Rescuing failed radiofrequency ablation: Pulsed field ablation in ventricular tachycardia

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Introduction

Pulsed field ablation (PFA) is an innovative ablative technique increasingly employed in managing atrial fibrillation. It uses high-energy, nonthermal, nanosecond electrical pulses to induce irreversible electroporation, selectively disrupting cellular structures. These tailored waveforms minimize damage to surrounding nonmyocardial tissues, such as pulmonary veins, esophagus, and nerves.[1] However, there is limited evidence on the efficacy of PFA for treating ventricular tachycardia (VT).

Here, we present 2 cases in which PFA technology was applied to manage scar-mediated VT. These cases will add to the initial experience of ablating VT in structural heart disease.

Cases reports

Case 1

A 76-year-old man with a history of ischemic cardiomyopathy, heart failure with reduced ejection fraction, and coronary artery disease post myocardial infarction presented with recurrent episodes of sustained monomorphic VT necessitating antitachycardia pacing and shocks.

Echocardiography revealed an ejection fraction of 25% with an aneurysmal apex, and nuclear stress testing confirmed a fixed perfusion defect at the apex. The patient was discharged on amiodarone (oral loading dose of 400 mg twice daily for 2 weeks), mexiletine, and carvedilol.

Four weeks after discharge, he experienced multiple episodes of VT despite medical therapy, accompanied by borderline blood pressure. Given the refractory nature of these events and frequent implantable cardioverter defibrillator (ICD) therapies, VT ablation was pursued.

KEY TEACHING POINTS

- Scar-related ventricular tachycardia often results from re-entry circuits within scarred myocardial tissue.
- Radiofrequency ablation is the primary modality for eliminating arrhythmogenic foci.
- Pulsed field ablation may offer a viable option in refractory cases of scar-mediated ventricular tachycardia.

During electrophysiological study, mapping using an Optrell Catheter (Biosense Webster, Irvine, CA) in the left ventricle identified low-voltage scar tissue at the apical aneurysm (Figure 1B) with fractionated potentials anteriorly, anterolaterally, and septally. Induction of VT during catheter manipulation necessitated termination due to severe hypotension, despite maximum inotropes and vasopressors. Further mapping at the inferolateral border of the apical scar and junction of healthy tissue (Figure 1C) revealed fractionated electrograms during diastole with a pace map showing 96% similarity on PASO Module mapping (Biosense Webster) and 12/12 visual alignment, suggesting it as the likely exit site (Figure 1D–1F).

We initially targeted this exit site with ablative power set at 40–50 W for 250–300 seconds with an irrigation speed of 8 mL/min, followed by ablation of fractionated areas within the apical scar (ie, apical septal, anterior septal, and anterolateral apical regions) with a total of 21 applications. Persistent late potentials despite long-duration radiofrequency (RF) application via the QDOT MICRO Catheter (Biosense Webster) prompted the use of PFA via the FARAWAVE Catheter (Boston Scientific, Marlborough, MA) (Figure 1A), requiring 16 sets of pulses, each comprising 5 pulses. Total radiofrequency ablation (RFA) time was 977 seconds. Post-PFA application, the electrogram amplitude diminished remarkably. Subsequent programmed ventricular stimulation with triple extra stimuli failed to induce VT. The patient remained free of VT at 3-month follow-up.

