Successful Catheter Ablation of Atrial Tachycardia and Atrial Fibrillation in Persistent Left Superior Vena Cava

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Summary

Atrial tachycardia (AT) and atrial fibrillation (AF) were observed in a 21-year old male who had a history of patch closure for an atrial septal defect (ASD) at the age of 5 and a persistent left superior vena cava (LSVC). During electrophysiologic study, atrial extrastimuli reproducibly induced AT which spontaneously terminated or changed into AF. Electroanatomical mapping revealed focal AT arising from the floor of the proximal LSVC. Radiofrequency applications within LSVC targeted to the earliest activation site of AT as well as the complex fractionated potential eliminated both AT and AF without trans-septal puncture. (Int Heart J 2010; 51: 72-74)

Key words: Atrial tachycardia, Atrial fibrillation, Atrial septal defect, Persistent left superior vena cava

Atrial arrhythmia often originates from thoracic veins such as the pulmonary veins (PVs), coronary sinus (CS), superior vena cava (SVC), and the ligament of Marshall. In patients with congenital heart disease, their anomalous structures also would be important for arrhythmias.

Although a left superior vena cava (LSVC) is really not rare as it occurs in approximately 0.4% of the general healthy population and is 10-fold more prevalent among patients with congenital heart disease including ASD,1,2 the electrophysiological properties of LSVC have not been well established.

In the present report, we describe a case of successful catheter ablation to AT and AF both arising from an arrhythmogenic persistent LSVC without ablation in the left atrium (LA) after ASD patch closure.

Case Report

A 21-year-old male was referred to our institute because of recurrent episodes of palpitations. He had a history of surgical repair using a Gore-Tex patch for ASD when he was 5 years old, and had not been given any antiarrhythmic agents. A 12-lead electrocardiogram (ECG) during palpitations revealed a narrow QRS regular tachycardia mimicking intra-atrial macroreentry with a ventricular rate at 120 bpm (Figure 1). A monitor ECG during hospital admission also revealed the presence of paroxysmal AF. Physical examinations were unremarkable. Echocardiography demonstrated normal cardiac function with marked dilatation of the coronary sinus (CS) and persistent LSVC.

After written informed consent was obtained, an electrophysiologic study was performed in the fasting state, free of antiarrhythmic agents. Propofol drip infusion was used for sedation. A steerable decapolar electrode catheter (Snake, Japan Lifeline Co., Ltd. Tokyo) was introduced into the LSVC via the right femoral vein. Likewise, 3 quadripolar electrode catheters were introduced into the right atrium (RA), His bundle region (HBE), and right ventricular apex (RVA) (Figure 2). A Navistar catheter (Biosense-Webster, Diamond Bar, CA, USA) was inserted as a mapping and ablation catheter. Because the patient had refused trans-septal puncture, assessment and intervention in the LA and pulmonary vein (PV) were not performed. Radiofrequency ablation was performed using a 500-kHz generator (EP Technologies, Sunnyvale, CA, USA) with temperature feedback. Targeted temperature and maximum power were 50°C to 55°C and 40W, respectively.

Figure 1. Twelve-lead electrocardiogram of the clinical tachycardia. The ventricular rate was 120 bpm, and the defect in the isoelectric line suggests intra-atrial macroreentry.
AT AND AF ARISING FROM L SVC

Extra stimuli could induce two types of AT whose cycle lengths were 220 msec (AT1) and 280 msec (AT2), respectively (Figure 3A, B). Detailed assessment including entrainment study was quite difficult, because the ATs switched with each other, terminated immediately, or changed into AF which required some electrical cardioversion to recover sinus rhythm and was time consumptive. Furthermore, as the session elapsed, both ATs showed a tendency to disorganize. Fortunately, we could obtain an activation map of AT2 using a CARTO system (Biosense-Webster, Diamond Bar, CA, USA) which revealed a discrete focus of origin at the bottom of the proximal LSVC and its centrifugal propagation (Figure 3C). A voltage map during sinus rhythm delineated no significant low voltage area (< 0.5 mV) in either the LSVC or RA. Incision scars and the intra-atrial patch did not seem to affect the ATs.

