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Empirical Ablation to Prevent Sequential Purkinje System Recruitment

A Novel Therapy for Idiopathic Ventricular Fibrillation

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ABSTRACT

We report 3 cases (mean age 48.3 ± 11.6 years) of idiopathic ventricular fibrillation (IVF), in which a triggering premature ventricular complex leading to IVF could not be identified. All patients underwent posterior fascicle transection with empirical linear ablation of the mid-Purkinje potentials identified along the left ventricular interventricular inferior septum, and no ventricular fibrillation recurrence was documented in any of the patients. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:517–22) © 2021 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/).

INTRODUCTION

Idiopathic ventricular fibrillation (IVF) is diagnosed in patients who have survived sudden cardiac arrest (SCA) from ventricular fibrillation (VF) without identifiable cardiac structural, metabolic, or electrical abnormalities, after extensive investigations. Short-coupled ectopy can trigger arrhythmia that can be successfully eliminated with premature ventricular complex (PVC) ablation (1). When the source PVC is not reproducible, source-sink continuity of Purkinje network (2) and its ability to initiate quantitative recruitment of Purkinje tissue may have to be targeted.

LEARNING OBJECTIVE

- To propose a novel empirical Purkinje transection as a possible effective ablation strategy in specific patients with IVF who have refractory ventricular arrhythmia after failed PVC ablation or noninducible triggered short-coupled PVCs during ablation.

We report 3 cases of refractory IVF without a clear origin; results of blood chemistry tests, cardiac magnetic resonance imaging, coronary angiography, and echocardiography, were unremarkable, with noninducible triggering PVCs by isoproterenol and programmed stimulation. The echocardiograms of every patient at follow-up did not show any evidence of cardiomyopathy. All cases were treated with novel empirical posterior fascicle transection and linear ablation of mid-Purkinje potentials through radiofrequency ablation. The purpose of this ablation was to prevent sequential, quantitative recruitment of Purkinje tissue network and passive recruitment of adjacent myocardium (**Figures 1A and 1B**). Although there is no strict definition of "empirical ablation of the posterior fascicle and Purkinje tissue," stopping further ablation if there is a new posterior fascicular block or an increase in the HV interval of 10 ms was

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ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

ICD = implantable cardioverter-defibrillator

IVF = idiopathic ventricular fibrillation

PVC = premature ventricular complex

SCA = sudden cardiac arrest

VF = ventricular fibrillation

VT = ventricular tachycardia

used as an arbitrary cutoff point, if it were to occur. The 3-dimensional Cartosound mapping system with the Soundstar intracardiac echocardiography catheter (Biosense Webster, Irvine, California) and the Thermocool Smarttouch SF ablation catheter (Biosense Webster) were used in all patients. Programmed stimulation with triple extrastimuli was performed at the end of the procedure in all cases, and it did not induce VF or ventricular tachycardia (VT). All patients were started on a beta-blocker before the ablation, which was briefly stopped preprocedurally and continued thereafter. None of the patients were receiving class I or class III antiarrhythmic therapy before or after the ablation.

CASE 1

A 56-year-old White woman with a past medical history of asthma and Graves disease was resuscitated from an episode of SCA by her husband, who performed chest compressions until paramedic arrival and shock therapy for VF, thus restoring spontaneous circulation.

