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EP Laboratory Nightmare

Catheter Ablation of Malignant Premature Ventricular Complex Complicated by Left Main Injury

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ABSTRACT

We present a case of ventricular fibrillation triggered by a premature ventricular complex. During ablation from the left coronary cusp, the ablation catheter dislodged inside left main coronary artery, thus resulting in cardiac arrest. We immediately performed angioplasty and stent implantation, and the procedure was accomplished with a guiding catheter left inside the vessel. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:2331–5) © 2020 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/).

HISTORY OF PRESENTATION

A 49-year-old female patient was referred to our center because she had sustained 14 episodes of ventricular fibrillation (VF) and 3 implantable cardioverter-defibrillator (ICD) shocks within the last 24 h despite beta-blocker and Class III antiarrhythmic therapy. No clinical signs of heart failure were detectable by physical examination.

LEARNING OBJECTIVES

- To emphasize the anatomic complexity of the left ventricular outflow tract in the context of EP procedures.
- To highlight safety concerns when performing ablation near the coronary ostia and the need for rapid management of a potentially lethal complication.

PAST MEDICAL HISTORY

The patient's past medical history included a survived sudden cardiac death secondary to VF in 2015, followed by ICD implantation for secondary prevention.

DIFFERENTIAL DIAGNOSIS

A structural heart disease was ruled out; the electrocardiogram (ECG) showed no sign of channelopathy; and common pathogenic long QT syndrome mutations were genetically excluded.

INVESTIGATIONS

The ICD interrogation and telemetry revealed repetitive monomorphic premature ventricular complexes (PVCs) with a short coupling interval, triggering VF episodes (R-on-T phenomenon) (**Figure 1B**). A 12-lead ECG (**Figure 1A**) showed sinus rhythm, a normal QTc interval, and frequent PVCs suggestive of a left ven-

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ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