KEYWORDS Pulsed field ablation; Ventricular tachycardia; Post-infarction; Radiofrequency ablation; Catheter ablation
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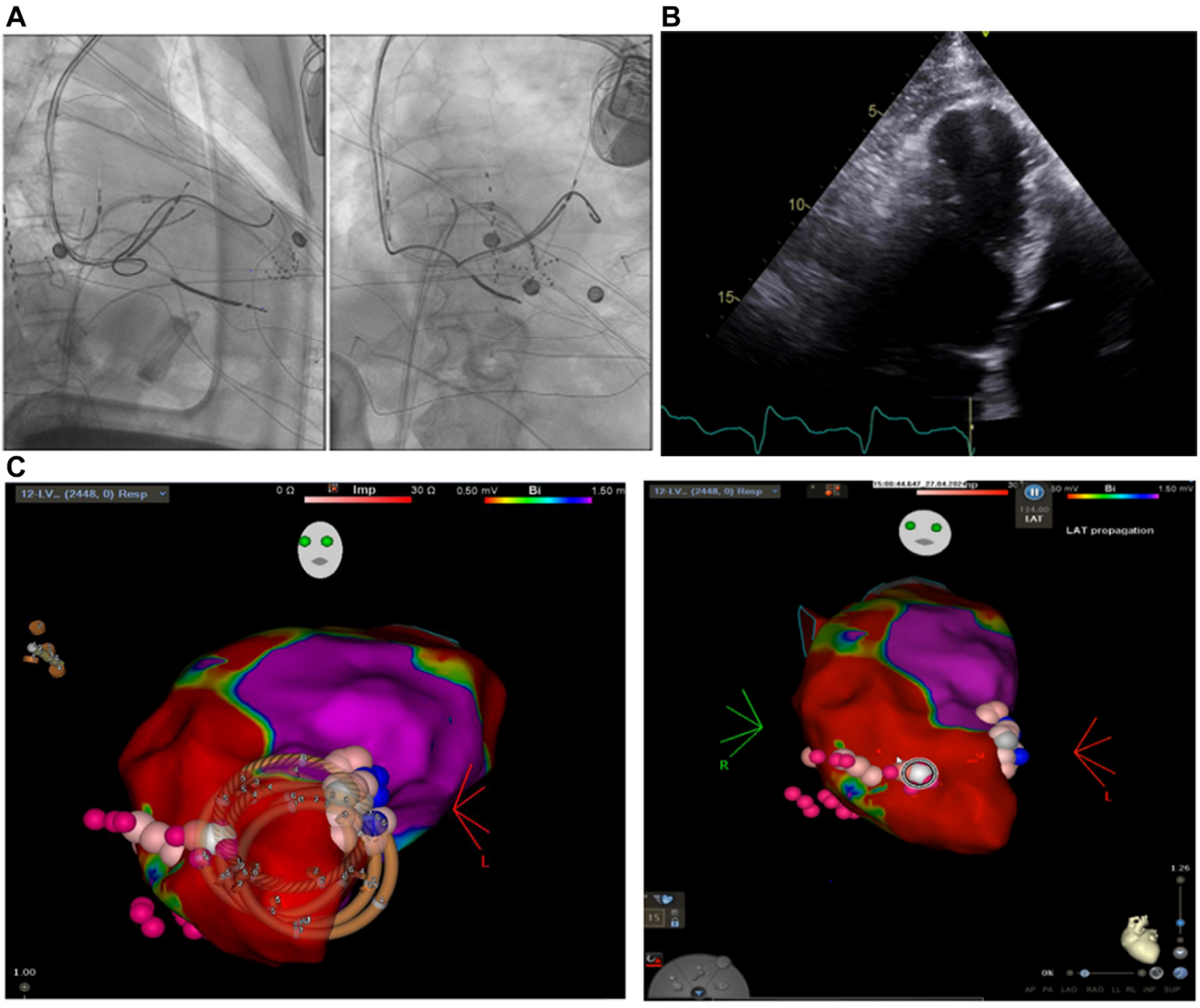

Figure 1 **A:** Fluoroscopy image showing FARAWAVE Catheter (Boston Scientific, Marlborough, MA) in the left ventricle. **B:** Ultrasonography scan demonstrating left ventricular apical aneurysm. **C:** Substrate map showing areas of ischemic scar (*red*) and healthy cardiac tissue (*pink*). **D:** Biventricular pacing vs S2 electrogram difference. **E:** Electrogram comparing pace map vs clinical ventricular tachycardia. **F:** Electrogram showing template match.

Case 2

A 62-year-old man with a medical history of systolic heart failure (ejection fraction of 48%) secondary to ischemic cardiomyopathy, coronary artery disease treated with percutaneous coronary intervention to the right coronary artery and obtuse marginal 7 years ago, and inducible VT with a dual-chamber ICD implantation, was admitted to the hospital after several ICD discharges for VT. He was discharged on oral amiodarone, which was continued up until the procedure.

The patient was advised to consider a VT ablation for his recurrent monomorphic VT to minimize therapies, and the ablation was subsequently performed.

