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Authors

Darden, Douglas
Hsu, Jonathan C
Shah, Sanjay
et al.

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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Ventricular Tachycardia Storm Originating From Moderator Band Requiring Extracorporeal Membrane Oxygenation

Douglas Darden, MD, Jonathan C. Hsu, MD, MAS, Sanjay Shah, MD, Kurt Hoffmayer, MD, PharmD, Gregory K. Feld, MD, Frederick T. Han, MD

ABSTRACT

A 67-year-old man presented with dizziness secondary to ventricular tachycardia (VT) originating from the moderator band. The VT was refractory to multiple antiarrhythmic medications requiring extracorporeal membrane oxygenation and eventual curative ablation. We highlight a malignant form of idiopathic VT, unique electrocardiogram characteristics, and ablation considerations. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:946–50) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 67-year-old man presented to his primary care physician after an episode of near-syncope. Electrocardiogram (ECG) showed ventricular bigeminy (Figure 1), and he was subsequently transferred to the emergency department. Physical examination and initial laboratory tests were unremarkable. Within 2 h of presentation, he developed hemodynamically stable sustained monomorphic wide complex tachycardia with left bundle branch block (LBBB) morphology at a rate of 227 beats/min (Figure 2) associated with near-syncope. Lidocaine infusion was initiated with conversion to ventricular bigeminy and resolution of symptoms.

LEARNING OBJECTIVES

- To recognize the 12-lead ECG characteristics of VAs originating from the MB.
- To appreciate the high risk for sudden cardiac death in VAs originating from the MB and the potential curative role of radiofrequency ablation in treatment.

PAST MEDICAL HISTORY

The patient had well-controlled hypertension on losartan. There was no family history of sudden cardiac death or unexplained death.

From the Division of Cardiology, Department of Medicine, Cardiovascular Institute, University of California San Diego, San Diego, California. Dr. Hsu has received honoraria from Medtronic, Abbott, Boston Scientific, Biotronik, Janssen Pharmaceuticals, Zoll, Biosense Webster, and Bristol-Myers Squibb; has received research grants from Biotronik and Biosense Webster; and has equity in Acutus Medical and Vektor Medical. Dr. Feld has served as Director of the Cardiac Electrophysiology Fellowship Training Program, which receives fellowship stipend support from Medtronic, Abbott, Boston Scientific, Biosense Webster, and Biotronik. Dr. Han has received honorarium from Abbott, Biosense Webster, and AltaThera Pharmaceuticals; has served as a consultant for Biosense Webster and AltaThera Pharmaceuticals; and has received research grants from Abbott.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Case Reports* author instructions page.

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DIFFERENTIAL DIAGNOSIS

The differential diagnosis for wide complex tachycardia generally includes ventricular tachycardia (VT), supraventricular tachycardia with aberrancy, and supraventricular tachycardia with antegrade conduction via an accessory pathway.

INVESTIGATIONS

The LBBB morphology with late precordial transition (>V_4/V_5) raises suspicion for a right lateral accessory pathway with pre-excitation, an atriofascicular pathway, a nodoventricular pathway with manifest pre-excitation, or VT originating from the right ventricle (RV). The presence of atrioventricular dissociation suggested VT. Echocardiogram was normal, and cardiac catheterization showed minimal coronary artery disease. Because there was no evidence of structural heart disease, we concluded the mechanism to be an idiopathic VT.

MANAGEMENT

After admission to the hospital, the patient continued to have ventricular arrythmias (VAs) requiring addition of antiarrhythmic infusions, including esmolol, diltiazem, procainamide, lidocaine, and amiodarone. VT persisted and became hemodynamically unstable requiring cardioversion. He was subsequently intubated and deeply sedated. However, VT continued with degeneration into ventricular fibrillation (VF) during profound hypotension and hypoxia, as well as premature ventricular contractions (PVCs) triggering VF. Due to acute severe cardiac failure, venoarterial extracorporeal membrane oxygenation (ECMO) was initiated. The patient required >20 defibrillations in a 12-h period. A subsequent echocardiogram showed severely depressed biventricular function with left ventricular ejection fraction 20%.

