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HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE

BEGINNER

A Multidisciplinary Approach to Electrical Instability and Cardiogenic Shock in Acute Myocardial Infarction

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ABSTRACT

New cardiogenic shock classifications allow prompt recognition and management of complications of acute coronary syndrome. A 59-year-old man presented after a delayed left anterior descending coronary artery ST-segment elevation myocardial infarction in Society of Cardiovascular Angiography and Interventions stage E cardiogenic shock and ventricular tachycardia storm. He underwent revascularization of the left anterior descending artery, percutaneous left ventricular assist device bridged to permanent assist device placement, epicardial and endocardial ventricular tachycardia ablation, and iatrogenic closure of an atrial septal defect. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:2053-9) © 2020 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/).

CASE DESCRIPTION

A 59-year-old man with diabetes (hemoglobin A1c 10%) presented to his local hospital with crushing substernal chest pain and was found to have an anterior ST-segment elevation myocardial infarction (STEMI). Blood pressure was 103/71 mm Hg, heart rate 95 beats/min, pulse oximetry 98% on 2L, and respiratory rate 18 breaths/min. Coronary angiography demonstrated an occluded proximal left anterior descending coronary artery (LAD) and a 90% ostial ramus lesion. Despite multiple attempts, there was failure to establish flow in the LAD; the ostial ramus lesion was stented to salvage some myocardium (Video 1, **Figure 1**). He was admitted to the intensive care unit after sustaining cardiac arrest caused by a monomorphic ventricular tachycardia (VT) storm that required direct current cardioversion in addition to amiodarone and lidocaine infusions. On hospital day 4, a cardiac resynchronization therapy-defibrillator (CRT-D) was placed. He subsequently had another VT storm and received 14 shocks from his CRT-D. He was intubated, sedated, and subsequently transferred to our institution (Loma Linda University Medical

LEARNING OBJECTIVES

- To discuss the rationale for revascularization in late-presenting myocardial infarction.
- To elaborate on the decision-making process for escalation of LV support.
- To discuss the role of ventricular tachycardia ablation during and after LVAD implantation.
- To discuss the rationale for percutaneous atrial septal defect closure following an iatrogenic shunt in a patient with LVAD.

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ABBREVIATIONS AND ACRONYMS

CPO = cardiac power output

CRT-D = cardiac resynchronization therapy defibrillator

LAD = left anterior descending (coronary artery)

LV = left ventricular

LVAD = left ventricular assist device

PCI = percutaneous coronary intervention

RV = right ventricular

STEMI = ST-segment elevation myocardial infarction

TIMI = Thrombolysis In Myocardial Infarction

VT = ventricular tachycardia

Center, Loma Linda, California) for evaluation for left ventricular (LV) assist device (LVAD) placement. On arrival to our hospital, his heart rate was 97 beats/min, and his blood pressure was 96/71 mm Hg while he was receiving infusions of amiodarone, lidocaine, epinephrine, and norepinephrine. His transthoracic echocardiogram demonstrated a LV ejection fraction of 10% to 15% with akinesis of the basal to apical anterior, anteroseptal, anterolateral, and lateral walls, as well as the apical inferior and inferolateral walls (Video 2).

