Clinical Case Report

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Complicated atrial tachycardia due to atrial fibrillation originating from the superior vena cava

A case report

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Abstract

Introduction: The superior vena cava (SVC) can act as an origin of atrial fibrillation (AF). The complex structure and special conductive properties of the SVC can result in complicated atrial tachycardia (AT), atrial flutter, or AF.

Symptoms and clinical findings: We report a case of the clinical observation of various kinds of AT and AF in 1 patient. Electrophysiological (EP) studies confirmed the muscle sleeve in the SVC to be the primary trigger and the only site or origin of tachycardia in this patient. Furthermore, we describe the mechanism of AT observed in clinic, which was misdiagnosed as multiorigin AF.

Intervention and Outcomes: Circumferential pulmonary vein isolation and circumferential SVC isolation resulted in termination of tachycardia.

Conclusion: We present an atypical case of AF originating from the SVC with unusual intra-atrial conduction characteristics and arrhythmogenic pulmonary veins. Stepwise EP studies were conducive to clarify the mechanism of this rare AT.

Abbreviations: AF = atrial fibrillation, AT = atrial tachycardia, CL = circle length, CPVI = circumferential pulmonary vein isolation, CS = coronary sinus, EP = electrophysiological, LA = left atrium, PV = pulmonary vein, RA = right atrium, SVC = superior vena cava, VA = venoarterial.

Keywords: atrial fibrillation, atrial tachycardia, catheter ablation, superior vena cava

1. Introduction

Atrial fibrillation (AF) is a commonly observed arrhythmia in the clinic, which often causes clinical complications such as stroke and heart failure, corresponding to a high rate of morbidity and mortality. Studies have shown that most AF originates from the pulmonary vein (PV) and that circumferential pulmonary vein isolation (CPVI) could cure approximately 70% of cases.[1] In addition, there are numerous studies suggesting that other special anatomical structures, such as the superior vena cava (SVC), Marshall ligament, crista terminalis, coronary sinus (CS), and the left and right atrial appendage, could also be sites of origin of AF.[2,3] We report a case of AF originating from the SVC, which demonstrated various atrial arrhythmias and was misdiagnosed as multiorigin AF.

2. Case presentation

A 53-year-old male patient with a 3-year history of tachycardia was admitted to our center for an electrophysiological (EP) study. Standard informed consent was given before the EP study. Standard CS leads (St. Jude Medical, St. Paul, Minnesota) were placed as shown in Figure 1A; the baseline intracardial electrocardiogram showed a normal sinus rhythm during the programmed stimulation. However, after intravenous administration of isoproterenol, narrow QRS wave tachycardia was induced by programmed stimulation (S1S2 400/210 ms on the CS 9,10 channel). The circle length (CL) of the initiated tachycardia was 262 ms, with an ambiguous atrial activation sequence; the A wave was earliest on CS 9,10, which was slightly earlier than on CS 1,2, and the A wave was latest on CS 5,6. This implied a focal atrial tachycardia (AT) originating from the roof of the left atrium (LA) (Fig. 1B). AT lasted for approximately 2 minutes before spontaneously changing into another kind of AT with a different CL (165 ms) and activation sequence; the A wave conducted from CS 9,10 to CS 1,2, implying that it originated from the right atrium (RA) (Fig. 1C). Finally, AF was induced (Fig. 1D). This led to the following questions: What were the potential mechanisms of tachycardia in this patient? Does this imply that ATs originating from different areas combined to induce AF?

Editor: Jacek Bil.

All authors listed above materially participated in the research and article preparation, W.H. and W.L. contributed the conception of the work and drafting the article. All authors were the main operators of electrophysiological study, and contributed the revising of the article.

The authors have no conflicts of interest to disclose.