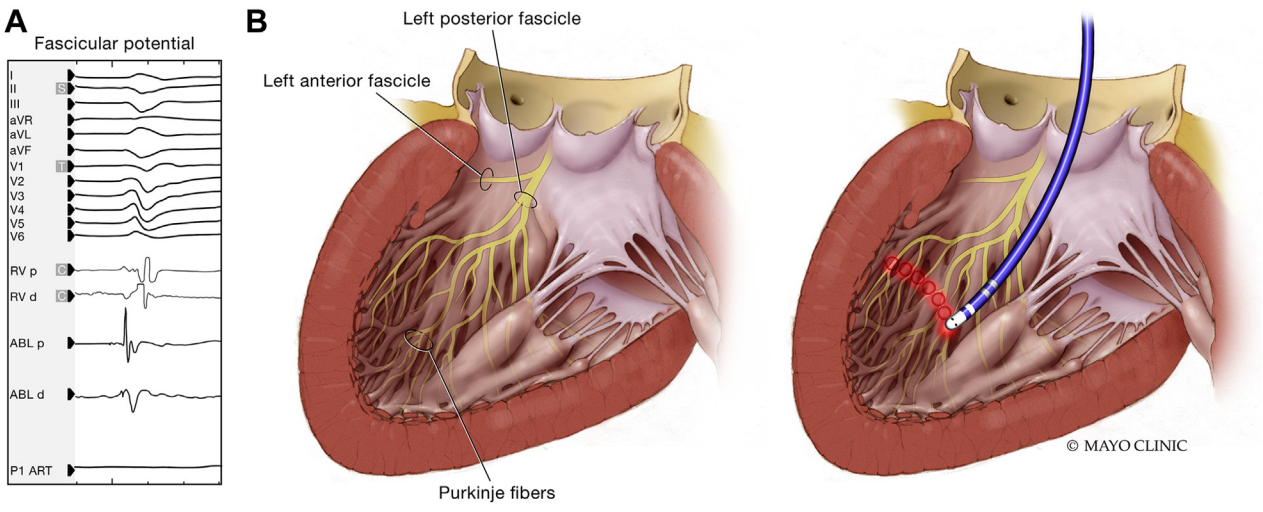
Her electrocardiogram (ECG) showed sinus bradycardia with left bundle branch block. She subsequently underwent insertion of an implantable cardioverter-defibrillator (ICD). She received multiple shocks from VF episodes at 2, 3, 4, and 5 months after ICD implantation triggered by PVCs of common ICD electrogram configuration (both near and far-field) (Figures 2A and 2B).

Because the ICD electrogram from a septal location showed that the initiating PVC had a far-field electrogram ahead of the near field electrogram from the right ventricle (Figure 2B), the left ventricular septum was considered as a possible location for the triggering PVC, which matched with a clinical PVC (asterisk in Figure 2C) in the electrogram during the electrophysiology study (Figure 2C). The clinical culprit PVC triggering polymorphic VT or VF could not be replicated with isoproterenol and programmed stimulation. An electroanatomic activation map was constructed of the left posterior fascicle with identification of fascicular potentials. Posterior fascicle transection and empirical linear ablation of mid-Purkinje potentials were performed (Figure 2D). Ablation was performed using a power output of 40 W and was discontinued after the HV interval increased by 10 ms. A total of 8 radiofrequency ablation lesions were administered to complete the linear ablation. She was followed up in the electrophysiology clinic at 66 months after the ablation, and no VF recurrence was documented.

CASE 2

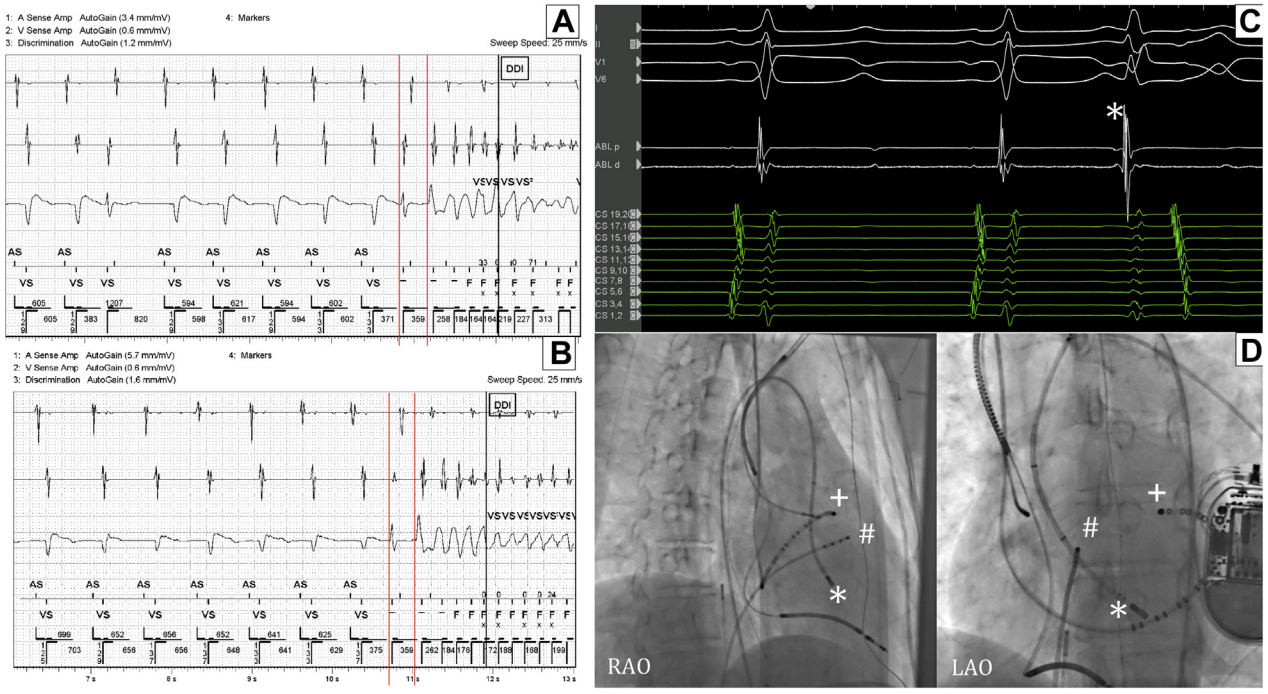
A 54-year-old White man without a cardiovascular history was resuscitated from SCA by his daughter, who performed chest compressions until emergency

