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Case Report

Irregular atrial flutter following pulmonary vein isolation for persistent atrial fibrillation

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ABSTRACT

A 65-year-old man with a history of refractory paroxysmal atrial fibrillation (AF) underwent catheter ablation for persistent AF lasting 2 months. AF was not terminated after complete isolation of the 4 pulmonary veins (PV). Instead, it was transformed to a sustained atrial tachyarrhythmia with beat-to-beat variability in the atrial cycle length. A 12-lead electrocardiogram during tachycardia showed negative flutter-like waves in the inferior leads. Entrainment pacing along the tricuspid annulus confirmed the diagnosis of irregular cavotricuspid isthmus (CTI)-dependent typical atrial flutter (AFL). Linear ablation of the CTI terminated AFL and restored sinus rhythm.

1. Introduction

The mechanism of typical counter clockwise (CCW) atrial flutter (AFL) is macroreentrant, and its cycle length is highly regular. Here, we present a rare case of typical AFL with irregular cycle lengths developing after pulmonary vein (PV) isolation. Further, we discuss the differential diagnosis of irregular atrial activities.

2. Case report

A 65-year-old man with a 6-year history of refractory paroxysmal atrial fibrillation (AF) underwent catheter ablation for persistent AF lasting 2 months. The left atrial (LA) and right atrial (RA) diameters were 39 and 32×43 mm, respectively. Extensive encircling PV isolation was performed with the double Lasso technique using a CARTO three-dimensional electro-anatomical mapping system (Biosense-Webster, Diamond Bar, CA, USA). At the beginning of the procedure, disorganized AF was present (Figs. 1A and 2), which was confirmed by the recording of a random activation sequence of the atrial potentials in the coronary sinus (CS). AF was not terminated after complete isolation of the 4 PVs. Instead, it was transformed to a sustained atrial tachyarrhythmia (ATA) with beat-to-beat variability of the atrial cycle length between 210 ms and 260 ms (Figs. 1B and 2). A 12-lead electrocardiogram during tachycardia showed negative flutter-like waves in the inferior leads and positive deflections in lead aV_L (Fig. 3). In contrast to AF before PV isolation, the irregular ATA exhibited a fixed proximal-to-distal CS activation sequence (Fig. 1B). The post-pacing interval following entrainment pacing of the anterior and posterior tricuspid annulus was nearly equal to the tachycardia cycle length (Fig. 4), consistent with irregular, cavotricuspid isthmus (CTI)-dependent, typical CCW AFL. Although the minimum cycle length of the tachycardia (210 ms) was slightly shorter than the pacing cycle length (220 ms), pacing during entrainment successfully captured the atrium. This was probably due to decremental conduction delay of the orthodromic wavefront. After the irregular ATA spontaneously transformed to regular ATA, we obtained an electro-anatomical activation map of the RA (Fig. 5), which showed a CCW tail-to-head activation sequence around the tricuspid annulus, confirming the diagnosis of typical AFL. Linear ablation of the CTI terminated AFL and restored sinus rhythm.

3. Discussion

The differential diagnosis of ATA with a constant CS activation sequence following PV isolation for AF includes (a) AF originating

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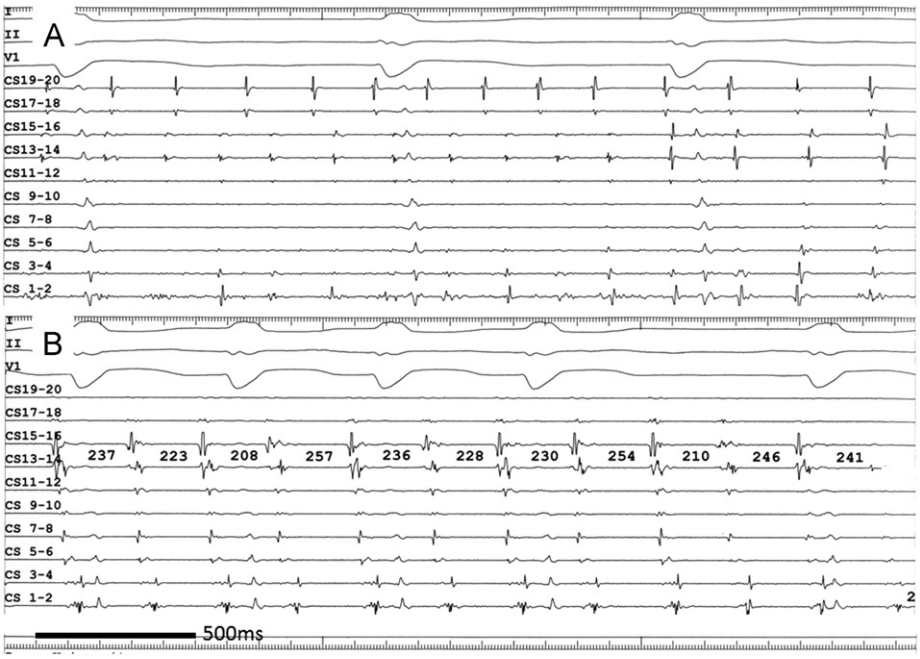