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Medicine (2017) 96:25(e7192)

Received: 21 March 2017 / Received in final form: 19 May 2017 / Accepted: 24 May 2017

http://dx.doi.org/10.1097/MD.0000000000007192

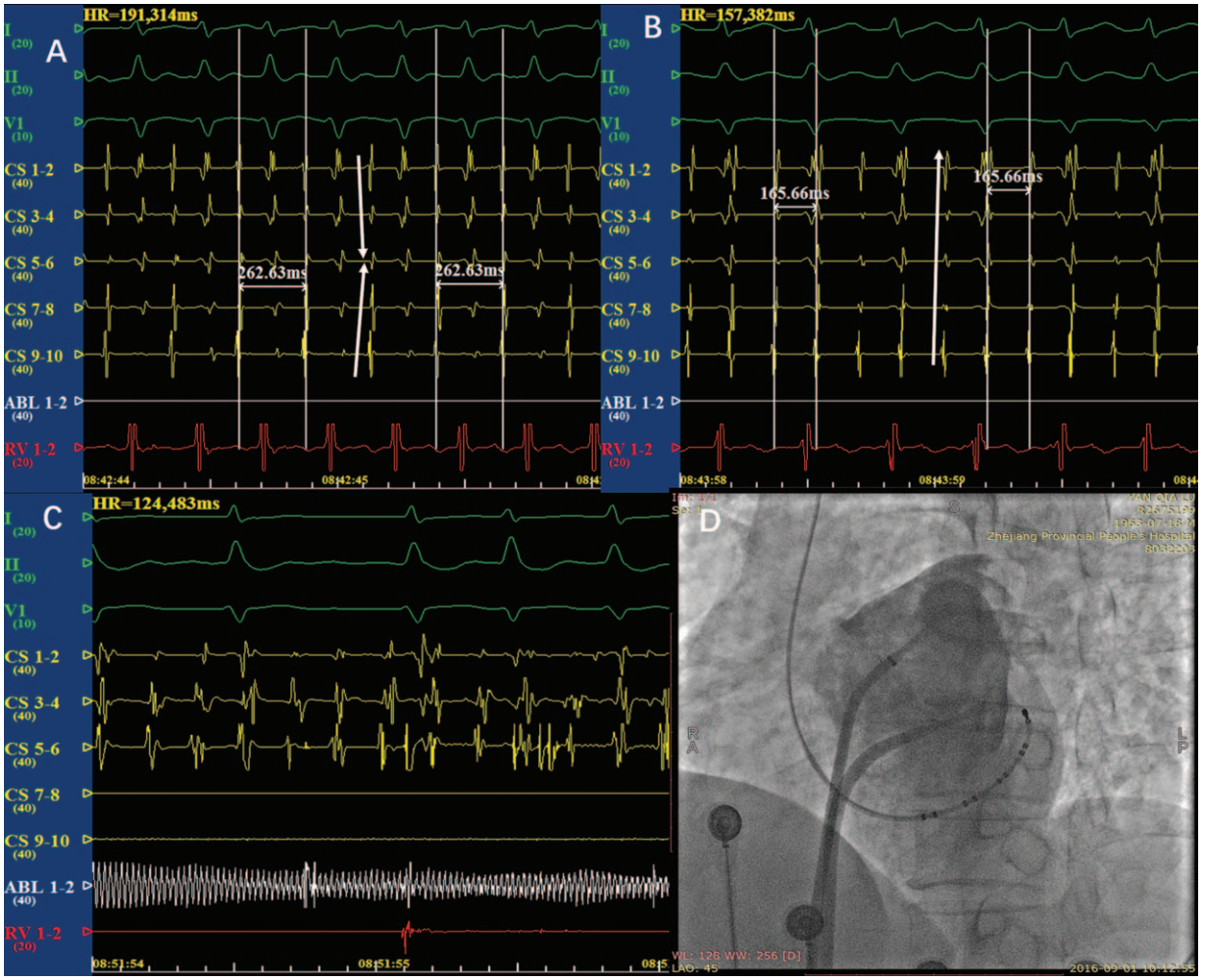

Figure 1. (A) Atrial tachyarrhythmia (AT) was induced by programmed stimulation and isoproterenol. The circle length (CL) was 262 ms. The white arrow represents the activation sequence on the coronary sinus (CS) lead; the A wave was earliest on CS 9,10, which was slightly earlier than that on CS 1,2, and the A wave on CS 5,6 was the latest. (B) The type AT changed. The CL was 165 ms. The white arrow represents the activation sequence of the A wave from CS 9,10 to CS 1,2. (C) AF was induced by the second AT. (D) The CS lead position in LAO 45 view.

