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CASE REPORT

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Complete free wall isolation of arrhythmogenic persistent left superior vena cava

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Abstract

Introduction: Persistent left superior vena cava (PLSVC) is one of the major sources of triggers and drivers of atrial fibrillation (AF). There has been no established PLSVC ablation procedure to eliminate the arrhythmogenicity along the entire length of PLSVC.

Methods and Results: A 70-year-old woman with a history of two previous catheter ablations for AF, mitral valvuloplasty, and an unroofed coronary sinus-type atrial septal defect closure underwent the redo AF ablations. The AF trigger and driver were identified within the patient's enlarged PLSVC. The AF was treated by complete PLSVC free wall isolation.

Conclusion: Complete PLSVC free wall isolation may be an effective ablation method to eliminate the arrhythmogenicity along the entire length of the PLSVC.

KEYWORDS

atrial fibrillation, catheter ablation, persistent left superior vena cava, ultrahigh-resolution mapping system

1 | CASE REPORT

A 70-year-old woman with a history of two previous catheter ablations for atrial fibrillation (AF) underwent the third ablation for recurrent AF. She also underwent surgical mitral valvuloplasty and closure of an unroofed coronary sinus type atrial septal defect via a transseptal approach before the AF ablations. The previous ablation and surgical lesions, including pulmonary vein isolation, left atrial posterior wall isolation, and cavotricuspid isthmus block, are shown in Figure 1A. She also had an enlarged persistent left superior vena cava (PLSVC, Figure 1B). The third session was performed with the Rhythmia HDx™ and IntellaNav StablePoint™ catheter (Boston Scientific). The patient was initially in sinus rhythm at the start of the session. However, an immediate recurrence of AF (IRAF) occurred spontaneously by the firing from the posterior side of the proximal PLSVC free wall (Figure 1C). In addition, the PLSVC showed rapid AF cycle length or fractionated electrograms along its entire length. Therefore, we performed ablation at the trigger site, circumferential ablation of the PLSVC ostium, distal PLSVC isolation, and substrate modification of mid- and proximal PLSVC (Figure 2A,B). Although these ablations successfully suppressed IRAF during the session, AF recurred the next day.

We performed the fourth session 2 months after the third session. At the beginning of the fourth session, the patient presented with biatrial tachycardia (BiAT) around the surgical transseptal lesion and the anterior LA scar (Supporting Information S1: Figure), which was terminated by the ablation in the

