Successful fluoroless ablation of an incessant atypical atrial flutter attributed to AtriClip usage during mini-MAZE surgery for persistent atrial fibrillation

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Introduction

The AtriClip left atrial appendage (LAA) exclusion device (AtriCure Inc, Westchester, OH) is a surgical LAA closure device frequently used in conjunction with thoracoscopy-guided mini-MAZE surgery to treat atrial fibrillation (AF).[1] It is composed of 2 parallel titanium bars, which provide constant tension when deployed. The AtriClip is coated with polyester fabric, which promotes tissue ingrowth and provides an atraumatic surface, preventing erosion into surrounding structures.[1] There is evidence that epicardial ligation of the LAA may have some antiarrhythmic effect, with reduced AF burden observed in patients who underwent successful LAA exclusion.[2] However, surgical intervention to the left atrium may also carry proarrhythmic potential. In this article, we report a rare case of incessant atypical atrial flutter that appears to be driven by the electrophysiologic remodeling of the local atrial tissue as a result of AtriClip application.

Case report

The patient is a 57-year-old man with a past medical history significant for hypertrophic cardiomyopathy and nonsustained ventricular tachycardia with previous dual-chamber implantable cardioverter-defibrillator (ICD) implantation for primary prevention. The patient subsequently developed persistent AF, peripheral thromboembolism, and chronic systolic heart failure with ejection fraction of 30%. After the patient developed persistent AF, right ventricular pacing became dominant due to very slow intrinsic atrioventricular conduction. Therefore, he was referred to cardiothoracic surgery for a combined mini-MAZE, LAA closure using the AtriClip device, and concomitant left ventricular epicardial lead placement.

The mini-MAZE was performed successfully and included pulmonary vein isolation and creation of roof and floor lines for posterior wall isolation, as well as LAA closure using the AtriClip device. The left ventricular epicardial lead was unable to be placed due to onset of ventricular fibrillation during attempted lead placement. After the surgery, the patient was able to maintain sinus rhythm for about 12 months, but he subsequently developed recurrent fatigue and dyspnea. Device interrogation demonstrated persistent atypical atrial flutter with a rate of about 130 beats per minute, refractory to antitachycardia pacing and cardioversion using the patient's existing ICD. Decisions were then made to proceed with repeat, catheter-based ablation in December 2016.

The procedure was performed using our standard, fluoroless approach to AF ablation, guided by the contact force sensing technology and intracardiac echocardiogram (ICE) (unpublished data). The patient was admitted to an outpatient unit and studied in a dedicated electrophysiology lab. After sterile draping in the usual fashion, 3 8F venous sheaths were placed in the right femoral vein. An ICE catheter was inserted into the right atrium for catheter visualization throughout the entire ablation. Another contact force sensing Carto ST catheter (Biosense Webster, Diamond Bar, CA) and a Lasso catheter were used to localize the pulmonary veins and ablate in the left atrium. The double transseptal access technique was used, guided entirely by the ICE. Heparin bolus was given prior to the transseptal access, followed by an intravenous drip to target activated clotting time around 350 seconds. The esophagus position was premapped by an esophageal mapping catheter (Biosense Webster, Diamond Bar, CA) prior to the ablation.

After successful establishment of double transseptal access guided by ICE, initial exploration using a circular Lasso catheter (Biosense Webster, Diamond Bar, CA) demonstrated residual potentials near the ostium of the left inferior pulmonary vein. The right inferior vein and the posterior wall also demonstrated reconnection. Therefore, our initial ablation strategy was to first completely redo the left- and right-sided wide-area circumferential ablation plus posterior wall isolation. A linear ablation was also performed over the

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mitral isthmus (from the left inferior pulmonary vein to the mitral annulus), given the presence of an atypical atrial flutter. The ablation was guided by the Lasso, contact force, and ICE imaging, as well as impedance and local electrogram changes, without use of fluoroscopy. The left- and right-sided veins were isolated first, followed by isolation of the entire posterior wall (Figure 1). Upon completion of repeat pulmonary vein isolation and posterior wall isolation, as well as mitral isthmus ablation, the patient's slow atypical flutter persisted. Decisions were then made to proceed with entrainment mapping of the arrhythmia. Initial mapping of the left atrial floor, septum, and roof resulted in a postpacing interval

