Intramural anterolateral mitral annular idiopathic ventricular tachycardia successfully ablated from the atrium

Tawseef Dar, MD, Bharath Yarlagadda, MD, Valay Parikh, MD,
Y. Madhu Reddy, MD, FHRS, Seth H. Sheldon, MD, FHRS

From the Department of Cardiovascular Medicine, The University of Kansas Health System, Kansas City, Kansas.

Introduction

Idiopathic ventricular arrhythmias (VAs) commonly originate from specific anatomic areas, including the ventricular outflow tracts, papillary muscles, fascicular system, mitral annulus, and tricuspid annulus.[1] Around 5% of idiopathic VA are mitral annular (MA) in origin.[2] Catheter ablation (CA) is an effective treatment option for symptomatic VAs, especially when refractory to medical therapy.[3] Advanced techniques and equipment and a better understanding of arrhythmia mechanisms have resulted in high success rates with CA of idiopathic VAs.[4] Septal and left ventricular intramural VAs, however, remain a challenge to successfully eliminate owing to difficulty achieving the necessary depth to ablate the focus. Numerous approaches have been reported, with variable success, including bipolar ablation,[5] irrigated unipolar radiofrequency CA from the endocardial and epicardial sites,[6] utilization of a needle catheter,[7] selective coronary venous or arterial ethanol injection,[8] stereotactic noninvasive ablation,[9] and delivery of high-power radiofrequency ablation with half-normal saline irrigation.[10] Here, we report a case of intramural MA ventricular tachycardia (VT) refractory to both endocardial and epicardial RF ablation, successfully ablated from the atrial aspect of the mitral annulus.

Case report

A 22-year-old woman with history of hypothyroidism, obesity, and symptomatic premature ventricular contractions (PVCs)/VT was referred for management of incessant VAs.

KEY TEACHING POINTS

- Mapping and ablation in adjacent structures is often helpful for intramural ventricular arrhythmias (VAs).

- The atrial aspect of the mitral annulus can be closer in proximity to the source of intramural mitral annular (MA) VAs than the subannular endocardial left ventricle or epicardial left ventricle approached subxiphoid or via the coronary sinus.

- Ablation at the atrial aspect of the MA can be effective for intramural MA VAs.

She reported a 10-year history of progressively worsening palpitations, shortness of breath, and fatigue. These symptoms had a sudden onset and offset and occurred at least a few times a month. Reported triggers included stress and menstruation. An event monitor demonstrated symptomatic episodes of nonsustained monomorphic VT with similar morphology to her PVCs, with rates of 110–120 beats per minute and up to 16 beats in duration (Figure 1A). An ablation was attempted a year prior at an outside medical center, although sedation suppressed the VAs and no mapping/ablation was performed. Medications, including metoprolol, flecainide, sotalol, mexiletine, and lidocaine, were subsequently utilized without effective suppression of VAs.