During the procedure, mapping using the Optrell Catheter showed a large scar on the anterior septum of the left ventricle, extending from the base to the apex. Slow conduction areas were identified (Figure 2A) and the QDOT MICRO RF catheter was initially used to ablate the slow zone gap. The patient repeatedly went into clinical VT during catheter manipulation, requiring cardioversion due to low blood pressure (40–50 mm Hg systolic, despite dopamine and dobutamine at maximum doses) (Figure 2B). Multiple location pace mapping surprisingly showed the base of the scar was a 92% pace map match (Figure 2C), whereas the apex produced a 25%–50% pace map match. Subsequently, widely separated double potentials were observed along the scar, particularly at the apex and base, with very long double potentials that were difficult to eliminate, even with a 50-W irrigated catheter (irrigation speed of 8 mL/min) with 32 applications of 240-second lesions (Figure 2D).

We switched to the FARAWAVE Catheter for further ablation. Monomorphic VT was no longer inducible after

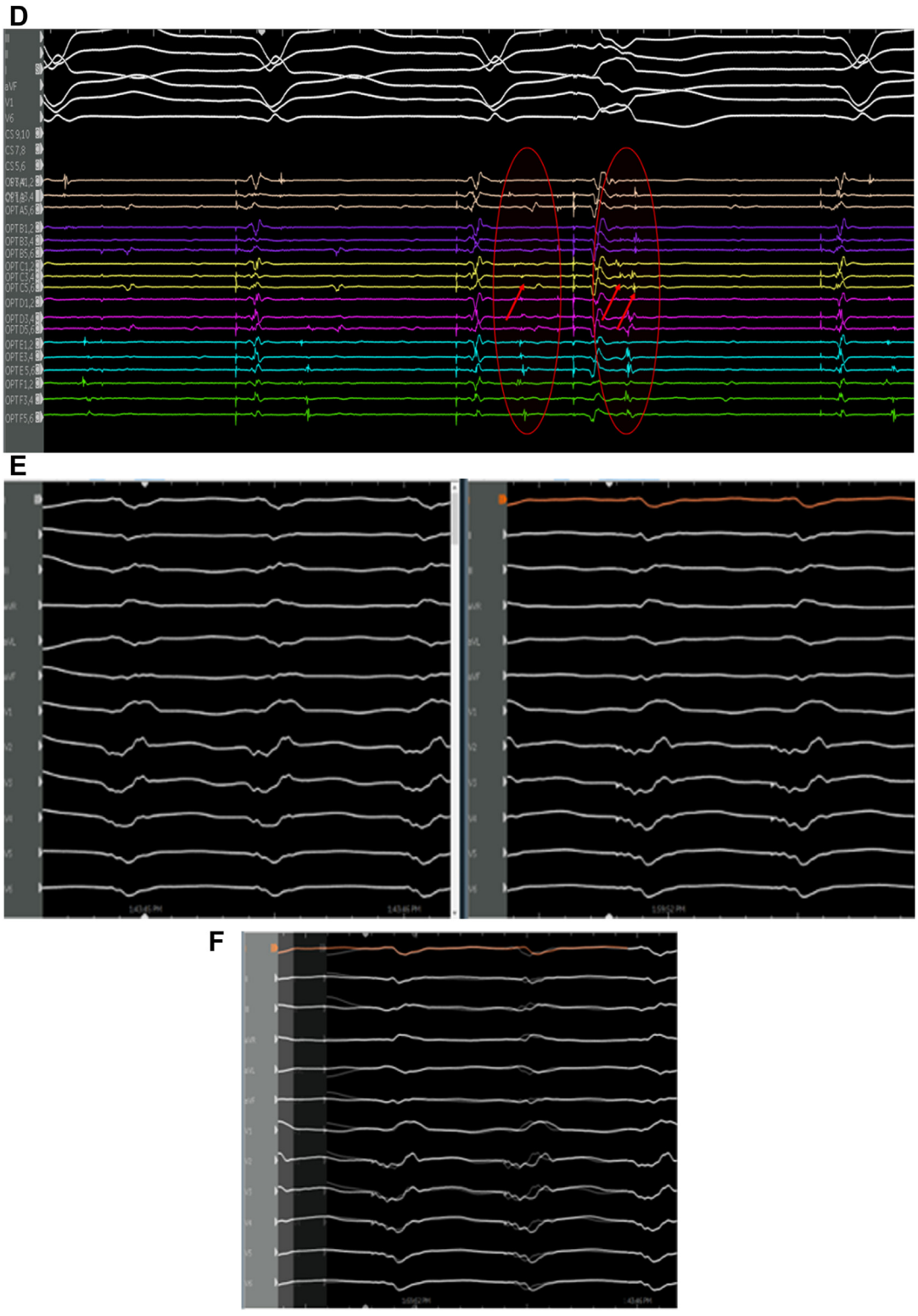

Figure 1 (*continued*).

