Focal atrial fibrillation presenting in the origin of atrial tachycardia

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Introduction

The preferential origin of atrial tachycardia (AT) is generally associated with specific anatomical atrial structures such as crista terminalis, tricuspid annulus, mitral annulus, and pulmonary veins.[1] There are 3 major mechanisms of focal AT: enhanced automaticity, triggered activity, and microreentry. Focal AT can be eliminated via radiofrequency ablation with fairly good long-term outcomes.[2] In previous reports,[3–5] atrial fibrillation (AF) with pseudo–atrial flutter had been presented; in those cases, the organized flutter rhythms were mostly confined far from AF origins. In our cases, we successfully entrained the narrow complex tachycardia. Fractionated potentials were present in the electrogram at the earliest tachycardia activation localized to a small area (0.25 cm^2), which suggested a mechanism of focal AF. Radiofrequency ablation at the earliest activation sites with fractionated potentials terminated the focal AF and AT. The objective of this case report was to demonstrate the underlying mechanisms of AT as a manifestation of focal AF.

Case report

Case 1

The first patient was a 54-year-old man with a history of slow-fast atrioventricular nodal reentry tachycardia and AF. He underwent slow pathway ablation and pulmonary vein isolation in the past. He had recurrence of palpitation. The electrocardiogram (ECG) showed a narrow QRS complex tachycardia consistent with AT (Figure 1A). Amiodarone and diltiazem therapy failed to suppress or convert the tachycardia. He was referred for electrophysiological study and ablation. After obtaining informed consent and withholding antiarrhythmic drug treatment for 5 half-lives, electrophysiological study and ablation were performed under local anesthesia. Recording catheters were placed in the right atrium (RA), right ventricular apex, His bundle region, and coronary sinus (CS) in standard fashion. The stimulation protocol consisted of programmed stimulation at 2 basic cycle lengths with up to 2 extrastimuli and burst pacing from either the RA or the CS and the right ventricular apex. Transseptal puncture was then performed using an anatomical approach with right atrial angiography. After this, biatrial mapping was performed. Atrial voltage map and activation map were constructed with a 3-dimensional electroanatomic mapping system (EnSite Velocity System, version 4.0, St Jude Medical, St Paul, MN) and a 4-mm irrigated-tip ablation catheter (Safire BLU, St Jude Medical). Radiofrequency ablation was performed with continuous temperature control and power output. The target temperature was 50°C, and the maximum power output was 50 W. An impedance drop of 15 Ω was observed during ablation, and radiofrequency energy was delivered for 120 seconds at the successful site.

The clinical AT was induced by CS pacing. Entrainment mapping was performed from the CS ostium, distal CS, posterior wall of the left atrium (LA), and high RA during the tachycardia. Manifest entrainment was demonstrated, and the duration of postpacing interval minus tachycardia cycle length was 106, 90, 46, 52, and 36 ms at high RA, atrial septum, CS ostium, distal CS, and posterior wall of the LA, respectively (Figure 1B). The earliest activation site was in the posterior roof of the LA, which was surrounded by low-voltage areas (Figure 1E). The fractionated electrogram potential was easily identified at the earliest activation site with an area of 0.25 cm^2 (Figures 1C and 1D).

Case 2

The second patient was an 82-year-old man with a history of typical atrial flutter who had received cavotricuspid isthmus isolation 7 years ago. He felt progressively increasing frequency of palpitation in the past 1 week. ECG and 24-hour ECG monitoring showed sustained AT (Figure 2). After a failed treatment with diltiazem and bisoprolol, the patient was referred for electrophysiological study and ablation.

The electrophysiological study protocol was prescribed before. Incessant AT was present at the start of

KEYWORDS Atrial tachycardia; Atrial fibrillation; Radiofrequency ablation

ABBREVIATIONS **AF** = atrial fibrillation; **AT** = atrial tachycardia; **CS** = coronary sinus; **ECG** = electrocardiogram; **LA** = left atrium; **RA** = right atrium (Heart Rhythm Case Reports 2015;1:18–21)

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KEY TEACHING POINTS

- Focal AT could be attributed to the presence of localized AF.

electrophysiological study. Manifest entrainment was demonstrated, and the duration of postpacing interval minus tachycardia cycle length was 71 ms at the high crista terminalis (Figure 2B). The earliest activation site was localized in a small area (0.25 cm^2) in the right atrial septum (Figures 2D and 2E) with local AF and 2:1 and 3:1 local conduction. Biatrial voltage mapping also revealed low-voltage areas surrounding the earliest activation site and the cavotricuspid isthmus. Intermittent conduction to the low-voltage area from the fractionated electrogram at the right intra-atrial septum resulted in an organized AT (Figure 2C).

