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Short Report

A case of typical atrial flutter causing unexpected advanced atrioventricular block despite lateral cavotricuspid isthmus ablation

Hiroaki Mano, MD, Yasuo Okumura, MD*, Ichiro Watanabe, MD, Koichi Nagashima, MD, Toshiko Nakai, MD, Kimie Ohkubo, MD, Tatsuya Kofune, MD, Masayoshi Kofune, MD, Kazumasa Sonoda, MD, Hironori Haruta, MD, Atsushi Hirayama, MD

Division of Cardiology, Department of Medicine, Nihon University School of Medicine, 30-1 Ohyaguchi kami-cho, Itabashi-ku, Tokyo 173 8610, Japan

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ABSTRACT

Here, we report a case of a 69-year-old patient with paroxysmal atrial fibrillation and inducible typical atrial flutter who required catheter ablation. After pulmonary vein isolation, cavotricuspid isthmus ablation was performed. During ablation at a lateral site of the cavotricuspid isthmus, a spiky potential appeared at the distal electrode of the ablation catheter, and subsequently, a 2:1 atrioventricular (AV) block occurred. Radiofrequency (RF) delivery at the same site caused a similar phenomenon, implying that the spiky potential may reflect a slow pathway potential as an anatomical variant of the rightward extension of the AV node.

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1. Introduction

Advanced atrioventricular (AV) block has been reported to occur, albeit rarely, during cavotricuspid isthmus (CTI) ablation of atrial flutter [1,2]. The main mechanism contributing to AV block is direct injury of the AV node via the delivery of radiofrequency (RF) energy, during which the ablation catheter is usually located at a septal site of the CTI near the AV node rather than at the lateral CTI. Here, we describe a unique case, in which a spiky potential appeared at the distal electrode of the ablation catheter immediately after initiating RF delivery at the lateral CTI, and unexpected AV block eventually occurred.

2. Case report

A 69-year-old man with symptomatic paroxysmal atrial fibrillation for 1 year was referred to Nihon University Itabashi Hospital for ablation of AF. He had hypertension, and his baseline electrocardiogram (ECG) revealed left ventricular hypertrophic changes without any conduction block. Coronary sinus (CS) angiography revealed no anatomical abnormalities in the CS. In baseline electrophysiological studies, the Wenckebach AV block cycle length was 350 ms, and there was no ventriculoatrial conduction during right

ventricular pacing. CARTO-guided extended ipsilateral pulmonary vein isolation was performed, and thereafter, typical atrial flutter was induced by rapid atrial pacing from the CS ostium. CTI ablation was performed during CS pacing at a cycle length of 600 ms with a deflectable 8-mm tip EPT catheter (EP Technologies Inc., San Jose, CA, USA); each RF application was performed at 60 °C and 50 W, for 60 s. The ablation catheter was positioned at 7:00 on the tricuspid annulus in the left anterior oblique view (Fig. 1A). One second after the first RF application, a spiky potential was noted at the distal electrode 1–2 of the ablation catheter and the Halo catheter, with a time interval of 80–90 ms from the local atrial potential. During the ablation, the spiky potential was reproducibly observed, consistently occurring 80–90 ms after the local atrial potential. The AH interval was gradually prolonged, and a 2:1 AV block occurred 34 s after the ablation (Fig. 1B). Although 1:1 AV conduction was resumed 3 s after the termination of RF delivery, it took 30 s for the AH interval to recover to baseline. The Wenckebach AV block cycle length was the same as the baseline value. No further RF application was delivered because an additional RF delivery at the same site caused a similar phenomenon.

3. Discussion

In previous studies, advanced AV block during RF application for type 1 atrial flutter raised immediate concerns that the catheter may have been positioned at the septum near the AV node, rather than in the CTI [1,2]. In our case, since the ablation catheter was positioned at

* Corresponding author. Tel.: +81 3 3972 8111; fax: +81 3 3972 1098.
E-mail address: yasuwo128@yahoo.co.jp (Y. Okumura).