Firstly, a standard CPVI was performed with 3D mapping using the Ensite NavX System (St. Jude Medical; Fig. 2A). During CPVI, we observed a spontaneous change from an AT with a CL of 303 ms and right to left activation sequence (Fig. 2B) to an AT with a CL of 278 ms and CS 9,10/1,2 to CS 5,6 activation sequence (Fig. 2C). In addition, the A wave on CS 1,2 was earlier than on CS 9,10. Finally, the latter AT was stabilized after CPVI.

Activation sequence 3D mapping was performed in the LA. The earliest A wave was located in the roof of the right LA, which was 61 ms earlier than the A wave on CS 9,10 (Fig. 2D). However, repeated ablation around the target was invalid (CL and activation sequence were unchanged); therefore, we suspected that the AT originated in the RA.

When we placed the ablation catheter into the SVC, chaotic fibrillation was recorded (Fig. 3A). In contrast, a LASSO lead (St. Jude Medical) at a slightly lower position (SVC-RA junction) recorded regular activation 91 ms earlier than the CS 9,10 (Fig. 3B and C). When the ablation lead was placed at the SVC-RA junction, atrial activation was in the distal to proximal direction (Fig. 3D). This indicated that activation of the whole atrium originated in the SVC, meaning that AF originating in the SVC was filtered into AT due to weak conduction and blocking of the myocardial sleeve. AT was then terminated when the SVC isolation was completed (Fig. 3E). 3D mapping showed that the ablation lesions at the SVC-RA joint were adjacent to the earliest point of activation in the LA roof (Fig. 3F).

3. Discussion

The incidence of SVC AF is 5% in patients with AF, as reported in studies by Miyazaki et al.[4,5] The mechanism of AF originating from the SVC is similar to typical PV-related AF, which is known as "muscle sleeve AF." In a report by Desimone et al,[6] cardiomyocyte elongation in the PV was observed in 99% of patients with PV-related AF; in contrast, cardiomyocyte elongation was found in 78% of patients with SVC AF. The EP heterogeneity of the sleeve cells can be arrhythmogenic, resulting in different types of atrial arrhythmia.[7,8]