In both patients, electrograms were characterized by the presence of fractionated potentials in small foci. The electrical wavefront spread centrifugally from these foci, and the electrogram showed a change from irregular AF to a more organized rhythm without beat-to-beat variation (Figures 1D and 2D). Tachycardias were terminated in 5 seconds of ablation at the foci in both patients, and tachycardia was no longer inducible with postablation stimulation. At 6-month follow-up, both patients were free from recurrence.

Discussion

These 2 cases demonstrated successful catheter ablation of focal AT, with the mechanism consistent with localized AF. In these 2 cases, AT origins were localized to a small area of 0.25 cm^2. During atrial voltage mapping, we found

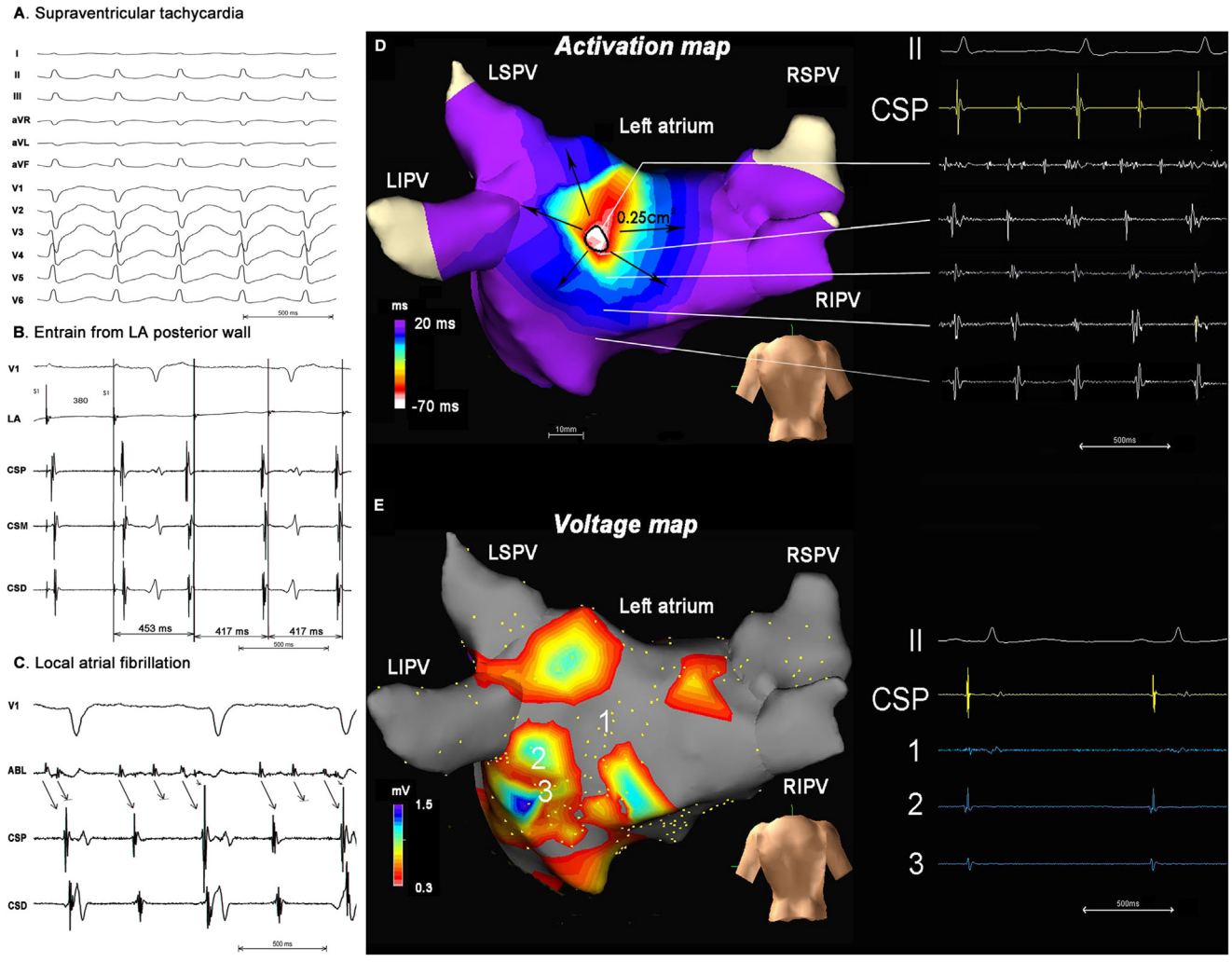

Figure 1 Electrocardiogram, intracardiac electrogram with entrainment, and 3-dimensional (3D) electroanatomic map of patient 1. **A:** The surface electrocardiogram. **B:** The electrogram of patient 1 shows atrial tachycardia. Entrainment was performed from the posterior wall of the LA with a postpacing interval of 453 ms. **C:** The electrogram recorded when the ablator tip in the atrial tachycardia origin site reveals fractionated irregular rhythm as atrial fibrillation with 2:1 and 3:1 conduction. **D:** The 3D atrial activation map reveals atrial activation starting rhythmically at a small area (0.25 cm^2) from where it spread centrifugally (black arrows). The electrogram on the right side reveals a transition from atrial fibrillation in the central area to an organized atrial tachycardia. **E:** The 3D left atrial voltage map reveals a large low-voltage zone in the posterior wall. The electrogram on the right side reveals a local signal in sinus rhythm after ablation. ABL = tip of ablator; CS = coronary sinus; CSD = distal coronary sinus; CSM = middle coronary sinus; CSP = proximal coronary sinus; LA = left atrium; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

