A perfect storm of ventricular fibrillation: Infarct, posterior fascicle, and the moderator band

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Introduction

Ventricular fibrillation (VF) is a highly morbid condition and is associated with subsequent mortality, even in patients with implantable cardioverter-defibrillator (ICD).[1–3] Early ablation should be considered given previously reported short-term mortality benefit.[2,4] Understanding the mechanisms of VF is crucial to enhance likelihood of ablation success.

Monomorphic premature ventricular contractions (PVCs) stimulated from Purkinje-like fibers have been previously described as a trigger for VF.[3] Arrhythmogenic Purkinje fibers can localize within abnormally structured myocardial tissue, including the border zone of ischemic scar.[3–6]

Electrical myocardial structures including the left posterior fascicle and the right ventricular (RV) moderator band have also implicated in the development of VF in the absence of ischemic scar.[7,8] Empiric ablation near the left posterior fascicle of Purkinje-like potentials and PVCs has previously been reported as an effective treatment for VF, even in the absence of ischemic or structural heart disease.[8–11] Idiopathic VF localized to the RV moderator band has also been reported to be suppressed after successful catheter ablation of PVCs arising from this site.[7,12] In part based on these studies, it is a class IIa recommendation to ablate drug-refractory, recurrent, monomorphic PVCs triggering VF and a class I recommendation to ablate non–outflow tract triggers of idiopathic VF.[3]

Here, we present a case in which VF trigger was in proximity of 3 structures that have typically been described in isolation for sustaining PVC-induced VF storm: postinfarct scar, the left posterior fascicle, and the RV moderator band.

KEY TEACHING POINTS

- Premature ventricular contraction (PVC) triggers of ventricular fibrillation (VF) have been described from a variety of sources, such as postinfarct scar or idiopathic PVCs arising from the right ventricular (RV) moderator band or posterior fascicle.
- In this case, VF was triggered by a PVC with a Purkinje-like potential originating from an inferoseptal left ventricular infarct, near the posterior fascicle, directly across the septum from the RV moderator band.
- This case highlights the arrhythmogenicity of structural and electrical substrate at the septal ventricle in the initiation and maintenance of VF.

Case report

A 61-year-old male patient with ischemic cardiomyopathy, heart failure with reduced left ventricular (LV) ejection fraction of 25%, multivessel coronary artery disease complicated by prior myocardial infarction, VF arrest 8 years prior, and obesity presented to our hospital after multiple ICD shocks.

Interrogation of his dual-chamber ICD demonstrated VF storm and 3 ICD shocks on the day of presentation, as well as 3 additional episodes of ventricular tachycardia within the prior month (Figure 1A).

Coronary angiogram did not show culprit disease, but redemonstrated chronic thrombotic occlusion of the left anterior descending and left circumflex coronary arteries, as well as intermediate stenosis of the right coronary artery. He was also treated for acute systolic and diastolic

KEYWORDS Ventricular fibrillation; Electroanatomic mapping; Activation mapping; Moderator band; Myocardial infarction; Posterior fascicle; Premature ventricular contractions; Catheter ablation
(Heart Rhythm Case Reports 2023;9:943–947)

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https://doi.org/10.1016/j.hrcr.2023.10.005

