

Percutaneous right ventricular assist device–supported ventricular tachycardia ablation in a patient with severe right ventricular dysfunction



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Introduction

Catheter ablation is an established therapy for the management of ventricular tachycardia (VT) but can be complicated by hemodynamic decompensation. Left ventricular assist devices (LVADs) have been used to support VT ablation. Percutaneous right ventricular assist devices (pRVADs) are a recent addition to the therapeutic armamentarium for the treatment of patients with right ventricular (RV) failure and there is no published literature for their use during VT ablation. Here, we present a first report of a patient treated with pRVAD-supported ablation for refractory RV VT in the setting of severe RV dysfunction.

Case report

A 71-year-old man with late surgical repair of a sinus venosus atrial septal defect, RV cardiomyopathy, permanent atrial fibrillation, complete heart block, and recurrent VT presented to an outside institution with appropriate cardiac resynchronization therapy defibrillator shocks for VT storm while on chronic amiodarone therapy. He was brought to the electrophysiology laboratory for VT ablation under general anesthesia. The patient became unstable during mapping of the clinical VT at the basal RV and suffered a pulseless electrical activity arrest. The procedure was aborted and the patient was successfully resuscitated. He was treated with therapeutic hypothermia and ultimately successfully extubated, with a good neurological outcome. He was transferred to our institution.

A transthoracic echocardiogram demonstrated moderately reduced left ventricular function, severe RV dilatation with moderate systolic dysfunction and diastolic flattening of the interventricular septum, severe right atrial dilatation, and

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KEY TEACHING POINTS

- Percutaneous left ventricular assist devices (pLVADs) are established devices; their net clinical benefit as adjuncts for ventricular tachycardia (VT) ablation is uncertain.
- Here we report the first case of isolated percutaneous right ventricular assist device (pRVAD)-supported ablation for refractory right ventricular (RV) VT in the setting of severe RV dysfunction.
- pRVADs represent a relatively new management option for patients with RV failure and more data are needed to better define the role and procedural indications for these devices.

no inspiratory collapse of the inferior vena cava, all consistent with right-sided pressure and volume overload (Figure 1A). Cardiac computed tomography demonstrated severe RV enlargement with akinesis of the basal/mid RV (indexed RV end-diastolic volume = 425 mL/m² [95th percentile of normal = 123 mL/m²]; RV ejection fraction 27%; Figure 1B). On day 3, the patient had recurrent VT (Figure 2A) while on maximally tolerated doses of beta-blocker, amiodarone, and intravenous lidocaine; the VT became antitachycardia pacing unresponsive and required repeated cardioversions. Given his VT refractory to medical therapy and noncandidacy for cardiac transplantation, the patient was considered for pRVAD-supported VT ablation.

On the day of the procedure, the patient was brought to the catheterization laboratory for insertion of a TandemHeart pRVAD using a Protek Duo double-lumen cannula (LivaNova, London, UK) via right internal jugular venous access; the Protek Duo inflow and outflow ports were positioned at the right atrium (RA) and main pulmonary artery (PA), respectively, and connected to the extracorporeal

