

CASE REPORT

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Vein of Marshall ethanol infusion for recurrent atrial flutter after ablation in a patient with dextrocardia

Zikan Zhong^{1†}, Xiaoyu Wu^{1†}, Jie An², Qitong Zhang¹, Cheng Cheng¹, Yong Wei¹, Songwen Chen¹, Shaowen Liu^{1*} and Genqing Zhou^{1*}

Abstract

Introduction Managing recurrent atrial flutter (AFL) following radiofrequency ablation (RFA) for atrial fibrillation (AF) poses significant clinical challenges.

Methods and results We report an 82-year-old male with mirror-image dextrocardia who developed AFL post-AF ablation. During the redo procedure, vein of Marshall ethanol infusion (VOM-Et) was employed as the initial intervention, successfully terminating the mitral isthmus (MI)-dependent AFL and transitioning into cavotricuspid isthmus (CTI)-dependent AFL during ethanol infusion. Subsequent ablation restored sinus rhythm.

Conclusion Using VOM-EI as the initial intervention in patients with recurrent AFL is a feasible and safe approach, and to our knowledge, this represents the first reported case of VOM-EI in a patient with recurrent AFL in the setting of dextrocardia.

Keywords Recurrent atrial flutter, Vein of Marshall ethanol infusion, Radiofrequency ablation

Case presentation

This 82-year-old male patient was admitted to our hospital one year ago due to “recurrent palpitations and shortness of breath.” He was diagnosed with persistent atrial fibrillation (AF) and underwent radiofrequency ablation (RFA) therapy. Pre-procedural CT imaging confirmed the patient’s mirror-image dextrocardia (Fig. 1A).

Intracardiac ultrasound ruled out intracardiac thrombus and clearly indicated left atrial structural abnormalities, complicating the ablation procedure (Fig. 1B). One year ago, during the initial ablation procedure, pulmonary vein isolation (PVI) was initially performed, followed by voltage mapping revealing extensive low-voltage areas in the anterior, posterior, and septal walls of the left atrium. Linear ablation was then performed in the roof and septal regions of the left atrium. After cardioversion, the patient reverted to sinus rhythm (Fig. 1C).

Three months ago, the patient was readmitted due to “recurrent palpitations and shortness of breath.” The electrocardiogram indicated atrial flutter (AFL), prompting a redo radiofrequency ablation procedure. High-density activation mapping of the left atrium indicated that the pulmonary veins remained electrically isolated, with the posterior wall electrically silent, and AFL’s tachycardia

[†]Zikan Zhong and Xiaoyu Wu are co-first authors.

*Correspondence:

Shaowen Liu
shaowen.liu@sjtu.edu.cn
Genqing Zhou
genqing.zhou@hotmail.com

¹Department of Cardiology, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China

²Department of Cardiology, The Third Affiliated Hospital of Zunyi Medical University (The First People’s Hospital of Zunyi), Guizhou, China



