Complex organized atrial arrhythmia with alternation between two circuits involving probable epicardial connections: An ultra-high-density mapping study

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Introduction

Atrial fibrillation (AF) is the commonest sustained arrhythmia in hypertrophic cardiomyopathy (HCM), with a prevalence of 20%.[1] Catheter ablation is an option for rhythm control, although freedom from AF/atrial tachycardia (AT) is lower than in non-HCM patients, both after a single procedure and after multiple procedures.[2] Transition to a permanent form of AF occurs in a quarter of patients,[1] and persistent AF is associated with lower success rate of ablation.[2,3] Recurrences after persistent AF ablation are more often atypical flutters than AF, and non–pulmonary vein (PV) sites are common.[3]

We report a case of a complex organized atrial arrhythmia occurring after a previous ablation of persistent AF in an HCM patient with a severely diseased left atrium (LA).

KEY TEACHING POINTS

- Complex atrial tachycardia may arise after previous extensive ablation in a severely diseased left atrium. In the present case an alternation between 2 circuits was observed.
- Multielectrode high-density mapping with fine tuning of scar burden mapping may be required to fully understand the tachycardia circuit.
- Epicardial connections were presumed to be part of the circuits, as endocardial activation mapping incompletely covered the cycle length.

Case report

A 42-year-old woman with a history of HCM and alcohol septal ablation was scheduled for a redo ablation procedure of AT. Left ventricular ejection fraction was 65% and LA surface was 40 cm^2.

The patient had had a first ablation procedure in September 2017 for persistent AF recurring despite amiodarone and nadolol. The initial procedure consisted of ablation of spatiotemporal dispersion areas widely distributed in the LA (anterior wall, posterior wall, septum, base of LA appendage, ridge, right superior PV, and right carina) and the right atrium (crista terminalis). PV isolation was not performed.[4] AF cycle length (CL) had increased from 220 ms to 300 ms and AF was cardioverted at the end of the procedure.

This second procedure was performed under general anesthesia and began in sinus rhythm. Burst pacing induced a sustained AT with 2 CLs of 519 ms and 665 ms alternating in a 1:1 fashion, each corresponding to a unique activation pattern on the decapolar coronary sinus (CS) catheter (Figure 1A). The electrocardiogram showed 2 slightly different P-wave morphologies, particularly in lead V_1 (Supplemental Figure 1). Activation mapping was acquired by continuous automated activation with an IntellaMap Orion multielectrode catheter (Boston Scientific, Marlborough, MA) using the Rhythmia three-dimensional electroanatomic mapping system (Boston Scientific). Electroanatomic mapping with the Rhythmia system[5] uses 2 reference electrograms, 1 main (R) and 1 additional (ΔR) chosen on the decapolar catheter. Cardiac beats are automatically accepted for inclusion in the map based on CL stability, stable relative timing of 2 reference electrograms, electrode location stability, and respiratory gating. The window of interest is automatically set by the system at the CL value and centered on the main reference electrogram.

We decided to map each circuit separately. To do so, the CL graph was used to discriminate between the 2 circuits.

