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CASE REPORT

ADVANCED

CLINICAL CASE

Successful Open Chest Epicardial Ablation for Refractory Ventricular Tachycardia in an LVAD Recipient

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ABSTRACT

A patient with history of dilated cardiomyopathy, a cardiac resynchronization therapy defibrillator, and endocardial ablation presented for refractory ventricular tachycardia 3 years after implantation of a Jarvik 2000 left ventricular assist device (Jarvik Heart, Inc., New York, New York). Open-chest epicardial ablation safely and effectively terminated the arrhythmia, without ventricular tachycardia recurrence at 9-month follow-up and in the absence of complications during the hospital stay. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:1055–60) © 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

Ventricular arrhythmias (VAs) are common in patients with a continuous-flow left ventricular assist device (LVAD) (1). No antiarrhythmic drug has been shown to improve survival compared with implantable cardioverter-defibrillator (ICD) therapy. However, both recurrent VAs and ICD shocks have been significantly associated with worsening morbidity and mortality (2,3).

LEARNING OBJECTIVES

- To recognize the cause of incessant VT in LVAD recipients by a correct application of differential diagnosis.
- To evaluate treatment options for incessant VT in LVAD recipients on the basis of clinical presentation and LVAD type.
- To understand that for patients with an LVAD, epicardial ablation of recurrent VT is safe and feasible and improves clinical outcomes.

Ablation of ventricular tachycardia (VT) in patients with LVADs has been shown to suppress recurrent VAs effectively. Additionally, freedom from recurrent VTs and ICD shocks after ablation has been associated with 1-year improved survival (4). However, there are limited reports of VT ablation in LVAD recipients, mainly because of technical and procedural challenges unique to these patients. Furthermore, endocardial ablation has not yet been described in the Jarvik 2000 LVAD (Jarvik Heart, Inc., New York, New York), possibly because of its totally intraventricular pump.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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ABBREVIATIONS AND ACRONYMS

CRT-D = cardiac resynchronization therapy defibrillator

ICD = implantable cardioverter-defibrillator

LV = left ventricular

LVAD = left ventricular assist device

RF = radiofrequency

RV = right ventricular

VA = ventricular arrhythmia

VT = ventricular tachycardia

HISTORY OF PRESENTATION

A 72-year-old woman, with a cardiac resynchronization therapy defibrillator (CRT-D) and an LVAD (implanted 10 and 3 years earlier, respectively), was admitted to our intensive care unit (Centro Cardiologico Monzino, Milan, Italy) for an electrical storm. In the previous 30 days, she had experienced multiple sustained VTs requiring antitachycardia pacing and ICD shocks (Figures 1A and 1B).

