

# Catheter ablation of atrial arrhythmias to prevent ventricular tachycardia in a patient with mobile left ventricular thrombus



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## Introduction

Atrial fibrillation (AF) is a leading cause of inappropriate shocks in patients with an implantable cardioverter-defibrillator (ICD), but can also trigger episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF), leading to appropriate ICD therapies.<sup>1</sup> Although many antiarrhythmic drugs have effects on both atrial and ventricular arrhythmias, catheter ablation is an effective therapy for both AF and VT, especially when refractory to antiarrhythmics. Left ventricular (LV) thrombus is an important contraindication to endocardial VT ablation.<sup>2</sup>

## Case report

A 70-year-old man, with a long history of coronary artery disease, remote coronary artery bypass grafting, asbestosis, paroxysmal AF, and a secondary prevention ICD implanted initially in 1993 for syncope with severe LV dysfunction, presented with recurrent appropriate ICD shocks for VT. These were preceded by days to weeks of symptoms of palpitations, shortness of breath, and reduced exercise tolerance. He had been treated with sotalol and mexiletine, although the dose of the former was limited to 80 mg twice daily owing to chronic kidney disease and sinus bradycardia, for which he had undergone upgrade to a dual-chamber ICD. Despite this, he had received 3 appropriate shocks in the last year. He also had a history of LV thrombus treated with warfarin, which had been stopped 6 months earlier after an echocardiogram demonstrated resolution. However, repeat echocardiography had shown inferior LV akinesis with apical dyskinesia and an ejection fraction of approximately 40%, with

## KEY TEACHING POINTS

- Atrial fibrillation predisposes to appropriate, as well as inappropriate, implantable cardioverter-defibrillator shocks in patients with structural heart disease.
- Mechanisms by which atrial fibrillation causes ventricular arrhythmias likely include production of short-long-short sequences.
- Atrial arrhythmias provide a therapeutic target to reduce ventricular arrhythmias in patients with structural heart disease.
- Left ventricular thrombus, especially fresh and protruding thrombus, is an important relative contraindication to endocardial ventricular tachycardia ablation.

reappearance of a mobile, protruding apical LV thrombus, and warfarin was restarted (Figure 1).

He complained of New York Heart Association class IIIa heart failure symptoms, including shortness of breath when showering. In addition to the above medications, the patient was receiving maximum tolerated guideline-directed medical therapy.

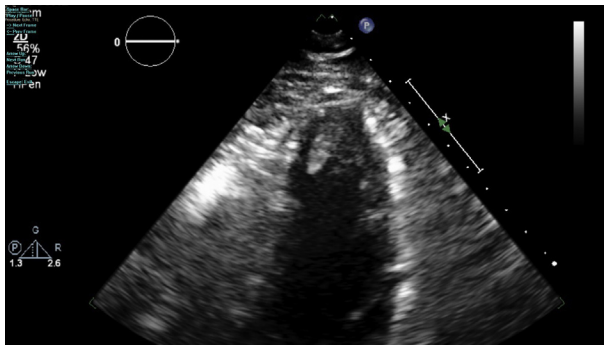
Twelve-lead electrocardiogram showed sinus rhythm with inferior Q waves and occasional premature ventricular contractions from the basal anterolateral LV. Interrogation of his dual-chamber ICD showed that VT events occurred solely during episodes of AF (Figure 2A), but intracardiac electrograms confirmed appropriate therapies for VT, and not inappropriate therapies for rapidly conducted AF (Figure 3). This was also suggested on review of records of his prior single-chamber device, which showed that VT had occurred at times when his heart rate had been elevated for the preceding several days. AF burden was 4.2% despite sotalol therapy.

Owing to the presence of mobile, protruding LV thrombus, endocardial catheter ablation was contraindicated; instead, catheter ablation of AF was pursued to eliminate this

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**Figure 1** Transthoracic echocardiogram image from the apical 4-chamber view showing a large protruding left ventricular thrombus.

