

Para-Hisian atrial tachycardia ablation in a patient with persistent left superior vena cava and absent right superior vena cava



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Introduction

A persistent left superior vena cava (PLSVC) is a relatively infrequent congenital anomaly, but the combination of PLSVC with an absent right superior vena cava (RSVC) is rare.¹ These anomalies are often discovered incidentally during various medical procedures. Focal atrial tachycardias (ATs) typically originate from specific sites in the atria, such as the crista terminalis, atrioventricular annuli, pulmonary vein ostia, coronary sinus (CS) ostium/musculature, atrial appendages, and the superior vena cava (SVC). Additionally, the para-Hisian region, particularly toward the apex of the triangle of Koch, has been identified as another distinct site of AT origin.² This report presents a novel case of para-Hisian AT ablation in a patient with a PLSVC and absent RSVC, which has not been previously reported.

Case report

A 57-year-old male patient with a medical history including hypertension, hyperlipidemia, obesity, aneurysmal aortic root dilatation, and paroxysmal atrial fibrillation (AF) underwent prior pulmonary vein isolation using cryo energy along with cavotricuspid isthmus ablation using radiofrequency at an external institution 18 months earlier. A preprocedural computed tomography scan revealed a left SVC without a corresponding RSVC (Figure 1A–1C). Transthoracic echocardiogram exhibited an ejection fraction of 60%, normal left ventricle size, mild left atrium enlargement, and a prominent CS, consistent with a PLSVC. Despite treatment with metoprolol and propafenone for symptomatic paroxysmal AF occurring weekly, there was no clinical improvement.

KEYWORDS Para-Hisian; Triangle of Koch; Atrial tachycardia; Atrial fibrillation; PLSVC; RSVC

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Consequently, the patient underwent a repeat ablation procedure at our institution.

Procedure

Catheters were guided into the right atrium via right femoral venous access. A CS catheter was attempted from the right internal jugular access to record the electrogram from the PLSVC (right internal jugular is our routine practice but were prepared to move to left internal jugular or left subclavian if necessary). Despite initial difficulty, a duodecapolar catheter (Boston Scientific, Marlborough, MA) was placed in the left CS. Contrast venography aided in the placement of the left-sided SVC. An intracardiac echocardiography (ICE) probe (CartoSound, Biosense Webster Inc, Irvine, CA) was introduced into the right atrium via the inferior vena cava. A dual transseptal puncture was performed to access the left atrium using ICE guidance with the help of 2 operators owing to absent RSVC (Figure 2A and 2B).

Electroanatomical mapping of the pulmonary veins was achieved using the Rhythmia (Boston Scientific, Marlborough, MA) mapping system, confirming the isolation from the prior procedure. Isoproterenol infusion triggered AF, revealing a triggering ectopic beat originating from the CS distal poles (owing to PLSVC, the distal poles were near the CS ostium). Subsequent attempts at initiation with burst pacing to induce the triggering beat led to AF with a ventricular rate of 200 beats per minute and hypotension requiring multiple cardioversions.

The Orion catheter (Boston Scientific, Marlborough, MA) was maneuvered into the left-sided SVC, revealing that the earliest signal for the premature atrial complex triggering AF was near the roof of the CS, closer to the ostium (Figure 3A–3C). Minimal activation owing to infrequent premature atrial contractions and multiple attempts at pace mapping were performed to template match on 12-lead surface electrocardiography and intracardiac matching. After satisfactory confirmation of coherence between activation and pace mapping, an ablation catheter was positioned at