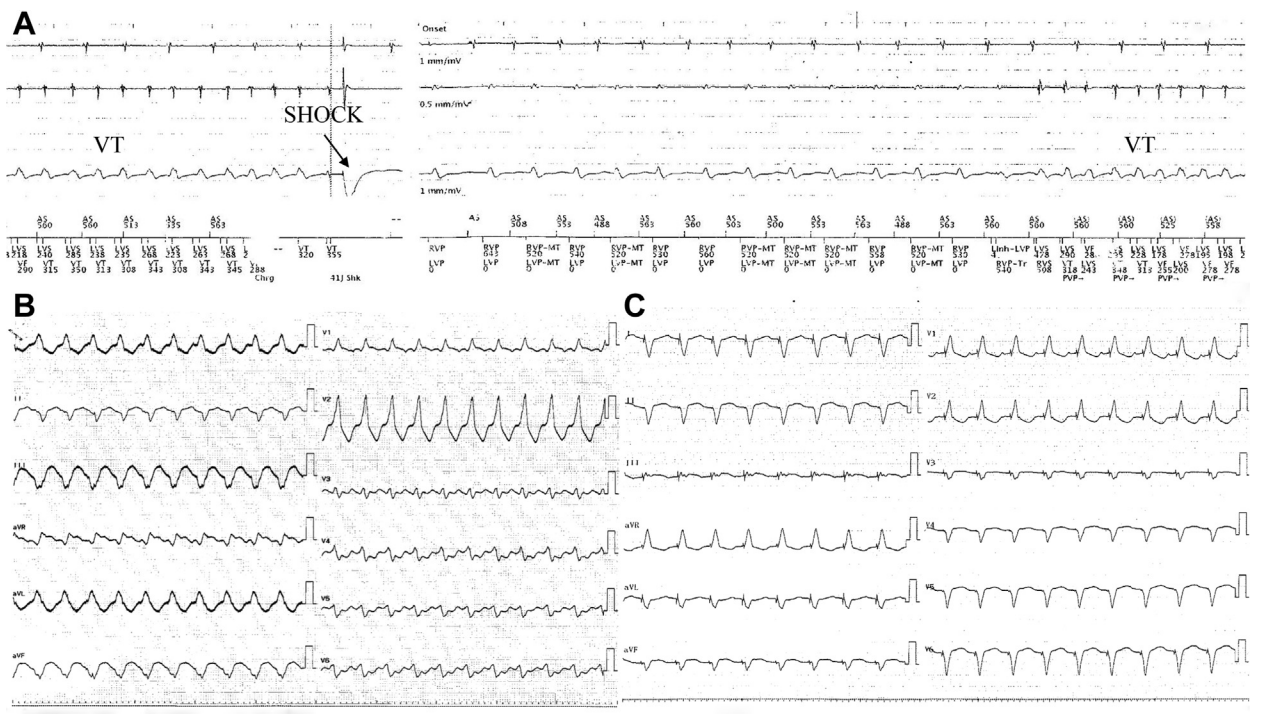
On examination, her mean arterial pressure was 80 mm Hg, and her heart rate was 80 beats/min. There were no signs of peripheral edema. Common LVAD parameters were normal (speed, 3 corresponding to 10.000 rpm and to a 4 to 5 l/min flow; pump power, 5 to 6 W; normal intermittent low speed, equivalent to 8 s every minute). An electrocardiogram showed a biventricular paced rhythm (Figure 1C).

Despite multiple antiarrhythmic drug combinations, both orally (mexiletine, flecainide, amiodarone) and intravenously (lidocaine and low-dose propranolol), sustained VTs kept recurring, leading to low cardiac output syndrome and ischemic colitis, treated with parenteral nutrition and antibiotic therapy for 14 days. Pacing inactivation did not reduce the episodes of arrhythmia, and the decision was to maintain the CRT.