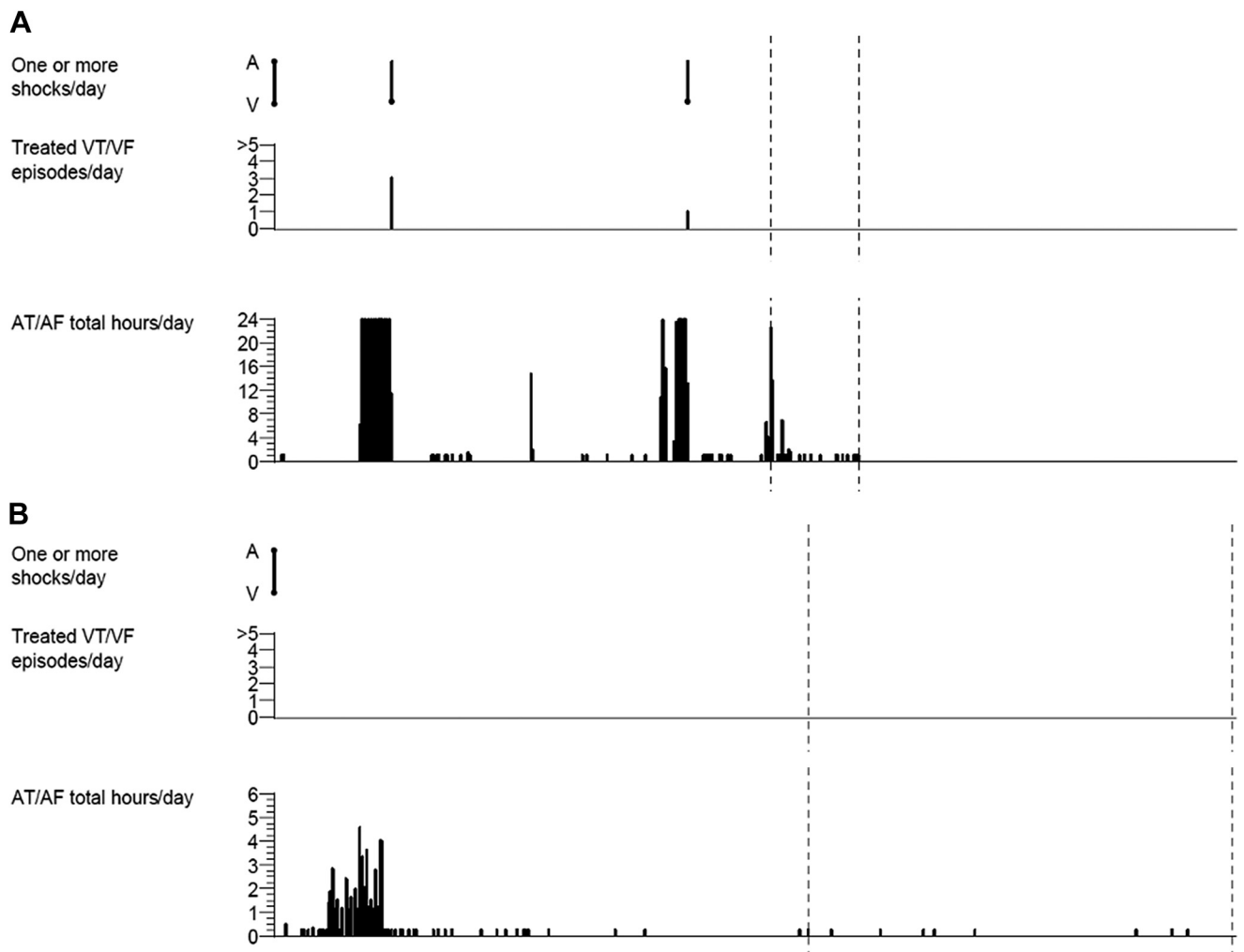
trigger. Intracardiac echocardiography confirmed the presence of LV thrombus. Although the patient presented in atrial paced rhythm, a single atrial extrastimulus from the high right atrium induced atypical atrial flutter. Via transseptal puncture, activation mapping of the left atrium (LA) using a multi-electrode catheter (PentaRay; Biosense Webster, Diamond Bar, CA) and electroanatomic mapping system (CARTO 3;

