Atrial fibrillation ablation with persistent left superior vena cava detected during intracardiac echocardiography

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Introduction

Atrial fibrillation ablation is an increasingly frequent procedure performed in the electrophysiology laboratory. Pulmonary vein foci account for more than 90% of triggers for atrial fibrillation, most commonly treated with complete pulmonary vein isolation most of the time.[1] Non–pulmonary vein triggers are the drivers of atrial fibrillation in a subgroup of patients. This patient had an atrial tachycardia originating from a persistent left superior vena cava (SVC).

Case report

A 67-year-old man with a 10-year history of symptomatic paroxysmal atrial fibrillation was having increasingly frequent episodes leading to hospitalization 5 times while being treated with sotalol 120 mg twice daily. The sotalol dose was increased briefly to 160 mg twice daily, although it was then reduced because of corrected QT interval prolongation. The patient was referred for pulmonary vein isolation for control of medically refractive symptomatic paroxysmal atrial fibrillation. Transesophageal echocardiography before the procedure showed normal left ventricular function and moderate left atrial enlargement.

After obtaining right femoral venous access, catheters for the procedure were advanced to the right atrium under fluoroscopic guidance, including a 3.5-mm irrigated tip ablation catheter (SmartTouch CARTO, Biosense Webster, Inc., Diamond Bar, CA), a Lasso catheter (Biosense Webster, Inc.), and an intracardiac echocardiogram probe. A duodecapolar catheter was placed in the coronary sinus (CS) after obtaining right internal jugular venous access. Surrogate geometry of the right and left atria was constructed with intracardiac echocardiography and CartoSound (Biosense Webster, Inc.), demonstrating normal anatomy of the pulmonary veins. Esophageal temperatures were monitored during ablation using an esophageal probe. The ablation catheter and Lasso catheter were advanced into the left atrium after transseptal punctures. The Lasso catheter was then placed into each of the 4 pulmonary veins, which showed pulmonary vein potentials and absence of entrance block. Wide antral circumferential ablation was performed around the pulmonary veins, as well as carina linear ablation procedures on the right and left with a power output of 25 W along the posterior wall and 35 W otherwise. Entrance block was demonstrated for each of the 4 pulmonary veins with the absence of pulmonary vein potentials recorded on the Lasso catheter from inside the ostium of each vein, with pacing from the distal and proximal CS for the left and right pulmonary veins, respectively. Entrance block for each pulmonary vein was reconfirmed during intravenous infusion of isoproterenol, and there was no evidence of acute pulmonary vein reconnection. In addition, spontaneous pulmonary vein potentials were recorded in the left superior pulmonary vein, which did not conduct outside of the circumferential ablation and therefore was highly suggestive of exit block. Circumferential pacing from the Lasso catheter was performed inside the remaining 3 pulmonary vein ostia without atrial capture, confirming exit block.

After isolating the pulmonary veins, we observed spontaneous isolated atrial extrasystoles and frequent runs of nonsustained atrial tachycardia with a cycle length of 181 ms. The ablation catheter was placed in the distal CS and advanced beyond the distal poles of the CS catheter (Figure 1A). The earliest potential was recorded with the mapping catheter and was found to be 24 ms earlier than the earliest electrogram recorded on the distal poles of the CS catheter (Figure 2). The focus of earliest activation was ablated with a power output of 15 W, terminating the tachycardia within 11 seconds of starting the ablation lesion, and continued for a total of 60 seconds. The frequently occurring spontaneous bursts of tachycardia were not noted to be active and remained noninducible with burst pacing, both with and without isoproterenol infusion, even after 30 minutes of waiting.

Marked dilation of the CS was observed on intracardiac echocardiography, raising suspicion for a persistent left SVC (Figure 3). A CS venogram was performed to further delineate the anatomy and showed marked dilation of the

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KEY TEACHING POINTS

- Although the pulmonary veins account for 90% of triggers for atrial fibrillation, nonpulmonary vein triggers may rarely be the drivers of the arrhythmia. A persistent left superior vena cava (SVC) may be one such trigger.
- A dilated coronary sinus on intracardiac echocardiography during the procedure may suggest the presence of a left SVC, if not known beforehand. This may be confirmed with a left upper extremity peripheral venography demonstrating contrast runoff into the left SVC.
- Ablation at the earliest site of activation in the left SVC may be sufficient to eliminate the triggering focus, deferring the need for circumferential ablation and associated risks in some cases.

vessel (Figure 1B). A left upper extremity peripheral venogram demonstrated contrast runoff into a persistent left SVC, which drained into the vein of Marshall before draining into a dilated CS (Figure 1C). After the procedure, the dilated CS was again visualized on transthoracic echocardiography. Our patient is no longer taking sotalol and has now been free from symptomatic atrial fibrillation for 6 months after ablation.