PAST MEDICAL HISTORY

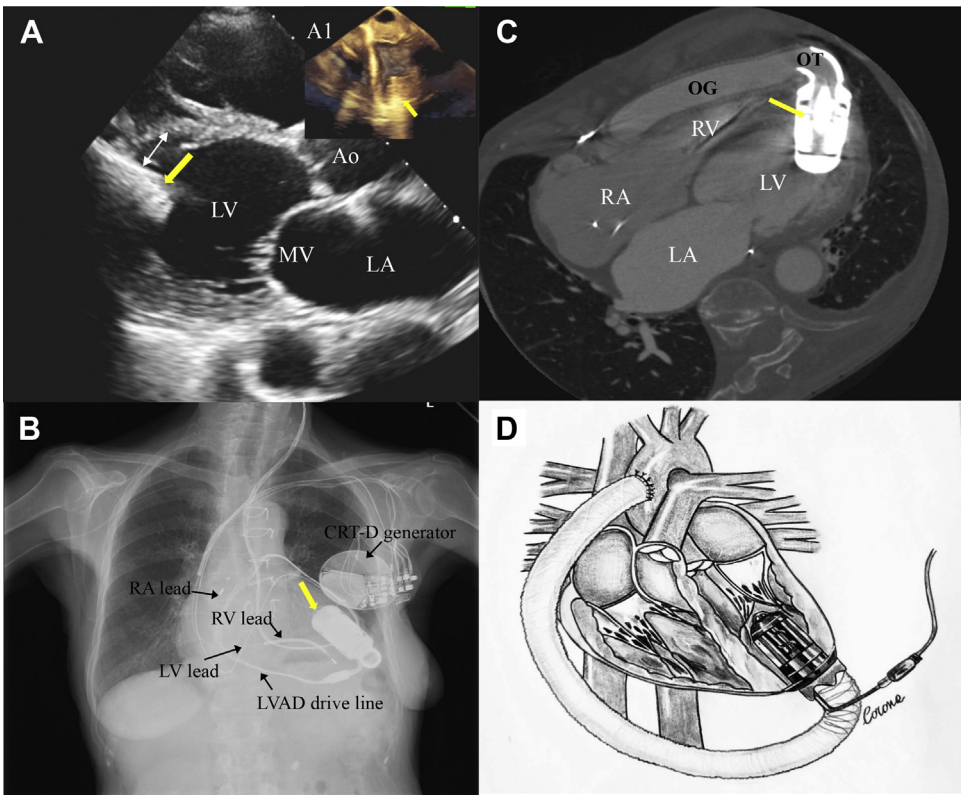
The patient had a known history of nonischemic dilated cardiomyopathy with a 20% left ventricular (LV) ejection fraction since 2002. She underwent CRT-D (Boston Scientific, Marlborough, Massachusetts) in 2009 and endocardial radiofrequency (RF) ablation of multiple VT configurations in 2015.Thereafter, while taking amiodarone and bisoprolol, she remained free of VT recurrences. In 2016, when she was in Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) class 3, an LVAD was implanted, as destination therapy, through off-pump left mini-thoracotomy in combination with J-mini-sternotomy.

FIGURE 1 Intracardiac Electrogram Recorded by the Implantable Cardioverter-Defibrillator and 12-Lead Electrocardiograms



(A) Recurrent ventricular tachycardia (VT) after implantable cardioverter-defibrillator shock therapy **(arrow). (B)** Electrocardiogram during ventricular tachycardia presented a different morphology from **(C)** an electrocardiogram during biventricular pacing.

FIGURE 2 Imaging Investigations



(A) Transthoracic and **(A1)** 3-dimensional transesophageal echocardiography, **(B)** chest radiograph, and **(C)** computed tomography oblique multiplanar reconstruction revealing the correct position of the left ventricular assist device (LVAD) pump **(yellow arrows). (A) White double arrow** points to a pump-interventricular septum distance of 7 mm, **(B)** absence of pulmonary edema and correct position of cardiac resynchronization therapy defibrillator (CRT-D) leads, and **(C)** absence of pump or outflow tract (OT) thrombosis, and relationship with adjacent tissues. **(D)** Drawing by the author showing the Jarvik 2000 (Jarvik Heart, Inc., New York, New York): a nonpulsatile axial-flow left ventricular assist device. It consists of a miniaturized intraventricular blood pump that lacks a real inflow conduit, unlike other devices. Ao = aorta; LA: left atrium; LV = left ventricle; MV = mitral valve; OG = outflow graft; RA = right atrium; RV = right ventricle.

DIFFERENTIAL DIAGNOSIS

Multiple mechanisms can trigger VTs in LVAD recipients: electrolyte disturbances, inotropic or vasopressor drugs, profibrotic remodeling around the inflow cannula, evolution of scar, LV pacing near a scar, volume depletion, and suction events.