FIGURE 1 Anatomic Representation of Posterior Fascicle and Site of Ablation to Prevent Sequential Recruitment



(A) The proximal and distal electrode shows a fascicular potential and a Purkinje potential. **(B)** Posterior fascicle and mid-Purkinje fiber transection through radiofrequency ablation (ABL). RV = right ventricle.

FIGURE 2 **Case 1: Implantable Cardiac Defibrillator Electrogram, Intracardiac Electrogram and Fluoroscopy**



(A) Premature ventricular complex–triggered ventricular fibrillation. **(B)** Implantable cardioverter-defibrillator electrogram from a septal location shows the initiating premature ventricular complex had the far-field ahead of the near-field electrogram from the right ventricle. **(C)** An electrogram shows a clinical premature ventricular complex **(*)** from the left ventricular septum during an electrophysiology study. **(D)** Ablation catheter **(*),** coronary sinus catheter **(+),** and right ventricular and right bundle catheter **(#).** ABL = ablation; CS = coronary sinus; LAO = left anterior oblique; RAO = right anterior oblique.

medical services arrived and defibrillated him twice from VF.

A 12-lead ECG showed complete left anterior fascicular block with nonsignificant PVCs (Figure 3A). A clinical PVC with a superior axis from the left ventricular inferoseptal region was recorded with an ablation catheter during an electrophysiology study (Figures 3C and 3D). Results of comprehensive panels of sudden cardiac death-related arrhythmia and cardiomyopathy genetic studies were unremarkable. A dual-chamber ICD was implanted. He received a shock while he was running on a treadmill, 8 months after ICD implantation. Device interrogation revealed PVC-triggered VF.

After PVC-triggered polymorphic VT or VF could not be replicated with isoproterenol, an electroanatomic activation map was constructed of the left ventricle and left posterior fascicle with identification of fascicular potentials, and ablation was performed at a power output of 40 W by transecting the posterior fascicle. A total of 16 ablation lesions were administered for a total of 705 s. No significant changes were noted on the 12-lead ECG after ablation (Figure 3B). He was followed up in the electrophysiology clinic at 22 months after the ablation, and no VF recurrence was documented.