minus tachycardia cycle length greater than 60 ms. Further mapping over the residual appendage area, near the site of previous AtriClip application, demonstrated very complex electrograms including fractionated, split, and very low-amplitude potentials (Figure 2A). Entrainment mapping over this area demonstrated a postpacing interval equal to tachycardia cycle length (Figure 2B), with no change in the flutter wave morphology (Figure 2C) during pacing and during tachycardia, consistent with concealed entrainment.[3] Decisions were then made to proceed with linear ablation from the superior mitral annulus to the left superior pulmonary vein, traversing the area where the complex electrograms were identified and where concealed entrainment was achieved. The atypical atrial flutter was easily terminated during linear ablation and was immediately followed by appropriate dual-chamber pacing from the patient's ICD, prior to completion of the ablation line (Figure 2D). As shown in Figure 2D, upon termination of the arrhythmia, there was still delayed atrial conduction to the Lasso that was placed in the remnant appendage area, between the left superior pulmonary vein and mitral annulus. The atrial electrograms on the Lasso were recorded after the ventricular signals, suggesting very delayed conduction into the remnant appendage area. Continued ablation to complete the line resulted in complete isolation of the entire remnant appendage area, as demonstrated by complete loss of atrial electrograms on the Lasso (Figure 2D), suggesting that conduction block
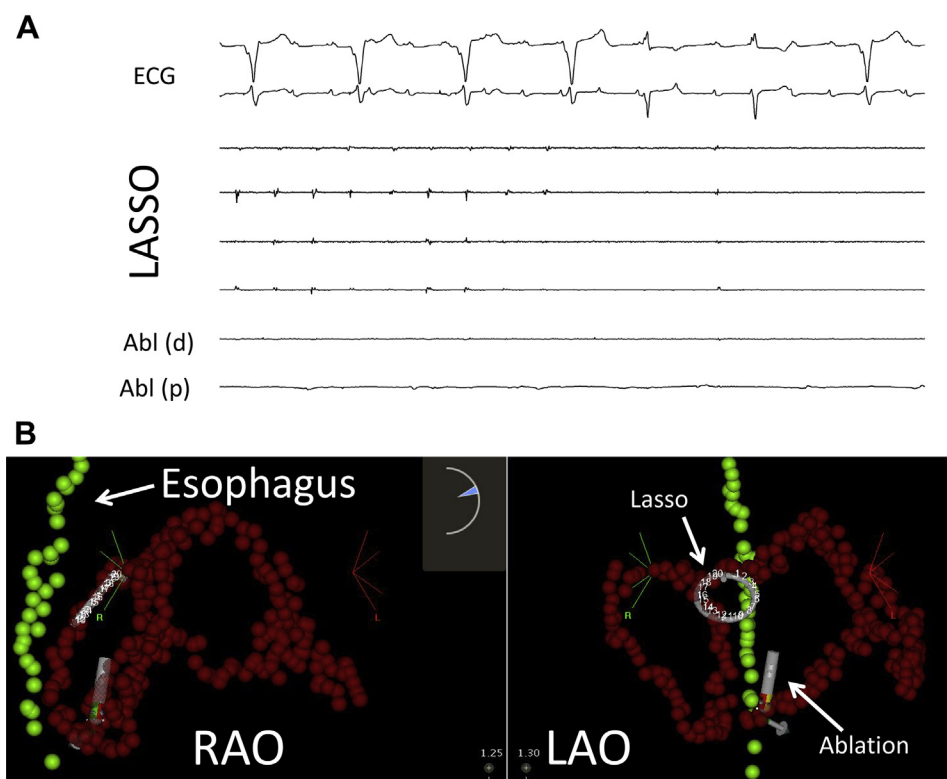