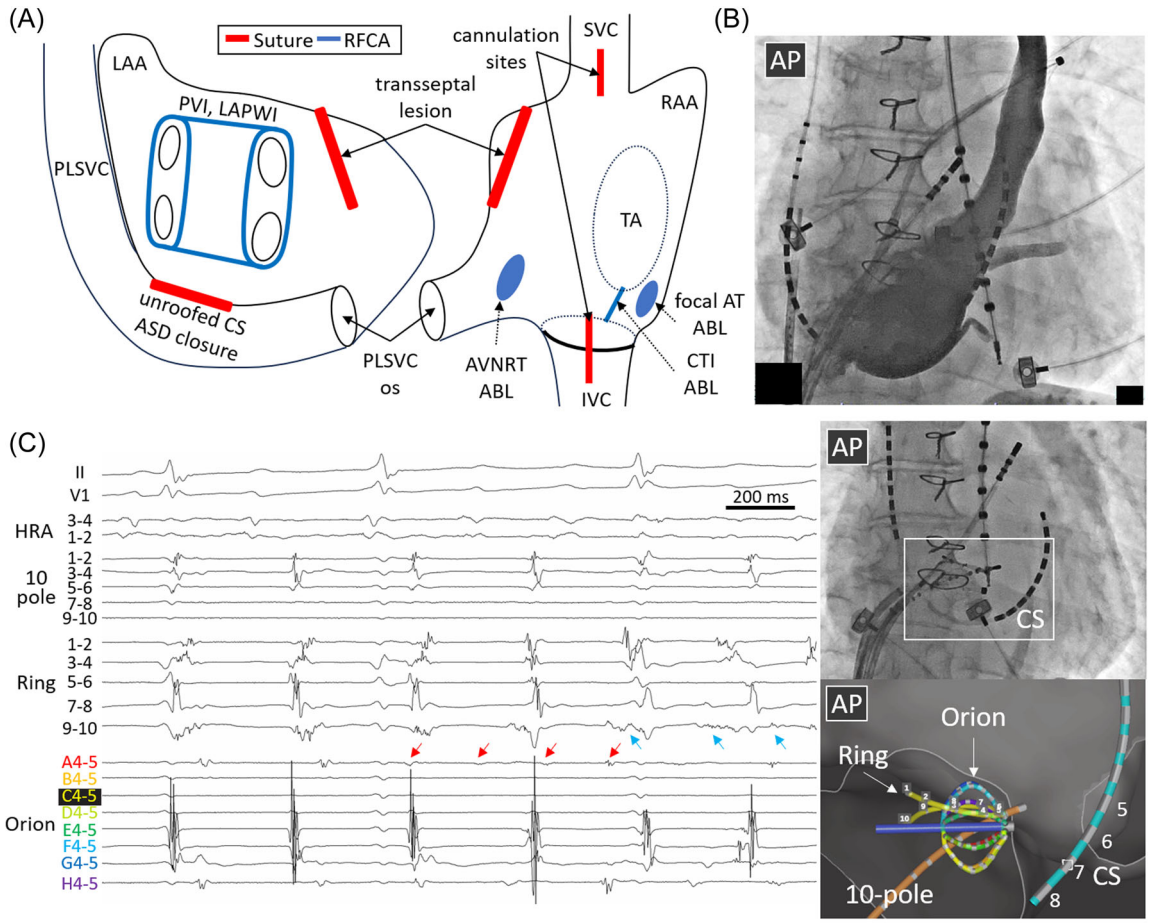

FIGURE 1 (A) A schema of the surgical and ablation lesion sets created before the third session. (B) PLSVC angiography performed during the fifth session. Enlargement of the PLSVC was observed along its entire length. (C) Left panel: AF onset by the firing from the posterior side of the proximal PLSVC free wall. The earliest activation was observed at Orion A4-5 (red arrows). Ring 9-10 also presented fractionated potentials during the AF initiation (blue arrows). Right panels: catheters position during the AF onset on fluoroscopy and the magnified view of the white square area on the mapping system. AF, atrial fibrillation; AP, anteroposterior; ASD, atrial septum defect; AVNRT, atrioventricular nodal reentrant tachycardia; CS, coronary sinus; CTI, cavotricuspid isthmus; HRA, high right atrium; IVC, inferior vena cava; LAA, left atrial appendage; LAPW, left atrial posterior wall; LAPWI, left atrial posterior wall isolation; PLSVC, persistent left superior vena cava; PVI, pulmonary vein isolation; RAA, right atrial appendage; RFCA, radiofrequency catheter ablation; SVC, superior vena cava; TA, tricuspid annulus.

anterior LA scar. Subsequently, we proceeded with PLSVC ablation to eliminate both AF triggers and substrates within the PLSVC. Because LAA activation was maintained via the inferior LA wall at that time due to the severe scarring of the anterior LA wall, we thought that further PLSVC ablation beside the inferior LA wall might pose a risk of LAA isolation. Therefore, we planned a selective isolation of the PLSVC free wall: the lesion set included the distal PLSVC isolation, linear ablations along the superior and inferior edge of the PLSVC-LA junction, vertical lesions adjacent to the esophagus (Figure 2C). Ablation within the PLSVC was performed at 25–30 W power (20 W near the esophagus) for up to 20 s, targeting a local impedance drop of 20 ohms. The ablation near the esophagus was frequently interrupted due to the rise in esophageal temperature. The lesion set successfully isolated the free wall of the PLSVC except for the area against the esophagus (Figure 2D). Although the PLSVC gaps-related AT was still inducible (Supporting Information S1: Video 1), these gaps near the esophagus were challenging to close because of the potential risk of esophageal injury. Therefore, we ended the session without closing the gaps.