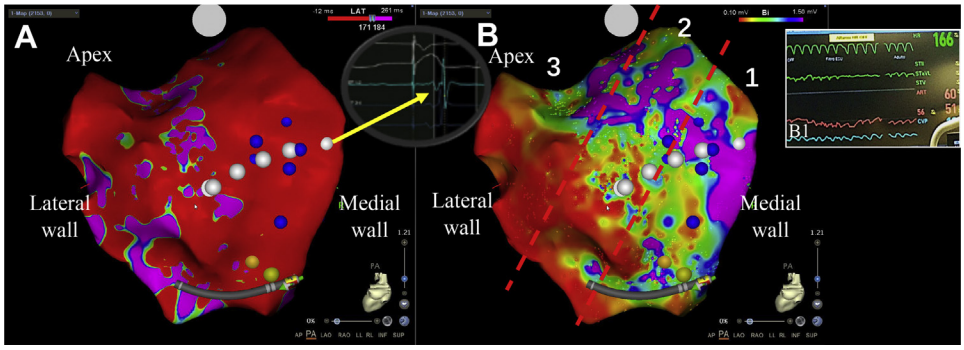
INVESTIGATIONS

Transthoracic echocardiography, computed tomography, and the chest radiograph (Figures 2A to 2C) revealed the absence of LVAD obstruction, suction events, displacement of the interventricular septum, pump thrombosis, and coronary stenosis. There was moderate aortic valve regurgitation with normal pulmonary artery systolic pressure. Blood count and electrolyte values were normal.

An electrocardiogram during VT (Figure 1B) showed characteristics suggestive of an epicardial origin. The clinical VT had a cycle length of 400 ms, right bundle branch configuration, superior axis, and transition $>V_3$, findings suggesting an inferoapical and inferolateral exit site. The clues to the epicardial origin of the VT were the history of nonischemic cardiomyopathy and the following specific criteria: intrinsicoid deflection time >85 ms in lead V_2 and time to earliest QRS complex nadir in precordial leads >121 ms.

In consideration of: 1) malignant VT, despite optimal medical treatment and LVAD support; 2) lack of evidence on the feasibility and safety of

FIGURE 3 3-Dimensional Electroanatomic Map of the Epicardial Left Ventricle



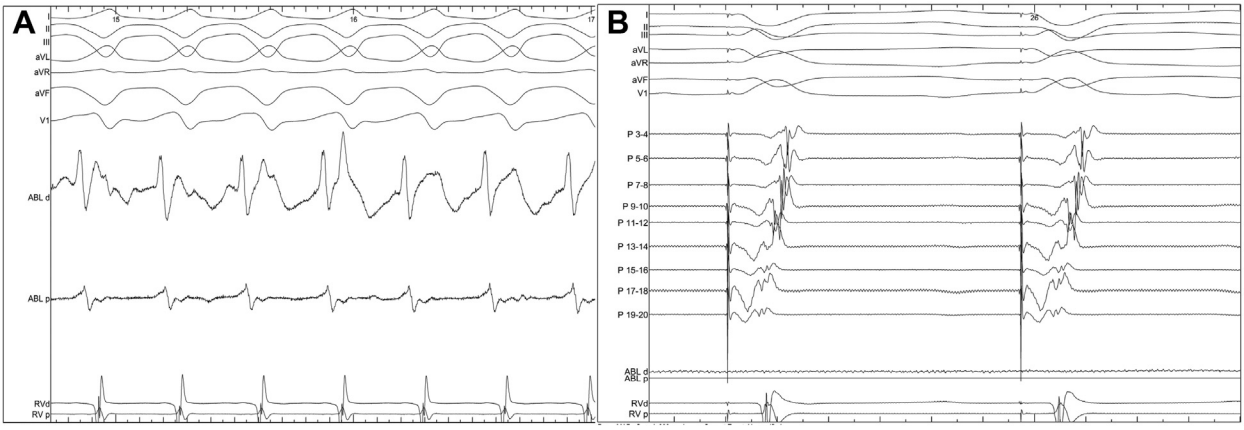
(A and B) Posteroanterior (PA) view. **(A)** The activation map in pacing identifies several small areas of late potentials in **purple;** the **blue** and **yellow dots** correspond to pathological fragmented potentials **(oval inset),** and the **line of white dots** represents a late potentials channel. **(B)** Bipolar voltage map: basal *(1),* medial *(2),* and apical *(3)* portions. Low-voltage areas, corresponding to scarred areas, appear in **red. (B1)** Ventricular tachycardia induced by standard right ventricular stimulation. AP = anteroposterior; INF = inferior; LAO = left anterior oblique; LL = left lateral; RAO = right anterior oblique; RL = right lateral; SUP = superior.

endocardial ablation in an LVAD; 3) the VT configurations suggesting their epicardial substrate; and 4) the possibility to create transmural lesions easily by using a surgical epicardial approach, we decided to perform a side-to-side surgeon-electrophysiologist procedure of open chest epicardial ablation as a "last resort" therapy.