EP = electrophysiology

ICD = implantable cardioverter-defibrillator

LMCA = left main coronary artery

LVOT = left ventricular outflow tract

PVC = premature ventricular complex

RFCA = radiofrequency catheter ablation

VF = ventricular fibrillation

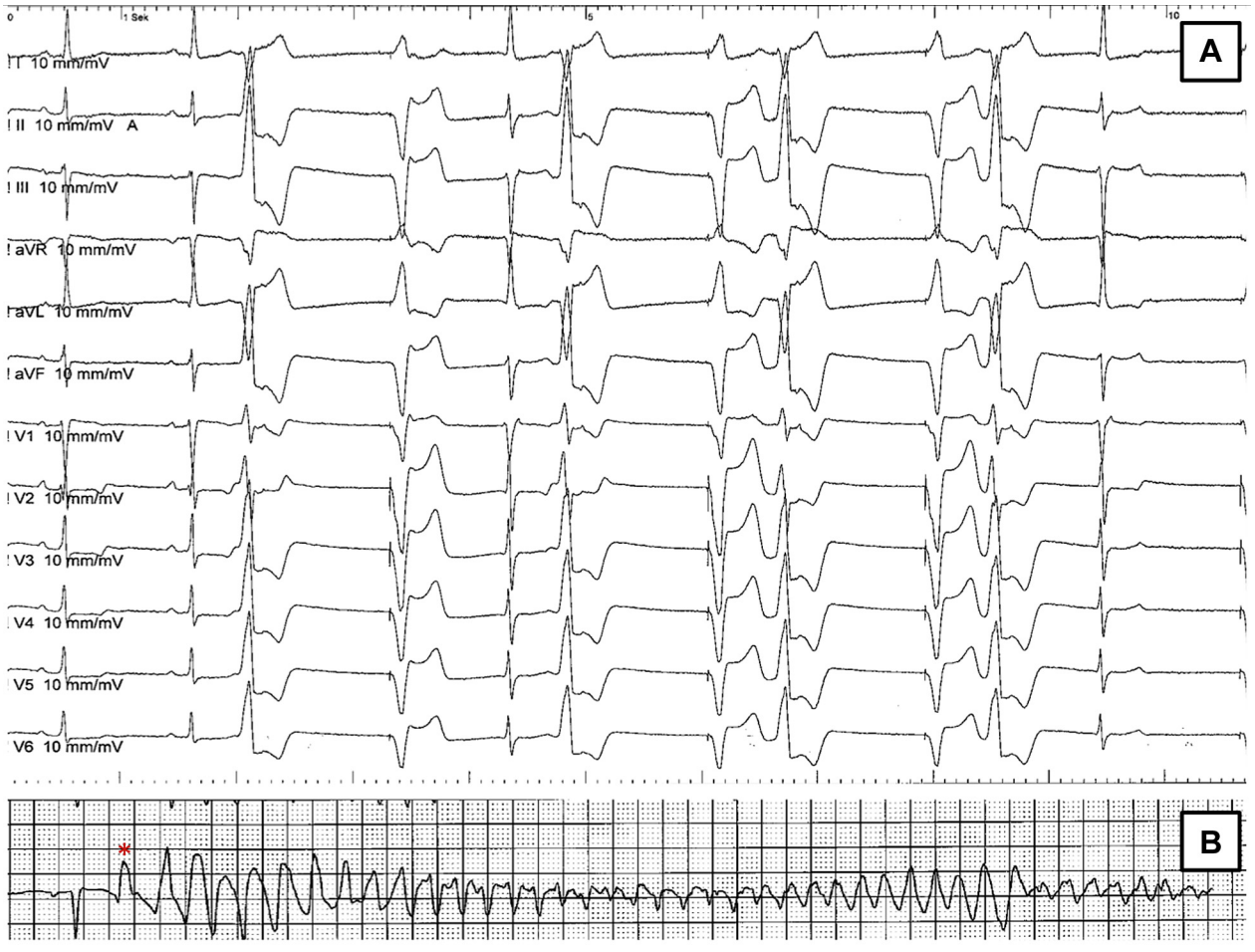
3D = 3-dimensional

tricular outflow tract (LVOT) origin (rS complex in V_1, early pre-cordial transition, inferior axis, I negative). Therefore, the clinical picture was suggestive of PVC-triggered idiopathic VF, and an electrophysiology (EP) study with PVC ablation was indicated.