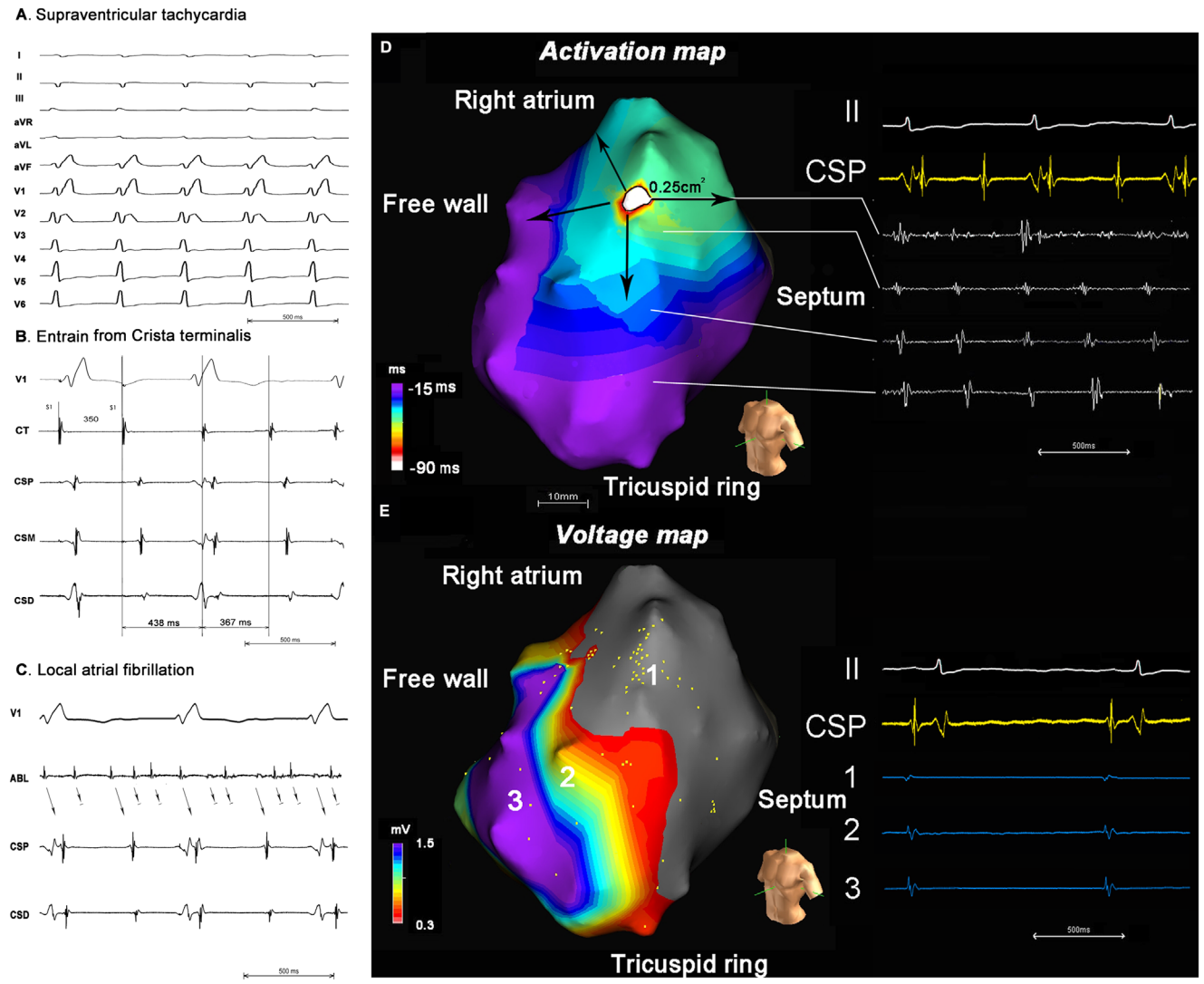

Figure 2 Electrocardiogram, intracardiac electrogram with entrainment, and 3-dimensional (3D) electroanatomic map of patient 2. **A:** The surface electrocardiogram. **B:** The electrogram of patient 2 shows atrial tachycardia. Entrainment was performed from the high crista terminalis with a postpacing interval of 438 ms. **C:** The electrogram recorded when the ablator tip in the atrial tachycardia origin site reveals fractionated irregular rhythm as atrial fibrillation with 2:1 and 3:1 conduction. **D:** The 3D atrial activation map reveals atrial activation starting rhythmically at a small area (0.25 cm^2) from where it spreads centrifugally (black arrows). The electrogram on the right side reveals a transition from atrial fibrillation in the central area to an organized atrial tachycardia. **E:** The 3D right atrial voltage map reveals a moderate low-voltage zone in the septum. The electrogram on the right side reveals a local signal in sinus rhythm after ablation. ABL = tip of ablator; CS = coronary sinus; CSD = distal coronary sinus; CSM = middle coronary sinus; CSP = proximal coronary sinus; CT = crista terminalis; RA = right atrium

low-voltage areas surrounding the earliest activation site. The reentry property was demonstrated by entrainment in both patients. The electrogram of the earliest activation site revealed fractionated potential, suggesting a mechanism of focal AF. Our cases illustrated the mechanism of focal AT as a result of localized AF within the boundary of low-voltage area. Catheter ablation is an effective strategy in the management of drug refractory AT. However, AT with a feature of focal AF has not been described previously. Our study suggests that by targeting the focal AF site, AT can be successfully ablated.

AT can be identified frequently in patients with AF after pulmonary vein isolation as a result of focal reentry through the gaps in the previous ablation lines.