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Case Report

Right atrial tachycardia with 2:1 intra-atrial conduction

Masayoshi Kofune, MD, PhD, Ichiro Watanabe, MD, PhD*, Yasuo Okumura, MD, PhD

Division of Cardiology, Department of Medicine, Nihon University School of Medicine, 30-1 Oyaguchi-kamimachi, Itabashi-ku, Tokyo 173-8610, Japan

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ABSTRACT

In a case of atrial tachycardia (AT) originating from the inferolateral right atrium, cycle length (CL) alternans was observed. Conduction at the longer CL was to the high right atrium (HRA), His bundle electrogram region (HBE), and coronary sinus (CS). Conduction at the shorter CL was to the HRA, with that to the HBE and CS blocked.

1. Case report

A 54-year-old woman presented to our clinic with palpitation and shortness of breath. Six years previously, she underwent successful catheter ablation for focal atrial tachycardia (AT) originating from the base of the right atrial appendage and right side of the fossa ovalis. However, AT originating from the inferolateral right atrium (RA) could not be ablated. The patient later developed multiple sclerosis, and steroid therapy was started. However, the AT recurred 2 months after the initiation of steroid therapy. Catheter ablation was again performed. The RA voltage map during sinus rhythm (Fig. 1, left) revealed a low-voltage area in the inferolateral RA and scarring at the inferolateral RA adjacent to the tricuspid annulus. Focal AT (AT1; tachycardia cycle length [CL]: 442 ms, total right atrial activation time: 23 ms) at 136 beats/min that originated from the inferolateral RA adjacent and anterior to the scar (Figs. 1 and 2, right) was induced by programmed atrial pacing and successfully ablated. Programmed atrial stimulation was performed to confirm the success of the ablation, but AT was easily induced by rapid atrial pacing, and the site of the earliest site was shifted to the lateral side along the right atrial scar area. Additional ablation was performed at the earliest activation site. Therefore, ablation points formed a linear ablation line along the right atrial scar site. Finally, another focal AT (AT2) was induced. The earliest AT2 activation site (hereafter referred to as the ABL site) was located lateral to the AT1 site (Fig. 3), and the AT2 was characterized by the presence of slight CL alternans (Fig. 4). The AT2 of the longer CL (184 and 182 ms) was conducted to the high right atrium (HRA), His bundle electrogram (HBE) region, and proximal and distal coronary sinuses (CSp and CSd, respectively). However, the AT2 of the shorter CL (167 and 174 ms) was conducted to the HRA, but conduction to the HBE and CS was blocked. The reason that the AT2 conducted to the RA appendage (HRA) but showed 2:1 conduction to the HBE region and left atrium might be explained as follows: the multiple ablation points actually created the linear lesion formation from the anterolateral tricuspid annulus to the inferolateral right atrium (Fig. 3) along the scar area (Fig. 1, left); thus, the activated AT2 could not conduct to the septal RA and LA in a 1:1 fashion. The surface electrocardiogram obtained during AT2 showed fairly regular results but no obvious P wave, which is typical of atrial fibrillation. AT2 was seen during the point-by-point ablation of the re-induced AT; therefore, we did not conduct a detailed examination of the mechanism of the AT2. The total RA activation of the AT1 was 233 ms, and the tachycardia CL of the AT1 was 442 ms; therefore, we speculated that the mechanism of the AT1 was focal AT. Because we did not perform entrainment pacing, we could not demonstrate the mechanism of the AT1 as reentry or automaticity. Because the earliest activation points shifted even after AT was terminated by ablation, we speculate that the mechanism of the AT2 might be reentrant mechanism. Radiofrequency ablation at the ABL site terminated AT2. Sinus rhythm was maintained thereafter. In 1975, Wu et al. [1] reported a case of left AT with separation of the left and right atrial components of the P wave; the surface electrocardiogram resembled that of atrial flutter. Mecca et al. [2] reported a case of a patient with a right AT and atrial fibrillation

* Corresponding author. Tel.: +81 3 3972 8111; fax: +81 3 3973 1098.
E-mail address: watanabe.ichirou@nihon-u.ac.jp (I. Watanabe).

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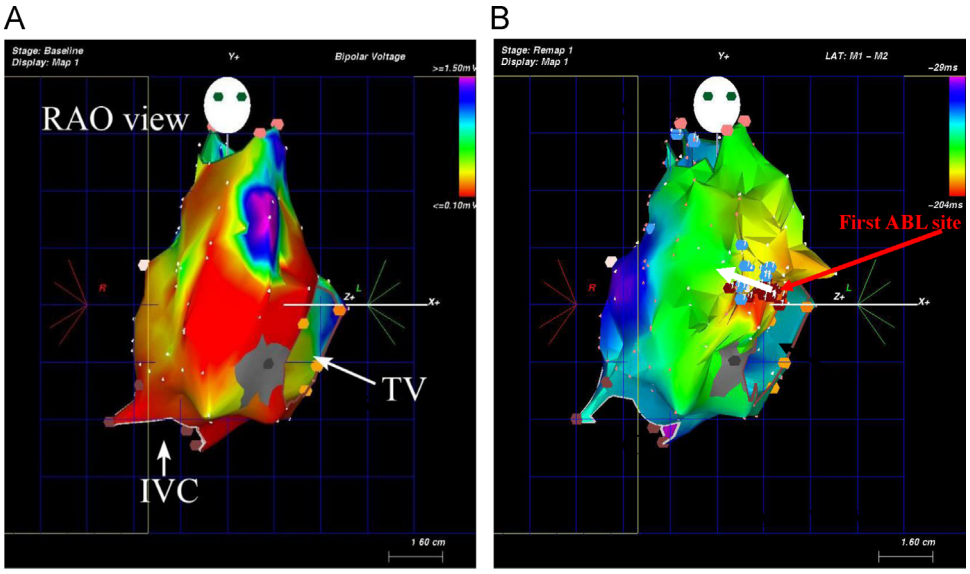

