Alcohol ablation of the vein of Marshall in a patient with persistent atrial fibrillation and prior surgical ligation of the ligament of Marshall

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Introduction

The ligament of Marshall (LoM) plays a key role in atrial arrhythmogenesis in patients with atrial fibrillation (AF)[1] and postablation atrial tachycardias (AT).[2,3] Apart from mediating myocardial connections to the left-sided pulmonary veins (PV), and conduction over the epicardial aspect of the (lateral) mitral isthmus, the LoM has also been implicated in the pathogenesis of AF based on an autonomic mechanism. The LoM contains segments of the vein of Marshall (VoM), a myocardial sleeve, ie, the Marshall bundle, and neural (both adrenergic and parasympathetic) elements. Prior to introduction of chemical ablation of the VoM, the (extracardiac) LoM was interrupted during surgical ablation of AF.[4,5] We present a case during which a patient underwent successful alcohol ablation of the VoM despite prior surgical interruption of the LoM.

KEY TEACHING POINTS

- Chemical ablation of the vein of Marshall (VoM) is effective in the treatment of epicardial reentrant circuits that arise during the course of catheter and surgical ablation of atrial fibrillation (AF).
- Prior surgical interruption of the ligament of Marshall (and lack of an obvious venous target on venography) should not preclude mapping of the VoM.
- The VoM oftentimes represents a critical target for elimination of difficult cases of post-AF tachycardias.

Case report

A 58-year-old male patient with a history of persistent AF and diastolic heart failure was referred for catheter ablation of recurrent AF and organized AT. He had undergone several prior catheter and surgical procedures at another facility. Targets during the former included the PVs as well as linear ablation at the left atrial (LA) roof, and the posterior left atrium. For recurrent AF and AT, he then underwent a minimally invasive surgical ablation. This involved LA appendage (LAA) ligation, placement of anterior and lateral mitral-isthmus lines, and surgical ligation of the LoM. The procedure was described as follows: "The pericardium was entered inferior to the phrenic nerve with a pair of thoracoscopic scissors. The incision was then carried superiorly using a Ligasure (vessel sealing system [Medtronic, Minneapolis, MN]). The phrenic [nerve] was visualized and preserved throughout. The LoM was then ligated using a Ligasure."

The patient was not felt to be a good candidate for antiarrhythmic therapy for recurrent atrial arrhythmias given his history of genotype-confirmed long QT syndrome, structural heart disease, and renal dysfunction, and therefore presented to our institution for another attempt at catheter ablation.

Electrophysiological study

An electrophysiological study was performed under general anesthesia. He presented to the electrophysiology laboratory in AF that spontaneously terminated, and then reinitiated. After transseptal puncture, heparin was administered, and an activated clotting time of 350 seconds was targeted. A ring catheter (Lasso; Biosense Webster, Inc., Diamond Bar, CA) was then used for creation of the LA geometry using a 3D electroanatomic mapping system (CARTO 3; Biosense Webster, Inc). Severe stenosis of the left superior PV was noted. Mapping revealed that the PVs remained isolated, except for a few antral areas below the left inferior and anterior to the right superior PV. There was recovery of the posterior left atrium. AF persisted despite radiofrequency (RF)

KEYWORDS Atrial fibrillation; Atrial tachycardia; Atrial flutter; Vein of Marshall; Alcohol ablation
(Heart Rhythm Case Reports 2024;10:151–154)

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https://doi.org/10.1016/j.hrcr.2023.11.008

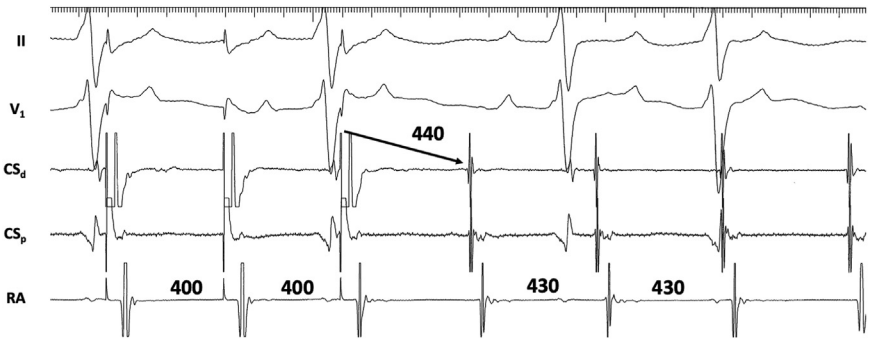

Figure 1 Entrainment mapping from the mid coronary sinus (CS) shows an in-circuit response.

ablation (ThermoCool SmartTouch STSF; Biosense Webster, Inc.) of these areas. Given prior mitral isthmus ablation, RF ablation (30 W, 20 seconds per lesion) was also performed at the lateral and posterolateral mitral isthmus targeting residual electrograms.

