Vein of Marshall partially isolated with radiofrequency ablation from the endocardium

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Introduction

The vein of Marshall (VOM) is widely known as a pathogenesis of atrial fibrillation (AF). To eliminate the triggers of AF, the Marshall potential can be ablated both from the endocardium with radiofrequency ablation (RFA)[1] and from the epicardium with ethanol injection.[2] This is a case report of successful catheter ablation from the endocardium to visualize the electrically isolated area of the VOM.

Case report

A 64-year-old man with dyspnea on effort and nonvalvular AF ($CHADS_2$-VASc = 0, LA = 42 mm) persisting for 8 years was referred for RFA. The posterior left atrial wall and pulmonary veins were simultaneously isolated by a single ring lesion. A lateral mitral isthmus linear ablation, both from the endocardium and in the coronary sinus (CS), and a cavotricuspid isthmus linear ablation were performed and complete conduction block was obtained at each line.

Within 3 months after the procedure, the patient began to suffer from symptomatic atrial tachycardia (AT). External electrical cardioversion under bepridil 100 mg twice daily terminated the AT and sinus rhythm (SR) recovered. However, soon AF developed and became sustained, which necessitated a second procedure 9 months after the first procedure.

Decapolar and tetrapolar electrode catheters (Inquiry; St. Jude Medical, St. Paul, MN) were positioned in the CS and right ventricular apex. A circumferential 20-pole ring catheter (Lasso 2515 NAV eco variable catheter; Biosense Webster, Diamond Bar, CA) was inserted into the left atrium (LA) via an SL1 long sheath.

Isolation of the bilateral pulmonary veins and posterior LA wall was confirmed by the ring catheter. The complex fractionated atrial electrograms (CFAE) were mapped using the ring catheter. Radiofrequency (RF) energy was delivered with an open irrigated 7 F, 3.5-mm-tip, deflectable ablation catheter (Thermocool Navistar; Biosense Webster) for the CFAE area in the LA for 21 minutes, which failed to terminate the AF or even convert it to AT. In the CS electrograms, the mid portion of the CS was sometimes preceded by the distal CS and proximal CS that was presumed to be the proximal region of the VOM. Hence, precise mapping of the VOM was attempted.

A 6 F multipurpose catheter was inserted into the CS via another SL1 sheath introduced from the right femoral vein and was engaged into the ostium of the VOM. After venography of the VOM was obtained, a 2 F octapolar catheter (EPstar Fixed; Japan Lifeline, Tokyo, Japan) was advanced into the VOM. In the VOM electrograms, without changing the catheter position, fractionated potentials and ectopic activity from the distal or middle portion of the VOM were detected (Figure 1A and B), exhibiting a shorter cycle length (CL) compared with that of the left atrial appendage (LAA) and CS. We began to ablate the VOM from the endocardium, targeting the 2 F catheter in the VOM, not with electrocardiographic guidance but with fluoroscopic guidance, because the VOM electrograms could hardly be recorded from the endocardium.

Although the main activation sequence of the VOM was from distal to proximal, some reflecting potentials were seen from VOM 3–4 to 1–2 and the duration of the local potentials of VOM 4–5 and 5–6 were long. These findings implicated that VOM 4–5, 5–6 could be a pivoting point. Moreover, we occasionally could see tiny preceding potentials in VOM 4–5 and 5–6 (Figure 2A, red arrow). First, ablation from the endocardium in the middle of the VOM (VOM4) dissociated the activation between the distal (VOM1–4) and proximal (VOM4–8) VOM (Figure 2B). Next, ablation at a more distal position (VOM2–3) eliminated activation of VOM2–3/3–4, but residual ectopic activities were still recorded at VOM1–2 (Figure 2C). Finally, ablation at VOM1 eliminated all activation of distal VOM, that was partial isolation of VOM electrically was accomplished (Figure 2D). The CL of the average 10 consecutive beats at the LAA were 174.6 ± 8.2 ms before VOM ablation,

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KEY TEACHING POINTS

- Ectopic activities can arise from the Marshall bundle, which can trigger atrial fibrillation (AF) in some patients. Isolation of the vein of Marshall should be useful method to cure AF.
- The Marshall bundle exists at the left atrial epicardium. From the endocardium, the local electrograms of the Marshall bundle are hardly recorded, but they can be recorded by the thin electrode catheter inserted into the Marshall vein.
- The Marshall bundle can be ablated from the endocardium by targeting anatomically the thin electrode catheter inserted in the vein of Marshall.

which was prolonged to 182.0 ± 10.3 ms after VOM ablation.