Figure 1   Complete isolation of the left atrial posterior wall during persistent atrial flutter. **A:** Demonstration of complete loss of electrogram on the Lasso catheter placed over the posterior wall. **B:** Position of the circular Lasso and ablation catheters at the time of posterior wall isolation, shown on the CARTO mapping system. LAO = left anterior oblique; RAO = right anterior oblique.
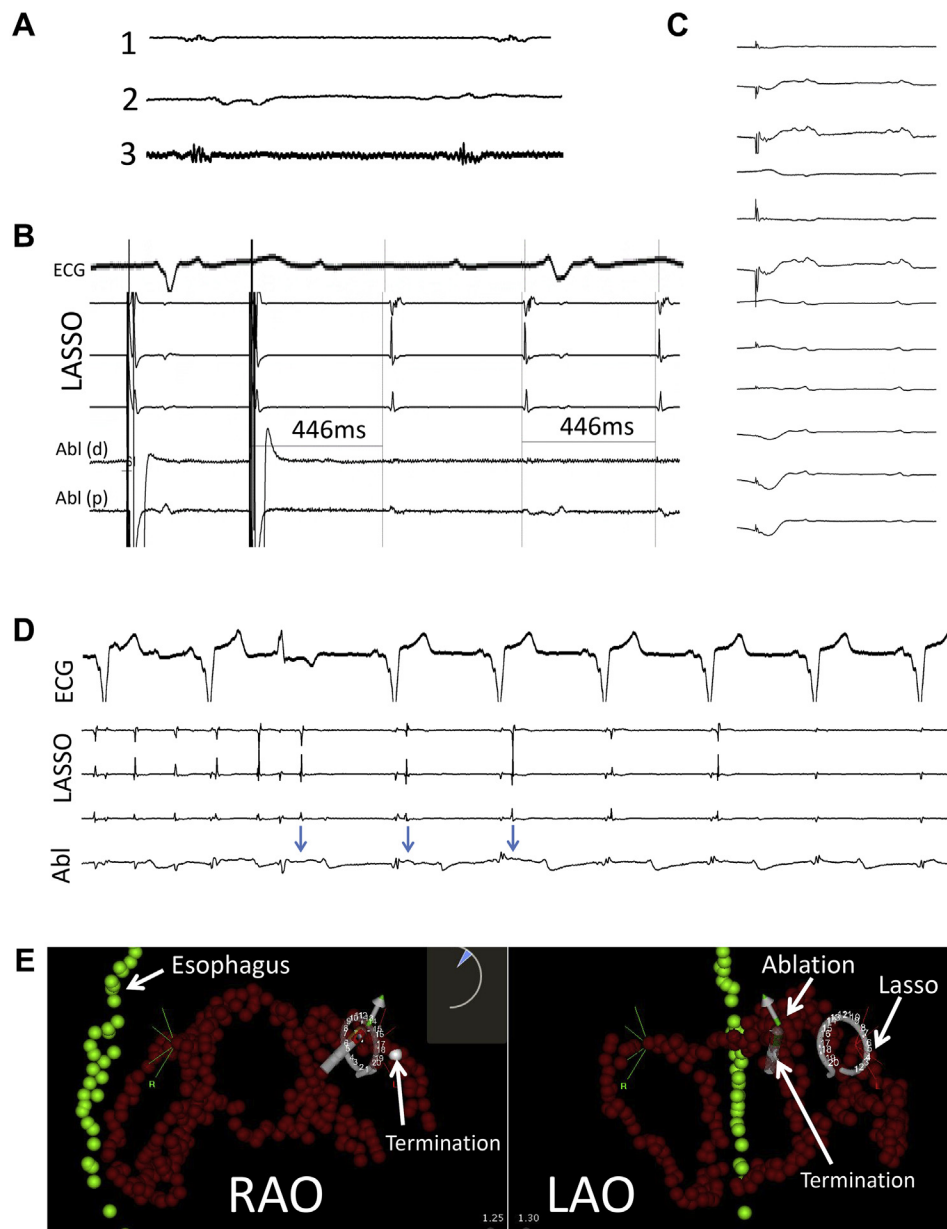
Figure 2 Entrainment and successful termination of the atypical atrial flutter during ablation. **A:** Examples of complex electrograms, including fractionated (1), split (2), and very low-amplitude signals (3), recorded near the ablation site. **B:** Demonstration of the postpacing interval equal to the tachycardia cycle length of 446 ms at the ablation site. **C:** Demonstration of no change in flutter wave morphology on the surface electrocardiogram (ECG) during entrainment pacing and spontaneous tachycardia. Only the last paced P wave and the first P wave during tachycardia were shown. ECG labels from top to bottom: I, II, III, aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5, V_6. **D:** Termination of the atypical atrial flutter during ablation, followed by dual-chamber paced rhythm with delayed atrial activation (recorded after ventricular electrogram) on the Lasso and ultimate complete electrical isolation of the residual appendage area. Note the near-instantaneous termination of the flutter as the low-amplitude atrial electrograms start to appear on the ablation catheter as it moves toward the concealed entrainment site (*arrows*). Only far-field ventricular signals were present on the Lasso catheter toward the end of the recording. The Lasso catheter was positioned near the remnant appendage area, between the left superior pulmonary vein and mitral annulus. **E:** Position of the ablation and Lasso catheters at the time of concealed entrainment and during flutter termination (*arrows*) on 2 orthogonal views on CARTO. LAO = left anterior oblique; RAO = right anterior oblique.

was achieved over both the superior and inferior mitral isthmus lines. Additional insurance ablations were performed to eliminate the low-amplitude, fractionated signals initially identified (Figure 2D and E).

