Ablation of Atrial Flutter in Congenital Heart Disease

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KEYWORDS

• Transposition of the great arteries • Mustard correction
• Electrophysiology • Radiofrequency ablation

A 42-year-old woman with a history of D-transposition of the great arteries (TGA) and a Mustard correction at age 5 years presented to an outside hospital with palpitations and chest pain. She was found to be in a narrow complex regular tachycardia with a ventricular rate of approximately 230 beats per minute. She was electrically cardioverted and discharged. She then presented to another outside hospital while on vacation with the same symptoms. She was again in a narrow complex tachycardia with a ventricular rate of approximately 200 beats per minute. She was treated with intravenous diltiazem that slowed her ventricular rate to approximately 115 beats per minute. She has continued to have episodes of palpitations and chest pain. A transthoracic echocardiogram revealed severe right atrial and right ventricular enlargement as well as mild tricuspid regurgitation and a baffle lead. She was thus referred for electrophysiology study and possible ablation.

ELECTROPHYSIOLOGY STUDY

At baseline, the patient was in a narrow complex supraventricular tachycardia with an atrial cycle length of 270 milliseconds and 2-to-1 atrioventricular conduction, most consistent with atrial flutter given the cycle length and pattern of atrial depolarization (**Fig. 1**). Multipolar catheters were placed in the left atrium (LA) and the subpulmonic (left) ventricular apex (LVA) via the right femoral vein. An electroanatomic activation map of the right atrium was performed using the Biosense Webster CARTO system (Diamond Bar, CA, USA). Multiple attempts to cross the superiorly located baffle leak seen on transesophageal echocardiogram failed. As such, a 4-mm irrigated tip ablation catheter was advanced retrograde from the right femoral artery to the right atrium via the subaortic right ventricle and tricuspid valve. The electroanatomic activation map was then completed. Attempts to pace from the cavotricupsid isthmus were unsuccessful with only intermittent capture. Using the electroanatomic map and the surface electrocardiogram, we hypothesized atrial activation to be most consistent with counterclockwise atrial flutter using the cavotricuspid isthmus. Therefore, ablation of the cavotricuspid isthmus was pursued.

STRATEGY FOR RADIOFREQUENCY ABLATION

Selection of sites for ablation was based on an anatomic approach. Radiofrequency (RF) applications were given along the inferior aspect of the tricuspid annulus, the isthmus of atrial tissue between the tricuspid annulus and the inferior vena caval (IVC) orifice. The mid-to-anterior aspect of the cavotricuspid isthmus ablation was performed retrograde via the aorta to the systemic right ventricle and tricuspid annulus. A 4-mm irrigated tip ablation catheter was positioned at approximately 6 o'clock on the tricuspid annulus. Discrete RF applications targeting a power of 30 W and a 10% decrease in impedance were made from the tricuspid annulus posteriorly

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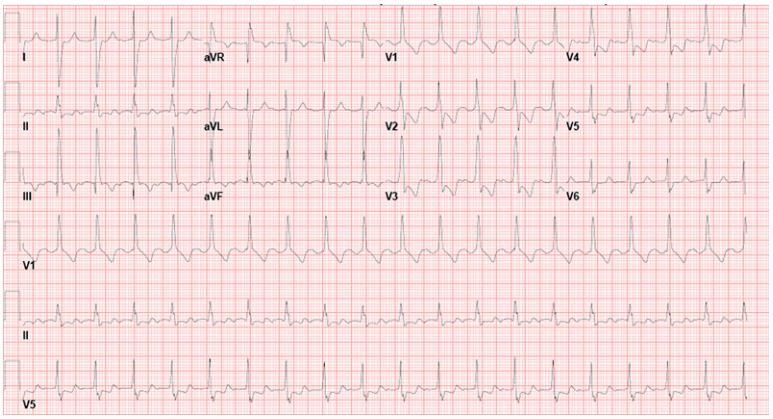

Fig. 1. Baseline electrocardiogram showing right ventricular hypertrophy with repolarization abnormality and a narrow complex supraventricular tachycardia most consistent with atrial flutter given the cycle length and pattern of atrial depolarization.

toward the IVC until the atrial baffle obstructed movement. The posterior aspect of the cavotricuspid isthmus ablation was performed by advancing the 4-mm ablation catheter to the juncture of the inferior vena cava and the atrial baffle. Discrete RF applications targeting a power of 30 W and a 10% decrease in impedance were made from the atrial baffle to the orifice of the IVC. A total of 22 discrete RF applications were made (**Fig. 2**). Although bidirectional conduction block could not be proved because of inability to pace, atrial flutter was terminated during RF ablation.

DISCUSSION

Sudden death is the leading cause of mortality in adult patients with TGA status after atrial switch correction. In a retrospective study of patients with TGA status after surgical baffling, only New York Heart Failure Association functional class and the development of supraventricular tachycardias were found to be independent risk factors for late mortality.[1] Supraventricular tachycardias have been shown to either degenerate into ventricular fibrillation resulting in sudden death or produce cardiogenic shock with subsequent death

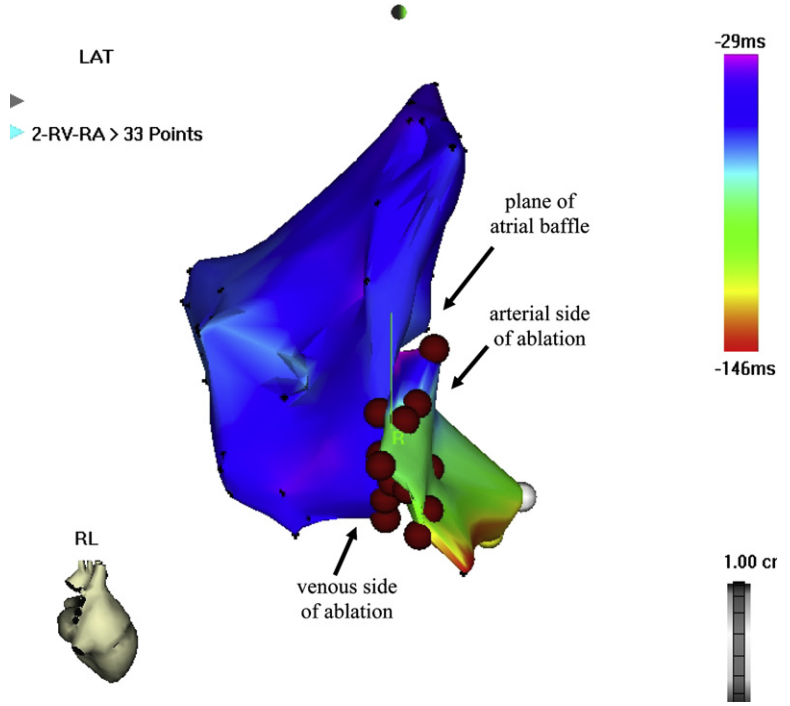

Fig. 2. Right lateral projection of electroanatomic map of atrial flutter and ablation sites (*red points*) on both sides of the atrial baffle.