Left atrial dissection as a trigger for recurrent atrial fibrillation

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Introduction

Ablation of atrial fibrillation (AF) consists of any of a combination of pulmonary vein (PV) isolation, targeting of non-PV triggers, linear ablation to target regions of substrate, and ablation of other presumed functional drivers of AF, such as complex fractionated electrograms and rotors. While the mainstay of ablation is PV isolation and much data exists to support the ablation of triggers identified via stimulation protocols, the value of empiric ablation of these other substrates is less clear.[1,2] However, the role of atrial substrate, whether created by atrial fibrosis due to primary atrial pathology or due to prior ablation, has been well recognized.[3,4] Such electrical substrate may also present as abnormalities of wall motion, given that electrical activation drives mechanical contraction.[5] Here we describe a case of an atrial wall motion abnormality that correlated with a region of electrical abnormality that served as a driver for AF.

KEY TEACHING POINTS

- Left atrial dissections are a rare but potential complication that can be seen after prior pulmonary vein isolation.
- Regional structural abnormalities such as left atrial dissection may serve as regional drivers/triggers for atrial fibrillation.
- Attention to wall motion during echocardiography and regional electrical abnormalities may help the electrophysiologist identify potential atypical causes of atrial fibrillation.

Case report

A 64-year-old man with a longstanding history of paroxysmal AF presented with shortness of breath and exertional dyspnea associated with his AF paroxysms. He was initially diagnosed with AF 17 years prior at the time of gastric bypass. He subsequently had multiple emergency department visits for rate control and electrical cardioversion to restore sinus rhythm and continued to have breakthrough episodes despite the use of sotalol.

Transthoracic echocardiogram showed a preserved ejection fraction and left atrial volume index of 41 cc/m^2. Preprocedural computed tomography (CT) imaging for his initial ablation demonstrated 2 left and 3 right PVs but no other structural abnormalities. Left and right wide area circumferential ablation had been performed one year prior to presentation with reported successful isolation of all PVs. This included wide-area circumferential ablation of the left and right veins, without carina lesions. Isolation was achieved with a single lesion set in both sets of veins using radiofrequency energy with no additional lines or targeted ablation of presumed substrate. Voltage mapping at that time showed normal voltage at baseline, but no repeat voltage mapping was done post-isolation. Postisolation testing including programmed stimulation, burst pacing, and isoproterenol infusion, which did not reveal any inducible AF or organized atrial arrhythmias. The prior ablation was done with CARTO3 (Biosense Webster, Irvine, CA) with a PentaRay catheter used for mapping (Biosense Webster, Irvine, CA). Because of continued paroxysms of symptomatic AF after prior ablation, he was taken back to the electrophysiology lab for repeat ablation.

Intracardiac echocardiography (ICE) was used to assess baseline status of the cardiac chambers and pericardial space. A discrete region with asynchronous contraction compared