After termination of the atypical flutter, further testing was performed using high-dose isoproterenol with target dose of 10 μG/min. In the presence of high-dose isoproterenol, no spontaneous or inducible atrial arrhythmias (through left

atrial burst pacing with cycle length down to left atrial refractory period) were seen. At the end of the procedure, additional ablations were performed over the cavotricuspid isthmus, with bidirectional block achieved. The patient tolerated the procedure well, with no apparent complications observed. The total procedure time, defined as the time from vascular access to removal of all intracardiac catheters, was 111 minutes. No fluoroscopy was used for the entire procedure.
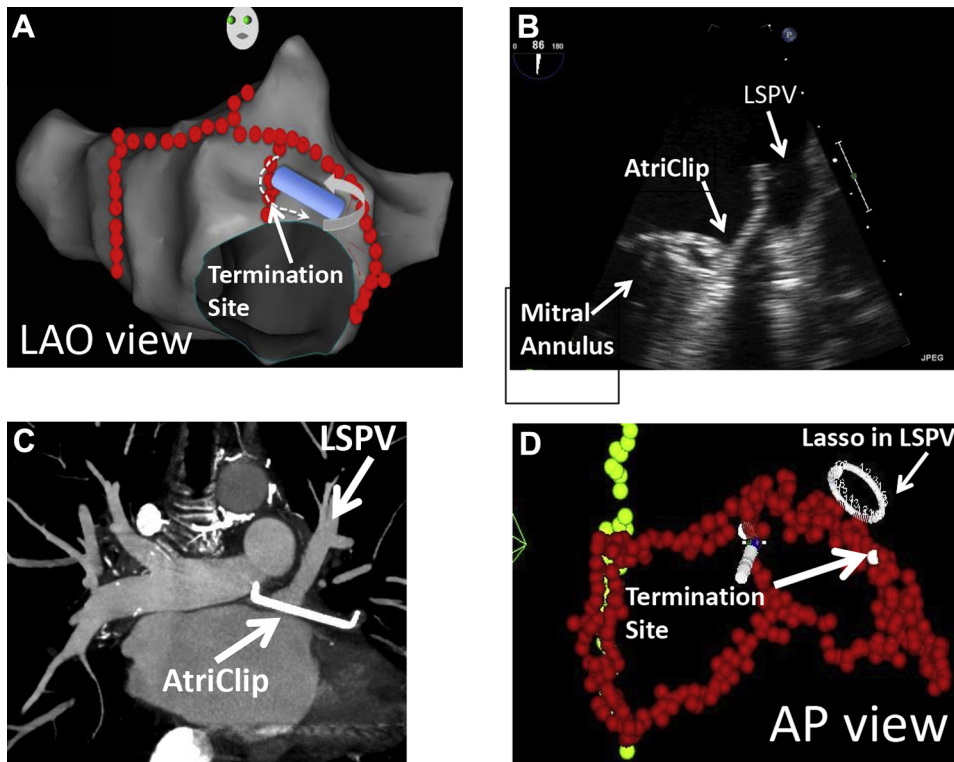
Figure 3 Schematic drawing of the successful ablation site in relationship to left atrial anatomy and depiction of AtriClip position based on intraoperative CARTO mapping as well as preoperative transesophageal echocardiogram (TEE) and computed tomographic (CT) imaging. **A:** A presumed flutter circuit was drawn around the AtriClip. Dotted line indicates areas of slow conduction detected during mapping. Solid arrow indicates area of relatively normal conduction. LAO = left anterior oblique. **B:** TEE showing the remnant of the left atrial appendage (LAA) and the closure line from the AtriClip device, between the left superior pulmonary vein (LSPV) and the mitral annulus. **C:** CT image (coronal view) showing the relationship between the LSPV and the AtriClip, similar to the relationship between the Lasso catheter placed in the LSPV and the successful ablation site as demonstrated in panel D. The arrow denotes the AtriClip LAA closure device. **D:** The arrow shows the successful ablation site leading to flutter termination. The circular Lasso catheter is placed inside the LSPV. AP = anteroposterior.

Following successful repeat ablation, the patient reported marked improvement in his symptoms at a 3-month follow-up. He has remained off antiarrhythmics and free of atrial tachyarrhythmias, based on recent device interrogation in March 2017. After maintenance of sinus or atrially paced rhythm, we were able to minimize right ventricular pacing percentage to <5%. A repeat echocardiogram in March 2017 showed an increased ejection fraction of 45%.