Five months after the fourth session, the AT recurred, and we performed the fifth session. Another gap-related AT using the inferior edge gap of the PLSVC free wall isolation and right atrial septum (Supporting Information S1: Video 2) was observed during the session. The AT was terminated by closing the gap, and we subsequently achieved complete PLSVC free wall isolation by ablation as far away from the esophagus as possible (25–30 W power for up to 30 s, targeting a local impedance drop of 20 ohms). Preservation of LAA activation via the inferior LA wall was also confirmed (Figure 3).

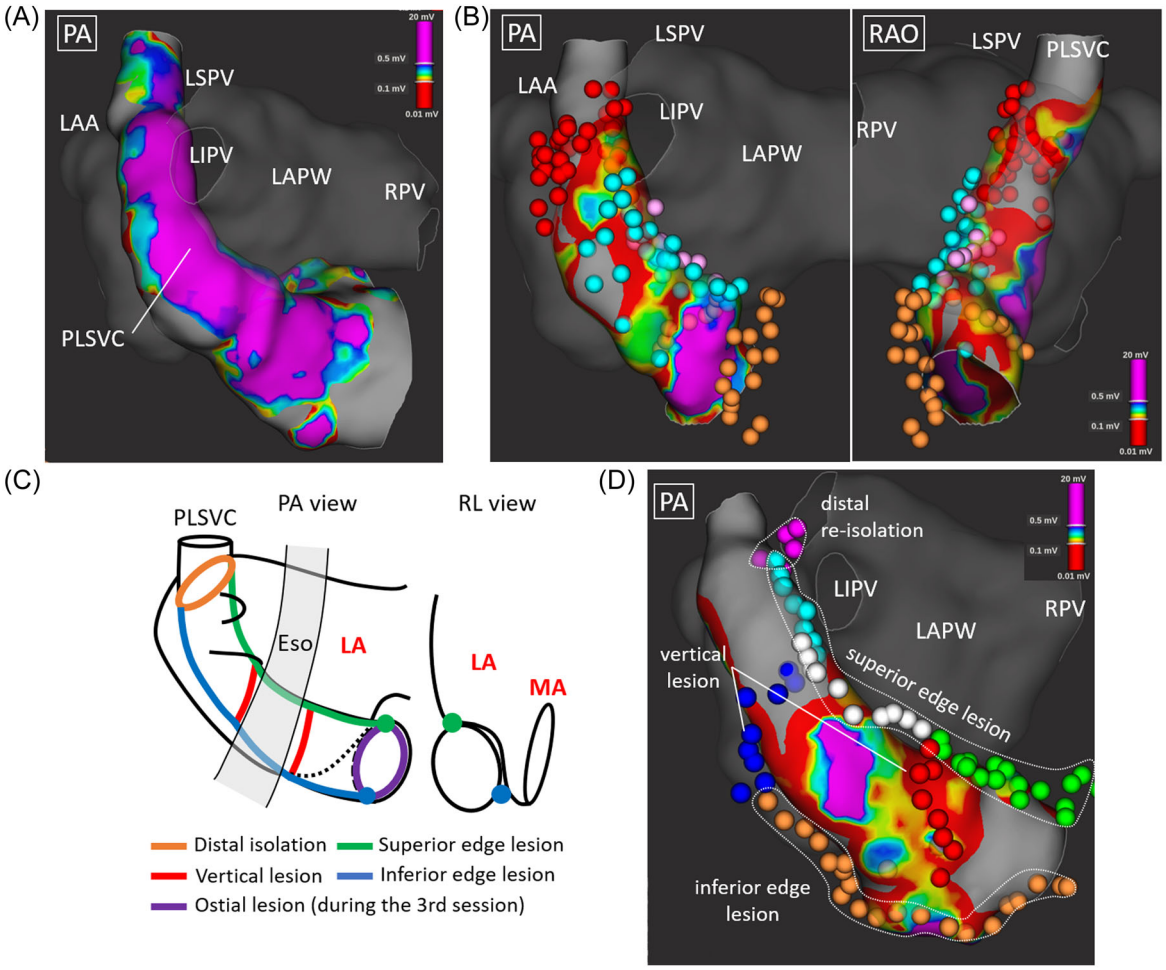

FIGURE 2 (A) The pre-ablation voltage map of the PLSVC in AF rhythm during the third session. (B) The postablation voltage maps of the PLSVC during right atrial pacing. Ablation points are superimposed on the map. Distal PLSVC was isolated, but the mid- and proximal PLSVC were still excitable. (C) A schema of the PLSVC free wall isolation strategy of the fourth session. (D) The postablation voltage map of the PLSVC on right atrial pacing during the fourth session. Ablation points are superimposed on the map. Note that the ostial lesion and the distal portion of the inferior edge lesion were established during the third session. AF, atrial fibrillation; Eso, esophagus; LA, left atrium; LAO, left anterior oblique; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; MA, mitral annulus; PA, posteroanterior; PLSVC, persistent left superior vena cava; RAO, right anterior oblique; RL, right lateral; RPV, right pulmonary vein. Other abbreviations as in Figure 1.