The cuff of tissue below the level of LAA ligation was then targeted, which terminated AF to an AT at a cycle length of 270 ms. Further ablation in this region terminated the AT, electrically isolated the stump of the LAA, and yielded another AT at a cycle length of 430 ms.

Owing to extensive scarring, high-density activation mapping was not feasible. Similarly, entrainment mapping could only be performed at a limited number of sites, and the results were compatible with perimitral reentry (Figure 1). Lack of voltage at the lateral, anterolateral, and anterior mitral annulus prompted epicardial mapping in the coronary sinus (CS).[6] Given tortuosity, it was difficult to advance the ablation catheter into the distal portion of the CS, from both the superior (internal jugular) and inferior (femoral) approaches. After multiple attempts, the ablation catheter was negotiated into the distal CS through a deflectable sheath. RF ablation (20 W) in the distal CS was also unsuccessful.

Alcohol ablation of the vein of Marshall

Despite prior surgical interruption of the LoM, CS venography was performed to determine the presence of the VoM, since the tachycardia could not be terminated with RF ablation alone. Venography did not disclose an obvious VoM, but it did reveal the location of the valve of Vieussens (Figure 2A). Using this landmark, the putative VoM was probed with an angioplasty wire (Prowater; Asahi, Irvine, CA). Selective venography through a 1.5 mm $\times$ 8 mm balloon (Boston Scientific, Marlborough, MA) confirmed the presence of the VoM (Figure 2B). After confirming occlusion (4 atmospheres), 4.75 cc of $\geq$99% dehydrated ethyl alcohol (Ablysinol; BPI Labs, LLC, Freehold, NJ) were slowly infused into the vessel, with immediate termination of the AT (Figure 2C). Because of complete absence of atrial electrograms at the lateral left atrium/LAA, conventional pacing maneuvers to confirm conduction block across the mitral isthmus could not be performed. But these observations in and of themselves, along with immediate response to chemical ablation of the VoM, should be reassuring. There were no arrhythmias inducible despite rapid atrial pacing at 180 ms during isoproterenol infusion (10 mcg/min). After 6 months following the ablation procedure, the patient remains free of arrhythmias in the absence of antiarrhythmic medications.

Discussion

To our knowledge, this is the first described case of alcohol ablation of the VoM in a patient who had previously undergone surgical ligation of the LoM. This case confirms the contribution of the LoM in mediating post-AF AT. It would have been very difficult to eliminate this arrhythmia without chemical ablation of the VoM. Surgical ablation might be considered to be the gold standard, since atrial myocardium can be visualized directly and is thought to be more likely to result in durable transmural lesions.[7] However, recurrent PV connections and recurrent AF and AT are well documented after surgical, as well as catheter-based, procedures.[8] Thus, it is important to verify the integrity of prior lesion sets to ensure completeness. For example, had the LoM been assumed to have been successfully ablated merely on the basis that it had been targeted during arrhythmia surgery, a successful outcome at the current procedure would have been unlikely. It should also be noted that during cardiac surgery, the extracardiac portion of the LoM is typically targeted, potentially leaving the patient vulnerable to the remaining potentially arrhythmogenic intracardiac components, specifically the myocardium drained by the VoM (Figure 3).[4]

One of the challenges encountered in this procedure was that the venous target could not be demonstrated by CS venography. In a large cohort study, nonidentification of the VoM occurred in about 6.2% of patients.[9] Of note, prior CS ablation was the most common predictor for failure of cannulation in that study. The valve of Vieussens at the junction of the CS and the great cardiac vein can be used as an anatomic landmark, in the vicinity of which venous cannulation with a soft-tipped angioplasty wire may be attempted. Although preprocedural imaging may be used to determine the presence and location of the VoM, its absence on such studies does not preclude successful cannulation and chemical ablation.[10]