Biosense Webster) suggested perimitral flutter. In sinus rhythm, bipolar voltage mapping revealed extensive LA scar, encompassing the septum and posterior wall, with the inferior and lateral LA and the LA appendage having normal voltage ( $>0.4$  mV). In addition, roof-dependent flutter, and a focal atrial tachycardia from the septal aspect of the mitral annulus, were induced and ablated. The final lesion set included pulmonary vein isolation, an anteroseptal mitral isthmus line through an area of scar, and box isolation of the posterior wall (Supplemental Figure).

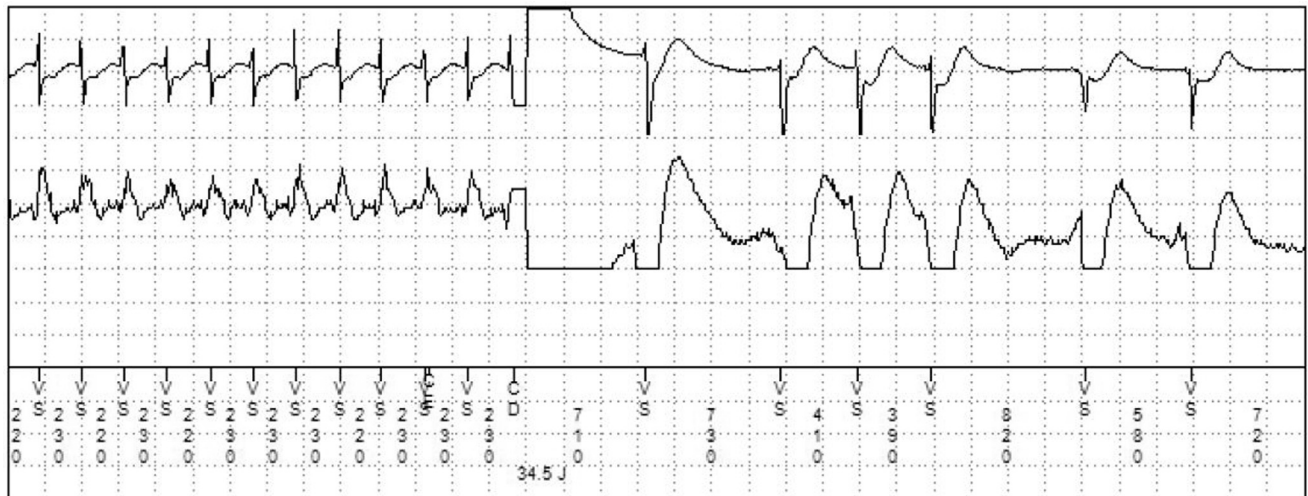
At follow-up at over 1 year postablation, with unchanged medical therapy, ICD interrogation revealed no further VT or ICD therapies and an AF burden of  $<0.1\%$  (Figure 2B). The patient reported freedom from symptoms of palpitation and reduced anxiety regarding ICD shocks, but unchanged exercise tolerance.

## Discussion

AF is common in patients with ICDs, with episodes lasting more than 24 hours occurring in 7.1% of dual-chamber ICD recipients without a prior history of atrial arrhythmia



**Figure 2** **A:** Ventricular tachycardia (VT) events occurred during episodes of atrial fibrillation (AF). **B:** Implantable cardioverter-defibrillator (ICD, Evera MRI XT DR; Medtronic, Minneapolis, MN) interrogation over 1 year post catheter ablation of AF shows no VT episodes or ICD therapies, and, apart from during the blanking period, a low AF burden of  $<0.1\%$ . The time scales on the x-axes represent 8 months (A) and 14 months (B). AT = atrial tachycardia; VF = ventricular fibrillation.



