arms dealer, satirist, financier, and revolutionary (both French and American)