Ablation of Persistent Left Superior Vena Cava in Atrial Fibrillation Case

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Key clinical message:

This case report discusses the successful ablation of a persistent left superior vena cava (PLSVC) as a trigger for atrial fibrillation. Following cryoballoon ablation and subsequent radiofrequency ablation, the patient experienced no recurrence of atrial fibrillation over a six-month follow-up period, confirmed by monthly ECG and Holter monitoring.

Introduction:

We report a case of persistent atrial fibrillation (AF) with a persistent left superior vena cava (PLSVC). The patient underwent cryoballoon ablation, improving cardiac function. Six months later, the patient experienced paroxysmal AF and underwent radiofrequency ablation. Intracardiac echocardiography and mapping confirmed PLSVC as the AF trigger. Isolation of the PLSVC successfully terminated AF. No AF episodes occurred during the six-month follow-up.

Case Presentation:

A 58-year-old female patient was admitted to the hospital on February 22, 2023, due to recurrent palpitations accompanied by shortness of breath that she had experienced for more than three years. The initial electrocardiogram (ECG) indicated atrial fibrillation (AF), and an echocardiogram revealed enlargement of both the left and right atria, along with a decreased ejection fraction (Left Atrial Size [LAS] 50mm, Left Ventricular Diameter [LVD] 55mm, Right Atrial Size [RAS] 39mm, Right Ventricular Diameter [RVD] 35mm, Ejection Fraction [EF] 33%). She underwent her first cryoballoon ablation procedure, after which her heart rhythm returned to normal sinus rhythm.

On November 23, 2023, she was readmitted to the hospital due to persistent palpitations that had lasted for over three months. During these episodes, the ECG again showed atrial fibrillation. A follow-up echocardiogram indicated a reduction in the size of both the left and right atria, and the ejection fraction had improved to normal (LAS 39mm, LVD 45mm, RAS 34mm, RVD 29mm, EF 61%).

Methods (Electrophysiological Study and Ablation Procedure):

The patient underwent her first surgery with considerations for heart failure, as her ejection fraction (EF) had decreased, making it difficult for her to tolerate lengthy radiofrequency ablation procedures and intraprocedural saline infusion. Therefore, cryoablation was chosen for treatment. After the procedure, the patient maintained sinus rhythm for six months before experiencing a recurrence of paroxysmal atrial fibrillation.

During the patient's second radiofrequency ablation procedure, pre-operative assessments showed she was in sinus rhythm (Figure 1A). Intracardiac echocardiography (ICE) revealed the presence of a Persistent Left Superior Vena Cava (PLSVC) (Figure 1B). The intracardiac three-dimensional mapping using the Carto 3 system demonstrated electrical reconnection in the left and right pulmonary veins (Figures 1C-F).

The ablation steps began with gap ablation within the pulmonary veins, followed by an expansion of the ablation area and linear ablation at the roof (Figure 1G).

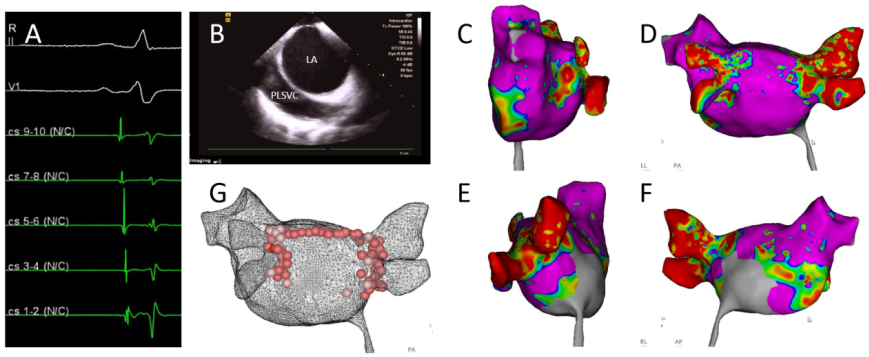

Figure 1: A: The preoperative electrocardiogram indicates sinus rhythm.

B: Intracardiac echocardiography (ICE) shows an echolucent shadow alongside the left atrium (LA), consistent with a Persistent Left Superior Vena Cava (PLSVC).

C: Three-dimensional electroanatomical map of the left atrium in sinus rhythm, viewed from the left side.

D: Three-dimensional electroanatomical map of the left atrium in sinus rhythm, viewed from the posterior-anterior perspective.