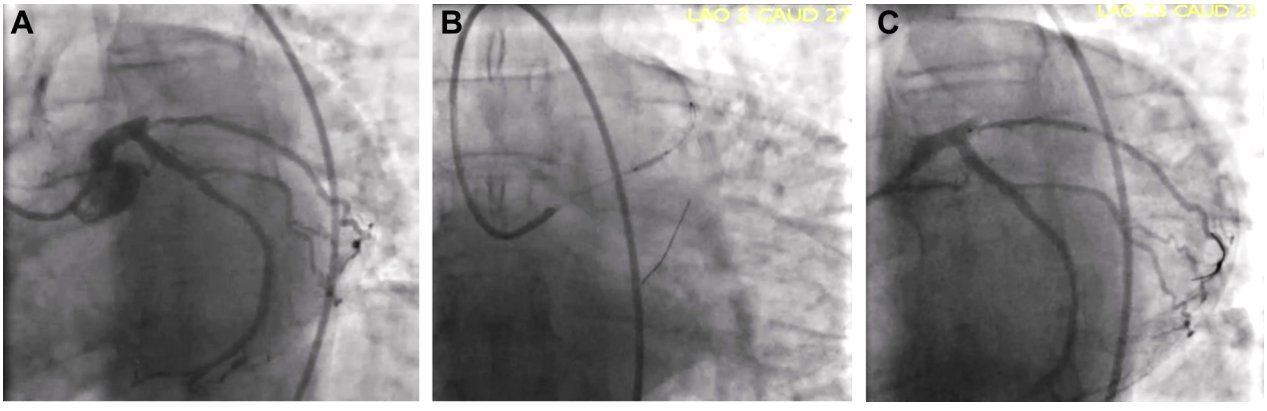
Given his electrical and hemodynamic instability, we proceeded with right-sided heart catheterization, insertion of a percutaneous LVAD, and revascularization of the LAD. An Impella CP device (Abiomed, Danvers, Massachusetts) was placed before percutaneous coronary intervention (PCI) for LV unloading. Angiography revealed Thrombolysis In Myocardial Infarction (TIMI) flow grade 0 in the LAD; the lesion, which consisted of organized thrombus, was crossed with a Pilot 200 wire (Abbott Vascular, Santa Clara, California) with a Finecross catheter (Terumo, Somerset, New Jersey) for support. After aspiration thrombectomy and multiple adenosine injections through a Pronto catheter (Vascular Solutions, Inc., Minneapolis, Minnesota), TIMI flow grade 2was restored, and a drug-eluting stent was deployed (Video 3, **Figure 2**). A Swan-Ganz catheter (Edwards Lifesciences, Irvine, California) was placed for hemodynamic monitoring, which demonstrated the following: right atrial pressure, 14 mm Hg; pulmonary artery pressure, 43/23 mm Hg; pulmonary capillary wedge pressure, 27 mm Hg (pulmonary capillary wedge pressure greater than pulmonary artery diastolic pressure as a result of functional mitral regurgitation); Fick cardiac output, 1.61; pulmonary artery saturation, 46%; pulmonary artery pressure index, 1.4; and cardiac power output (CPO), 0.28 (calculated by $\frac{MAP \times CO}{451}$) (where MAP is mean arterial pressure and CO is cardiac output).

CLINICAL PERSPECTIVE 1: WHY REVASCULARIZE THE LAD SO LATE AFTER STEMI PRESENTATION?

Primary PCI with a door-to-balloon time of <90 min is the standard of care for patients with STEMI (1). Notably, late-presenting patients with STEMI have not been shown to benefit from revascularization unless they have persistent symptoms of ischemia (2,3). However, these studies have excluded patients in cardiogenic shock and with electrical instability (3,4). Although monomorphic VT is more commonly associated with re-entrant circuits in myocardial scar, we could not definitively rule out active ischemia as a reason for his arrhythmias.