2 | DISCUSSION

PLSVC is a common thoracic venous anomaly occurring in 0.2% of the normal population and 5.9% of the patients with congenital heart disease.[1] PLSVC is one of the significant non-pulmonary vein triggers and drivers in patients with AF,[2] thus eliminating the arrhythmogenic substrate within the PLSVC is sometimes required during AF ablation. However, the strategy of PLSVC ablation has not been fully established. If the AF triggers and drivers are confined within the distal PLSVC, distal PLSVC isolation is an effective strategy.[3] The driver ablation or defragmentation strategy has also been reported.[2,4] The previous investigations on PLSVC ablation are summarized in the Supporting Information S1: Table.

In the present case, the AF trigger was identified at the posterior side of the proximal PLSVC free wall, and the arrhythmogenic substrate was suspected to extend over the entire PLSVC. Therefore, we completely isolated the PLSVC free wall by radiofrequency ablation.

The recently emerging pulsed field ablation (PFA) technology is one of the promising solutions for PLSVC ablation.[5,6] PFA is reported to have little or no possibility of causing phrenic nerve or esophageal injury that can be problematic during radiofrequency ablation.[7] However, PLSVC isolation by PFA potentially ablates adjacent LA myocardium at the same time.[6] This effect is advantageous when planning a mitral isthmus block line but should be avoided from the perspective of preserving the LAA