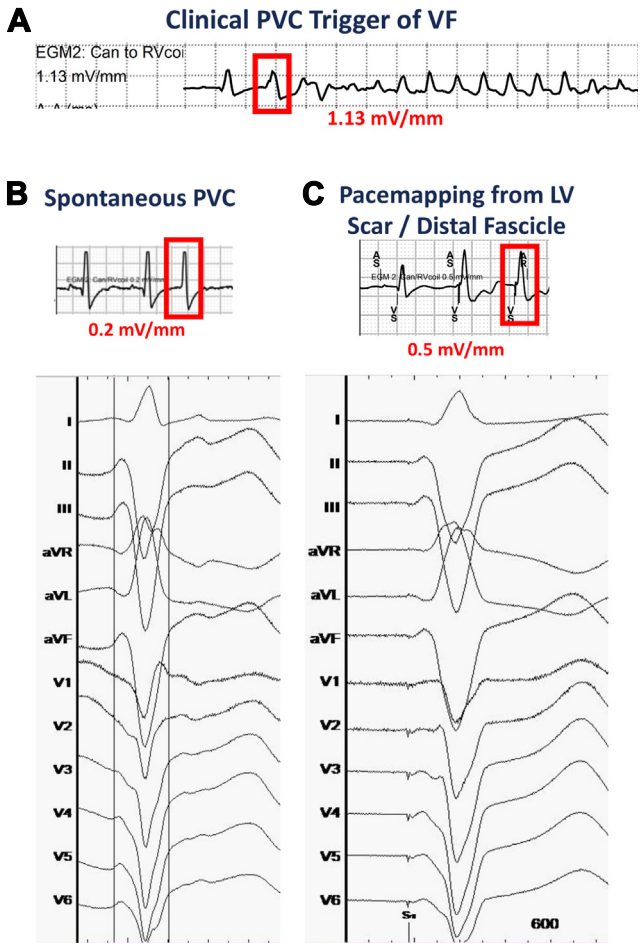

Figure 1 The clinical premature ventricular contraction (PVC) triggering ventricular fibrillation (VF) (**A**) had a similar implantable cardioverter-defibrillator (ICD) electrogram far-field morphology as the spontaneous PVC targeted during ablation (**B**). Pace mapping from the earliest site at the inferoseptal left ventricle (LV) (**C**) also had similar morphology. The amplitudes of the electrograms were automatically saved differently by the ICD, as noted.

heart failure exacerbation with intravenous diuretic. Transthoracic echocardiography demonstrated LV ejection fraction of 25% with global hypokinesis and apical akinesis, with normal RV and valvular function.

As no culprit coronary artery occlusion was noted and a high burden of PVCs remained (Figure 2A), his VF storm was suspected to be PVC mediated. Electrophysiology study was performed to target potential PVC VF triggers.

Procedure management

PVCs were spontaneously observed at baseline under general anesthesia (Supplemental Figure 1). Dopamine was titrated as needed up to 5 mcg/kg/h to allow PVCs to be further induced for activation mapping. The most frequent spontaneous PVC was recorded and templated. The ICD electrogram (EGM) of this PVC was recorded and visually matched with the ICD EGM of the PVC recorded during clinical VF, suggesting this was the clinical PVC trigger (Figure 1, Supplemental Figure 2).

The completely negative precordial transition of the predominant clinical PVC initially suggested a RV origin (Figure 2A). The RV was mapped first using a high-density catheter (Advisor™ HD Grid; Abbott Laboratories, Chicago, IL), and the moderator band was visualized with intracardiac echocardiography. Pacing at the RV moderator band demonstrated a pace map match of 94% to the clinical PVC and local activation was slightly early (local activation time -15 ms relative to QRS onset).[11] Ablation was performed here empirically at 40 W, from the moderator band at the septal insertion point to the RV anterior papillary muscle (Supplemental Video 1). Ablation performed there did not suppress PVCs, but did alter morphology of the predominant clinical PVC, suggesting close proximity (Supplemental Figure 3). Therefore, we performed a transseptal puncture to access and map the left ventricle.

LV substrate map and geometry demonstrated a scar and low voltage (<0.5 mV) in the anteroseptal and inferoseptal LV wall (Figure 2C). An apical aneurysm was also identified. The clinical PVC was localized to the border zone of the scar (activation time -40 ms with a Purkinje-like potential), directly across the septum from the RV moderator band (Figure 2A–2C, Supplemental Figure 4). It was also noted that the PVC origin was just distal to the left posterior fascicle, as shown in a first deflection activation map of sinus rhythm (Figure 3). Intracardiac EGMs show Purkinje-like signals at the earliest site during PVC (arrow, local activation time -40 ms) and during sinus rhythm (asterisk, local activation time -10 ms). During mapping of the PVC in the inferoseptal left ventricle, catheter manipulation induced VF requiring defibrillation, supporting arrhythmogenicity in this region. Ablation was performed with an open-irrigated ablation force-sensing catheter (Tacticath SE™; Abbott Laboratories, Chicago, IL) at 40 W at the earliest activation site at the border zone of the inferoseptal LV apex. The PVC was eliminated, and VF was thereafter not inducible. The patient has not had any VF in more than 18 months of follow-up.