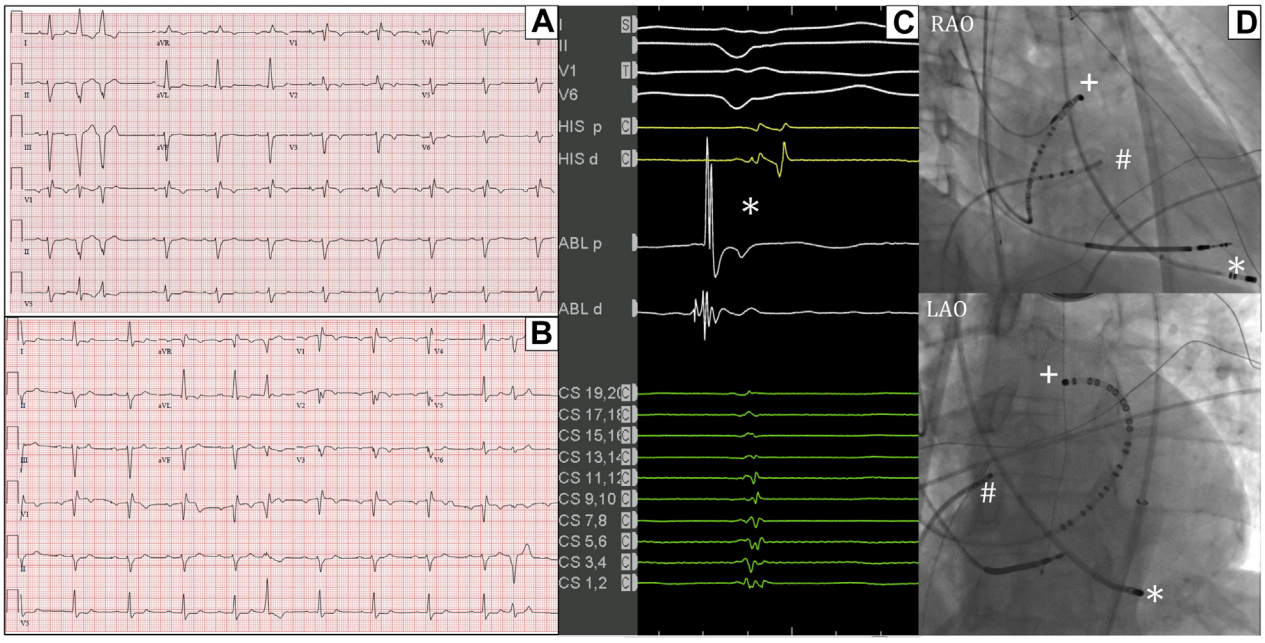
CASE 3

A 35-year-old African American man with a past medical history of hypertension was resuscitated from SCA with chest compressions. VF was documented, which required 2 cardiac defibrillations during transportation to the hospital.

His ECG showed normal sinus rhythm with incomplete right bundle-branch block. The result of a procainamide challenge was negative, and an exercise stress test was unremarkable. A single-lead ICD was implanted. He presented to the emergency department with PVC-triggered VF, requiring 3 ICD shocks at 1 year after ICD implantation. Twelve-lead ECG Holter monitoring showed 2 clinical PVCs likely originating from left anterior fascicle and right ventricular septum.

The His Purkinje system and right bundle were mapped and tagged on the mapping system. During

FIGURE 3 Case 2: Surface ECG, Intracardiac Electrogram and Fluoroscopy



(A) A 12-lead electrocardiogram shows complete right bundle branch block with left anterior fascicular block before the ablation with premature ventricular complexes. **(B)** No significant conduction changes noted on 12-lead electrocardiogram after ablation with premature ventricular complexes. **(C)** An electrogram shows a clinical premature ventricular complex **(asterisk)** with a superior axis originating from the left ventricular inferoseptal region. **(D)** Ablation catheter **(*),** coronary sinus catheter **(+),** and His-bundle catheter **(#).** Abbreviations as in Figure 2.

initial catheter placement into the right ventricle near the moderator band, dissociated potentials were noted (Figure 4A). Despite isoproterenol administration, PVC-triggered polymorphic VT or VF could not be replicated. Empirical ablation was performed at a power output of 30 W in the right ventricular septum anterior to the right bundle (dissociated high-frequency potential was noted on the right side of the septum after a comprehensive map of the right bundle was performed), and it was extended to the interventricular septum (Figure 4B) (although not proven to participate in VF and not consistent with the approach of the previous 2 cases, this was included in the ablation because of a remote possibility of aberrant Purkinje-like fibers with unidirectional conduction precipitating VF).

