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Change in Atrial Activation Pattern during Ablation of Atrial Flutter

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Abstract

Different types of supraventricular tachycardia have been reported in patients with history of surgical repair of Tetralogy of Fallot. This report presents appearance of focal atrial tachycardia during radiofrequency ablation of the cavotricuspid isthmus in typical atrial flutter.

INTRODUCTION

Supraventricular tachycardia occurs in about 20% of patients with previous surgical Tetralogy of Fallot (TOF) repair [1, 2]. Increased right atrial pressure due to chronic right ventricular pressure overload and anatomical obstacle due to atriotomy scar can cause atrial arrhythmias, mainly cavotricuspid (CTI)-dependent atrial flutter (AFL) and non-CTI-dependent AFL around the atriotomy scar or both [3].

Radiofrequency ablation of these arrhythmias has been reported in this group of patients. Electroanatomical mapping has an important role in identification of reentry circuits in patients with postoperative right atrial incisional scar and flutter and it is necessary to ablate both scar-related and typical AFL to prevent long-term recurrence [4].

CASE PRESENTATION

A 31-year-old man who performed total surgical correction of TOF at 5 years old, was referred for catheter ablation of persistent typical AFL. His baseline electrocardiogram revealed negative flutter waves in leads II, III, aVF, V5 and V6, and positive waves in lead V1 consistent with typical counterclockwise AFL with 4:1 AV conduction and right bundle branch block (RBBB) pattern.

A decapolar catheter was advanced with its proximal 9 and 10 poles placed in the proximal coronary sinus (CS) and a duodecapolar halo catheter was positioned along the tricuspid annulus, such that the distal bipole of the halo catheter was in the low lateral right atrium.

The atrial activation sequence and entrainment mapping were compatible with typical counterclockwise AFL. Linear radiofrequency ablation of CTI during arrhythmia was performed which resulted in termination of AFL, but appearance of another arrhythmia (change in atrial cycle length from 300 milliseconds to 326 milliseconds and with a different sequence of atrial activation) without restoration of sinus rhythm. P waves became positive in leads II, III and aVF and biphasic in lead V1 (Fig. 1A). This arrhythmia was compatible with focal atrial tachycardia as the atrial activation accounted for less than 60% of atrial cycle length [5]. Activation mapping was performed conventionally and arrhythmia was successfully ablated at the lateral of right atrium where the earliest activation signal achieved (96 milliseconds) (Fig. 1B).

DISCUSSION

Change of the first arrhythmia without restoration of the sinus rhythm can be described by different theories. It is probable that the second arrhythmia started de novo at the termination of the first arrhythmia. The other theory is that both arrhythmias were presented initially, but as the AFL rate was faster than the atrial tachycardia, the second arrhythmia could not be manifested. In this theory, one can argue that the mechanism of atrial tachycardia was mircoreentry (maybe around previous surgical scar of lateral atriotomy), so AFL

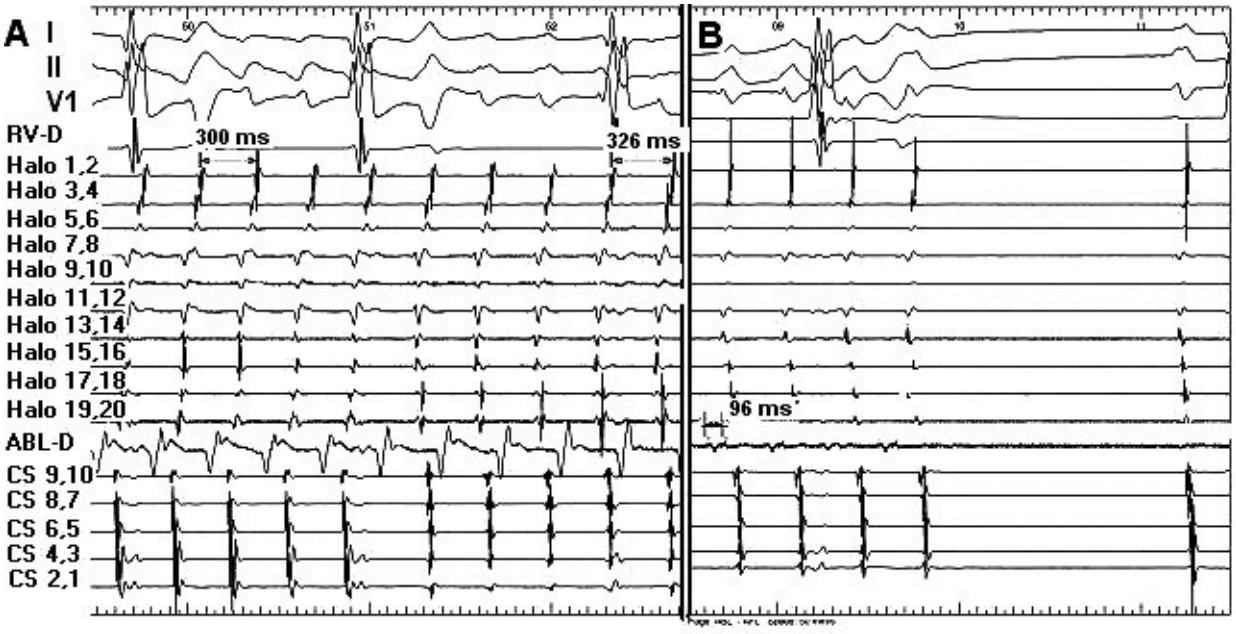

Figure 1: A) Termination of atrial flutter (300 ms) and appearing of focal atrial tachycardia (CL = 326 ms) during ablation of cavotricuspid isthmus. Note Change of intracardiac sequence and cycle length, and P wave morphology. B) Termination of focal atrial tachycardia during radiofrequency application in lateral of right atrium. Signal in ablation catheter is 96 ms earlier than surface P wave.

entrained the cycle of the micro reentry, but could not terminate it, or if the mechanism of atrial tachycardia was automaticity, due to a lower rate, it was hidden under AFL. Although it is difficult to define the mechanism of a focal AT, some principles may help to find that. Initiation and termination with programmed atrial stimulation and insensitivity to adenosine can be in favor of reentrant mechanism. Criteria for entrainment may be useful to prove the mechanism of reentrant, but due to small circuit size, it is difficult to demonstrate. By the way, regardless of the mechanism of focal atrial tachycardia, ablation can be performed with mapping the tachycardia focus. In our case we did not try to find out the main mechanism and just found its focus and ablated it [6].

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CONFLICT OF INTEREST

None of the authors had any conflicts of interest.

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