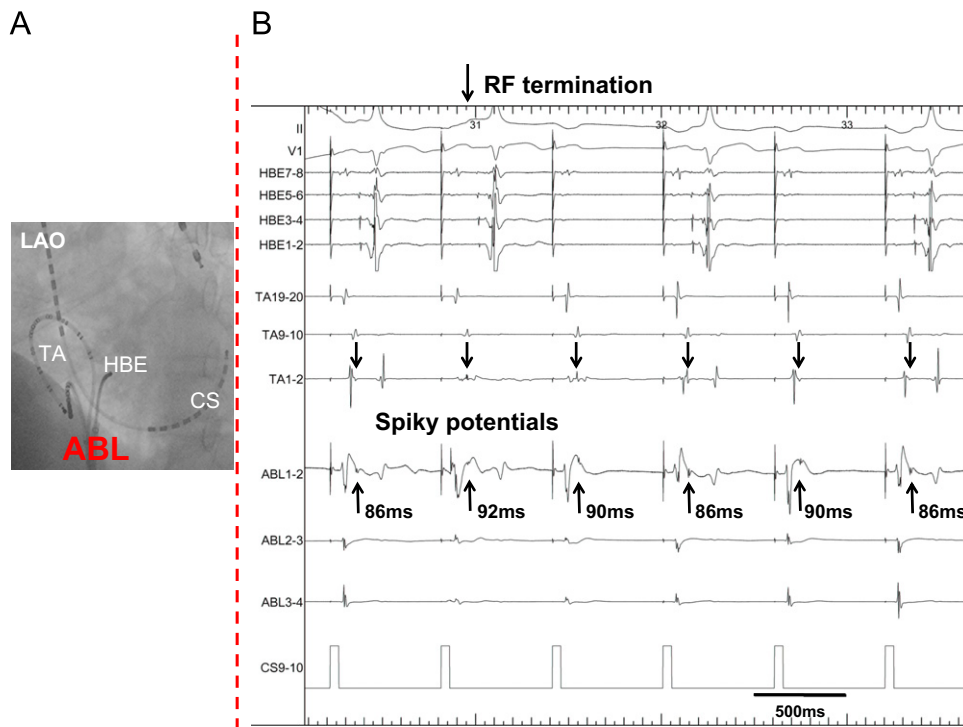


Fig. 1. Catheter position during cavotricuspid isthmus ablation (A) and the intracardiac electrograms of 2:1 atrioventricular (AV) block during ablation (B).

1. The ablation catheter (ABL) was positioned at 7:00 on the tricuspid annulus (TA) in the left anterior oblique (LAO) view at 45°, which corresponded to the lateral isthmus. The distal electrodes of the ablation catheter were also located at a site adjacent to the distal electrodes of the Halo catheter.
2. A 2:1 AV block occurred during ablation and was sustained after termination of RF delivery. Note that spiky potentials were reproducibly observed (see arrows) with an 80–90 ms delay from the local atrial electrogram wave from the distal electrode pair of the ablation and Halo catheters.

HBE: His bundle electrograms, CS: coronary sinus.

a lateral isthmus site according to fluoroscopy, the potential risk of AV node injury by RF delivery at that site was expected to be low. Besides catheter placement, there are 3 other potential mechanisms for AV block during CTI ablation. The first potential mechanism is coronary ischemia. RF delivery at the CTI has been reported to increase the risk of coronary artery damage because the right coronary artery is located in the AV groove just below the CTI [3,4]. However, the possibility of coronary ischemia may be low because neither chest pain nor ST-T changes on the surface ECG were observed during ablation, and there was no evidence of stenoses on coronary angiography. The second potential mechanism is through vagal tone. Sinus bradycardia or AV node block have been reported to occur with excessive vagal tone due to pain during ablation [5]. Recently, a study has also demonstrated that catheter ablation of the left atrial ganglionated plexi can mediate AV node block or sinus bradycardia [5]. In fact, ganglia have been shown to be located in the inferior vena cava-left atrial fat pad, which is not far from the CTI. Therefore, CTI ablation may potentially affect these ganglia, leading to AV block. The absence of AV block during ablation after intravenous atropine supports the mechanism of vagal tone; however, atropine was not administered in the present case. The final potential mechanism involves an anatomical variant of the slow pathway that would place these structures more inferolaterally than normally expected. Transient AH interval prolongation despite RF termination suggested direct thermal injury to the AV node. Even if such an anatomic variant existed, our patient should not have experienced advanced AV block because a shorter AH interval during CS ostial pacing implied that the AV conduction was dependent on the fast pathway. Therefore, RF energy delivered by the slow pathway may have affected the AV node via a specific pathway bridging the slow pathway to the fast pathway and His bundle region, i.e., a lower common pathway. Interestingly, in this case, slow

pathway-like spiky potentials were reproducibly observed during ablation from the distal electrodes of the ablation catheter that was placed at the lateral isthmus. Therefore, these spiky potentials may reflect slow pathway potentials as anatomical variants of the rightward extension of the AV node. However, it is also possible that these spiky potentials simply reflect the local atrial electrograms with an intra-atrial conduction delay because the spiky potentials were recorded from the distal electrodes of both the Halo and ablation catheters, and the direction of activation of the spiky potentials was counterclockwise around the tricuspid annulus.

Conflict of interest

None.

References

- [1] Belhassen B, Glick A, Rosso R, Michowitz Y, Viskin S. Atrioventricular block during radiofrequency catheter ablation of atrial flutter: incidence, mechanism, and clinical implications. *Eurpace* 2011;13:1009–14.
- [2] Passman RS, Kadish AH, Dibs SR, Engelstein ED, Goldberger JJ. Radiofrequency ablation of atrial flutter: a randomized controlled study of two anatomic approaches. *Pacing Clin Electrophysiol* 2004;27:83–8.
- [3] Weiss C, Becker J, Hoffmann M, Willems S. Can radiofrequency current isthmus ablation damage the right coronary artery? Histopathological findings following the use of a long (8 mm) tip electrode. *Pacing Clin Electrophysiol* 2002;25:860–2.
- [4] Brembilla-Perrot B, Filali ML, Beurrier D, et al. Complete atrioventricular block during ablation of atrial flutter. *Pacing Clin Electrophysiol* 2010;33:516–9.
- [5] Verma A, Saliba WI, Lakkireddy D, et al. Vagal responses induced by endocardial left atrial autonomic ganglion stimulation before and after pulmonary vein antrum isolation for atrial fibrillation. *Heart Rhythm* 2007;4:1177–82.