Electrogram-guided isolation of the left superior vena cava for treatment of atrial fibrillation

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Aims Radiofrequency ablation targeting the pulmonary veins offers potential cure for patients with symptomatic paroxysmal atrial fibrillation (AF). Initiating ectopics can also arise from other sites including the major thoracic veins, vein of Marshall and more rarely, persistent left superior vena cava (LSVC). We report our experience with arrhythmogenic persistent LSVC initiating AF.

Methods and results The LSVC was present in four patients from an overall series of 204 patients undergoing AF ablation at our centre. All were males, mean age 50 ± 11 years. All patients underwent pre-procedure transesophageal echocardiography. The mapping of the LSVC was performed with a circumferential mapping catheter following pulmonary vein isolation. Atrial ectopics from the LSVC were observed to initiate AF. Catheter ablation (power controlled mode; 65°C and 30 W at irrigation flow rate of 30 mL/min) resulted in electrical isolation of the LSVC in all patients and was accompanied by termination of AF in one of four patients. There were no complications. All patients underwent multiple procedures (three procedures in one patient, two procedures in three patients). After a mean follow-up of 18 ± 7 months (range 7–24 months), three of the four patients remained free of AF off antiarrhythmic medications.

Conclusion Arrhythmogenic foci within persistent LSVC can result in AF despite electrical isolation of pulmonary veins. This report demonstrates the importance of the LSVC as a potential source of atrial ectopics initiating and perpetuating AF.

Introduction

The role of the thoracic veins in atrial arrhythmogenesis is most clearly established for the pulmonary veins while the superior vena cava (SVC),⁴⁵ coronary sinus (CS),⁴ vein of Marshall and more rarely, the inferior vena cava (IVC)⁸⁹ have also been implicated. The left superior vena cava (LSVC) usually regresses during embryological development to form the diminutive vein of Marshall. Uncommonly, the LSVC persists and forms the major conduit for the upper extremity venous return. Recently, five cases of atrial fibrillation (AF) originating from the LSVC were described in a multicentre experience.¹⁰ We describe our single centre experience of an additional four cases.

Methods

Patients

Four patients (all males, mean age 50 ± 12 years) from a consecutive catheter ablation series of 204 patients with symptomatic drug-refractory AF were found to have arrhythmogenic activity in the LSVC. Two of the four patients were amateur sportsmen who identified vigorous exercise as an AF precipitant (Table 1). All patients underwent pre-procedure imaging by magnetic resonance (MR) imaging (three patients) or 64-slice computed topography (CT) scanning (one patient). All MR examinations were performed using a Siemens Sonata 1.5 T machine with gradient strength of 40 mT/m and a slew rate of 200 T/m per second (Figures 1 and 2).

Electrophysiology study

Written informed consent was obtained from all patients. A pre-procedure transesophageal echocardiogram was performed to exclude left atrial thrombus. Multi-electrode catheters were placed within the CS, right atrium, and transseptally into the left atrium. In Patient 1, a venogram of the LSVC was performed using contrast injection via a Preface long sheath (Figure 3). Mapping of the LSVC was performed with a circumferential mapping catheter following pulmonary vein isolation by a standard previously described technique.¹¹ The circumferential mapping catheter (Lasso) was introduced retrogradely through the CS via a long sheath (Preface 62 cm, Biosense-Webster, Diamond Bar, CA, USA) and was positioned at the level of the left superior pulmonary vein (LSPV) ostium (Figure 4). As previously described by Hsu et al.,¹² it was possible to identify a signal with two components: a far-field LA and local LSVC potential. During sinus rhythm, the LA potential preceded the LSVC potential and this sequence was reversed during ectopy and initiation of AF. Pacing the LA and

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Table 1  Patient characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>LA size (mm) (M mode parasternal)</th>
<th>AF type</th>
<th>Duration AF (months)</th>
<th>Failed AAD</th>
<th>Number of procedures</th>
<th>LSVC ablation (min)</th>
<th>Follow-up (months)</th>
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<td>38</td>
<td>PAF</td>
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<td>3</td>
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<td>PAF</td>
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<td>BB,A,S,F</td>
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</tbody>
</table>

S, Sotalol; F, Flecainide; A, Amiodarone; BB, Beta blocker; PAF, Paroxysmal atrial fibrillation.