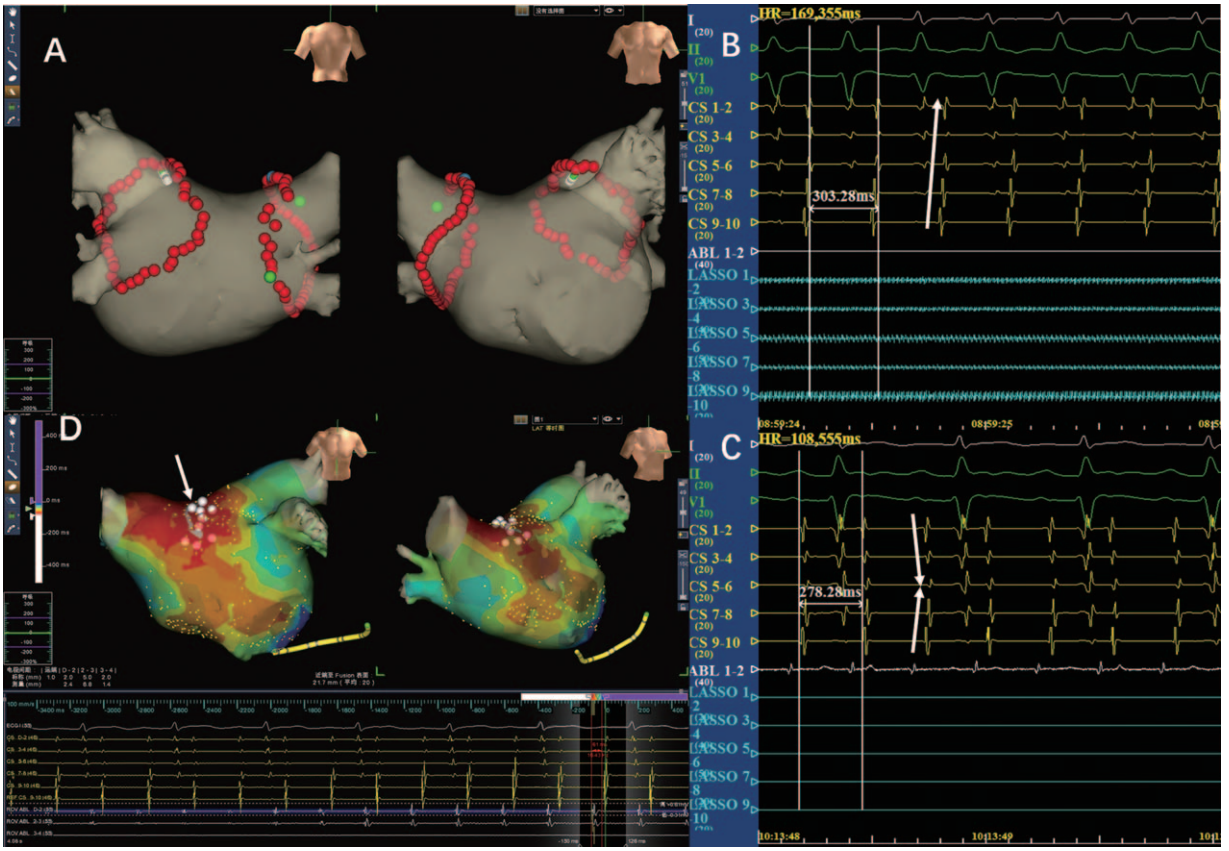

Figure 2. (A) Circumferential pulmonary vein isolation (CPVI); red dots represent the isolation line and green dots represent the key point of pulmonary vein (PV) isolation. (B) Atrial tachyarrhythmia (AT) occurred during CPVI. The circle length (CL) was 303 ms and the activation sequence was from coronary sinus (CS) 9,10 to CS 1,2, as showed by the white arrow. (C) Another AT was evoked during CPVI; the CL was 278 ms and the activation was conducted from CS 9,10/1,2 to CS 5,6. This AT was stabilized after completion of CPVI. (D) 3D activation sequence mapping of AT in the left atrium (LA). White dots on the roof of the LA indicate the earliest activation area (61 ms earlier than the A wave on CS 9,10, as indicated by white arrow).

In this case, AF in the SVC was considered to be the primary cause of arrhythmias; however, it is interesting that several types of AT or AF were observed in this patient. This could be explained by the following points. First, an intracaval conduction delay and decremental conduction properties of the venoarterial (VA) junction is common, as reported by Fukumoto et al.[8] We do not have direct evidence to prove that a relationship exists between AF in the SVC and AT in the atrium; however, the activation sequence shown by the ablation catheter demonstrated that the direction of atrial activation was from the SVC to the atrium. We considered that the conduction delay in the muscle sleeve at the VA junction resulted in filtering of the fibrillation from the SVC to induce AT. Second, the ATs had 2 different conduction directions; one conducted from the RA to the LA through the CS (typical RA originating AT), and the other penetrated the superior part of the atrial septum to activate the roof of the LA first. Third, there was a pathogenetic basis for AF in the LA.

The atrial activation sequence of the first AT after programmed stimulation was a fusion of activations from 2 different conduction directions, as described above. We inferred that conduction via the CS may predominate because the A wave on CS 9,10 was slightly earlier than on CS 1,2. This AT could change to a typical RA originating AT, providing circumstantial evidence to prove our hypothesis. After CPVI, the tachycardia changed; AT with a CL of 278 ms and CS 9,10/1,2 to CS 5,6 activation sequence stabilized. This implied that CS conduction was no longer predominant; from this, we inferred that conduction via the CS may have been weakened by CPVI.

We finally diagnosed the primary lesion as being AF in the SVC in this patient; however, 1 last question arose. In theory, isolating the SVC could terminate tachycardia in this patient; therefore, was CPVI necessary in this patient? As the EP result showed, AF in the SVC was filtered by the muscle sleeve, turning it into AT; clinical AF was induced by the AT, and was terminated by CPVI. This provided evidence that the LA was electrophysiologically unstable in this patient, which was the basis of AF. Furthermore, it implied that there may be one or more arrhythmogenic PVs. Data from Miyazaki et al[5] demonstrated that half of patients with an arrhythmogenic SVC also had an arrhythmogenic PV. Thus, CPVI was necessary to prevent future episodes of AF.

