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Ventricular Tachycardia Storm After Standard Radiofrequency Pulmonary Vein Isolation

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 69
Final Diagnosis: Ventricular tachycardia storm post PVI ablation
Symptoms: Recurrent premature ventricular contractions at 180–200 bpm rate that progressed into a VT storm
Medication: —
Clinical Procedure: Radiofrequency PVI ablation
Specialty: Cardiology

Objective: Unknown ethiology

Background: The occurrence of ventricular arrhythmias (VAs), particularly premature ventricular complexes, following pulmonary vein isolation (PVI) is a documented phenomenon, but monomorphic scar-related ventricular tachycardia (VT) following PVI is an unusual phenomenon. In this case report, we present a case of new-onset VA after radiofrequency PVI in a patient with no prior history of sustained VTs.

Case Report: Our patient was a 69-year-old man with a history of symptomatic persistent atrial fibrillation, with an apparently structurally normal heart with subtle regional wall motion abnormalities. He underwent radiofrequency directed pulmonary vein isolation ablation. On the night of an uneventful procedure, the patient for the first time experienced a sustained ventricular tachycardia that exacerbated into a VT storm. Each arrhythmia was terminated by cardioversion due to hemodynamic instability. Antiarrhythmic treatment with lidocaine was initiated immediately. The patient settled from sustained ventricular arrhythmia and received further ablation to monomorphic ventricular tachycardia.

Conclusions: The incidence of ventricular ectopics after PVI ablation has been previously described, but a sustained monomorphic ventricular storm has not been reported before with RF ablation. We attribute the pathophysiology to an increase in myocardial excitability and/or ventricular autonomic modulation. This is a very rare phenomenon, but any subtle imaging abnormality before planning RF-PVI should be taken into consideration.

MeSH Keywords: Atrial Fibrillation • Catheter Ablation • Tachycardia, Ventricular

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/918432

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Background

Occurrence of ventricular arrhythmias (VAs), particularly premature ventricular complexes, following pulmonary vein isolation (PVI) is a documented phenomenon, but monomorphic scar-related ventricular tachycardia (VT) following PVI is an unusual phenomenon. In this case report, we present a case of new-onset VA after radiofrequency PVI in a patient with no prior history of sustained VTs.

Case Report

A 69-year-old man with a history of symptomatic persistent atrial fibrillation was referred for catheter ablation (CA). He had had 2 events of pre-syncope and his echocardiogram showed normal left ventricular (LV) systolic function and a normal LV cavity. The left atrium (LA) was mildly dilated. His full history of comorbidities was traced and he was only on Sotalol 40 mg BD and Apixaban 5 mg/BD. He had no serious cardiac problems requiring assessment or investigation prior to developing AF, he had no chest pain and denied any recent history of flu-like symptoms or any other viral illness, and he had no family history of SCD (e.g., congenital arrhythmia) before the age of 40.

Wide antral circumferential ablation (WACA) was performed under general anesthesia. Antiarrhythmic medication was withheld 3 days prior to the procedure. He was in AF. All 4 pulmonary veins were successfully isolated. The procedure was uneventful. However, that night he developed frequent premature ventricular contractions – "PVCs" – that quickly provoked a sustained event of monomorphic broad-complex VT at 180–200 bpm documented in 12 leads ECG (Figures 1, 2). When these events developed, he had not been given his evening Sotalol dose. Initial management with IV Metoprolol was unsuccessful. Subsequently, due to hemodynamic instability,

