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Mechanistic Insights into Roof-Dependent Macroreentrant Atrial Flutter with Endocardial Block of the Roof Line: A Case Report

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Abstract: *Background:* Epicardial roof-dependent atrial tachycardia is rare among macroreentrant tachycardias. The importance of epicardial structure or fiber involving septopulmonary bundle (SPB) has not been realized generally. *Case presentation:* A 74-year-old woman who underwent catheter ablation of atrial fibrillation previously accepted a second-time radiofrequency ablation due to atrial flutter. The mapping and entrainment results of the tachycardia tended to be an epicardial SPB-dependent macroreentrant atrial tachycardia and it was ablated to sinus rate at the first single targeting site, just located in the breakout site of SPB into the posterior wall (PW) of left atrial (LA). The twice-activation mapping of PW of LA also proved the presence of SPB. No recurrent arrhythmia was seen at follow-up at 3 months. *Conclusion:* In this case, an uncommon phenomenon was observed post-ablation for persistent atrial fibrillation, where the epicardial muscular structure of the LA-SPB was involved in atypical atrial flutter. This should be considered as a potential factor in such cases. Further similar cases may be required to improve diagnostic accuracy and to formulate effective ablation strategies for this type of tachycardia.

Keywords: Catheter ablation; Macroreentrant atrial tachycardia; Septopulmonary bundle; Atrial flutter

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1. Background

Atypical atrial flutter (AFL) in the left atrial commonly occurs after ablation for atrial fibrillation (AF) and/or cardiac surgery or in patients with atrial myopathies [1]. Epicardial roof-dependent atrial tachycardia (ER-AT) is one in a thousand among macroreentrant atrial tachycardia (MRAT). The importance of epicardial structures and fibers is well-detailed, involving septopulmonary bundle (SPB) and septoatrial bundle (SAB). The general arrangement of the myofibers that make up the atrial walls is reviewed to provide a morphologic basis for atrial conduction and potential substrates of arrhythmias. Overlapping is observed at the dome and the posterior

wall of the left atrium, with the SAB running just beneath the SPB [2,3]. Successful identification and location of epicardial connections with endocardium and circuits of the re-entry are of extreme difficulty and thus take a long time to accomplish the procedure. Furthermore, an inadequate understanding of the re-entrant circuit probably leads to failed ablation results, consequentially increasing the risk of complications. To the best of our knowledge, epicardial roof-dependent macroreentrant atrial tachycardia consists of four types: epicardial bridging across structures and regions involving Bachmann's region, septopulmonary bundle, coronary sinus, and vein of Marshall, as described by Nayak *et al.* [4]. Whereas detailed explanations and diagnoses of such cases and the reason septopulmonary bundle-dependent flutter comes into existence are extremely rare. In this case report, we describe the subtle reentrant circuit through endocardial activation, as well as entrainment mapping, providing insights that the anatomical bridge formed by the SPB serves as the substrate for reentrant atrial tachycardia following ablation lesions for previous atrial fibrillation.