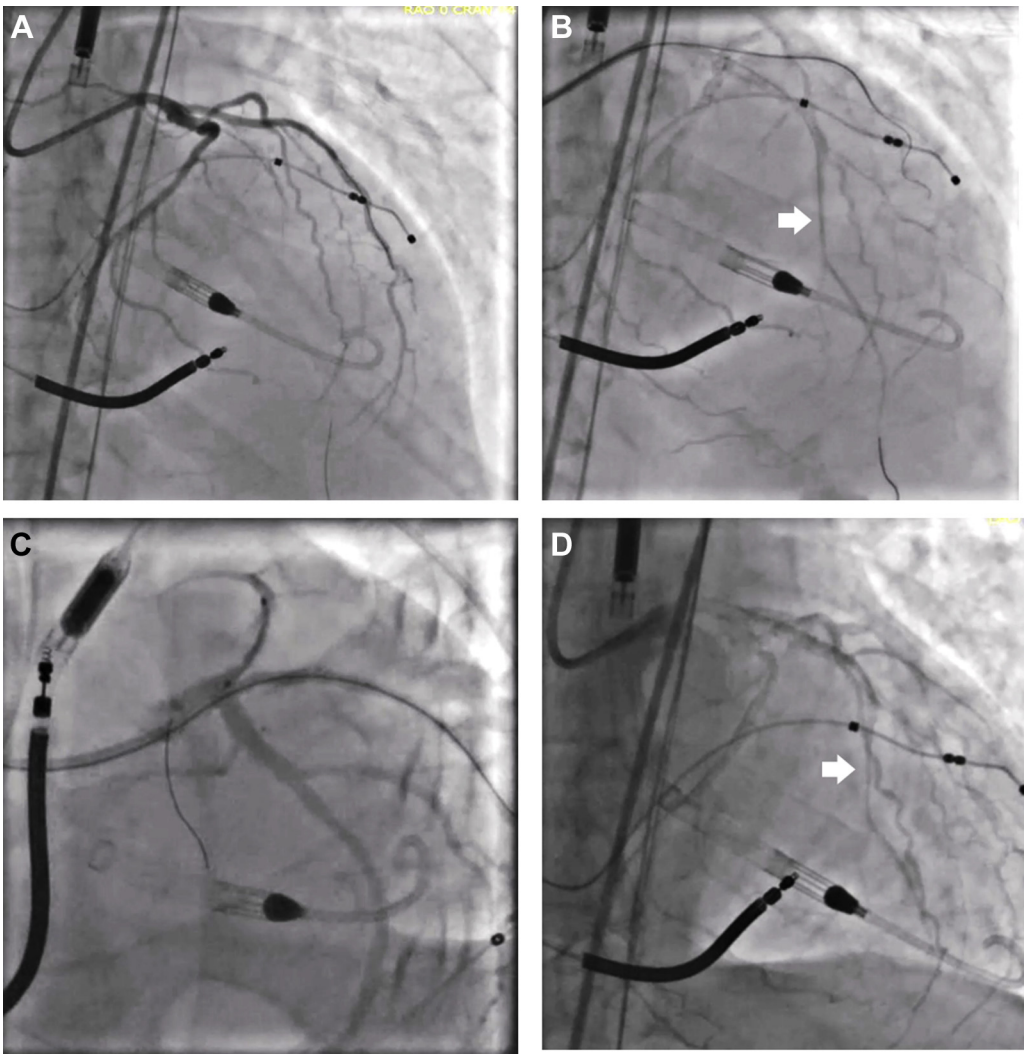
Our patient's PCI was complicated by ventricular fibrillation, resulting in 47 CRT-D shocks, 20 external direct current cardioversions, and over 40 min of cardiopulmonary resuscitation before he returned to an A-sense V-paced rhythm. He returned to the intensive care unit on a regimen of lidocaine and amiodarone, sedated with midazolam and propofol, as well as pressor and inotropic support with epinephrine, dobutamine, norepinephrine, and

FIGURE 1 Initial Cardiac Catheterization Images From the Local Hospital



(A and B) Angioplasty of a 100% thrombotic occlusion of the left anterior descending artery was unsuccessful. **(C)** Therefore, a drug-eluting stent was placed in the ramus intermedius.

FIGURE 2 **Percutaneous Coronary Intervention at Loma Linda University Medical Center**



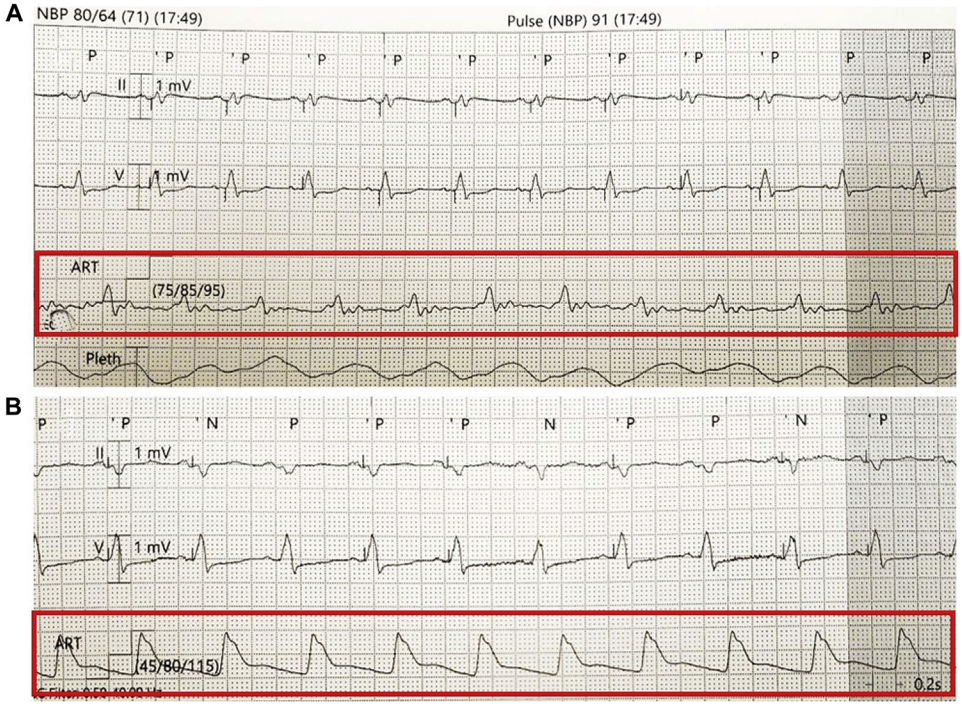
After Impella device (Abiomed, Danvers, Massachusetts) placement, **(A and B)** the left anterior descending artery lesion crossed, and a **(C)** drug-eluting stent was placed, with **(D)** faint Thrombolysis In Myocardial Infarction flow grade 2 **(arrow, D)**.