[6,7] In our cases, AT origins were not associated with previous ablation lines. Haissaguerre et al[8] reported a case with focal AF in the left atrial septum near the CS and recorded AT in the right atrial free wall. In contrast to their report, our cases demonstrated that AF foci were located in the posterior roof of the LA and right intra-atrial septum within the boundary of low-voltage areas. Coexistence of focal AF and AT originating from the superior vena cava has been documented in previous studies,[4,5] but apparently an irregular property of the arrhythmia was observed.

In our patients, atria were mapped and the activation maps exhibited consistently regular rhythm with minimal beat-to-beat variation in CS. The earliest activation site surrounded by low-voltage areas showed a localized AF signal,

suggesting the sink-to-source phenomenon.[9] Our cases demonstrated the distinct feature and substrate characteristic of AT relating to focal AF at the origin. However, whether the low-voltage substrate and entrainment properties in patients with AT secondary to focal AF display uniformity warrants further investigation.

Entrainment could be performed on the periphery of AT, where activation was regular. Irregular fractionated potential was found at the center of AT, where entrainment mapping is difficult. The findings of entrainment support the possible mechanism of focal and small radius reentry in AF, whereas the other mechanism of AF could not be excluded.[10] The low-voltage area adjacent to the AT origin site could contribute to discontinuity of conduction. Conduction abnormalities at these low-voltage sites might interact with one another to generate reentry circuits, which could explain the property of entrainment.

Disturbance in wavefront propagation can be due to substrate discontinuity in excitable atrial myocytes. These correspond to the surviving tissue strands connecting islands of intact myocardial tissue inside atrial scars. During substrate discontinuity, a small current source is connected to a large current load, resulting in the sink-to-source mismatch and therefore predisposition to AF.[11]

In conclusion, we presented 2 cases demonstrating successful catheter ablation of focal AT, which could be attributed to the presence of localized AF surrounded by low-voltage areas.

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