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CASE REPORT

Termination of Recurrent Atrial Fibrillation by Superior Vena Cava Isolation: A Case Report

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Abstract

Background: Paroxysmal atrial fibrillation can be triggered by non-pulmonary vein foci, such as the superior vena cava. Here, we report the case of a patient with a 6-year history of paroxysmal atrial fibrillation who received cryoballoon ablation in 2012 but relapsed in 2014. He then received cardiac radiofrequency ablation, which successfully isolated the left pulmonary vein and superior vena cava, but the arrhythmia recently relapsed again. The tachycardia was finally successfully terminated by ablation on the free wall without recurrence during a 2-year following up.
Conclusion: Superior vena cava isolation may not require ablation isolation with a full circle way and can be accomplished by ablating several connection points between the superior vena cava and the right atrium.

Keywords: Recurrent atrial fibrillation; superior vena cava; arrhythmia; tachycardia; electrocardiogram

A 63-year-old man presented to our hospital with aggravation of paroxysmal palpitations and shortness of breath. He had a history of diabetes for 5 years and regular hypoglycemic therapy. He denied a history of coronary heart disease or hypertension, and had no prior history of smoking and drinking. He had a 6-year history of paroxysmal atrial fibrillation, and he had received cryoballoon ablation for atrial fibrillation in 2012 but relapsed in 2014. The patient was admitted to the hospital for electrophysiologic study under local infiltration anesthesia, which confirmed atrial fibrillation and atrial tachycardia (originating from the superior vena cava). Hence, he received cardiac radiofrequency ablation, and the left pulmonary vein and superior vena cava were successfully isolated. Recently, the patient presented for further treatment at our hospital because the arrhythmia had relapsed again in the absence of antiarrhythmic drugs.

At the time of presentation to our hospital, the patient's blood test results showed a TnI level of 0.86 pg/mL, CK-MB level of 0.45 ng/mL, and brain natriuretic peptide (BNP) level of 10.3 pg/mL, all of which were within normal ranges. No other hematological test findings were abnormal. An echocardiogram indicated that the left atrium had an anteroposterior diameter of 36 mm, and the left ventricular ejection fraction was 70%. Transesophageal echocardiography indicated no clear mass-like thrombi in the left atrium and left auricle. No abnormalities were observed on chest X-ray. The ECG obtained at initial presentation showed a sinus rhythm (Figure 1A). On the 2nd day after admission, the patient developed palpitations and shortness of breath. Urgent ECGs were performed