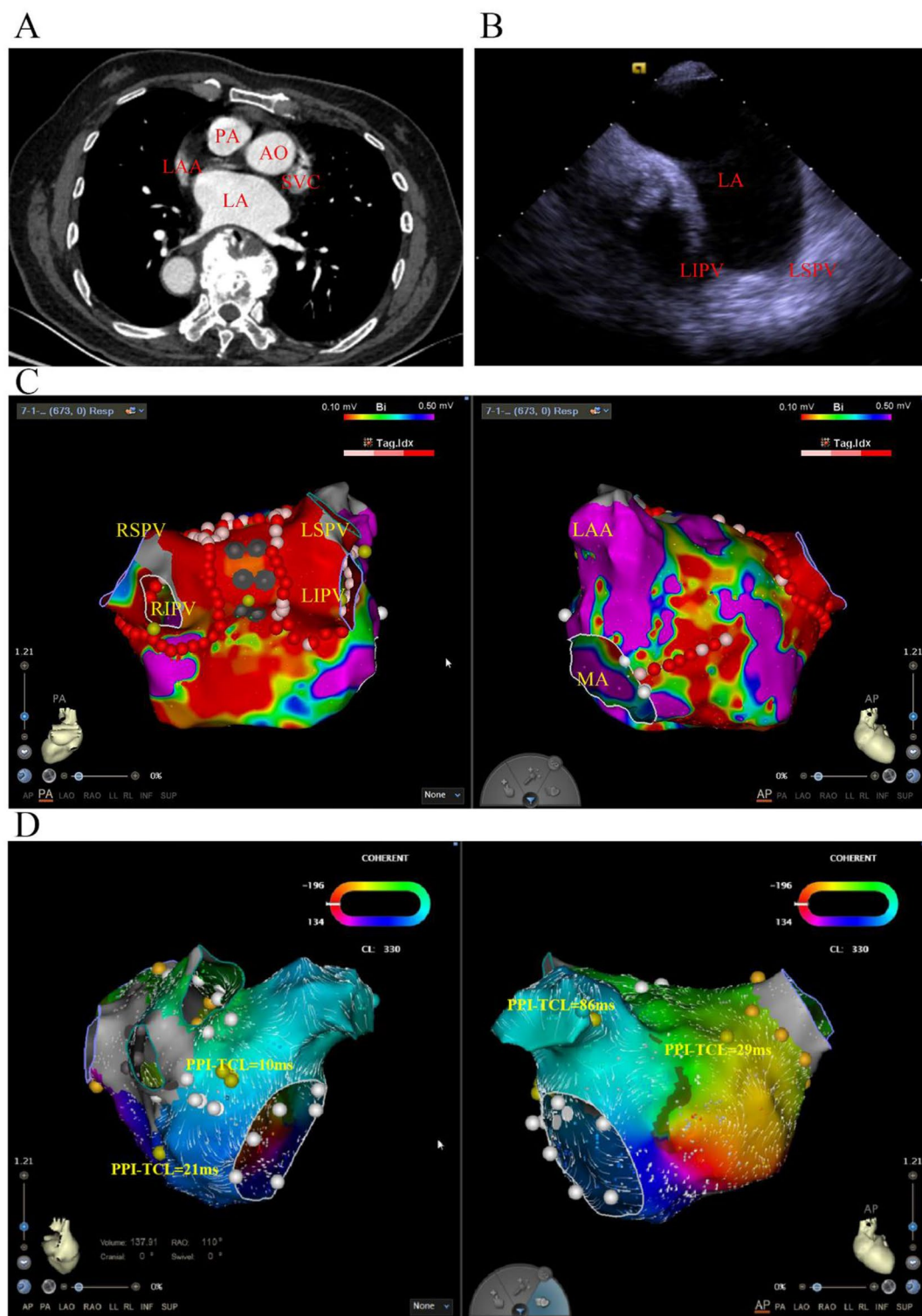


Fig. 1 The Mirror-Image Dextrocardia and Mapping of the Recurrent AFL in left atrium (LA). **(A)** Contrast-enhanced computed tomography examination: Confirmed the patient's rare mirror-image dextrocardia anatomy. **(B)** Intracardiac echocardiography ruled out intracardiac thrombus and revealed severe compression of the left atrium, increasing the difficulty of ablation. **(C)** PVI and additional linear ablation was performed during the index radiofrequency ablation procedure (CARTO). **(D)** Entrainment mapping within the LAA and at three different sites in LA. PA = pulmonary artery, AO = aorta, LA = left atrium, SVC = superior vena cava, LAA = left atrial appendage, LIPV = left inferior pulmonary vein, RIPV = right inferior pulmonary vein, RSPV = right superior pulmonary vein, LSPV = left superior pulmonary vein, MA = mitral annulus