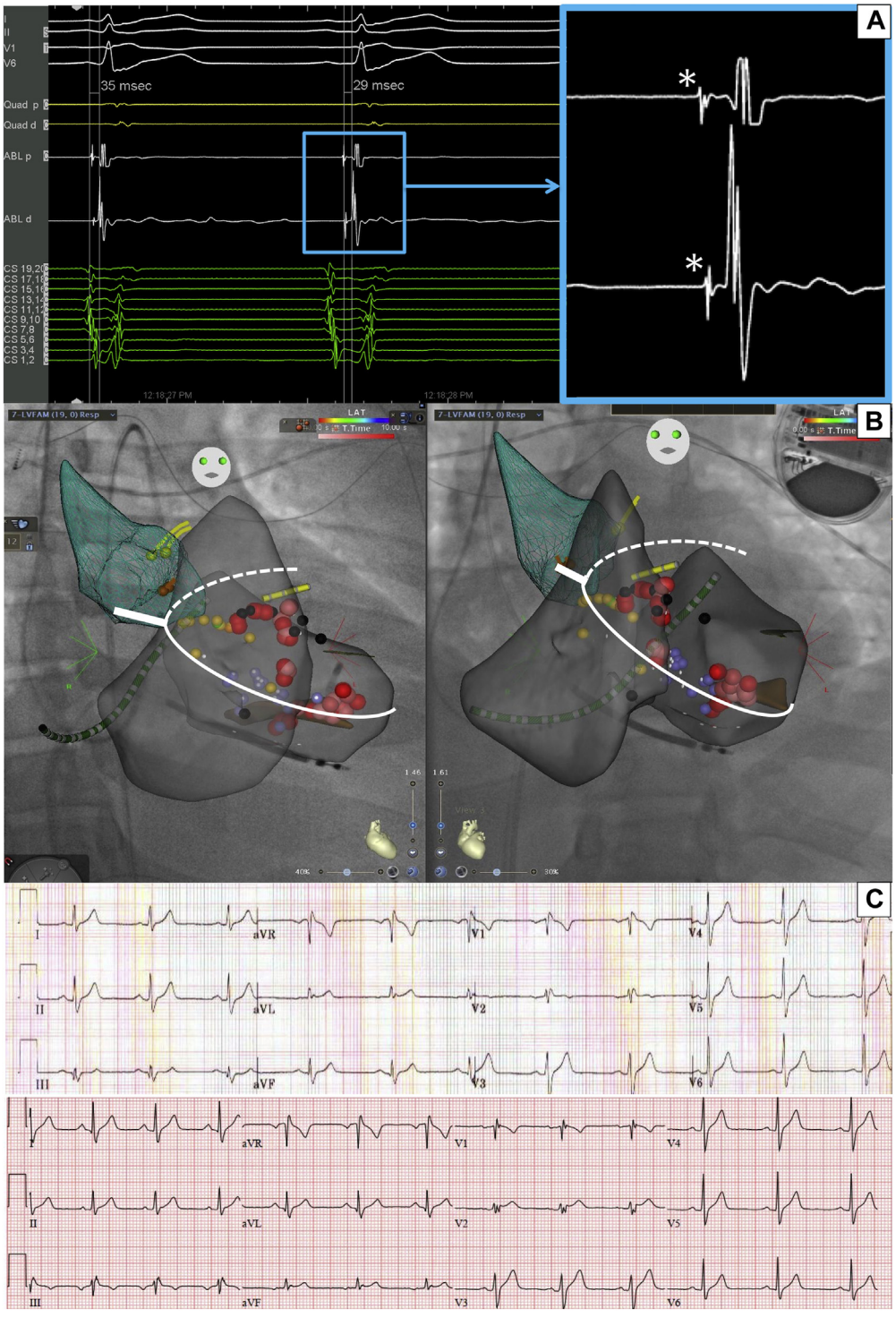
The left posterior fascicle was mapped with identification of fascicular potentials proximally and Purkinje potentials distally. Ablation of mid-Purkinje fibers and transection of the posterior fascicle were performed at 40 W. Minimal prolongation of the HV interval from 55 to 63 ms was noted. The ECG was similar before (Figure 4C) and after (Figure 4C) ablation, and the particular PVC was no longer noticed. The patient was followed up in the electrophysiology clinic at 18 months after the ablation and had no VF recurrence or shocks.