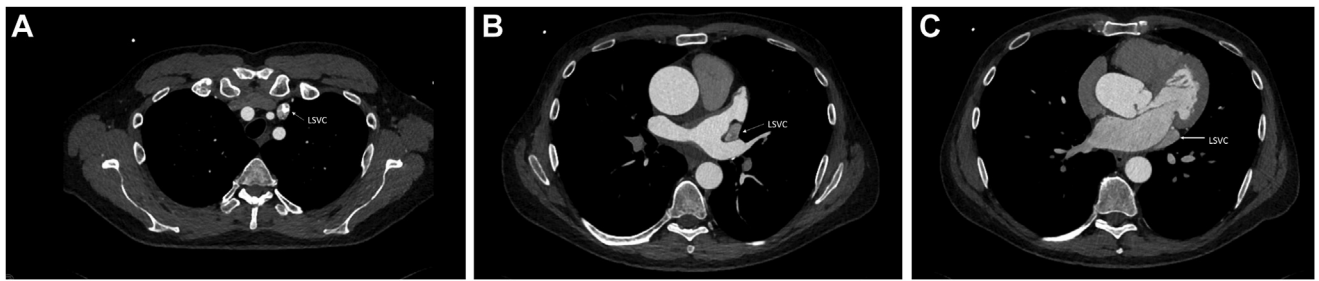


Figure 1 A–C: Preprocedural computed tomography imaging showing course of LSVC. LSVC = left superior vena cava.

the CS ostium (Figure 3C and 3D) and initiated at 20 W with titration to 30 W. During ablation, a junctional rhythm was immediately noticed, exhibiting continuous 1:1 VA conduction. Insurance lesions were applied, followed by attempts to reinduce AF with an identical initiation protocol, including the use of adrenergic agents like preablation. Neither the triggering beat nor AF was inducible. The Halo catheter confirmed the bidirectional block along the tricuspid annulus from the prior procedure. After the procedure, the patient remained asymptomatic during the 12-month follow-up.

Discussion

This case underscores the complexities encountered in managing patients with PLSVC and absent RSVC during ablation procedures, emphasizing the anatomical challenges posed by the para-Hisian region.

Understanding tachycardias originating from this specific region is crucial, as they must be distinguished from those originating from the broader tricuspid annuli category.² The mechanism behind focal ATs can be elusive clinically but can often be inferred from their initiation, termination, and response to overdrive pacing.³