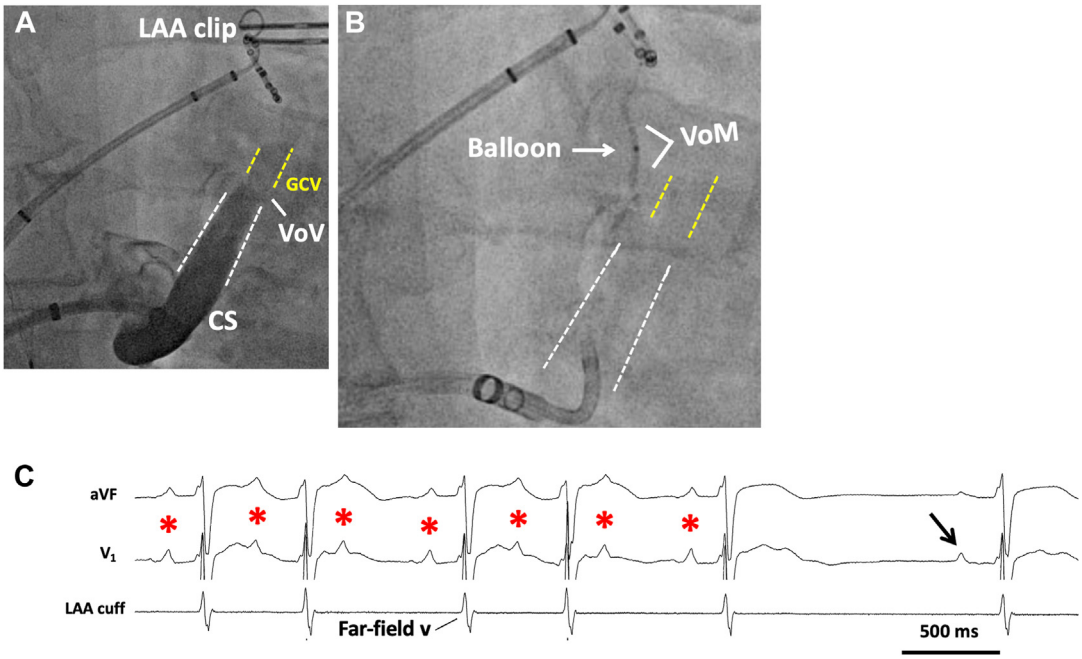

Figure 2 **A:** Venography of the coronary sinus (CS) did not reveal a vein of Marshall (VoM) but showed the valve of Vieussens (VoV), which was subsequently used as an anatomical landmark. The CS is depicted by the white dashed lines while the great cardiac vein (GCV) by yellow dashed lines. **B:** Selective balloon-occlusion venography demonstrates the VoM and the related branches. **C:** Termination of the atrial tachycardia during injection of ethanol into the VoM. The black arrow depicts the P wave at the time of restoration of sinus rhythm. LAA = left atrial appendage.

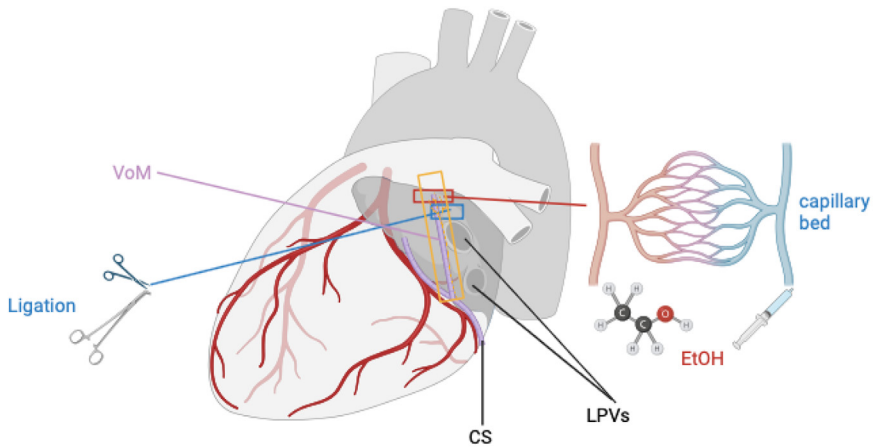

Figure 3 A schematic highlighting the difference between surgical ligation of the extracardiac components of the ligament of Marshall (LoM) and chemical ablation of the vein of Marshall (VoM) targeting the intracardiac components. The orange rectangle depicts the Marshall bundle. The blue box shows the level at which surgical ligation of the LoM is performed. The red box depicts a portion of the lateral left atrium that is drained by the VoM, the target of chemical ablation. CS = coronary sinus; LPVs = left-sided pulmonary veins.

We should also note that definitive diagnosis of perimitral reentry was not feasible owing to areas of scar at the lateral and posterolateral mitral isthmus and the region of the LAA. However, entrainment mapping from the CS and the immediate response to ethanol infusion provide *circumstantial* evidence of macroreentry—more specifically, perimitral reentry. If the AT were due to a focal/centrifugal mechanism involving the left lateral ridge or Marshall bundle, entrainment mapping from the mid CS, an area relatively remote from the distal aspect of the VoM and ridge, would have yielded a longer postpacing interval. It may also be difficult to reconcile the presence of classic perimitral reentry with LAA disconnection. However, not uncommonly, one encounters an "oblique" variant of mitral isthmus–dependent atrial flutter that still uses the inferior and lateral annular tissue but not the anterior portion, ie, the myocardium between the base of the LAA and the anterior annulus.[11,12]

Conclusion

Chemical ablation of the VoM is effective in the treatment of epicardial reentrant circuits that arise during the course of catheter and surgical ablation of AF. A prior history of

surgical interruption of the LoM (and lack of an obvious venous target on venography) should not preclude mapping of the VoM, which may be critical in eliminating challenging post-AF tachycardias.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: None.

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