There was still ectopic activity from the proximal VOM, and ethanol injection was needed to achieve additional isolation of VOM. The 2 F catheter was removed and 4 separate slow injections of 1 mL 98% ethanol were administered from distal to proximal VOM via a balloon catheter (Apex; Boston Scientific, Marlborough, MA) for percutaneous catheter intervention, which failed to terminate the AF. However, the CL of the LAA was prolonged to 196.8 ± 6.5 ms after the ethanol injection to the VOM. The reinsertion of the 2 F catheter failed owing to a VOM obstruction caused by ethanol. Internal cardioversion terminated the AF and SR resumed. Only nonsustained AT could be induced by atrial burst pacing even under a 15 μg/min infusion of isoproterenol. During a follow-up of 1 year, SR was maintained despite a dose reduction of the bepridil to 50 mg twice daily, which demonstrated the reduction of AF recurrence after catheter ablation for persistent AF.[3]

Discussion

Histologically, the extracardiac ligament of Marshall contains portions of the intracardiac VOM and surrounding myocardial sleeve, the Marshall bundle (MB).[1,4,5] The MB connects the atrial muscle and CS muscle sleeve, which is considered to be important as a source of triggers and drivers of AF.[1,6,7] RF applications to the MB are reported to be useful in controlling atrial arrhythmias.[8] The same author reported the onset of rapid activation of the MB recorded in the VOM, and an RF delivery from the endocardium eliminated all tachyarrhythmias without any pulmonary vein isolation.[1]

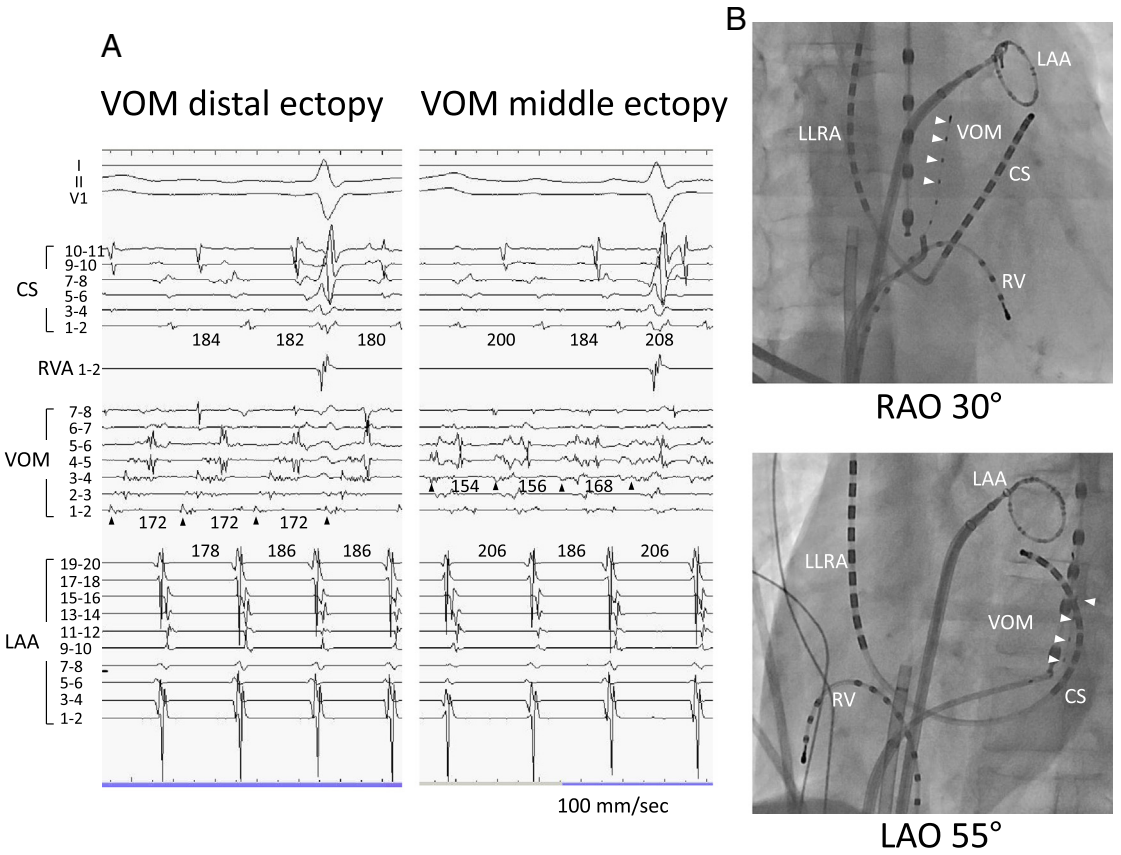

Figure 1 **A:** An ectopic activity from the distal or middle part of the vein of Marshall (VOM) was recorded during atrial fibrillation. The cycle length of coronary sinus (CS) and left atrial appendage (LAA) was longer than the VOM. CS and LAA were passively activated. **B:** Catheter position (RAO 30 degrees/LAO 55 degrees). A circumferential 20-pole catheter was located in the LAA. A 2 F catheter was inserted into the VOM. LAO = left anterior oblique; LLRA = lower lateral right atrium; RAO = right anterior oblique; RVA = right ventricular apex.