Figure 1  Pre-procedure MR imaging. (A) Patient 1: single frame from a coronal acquisition time resolved 3-D gadolinium enhanced MR angio-
gram demonstrating LSCV draining into a dilated CS. (B) Patient 2: coronal maximum intensity projection image from a 3-D gadolinium enhanced MR angiogram. Venous and arterial tree are demonstrated. A LSVC is seen. (C) Patient 3: single frame from a coronal steady-state free precession image demonstrating dual SVC system.

Figure 2  Pre-procedure CT imaging (Patient 4). (A) Posteroanterior view demonstrating the relationship between LSVC and LSPV. RA, Right atrium; CS, Coronary sinus. (B) Coronal section (anteroposterior view). (C) Left lateral view.
LSVC at low output was used to validate the LA and LSVC signals. Low-output pacing of the LA captured the first signal while pacing LSVC resulted in capture of the second signal (Figure 5). Atrial ectopics from the LSVC were observed to initiate AF spontaneously in one patient and during isoproterenol infusion in the remaining three patients. In Patient 4, the CARTO electroanatomical-mapping system was used to create a CARTO merge image. The position of the circular mapping catheter within the LSVC was tagged to indicate the level of isolation on the CT image (Figure 6).

Catheter ablation

Ablation was performed using a D-curve 5 mm or 3.5-mm tip externally irrigated catheter (Thermocool or Navistar Thermocool, Biosense-Webster, Diamond Bar, CA, USA) positioned retrogradely within the LSVC via the right femoral vein. Radiofrequency (RF) applications were delivered within the LSVC targeting the LSVC potentials with temperature and power limited to 65°C and 30 W at irrigation flow rate of 17–30 mL/min. The temperature limit was never reached because of the irrigation. The desired end-point of ablation was loss or dissociation of the local venous potential from the LSVC. Repeat procedures were performed if patients had on-going AF >1 month following ablation. At repeat procedures, pulmonary veins and the LSVC were checked for conduction recovery and ablation was performed as required to achieve isolation.

Follow-up

All patients were followed-up by the treating clinician for symptom recurrence. A repeat 24-h Holter monitor was performed at 6–9 months following the last ablation procedure to check for silent AF. Post-procedure imaging by CT or MR was performed at 6 months after the last ablation procedure to exclude high-grade venous stenosis.

Results

Cardiac anatomy

Dual SVC system with both left and right SVC was observed in one patient. The remaining three patients had LSVC only.

Arrhythmias

The LSVC was mapped with a Lasso catheter following pulmonary vein disconnection. The patients were all in sinus rhythm at baseline. Ectopics originating from the LSVC were seen to initiate AF in all patients, spontaneously in one patient and with isoproterenol in the remaining three patients. Typical atrial flutter was also seen in one patient.

Mapping and ablation

All patients underwent multiple ablation procedures because of clinical recurrence of AF (three procedures in one patient, two procedures in three patients). The mean duration of RF energy applied to LSVC during the first procedure was 16 ± 12 min (temperature controlled mode; 65°C and 30 W at irrigation flow rate of 30 mL/min). One of the four patients also underwent cavotricuspid isthmus ablation for typical atrial flutter. Electrical isolation of the LSVC occurred in all patients and was characterized by the
Figure 5  Left SVC potential validation. (A) During sinus rhythm, the two components (left atrial far-field and LSVC potential) are fused. (B) During left atrial appendage pacing, the LSVC potential follows the LA far-field signal in LSVC 1,2. (C) During LSVC pacing via the circular mapping catheter, the LSVC potential precedes the far-field LA in LSVC 1,2. (D) Following ablation, the LSVC potentials are abolished and only far-field LA signals remain.