Discussion

The pulmonary veins account for the majority of foci that trigger atrial fibrillation, although nonpulmonary vein foci may explain arrhythmia recurrence in some patients after pulmonary vein isolation.[2] One such trigger is a persistent left SVC, which may contain remnant muscular and pacemaker tissue carried over from embryonic life.[3] The incidence of a left SVC is reported to be 0.2% in patients with normal hearts and 2.9% in patients with other congenital heart defects.[3,4]

The normal size of the CS at the ostium ranges from 4×5 to 11×16 mm.[5] Dilation of the CS on transthoracic echocardiography may suggest the presence of a persistent left SVC, although other causes of CS dilation include pulmonary hypertension, anomalous pulmonary venous return, an unroofed CS, and a coronary artery–CS fistula.[6] In the parasternal long-axis view, a left SVC can be differentiated from other causes of CS dilation with opacification of the CS before the right ventricle when contrast medium is injected into the antecubital vein of the left arm. Dilation of the CS can also be appreciated on intracardiac echocardiography and should prompt further investigation into the possibility of a left SVC, if not known beforehand. Before the ablation procedure, a chest computed tomography angiogram will also identify a left SVC.

Separate electrical connections may exist from the left SVC to the CS and to the left atrium, which is at the level of the left superior pulmonary vein.[7] These connections allow an arrhythmogenic focus in the left SVC to propagate atrial arrhythmias. Atrial extrasystoles originating beyond the distal CS should prompt further mapping in the left SVC to identify the earliest electrogram. Ablation at the earliest site of activation in the left SVC may be sufficient to eliminate the triggering focus in some cases. Pacing from the left SVC with the Lasso catheter in the left superior pulmonary vein can also determine whether an electrical connection remains from the left SVC to the left atrium. Although complete electrical isolation of a persistent left SVC may prevent a potential new trigger causing arrhythmia recurrence in the future, the long-term benefit of such an extensive ablation is unclear. The risk of complete isolation needs to be weighed against the potential benefits of limited ablation.

The success of a focal ablation strategy for our patient has been demonstrated in a prior report, where focal

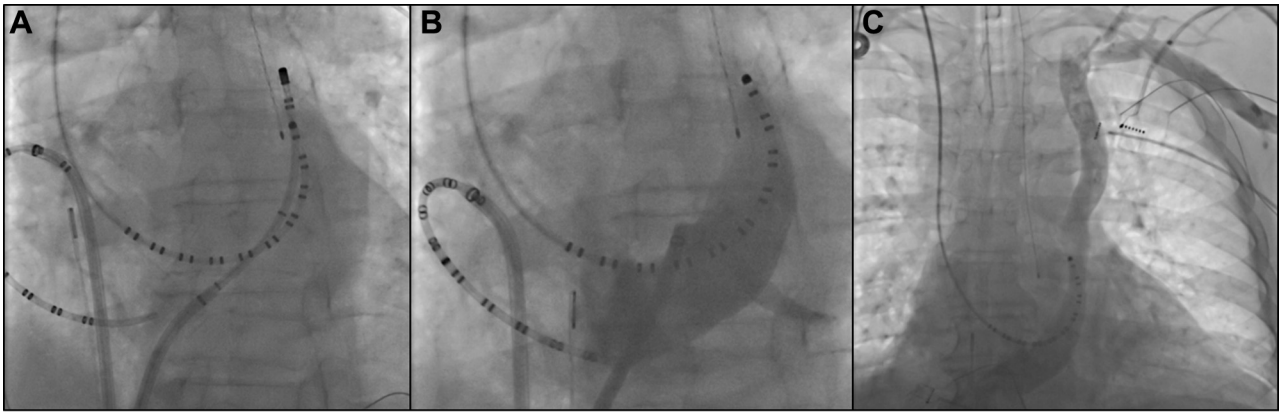

Figure 1 Fluoroscopic images. **A:** Ablation catheter placed at the focus of earliest activation, distal to the distal poles of the duodecapolar coronary sinus catheter. **B:** Coronary sinus venogram with the duodecapolar catheter placed in a dilated coronary sinus. **C:** Left upper extremity peripheral venogram showing contrast runoff into a persistent left superior vena cava.