On day 2, the patient was then emergently brought to the electrophysiology laboratory for mapping and ablation. Despite numerous defibrillations, invasive hemodynamic monitoring revealed optimal filling pressures, lactate and arterial blood gases were normal, and no vasopressor medications were required. Therefore, venting of the left ventricle was deferred and ECMO flows remained low around 3 to 4 l/min. The VT ECG demonstrated LBBB morphology, with late precordial transition at V_6 and rapid downstroke of the QRS in the precordial leads, with a left superior axis localizing the VT exit site to the RV, specifically the moderator band (MB) (Figure 2). Electroanatomic activation mapping of the endocardial RV was performed guided by intracardiac echocardiography while the patient was in sustained stable monomorphic VT. The MB was observed extending from the septal apex of the RV to the inferolateral free wall of the RV, while the tricuspid valve anterior papillary muscle (APM) extended from the RV apex perpendicular over the MB (Figure 3). The earliest activation

ABBREVIATIONS AND ACRONYMS

APM = anterior papillary muscle
ECG = electrocardiogram
ECMO = extracorporeal membrane oxygenation
ICD = implantable cardioverter-defibrillator
MB = moderator band
PF = Purkinje fibers
PVC = premature ventricular contraction
RV = right ventricle
VA = ventricular arrhythmia
VF = ventricular fibrillation
VT = ventricular tachycardia

FIGURE 1 Initial Electrocardiogram

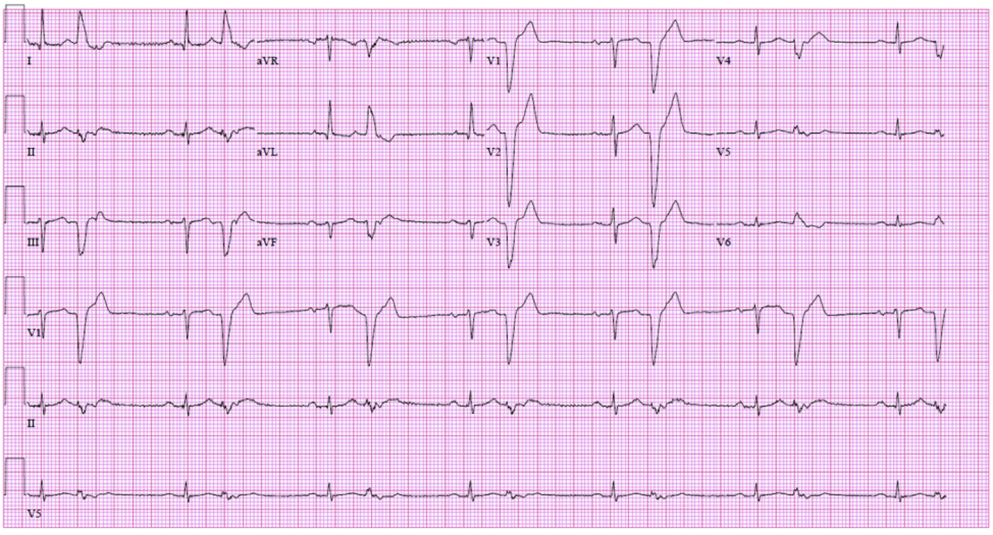

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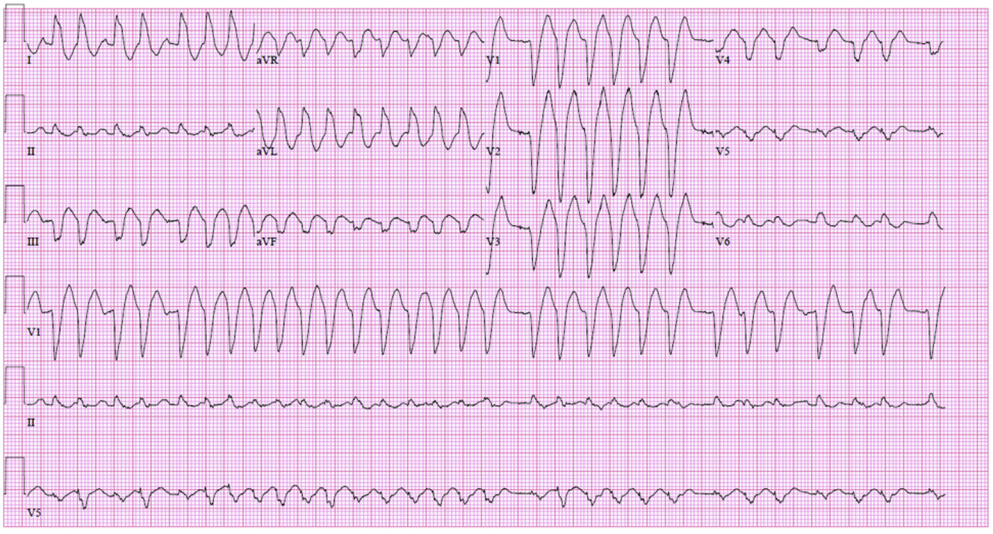
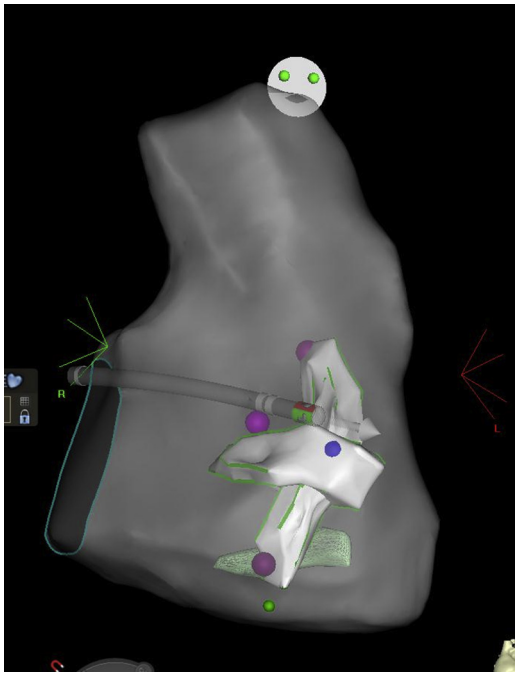
FIGURE 2 Sustained Ventricular Tachycardia