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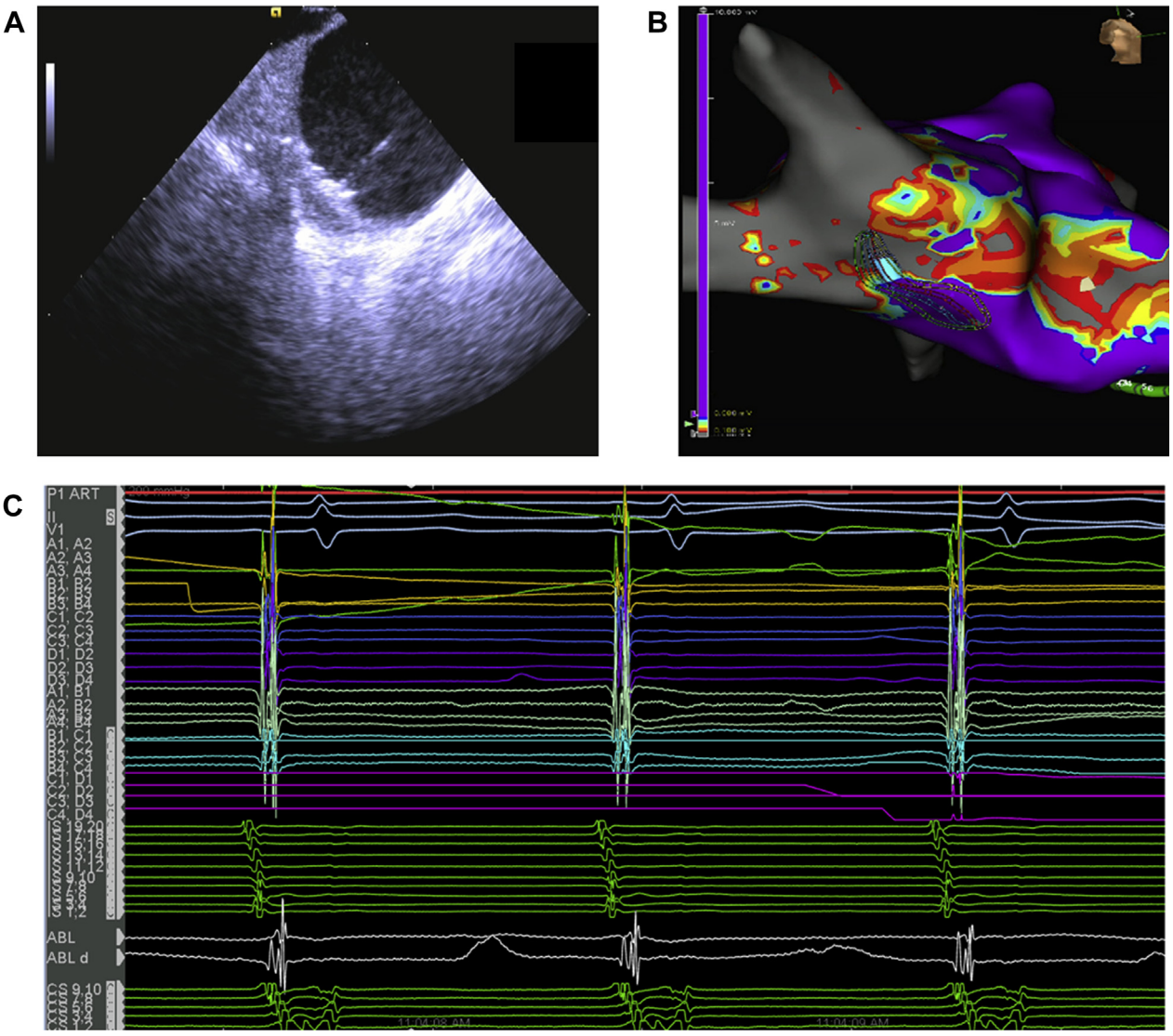

Figure 1 Local region of dyssynchronous contraction. **A:** Location of the multipolar catheter on intracardiac echocardiography; **B:** the corresponding location on the voltage map; **C:** the signals in this region during sinus rhythm. Pole A, B, C, and D correspond with the local signals seen on the multipolar catheter.

to the rest of the left atrium (LA) was noted (Supplementary Video 1, A and B). When compared with ICE images obtained during the index procedure, the region of abnormal contraction was not previously seen. We performed a voltage map of the PVs and LA using the HD Grid catheter and EnSite Precision mapping system (Abbott, St. Paul, MN). All PVs were found to be isolated. The mapping catheter was placed on the region of asynchronous contraction under visualization with ICE, which demonstrated 2 tissue planes. Figure 1 shows the multipolar mapping catheter over the region noted on ICE, which demonstrated normal voltage, though local potentials were split and thus abnormal. During coronary sinus pacing, this region demonstrated local fibrillation with sinus in the rest of the atria (Figure 2, top panel; Supplementary Video 2A). Longer periods of pacing would eventually result in global AF, preceded by local fibrillation (Figure 2, bottom panel; Supplementary Video 2B). There were also periods of transient AF seen in this local region spontaneously (Supplementary Video 3), with differences seen in local activation during periods of transient local AF and when the atrium was generally in AF (Supplementary Video 4). Comparison of the sinus bipolar voltage map to the CT image showed no evidence of accessory PV, remnant PV, or LA diverticulum.

In sinus rhythm, we then performed endocardial ablation along this region of the LA (Figure 3). The entire region was ablated in order to affect the split atrial electrograms. On ICE, we noted loss of contractility, increased echogenicity of this area, and no flow between the presumed 2 tissue planes.