Discussion

We have presented a case of VF storm triggered from a PVC originating from a region in close proximity with 3 structures that have typically been described in isolation as VF-sustaining substrate: postinfarct scar, the left posterior fascicle, and the RV moderator band.[3] Our case illustrates the importance of delineating all possible sources of VF substrate. In the context of ischemic scar, VF sources usually arise from vulnerable areas at scar border zones. Although there was heavy scar burden in this case, it was a border zone that harbored the clinically implicated PVC.

Multiple studies have demonstrated an association of Purkinje-like potential-mediated PVC ablation and

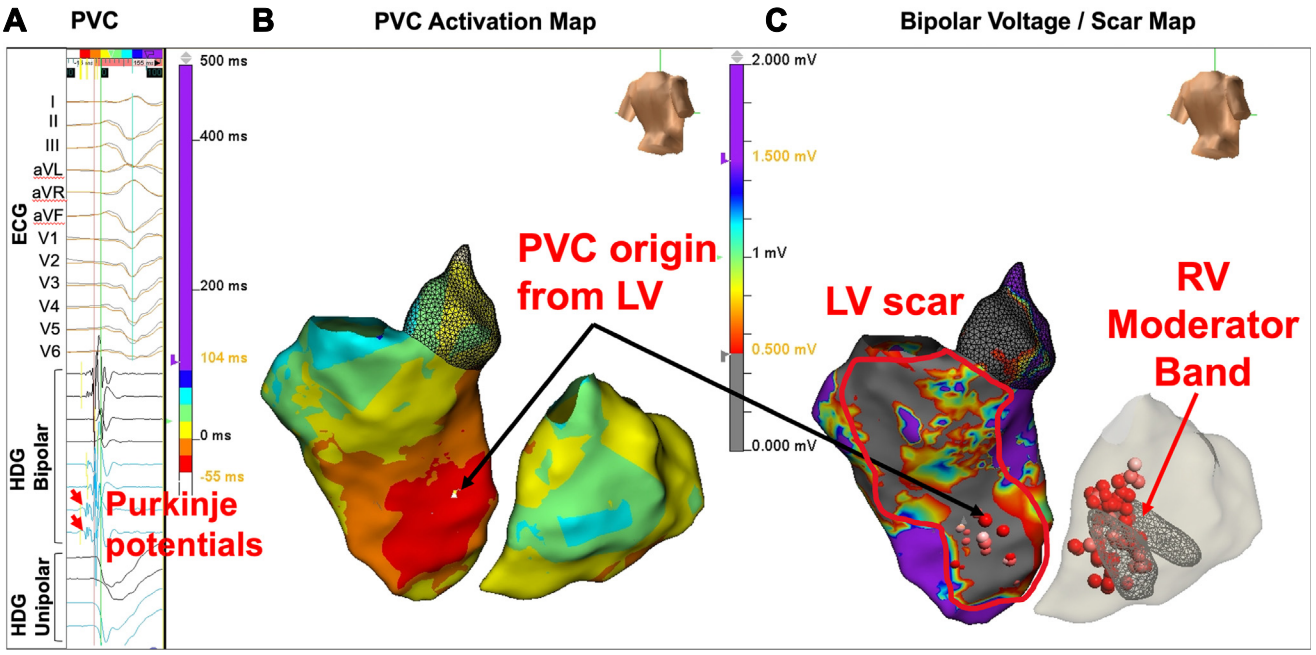

Figure 2 **A:** Twelve-lead electrocardiogram and earliest local activation time (LAT -40 ms) with a Purkinje-like potential of the clinical premature ventricular contraction (PVC), recorded using a multielectrode catheter (high-density grid). **B:** Biventricular activation map showing origin of the PVC from the inferoseptal left ventricle (LV). **C:** Bipolar voltage map showing large scar at the inferoseptal LV, with PVC origin directly across the septum from the right ventricular (RV) moderator band.

suppression of VF.[3–6] In the largest multicenter retrospective observational study to date, Komatsu and colleagues[4] evaluated patients who underwent catheter ablation of post–myocardial infarction refractory VF storm (included remote and index admission cases), finding that greater than 80% of patients sustained in-hospital suppression of VF storm. Review of these ablations redemonstrated findings from smaller studies that ablation of Purkinje-related triggers from the scar border zone at the LV septum was often associated with cure of VF storm. Although it has been considered that perhaps a broad ablation of all potential Purkinje-like triggers should be performed along the scar border zone, this has not been necessary in multiple observational studies.[4,5] Rather, ablation of the Purkinje potentials earliest to the clinically observed PVC that has induced VF appears most essential to successful ablation.