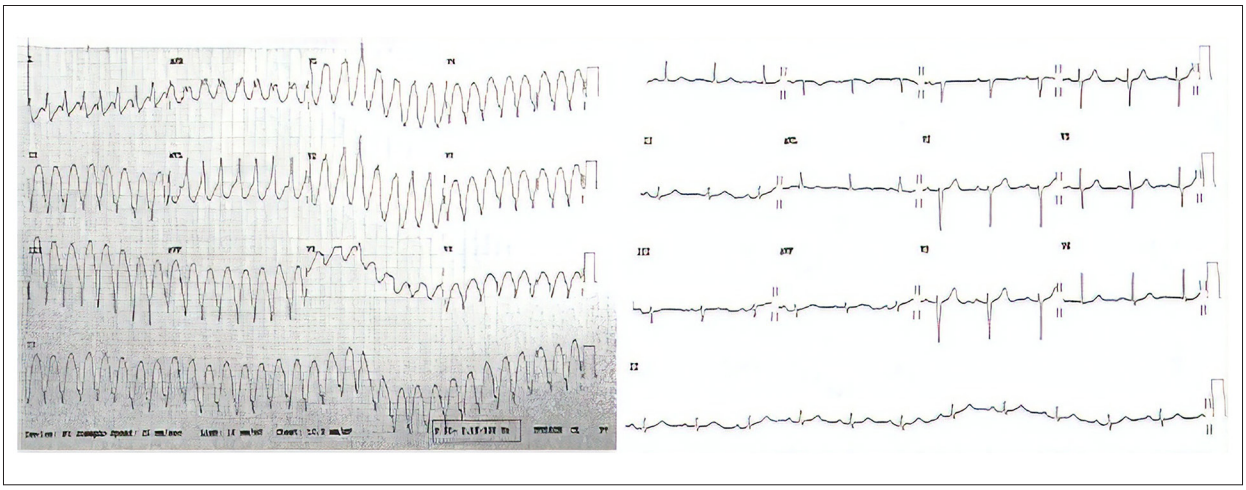

Figure 1. ECG representing a monomorphic VT and a baseline ECG in normal sinus rhythm.

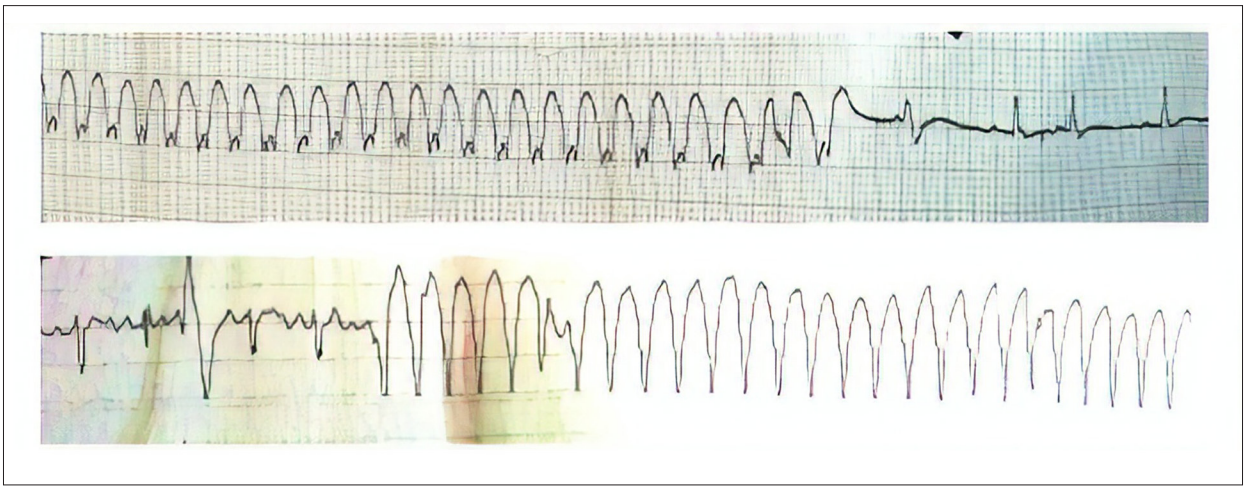

Figure 2. Tracings from ward telemetry showing the initiation of a non-sustained VT event before developing sustained events that required electrocardioversion.

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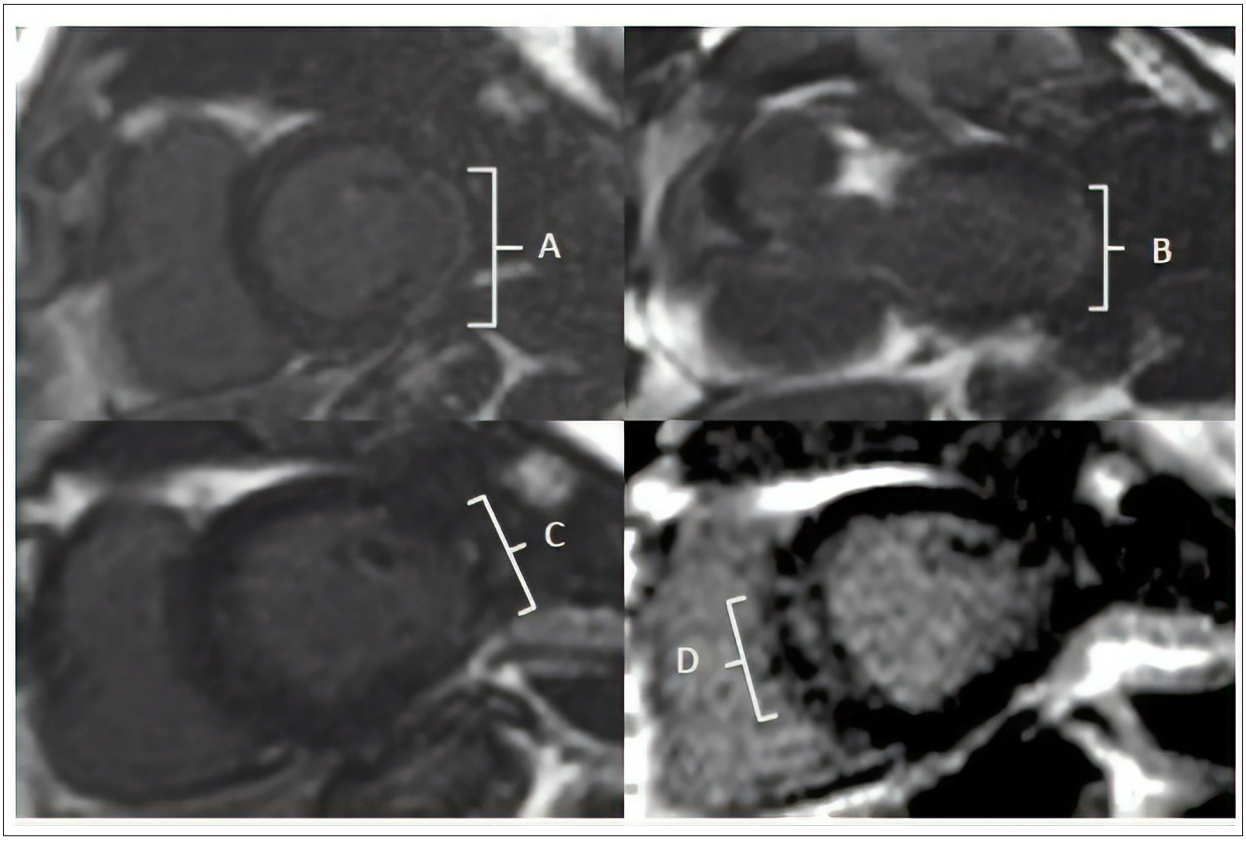