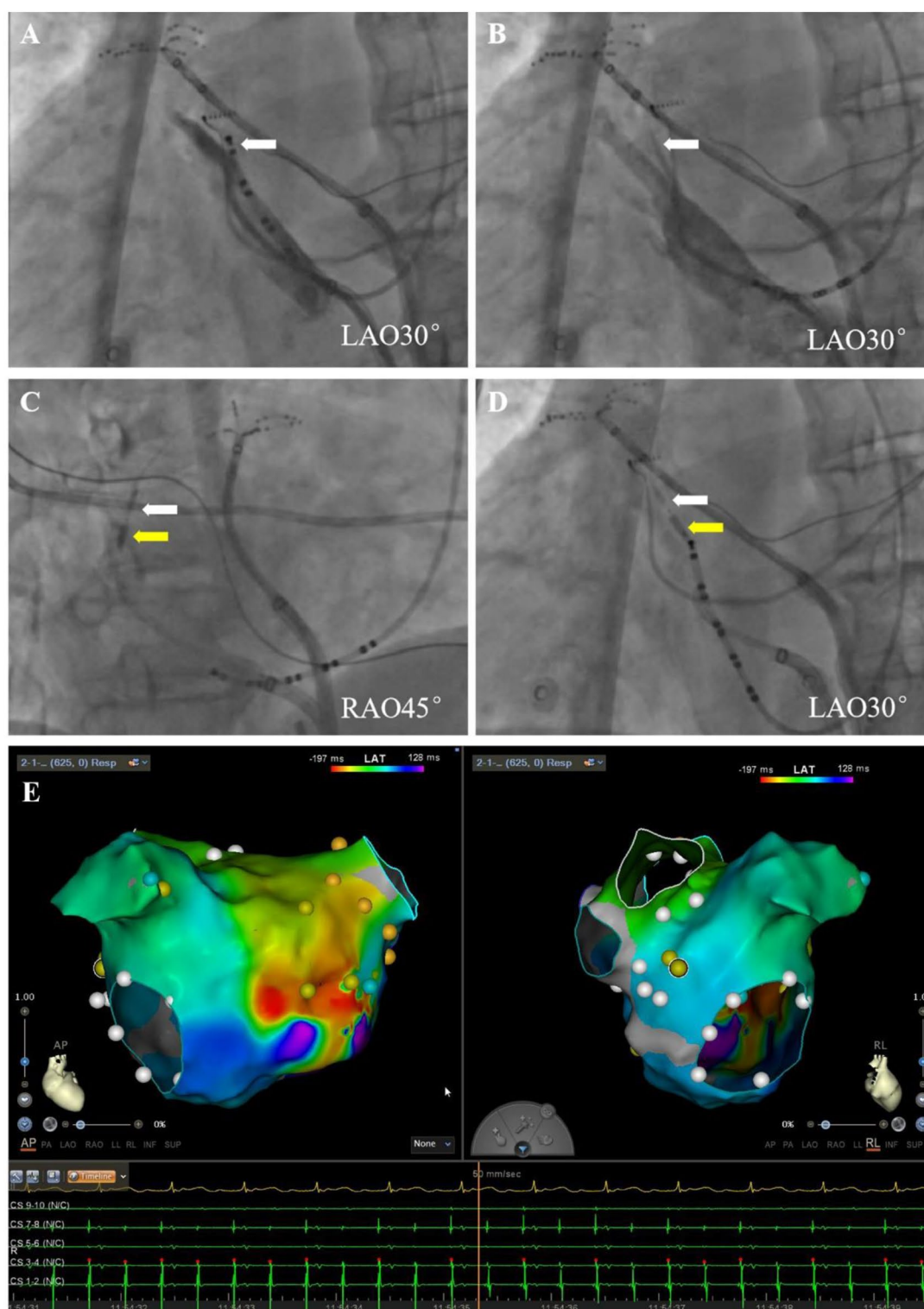


Fig. 2 Vein of Marshall angiography and ethanol ablation. Activation mapping demonstrated the recurrent AFL was MI-related. Considering the patient's suboptimal substrate and the mechanism of AFL, VOM-Et was undertaken as the primary intervention step. **(A):** LAO 30° view, the distal end of the CS electrode (arrow) inserted into the VOM. **(B):** LAO 30° view, angiography (white arrow) of the VOM. **(C):** RAO 45° view, selective angiography of the VOM after proximal balloon occlusion (yellow arrow). **(D):** LAO 30° view, successful VOM ethanol ablation. **(E):** During ethanol infusion, a change in AFL morphology was observed. LAO=left anterior oblique, RAO=right anterior oblique

cycle length (TCL) remained stable without any gaps. Entrainment mapping at specific sites including left atrial