MANAGEMENT

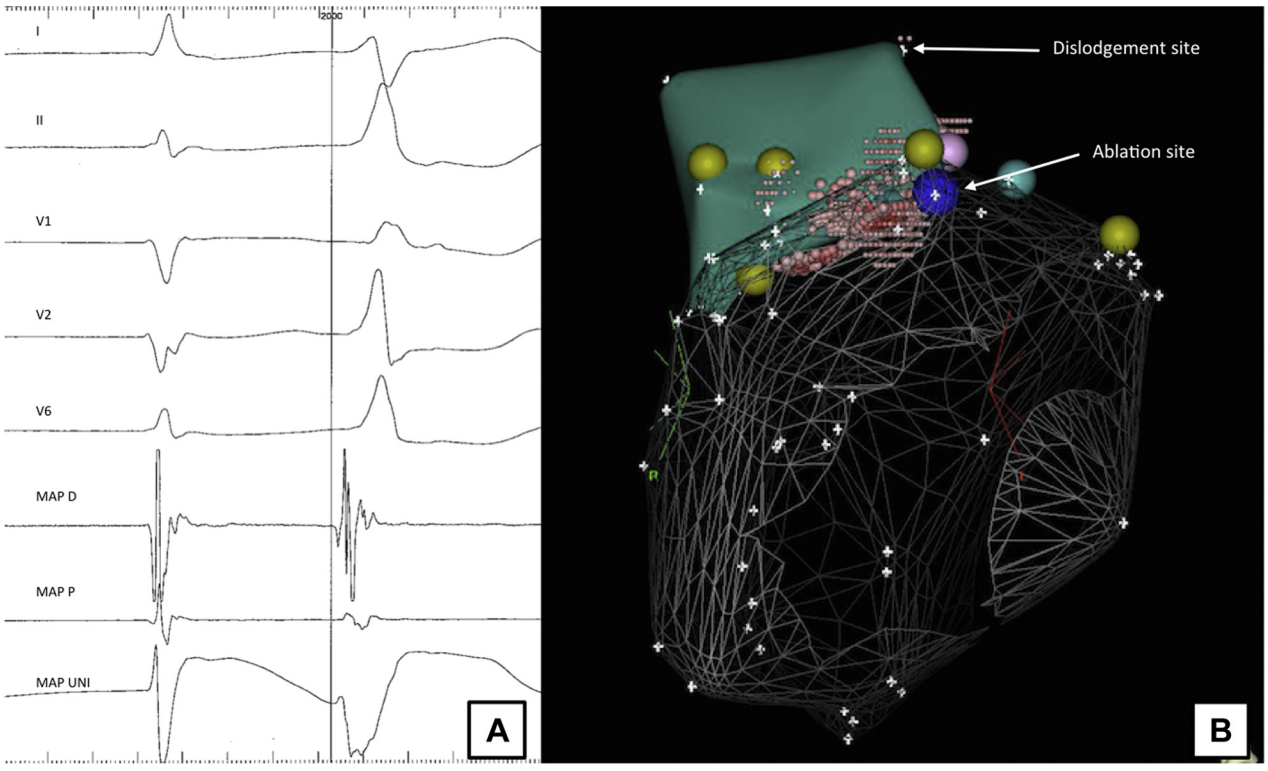
The procedure was performed without any sedation of the patient to avoid PVC suppression and because of the patient's history of allergies (propofol, midazolam, fentanyl). Programmed ventricular stimulation did not induce sustained ventricular arrhythmias. A 3-dimensional (3D) electroanatomic CARTO (Biosense Webster, Irvine, California) reconstruction of the left ventricle and LVOT was performed. Critical activation and pace-map sites were tagged in the 3D electroanatomic map (Figure 2B). The earliest activation of the PVC origin could be identified in the lower left coronary cusp (Figure 2A). Despite a nonperfect unipolar electrogram (rS morphology), the earliest bipolar signal could be identified at this site (36 ms before QRS complex onset), and irrigated-tip radiofrequency catheter ablation (RFCA) was initiated (30 W, 8 ml/min irrigation, noncontact force-sensing catheter). Despite our effort in reassuring the patient, during the first radiofrequency energy delivery she moved suddenly, and the ablation catheter became

FIGURE 1 Culprit Premature Ventricular Complexes Triggering Ventricular Fibrillation



(A) A 12-lead electrocardiogram showing sinus rhythm with a normal QT interval and frequent short coupled monomorphic premature ventricular complexes suggestive of a left ventricular outflow tract origin; intermittent paced complexes are also present. **(B)** Telemetry electrocardiographic tracing showing premature ventricular complex **(asterisk)** triggering ventricular fibrillation.