MANAGEMENT

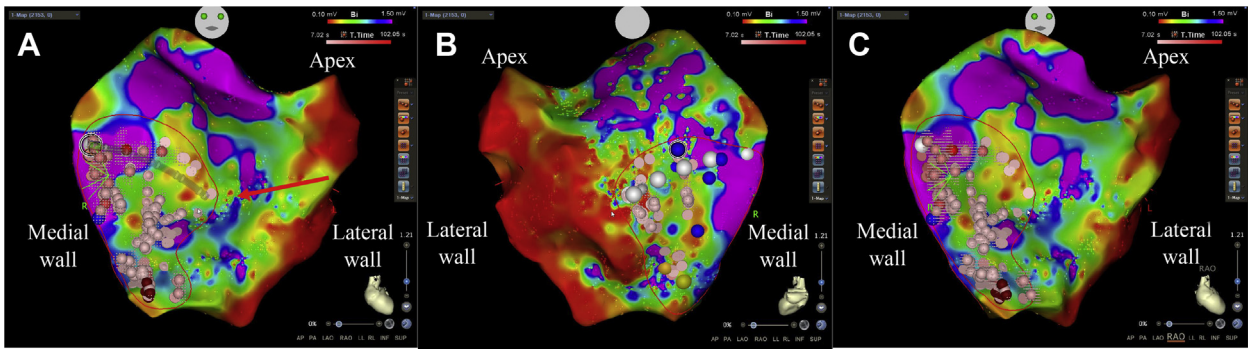
We performed the procedure in the hybrid operating room, with the patient under general anesthesia and with the use of continuous transesophageal echocardiography monitoring. Heparin was administrated to achieve activated clotting time >300 s. LVAD flow

FIGURE 4 Intracardiac Electrograms During Ventricular Tachycardia Ablation



(A) The site of successful ablation of ventricular tachycardia-1. The tracing shows a pre-systolic potential that turned out to be the successful site of ventricular tachycardia-1 termination. **(B)** Ventricular tachycardia-2 was abolished by ablating fragmented and late potentials uncovered by paced rhythm. An example of the pattern of late potentials is shown. Please note the bracketing pattern of late potentials that fall in the offset of the QRS complex.

FIGURE 5 3-Dimensional Electroanatomic Map Showing the Ablation Sites



(A) Anteroposterior (AP), **(B)** posteroanterior (B), and **(C)** right anterior oblique (RAO) view. Bipolar maps in pacing. The **pink and red dots** show the radiofrequency application sites. The **blue and yellow dots** correspond to pathological potentials. The **line of white dots** represents a late potential channel. Ablation sites (basal and medial portions, **red outlines**) and late potentials homogenization are shown. **(A) Red arrow** indicates the ventricular tachycardia interruption site. Abbreviations are as in **Figure 3**.

was continued unchanged, and pacing was maintained. Patches were arranged to limit electroanatomic mapping interference (Supplemental Figures 1A to 1C). A diagnostic quadripolar catheter was advanced to the right ventricular (RV) apex through the femoral access.

Redo left anterior thoracotomy at the fifth intercostal space, fifth rib disarticulation, and lysis of adhesions were performed, exposing the whole LV surface (Video 1).

Epicardial mapping was performed, with a decapolar catheter (DECANAV, Biosense Webster, Inc., Diamond Bar, California), using a 3-dimensional electroanatomic system, CARTO3 (Biosense Webster, Inc.). Pathological low-voltage scar areas (≤1 mV) were identified on the inferolateral wall (**Figures 3A and 3B**), where fractionated and late potentials were recorded. The epicardial map was completed using a 3.5-mm-tip irrigated catheter (ThermoCool SmartTouch, Biosense Webster, Inc.).