32 FARAWAVE applications, and only nonsustained, nonclinical polymorphic VT was inducible (Figure 2E–G).

At the 3-month follow-up appointment, the patient was doing very well, with no episodes of VT recorded on the ICD.

Discussion

PFA is a new energy source that is increasingly used in treating atrial fibrillation by pulmonary vein isolation. It is an emerging modality in cardiac electrophysiology, particularly

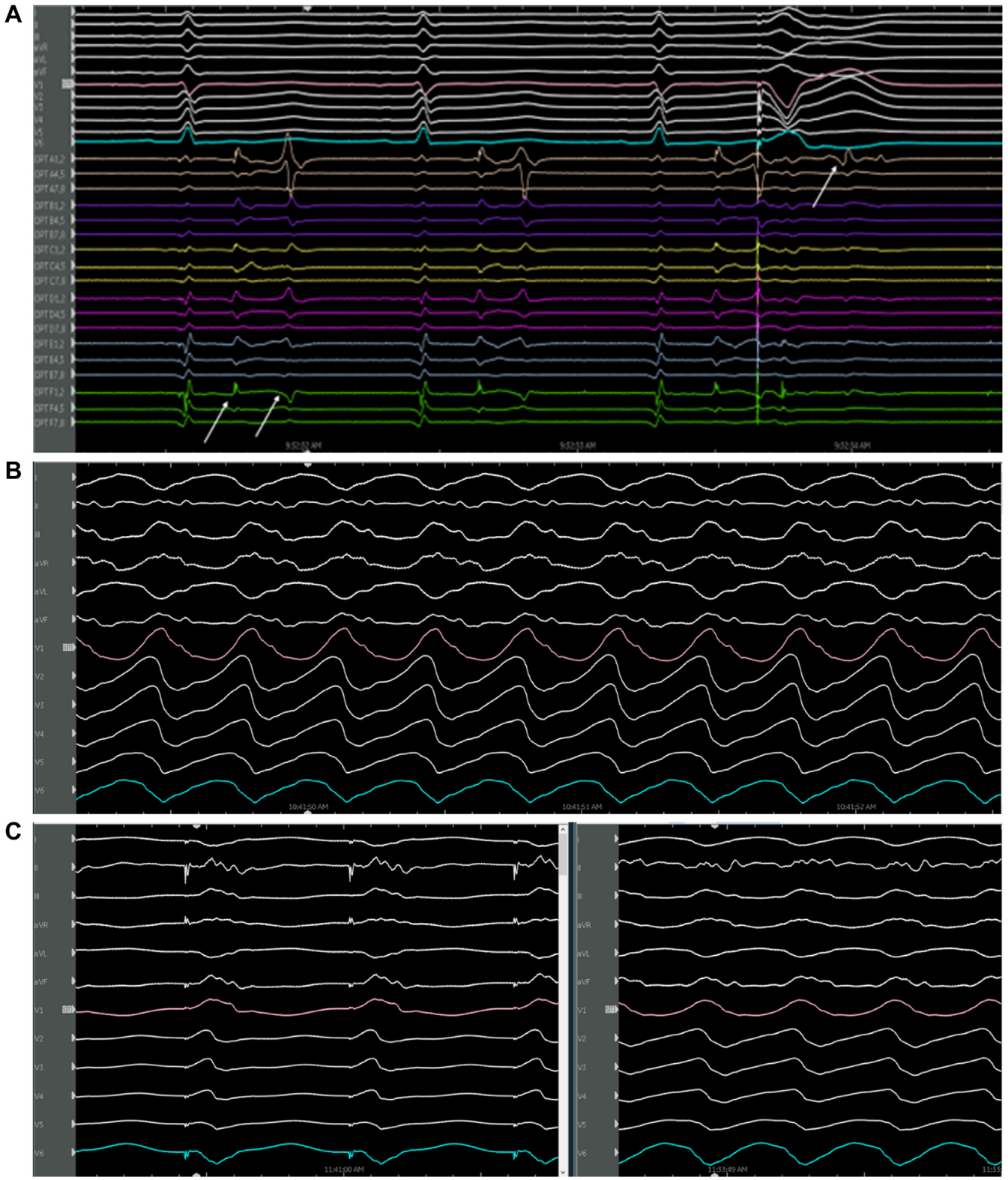

Figure 2 **A:** Decremental evoked potentials mapping and late potentials in sinus rhythm. **B:** Catheter movement induced clinical ventricular tachycardia (VT) requiring cardioversion due to poor hemodynamic tolerance. **C:** Pace mapping near the exit site with close resemblance to clinical VT. **D:** Electrogram showing persistence of potentials despite extensive radiofrequency ablation at 50 W. **E:** Electrogram before FARAPULSE (Boston Scientific, Marlborough, MA) vs after first FARAPULSE. **F:** Electrogram after completion of FARAPULSE. **G:** Electrogram showing post-PFA nonsustained polymorphic VT.