Figure 3. Cardiac MRI scan showing the extension of the scar on lateral and basal lateral walls (in A, B, and C). "D" corresponds to an area of patchy scar in the basal interventricular septal region.

he received a biphasic 200-J shock, reverting him into sinus rhythm. The patient had developed recurrent VT events treated by electrical shocks, so he was transferred to the CCU. His VT storm settled using Class antiarrhythmic. The patient's QTc was relatively high, at 465 ms, even though he had not received his evening dose of Sotalol. Relevant electrolyte tests were performed, and hypokalemia was ruled out, as his K^+ levels were normal, at 4.6 mmol/L.

An urgent coronary angiogram confirmed the patency of the coronary arteries. Cardiac MRI revealed a scar, as shown in Figure 3. He received ICD for secondary prevention and was started on Mexiletine when his arrhythmia has completely settled. Ventricular tachycardia ablation was planned as an outpatient procedure.

Discussion

We report a case of electrical storm after RF-PVI. Despite a vague history of 2 short episodes of dizziness, he had no previously recorded incidence of sustained VA. It is feasible that the 2 events of pre-syncope could be related to prior VA episodes, but they had occurred more than 2 years ago and were not frequent. However, the main presenting complaint was consistent with AF, as documented. Again, it is a speculation with no documented events. More importantly and interestingly, the VT and electric storm were more pronounced after atrial ablation, and the patient had another event later on (2 month later, when we tried weaning him off Mexiletine). This suggests that ablation may have triggered the autonomic modulation and hence the electric storm.

The patient had a structurally normal heart except for subtle hypokinetic wall motion abnormalities in rather normal LV systolic function. A similar case was reported after pulmonary vein isolation using cryoablation [1]. To the best of our knowledge, this is the first case report of a VT storm following radiofrequency pulmonary vein isolation.

The mechanism causing the VA post-procedurally is unknown; however, ventricular arrhythmias in the form of premature contractions following PVI ablation are well documented and are associated with benign prognosis [2]. One possible explanation is the increased myocardial excitability and/or ventricular autonomic modulation after ablation targeting a ganglionated plexuses around PVs [3–5]. However, the presence of a previously formed patchy scar, likely related to pericarditis,

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was responsible for the re-entry ventricular tachycardia [6], so we can easily speculate from the inhomogeneous distribution of the scar and patency of his coronaries that myo-pericarditis is the most likely diagnosis. Mexiletine (class IB) was used, which has been shown to be effective in reducing ventricular arrhythmia burden. This phenomenon of sustained VT storm after radiofrequency PVI ablation is uncommon, although it has been documented with a different modality of PV isolation (i.e., cryoballoon-based ablation) [1].

Conclusions

The incidence of ventricular ectopics after PVI ablation has been previously described, but, to the best of our knowledge, there has been no previous report of sustained ventricular storms associated with RF ablation. We attribute the pathophysiology to an increase in myocardial excitability and/or ventricular autonomic modulation, possibly secondary to the ablation of atrial ganglionated plexus. This is a quite rare phenomenon, but any subtle imaging abnormalities should be taken into consideration when pre-planning RF-PVI.

Institutions where work was done

Heart and Vascular Center, Mater Private Hospital, Dublin, Ireland

Conflict of interest

None.

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