After ablation, the patient was no longer inducible for AF, including with use of atrial burst pacing; atrial single, double, and triple extrastimuli from 2 locations; and isoproterenol up to 15 mcg/kg/min. The patient had 1 episode of AF during an infectious illness 1 month post-ablation, but has been free of AF over 1 year of follow-up, off of antiarrhythmic drugs.

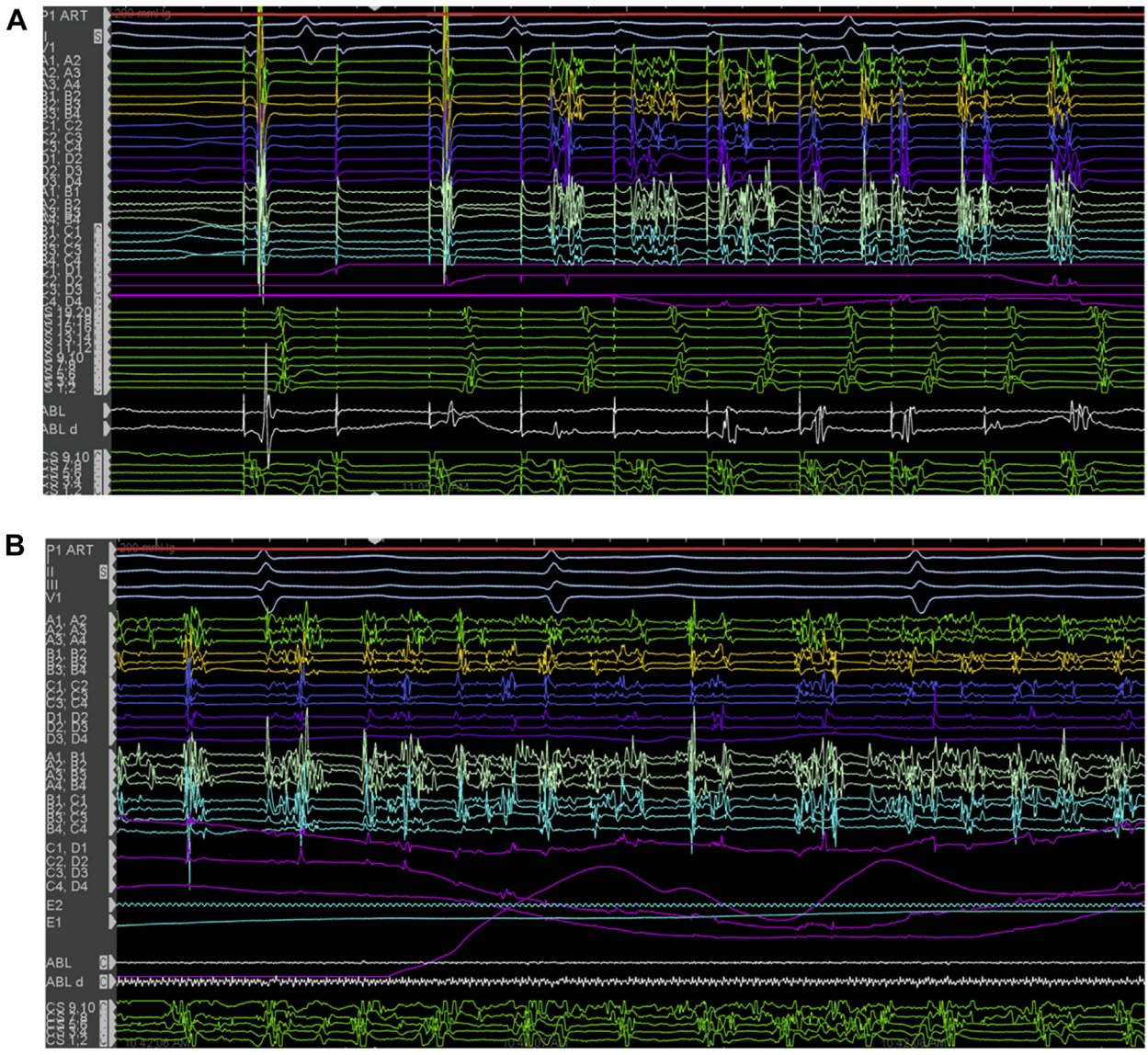

Figure 2 Effect of coronary sinus (CS) pacing on generation of atrial fibrillation (AF) signals. Shown are examples of the signals on the multipolar catheter (A, B, C, D labeled electrograms) during pacing of the CS. **A:** Disorganization of the local signals in the region of interest shown in Figure 1, though the isthmus labeled catheter (along the lateral right atrium) and CS labeled catheter (in the coroanry sinus) show an organized rhythm. **B:** Sustained AF throughout the heart. For review of induction of each see Supplementary Video 2, A and B.

Discussion

This case illustrates a rare cause for recurrence of AF following prior ablation and approach to intervention. In general, recurrence of AF post-ablation can occur from several known causes. The most common culprit is reconnection of the PVs to the LA. In addition, AF can be triggered from non-PV triggers such as the coronary sinus, left atrial appendage, vein of Marshall, superior vena cava, and posterior LA.[6] Here we demonstrated a region of dyssynchronous contraction external to the PVs associated with localized voltage abnormalities that was serving as a driver for AF.

There are several potential mechanisms by which this atrial region may have served as an AF driver. For example, tissue heterogeneity and fibrosis/scar from the prior ablation may have created an electrophysiologic milieu for functional reentry to occur.[7] Owing to the proximity of this region to the PV-LA junction, there could also be proximate epicardial ganglionated plexi and/or abnormalities in local innervation post-ablation leading to a difference in local arrhythmogenic potential.[8] While we cannot definitively rule out these possibilities, it is difficult to rectify these as the culprits, as this was a re-do ablation and the problem did not manifest during the first procedure. The only way to support the premise is if one were to consider nerve sprouting and resulting hyperinnervation of the border zone of the prior ablated region or progression in the electrophysiologic milieu after prior ablation as potential mechanisms.

Another reason for the arrhythmogenic potential of this region may be local structural anomalies. For example, aberration in normal PV architecture could provide the anatomical basis for focal sources of AF triggers. In a prior study, an accessory PV was reported in 17% of 188 patients prior to