The 12-lead electrocardiogram of VAs showed a right bundle branch block morphology with positive precordial concordance and right inferior axis (Figure 1B) suggestive of anterolateral MA exit site. A slurred initial onset with maximum deflection index of 0.59 and qS in lead I was suggestive of epicardial origin. Cardiac magnetic resonance imaging showed a normal left ventricle (LV) size and function without delayed gadolinium enhancement. Cardiac positron emission tomography scan was equivocal for myocarditis with mean blood pool standardized uptake value of 2.9 and maximum myocardial standardized uptake value
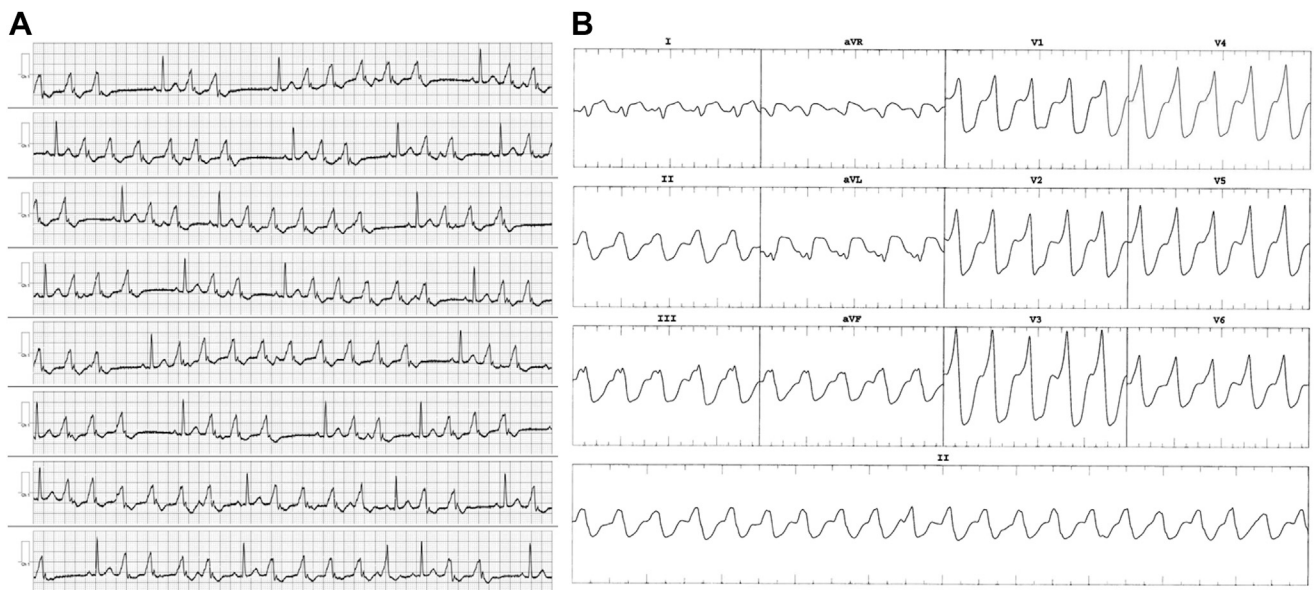
A

B

Figure 1   Event monitor tracing and 12-lead electrocardiogram of ventricular tachycardia. **A:** Single-channel event monitor tracing demonstrates sinus rhythm with frequent ventricular ectopy including runs of nonsustained ventricular tachycardia. **B:** A 12-lead electrocardiogram of ventricular tachycardia with right bundle branch block, positive precordial concordance, and right inferior axis suggestive of anterolateral mitral annular exit site.

of 4.2. She was treated for subacute myocarditis with prednisone at up to 30 mg daily for 2 months without improvement in symptoms or burden of VAs. She went on to have incessant sustained VT at an outside hospital and transferred for electrophysiology study and potential ablation. She was treated with intravenous lidocaine with incomplete suppression of VAs.

Lidocaine was discontinued 4 hours before the procedure. She had frequent PVCs and nonsustained VTs upon arrival to the lab with morphology similar to her clinical VAs. Propofol was used to achieve moderate sedation. She continued to have frequent VAs despite sedation. Left ventricular geometry was created using the CARTO-SOUND module in conjunction with the CARTO 3-dimensional mapping system (Biosense Webster, Diamond Bar, CA). Transseptal puncture was performed and anticoagulation provided with unfractionated heparin to maintain an activated clotting time between 350 and 400 seconds. An Agilis (Abbott Medical, Chicago, IL) sheath was utilized in conjunction with a Biosense Webster SmartTouch D-F curve ablation catheter (Diamond Bar, CA) for mapping and ablation. Activation mapping of the PVCs and nonsustained VT in the LV via a transseptal and later via a retrograde aortic approach was only 15 ms presystolic with a small R wave on the unipolar electrogram (Figure 2A) with earliest activation at the anterolateral mitral annulus. Ablation was performed from the endocardial LV in this location at up to 50 W without suppression of VAs. Rather, the incessant nonsustained VT became sustained, hemodynamically stable VT (cycle length 400 ms).

Mapping was then performed in the adjacent coronary sinus (CS). Ventricular activation remained far-field in the CS and was 28 ms presystolic during VT (Figure 2B). Although the activation was earlier in the CS than the endocardial LV, ablation performed within the CS between 15 and 25 W with

high-flow irrigation and careful impedance monitoring did not result in termination of the VT.