Fig. 1. Intracardiac electrograms during AF recorded at baseline (A) and when ATA evolved from AF (B). (A) The atrial activation sequence at baseline was variable. (B) The activity during ATA was irregular in the atrium, while the activation sequence in the CS was fixed. The numbers indicate the intervals between adjacent atrial potentials. I, II, and V1 = surface electrocardiogram; CS 19–20 to 1–2 = proximal-to-distal CS.

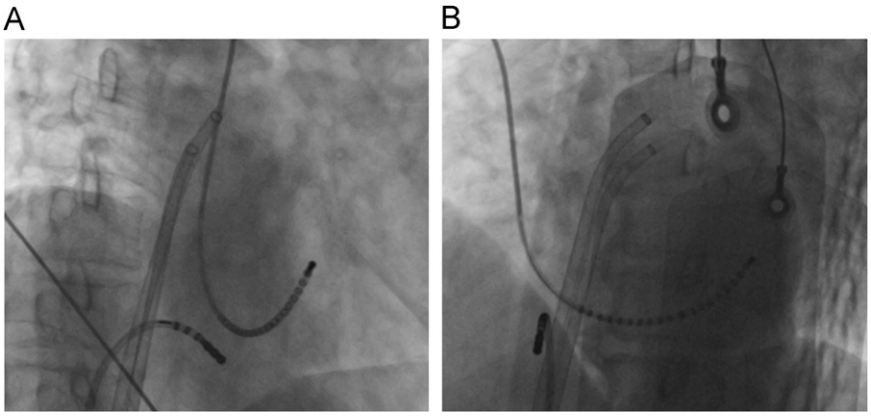

Fig. 2. Fluoroscopic views of catheter position in the right (A) and left (B) oblique projections.

from a non-PV focus [1], (b) focal ATA, and (c) macro-reentrant AFL of LA or RA origin [2–4]. First, the proximal-to-distal CS activation sequence during tachycardia excluded focal ATA originating from left side of LA outside the PV, such as the ligament of Marshall or the posterior wall [1,2]. However, it did not exclude an ATA originating from a RA focus, such as the CS ostium, the interatrial septum, or the crista terminalis [1] or a focal ATA originating from right side of the LA outside the PV [2]. Although macroreentrant AFL is an unlikely mechanism for irregular tachycardia, it was not excluded [6]. Second, the electro-anatomical activation map of the LA during tachycardia showed no atrial activity preceding the proximal CS, excluding focal ATA or macroreentrant AFL originating from the LA outside the PV. Finally, to distinguish focal ATA from macroreentrant AFL originating from the RA, we performed entrainment pacing along the tricuspid annulus (TA) and electro-anatomical activation mapping of the RA, confirming the diagnosis of typical CCW AFL.

Although spontaneous transition from AF to CTI-dependent typical AFL during and after LA ablation of AFL is often observed [3,4], irregular AFL is uncommon [5–7]. In this case, a *fixed* CS activation sequence revealed the diagnosis of AFL. Atypical flutter waves may result from abnormal LA activation [4]. Entrainment mapping at the CTI early in the procedure, even in absence of typical electrocardiographic characteristics, may prevent unnecessary LA mapping [4].