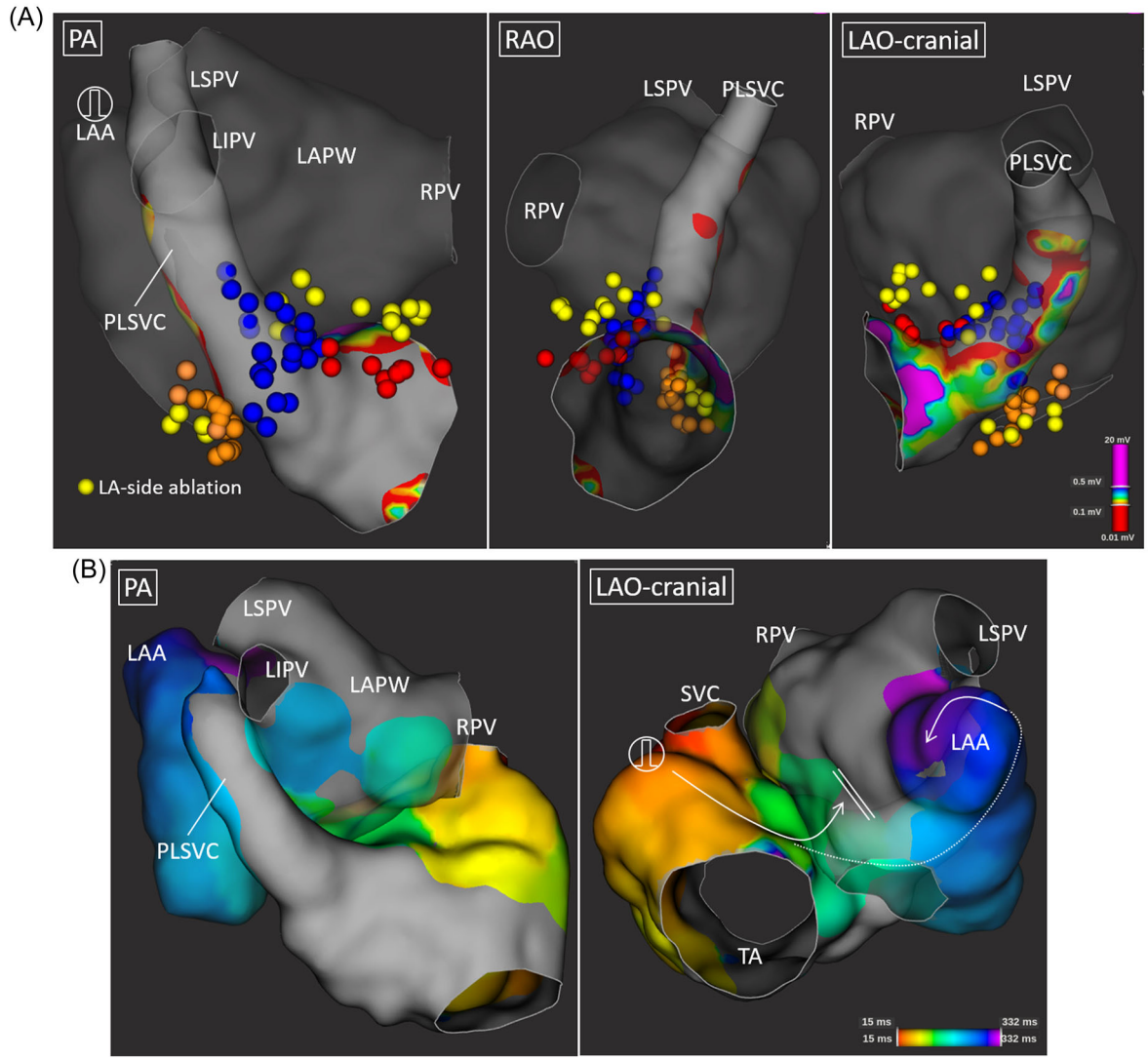

FIGURE 3 (A) The postablation voltage maps during LAA pacing after complete PLSVC free wall isolation. Yellow tags indicate the ablation points from the LA side and the other tags from the PLSVC side. The distal PLSVC and PLSVC free wall became electrically silent (gray-colored area: <0.03 mV local amplitude) along the entire length. (B) The postablation RA and LA activation map during high right atrial pacing. The LAA activation was maintained via the inferior LA wall. LA, left atrium; LAO, left anterior oblique; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; PA, posteroanterior; PLSVC, persistent left superior vena cava; RAO, right anterior oblique; RPV, right pulmonary vein.

activation from the inferior wall. Selective PLSVC free wall isolation may be beneficial in this regard.

In conclusion, we have demonstrated that PLSVC free wall isolation may be an effective ablation method to eliminate the AF substrate within PLSVC, especially when the arrhythmogenic substrates are located on the free wall side or when the preservation of the LAA activation via the inferior LA wall is necessary. As this is the first report describing the PLSVC free wall isolation, further investigation regarding the efficacy and safety should be performed.

AUTHOR CONTRIBUTIONS

Takayuki Sekihara: Conceptualization, methodology, investigation, original draft writing. **Takafumi Oka, Kentaro Ozu, and Yasushi Sakata**: Review and editing.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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