Ectopic Tachycardia Originating from the Superior Vena Cava

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Ectopic Tachycardia from the SVC. We report a 65-year-old female patient with a 3-year history of symptomatic paroxysmal supraventricular tachycardia. Electroanatomic and basket catheter mapping revealed a focal tachycardia originating in the superior vena cava (SVC), 5 cm above the SVC-right atrium (SVC-RA) junction. An area of fractionated potentials and slow conduction was found on the anterior wall of the SVC. A line of conduction block extending downwardly and obliquely from the anteroseptal aspect to anterolateral aspect of the SVC forcing the impulse to enter the RA via the posterior aspect of SVC-RA junction was observed. Entrainment attempts from multiple sites within the SVC failed to demonstrate reentry as a mechanism of arrhythmia. The ablation approach consisted of isolation of the arrhythmogenic area from the rest of the SVC. (J Cardiovasc Electrophysiol, Vol. 13, pp. 620-624, June 2002)

ectopic tachycardia, superior vena cava, radiofrequency ablation

Introduction

Radiofrequency ablation of atrial premature complexes that originate from the superior vena cava (SVC) and initiate atrial fibrillation has been reported.1, 2 Ino et al.3 reported a patient with atrial tachycardia in whom a rapidly firing focus was located and ablated successfully within the SVC. We report a patient who had a sustained focal tachycardia originating from the SVC. Mapping and successful radiofrequency ablation were performed under the guidance of electroanatomic and multielectrode basket catheter mapping systems.

Case Report

A 65-year-old woman was referred to our hospital for electrophysiologic study and radiofrequency ablation of paroxysmal supraventricular tachycardia that she had for 3 years. During intermittent tachycardia episodes, the patient complained of palpitations, nausea, and dizziness. Twelve-lead surface ECG recorded at the time of the episodes documented a narrow complex tachycardia with irregular AV conduction. During the last 2 weeks, episodes of tachycardia occurred daily and lasted for hours. The patient was treated unsuccessfully with metoprolol and digoxin. She has suffered from arterial hypertension for 3 years. ECG taken at admission and 12-lead 24-hour Holter monitoring showed a sustained supraventricular tachycardia with a Wenckebach-type AV conduction or long intervals of 2:1 AV block. Results of physical examination and chest radiography were unremarkable. Thyroid gland function was normal. Transthoracic echocardiography revealed mild left ventricular hypertrophy with preserved left ventricular function.

Electrophysiologic Study and Radiofrequency Ablation

At the time of electrophysiologic study, the patient was in tachycardia having a cycle length of 270 msec and 2:1 AV block. The surface ECG was highly suggestive of a high right atrial tachycardia with positive P wave on inferior leads and leads I and aVL (Fig. 1A). A 6-French octopolar deflectable catheter (Bard LabSystem; Bard Electrophysiology, Billerica, MA, USA) was inserted in the coronary sinus. Activation mapping during tachycardia was performed with an electroanatomic mapping system (CARTO™, Biosense Webster Ltd., Tirat-HaCarmel, Israel). The earliest local activation was observed on the anterolateral wall of the SVC, 5 cm above the SVC-right atrium (SVC-RA) junction (Fig. 1B). Tachycardia was terminated mechanically by manipulation of the mapping/ablation catheter at that region. Atrial burst pacing reproducibly induced self-terminating tachycardia episodes of the same morphology and location. Activation mapping during tachycardia revealed a line of block about 2 cm beneath the arrhythmogenic area running downwardly and obliquely from the anteroseptal to anterolateral wall of the SVC. Thus, during tachycardia, the impulse was forced to enter the RA via the posterior aspect of the SVC-RA junction. During sinus or atrial paced rhythms, the arrhythmogenic area was the latest region to be activated. Tachycardia could be suppressed by overdrive pacing. Entrainment attempts from several positions within the SVC, including the region with the earliest activation and the zone of fractionated potentials, failed to demonstrate reentry as a mechanism of this tachycardia. The pacing output during entrainment attempts was increased up to a level to enable local capture.

Because the final electroanatomic mapping-based activation map was derived from multiple tachycardia episodes, we decided to perform a simultaneous mapping of the SVC with a basket catheter. For that reason, a 64-electrode basket catheter with a diameter of 38 mm (Constellation™, EP Technologies, San Jose, CA, USA) was inserted from the right femoral vein and deployed in the SVC. The basket catheter enabled mapping of the SVC with 56 bipolar electrograms derived by combining sequential pairs 1-2, 2-3, up to 7-8 of the electrodes on each spline. Electrode positions relative to the SVC were determined in the right anterior oblique 30° and left anterior oblique 45° fluoroscopic views with the help of radiopaque markers. The position of the basket catheter was verified by contrast injection within the SVC (Fig. 2). Anticoagulation was performed by bolus administration of 5,000 IU heparin followed by continuous intravenous heparin infusion to maintain an activated clotting time at about 200 seconds.

SVC potentials were recorded during ongoing tachycardia.
Figure 1. Twelve-lead surface ECG and electroanatomic activation map of the superior vena cava (SVC) and the right atrium (RA) during tachycardia. (A) Surface ECG shows an atrial tachycardia with positive P waves in the inferior leads and 2:1 AV conduction ratio. (B) Electroanatomic activation map of the SVC and RA during tachycardia in right anterior oblique 30° and left anterior oblique 45° views. Red indicates the earliest activation, whereas purple indicates the latest activation. The arrhythmogenic focus (red) was located 5 cm above the SVC-RA junction. Yellow points show the areas of double and fractionated potentials during tachycardia. The color bar in the middle indicates local activation times relative to the reference catheter placed in the coronary sinus. Dark brown points indicate the ablation sites. IVC = inferior vena cava; TA = tricuspid annulus.