After coronary angiography to confirm the locations of the major coronary arteries, we delivered radiofrequency energy during AF to the bottom of the LSVC at a point approximately 1 to 2 cm distal to the ostium at which the earliest activation of AT2 was recorded. After several applications, RA extra stimuli could no longer induce AT1, but AT2 and AF were still inducible. Further mapping during AF demonstrated complex fractionated potential in the proximal bottom portion of the LSVC and in the distal portion of the LSVC near the ligament of Marshall and LSPV, but not in the RA (Figure 4). Ablation targeting complex fractionated potential gradually resulted in AF organization. Finally, AF terminated during ablation. Thereafter, atrial extra stimulation could induce neither AT nor AF in the basal condition or under infusion of 2 μg/kg/min of isoproterenol. The cumulative total delivered energy amounted to 14,006 J. The postprocedural course was uneventful. No recurrence was observed within a 1 year period during which no antiarrhythmic agents were administered.

**Discussion**

**LSVC as an arrhythmogenic substrate:** Though the unsteadiness of ATs made it quite difficult for us to determine their properties during electrophysiological study, we could eliminate AT1, AT2, and even AF only in RA and LSVC. Considering this result retrospectively, both AT1 and AT2 would have a common origin at proximal LSVC, and slow conduction expressed as complex fractionated potential during AF might be a critical substrate of the AF.

Thoracic veins including PVs, 3 coronary veins, or the ligament of Marshall have been regarded as a substrate for initiating or perpetuating atrial arrhythmia. Abnormal automaticity, triggered activity, and reentry could all potentially function as their arrhythmogenicity. Since the LSVC is also a thoracic vein, it seems...
reasonable that the LSVC can play an important role in the pathogenesis of atrial arrhythmia. S6

Actually, Maruyama, et al S3 characterized the electrical activity within LSVC, and demonstrated the presence of multiple LSVC-LA connections and the resemblance between LSVC and the ligament of Marshall. Hsu, et al S6 reported 5 cases whose LSVC were the source of ectopies initiating AF, and emphasized the necessity of the electrical isolation of LSVC from both atria to terminate AF. Elayi, et al S10 and Hao, et al S11 reported 4 and 5 similar cases, respectively, and concluded that electrical isolation of 4 PVs is not sufficient to prevent AF recurrence in most patients with LSVC.

Attention should be paid to LSVC not only as an anomalous structure but also as an arrhythmogenic substrate in patients with atrial arrhythmias and congenital heart disease.

Safety, efficacy, and complications of ablation in LSVC: To the best of our knowledge, there have been no reports on the specific success rate, recurrence rate, or complications related to ablation within an LSVC. Katsouras S12 and colleagues reviewed the implications, efficacy, and safety of CS ablation. However, they also warned about potential complications, such as coronary stenosis, AV block, pain, vein thrombosis, and cardiac tamponade. Attention should be paid to nearby structures such as coronary arteries, the conduction system, and esophagus. Judiciously titrated lower energy, cryoaolation, and coronary angiography before ablation may reduce the risk of complications even in LSVC.

Trans-septal puncture: The patient refused to undergo a trans-septal puncture, which made us unable to assess the electrical properties in the LA, the relationship between the LA and LSVC, which might be vital for a comprehensive understanding of our case, and even pulmonary vein isolation. Fortunately, we were able to treat the patient without LA access. Although El-Said, et al S6 concluded trans-septal puncture over a prosthesis is safe and feasible, we respected the patient’s decision to refuse a trans-septal puncture.

In summary, we experienced a case with AT and AF that originated from persistent LSVC after ASD repair. Both the AT and AF were successfully eliminated by radio-frequency ablation only within LSVC. This report demonstrates the importance of an LSVC as a potential source of atrial tachycardia and a substrate perpetuating AF.

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REFERENCES