Predetermination as to which anatomic structure from which a clinically observed VF trigger may arise should be met with caution. Salazar and colleagues[11] have demonstrated that interrogation of previously implicated anatomic regions for VF with pace mapping to the stored ICD EGM template can be an effective guide to ablation. In this case, far-field ICD EGMs of the clinical VF trigger matched the morphology of the most frequently occurring spontaneous PVC during the case, and helped identify this PVC as the culprit VF trigger. In addition, the far-field EGM morphologies of the spontaneous clinical PVC and pace mapping from the inferoseptal left ventricle at the distal posterior fascicle were similar. The near-field tip-ring EGM with far-field EGM was on time for both the culprit PVC and during pace mapping from the inferoseptal LV site, giving further evidence of the inferoseptal PVC origin close to the posterior fascicle. Finally, the ICD EGMs during pace mapping at both the RV moderator band and the inferoseptal LV infarct also matched to the clinical VF trigger morphology. This did help further confirm the involvement of this region, but it did not help distinguish the actual PVC origin in the LV inferoseptum compared to the RV. Fortunately in this case, there were enough spontaneous culprit PVCs that facilitated precise activation mapping to eventually localize the true origin. Maintaining consideration of multiple anatomic sites for the formation of VF-sustaining substrate is critical, as we suspect ablation at the moderator band alone would have been insufficient to suppress VF. Indeed, a published case of attempted radiofrequency catheter ablation at the RV moderator band alone for a symptomatic PVC was insufficient for suppression and temporarily induced more ventricular arrhythmia.[13]

Our study is limited by the fact that we cannot definitively state that the PVCs ablated in the LV septum near LV scar and the RV moderator band were the culprits of VF.