Discussion

This case illustrates that epicardial application of the AtriClip LAA closure device in this patient appears to be associated with eletrophysiologic changes in the underlying atrial tissue, manifested as localized areas of low-amplitude, fractionated, and split atrial electrograms recorded endocardially. There were no previous surgical interventions or ablations performed in the area where complex atrial electrograms were discovered. It appears that these abnormal signals are indicative of areas of slow conduction that may facilitate formation of a reentrant circuit.[4] As shown in Figure 2B, a very prolonged pacing spike to P wave on the surface electrocardiogram was observed, suggestive of very slowed conduction out of the slow zone during entrainment mapping.[4] To our knowledge, this case appears to be the first report of a

potential proarrhythmic effect resulting from the use of AtriClip, an epicardial LAA closure device.

The ability to reliably entrain the tachycardia confirms the arrhythmia mechanism to be reentry driven.[3,4] The reentrant circuit underlying the atypical flutter does not appear to be a classic mitral isthmus–dependent type, even though the flutter termination site was over the anterior isthmus. As shown in Figure 2D, the ability to completely isolate the appendage while ablating the anterior mitral isthmus suggests that the previously placed traditional mitral isthmus line (from the left inferior pulmonary vein to the mitral annulus) was already blocked. However, it is clear that the arrhythmia continued after initial ablation of the inferior mitral isthmus, as shown in Figure 1. The extensive tissue remodeling detected in the atrial tissue under and adjacent to the AtriClip site appears to serve as a central core of conduction delay, sustaining an atypical flutter circuit (Figure 3A). This was supported by the ability to achieve concealed entrainment over the area and almost instantaneous termination of the arrhythmia as the ablation catheter reaches this site from the anterior mitral annulus during linear ablation (Figure 2D).

It is noteworthy that the entire procedure was performed without using fluoroscopy. Neither the operator nor any of

the electrophysiology personnel had radiation protection throughout the procedure. Unfortunately, this led to one of the limitations of the current case: We were not able to provide fluoroscopic images to demonstrate the direct anatomic relationship of the catheters and the AtriClip device. Yet, we feel there is ample evidence that the critical zone of the atypical flutter site was near the endocardial tissue underlying the AtriClip. This is supported by the fact that the patient had a preprocedural transesophageal echocardiogram and a computed tomography scan that clearly documented the anatomic location of the AtriClip between the left superior pulmonary vein and mitral annulus (Figure 3B and C). Moreover, the position of the AtriClip device was further confirmed by intraoperative ICE imaging. Finally, in this case, no surgical ablation or intervention other than the AtriClip device application was performed near the area (anterior mitral isthmus) where the complex electrograms, including signals with amplitude similar to baseline noise, were recorded. Based on these findings as well as the ablation points registered on the CARTO map, there is strong evidence that the ablation site resulting in termination of the atypical flutter was located near the endocardial tissue underlying the AtriClip (Figure 3C and D).

The complete isolation of the entire residual appendage area demonstrated in this case was neither a predesigned outcome nor required for arrhythmia termination. As shown in Figure 2D, the successful termination of the atypical flutter preceded the complete lateral wall isolation. This suggests that the mechanism of termination is a result of direct ablation of the critical zone in the flutter circuit, rather than a nonspecific consequence of complete isolation of a large area that contains the flutter circuit.

Conclusion

This case demonstrates that epicardial LAA exclusion using the AtriClip device may be associated with areas of electrophysiologic remodeling in the underlying atrial tissue, thereby creating a potential substrate for reentry-based arrhythmia. Careful mapping of these areas may lead to successful ablation for potential associated arrhythmias. The case also illustrates that even complex, postsurgical arrhythmias in patients with multi-lead devices can be potentially ablated using a fluoroless, efficient, and non–activation time mapping–based approach through meticulous entrainment mapping and attention to the left atrial anatomy.

Acknowledgments

References

1. Fumoto H, Gillinov AM, Ootaki Y, et al. A novel device for left atrial appendage exclusion: the third-generation atrial exclusion device. J Thorac Cardiovasc Surg 2008;136:1019–1027.
2. Afzal MR, Kanmanthareddy A, Earnest M, et al. Impact of left atrial appendage exclusion using an epicardial ligation system (LARIAT) on atrial fibrillation burden in patients with cardiac implantable electronic devices. Heart Rhythm 2015;12:52–59.
3. Waldo AL. Atrial flutter: entrainment characteristics. J Cardiovasc Electrophysiol 1997;8:337–352.
4. Olshansky B, Okumura K, Hess PG, Waldo AL. Demonstration of an area of slow conduction in human atrial flutter. J Am Coll Cardiol 1990;16:1639–1648.