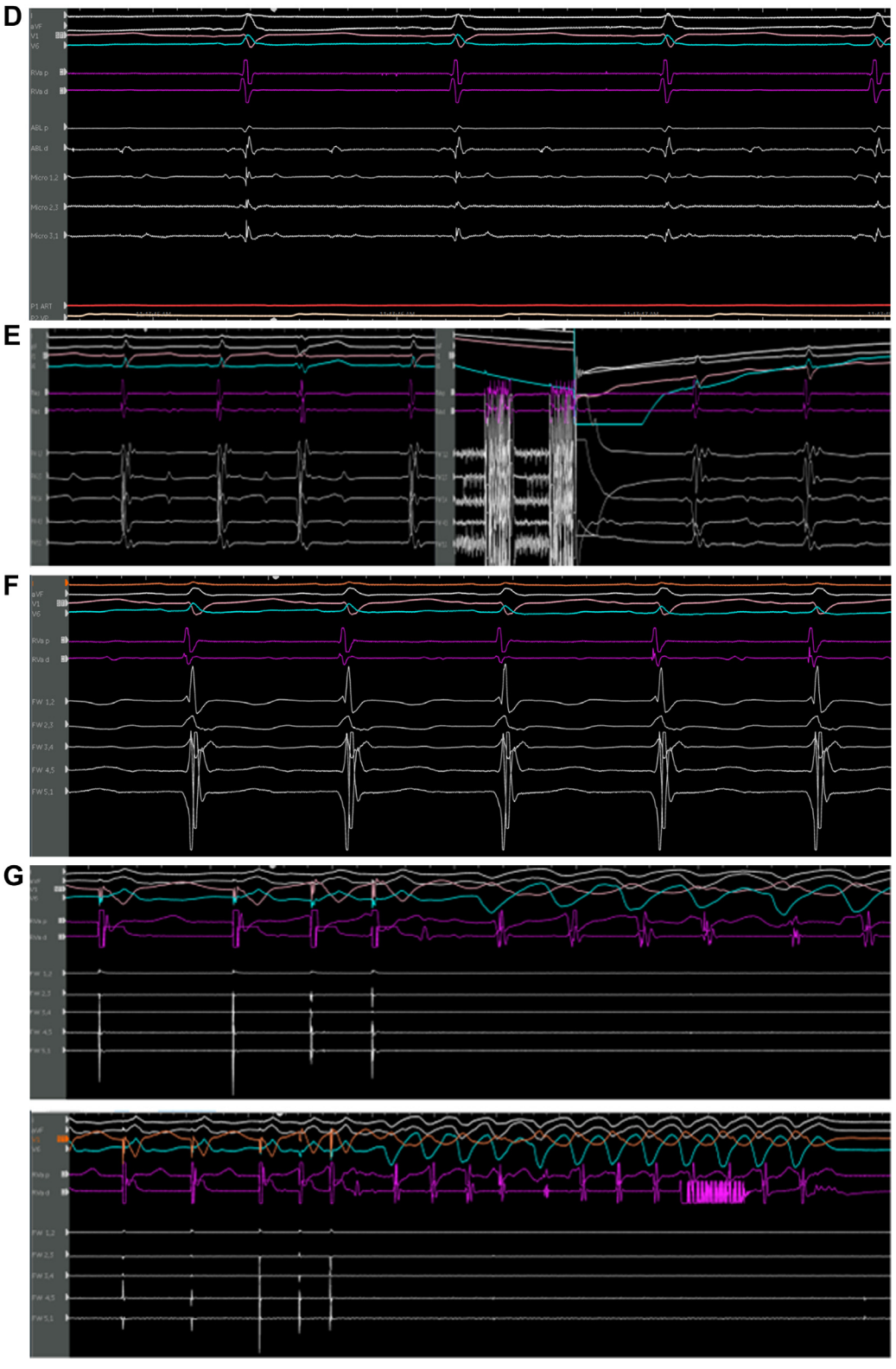

Figure 2 (*continued*).

for cases refractory to conventional RFA methods. Unlike RFA, PFA uses nonthermal energy to achieve targeted cell disruption via irreversible electroporation, sparing adjacent structures like blood vessels and nerves.[1]

If necessary, initial treatment for hemodynamically stable VT includes antiarrhythmic drugs and electrical cardioversion.[2] For patients with VT experiencing ICD shocks despite antiarrhythmic therapy, ablation is advised.[3]

Scar-related VT is a potentially life-threatening condition. It is usually caused by re-entry due to scar tissue. with a few poorly coupled, slowly conducting myofibrils acting as a slow zone formed after a myocardial infarction or due to other types of nonischemic pathologic myocardial tissue.[4]

PFA is currently in the experimental phases for VT ablation. In our cases, the decision to ablate was made based on the PARTITA trial to minimize ICD therapies and improve prognosis.[5]

RFA is used extensively to treat VT. However, in scarred myocardium, RFA results in unpredictable and suboptimal lesion formation in the residual myocytes.[6] This may be due to the heterogeneity of biophysical and thermodynamic characteristics of scar tissue compared with healthy myocardium.

Although there have been multiple advancements in catheter ablation techniques in recent years, the application of thermal energy using RFA in scar tissue remains a concern due to its unpredictable lesion formation.

We decided to use PFA for multiple reasons. Firstly, the QDOT RF catheter could not effectively eliminate the abnormal signals in our cases, despite using high energy levels and ablation indices with RFA. Secondly, our patients had scar-mediated VT, and PFA's mechanism of creating cell membrane pores might have been more effective in disrupting the abnormal electrical pathways within the dense scar tissue compared with the thermal injury caused by RFA.[7]