Earlier in the case, from a transseptal approach, very early far-field ventricular activation was seen at the atrial aspect of the mitral annulus. It was elected to return to this site. Despite a large near-field atrial electrogram, the far-field ventricular electrogram was 44 ms presystolic and the unipolar ventricular electrogram all negative (Figure 2C). Ablation was performed at 30 W, which terminated the VT within 2 seconds (Supplemental Figure 1). Reinforcement lesions were delivered at this site at up to 40 W. No further VAs occurred or were inducible. Isoproterenol was provided at up to 4 mcg/min and sedation turned off without ectopy. Ventricular stimulation was performed from 2 right ventricular sites with 4 extrastimuli down to 200 ms at drive trains of 350, 400, and 550 ms with no inducible VT or ventricular fibrillation. We waited over an hour and there was no further ventricular ectopy. Given the absence of inducible VAs postablation, the hemodynamically stable nature of the VAs, and no evidence of scar on cardiac magnetic resonance imaging, implantable cardioverter-defibrillator implantation was not recommended.

At clinical follow-up 10 months later, she had no further symptoms and 7-day event monitoring showed no VT.

Discussion

Intramural VAs have a higher failure rate with ablation than nonintramural VAs.[11] This case demonstrates the potential utility of mapping and ablation at the atrial aspect of the mitral annulus for intramural MA VA.

Mapping and ablation within adjacent structures is often necessary and successful. Ablation from the right atrium can be successful for VAs arising from the left posterior-superior process of the LV.[12] Epicardial VAs are often