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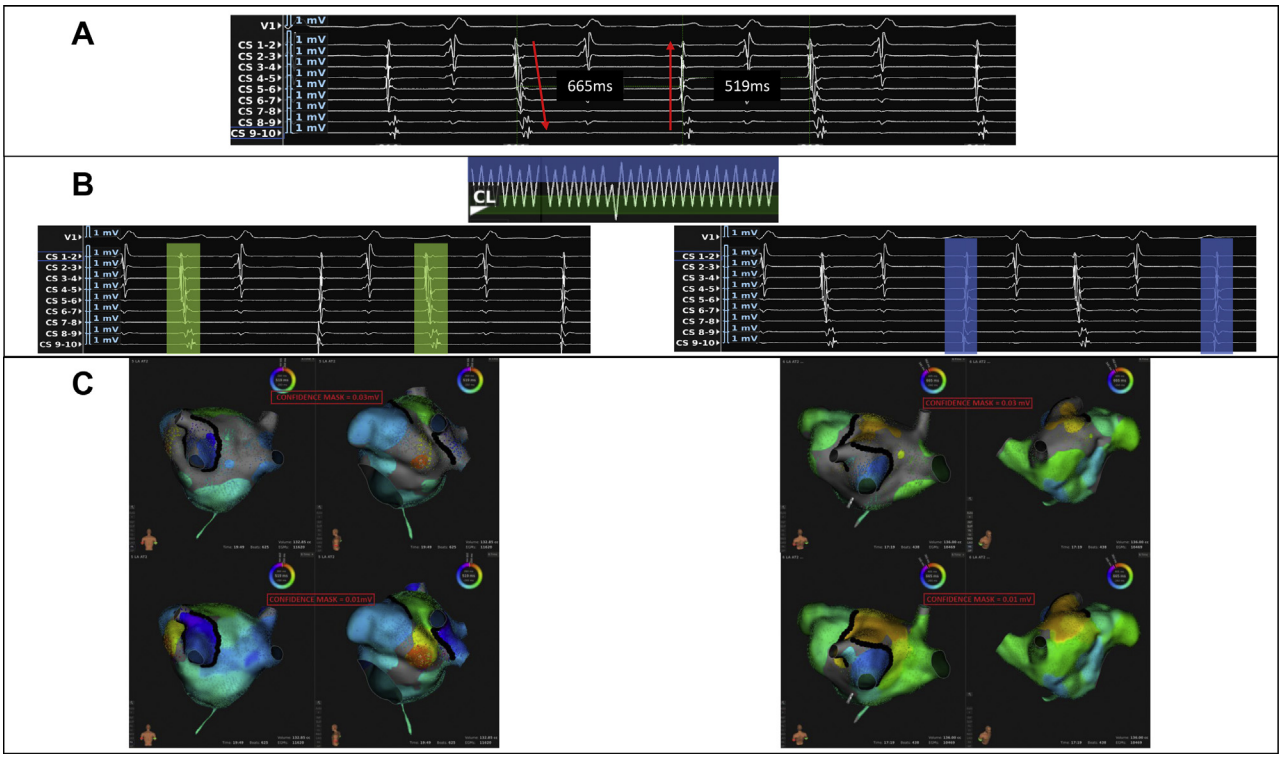

Figure 1 Tuning of the Rhythmia (Boston Scientific, Marlborough, MA) settings. **A:** The 2 alternating cycle lengths (CL) with different activation patterns on the coronary sinus catheter. **B:** The CL graph with a sawtooth pattern and the 2 activations mapped separately: in *green*, the lower points corresponding to the short CL of 519 ms (circuit 1); and in *blue*, the upper points corresponding to the long CL of 665 ms (circuit 2). **C:** For both circuits, the voltage map is shown with the nominal confidence mask at 0.03 mV and at the lowest possible value of 0.01 mV, which was required to understand the activation.

It exhibited a sawtooth pattern with lower points corresponding to the short CL and upper points corresponding to the long CL (Figure 1B). The "CL stability" box had to be checked and for each map the window of interest was set exactly at the considered CL value, thus excluding beats corresponding to the other CL values.

Circuit 1 (CL 519 ms) was mapped in 20 minutes and 11,621 points were collected (Supplemental Figure 2, Video 1). Voltage mapping is nominally displayed with the confidence mask tool set at 0.03 mV, meaning that points in an area with electrogram bipolar amplitude below 0.03 mV have no color code and are displayed in gray. With such a dense scar setting, large areas in the posterior wall, PV antrum, mitral isthmus, and ridge were displayed in gray and the circuit could not be apprehended. By setting the confidence mask to the lowest possible value of 0.01 mV, most areas became color-coded and the activation became intelligible (Figure 1C). Starting from the lower ridge, activation propagated superiorly, then traveled posteriorly around the left PV antrum, entered the left inferior PV (LIPV) by its inferior pole, and ended at the left carina. Activation from the lower ridge followed a second path epicardially to the distal CS, where it split into 2 wavefronts, 1 activating the CS in a distal-to-proximal fashion and another ascending toward the posterior mitral isthmus and joining the descending posterior wavefront entering the LIPV. The anterior wall was not part of the circuit, being activated by 3 colliding wavefronts, originating from the roof, the septum, and the mitral isthmus.