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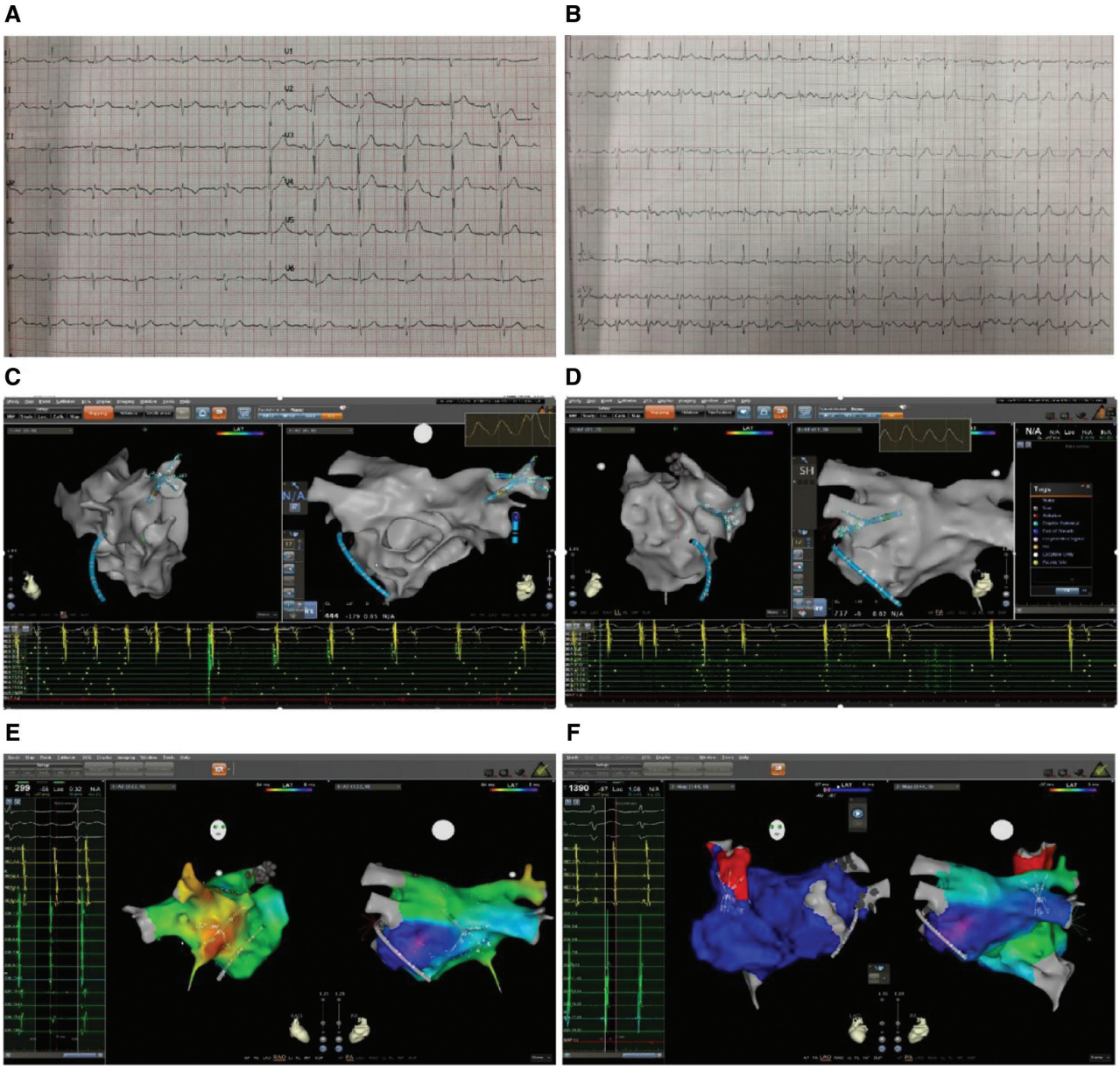

Figure 1 Target Mapping.
(A) ECG obtained at initial presentation. (B) ECG obtained on the second day after admission. (C and D) Confirmation of complete pulmonary vein isolation by pulmonary vein mapping. (E) The atrial septum is activated earlier than other areas of the left atrium. It means that excitation waves are from the right atrium. (F) Combined activation mapping, indicating that the superior vena cava activated first and was followed by the right atrium, and the left atrium was activated last.

and indicated atrial tachycardia with P′ waves in the inferior wall leads (II, III, and aVF) (Figure 1B). On the basis of these findings, the patient was diagnosed with paroxysmal atrial fibrillation, short bursts of atrial tachycardia, atrial premature contraction, and noninsulin-dependent diabetes mellitus. Because of the patient's strong desire to terminate the arrhythmia, we decided to perform a catheter ablation.

An ECG mapped by a coronary sinus electrode under electrophysiological examination is shown in Figure 2C. Mapping at the pulmonary vein confirmed complete pulmonary vein isolation (Figure 1C and D). Mapping at the left atrium indicated that the atrium was activated earlier in the atrial septum (Figure 1E). Mapping of the right atrium indicated that the superior vena cava is activated earlier than the right atrium. It means that excitation waves are from the superior vena cava. Combined activation mapping (Figure 1F) revealed that the superior vena cava was activated first and was followed by the right atrium, and the left atrium was activated last. Mapping at the superior vena cava revealed that the