E: Three-dimensional electroanatomical map of the left atrium in sinus rhythm, viewed from the right side.

F: Three-dimensional electroanatomical map of the left atrium in sinus rhythm, viewed from the anterior-posterior perspective.

G: Ablation steps include gap ablation within the pulmonary veins, expansion of the ablation area, and linear ablation at the roof.

Post-operatively, AF was induced using an S1S1 pacing protocol (Figure 2A), suggesting ectopic electrical activity originating from non-pulmonary vein sources. Subsequently, a three-dimensional electroanatomical mapping of the right atrium was performed, identifying the presence of a PLSVC (Figures 2B, 2D). The electrical potentials in the superior vena cava (SVC) were regular and slower compared to those in the coronary sinus (CS), indicating passive activation (Figure 2C). In contrast, the PLSVC exhibited prolonged, fragmented, and regular potentials, suggesting it as a driver of the AF (Figure 2E).Ablation was performed at the site of fragmented potentials within the PLSVC, with an ablation index (AI) of 280-300, and the ablation duration was 10 seconds. Complete electrical isolation of the PLSVC successfully terminated the AF (Figures 2F, 2G). Following the procedure, intravenous isoproterenol was administered, and repeated electrophysiological tests with atrial pacing were conducted, none of which could induce any arrhythmias.

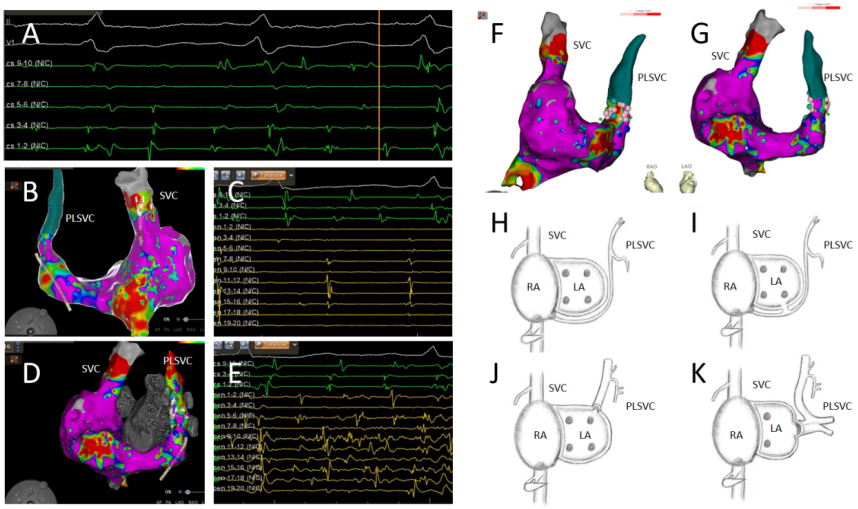

Figure 2:A: Atrial fibrillation (AF) was induced using an S1S1 pacing protocol at 260 ms.

B: A three-dimensional electroanatomical mapping of the right atrium shows the presence of a Persistent Left Superior Vena Cava (PLSVC). The mapping electrode located within the superior vena cava (SVC) recorded potentials in the posterior-anterior view.

C: The mapping electrode (yellow) displays regular electrical potentials in the SVC, which are slower in frequency relative to those in the coronary sinus (CS) (green).

D: The three-dimensional electroanatomical map of the right atrium with the mapping electrode positioned in the PLSVC records rapid and disorganized potentials in the anterior-posterior view.

E: The mapping electrode (yellow) in the PLSVC shows prolonged, fragmented, and regular potentials, which are faster than those in the coronary sinus (green).

F: A three-dimensional model from the right anterior oblique perspective showing the ablation points (pink) at the proximal end of the PLSVC.

G: A three-dimensional model from the left anterior oblique perspective also showing the ablation points (pink) at the proximal end of the PLSVC.

H-K: Three types of PLSVC configurations.

H: The PLSVC drains into the right atrium via the coronary sinus.

I: The PLSVC drains into the left atrium via the coronary sinus.

J: The PLSVC drains directly into the left atrium.

K: The PLSVC drains into the left atrium and is associated with pulmonary vein anomalies.