dopamine infusions. He was dependent on the Impella CP for cardiac output with very minimal pulsatility, but his intrinsic cardiac contractility improved after 3 days of LV unloading, with a return of a pulsatile waveform and improving renal, hepatic, and mental function (Figure 3). However, the following day, he again had sustained monomorphic VT (Figure 4), leading to hemodynamic deterioration, with persistently reduced CPO (<0.6) and worsening end-organ dysfunction. After a multidisciplinary team discussion that included advanced heart failure, interventional cardiology, electrophysiology, and cardiothoracic surgery, it was decided to proceed with LVAD as destination therapy. Subsequently, a HeartMate 3 (HM3) LVAD (Abbott, North Chicago, Illinois) was implanted.

CLINICAL PERSPECTIVE 2: WHEN SHOULD ESCALATION OF THERAPY BE CONSIDERED IN CARDIOGENIC SHOCK?

The decision to escalate LV support should be based on the patient's hemodynamic requirements, end-organ dysfunction, anatomic feasibility, and long-term plan (either native heart recovery or bridge to LVAD or cardiac transplantation). The National

FIGURE 3 Improved Pulsatility on Arterial Line After Unloading With Impella CP



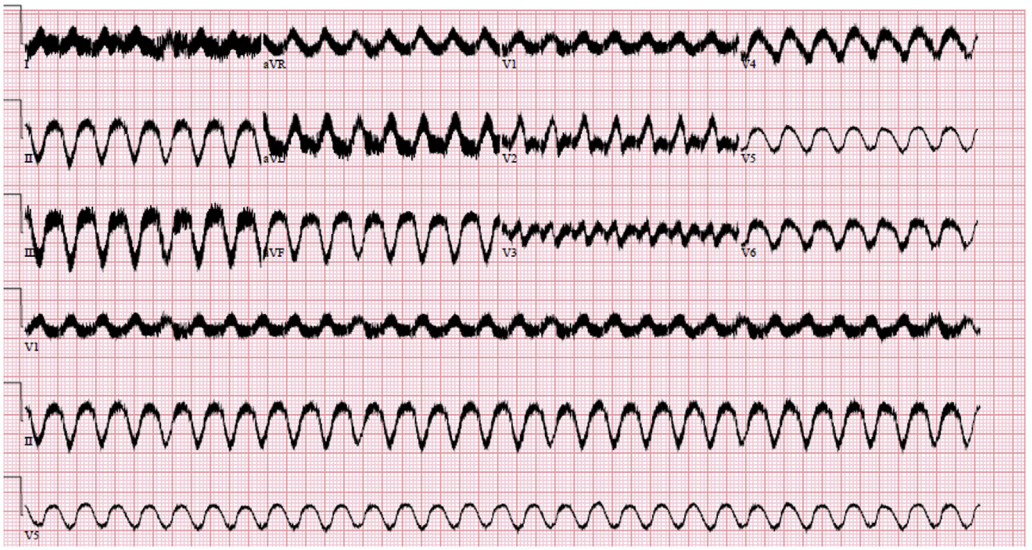
(A) Low pulsatility immediately on return to the intensive care unit from the cardiac catheterization laboratory, which **(B)** improved significantly after 24 h of unloading with an Impella CP device (Abiomed, Danvers, Massachusetts). ART = arterial line tracing; N = native; NBP = noninvasive blood pressure; P = paced; Pleth = plethysmograph.