Figure 6  Sinus rhythm Carto merge (AP view) map from Patient 4. The white circle indicates the level of the circular mapping catheter. Green tag indicates the ostium of dilated CS, yellow tag is the His bundle, Orange tags are the tricuspid annulus, light blue tag is the IVC, and dark blue tag is the circular mapping catheter.
loss of the local venous potential (rather than dissociation). At the repeat procedures, incomplete recovery of LSVC potentials was seen in each patient and was characterized by delayed and fractionated electrograms. During repeat procedures, a mean of 9 ± 6 min RF applications (temperature controlled mode; 65°C and 30 W at irrigation flow rate of 17–30 mL/min) was required to re-isolate the LSVC.

Follow-up
No complications were observed in all patients during the mean follow-up of 18 ± 7 months (from the last procedure). There were no stenoses in the LSVC on either cardiac CT or MR at 6 months post-ablation. All patients underwent routine ambulatory ECG recordings and remained free of silent AF at 6 months. One patient remained on anti-arrhythmic therapy (Flecainide) and had symptomatic recurrence at 10 months.

Discussion
Arrhythmogenic foci can arise from not only within the pulmonary veins but also in major thoracic veins, vein of Marshall, CS, IVC, and in persistent LSVC. With the exception of IVC, these veins have electrically active muscle sleeves in most individuals. Persistent LSVC is an uncommon finding with a prevalence rate estimated at 0.3% in patients without other congenital heart malformations. The presence of a persistent LSVC is particularly uncommon in, otherwise, structurally normal hearts. While the true incidence of LSVC among the patients with AF is unknown because of its rarity, Hsu et al. reported 3 cases in 851 consecutive patients undergoing the catheter ablation of AF. The presence of a large CS on echocardiography should raise suspicion of a persistent LSVC, which can be confirmed with agitated saline injection via the antecubital vein. Diagnosis can similarly be obtained with transesophageal echocardiography, which not only allows demonstration of the venous anomaly but also excludes any co-existent congenital cardiac anomalies. Dual SVC system is most prevalent with LSVC drainage into right atrium via the CS, easily demonstrated on echocardiography. Other imaging techniques such as cardiac CT and MR may also detect cardiac venous anomaly as demonstrated in the present experience.

The embryonic remnant of the LSVC is the ligament of Marshall (LOM). The LOM is comprised of a vestigial fold in the back of the left auricle, extending from the CS to the orifice of the LSPV and containing the vein of Marshall as well as muscle sleeves from the CS. Repetitive activities from the LOM initiating AF have been described.

In our experience, electrical isolation of the LSVC was similar to the previous report except that in the description of Hsu et al. ablation was carried out from the LA and LSVC while we limited energy delivery to the LSVC only. This could have accounted for the greater need for repeat procedures in our series. Despite extensive energy delivery, the stenosis of the LSVC was not seen. In the developing embryo,pacemaker cells are found at the junction of the common cardinal veins with the horns of the sinus venosus. The sinoatrial node derived from the right pacemaker cells, with regression of the left sided veins, represents the principal pacemaker of the cardiac conducting system in adults. Developmental abnormalities leading to a persistent LSVC may therefore be associated with abnormalities of cardiac conduction and ectopic pacemaker cells. It was interesting that AF in two of four of our patients suffered AF, which was largely exercise-induced and one may speculate that initiating ectopics from the LSVC arise from ectopic pacemaker cells.

In conclusion, arrhythmogenic foci within persistent LSVC can result in AF despite electrical isolation of pulmonary veins. This report demonstrates the importance of the LSVC as a source of atrial ectopics initiating AF. Consideration should be given towards isolating the vein if it is found to be arrhythmogenic.

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