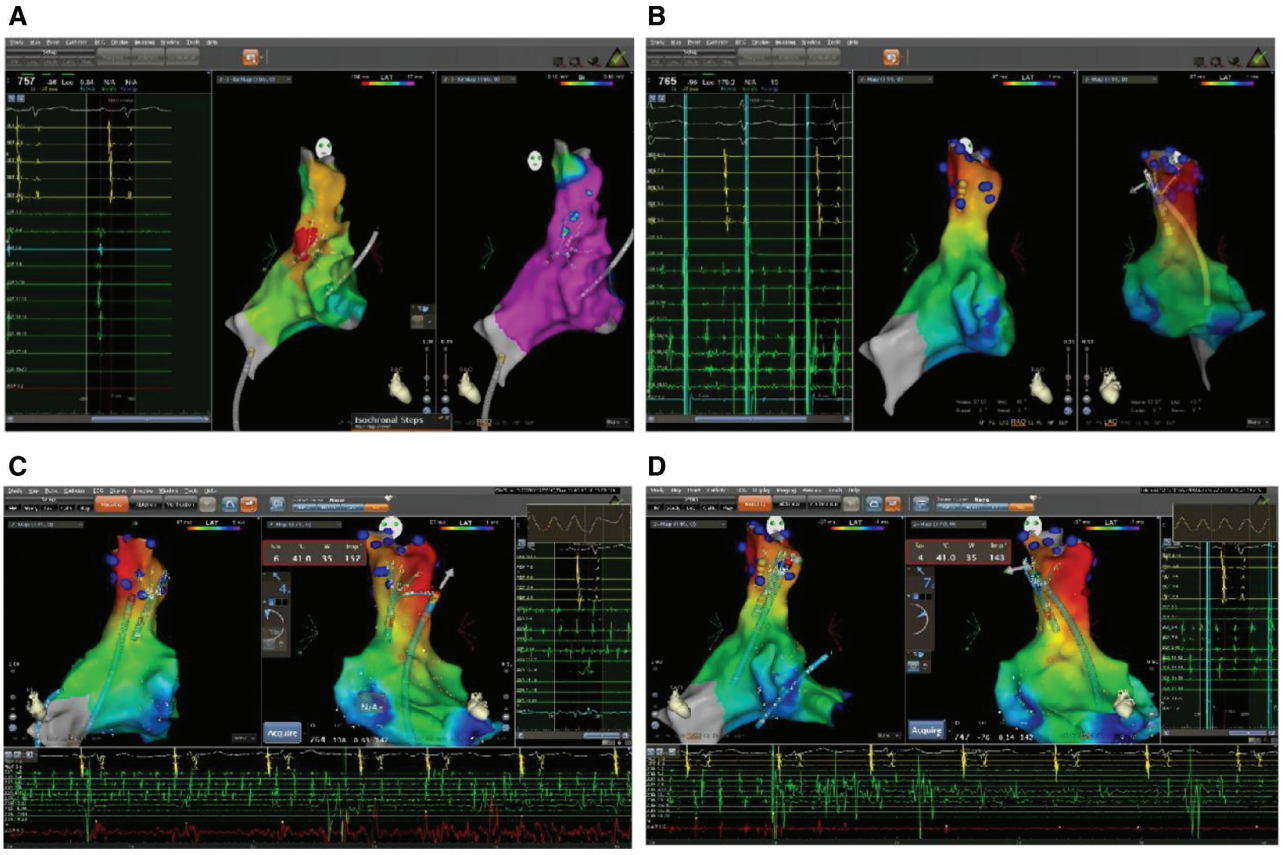

Figure 2 Target Ablation.
(A) Mapping at the superior vena cava showed that the arrhythmia was atrial fibrillation. The ablation catheter marked the phrenic nerve by pacing in the superior vena cava. (B) Sinoatrial node mapping was performed to avoid damaging the sinoatrial node. (C) Superior vena cava isolation was performed but did not terminate the tachycardia on the septal side. (D) The tachycardia was finally successfully terminated by ablation on the free wall.

arrhythmia was atrial fibrillation (Figure 2A). Next, sinoatrial node mapping was performed to avoid damaging the sinoatrial node (Figure 2A). The ablation catheter marked the course of the phrenic nerve by pacing in the superior vena cava (Figure 2B). Superior vena cava isolation was performed but did not terminate the tachycardia on the septal side (Figure 2C). The tachycardia was finally successfully terminated by ablation on the free wall (Figure 2D). Routine treatment was administered after operation. During a 2-year follow-up, sinus rhythm was maintained without tachycardia.