4. Conclusion

This case report describes an example of AF in the SVC being the primary loci of tachycardia. Because of different conduction directions and electrophysiological vulnerability of the LA, different types of AT were observed in this patient.

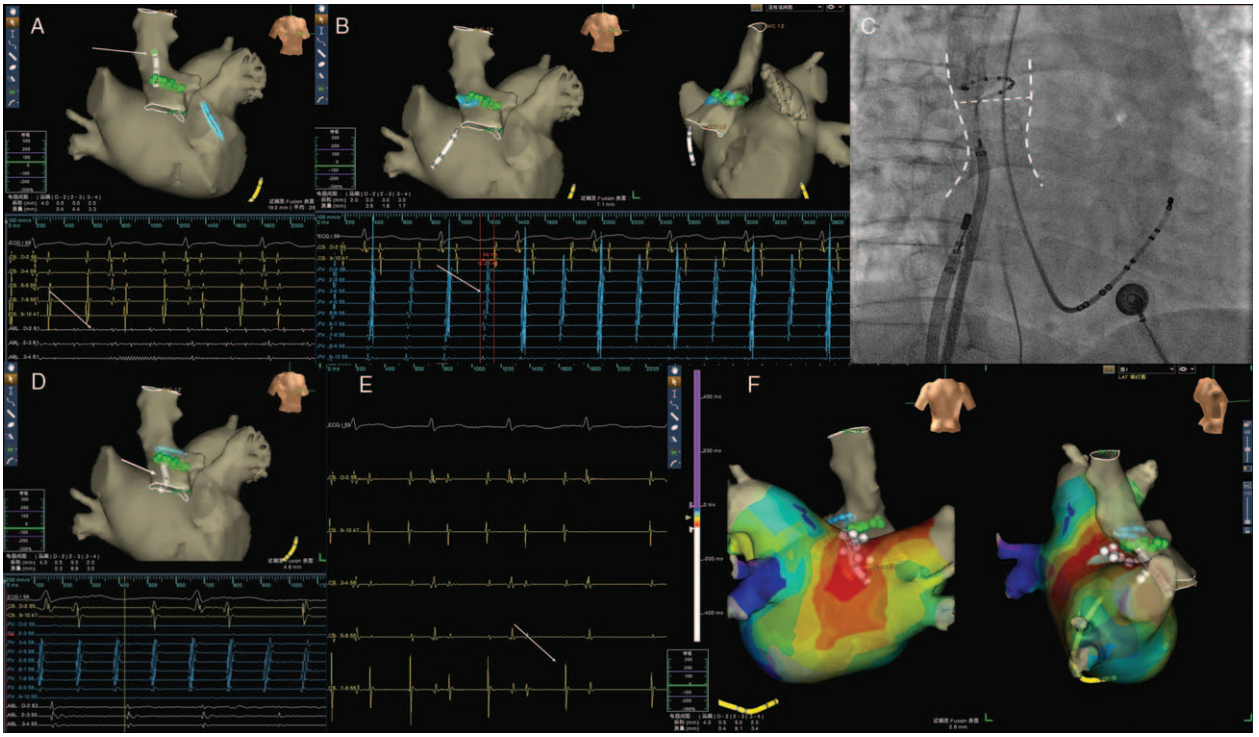

Figure 3. (A) The ablation catheter in the superior vena cava (SVC) is highlighted by the white arrow. The intracardiac electrocardiogram showed a fibrillation wave on the ablation catheter; at the same time, the coronary sinus (CS) lead recorded regular AT in atrium. (B) The LASSO lead (blue) at SVC-right atrial (RA) junction recorded regular activation 91 ms earlier than CS 9,10 (red lines). (C) The fluoroscopy image on a RAO 20 view, which coincides with image B, shows the positional relationship of the leads in the heart. The white straight dotted line represents the ablation position later and the white dotted curve shows the shape of the SVC and RA. (D) When the ablation catheter was placed in the SVC-RA junction (white arrow), the direction of atrial activation was from distal to proximal. (E) AT was terminated when SVC isolation was completed (highlighted by the white arrow). (F) 3D mapping of the patient. The ablation lesions (green dots) at the SVC-RA joint were adjacent to the earliest point of activation on the LA roof (white dots), highlighted by a white arrow.

Accurate diagnosis required careful and stepwise EP mapping and analysis.

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