Cardiogenic Shock Initiative, whose protocol we follow, recommends the use of CPO <0.6, which portends a poor outcome in cardiogenic shock, as a parameter for considering escalation of LV support (5). Anatomic considerations include the caliber of the femoral or axillary artery or other vascular disease of the aorta, the presence and severity of aortic stenosis, and the presence of a ventricular septal defect. In our case, the patient presented with Society of Cardiovascular Angiography and Interventions stage D/E cardiogenic shock and Interagency Registry for Mechanically Assisted Circulatory Support profile 1, and placement of an Impella CP first provided the necessary hemodynamic support for PCI and continued LV unloading (6,7). After LV unloading, his right ventricular (RV) function improved, so upgrade to an LVAD rather than biventricular support was pursued. He was not a candidate for heart transplantation given his poorly controlled diabetes.

In view of the recurrent episodes of hemodynamically unstable VT, intraoperative mapping and ablation were performed during the implantation of the HeartMate 3 device (Video 4, **Figure 5**). Using an HD grid mapping catheter (Abbott, St. Paul, Minnesota), the epicardial substrate was extensively mapped, and areas of scar were encircled with the cryoablation probe. Pacing within the encircled lesions indicated epicardial scar core isolation. However, after creation of the core for the LVAD, there was evidence of endocardial capture despite attempted endocardial cryoablation, thus indicating that the lesions were not transmural.

He continued having episodes of VT despite treatment with amiodarone, lidocaine, and procainamide (**Figure 4**), so endocardial VT ablation was performed on post-operative day 2 (Video 5, **Figure 6**). Transseptal access was performed to map and ablate within the LV endocardium. Intracardiac echocardiography revealed that the inflow cannula was abutting the LV septum, which correlated with the best pace map for the VT (Videos 5A and 5B). Extensive ablation was performed from both the RV and LV aspects of this area (Video 5C). Following this procedure, a second VT configuration was inducible (Video 5D) that appeared to exit the basal septum. Extensive

FIGURE 4 Clinical Ventricular Tachycardia

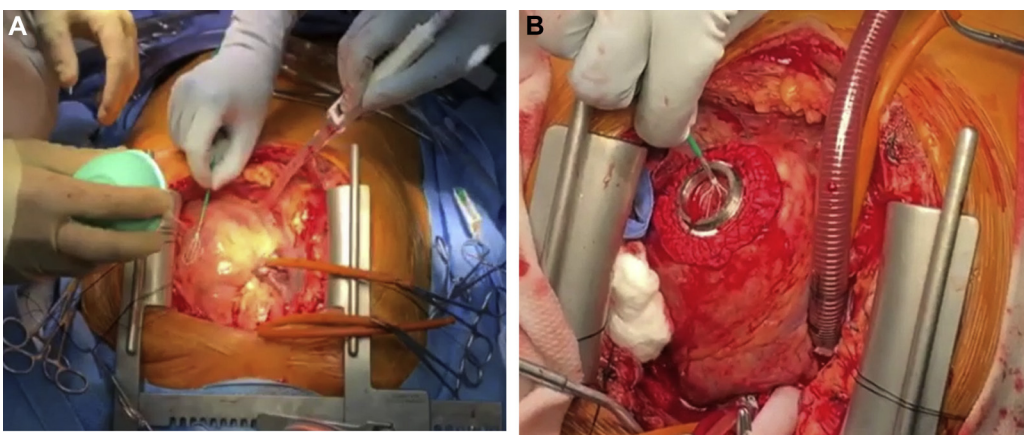


Electrocardiogram of clinical ventricular tachycardia shows rate of 160 beats/min, with a right bundle pattern and left superior axis.

substrate modification from both the RV and LV aspects of the entire septum was performed (Video 5E). A third VT was inducible with entrainment (Video 5F) revealing a biventricular "flutter" (Video 5G). This VT terminated during mapping, and given the significant amount of time spent during the procedure, the procedure was concluded. The patient remained arrhythmia-free after this ablation procedure and was transitioned to oral amiodarone and mexiletine.