PFA application in VT and the ventricular myocardium remains investigational, with potential advantages in challenging cases, such as ours, when traditional RFA failed to eliminate arrhythmogenic substrates effectively. PFA could be a viable alternative for patients who experienced VT recurrence after RFA due to its potential for deeper and more effective lesion formation.[8] In a review article by Zhang and colleagues,[9] compiling 10 cases of PFA application to treat patients with VT, which included 8 publications, no significant adverse clinical outcomes, including VT recurrence, or procedure-related complications were reported in the mean $\pm$ SD follow-up period of 3.1 $\pm$ 2.0 months in that study, indicating a favorable initial safety profile. The mean age of patients in that study was 58.5 years, which is younger than the mean age of 70.3 years in our study. We also encountered difficulty maneuvering the catheter, similar to the cases referenced in Zhang and colleagues' review article. In 1 case involving VT originating from the right ventricular inferolateral free wall in the case series by Lozano-Granero and colleagues,[10] PFA application induced VT, which was not observed in either of our cases, in which the VT originated from the left ventricle. A recent case by Katrapati and colleagues,[11] which demonstrated the first successful application of PFA for VT ablation in the United States, showed successful elimination of 2 clinical VTs originating from an ischemic apical scar extending from the lateral apex to apical septum. This case demonstrated similarities to ours with comparable outcomes observed during follow-up. However, the number of applications required to ablate the clinical VTs in our cases was notably lower, with 74 applications in the reported case compared with 16 and 32 in our cases.

The correlation between acute and long-term outcomes remains unknown. Utilization of PFA for VT ablation may require randomized evaluation.

Conclusion

PFA shows promise as an adjunctive therapy for VT ablation, particularly when traditional methods are inadequate. Our case series demonstrates the short-term success of PFA in treating VTs. However, due to the recent nature of these procedures, long-term efficacy remains unverified, necessitating further studies.

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