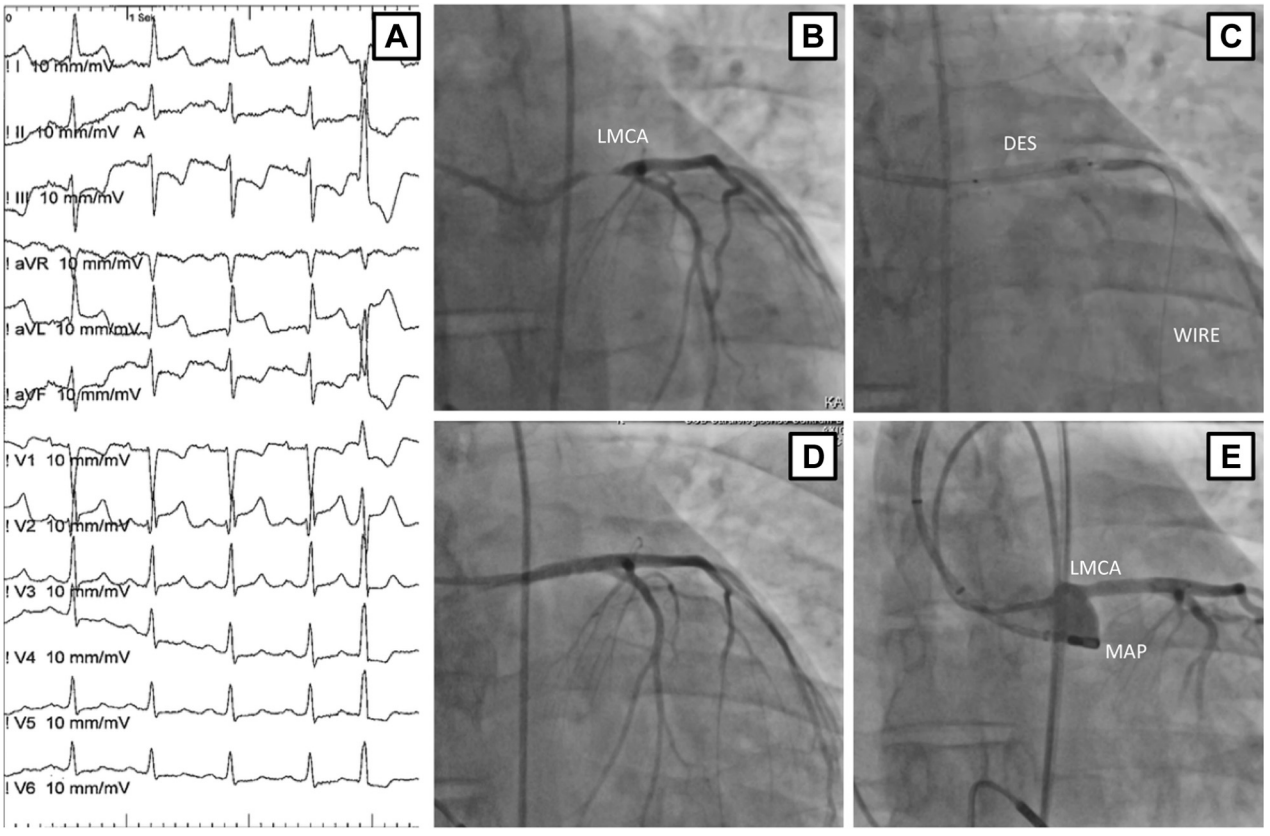
FIGURE 2 Intracavitary Signals and Anatomical Position of Successful Ablation Site



(A) Surface electrocardiographic tracings (I, II, V_1, V_2, V_6) and mapping catheter intracavitary signals recorded on the left coronary cusp during premature ventricular complexes: activation shows early bipolar signals (MAP D, MAP P) and a nonperfect morphology (rS complex) of unipolar recording (MAP UNI). **(B)** A 3-dimensional CARTO anatomic map (Biosense Webster, Irvine, California) of the left ventricle and aortic root in the left anterior oblique projection; the **blue point** marks the site of early activation and successful ablation. Analysis of the ablation grids shows the site of dislodgment in the left main coronary artery compared with the initial and finally successful ablation site.

dislodged inside the left main coronary artery (LMCA) ostium (Figure 2B), thus leading to an immediate radiofrequency interruption. The patient remained agitated and appeared to be in massive distress. A 12-lead ECG revealed ST-segment elevation (I, aVL, aVR, V_1 to V_2) along with ST-segment depression (II, III, aVF) (Figure 3A). Therefore, immediate coronary angiography was performed, and it showed LMCA occlusion (XB 3.5, 6-F, Cordis, Gaithersburg, Maryland) (Figure 3B). In the meantime, pulseless electrical activity developed in this patient, so cardiopulmonary resuscitation with orotracheal intubation was performed. A Runthrough wire (Terumo, Tokyo, Japan) was positioned in the distal left anterior descending artery, and LMCA balloon dilatation (Trek 3.0 × 20 mm, Abbott, Abbott Park, Illinois) was performed, thereby restoring subtle blood flow. Subsequently, a drug-eluting stent (3.5 × 23 mm, Xience Pro, Abbott) was successfully implanted in the LMCA, followed by Thrombolysis in Myocardial Infarction flow grade 3 (Figures 3C and 3D). Within a few minutes, the patient's cardiac function recovered, and her blood pressure regained normal values. The ST-segment recovered too, but the clinical PVCs were still present. Therefore, it was decided to continue the EP procedure. A second femoral arterial access was performed, and the LVOT was remapped: the origin of the PVC could be confirmed in the lower left coronary cusp, where a perfect pace map could also be obtained. RFCA at this site immediately eliminated the PVC. Importantly, during ablation, the XB 3.5 guiding catheter was left in the LMCA to mark and protect the ostium. Therefore, RFCA application could be safely deployed (Figure 3E). The patient was then transferred to the intensive care unit and was able to be extubated on