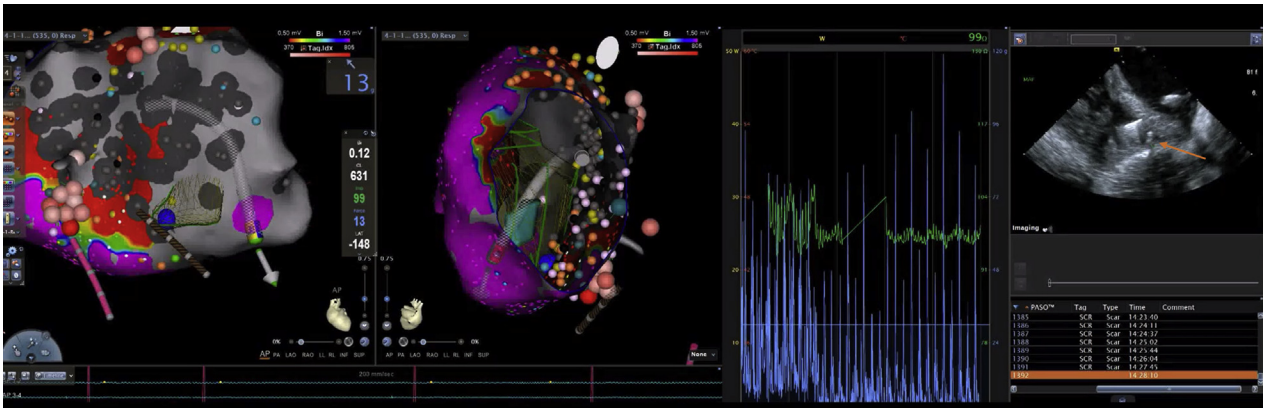
CLINICAL PERSPECTIVE 3: WHAT ARE THE UTILITY AND SAFETY OF CATHETER ABLATION FOR VT DURING AND AFTER LVAD IMPLANTATION? Most monomorphic VT is attributed to re-entrant circuits in established arrhythmic substrates within chronic myocardial scar (8). In patients with an

FIGURE 5 Intraoperative Mapping and Epicardial Ventricular Tachycardia Ablation During HM3 Placement



(A) Epicardial mapping and **(B)** subsequent pacing to ensure block performed during HeartMate 3 (HM3) (Abbott, North Chicago, Illinois) placement.

FIGURE 6 Endocardial Ventricular Tachycardia Ablation

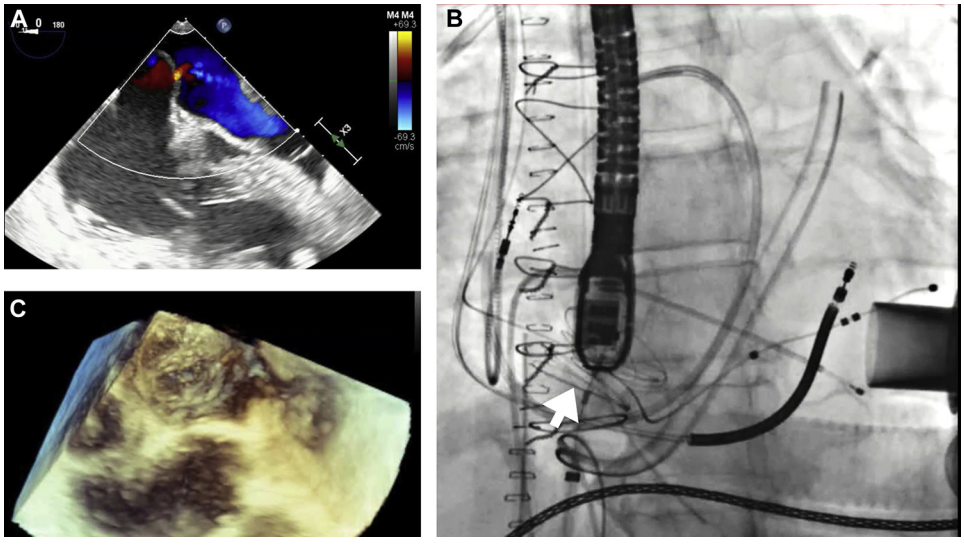


Extensive ablation performed of ventricular septum, particularly next to the area of septum touched by the inflow cannula **(arrow)**.