Figure 2. Fluoroscopic view of the 64-electrode basket catheter and the mapping/ablation catheter positioned within the superior vena cava (SVC) and the reference catheter placed in the coronary sinus (CS) during contrast injection in the upper segments of the SVC. The tip of the mapping/ablation catheter (MAP) points to the earliest activation area during tachycardia (electrode pair D2/3). Longer electrode seen on the fourth position starting from the SVC-right atrium junction (below the tip of the mapping catheter) marks the position of the spline D. The remaining splines were oriented in a clockwise direction, with the SVC seen from the above.
and sinus or paced rhythms (Fig. 3). The SVC potentials recorded from the posterior wall of the structure consisted of high-amplitude signals. Conversely, highly fractionated and low-amplitude signals suggestive of slow conduction were recorded over the anterior wall of SVC. This region corresponded to the line of block documented by electroanatomic activation maps. During tachycardia, the earliest activation was observed close to the D2/3 electrode pair. Thereafter, the activation front propagated downwardly in an oblique direction and entered the RA at the posterior aspect of SVC-RA junction. Double potentials recorded in electrode pairs C6/7, C7/8, D4/5, D5/6, and E6/7 coincided with the line of block demonstrated by electroanatomic activation maps (Fig. 3A). During sinus and paced atrial rhythms, the impulse followed the same pathway but in a reversed direction. Decremental conduction between the high RA and SVC was demonstrated using programmed electrical extrastimulation, suggesting a rate-related conduction delay at the RA-SVC junction (Fig. 3B).

The goal of radiofrequency ablation was to isolate the arrhythmogenic area. Radiofrequency ablation was performed during sinus rhythm. Radiofrequency energy was delivered at the distal electrode of the thermocouple-equipped catheter (4-mm tip, Navi-Star™; Biosense Webster) with a power limit of 20 W for 30 to 60 seconds and temperature limit of 55 °C at each site. A horizontal ablation line was drawn 2 cm beneath the arrhythmogenic area from the lateral aspect to septal aspect of the SVC. After completion of the ablation line with a total of 13 radiofrequency applications, the arrhythmogenic area was electrically disconnected from the rest of the SVC (Fig. 4). Orciprenaline infusion, programmed atrial extra-stimulation, and burst pacing could not induce arrhythmia immediately or 30 minutes after radiofrequency ablation.

No complications were observed. Angiography after radiofrequency ablations did not show any signs of narrowing of the SVC. The patient was discharged without antiarrhythmic drugs. No stable atrial tachycardias were found on two 24-hour Holter monitoring tests performed 3 and 6 weeks after radiofrequency ablation. During 3-month follow-up, the patient was free of arrhythmia symptoms.

**Discussion**

It has been demonstrated for a long time that cardiac muscle extends for a distance into the SVC in mammalian and human hearts. Zipes and Knope showed that the electrophysiologic characteristics of the SVC and RA muscle were similar. Ito et al. found in rabbit preparations that action potentials obtained from the SVC-RA junction...
showed slight diastolic slow depolarization and under certain circumstances might initiate arrhythmias. Ablation studies in patients with atrial fibrillation demonstrated that SVC could become an arrhythmogenic region.\textsuperscript{1,2}

Ino et al.\textsuperscript{3} reported a patient in whom a tachycardia originated from the SVC and exhibited 2:1 exit block to the atria. At shorter cycle lengths, conduction at the RA-SVC junction was delayed or blocked.\textsuperscript{3} Tsai et al.\textsuperscript{1} described a cohort of eight patients with ectopic beats originating from the SVC; six of these patients showed a Wenckebach or 2:1 SVC-RA conduction pattern. To the authors’ knowledge, this is the first report delineating the activation pattern of sustained ectopic tachycardia originating from the SVC using an electroanatomic mapping or multielectrode basket catheter. In the present case, the electroanatomic mapping and multielectrode basket catheter contributed to our understanding of conduction at the RA-SVC junction and inside the SVC. Conduction delay at the RA-SVC junction was demonstrated by programmed extrastimulation. During sinus rhythm or paced atrial rhythms, the same region manifested characteristics of slow conduction. Moreover, a line of block inside the SVC found during tachycardia was considered a bystander rather than directly involved in the genesis of arrhythmia. Based on the activation mapping, the tachycardia was of focal origin and entrainment technique did not substantiate reentry as a mechanism for arrhythmia. However, we could not elaborate further whether microreentry or triggered activity was the underlying mechanism. Demonstration of large areas of slow conduction located in the SVC might be seen as an index of a degenerative process developed in the SVC. Degenerative changes in the muscle sleeves that surround the human pulmonary veins were demonstrated in a histologic study.\textsuperscript{3} However, a direct link between diseased tissue in the veins entering the heart and arrhythmogenesis is still conjectural rather than well established.

With regard to radiofrequency ablation, we preferred isolation of the arrhythmogenic area rather than a focus-oriented approach. The rationale behind this ablation strategy is supported by the assumption that firing from automatic foci is unpredictable, which weakens the strength of noninducibility as a criterion for ablation. Experience with radiofrequency ablation for paroxysmal atrial fibrillation by electrical disconnection of the pulmonary veins\textsuperscript{8,10} has proven the applicability and accuracy of the isolation strategy as an endpoint for ablation. Complete SVC isolation was not attempted because of the risk of SVC stenosis. Using intracardiac echocardiography, Callans et al.\textsuperscript{11} revealed narrowing of the SVC-RA junction during radiofrequency ablation for inappropriate sinus tachycardia.

Studies of a larger population are warranted to evaluate the efficacy and safety of this ablation strategy and the importance of the SVC as a structure that generates supraventricular arrhythmias.

References
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