An interesting case of reverse electrical remodeling of the left atrium after radiofrequency ablation



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Introduction

Atrial remodeling caused by a variety of comorbidities culminates in the onset and perpetuation of atrial fibrillation (AF). AF by itself may result in atrial remodeling and contributes to the progressive nature of the arrhythmia.¹ Radiofrequency ablation (RFA) is a well-established strategy for rhythm control in patients with symptomatic and drug-refractory AF.² Successful RFA is associated with significant reverse remodeling of the left atrium (LA). Reverse electrical remodeling starts within hours to days and structural remodeling ensues over a period of months.³ There is a large body of literature suggesting a decrease in LA size and concurrent preservation of LA function after ablation during long-term follow-up.^{4–6} We present a case of reverse electrical remodeling of the LA as seen on 3-D mapping after AF ablation.

Case report

A 68-year-old man with past medical history of coronary artery disease, obstructive sleep apnea, and morbid obesity presented for management of early persistent AF (CHA₂DS₂-VASc score of 3 on rivaroxaban) 18 months from diagnosis. He had failed 2 different antiarrhythmic drugs. At the time of his first ablation, the patient was symptomatic and had 100% AF burden despite being on flecainide for rhythm control. A transesophageal echocardiogram showed borderline systolic function (ejection fraction of 50%), mild-to-moderate biatrial enlargement, and no left atrial or left atrial appendage thrombus. He underwent electrical cardioversion with conversion to sinus rhythm prior to the procedure. The atrial electroanatomic maps were constructed using a LASSO mapping catheter and CARTO mapping system (Biosense Webster, Diamond

KEYWORDS Atrial fibrillation; Atrial remodeling; Left atrium; Radiofrequency ablation; Reverse atrial remodeling (Heart Rhythm Case Reports 2018;4:177–179) Bar, CA) while the patient was in sinus rhythm. A Thermo-Cool Navistar SmartTouch (Biosense Webster, Diamond Bar, CA) contact force catheter (3.5-mm tip) with an interelectrode distance of 5 mm was used for RFA. All 4 pulmonary veins along with the cavotricuspid isthmus were successfully isolated, with noninducibility of tachyarrhythmias at the end of the procedure. The 3-D map during the first ablation is shown in Figure 1A and B. During postprocedure follow-up the patient was doing well, without recurrence of AF, and his antiarrhythmic drug was discontinued.

The patient had relapses of AF 6 months after the procedure. He remained symptomatic with these episodes even though his AF burden was 5%, as seen on looping and nonlooping event monitors. He underwent cardioversion and sotalol initiation, with subsequent recurrence of AF. Given his recurrent symptomatic AF refractory to antiarrhythmic drugs, the patient opted to undergo repeat pulmonary vein isolation. The patient underwent repeat RFA 433 days after the first ablation. He was in sinus rhythm prior to the procedure. The 3-D map during the second ablation is shown in Figure 1D and E. A total of 3560 point maps were collected during the first ablation and 3892 during the second ablation, with consistent point density throughout the LA. Voltage < 0.30 mV was considered abnormal and > 1.0 mV was considered normal atrial tissue.⁷ The cumulative voltage abnormality in the LA was 79% at the time of the first ablation. It decreased to 20% during the second ablation after considerable time in sinus rhythm. LA pressures were 24/8 mm during the initial ablation and decreased to 10/5 mm during the second ablation. Furthermore, atrial volumes reduced from 138 cc to 108 cc on computed tomography scan prior to each ablation (Figure 1C and F). All 4 pulmonary veins along with the cavotricuspid isthmus, typical atrioventricular nodal reentry tachycardia, and superior vena cava / right atrium junction were successfully isolated during the second procedure, with noninducibility of tachyarrhythmias at the end of the procedure. Our case demonstrates significant reverse electrical and structural remodeling of the LA after RFA.

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- Electrical and structural remodeling of the atria plays a central role in the onset and preservation of atrial fibrillation (AF).
- Successful radiofrequency ablation (RFA) has the potential to cause reverse atrial remodeling in AF patients, unlike other pharmacologic therapies.
- RFA can play an important role in AF substrate modification if considered earlier in the course of management.

Discussion

Electrical and structural remodeling of the atria plays a central role in the onset and maintenance of AF.¹ Studies have shown positive effects of RFA on atrial remodeling.⁸ In a recent meta-analysis by Zhuang and colleagues,⁴ RFA caused reverse structural remodeling of the LA by reducing the atrial size and volume. This is likely because of atrial shrinkage caused by reverse remodeling from establishing sinus rhythm.^{9,10} Reverse structural remodeling is demonstrated very clearly in our case. Figure 1C shows LA volume of 138 cc at the time of the first ablation and Figure 1F shows a significant reduction in the LA volume to 108 cc during the second ablation. Similarly, reverse atrial electrical remodeling, closer to normal physiology, was demonstrated in earlier reviews.^{3,11} This is seen in our case as a significant reduction in abnormal atrial tissue during the second ablation (Figure 1D and E) when compared to the first ablation (Figure 1A and B). Our patient, who was in long-standing persistent AF prior to RFA, converted to paroxysmal AF with intermittent episodes of AF after the first ablation. The cumulative AF burden has gone down from 100% to less than 5% on looping and nonlooping monitors post ablation, which is the reason why his LA remodeled.

Therapies like antiarrhythmic drugs and rate control medications focus on managing symptoms of AF but do not address the underlying substrate that perpetuates it. Over the last decade, recognition of the importance of atrial remodeling in AF led to the notion of developing therapies to avert remodeling. As demonstrated in our case, RFA can cause positive reverse remodeling of the atria and can modify the underlying substrate that propagates AF. However, this approach is not feasible if underlying LA fibrosis



Figure 1 Electroanatomic bipolar voltage maps generated with 3-D CARTO mapping system during sinus rhythm. A: Voltage map during the first ablation, anteroposterior (AP) view. B: Voltage map during the first ablation, posteroanterior (PA) view. C: Left atrium (LA) volume on computed tomography scan prior to the first ablation. D: Voltage map during the second ablation, AP view. E: Voltage map during the second ablation was done 433 days after the first ablation. F: LA volume on computed tomography scan prior to the second ablation. AF = atrial fibrillation; AT = atrial tachycardia.

has progressed markedly. These patients are relatively resistant to reverse remodeling and there is a high chance of recurrence of AF after RFA.¹² In summary, our experience shows that RFA can play an important role in AF substrate modification if considered earlier in the course of management. Further large prospective studies are required to validate our findings.

Conclusion

RFA is a well-established strategy for rhythm control in patients with symptomatic and drug-refractory AF. Successful RFA has the potential to cause reverse atrial remodeling, thus allowing AF substrate modification. We present one such case of reverse electromechanical remodeling of the LA after RFA, with a reduction in LA volumes and nearnormalization of atrial voltages on 3-D mapping.

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