was mapped to the septal insertion of the MB. A ThermoCool SmartTouch F/J curve catheter (Biosense Webster, Irvine, California) was then used for successful radiofrequency ablation. After ablation, burst ventricular pacing triggered a second VT morphology localized to a more lateral location on the MB, which was also ablated. Subsequently, VT was not inducible with burst pacing at isoproterenol infusion of 20 μg/min.

FIGURE 3 Anatomy of the Right Ventricle and Intracavitary Structures in Right Anterior Oblique View



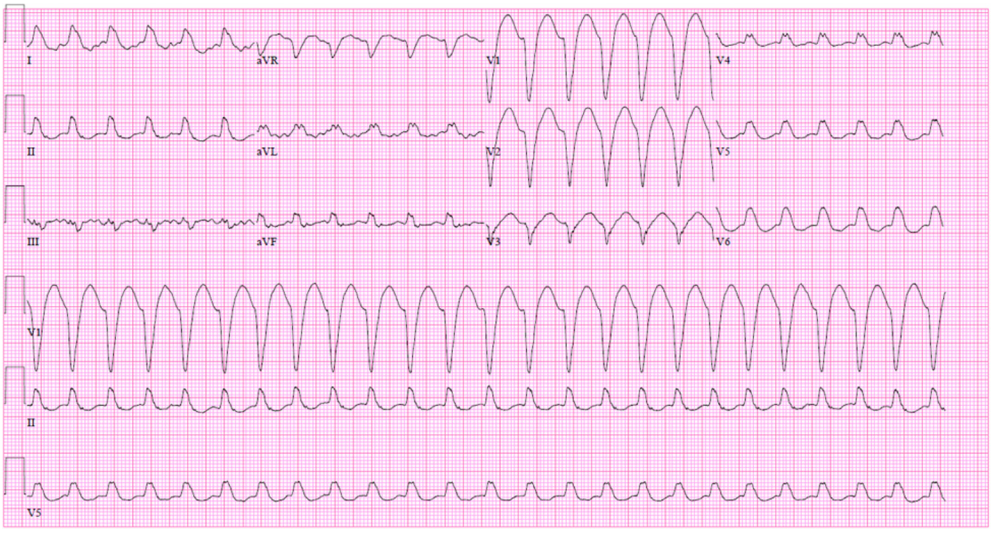
The patient remained on ECMO after the procedure with no antiarrhythmic therapy. However, he converted to a hemodynamically stable VT 2 h after conclusion of the ablation, with a different LBBB morphology with precordial transition at V_4 and a change in axis to +30° (Figure 4). He was restarted on lidocaine infusion with intermittent control of VT and returned to the electrophysiology laboratory the following morning for repeat ablation. A stable VT was triggered when the patient was positioned on his left side. The VT localized to the middle of the MB at the intersection of the APM, and ablation was performed with successful termination (Figure 5). Afterward, no VT or VF were induced.

After the second ablation, the patient remained free of VT with no antiarrhythmic therapy. On day 5, the patient was removed from ECMO. He was started on metoprolol succinate. Echocardiogram prior to discharge showed left ventricular ejection fraction 55%. ECG showed sinus rhythm with an incomplete right bundle branch block. The patient deferred an implantable cardioverter-defibrillator (ICD).

FOLLOW-UP

Cardiac magnetic resonance imaging was performed 2 weeks after discharge that showed normal biventricular function and absence of late gadolinium enhancement. The patient has been free of VAs for 1 year.