The activation at the left carina did not complete the circle to the lower ridge but switched to the second circuit. The second circuit started on the posterosuperior aspect of the left superior PV. There was a delay of 157 ms in the endocardial activation between the end of circuit 1 and the beginning of circuit 2 (Supplemental Figure 3), and we hypothesized that there was an unmapped epicardial connection between the 2 circuits.

Circuit 2 (CL 665 ms) was mapped in 17 minutes and 10,469 points were collected (Supplemental Figure 4, Video 2). As for circuit 1, the confidence mask had to be lowered at 0.01 mV in order to visualize the activation in the entire LA (Figure 1C). Activation from the posterior wall proceeded both to the ridge, activated in a superior-to-inferior way, and inferiorly, where it turned around the LIPV, entered the LIPV at its anterior pole, and ended at the posterior aspect of the LIPV. Again, there was a missing endocardial activation of 130 ms duration from this point to the start of circuit 1 at the lower ridge level (Supplemental Figure 5). The missing activation could probably be supported by an epicardial connection along the ligament of Marshall. As in circuit 1, the anterior wall was not part of the circuit.

Figure 2 and Video 3 display the sequential activation mapping of circuit 1 and circuit 2. For ease of interpretation,

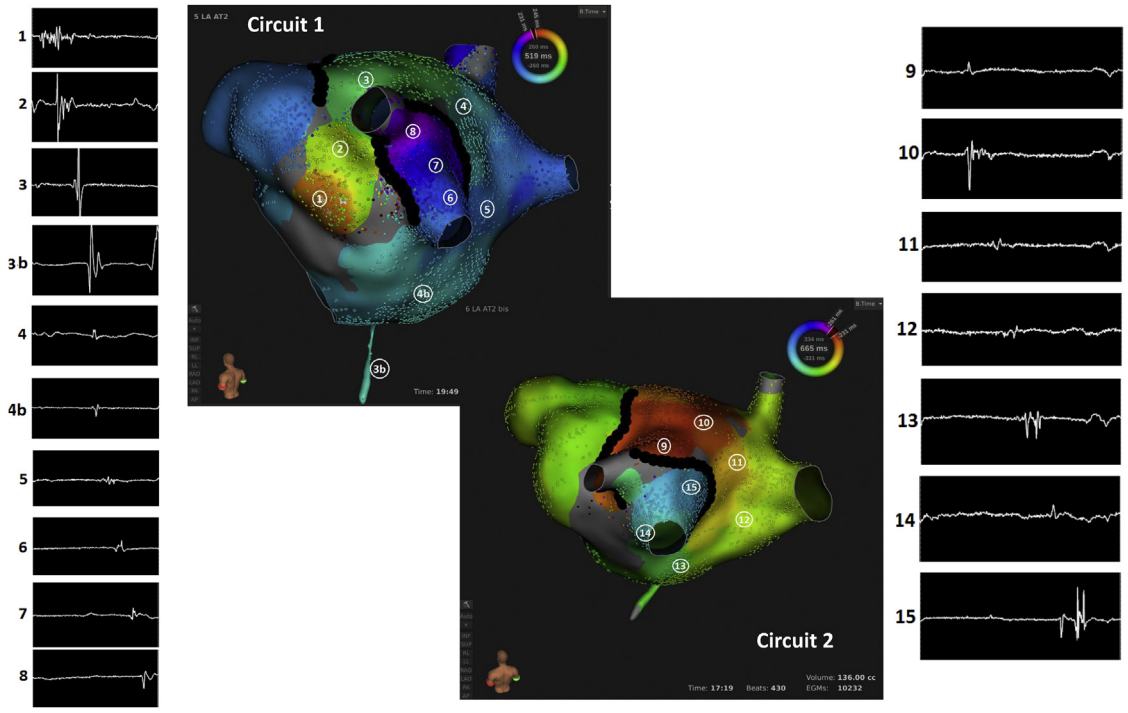

Figure 2 Activation mapping of circuit 1 and circuit 2. Sequential activation propagation is annotated by numbers. The missing endocardial activation is between points 8 and 9 (connecting circuit 1 to circuit 2) and between points 15 and 1 (connecting circuit 2 back to circuit 1). Local electrograms along the circuits are displayed.

activation propagation is annotated by numbers and the missing endocardial activation connecting both circuits is represented on the video in dashed lines.