2. Case presentation

A 74-year-old female patient, who had an operation of persistent atrial fibrillation (circumferential pulmonary vein isolation, posterior wall box isolation, and left atrial appendage occlusion) on 27 April 2021, suffering from frequent palpitations due to recurrent atrial arrhythmia was referred for radiofrequency catheter ablation again after signing the informed consent. Endocardial electroanatomic mapping of the atrium was performed by a skilled electrophysiology physician of our institution using a PentaRay catheter (Biosense Webster, Diamond Bar, CA, USA) with the CARTO 3 system (Biosense Webster). The activation mapping of the left atrium (LA) and right atrium (RA) was done separately during atrial tachycardia (AT) with a tachycardia cycle length (TCL) of 286 ms. The LA activation result (**Figure 1A**) seemed like a focal AT originated from the LA posterior wall located between inferior pulmonary veins at first glimpse, and we saw the earliest activation position of RA was located near the coronary sinus ostium (CSO) (**Figure 1B**). It is noteworthy that the previous box isolation failed to block the floor line but an endocardial block of the roof line was presented and the activation cycle length (ACL) of LA (183 ms) and RA (127 ms) were both shorter than TCL, showing a missing endocardial cycle length. In consideration of the patient history and organized tachycardia, a reentrant AT was suspected. We conducted multiple positions entrainment mapping of LA and RA. The post-pacing interval (PPI) transcriptions, as shown in **Figure 1C**, strongly support that the AT we studied operates through a macroreentrant mechanism. The concealed entrainment sites were precisely located within the reentrant loop. The assumed circuit was suspicious to be SPB-dependent. We designed lines to describe the circuit of roof-dependent AFL on the activation map of LA and drew an optimal activation model in contrast with the anatomy of SPB (**Figure 2**). The dynamic propagation of LA and double potentials from both sides of the roof line suggested that further evidence of endocardial block of the roof line with recurrent conduction of the floor line was also displayed clearly. We noticed that the earliest activation site of the LA endocardium with a long duration, low amplitude, and fragmented potential was nearly at the same position as the SPB inserted posterior wall of LA, presenting a concealed entrainment result and considered as an optimal ablation target. Thus, an AT with a reentrant circuit involving the epicardial bridge of SPB was diagnosed. With no hesitation, we targeted this single site with radiofrequency applications (40 W/15s). The tachycardia terminated and turned to sinus rate (SR) in the first second after ablation (**Figure 3A**). Then, the activation map of the LA posterior wall using PentaRay catheter during SR was acquired. We could see from the picture that the relatively earlier sites of the

LA posterior wall showed no difference with the location of the ablation target, also meaning that there was an insertion point from epicardial SPB into the endocardium of the LA posterior wall (**Figure 3B**). Additionally, we began to ablate this area to homogeneousness. The remapping result of the LA posterior wall during SR exhibited the earlier sites of LA were near CSO, representing the disconnection between SPB and LA posterior wall. As the thickness of fat tissue in the LA dome may vary among different patients, the block of roof line with radiofrequency energy is difficult to accomplish transmural lesions for all designed roof lines. That is the reason such a case emerged with a previous endocardial block of the roof line of LA. In order to consolidate the effect of the procedure and prevent the possible recurrence of AFL and reentrant AT involving the other epicardial structures, the floor line was ablated to a bidirectional block and Box isolation of the post wall (**Figure 4**). The recurrent pulmonary veins were also ablated to isolation. Another sequence AT with a TCL of 228 ms, CS90 to CS12, was induced by using burst pacing, then proved to be a typical AFL via EPS and activation mapping of RA. The second tachycardia was terminated (**Figure 3A**) during cavotricuspid isthmus ablation with SmartTouch SF catheter and after the ablation procedure, the line of CTI was proved a bidirectional block. Finally, we repeated the inducing procedure and no tachycardia was induced. There was no recurrence during the 3-month follow-up.