FIGURE 4 Post-Ablation Ventricular Tachycardia

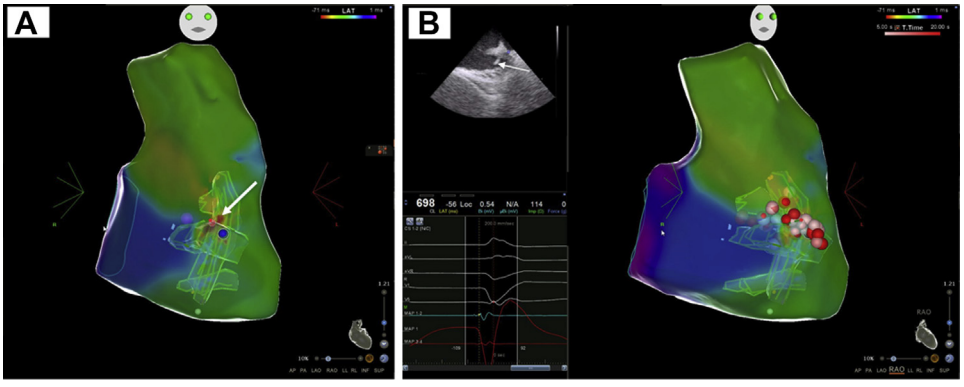


DISCUSSION

The case highlights a malignant form of idiopathic VT that required mechanical circulatory support and ultimately successful radiofrequency ablation targeting the exit site from the MB. Most malignant VAs occur in the presence of structural heart disease, whereas idiopathic VAs occur in structurally normal hearts and are thought to be mostly benign (1). Idiopathic VAs typically originate from the outflow tracts, predominately the RV outflow tract. Although not as common, idiopathic VAs can also originate from intracavitary structures, including the papillary muscles and MB, where the reported risk of sudden cardiac death is higher (2).

The MB contains the right bundle branch of the conducting system as it exits the interventricular septum and spreads into Purkinje fibers (PFs) as it joins the APM and RV free wall. The mechanisms underlying VAs originating from the MB remain

FIGURE 5 Electroanatomic Activation Map for Second Ventricular Tachycardia



(A) Activation map of the second ablation procedure localizing the ventricular tachycardia by the earliest site of activation to the mid-anterior moderator band (MB) at the intersection of the tricuspid valve anterior papillary muscle as indicated by the **white arrow**. **(B) (Top left)** Intracardiac ultrasound image of the MB **(arrow)** intersecting the tricuspid valve anterior papillary muscle. **(Bottom left) Blue tracing** (MAP 1-2) indicates bipolar electrogram that preceded QRS onset by 20 ms. **Red tracing** (MAP 1) indicates unipolar electrogram with a QS complex. **(Right)** Labeled ablation points resulted in successful termination of VT. RAO = right anterior oblique.

speculative currently. The PFs at the MB may have abnormal refractory periods that lead to re-entry, or the PFs may be more susceptible to early afterdepolarizations and subsequently inward calcium current promoting Vas (2). The latter may be susceptible to stretch and patient position (left lateral decubitus), which was utilized to sustain the VT during our second procedure (3). In addition to PVC-triggered VF, VT likely degenerated into VF in this case due to profound hypotension and hypoxia in the setting of sustained rapid VT (4).

The ECG pattern of MB VAs consists of LBBB morphology with a left superior frontal plane axis, a rapid downstroke of the QRS in the precordial leads, relatively narrow QRS width, and a late precordial transition pattern, usually after V_4. The transition is always later compared with sinus rhythm (as shown in **Figure 1**). Importantly, ECG characteristics may vary depending on the morphology and insertion sites of the MB and may change slightly in the same patient given differing exit sites.

Ablation remains first-line treatment, but challenges remain. Sadek et al. (2) reported outcomes in 10 patients presenting with VAs, including PVC-induced VF, mapped throughout the MB. In total, 6 of 10 patients required a repeat ablation, possibly related to difficulty with catheter contact and variable exit points, similar to our patient. Performing additional ablation to include both the septum and free wall insertion sites, as well as utilizing intracardiac echocardiography, cryoablation, and contact force technology, may prove useful in completely eliminating VAs (5). Importantly, all patients were free of sustained VAs at almost 2-year follow-up.

Last, the role of ICD therapy for prevention of sudden death following ablation for MB VAs remains unknown. Cardiac magnetic resonance imaging may be a valuable tool to risk stratify patients after ablation by detecting scar that could potentially act as a source for VAs, although supportive data is lacking. After a shared decision-making conversation including the patient's cardiac magnetic resonance imaging results, our patient deferred ICD therapy.

CONCLUSIONS

We describe a rare case of incessant idiopathic VT originating from the MB that required emergent ECMO and was cured with radiofrequency ablation. Awareness of the unique electrocardiogram characteristics of MB VAs and challenges with ablation are vital for favorable outcomes.

ADDRESS FOR CORRESPONDENCE: Dr. Frederick T. Han, Division of Cardiology, Cardiovascular Institute, University of California-San Diego, 9452 Medical Center Drive, MC7411, Altman Building, 3rd Floor, Room 3E-313, La Jolla, California 92037. E-mail: frhan@health.ucsd.edu.

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KEY WORDS ablation, acute heart failure, cardiac assist devices, ventricular tachycardia