LVAD, the inflow cannula itself can serve as an anatomic barrier at the LV apex for re-entrant VT, and it can also cause mechanical irritation triggering VT in an excessively unloaded left ventricle. Refractory VT despite medical therapy is a Class I indication for catheter ablation (9). Although there are no specific recommendations in patients with LVADs, the same approach is generally used. Similarly, there are no guidelines for intraoperative VT ablation at time of LVAD placement, but the open chest provides a unique opportunity to define as well as ablate epicardial substrate in patients at high risk of VT recurrence post-LVAD placement.

The patient subsequently developed persistent hypoxemia secondary to a right-to-left shunt from the iatrogenic atrial septal defect (ASD) created by the transseptal puncture during the endocardial ablation. The ASD was closed 3 days after the second VT ablation with a 30-mm Cardioform device (W.L. Gore &

FIGURE 7 Percutaneous Closure of Iatrogenic Atrial Septal Defect



(A) Right-to-left shunt was noted continuously, so **(B and C)** a 30-mm Cardioform device (W.L. Gore & Associates, Newark, Delaware) was deployed, with resolution of hypoxemia.

Associates, Newark, Delaware), with resolution of hypoxemia (Video 6, **Figure 7**).

CLINICAL PERSPECTIVE 4: WHY WAS AN ASD CLOSURE DEVICE NECESSARY? The patient's persistent hypoxemia was likely the result of the iatrogenic ASD in the setting of the LVAD. With the LVAD's constant unloading of the left ventricle, the left atrial pressure is lowered below the right atrial pressure, thereby allowing for a large right-to-left shunt and causing hypoxemia. This phenomenon has been reported in patients with undiagnosed patent foramen ovale, but it has not yet been reported for an iatrogenic ASD after endocardial VT ablation (10). A catheter-based closure of the ASD was the best and only way to treat this patient's right-to-left shunt and resultant hypoxemia.

He made a remarkable neurological and functional recovery, and at 9 months of follow-up, he continues to do well with the LVAD, with improvement of LV ejection fraction to 30% and preservation of inferolateral and inferior wall motion, and he has been free of hospitalization and VT. This case demonstrates the importance of a multidisciplinary approach to recognizing and aggressively managing cardiogenic shock through the use of best practices.

AUTHOR DISCLOSURES

Dr. Bharadwaj is an advisor, proctor, and member of the Speakers Bureau for Abiomed. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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REFERENCES

1. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61:485–510.

2. Levine GN, Bates ER, Blankenship JC, et al. 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. J Am Coll Cardiol 2011;58:e44–122.

3. Hochman JS, Lamas GA, Buller CE, et al. Coronary intervention for persistent occlusion after myocardial infarction. N Engl J Med 2006;355:2395–407.

4. Schömig A, Mehilli J, Antoniucci D, et al. Mechanical reperfusion in patients with acute myocardial infarction presenting more than 12 hours from symptom onset: a randomized controlled trial. JAMA 2005;293:2865–72.

5. Basir MB, Kapur NK, Patel K, et al. Improved outcomes associated with the use of shock protocols: updates from the National Cardiogenic Shock Initiative. Catheter Cardiovasc Interv 2019;93:1173–83.

6. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock. Catheter Cardiovasc Interv 2019;94:29–37.

7. Stevenson LW, Pagani FD, Young JB, et al. INTERMACS profiles of advanced heart failure: the current picture. J Heart Lung Transplant 2009;28:535–41.

8. Moss JD, Flatley EE, Beaser AD, et al. Characterization of ventricular tachycardia after left ventricular assist device implantation as destination therapy: a single-center ablation experience. J Am Coll Cardiol EP 2017;3:1412–24.

9. Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. J Am Coll Cardiol 2018;72:e91–220.

10. Loforte A, Violini R, Musumeci F. Transcatheter closure of patent foramen ovale for hypoxemia during left ventricular assist device support. J Card Surg 2012;27:528–9.

KEY WORDS ASD closure, cardiogenic shock, delayed STEMI presentation, mechanical assist devices, ventricular tachycardia storm

APPENDIX For supplemental videos, please see the online version of this article.

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