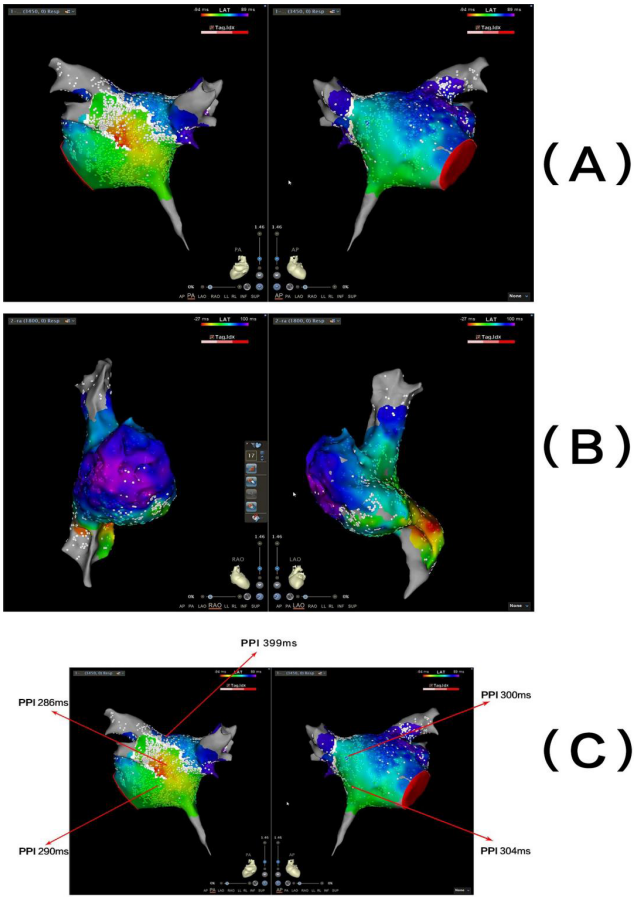

Figure 1. (A) Endocardial activation map of LA showing an activation gap (183 ms) with a block line in the LA roof. The relatively earlier sites were located between the inferior pulmonary veins. (B) Endocardial activation map of RA also showing an activation gap (127 ms) with the relatively earlier sites located around CSO. (C) Entrainment mapping of different sites of LA and RA. Each PPI was noted. (A) and (B) illustrated the focal-like sites of LA and RA were discrete from each other anatomically.

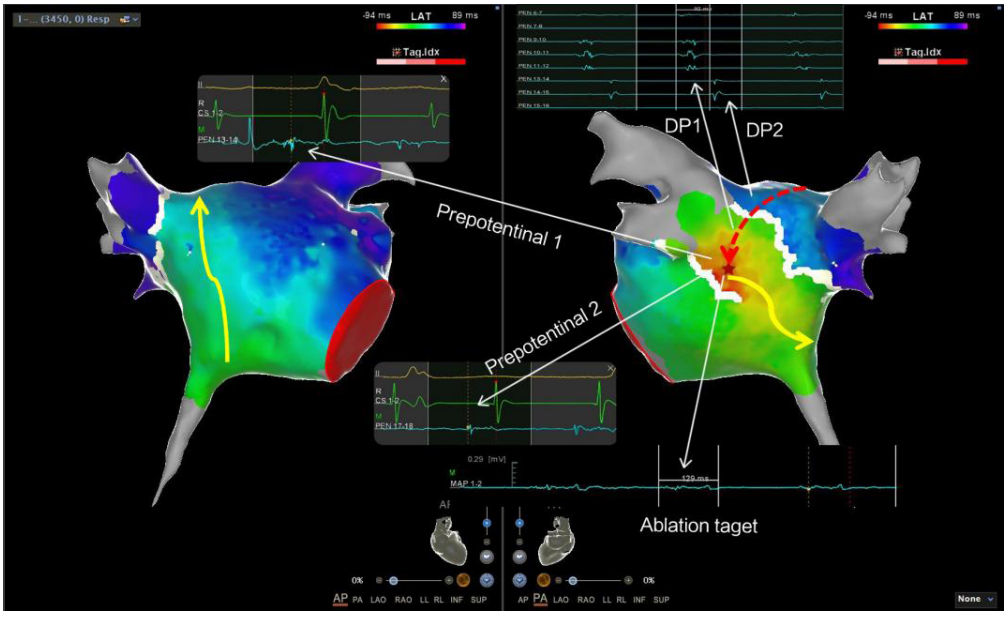

Figure 2. Solid yellow arrows (endocardial conduction) and dashed red arrow (epicardial conduction) represented the assumed loop of the AFL, noting that double potentials with a 92 ms interval meant an endocardial block of the roof line, the prepotentials were obtained around the breakthrough site of SPB into LA posterior wall, SPB-LA connection-ablation target exhibited a long duration of 129 ms, with low amplitude and fragmented potential.