Fig. 1. Right atrial voltage map during sinus rhythm (left panel) and activation map of the AT1. The white arrow indicates the shift of the earliest activation site during the successive ablations. RAO: right anterior oblique, TV: tricuspid annulus, IVC: inferior vena cava, and ABL: ablation.

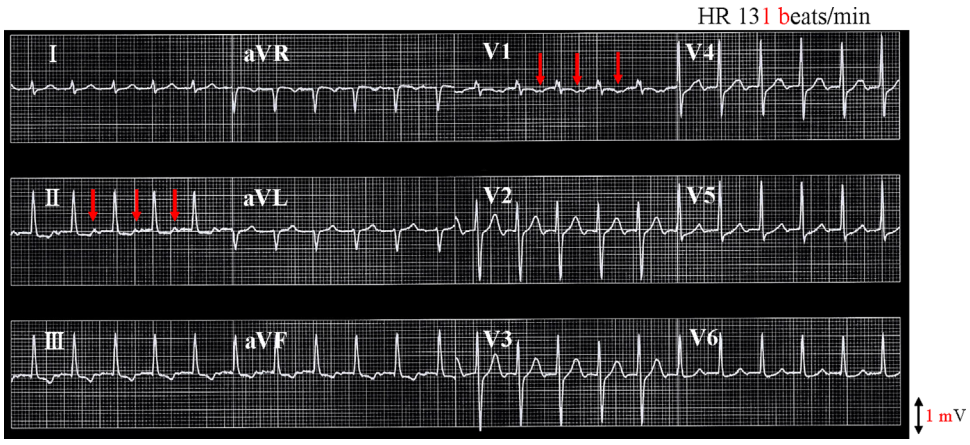

Fig. 2. A 12-lead electrocardiogram of the AT1. P waves are shown by the red arrows.

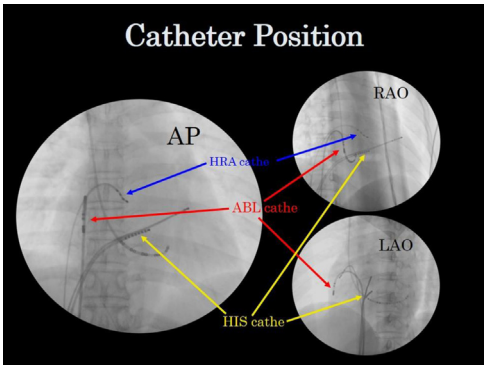

Fig. 3. Catheter position for AT2 ablation. Note that the ablation catheter was located at the mid-lateral right atrium. cathe: catheter, AP: antero-posterior, RAO: right anterior oblique, LAO: left anterior oblique HRA: high right atrium, ABL: ablation, HIS: His bundle and HRA: high right atrium (located at the right atrial appendage). ABL: ablation catheter. HIS: His bundle electrogram recording catheter.

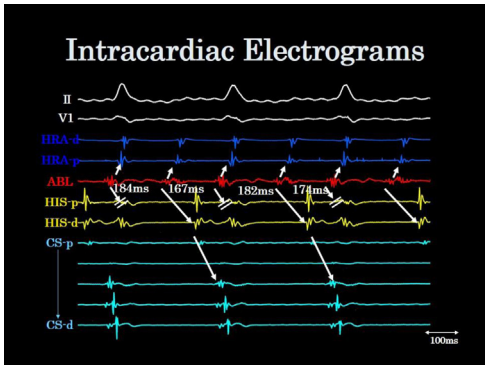

Fig. 4. Intracardiac electrograms of the AT2. HRA-d: distal pair of the electrogram from the right atrial appendage, HRA-p: proximal pair of the electrogram from the right atrial appendage, ABL: ablation catheter, HIS-p: proximal pair of the His bundle electrogram, HIS-d: distal pair of the His bundle electrogram, CS-p: proximal pair of the decapolar electrodes placed in the coronary sinus, and CP-p: proximal pair of the decapolar electrodes placed in the coronary sinus.

in whom a single site was found responsible for both conditions. In 2000, Ino et al. [3] described focal repetitive activity within the superior vena cava at a CL of 120–175 ms and 2:1 exit block to the atria masquerading as the atrial activation observed with high right atrial AT. In the present case, the P wave was not apparent during the AT2. This may be explained by the rapid focal activity, 2:1 intra-atrial conduction block, and/or the low-voltage area and scarring in the RA.

Conflict of interest

There are no competing financial interests for this manuscript.

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