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A Perfect Storm of Ventricular Fibrillation: Infarct, Posterior Fascicle, and the Moderator Band

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Abbreviations

Implantable cardioverter-defibrillator (ICD)

Left ventricular (LV)

Premature ventricular contraction (PVC)

Right ventricular (RV)

Ventricular Fibrillation (VF)

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 three structures that have typically been described in isolation for sustaining PVC-induced VF storm: post-infarct scar, the left posterior fascicle, and the RV moderator band.

Case Report

A 61-year-old male 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 three 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, was well as intermediate stenosis of the right coronary artery. He was also treated for acute systolic and diastolic 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 S1**). Dopamine was titrated as needed up to 5 mcg/kg/hr to allow PVCs to be further induced for activation mapping. The most frequent spontaneous PVC was recorded and templated. The ICD electrogram of this PVC was recorded and visually matched with the ICD electrogram of the PVC recorded during clinical VF, suggesting this was the clinical PVC trigger **(Figure 1)**.

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, IL, USA), 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 milliseconds relative to QRS onset).[11] Ablation was performed here empirically at 40W, 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 S1)**. Therefore, we performed a trans-septal puncture to access and map the LV.

LV substrate map and geometry demonstrated a scar and low voltage (< 0.5mV) 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 -40ms with a Purkinje-like potential), directly across the septum from the RV moderator band (**Figure 2A-C**). 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 electrograms show Purkinje-like signals at the earliest site during PVC (arrow, LAT -40ms) and during sinus rhythm (asterisk, LAT -10ms). During mapping of the PVC in the inferoseptal LV, 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, USA) at 40W 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 follow-up.

Discussion

We have presented a case of VF storm triggered from a PVC originating from a region in close proximity with three structures that have typically been described in isolation as VF-sustaining substrate: post-infarct 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 suppression of VF.[3–6] In the largest multicenter retrospective observational study to date, Komatsu et al[4] evaluated patients who underwent catheter ablation of post-MI 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 left ventricular 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 et al[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 pacemapping from the inferoseptal LV 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 pacemapping 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 arrythmia.[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. However, the

induction of VF from catheter irritation in the LV septum and 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 three distinct features that have previously been separately described as VF triggering and sustaining substrate: post-infarct 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.

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Figure 1

The clinical PVC triggering VF (A) had a similar ICD electrogram far-field morphology as the spontaneous PVC targeted during ablation (B). Pace mapping from the earliest site at the inferoseptal LV (C) also had similar morphology. The amplitudes of the EGMs were automatically saved differently by the ICD, as noted.

Figure 2

(A) 12-lead electrocardiogram and earliest local activation time (LAT -40ms) with a Purkinje-like potential of the clinical PVC, recorded using a multielectrode catheter (HDG). (B) Biventricular activation map showing origin of the PVC from the inferoseptal LV. (C) Bipolar voltage map showing large scar at the inferoseptal LV, with PVC origin directly across the septum from the RV moderator band.

Figure 3

Activation map of the PVC (left panel) originating from the 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 (HDG) show Purkinje-like signals at the earliest site during PVC (arrow, LAT -40ms) and during sinus rhythm (asterisk, LAT -10ms).