FIGURE 3 **Electrocardiogram After Left Main Occlusion, Coronary Angiography, Subsequent Stent Implantation With Reperfusion, and Restart of Ablation**



(A) A 12-lead electrocardiogram after left main coronary artery occlusion, showing ischemic ST-segment alterations. **(B)** Fluoroscopic right anterior oblique projection showing left main coronary artery (LMCA) occlusion, **(C)** subsequent stent implantation, and **(D)** restoration of normal flow. **(E)** Guiding catheter inside the left main coronary artery ostium as a radiological marker and protection against ablation catheter dislodgment during left coronary cusp radiofrequency ablation. DES = drug-eluting stent.

the same day. No obvious neurological deficit was present, and the echocardiogram demonstrated normal left ventricular function.

DISCUSSION

RFCA of arrhythmias arising from the aortic cusp is challenging because of the need to manipulate catheters and energy delivery close to the coronary ostia. The LMCA provides the greatest part of left ventricular perfusion, and its acute occlusion is usually associated with cardiogenic shock. Each of the aortic sinuses of Valsalva, positioned at the base of the aortic root, is in contact with the ventricular myocardium at their bases, and this structure enables mapping and ablation of arrhythmias with an outflow tract origin (1). A retrospective analysis of all described cases of severe LMCA injury during catheter ablation identified 22 cases between 1987 and 2018 (2): 86% of cases manifested dramatically as a life-threatening arrhythmia, cardiogenic shock, or severe hypotension. The in-hospital mortality rate was 32%, and direct stenting was found to be the most successful treatment strategy. Direct mechanical trauma inflicted by unintentional LMCA cannulation followed by vessel dissection was most often reported as the suspected mechanism of injury, although another study found coronary spasm to be the leading mechanism (3). In our case, it is likely that radiofrequency delivery inside the LMCA caused a severe spasm of the vessel. In cases of PVCs originating close to the coronary ostia, an understanding of a patient's anatomy and of catheter position can be helped by intracardiac echocardiography (4). However, this imaging modality is not available in every EP laboratory. In addition, in older patients, calcifications of the

coronary arteries can help in predicting their location. In the present case, integrating data from fluoroscopic imaging and 3D mapping, the position of the catheter was deemed to be at the bottom of the aortic cusp, far from the LMCA ostium, and the dislodgment resulted from a sudden, uncontrolled patient motion. Luckily, in our laboratory electrophysiologists also perform coronary angiograms and interventions, so the operators could immediately perform a coronary angiogram, identify the problem, and treat it. After this case, we started to perform a coronary angiogram to assess the coronary anatomy before delivering energy close to the coronary ostia, and, more importantly, we began to sedate the patient to avoid dislodgments when performing ablations at these sites.

FOLLOW-UP

The patient was discharged home after 2 days surveillance and remained free of any ICD therapy and VF episode for >8 months.

CONCLUSIONS

Catheter ablation in the LVOT can be complicated by proximal coronary artery injury, with a potentially dramatic outcome in cases of LMCA involvement. Coronary artery visualization with either angiography or intracardiac echocardiography should be strongly considered in aortic root ablation. Sedation of the patient is important to improve catheter stability. EP operators with experience in coronary angiograms and interventions can ensure rapid management of this rare but life-threatening complication.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS ablation, complication, coronary angiography, ventricular fibrillation