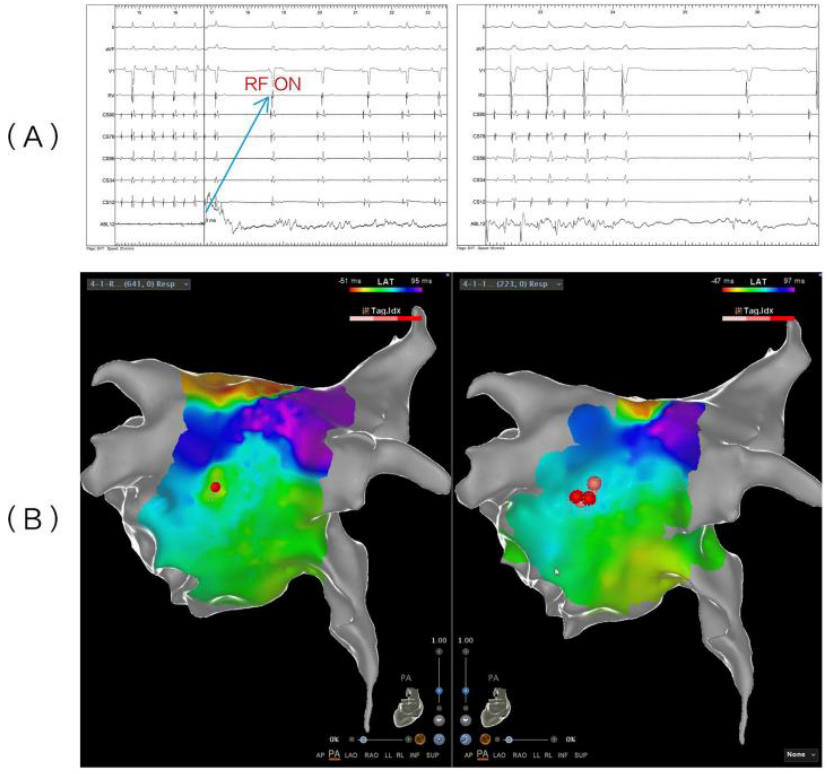

Figure 3. (A) The termination of SPB-dependent AFL and the typical AFL. From the left panel, tachycardia was terminated at a single site-breakthrough site of SPB. (B) Twice activation maps of LA post wall after terminating the roof-dependent AFL, the right one was obtained after further ablation of SPB-LA connection, meaning a disconnection between SPB and LA post wall.