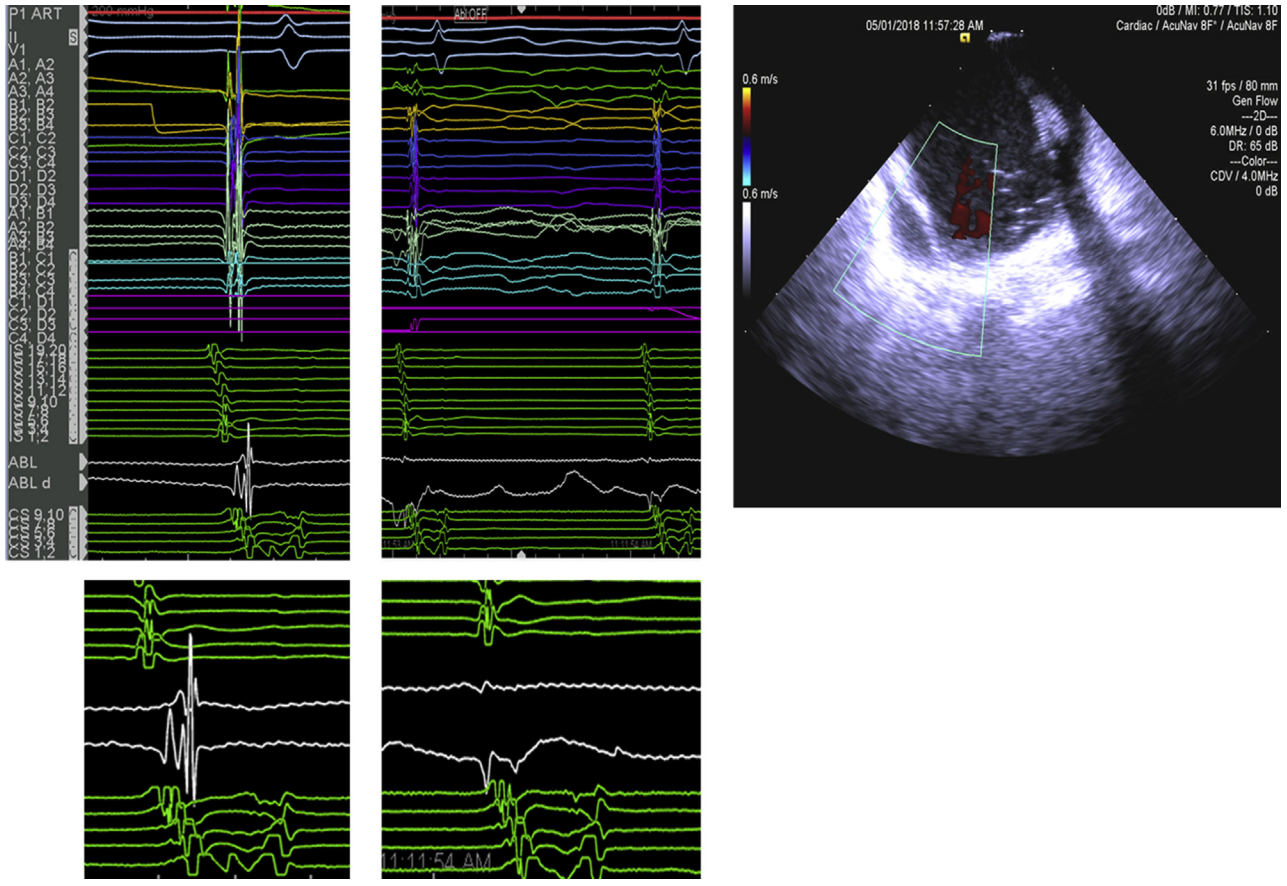

Figure 3 Ablation of endocardial surface to eliminate complex, near-field potentials. Ablation was performed along the endocardial surface to eliminate the complex signals. Left and middle panels show pre- and post-ablation, respectively (below: noted inset of atrial electrogram on ablation catheter pre- and post-ablation). Right panel demonstrates the echogenicity of the ablated tissue.

ablation for AF.[9] These should be included within the ablation set when identified. There have also been reports of a remnant PV as a cause for recurrent AF.[10–12] Finally, left atrial diverticula, defined as thin-walled outpouchings of the left atrial wall that communicate with the left atrial lumen, are commonly found on CT scan, with a prevalence of over 40%.[13] It is plausible that such a focal region of atrial tissue could serve as a trigger for AF. However, we did not note any of these on preprocedural CT scanning or with the use of intraprocedural ICE.

A final cause that can be hypothesized as being the principal physiology underlying the region's arrhythmogenic potential is a focal dissection of the left atrial wall. A left atrial dissection is a false, blood-filled cavity in the LA that forms a new chamber with or without communication into the true LA. It is exceedingly rare and most commonly associated with complications from mitral valve surgery.[14] However, it can also occur from catheter ablation, albeit uncommonly.[14] Left atrial dissection can be missed if the patient remains hemodynamically stable during ablation. This can result in atrial remodeling over time.[15] It is plausible that this may therefore create a potential substrate for AF.

We hypothesize that the AF trigger seen in our study likely resulted from prior ablation, which caused an iatrogenic left atrial dissection, thereby creating a localized region of fibrillating tissue with the ability to trigger global AF. There are several reasons for us to conclude this. The first is that this "trigger" for AF was found well after the first ablation and ICE suggested a second tissue plane with associated dyssynchronous contraction. While it is possible that this region was always arrhythmogenic, even at the time of the first ablation, we can neither prove nor disprove this with the available data. The lack of inducibility at the end of the first procedure, however, argues against this case. Furthermore, the success seen with relatively targeted ablation to this area suggests the region served as the principal arrhythmogenic substrate for AF at least subsequent to the first ablation.

Conclusions

Our case demonstrates that a left atrial dissection following catheter ablation can serve as a proarrhythmic substrate and trigger of AF. It is important to be aware of iatrogenic causes from prior ablation that can lead to damage to the integrity and synchrony of the left atrial wall and can serve as potential "non-PV" triggers for recurrent AF. This case also highlights the utility of ICE in evaluating the electroanatomical-physiologic substrate for AF to enhance successful re-do procedures.

Appendix A

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.hrcr.2020.02.011.

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