Discussion

In our case, the patient underwent two ablations for atrial fibrillation: cryoballoon and cardiac radiofrequency ablation. The electrocardiogram at the time of the attack indicated atrial tachycardia, which was characterized by up-right, high atrial P′ waves in leads II, III, and aVF. We considered that the atrial tachycardia originated from a position closer to the roof of the atrium, according to the 12-lead ECG. Catheter mapping also indicated that this excitation occurred first in the superior vena cava, second in the right atrium, and finally in the left atrium; therefore, 12-lead ECG is highly valuable in assessing the general orientation of atrial tachycardia. However, careful interpretation of the readout is required before ablation for atrial tachycardia, particularly when the circumference is unfixed. Because of possible difficulties in excitation mapping, greater attention should be paid to the interpretation of the ECG before ablation.

Non-pulmonary vein triggers play a role in the initiation of atrial fibrillation [1, 2]. In our case, when the Pentaray electrode was sent to the superior vena cava, the arrhythmia should have been atrial fibrillation for the completely unequal AA interval, and the various potential amplitudes and the potential period

should have been less than 250 ms. Therefore, assessment of atrial tachycardia on the basis of atrial waves recorded by a 12-lead electrocardiogram or coronary sinus electrodes is inaccurate, because of the accuracy of mapping. Atrial fibrillation originating from the superior vena cava often presents as atrial tachycardia in terms of atrial potential determined in both the right atrium and the coronary sinus, because of the rectification by the myocardium at the junction of the superior vena cava and the right atrium. Therefore, the existing definitions of atrial fibrillation and atrial tachycardia must be further improved to clarify the mapping electrodes, the mapping method, and the mapping location, which are the basis for diagnosis. With advances in density mapping and other mapping methods, subtle reentrant mechanisms of atrial tachycardia will increasingly be revealed. Comparing with high density mapping, current mapping does not reflect complete information of arrhythmia mechanisms. Shah et al. [3] have reported that approximately 24% of atrial fibrillation are of non-pulmonary vein origin. The sites of non-pulmonary vein origin include the Marshall ligament, left posterior wall of the atrium, superior vena cava, boundary ridge, oval fossa, coronary sinus, and right atrium. For this type of atrial fibrillation, pure pulmonary vein electrical isolation is not effective; however, superior vena cava electrical isolation and focal ablation are effective, thus suggesting the necessity of detailed electrophysiological examination during the ablation process. To develop better mapping methods and techniques, attention should also be paid to mapping of the foci of origin of atrial fibrillation outside the pulmonary veins. In our case, if the atrial fibrillation lesions had been accurately located during the first ablation for atrial fibrillation, the patient might not have needed to undergo the subsequent two ablations. New mapping methods and mapping techniques for identifying the foci of origin of atrial fibrillation outside the pulmonary veins must be developed. In addition, in a study by Kholova et al. [4], 19 of 25 hearts had a myocardial sleeve extending from the right atrium to the superior vena cava, and in most cases, the superior vena cava and the right atrium myocardium were not connected. Xia Yunlong et al. [5], in a study of 16 superior vena cava muscle sleeves, have found that most of the connections between the superior vena cava and the right atrium were single or double fasciculate.

In our case of superior vena cava isolation, only two points on the septal and free wall side were ablated to achieve isolation of the superior vena cava. Simultaneously, the atrial tachycardia of this activation sequence disappeared completely, thus suggesting that superior vena cava isolation may not require ablation isolation with a full circle way of the superior vena cava; instead, the superior vena cava can be successfully isolated by ablating only several connection points between the superior vena cava and the right atrium. In the future study, it is necessary to determine which types of atrial fibrillation require superior vena cava ablation isolation in the first treatment, and whether the superior vena cava ablation isolation be performed in persistent atrial fibrillation treatment strategies.

Conflict of Interests

The authors declare that they have no conflict of interest.

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