Two VTs were induced by standard RV stimulation (**Figure 3B1**), and LVAD support allowed for activation mapping of both.

The first VT had a cycle length of 440 ms, right bundle branch block configuration, superior axis, and transition $>V_3$. Electrogram tracings showed presystolic potentials (**Figure 4A**) at the scar area's apical boundary in the lateral wall. RF applications at 30 to 40 W (target temperatures <42°C) terminated the tachycardia (**Figure 5A**). The second slightly different VT (cycle length 375 ms, transition $>V_4$) was mapped without identifying a diastolic and pre-systolic activity. Thus, fragmented and late potentials uncovered by paced rhythm (**Figure 4B**) were ablated within the inferolateral wall to homogenize the scar (**Figures 5A to 5C**).

Ablation lesions were performed with the surgical ablation tool at 30 W for 40 s and covered the whole region of interest. At the end of the procedure, the abolition of all late potentials and substrate homogenization were assessed by an accurate remapping. In our case, the pure anatomic approach (with diastolic activity recording during VT), without entrainment maneuvers before RF delivery, effectively terminated the arrhythmia. A final standard RV stimulation demonstrated complete success with no VT inducibility. Thoracotomy closure was performed using routine protocol.

The surgical procedure was well tolerated, and the patient had an uncomplicated postoperative course and no VA recurrences at the telemetry monitoring. Pre-discharge transthoracic echocardiography showed regular LVAD function without pericardial effusion. The patient was discharged on postoperative day 13.

DISCUSSION

Because VTs may be well tolerated, there is still debate regarding ablation in LVAD recipients. However, refractory VTs may lead to distress and RV dysfunction and consequently may worsen LVAD function. Catheter ablation is an established and effective treatment because LVAD support allows for hemodynamic tolerance of otherwise unstable VTs, thus favoring detailed electroanatomic mapping.

Most VTs are caused by the pre-existent arrhythmogenic substrate related to the underlying cardiomyopathy, rather than by a new substrate related to LVAD surgery itself and the inflow cannula (4). Pre-LVAD VTs are the main predictors of post-LVAD VTs (1). Furthermore, cannula-adjacent VTs frequently occur earlier in patients with a pre-existing apical scar, the usual ablation target.

In patients with previous Vas who are undergoing LVAD implantation, optimal management is still unknown. To date, performing ablation before LVAD implantation represents an effective treatment option if VTs are documented. Conversely, recurrent VTs after ablation appear to be more common in patients without known pre-LVAD VAs, a finding suggesting a new LVAD-related mechanism that may not be fully treatable by endocardial ablation (4). An alternative option is to perform substrate-guided ablation during LVAD implantation (5).

There are some technical challenges of VT treatment in LVAD recipients: potential for catheter entrapment, although not described yet, difficult maneuverability within a decompressed cavity, electroanatomic mapping interference, and a more challenging retrograde approach resulting from the aortic valve closure often seen (6).

No endocardial ablation procedure cases are reported in patients with an LVAD, perhaps because of the higher perceived risk related to the totally intraventricular turbine (**Figure 2D**). Thus, surgical ablation of recurrent VTs can be considered during or after LVAD implantation in patients with evidence of epicardial arrhythmias, but further data are needed (6,7).

FOLLOW-UP

At 9-month follow-up, no additional VAs occurred under treatment with oral amiodarone 200 mg/day in combination with propranolol 80 mg/day for the first month, followed by bisoprolol 2.5 mg/day monotherapy, with clinical improvement.

CONCLUSIONS

This case demonstrates that in LVAD recipients with incessant and drug-refractory VTs, the hybrid procedure of electroanatomic mapping and epicardial ablation through an open chest procedure is feasible and effective. This approach promotes fast recovery, given the limited surgical access, and an overall better clinical outcome. Interdisciplinary collaboration between the cardiac surgeon and the electrophysiologist is needed in arrhythmia management in these delicate patients.

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KEY WORDS epicardial ablation, Jarvik 2000, LVAD, ventricular tachycardia

APPENDIX For a supplemental video and figure, please see the online version of this paper.