A Endocardial LV   B Epicardial (CS)   C Left atrium

Map proximal

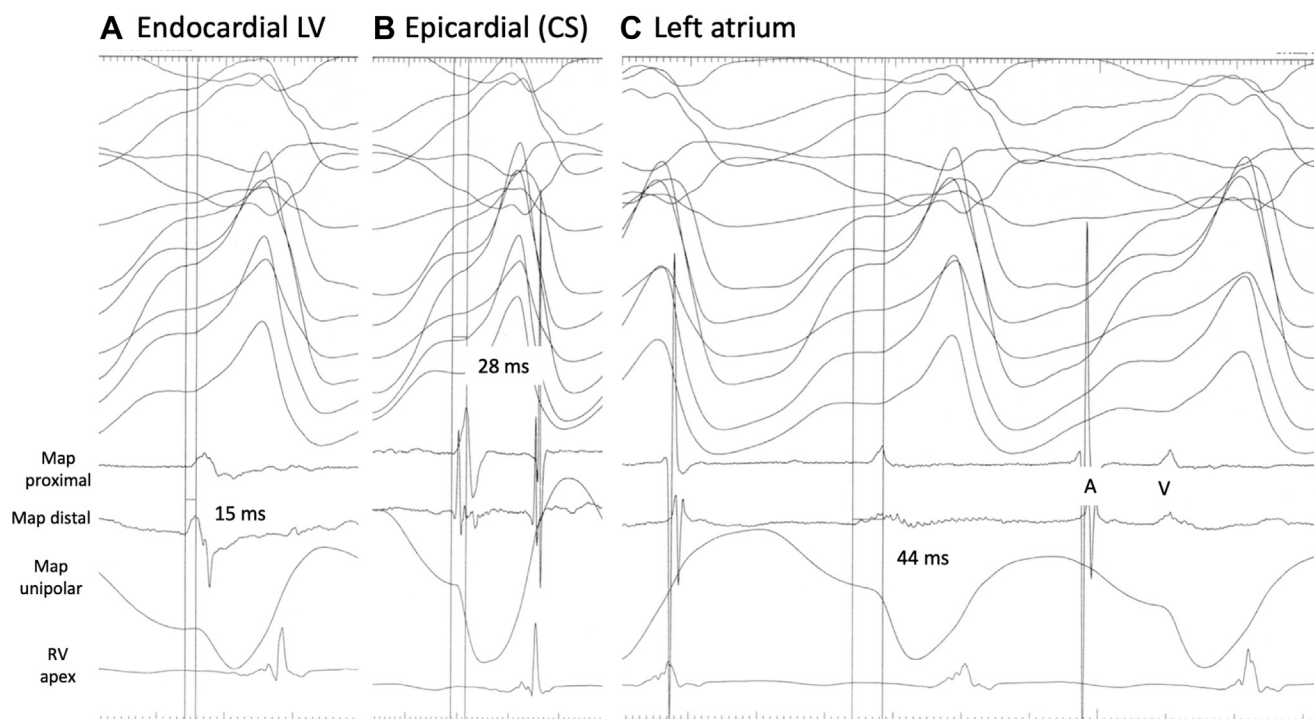Map distal — 15 ms

Map unipolar

RV apex

28 ms

44 ms

A   V

Figure 2 Mapping signals at the endocardial left ventricle (LV), adjacent epicardium via the coronary sinus (CS), and the left atrium. **A:** The earliest endocardial activation in the left ventricle was 15 ms presystolic with a small R wave on the unipolar electrogram. **B:** The earliest epicardial activation from the distal CS was 28 ms presystolic. **C:** Far-field activation at the atrial aspect of the mitral annulus was much earlier at 44 ms pre-systolic with an all negative unipolar electrogram. Note the labeled ventricular (V) and atrial (A) electrograms. Ventricular-atrial dissociation was present. The atrial electrogram was near-field. Ablation at site C terminated the ventricular tachycardia within 2 seconds. RV = right ventricular.

mapped and ablated from the coronary venous system (CS/middle cardiac vein).[13] Mapping and ablation in the left atrial appendage can help eliminate accessory pathways as well as VAs with epicardial origin, including the LV summit.[14] Furthermore, ablation in the left atrium for VT with structural heart disease (prior mitral valve repair) has been described.[15] We report successful ablation in the atria to eliminate idiopathic intramural MA VAs.

Figure 3 demonstrates the anatomic proximity of the endocardial LV, atrial aspect of the mitral valve, and epicardium with a hypothetical intramural origin of VA. The atrial aspect of the mitral annulus can be in closer proximity than the endocardial LV or epicardium (via either the CS or subxiphoid epicardial approach). At the true annulus, it can be difficult to ablate successfully from a subxiphoid epicardial approach owing to the left atrial appendage and overlying
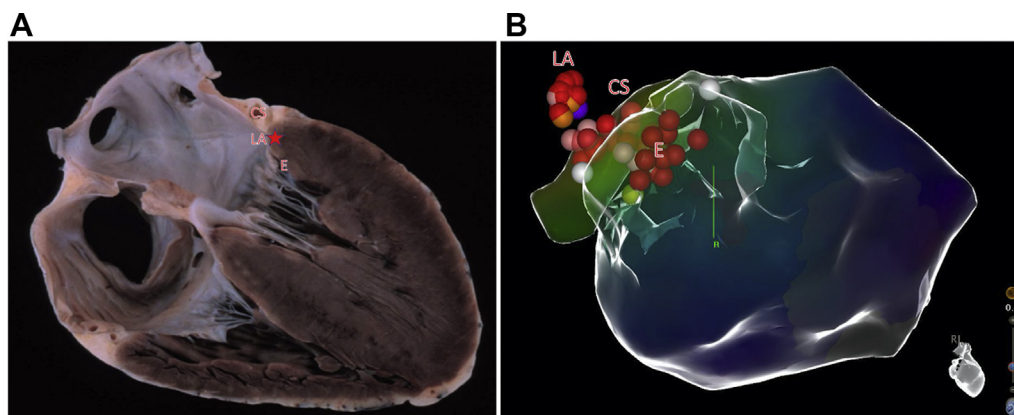
A   B

Figure 3 Mitral annular anatomy and proximity to intramural ventricular arrhythmia site of origin. **A:** Image demonstrates the relationship between the *star*, which represents the focus of the mitral annular ventricular arrhythmia, and adjacent sites. Site LA, the atrial aspect of the annulus, is in closer proximity to the site of origin than site E (endocardial subvalvular left ventricle) or site CS (epicardial coronary sinus approach). Note the pericardial fat overlying the epicardial aspect of the annulus that would make this region difficult to ablate from a subxiphoid epicardial access. **B:** CARTO map (Biosense Webster, Diamond Bar, CA) demonstrating red ablation lesions in the endocardial left atrium (site E), epicardial coronary sinus (site CS), and left atrium (LA). The *purple dot* represents the successful site. (Used with permission from J Cardiovasc Electrophysiol 2010;21:245–254.)

fat at the annulus. Ablation on the atrial aspect of the mitral annulus is analogous to ablation at the atrial insertion of a concealed accessory pathway, although ablation must be performed with sufficient power and duration to reach the deeper ventricular tissue. When ablating intramural VT from the atrial aspect of the mitral valve, one must be mindful of the ablation power and duration, given the risk of atrial perforation. Furthermore, care must be taken not to damage surrounding structures such as the mitral valve apparatus or coronary vessels. In our patient with intramural source of VA, a far-field ventricular electrogram in the atrium was the site of earliest activation and successful site of ablation.