The oscillatory AFL cycle length is mediated by variations in conduction velocity and refractoriness among sites within the reentry circuit. It depends on the duration of the preceding diastolic interval and results in a complex oscillation of the cycle length, as demonstrated in animal models [8,9]. Albeit rare, cases of AFL exhibiting irregular atrial cycles have been described. Ibutilide increases the cycle length variability and diastolic interval of AFL perhaps by varying the beat-to-beat duration of action potential, refractoriness, and diastolic interval [5]. Alternating typical AFL and lower-loop reentry may cause cycle length

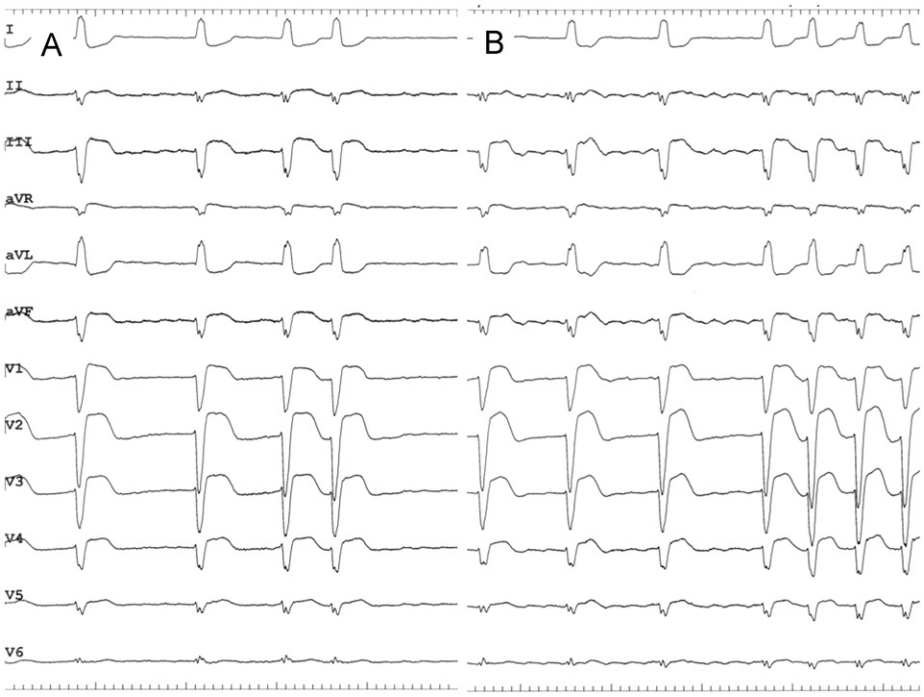

Fig. 3. Twelve-lead ECG during AF recorded at baseline (A) and when ATA evolved from AF (B).

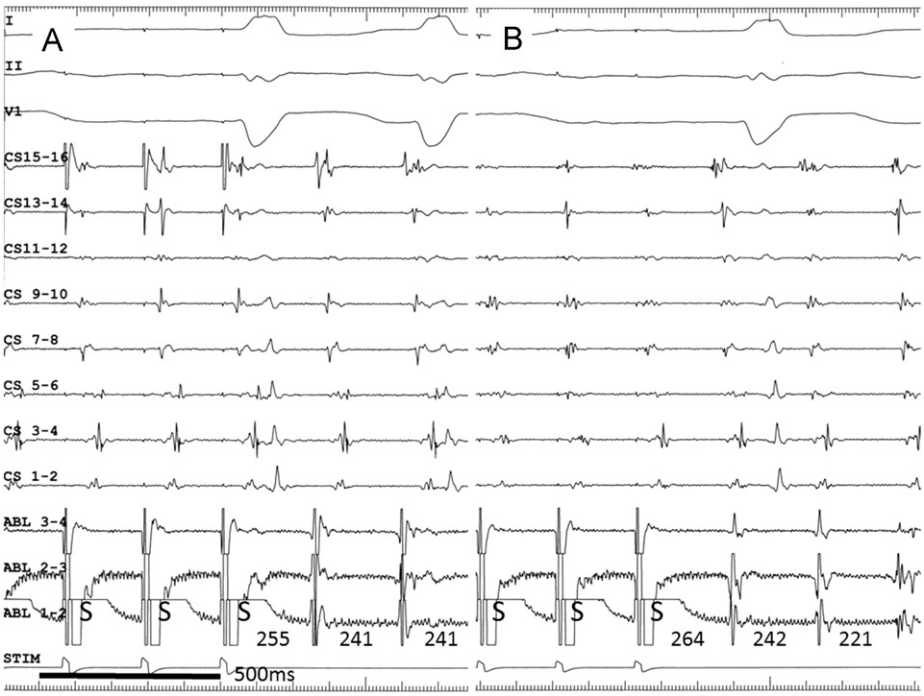

Fig. 4. Entrainment pacing at an S–S pacing cycle length of 220 ms from the cavotricuspid isthmus (A) and at an S–S pacing cycle length of 220 ms from the anterior RA along the tricuspid annulus (B). The numbers indicate the atrial cycle lengths in milliseconds. The post-pacing interval was nearly equal to the tachycardia cycle length in panels A and B. Other abbreviations are the same as in Fig. 1.