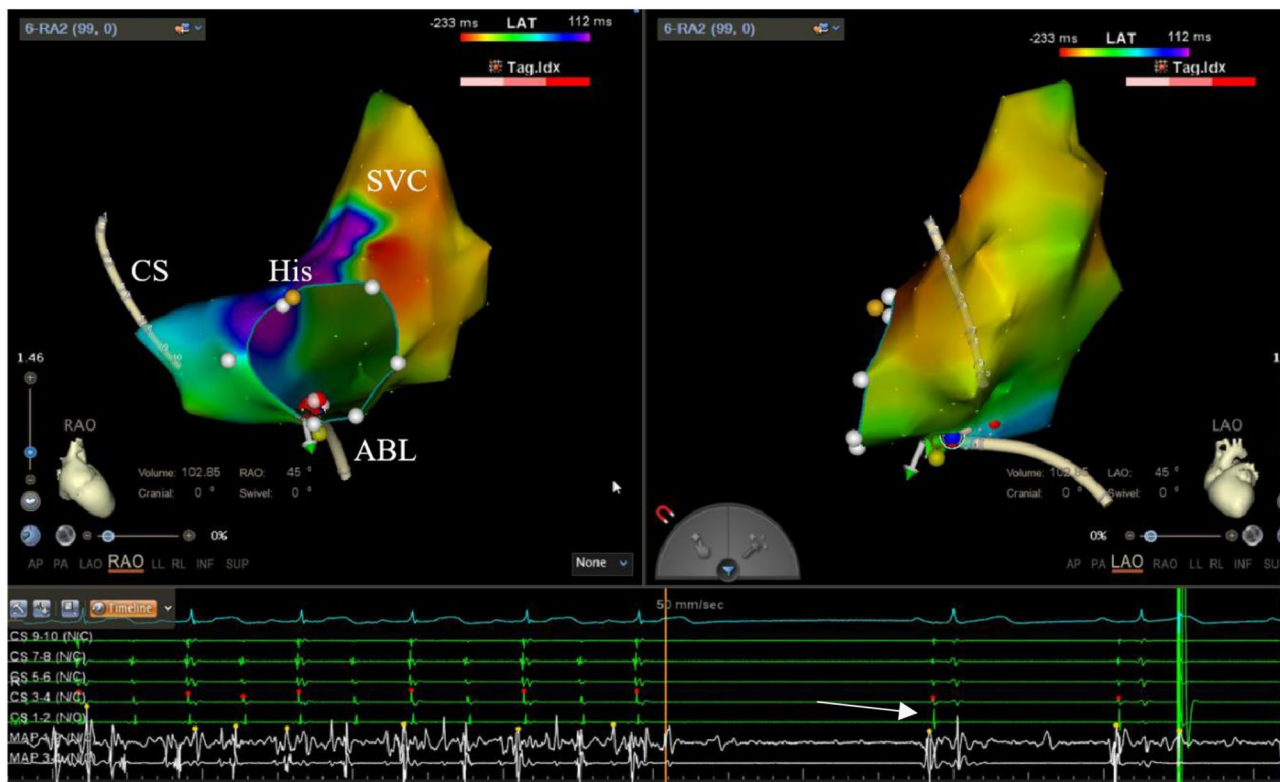


Fig. 3 Mapping and ablation of cavotricuspid isthmus-related AFL. Linear ablation was performed at the tricuspid isthmus, and during the ablation, the altered AFL was converted to sinus rhythm (blue dot), Bidirectional conduction block of the CTI was achieved after CTI linear ablation. His bundle potential (yellow dot), and linear ablation along the CTI by the ablation catheter (red dots)

vestibule, anterior wall, and near the typical mitral isthmus (MI) revealed post-pacing intervals (PPI) minus TCL values of 10 ms, 21 ms, and 29 ms, respectively. Additionally, entrainment mapping in the left atrial appendage (LAA) showed a PPI-TCL of 86 ms, combined with coherent activation mapping suggesting AFL related to the MI (Fig. 1D).

Activation mapping confirmed that the recurrent AFL was MI-related. Considering the patient's suboptimal substrate and the mechanism of AFL, vein of Marshall ethanol infusion (VOM-Et) was employed as the primary intervention (Fig. 2A and D). We slowly infused 3 mL of anhydrous ethanol over approximately 2 min. After the ethanol infusion, we simultaneously injected an appropriate amount of contrast medium to observe any leakage of the contrast agent. This procedure was performed twice, with a total of 6 mL of anhydrous ethanol injected. During ethanol infusion, changes in AFL morphology were observed (Fig. 2E and F). The TCL extended to 346 milliseconds, accompanied by a change in the coronary sinus (CS) activation sequence from CS 1–2 being earliest to CS 7–8 being earliest (video 1). This suggests a potential deviation in the AFL substrate, no longer entirely consistent with the original AFL reentrant circuit.