**Figure 3** Single-chamber implantable cardioverter-defibrillator (ICD, Protecta XT VR; Medtronic, Minneapolis, MN) electrogram showing ventricular tachycardia treated by an ICD shock followed by a slower, irregular rhythm consistent with atrial fibrillation. Top: right ventricle (RV) tip to ring; middle: RV coil to can; bottom: marker channel.

during follow-up of  $22 \pm 9$  months in a recent study,<sup>3</sup> and a prevalence of 20% in a contemporaneous cross-sectional study.<sup>4</sup> AF is also the most common cause of inappropriate ICD shocks, when it is rapidly conducted to the ventricles.<sup>5</sup> Perhaps less well recognized, however, is that the risk of VT/VF and therefore appropriate ICD therapies also rises during episodes of atrial arrhythmia. Early investigations of stored electrograms from single-chamber ICDs suggested that 6%–10% of VT/VF episodes were preceded by AF.<sup>6,7</sup> An analysis of adjudicated dual-chamber ICD electrograms from 537 patients found that AF preceded VT/VF in 8.6% of VT/VF episodes, within 1 hour half of the time.<sup>1</sup> In addition, continued AF after termination of VT/VF was associated with a much shorter time until the next VT/VF episode vs cases where AF terminated with the ICD shock, suggesting a role for AF in facilitating initiation of ventricular arrhythmia. Another prospective study found that AF was an independent predictor of appropriate (as well as inappropriate) ICD shocks, often preceded by short-long-short sequences.<sup>8</sup> These findings have been reproduced by several other investigators.<sup>9</sup> Effective rhythm control of AF therefore presents a therapeutic opportunity for patients with VT. While many of the antiarrhythmic drugs used for AF will also act to reduce episodes of VT, when refractory, catheter ablation may provide an effective option.

There may be several mechanisms by which AF can trigger VT/VF. Firstly, irregular rhythms such as AF produce long-short sequences, which may induce reentry.<sup>8</sup> This may be particularly important in patients with ICDs, in whom ventricular pacing can result, and is associated with an increased risk of appropriate ICD shocks.<sup>10</sup> Irregularity is associated with QTc prolongation and increased QT dispersion compared to a regular rhythm, suggesting that irregularity itself leads to electrical remodeling, which predisposes to ventricular arrhythmia.<sup>11</sup> The increased risk of ventricular arrhythmia in patients with AF has been found to be enhanced by the presence of QRS and QT prolongation,

further suggesting that both abnormal depolarization and repolarization play a role in these events.<sup>9</sup> Other possible mechanisms include hemodynamic impairment, increased sympathetic tone, rate-related ischemia, and tachycardia-induced cardiomyopathy; further study is needed to clarify the role of each.

LV thrombus is generally regarded as a contraindication to endocardial ablation.<sup>2</sup> Successful ablation has been reported in a very limited number of patients with old, laminated thrombus, although not without a risk of stroke.<sup>12,13</sup> This patient, however, had a relatively new, recurrent, and mobile thrombus that protruded into the LV cavity, which would likely lead to a higher risk of embolism. Alternatively, epicardial-only ablation has been reported in a small number of patients, including cases with LV thrombus.<sup>14</sup> This patient had a history of coronary artery bypass grafting, which makes epicardial access particularly challenging, with a success rate of 60% in 1 study.<sup>15</sup> Therefore, ablation of atrial arrhythmias in this case was a feasible alternative to VT ablation.

## Conclusion

AF acts as a trigger of VT/VF in patients with ICDs. In these patients, AF presents a therapeutic opportunity, as illustrated by this case, in which ablation of atrial arrhythmias was associated with control of drug-refractory VT.

## Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2021.07.005>.

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