Unrecognized left atrial activation patterns of Marshall bundle–related atrial tachycardia following atrial fibrillation ablation

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Introduction
Atrial tachycardia (AT) commonly occurs after pulmonary vein isolation (PVI) for atrial fibrillation (AF) treatment. The ligament of Marshall (LOM) reportedly has a critical role as a mechanism of AT after PVI.1 We identified 2 cases of unrecognized left atrial activation patterns of LOM-related AT after PVI that may be misleading and prevent us from achieving correct diagnosis and treatment.

Case report
Case 1
A 70-year-old man was admitted to our institution to undergo catheter ablation for persistent AT (AT1; tachycardia cycle length [TCL] = 216 ms) following PVI, which was performed 3 months earlier. The coronary sinus (CS) activation exhibited a chevron pattern (Figure 1C), and the local activation time map on the 3D electroanatomical mapping system (CARTO3; Biosense-Webster, Inc, Diamond Bar, CA) represented a centrifugal but “broad breakout” activation pattern arising from the posterior wall, though the local activation time was apparently late compared with the P wave (Figure 1A, Supplementary Movie, and Supplementary Figures 1 and 2). Thus presystolic activation should have been missed in the left atrium (LA). With the detailed mapping around the left PVI circle using the Pentaray multispline catheter (Biosense-Webster, Inc), we identified a high-frequency, long-duration amplitude multicomponent electrogram (EGM) that preceded by 31 ms from the P-wave onset and that covered 144 ms (67% of TCL) between the mid-diastole and the systole at the left lateral ridge (fractionated potential [FP] 1, Figure 1A and B), though no EGM was detected on the line or within the circle of the left PVI at all. Entrainment study at the FP1 recording site (Figure 1C) demonstrated a concealed entrainment of AT1 with a little shortening of the postpacing interval (203 ms) as compared to AT1 cycle length, presumably because the critical isthmus was extensively captured by the pacing. Based on these findings, we recognized that AT1 was the localized reentrant AT arising from the LOM, and speculated that some epicardial connections play a role in conducting the local activation at the LOM directly to the LA posterior wall. AT1 was successfully eliminated by radiofrequency application (35 W, 18 s) at the FP1 recording site (Supplementary Figure 3). No AF/AT recurrence was observed after the procedure.

KEY TEACHING POINTS
- The Marshall bundle (MB) is related to the source of atrial tachycardia (AT) following atrial fibrillation ablation besides macroreentrant AT or gap-related AT.
- In this type of AT, a centrifugal but broad breakout pattern may emerge at unexpectedly distant sites from the true focus at the left lateral ridge, such as the posterior wall of the left atrium, possibly through the epicardial conduction.
- Conventional analysis of the local electrogram is the key to reach the correct diagnosis (“the earliest site is not early”). Detailed endocardial mapping of the left lateral ridge as the interface of the epicardium and the endocardium with a multispline mapping catheter can find the characteristic MB mid-diastolic electrogram (high-frequency, long-duration amplitude multicomponent) at which the ablation is effective.

KEYWORDS
Atrial tachycardia; Catheter ablation; Electroanatomical mapping; Marshall bundle; Epicardial connections

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Case 2

A 50-year-old woman with complaints of frequent palpitations 2 months after the PVI underwent a repeat procedure. In this second procedure, mitral isthmus linear ablation was successfully performed for inducible mitral annular flutter. Subsequently, burst pacing from the CS electrodes induced the AT2 (TCL = 366 ms), which demonstrated the same broad breakout patterns arising from the posterior wall as the AT1 in case 1 on the CARTO map (Figure 2). The local activation of the “earliest site” at the LA posterior wall was late compared with the P-wave onset. On the left lateral ridge, a fragmented mid-diastolic EGM component was observed (FP2; Figure 2A). Furthermore, no EGM could be tracked within the PVI line. As in case 1, we speculated that AT2 activation was propagated through the LOM–LA posterior wall connections. Although entrainment pacing could not be evaluated because the local activation could not be captured, AT2 was terminated and eliminated by radiofrequency application (40 W, 10 s) at the FP2 recording site on the left lateral ridge. No arrhythmia recurrence was reported after the procedure.

Discussion

The LOM is an epicardial vestigial fold that contains the vein of Marshall; myocardial sleeve, which is described as the Marshall bundle (MB); and autonomic nerves. The LOM located in the epicardial aspect of the left lateral ridge is important as the focus of AF because it demonstrates a rapid electrical activity2 and particularly, the MB plays a critical role in conducting the electrical activities through various connections to the adjacent myocardium. Patients who have more than 2 connections between the MB and the myocardium of the CS, LA, or pulmonary veins possess the anatomical substrate to generate localized reentry circuits or macroreentrant ATs around the mitral isthmus using the epicardial MB.3

Vlachos and colleagues1 reported that the MB-related AT of post-AF ablation accounted for approximately 30%.
However, determining the MB involvement in a post–AF ablation AT may be difficult because the MB is electrically protected and the reentrant circuit may be contained within a small area; in addition, previous ablation procedures, such as PVI or mitral isthmus linear ablation, may make the MB potential more difficult to distinguish. The MB may also have an epicardial-to-endocardial breakthrough that prevents tracking presystolic activations from the endocardial side.

In our case series of ATs following AF ablation, unusual broad breakout patterns arising from the left posterior wall were detected by activation mapping. In both case 1 and case 2, the presystolic or mid-diastolic multicomponent EGM at the left lateral ridge (FP1 and FP2) consisted of initial “dull” signals followed by the “spiky” signal components (Figures 1B and 2A). It is reasonable to consider that this multicomponent EGM is a complex of far-field and near-field signals, which could be explained by the epicardial connections manifesting as an earlier epicardial signal followed by an endocardial signal at the left lateral ridge. Considering the nature of these FPs (low amplitude, high frequency, and long duration) and the possible conduction of electrical activities through the epicardial connections between the MB and the posterior wall, the electroanatomical mapping system could not describe the true earliest site. Chik and colleagues reported the characteristics of MB-related ATs following left posterior wall isolation. In their case series, activation maps revealed broad endocardial breakout sites, especially at the floor of the LA or adjacent to the ridge that corresponded to the previously reported anatomical distribution of MB–LA or CS connections.

In the current report, the endocardial breakouts of MB-related ATs were located at the left posterior wall where the anatomical distributions of MB–LA connections have not been reported. We speculated 2 possible mechanisms. First, the epicardial breakthrough at the posterior wall played an important role in demonstrating these unique activation patterns as a kind of a preferential conduction of electrical activities from the MB. Second, an epicardial bridge between the MB and posterior LA wall may not be anatomically recognized. An interplay of these 2 mechanisms is also possible. In our case series, considering that previous PVI or additional mitral isthmus linear ablation had blocked the MB–CS connections, the MB activities might have been preferentially conducted through the electrical or anatomical pathway toward the posterior wall.

Conclusions

This report revealed the unique activation patterns of the MB-related ATs after PVI. The MB electrical activities may have been conducted preferentially to unexpected LA sites through anatomical or electrical epicardial breakthroughs. Detailed high-density mapping at the LOM or the lateral ridge may help identify the true source of the AT after AF ablation.
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Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2020.05.005.

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