Alternative approaches could have been utilized in this scenario and have been described with intramural VAs. The use of half-normal saline irrigation with ablation may have facilitated a deeper ablation lesion, resulting in success from an endocardial LV approach.[10] Simultaneous unipolar or bipolar ablation from the endocardium and epicardium can be helpful with intramural VAs.[5,6] A novel, investigational infusion needle catheter could also be utilized with a deep site of origin.[7] A hybrid or surgical approach could also be utilized for intramural VAs. Transcoronary venous or arterial ethanol ablation can be effective for intramural VA, especially at the interventricular septum.[8] Fortunately, these alternative approaches were not necessary in this case, as atrial ablation was successful in eliminating this patient's VAs.

Conclusion
This case demonstrates the importance of mapping and ablation at the atrial aspect of the mitral annulus for refractory intramural MA VAs. The safety and effectiveness of this approach warrants further investigation.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2019.04.002.

References

1. Sheldon SH, Gard JJ, Asirvatham SJ. Premature ventricular contractions and non-sustained ventricular tachycardia: association with sudden cardiac death, risk stratification, and management strategies. Indian Pacing Electrophysiol J 2010; 10:357–371.
2. Tada H, Ito S, Naito S, et al. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmias. J Am Coll Cardiol 2005;45:877–886.
3. Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Heart Rhythm 2018;15:e73–e189.
4. Latchamsetty R, Yokokawa M, Morady F, et al. Multicenter outcomes for catheter ablation of idiopathic premature ventricular complexes. JACC Clin Electrophysiol 2015;1:116–123.
5. Koruth JS, Dukkipati S, Miller MA, Neuzil P, d'Avila A, Reddy VY. Bipolar irrigated radiofrequency ablation: a therapeutic option for refractory intramural atrial and ventricular tachycardia circuits. Heart Rhythm 2012; 9:1932–1941.
6. Iyer V, Gambhir A, Desai SP, Garan H, Whang W. Successful simultaneous unipolar radiofrequency ablation of septal ventricular tachycardia using 2 ablation catheters. Heart Rhythm 2014;11:710–713.
7. Sapp JL, Beeckler C, Pike R, et al. Initial human feasibility of infusion needle catheter ablation for refractory ventricular tachycardia. Circulation 2013; 128:2289–2295.
8. Sacher F, Sobieszczyk P, Tedrow U, et al. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. Heart Rhythm 2008; 5:62–68.
9. Cuculich PS, Schill MR, Kashani R, et al. Noninvasive cardiac radiation for ablation of ventricular tachycardia. N Engl J Med 2017;377:2325–2336.
10. Nguyen DT, Tzou WS, Sandhu A, et al. Prospective multicenter experience with cooled radiofrequency ablation using high impedance irrigant to target deep myocardial substrate refractory to standard ablation. JACC Clin Electrophysiol 2018;4:1176–1185.
11. Baser K, Bas HD, Belardi D, et al. Predictors of outcome after catheter ablation of premature ventricular complexes. J Cardiovasc Electrophysiol 2014;25:597–601.
12. Santangeli P, Hutchinson MD, Supple GE, Callans DJ, Marchlinski FE, Garcia FC. Right atrial approach for ablation of ventricular arrhythmias arising from the left posterior-superior process of the left ventricle. Circ Arrhythm Electrophysiol 2016;9:e004048.
13. Yokokawa M, Latchamsetty R, Good E, et al. Ablation of epicardial ventricular arrhythmias from nonepicardial sites. Heart Rhythm 2011;8:1525–1529.
14. Di Biase L, Schweikert RA, Saliba WI, et al. Left atrial appendage tip: an unusual site of successful ablation after failed endocardial and epicardial mapping and ablation. J Cardiovasc Electrophysiol 2010;21:203–206.
15. Itoh T, Doppalapudi H, Yamada T. Epicardial ventricular tachycardia successfully ablated from the left atrium in a case with a prior mitral valve repair. Europace 2017;19:1356.