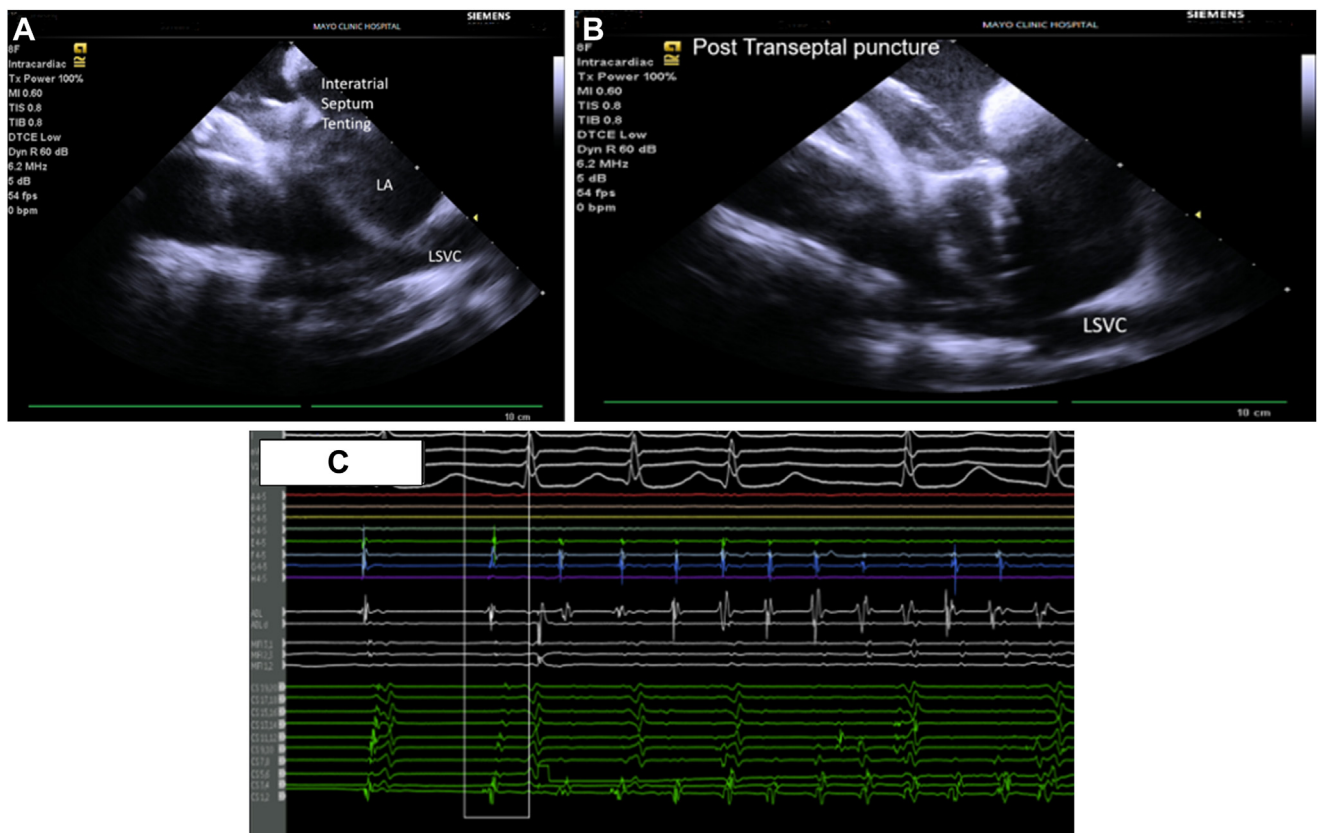


Figure 2 A, B: A dual transeptal puncture was performed to access the left atrium (LA) using intracardiac echocardiography guidance. LSVC = left superior vena cava. C: Coronary sinus (CS) distal electrogram near CS ostium showing initiating beat with atrial fibrillation onset.