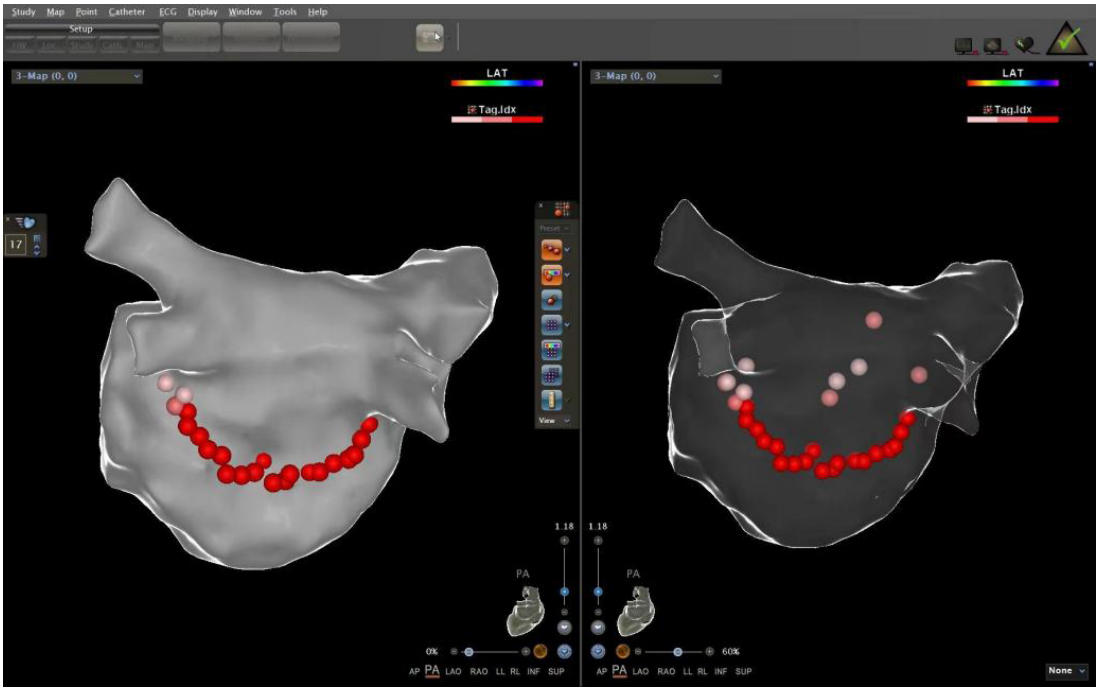

Figure 4. Isolation of post wall of LA as the ablation strategy of the atypical SPB-dependent AFL

3. Discussion

We presented a case of a macroreentrant AT involving epicardial bridge-SPB. Although such tachycardia has been reported before, the analogous cases are exceedingly rare. This case may provide further evidence for the AT correlated with SPB and supply our electrophysiological fellows with reference experience. Macroreentrant perimitral AFL often happens following persistent atrial fibrillation ablation with Marshall bundle-LA connection, most commonly due to prior non-transmural linear lesions of the mitral isthmus [5,6]. Such perimitral flutter has a common mechanism with our case in previous relevant linear ablation with an epicardial muscular connection. Moreover, it is the endocardial ablation that facilitates the formation of the epicardial roof-dependent reentrant ATs. Thus, the ablation strategy and efficiency of linear ablation in persistent atrial fibrillation need provoking thinking.

The activation gap is regarded as the missing part of cycle length with endocardial high-density mapping. In this regard, any activation gap that spanned > 5% of the tachycardia cycle length is considered significant. In this situation, the AT is proved to be reentrant by entrainment mapping. Then, an AFL using epicardial connections as a component part of the circuit is suspected with reasonable explanations. Plus, two discrete focal regions obtained from the endocardium of RA and LA can further illustrate the reason the endocardial ACL is shorter than the TCL. Entrainment pacing plays an important role in a pseudo-focal atrial tachycardia case [7]. We should recognize the pitfall of endocardial mapping results. As Thomas Pambrun mentioned in his article [3], the endocardial prepotentials of the posterior wall recorded after roof linear ablation probably mean a conduction recovery of epicardial structure-SPB. A long-duration, low-amplitude, fragmented potential with mid-diastolic activation at the site was validated by entrainment, indicating that the breakthrough sites are key points within the circuit-isthmus. The mid-diastolic activated isthmus is likely the weakest part of the re-entry circuit and thus represents an optimal ablation target [8]. In our case presented, tachycardia was terminated by

a single ablation site, which accounts for the reasonable descriptions above. However, more relevant cases are needed to explore the optimal ablation strategy, targeting SPB-LA connection or anatomical isthmus. Besides, when a patient with persistent AF operated circumferential pulmonary vein isolation plus linear ablation such as roof and floor line accepted a second-time ablation due to AFL, a single-loop reentrant atypical AFL involving the epicardial bridge of LA should be considered, if the following conditions were met: (1) Each of the atrial mapping shows a focal-like result via endocardial high-density mapping, with the two earliest sites from LA and RA separating with each other anatomically. (2) The ACL of the two are both shorter than TCL. (3) Good PPI along the assumed circuit, except for the endocardial sites corresponding to the EB anatomically, including the breakthrough site, is obtained via endocardium entrainment. (4) Poor PPI along the endocardial sites corresponding to the EB anatomically is obtained via endocardium entrainment. It is with no doubt that epicardial mapping and entrainment is the optimal maneuver for diagnosis and ablation of AFL involving epicardial structures in some cases, but whether it deserves to puncture pericardium at risk shall be taken into consideration seriously.

4. Conclusion

In conclusion, the involvement of the epicardial muscular structure of the LA-SPB in atypical AFL that occurred following ablation for persistent atrial fibrillation should be carefully considered. More cases of this nature may be needed to improve diagnosis and refine ablation strategies for such tachycardias.

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Disclosure statement

The authors declare no conflict of interest.

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