Ablation was started at the lower ridge, which was thought to be a crucial point connecting the 2 circuits. A wide complex fractionated electrogram of 109 ms duration was present in this area (Figure 2, Supplemental Figure 2). The first radiofrequency application resulted in a single CS activation and the second application prolonged the CL and converted the AT to sinus rhythm (Supplemental Figure 6).

Discussion

We report a complex organized atrial arrhythmia with alternation of 2 circuits. Although dual-loop and triple-loop tachycardias have been described in post-AF ablation atria,[6] these loops coexist and share a common isthmus. To our knowledge, a tachycardia with 2 different, successive circuits has not been previously reported.

The mechanism of this arrhythmia with LA activation mapping incompletely covering the CL was challenging to understand.

A biatrial tachycardia was excluded because the Bachmann bundle/high septum and the proximal CS (from poles 6 to 10) were both activated in a LA-to–right atrium fashion.

The first mechanism is a macroreentrant atrial flutter with the circuits detailed above. The main issue in this hypothesis is related to the unmapped connections between the 2 circuits. One explanation is that all the circuits are totally endocardial but with (1) extremely slow endocardial conduction velocities in this region, to such an extent that a "pause" in conduction is erroneously diagnosed; (2) imprecise endocardial mapping with insufficient activation points; and/or (3) missing activation points in an extremely scarred region <0.01 mV. Although these explanations cannot be totally excluded, we believe they are unlikely, respectively because (1) the conduction velocities in the regions immediately surrounding the "missing areas" do not seem particularly low, (2) the mapping density in this region seems satisfactory, and (3) the capability of regions below 0.01 mV to sustain propagation of an electrical impulse is probably low.

The other explanation is the presence of an epicardial connection over the Marshall bundle. Epicardial muscle bundles often connect the ligament of Marshall and the CS to the PVs and the ridge.[7] These anatomic structures are increasingly involved in postablation ATs.[8] In our case, these presumed connections from the lower ridge to the CS and encompassing the left PV-ridge region, which are mandatory to understand the whole activation of a macroreentrant double circuit, could be supported by epicardial fibers present in the lateral LA region.

The second mechanism is a focal AT situated in the ligament of Marshall with alternating unidirectional block. A block in its superior portion would lead to an exit at its inferior part, at the lower ridge, giving rise to circuit 1. A block in its inferior portion leading to an exit at its superior part, at the carina–posterosuperior LA junction, could give rise to circuit 2 (Supplemental Figure 7).

It is noteworthy that both hypotheses, focal AT and macroreentrant flutter, require the involvement of a long ligament of Marshall. Resetting would have been important to elucidate the mechanism (ie, resetting with fusion leading to the diagnosis of reentry).

Another interesting feature of our case was the need for tuning the confidence mask owing to the very low voltage of the LA, the latter being related to both extensive fibrosis and previous radiofrequency lesions. The confidence mask had to be lowered to its minimal value to visualize most of the circuit. The mini-basket Orion catheter has flat and small (0.4 mm^2) electrodes and is characterized by particularly low background noise,[5] which allows for accurate critical isthmus identification in the scarred LA. Mapping and understanding the circuit would probably not have been possible with a standard 3.5-mm-tip catheter.

Finally, the ablation strategy adopted in the first ablation procedure could have contributed to the tachycardia seen during this repeat procedure. The nonisolated PVs were part of the circuits and, as in the study by Seitz and colleagues,[4] the arrhythmia recurrence was in an organized form. Whether such an ablation strategy should have been used in a persistent AF with a severely diseased LA can be debated.

Conclusion

We describe the case of a complex organized atrial arrhythmia arising after previous extensive ablation. Multielectrode high-density mapping with tuning of scar burden mapping was required to fully understand the tachycardia, which consisted of 2 successive circuits. This alternation could be explained either by a complex macroreentry with connections over a presumed epicardial path or by a focal ligament of Marshall–mediated AT with alternating unidirectional block. Epicardial connections can sometimes be part of the circuit and should be imputed in case of an endocardial activation mapping incompletely covering the CL.

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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.2019.01.013.

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