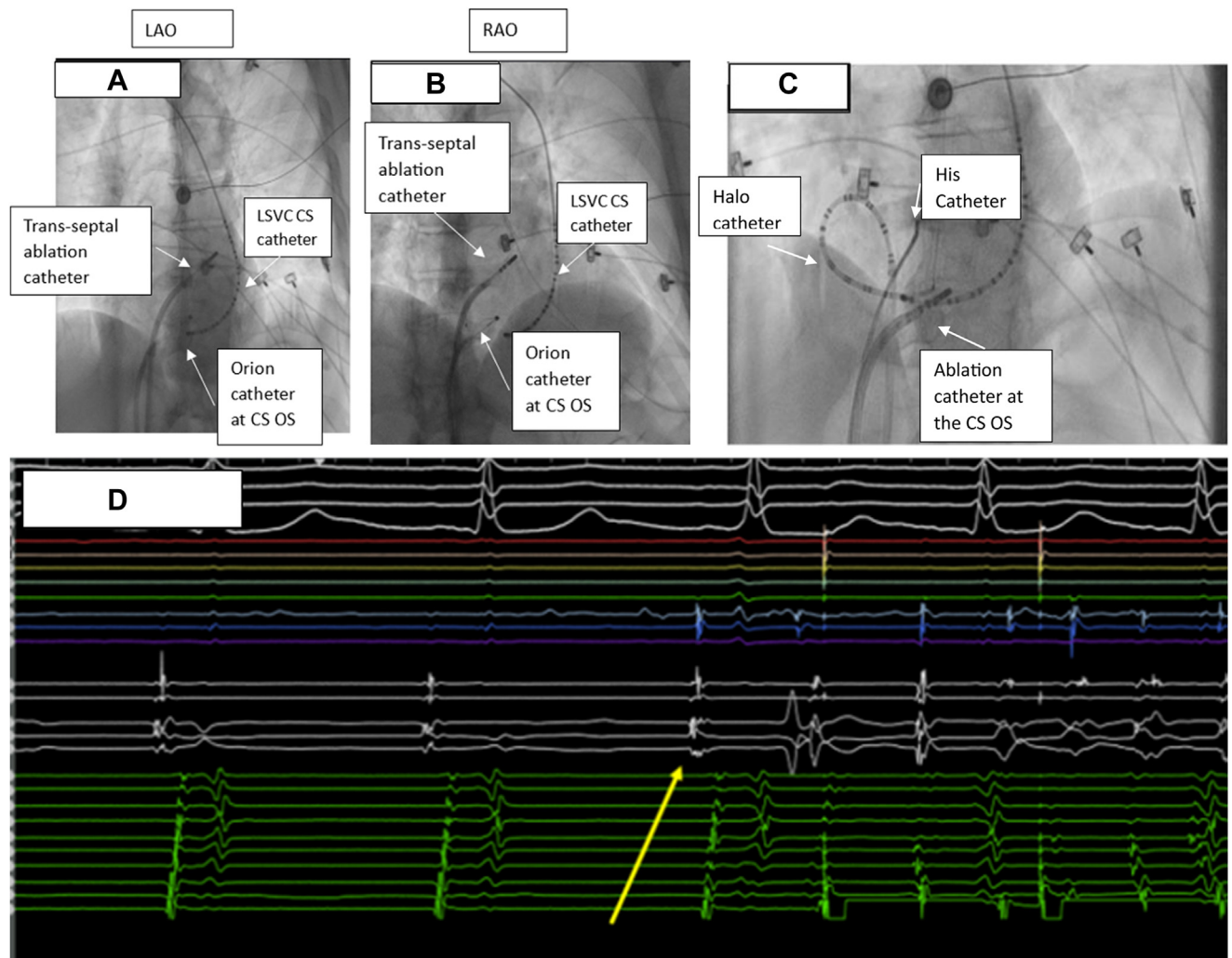


Figure 3 A–C: Fluoroscopy images showing left anterior oblique (LAO; **A**) and right anterior oblique (RAO; **B**) mapping with the Orion catheter (Boston Scientific, Marlborough, MA) of the coronary sinus ostium (CS OS, LAO; **C**). LSVCS = left superior vena cava. **D**: Ablation electrogram showing fractionated electrogram from the ablation site.

When a persistent left cardinal vein transforms into a PLSVC, it drains into the right atrium via an enlarged CS, a characteristic observed on transthoracic echocardiograms. In such cases, the conventional approach to sheath and dilator insertion into the SVC and engagement with the fossa ovalis becomes challenging owing to complex anatomy, often necessitating the use of ICE for transseptal puncture.⁴

The absence of the RSVC results in the CS being the sole drainage route in PLSVC patients, and the enlarged CS ostium can distort normal anatomy, potentially altering the location of the His bundle and causing electrophysiological changes or arrhythmogenic foci. Additionally, it reduces the space between the CS roof and the location of the His bundle. Ablation near an enlarged CS ostium, particularly at the roof of the CS, can inadvertently damage nodal tissue, leading to atrioventricular block.

Studies have identified PLSVC as a trigger for AF, underscoring the technical challenges associated with isolating PLSVC and the importance of recognizing coexisting ar-

rhythmias during ablation in patients without an RSVC.⁵ Risks associated with ablation in PLSVC include left phrenic nerve injury, cardiac tamponade, and SVC syndrome owing to vascular stenosis.⁵

The concurrent association of PLSVC and AT within the Koch triangle has not been previously described.

Conclusion

This case underscores the importance of identifying the para-Hisian region as a potential trigger for atrial arrhythmias in patients with PLSVC. Preprocedural imaging via computed tomography or cardiac magnetic resonance imaging is recommended to identify such anomalies. Unusual CS appearances on ICE should alert operators to the possibility of a PLSVC and confirmation can be aided by a CS venogram. Given the propensity for para-Hisian AT to induce AF, a trigger search after pulmonary vein isolation may be necessary for all such patients.

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