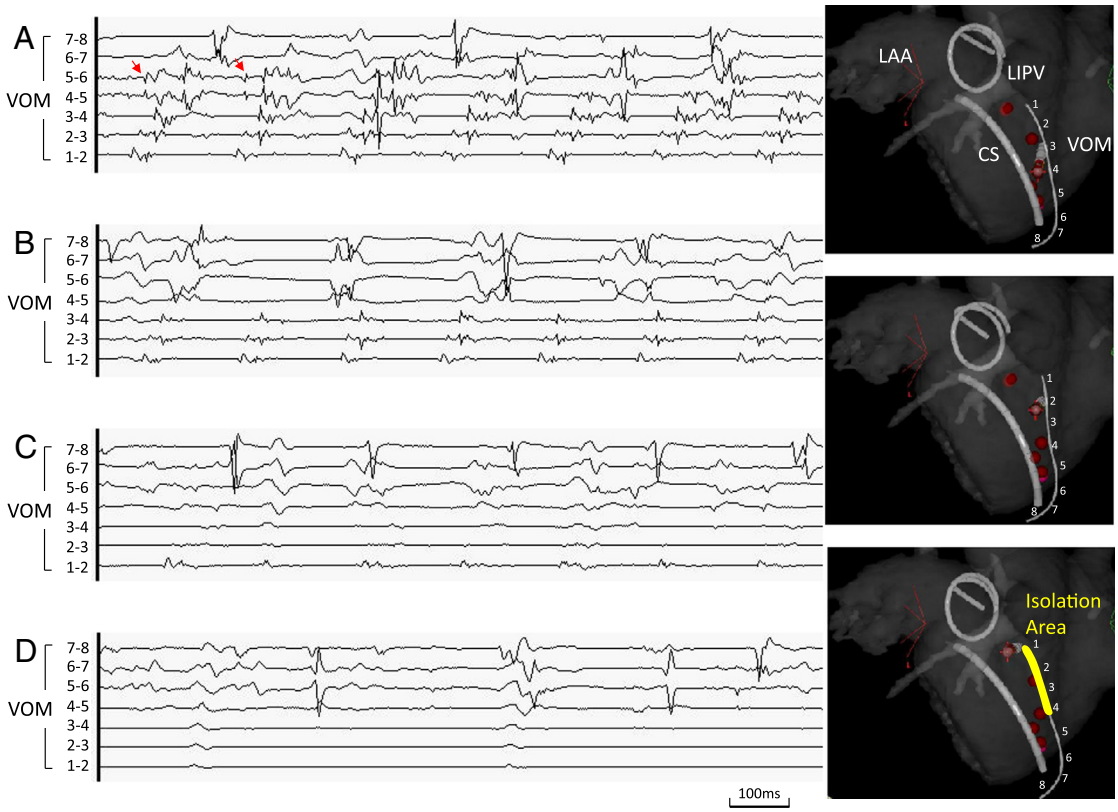

Figure 2 **A:** Before ablation. An ectopic activity was recorded in the distal vein of Marshall (VOM). A fractionated electrogram was recorded in the middle of the VOM. **B:** Ablation near electrode VOM4 resulted in dissociation of the electrical activity from the distal and the proximal segments of the VOM. **C:** Ablation at VOM2–3 eliminated activation at VOM2–3/3–4. An ectopic activity from the distal VOM continued. **D:** Ablation at VOM1 eliminated all activation of distal VOM.

Regarding the arrhythmogenicity of the LAA, it is reported that 27% of patients with AF have triggers from the LAA.[9] However, the VOM electrograms were not investigated in that study, and triggers from the VOM might be included in that population.

We demonstrated the partial electrical isolation of the VOM by RF applications from the endocardium. Valderrábano et al[2] showed that injection of echocardiac contrast in the VOM demonstrated earliest contrast appearance in the LA adjacent to the left pulmonary vein, and that ethanol infusion in the VOM generated a low-voltage area on the posterior atrial wall and anterior aspect of the left pulmonary vein. Kim et al[10] reported that 1 or more myocardial tracts insert directly into the left atrial free wall and CS from the VOM. These studies indicated that connection sites between the VOM and LA could be the target to cure atrial arrhythmia. In our case, the most distal part of the VOM was considered to be the focus of the ectopic activity and the proximal part of the VOM connected to the CS musculature. Ablation of the middle and distal parts of the VOM interrupted the electrical connection and the distal half of the VOM became electrically isolated. The ablation sites were thought to be the insertion sites between the MB and epicardial aspect of the LA because electrograms could not be recorded from the endocardium. Hwang et al[11] reported that in $<4\%$ of the patients, an endocardial ablation alone could not eliminate all connecting fibers. In our case, AF persisting for 8 years was successfully treated and SR was maintained for 1 year after the second ablation. Although CFAE ablation was appended in the second session, the CL of the LAA was prolonged by the intervention on the VOM, which could have contributed some antiarrhythmic effect in this patient.

Conclusion

The VOM is recognized as one of the non–pulmonary vein foci. As the VOM is located epicardially, an ethanol injection is a useful method to cure VOM-origin AF. The potentials from the VOM could barely be recorded from the endocardium. Hence, when the VOM is targeted for RF ablation, the insertion of an electrode catheter into the VOM could function as an anatomic indicator of the target site, which could also tell us when the electrical isolation of the VOM is achieved, as a possible endpoint of the procedure.

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