Outcome and follow-up:

After the ablation, isoproterenol infusion and atrial pacing were used to repeatedly verify that the patient did not experience atrial fibrillation. The patient was then followed up via telephone for six months, with monthly check-ins. The follow-up outcomes were defined as the patient experiencing palpitations accompanied by ECG-confirmed atrial fibrillation, or the presence of atrial fibrillation lasting more than 30 seconds on a 24-hour Holter monitor, indicating a recurrence of atrial fibrillation. Regardless of the presence of palpitations, the patient underwent a 24-hour Holter monitor every three months. During the entire follow-up period, monthly phone calls and Holter monitoring showed no arrhythmias.

Discussion:

AF is the most common cardiac arrhythmia, characterized by rapid and irregular contractions of the atrial myocardial cells. The incidence of AF increases with age and has been rising annually. It often occurs due to spontaneous depolarization of atrial tissue outside the sinoatrial node, primarily due to ectopic activity that commonly originates from the pulmonary veins (95%) and, less frequently, from the inferior and superior vena cava (5%)[1]. Rarely, it arises from abnormal structures such as the Persistent Left Superior Vena Cava (PLSVC)[2,3].

In terms of treatment, rhythm control is preferred for patients with concomitant heart failure or severe symptoms[4,5].

PLSVC is a common congenital venous anomaly, also known as bilateral superior vena cava. Normally, the left superior vena cava regresses to form the ligament of Marshall. However, in approximately 0.3% to 0.5%[6,7] of the population, this regression does not occur, resulting in the formation of PLSVC. Most PLSVC cases drain into the right atrium via the coronary sinus, while in some patients, the right superior vena cava is atretic, and in very rare cases, the PLSVC drains into the left atrium[8] (Figures 2H-K).

Studies have shown that in patients with AF combined with PLSVC, 68.8% of the PLSVC can act as an ectopic trigger or maintenance substrate for AF, necessitating electrical isolation of the left superior vena cava. Additionally, 44.4% of these patients may have other types of arrhythmias, including atrial flutter, AVNRT (Atrioventricular Nodal Reentrant Tachycardia), and junctional tachycardia[8].

The mechanism of arrhythmias in PLSVC is complex and may be related to residual tissues from cardiac development during embryogenesis. During embryonic development, the primitive heart tube exhibits autonomic electrical activity, initially occurring in the sinus horn and main veins. As the heart matures, the pacemaker function gradually shifts to the sinoatrial node in the right heart. However, in some cases, such as with PLSVC, the original pacemaker tissue may not completely regress and remains in the PLSVC. These residual pacemaker tissues can cause electrophysiological abnormalities, particularly at the junction between the PLSVC and the coronary sinus. This structural abnormality can form ectopic trigger points, thereby initiating and maintaining AF. Additionally, overlapping myocardial sleeves in the PLSVC can also be a potential source of abnormal electrical activity and arrhythmias[8,9].

Different scholars have varied strategies for the ablation of PLSVC. Successful catheter ablation has been reported at the proximal, mid, and distal segments of the PLSVC. Regarding ablation power settings, some researchers have used a maximum power control mode of 65°C and 30W with a flow rate of 30 mL/min, while others have suggested using a power of 15-20W for ablation within the PLSVC, with each ablation point limited to a maximum duration of 20 seconds and a flow rate of 17 mL/min, targeting a maximum temperature of 43°C[9]. Additionally, some scholars have employed cryoablation, which has also been successful in eliminating AF[10].

During the procedure, it is crucial to consider the anatomical proximity of surrounding tissues. The left phrenic nerve descends along the anterolateral side of the PLSVC, extending down to the pericardium at the obtuse margin of the left ventricle[11]. Therefore, when performing ablation on the anterolateral side of the PLSVC, phrenic nerve pacing should be employed to avoid nerve damage.

In this case report, the patient was initially admitted with persistent atrial fibrillation. The preoperative echocardiogram showed a left atrial end-systolic diameter (LA) of 50mm and an ejection fraction (EF) of 33%. After nine months of rhythm control to maintain sinus rhythm, a follow-up echocardiogram revealed a reduction in heart size and normalization of the ejection fraction, further confirming that rhythm control is the preferred treatment for AF patients with heart failure to improve cardiac function.

Additionally, atrial substrate mapping identified the PLSVC as the trigger for paroxysmal AF, further confirming that AF can originate from abnormal structures. For ablation, the strategy involved targeting the fragmented potentials at the proximal PLSVC with an AI index of 280-300 and performing a 10-second

ablation. Under the protection of phrenic nerve pacing, electrical isolation of the PLSVC during AF successfully terminated the tachycardia, providing an effective treatment strategy for arrhythmias originating from similar anatomical anomalies.

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