DISCUSSION

We report 3 cases of IVF in patients with triggering short-coupled PVCs that could not be replicated in the electrophysiology laboratory. All 3 patients were treated with empirical fascicular ablation, which prevented sequential and quantitative recruitment of Purkinje tissue, without recurrence of clinical arrhythmia over a significant period.

Short-coupling triggering PVCs were from the left, right, or both Purkinje systems in up to 85% of the IVF cases. The locations of Purkinje potential likely originated from peripheral Purkinje fibers (1). The target ablation sites are either the earliest electrogram site preceding the PVC onset or the site of best-matched morphology by pace mapping. The recommended ablation endpoints are complete elimination of culprit PVCs, local Purkinje potentials, and localized substrate (3). However, not infrequently, spontaneous or inducible PVCs triggering ventricular

FIGURE 4 **Case 3: Intracardiac Electrogram, Electroanatomic Mapping and Surface ECG**



(A) Fascicular potential **(*)** with Purkinje premature ventricular complex. **(B)** Left anterior fascicle **(white solid line),** left posterior fascicle **(white dotted line),** left bundle **(white thick solid line),** fascicular potentials **(yellow),** Purkinje potential **(blue),** coronary sinus catheter **(green),** and mid-Purkinje fiber transection and empirical ablation of left posterior Purkinje potentials **(red)** (right anterior oblique and left anterior oblique views from left to right). **(C)** The electrocardiograms were similar before **(top)** and after **(bottom)** ablation.

fibrillation may not be reproduced in the electrophysiology laboratory.

Purkinje systems play a role in the initiation and maintenance of VF or polymorphic VT (4). The triggering PVC is the source of initiation of ventricular arrhythmia, and the Purkinje system acts as the synchronization with secondary recruitment of myocardium with volumetric changes in the conducting medium (2). Therefore, empirical mid-Purkinje fascicular ablation and posterior fascicular ablation (5) could decrease critical volume of conducting medium and thus inhibit re-entrant ventricular tachycardia acceleration.

We propose a novel method of empirical fascicular ablation and mid-Purkinje transection through radiofrequency ablation in specific patients with IVF who have VF with documented short-coupled PVCs but a lack of spontaneous or inducible short-coupled PVCs. Empirical mid-Purkinje transection to interrupt sequential continuity may yield significant efficacy given the larger mass of Purkinje tissue within the posterior fascicle (6). In our cases, all patients underwent aggressive programmed ventricular stimulation at the end of the empirical ablation, and no sustained ventricular arrhythmia could be induced.

During follow-up, PVCs were still documented among 3 patients, but no VF recurrence was documented in any of the patients. This finding supports our hypothesis that empirical fascicular ablation and mid-Purkinje transection effectively interrupt sequential recruitment of Purkinje fibers that initiate and maintain VF.

In Case 3, the post-ablation HV interval was 63 ms. Ablation was discontinued if the HV interval increased by 10 s or if left posterior block occurred. No significant atrioventricular block was observed post-procedure and during follow-up. Empirical mid-Purkinje transection may cause minimal QRS complex widening without symptomatic progression to heart block or advanced His-Purkinje disease.

LIMITATIONS

It is possible that empirical ablation inhibits PVC conduction into the fascicular system and the initiation of VF. Two patients had a follow-up duration of <5 years. The risk of posterior fascicle ablation includes posterior fascicular block after ablation. Beta-blockers were used in all patients before and after procedures, and this use may have contributed to antiarrhythmic effects. Moreover, not every patient underwent genetic testing, because of patient financial limitations. Procainamide challenge was done if there was a family history of sudden cardiac death, any baseline ECG changes, or clinical features such as ECG changes with fever.

CONCLUSIONS

A novel empirical Purkinje transection through radiofrequency ablation may be an effective ablation strategy in specific patients with IVF who have frequent ventricular arrhythmia requiring therapy but a lack of short-coupled spontaneous PVCs that cannot be replicated with isoproterenol administration. QRS complex widening may be observed after ablation, with a mild increase in HV interval without significant atrioventricular block in our limited experience. This approach may need further validation before widespread use and may only be used when other options have failed.

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KEY WORDS ablation, idiopathic ventricular fibrillation, sudden cardiac death