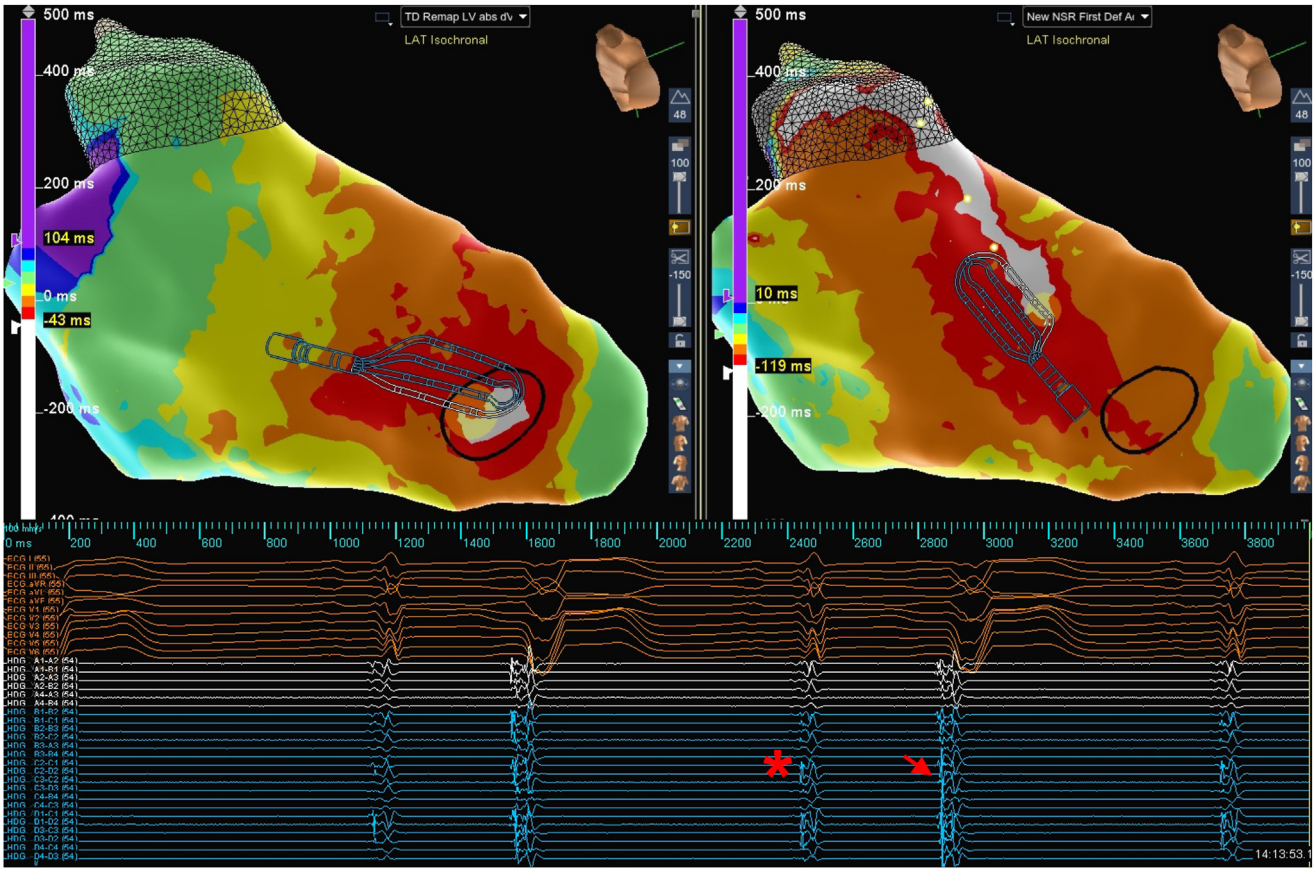

Figure 3 Activation map of the premature ventricular contraction (PVC; left panel) originating from the left ventricular (LV) inferoseptum, just distal to the posteroseptal fascicle, as shown in the activation map of the intrinsic conduction system recorded during sinus rhythm (right panel). Intracardiac electrograms recorded by a multielectrode catheter (high-density grid) show Purkinje-like signals at the earliest site during PVC (*arrow*, local activation time [LAT] -40 ms) and during sinus rhythm (*asterisk*, LAT -10 ms).

However, the induction of VF from catheter irritation in the LV septum and the fact that the patient has been free from VF for 18 months of follow-up after elimination of this PVC are strongly suggestive of its mechanism.

Conclusion

We present a case of PVC-induced VF storm, originating from a particularly arrhythmogenic region exhibiting 3 distinct features that have previously been separately described as VF-triggering and VF-sustaining substrate: postinfarct scar, the left posterior fascicle, and the RV moderator band. Our case enhances previously established literature describing arrhythmogenic Purkinje fibers associated with myocardial scar and PVCs arising from heterogenous myocardial structures that should be considered for mapping and ablation in order to suppress VF storm.

Acknowledgments

We thank Taylor Davis, MS, CEPS, CCDS; Tyler Tackabery, CEPS; Tristen Barbeau, CVT, RCIS; and Kenneth Hopper, CVT for technical and mapping support; and Susan Van Nordheim, RN, MS, NP-C; Christine Clayton, NP-C; and Brooke Conner RN, MS for clinical support.

Funding Sources: This work was supported by National Institutes of Health (NIH 1KL2TR001444).

Disclosures: Dr Ho has received grant support from the American Heart Association (AHA 19CDA34760021), National Institutes of Health (NIH 1KL2TR001444), and Muggleton Family via the Artificial Intelligence Arrhythmia Research Fund at UC San Diego Health, founder equity in Vektor Inc, consulting for Abbott and Kestra. Dr Hoffmayer: Grants from the NIH (F32 HL10472702 and LRP), consulting for Samsung Electronics, Inc, and Vektor Medical Inc. Dr Feld: Equity and consulting for Acutus Medical, Adagio, Medwaves, Varian, and co-founded and consulted for Perminova; received CCEP Fellowship Training program stipend support from Medtronic, Biotronik, Biosense Webster, Boston Scientific, and Abbott. Dr Krummen: Grant from the UCSD Galvanizing Engineering in Medicine Foundation and equity in Vektor Medical Inc. Dr Ahlers and Dr Darden have no conflicts of interest to disclose.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.10.005.

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