oscillation [6]. The cycle-to-cycle length variability of non-isthmus-dependent AFL wavefronts may be greater than those of isthmus-dependent AFL [7]. In the present case, the variations in cycle length were possibly due to the atrial electrical and structural remodeling caused by long-standing AF, resulting in abnormally slow conduction and random functional block in the RA during ongoing typical AFL [10]. Unfortunately, we did not evaluate activation sequences along the tricuspid annulus with Halo catheter and could not determine whether the variation in cycle length depended on an oscillation in conductivity through the inferior vena cava (IVC)-TA isthmus, lateral RA, or transverse conductivity through the crista terminalis with conduction gap. Moreover, we did not evaluate the activation pattern in the RA during pacing from CS after restoring sinus rhythm.

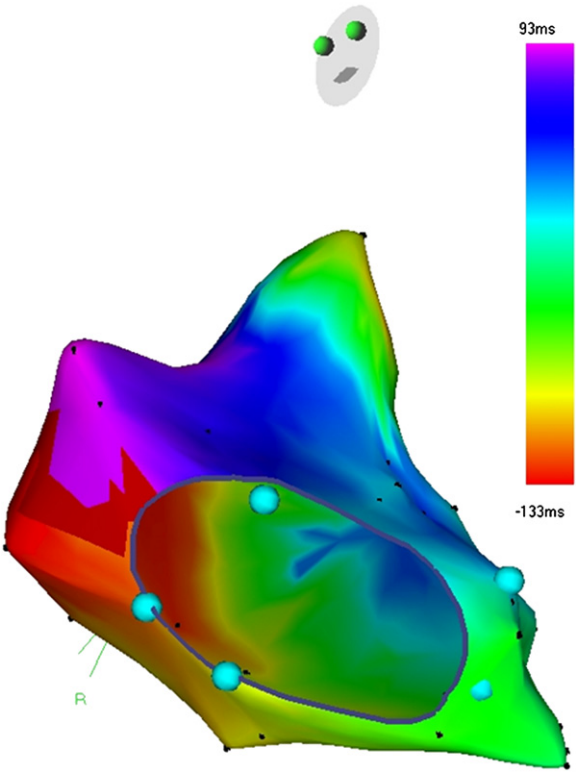

Fig. 5. Electroanatomical activation map of the RA.

Conflict of interest

The authors have no conflict of interest to disclose.

References

[1] Lin WS, Tai CT, Hsieh MH, et al. Catheter ablation of paroxysmal atrial fibrillation initiated by non-pulmonary vein ectopy. Circulation 2003;107:3176–83.
[2] Satomi K. Electrophysiological characteristics of atrial tachycardia after pulmonary vein isolation of atrial fibrillation. Circ J 2010;74:1051–8.
[3] Hsieh MH, Tai CT, Chan P, et al. Spontaneous transition from atrial fibrillation to typical atrial flutter during catheter ablation of the pulmonary vein. J Interv Card Electrophysiol 2004;10:289–91.
[4] Chugh A, Latchamsetty R, Oral H, et al. Characteristics of cavotricuspid isthmus-dependent atrial flutter after left atrial ablation of atrial fibrillation. Circulation 2006;113:609–15.
[5] Guo GB, Ellenbogen KA, Wood MA, et al. Conversion of atrial flutter by ibutilide is associated with increased atrial cycle length variability. J Am Coll Cardiol 1996;27:1083–9.
[6] Cheng J, Cabeen Jr WR, Scheinman MM. Right atrial flutter due to lower loop reentry: mechanism and anatomic substrates. Circulation 1999;99:1700–5.
[7] Narayan SM, Hassankhani A, Feld GK, et al. Separating non-isthmus from isthmus-dependent atrial flutter using wavefront variability. J Am Coll Cardiol 2005;45:1269–79.
[8] Frame LH, Simson MB. Oscillations of conduction, action potential duration, and refractoriness. A mechanism for spontaneous termination of reentrant tachycardias. Circulation 1988;78:1277–87.
[9] Frame LH, Rhee EK. Spontaneous termination of reentry after one cycle or short nonsustained runs. Role of oscillations and excess dispersion of refractoriness. Circ Res 1991;68:493–502.
[10] Nattel S, Burstein B, Dobrev D. Atrial remodeling and atrial fibrillation: mechanisms and implications. Circ Arrhythm Electrophysiol 2008;1:62–73.