After the change in AFL morphology, activation mapping was repeated in the left atrium, and entrainment mapping near the anterior wall and MI region showed PPI-TCL values greater than 30 ms, indicating no concealed entrainment. Despite reinforced linear ablation of the MI and septal wall, the altered AFL morphology persisted unchanged, indicating that the altered AFL is not directly associated with the MI. Therefore, our next plan involves activation and entrainment mapping in the right atrium. The STSF catheter was repositioned in the right atrium, where activation mapping was performed. The activation indicated that the altered AFL was clockwise AFL dependent on the cavotricuspid isthmus (CTI), with a complete circuit length TCL of 345 milliseconds (Fig. 3A). Subsequently, linear ablation was performed at the CTI, during which the altered AFL converted to sinus rhythm (blue dot), achieving bidirectional conduction block of the CTI. The STSF catheter returned to the left atrium, and the Pentaray catheter was positioned in the left atrial appendage (LAA). Pentaray pacing in the LAA revealed a conduction sequence from CS 9–10 to CS 1–2. Verification of bidirectional block was then performed by pacing with CS 1–2, CS 3–4, and CS 5–6.

Discussion

Managing recurrent AFL following radiofrequency ablation for AF poses a significant clinical challenge [1]. The vein of Marshall (VOM), recognized as a source of arrhythmia in AF [2], has garnered attention in clinical research, with VOM-Et proposed as a potential effective strategy for treating AF and promoting mitral isthmus ablation [3, 4]. To our knowledge, this is the first reported case of redo RFA in a patient with mirror-image dextrocardia, situs inversus totalis, and recurrent AFL after AF ablation, where VOM ethanol infusion was employed as the initial intervention.

Mirror-image dextrocardia can occur with either normal visceral arrangement or complete mirror imaging of thoracic and abdominal organ arrangements. Performing intracardiac electrophysiology procedures and VOM ethanol infusion in these anatomical variant patients presents significant challenges, especially after the initial pulmonary vein isolation and left atrial linear ablation. Additionally, intraoperative transesophageal echocardiography revealed severe compression of the left atrium, significantly increasing the difficulty of the procedure.

Our observations revealed a unique electrophysiological phenomenon wherein, during ethanol infusion, MI-dependent AFL transitioned into CTI-dependent AFL. The decision to use VOM-EI was based on the patient's complex anatomy and extensive prior scarring, which made traditional methods (such as anterior line ablation) less feasible. Although focal point ablation or linear ablation of critical isthmuses and epicardial ablation are common treatment strategies for recurrent AFL, these methods may be less effective in patients with dextrocardia due to the unique anatomical challenges. In contrast, VOM-EI offers a more targeted approach, which may be more appropriate in such cases. This case highlights the utility of VOM-EI in managing complex arrhythmias and suggests the need for further investigation into its efficacy and safety.

Conclusion

In conclusion, VOM-EI represents a promising adjunctive therapy in managing recurrent AFL after AF ablation, especially in patients with complex anatomical variants. This case demonstrates successful AFL termination using VOM-EI and subsequent CTI ablation, achieving sinus rhythm restoration in a challenging patient population.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12872-025-04716-6>.

Supplementary Material 1: Supplementary data 1 (Video 1). Alterations in AFL Morphology and Characteristics During Ethanol Infusion Ablation.

During ethanol infusion, a change in AFL morphology was observed. The TCL extended to 346 ms, accompanied by a shift in the conduction sequence within the coronary sinus (CS). The CS activation sequence transitioned from CS 1–2 being the earliest to CS 7–8 being the earliest. This suggests a potential deviation in the substrate of AFL, which may no longer be entirely consistent with the reentrant circuit of the original AFL.

Supplementary Material 2: Supplementary data 2. The Bidirectional Conduction Block Verification of MI.

Supplementary Material 3: Supplementary data 3. The Follow-up of the patient.

Supplementary Material 4: Supplementary data 4. The table of Ablation Strategy.

Supplementary Material 5: Supplementary data 5. Check list.

Author contributions

Conceptualization, G.Z. and S.L.; investigation, X.W. and Q.Z.; writing—original draft preparation, C.C., Z.Z. and J.A.; writing—review and editing, J.X, X.L and G.Z.; supervision, Q.Z., Y.W., and S.C.; All authors have read and agreed to the published version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethical approval

Patients have given informed consent for their clinical data to be published in this study.

Consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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