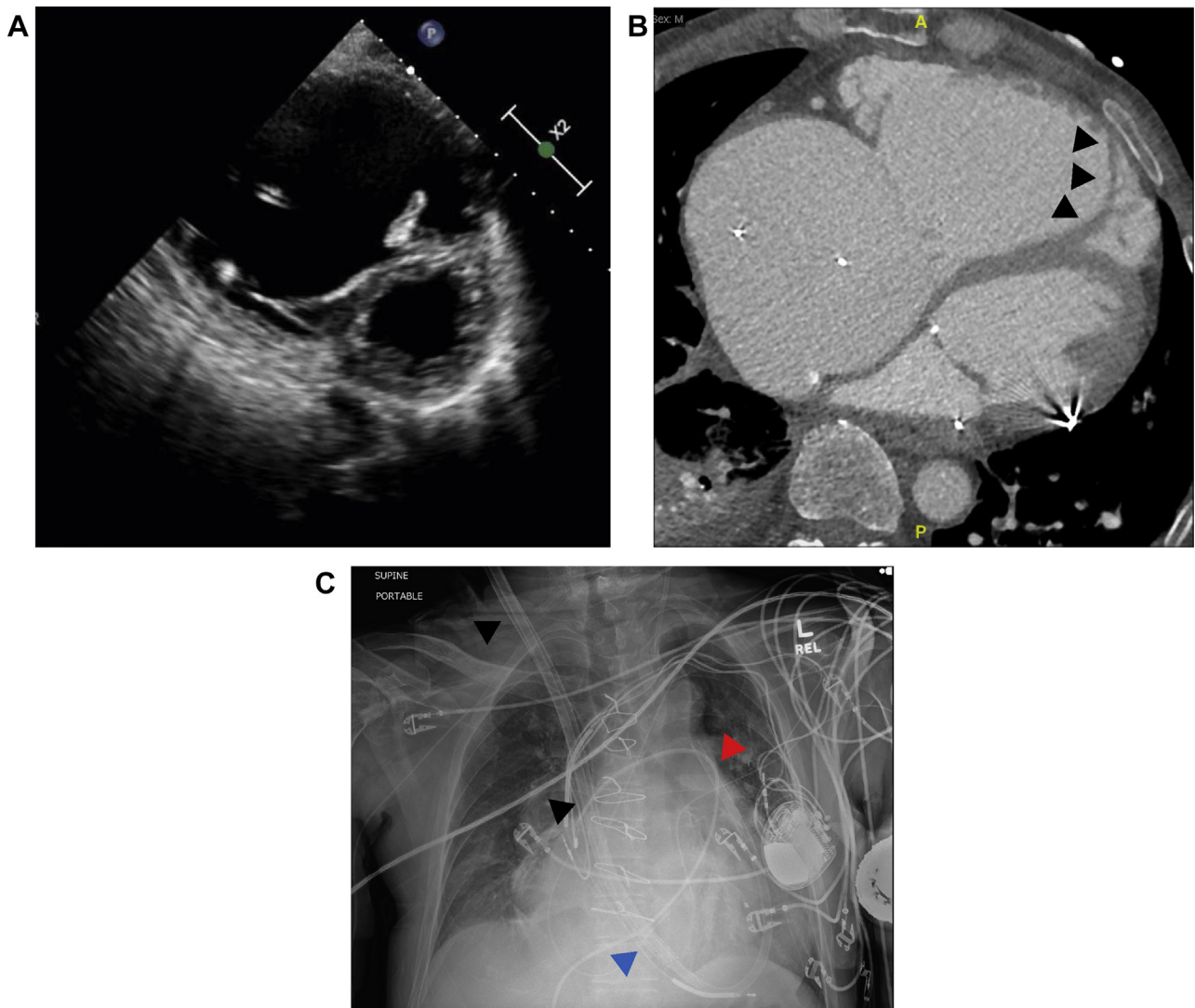


Figure 1 **A:** Echocardiographic short-axis view at end-diastole demonstrating severe right ventricle (RV) dilatation with flattening of the interventricular septum and normal left ventricular dimensions. **B:** Computerized tomographic 4-chamber view demonstrating severe right atrial (RA) dilatation, a prominent moderator band (*arrowheads*), and the entire apex taken up by the RV. **C:** CXR demonstrating the position of the Protek Duo cannula (LivaNova, London, UK) (*black arrowheads*), RA inflow port (*blue arrowhead*), and pulmonary artery outflow port (*red arrowhead*).

TandemHeart pump (Figure 1C). The patient was anticoagulated with heparin to a target activated partial thromboplastin time of 60–80. Hemodynamic parameters recorded before activation of the pRVAD were consistent with RV failure (RA 21 mm Hg; pulmonary artery pressure 36/20 mm Hg; pulmonary capillary wedge pressure (PCWP) 21 mm Hg; RA/PCWP = 1.0 [>0.86 suggestive of RV failure]). The patient was transferred to our electrophysiology laboratory. Monitored anesthesia care was administered by a cardiac anesthesiologist; an arterial line was obtained for continuous blood pressure monitoring. A voltage map of the RV revealed reduced voltage around the tricuspid annulus extending to the mid-lateral RV (Figure 3A). Three VTs were induced, mapped, and successfully ablated with radiofrequency energy around the tricuspid annulus, including the clinical VT; the patient remained stable during entrainment mapping (Figures 2B and 3B). The mapping and ablation catheters could be manipulated in the RV without hindrance by the

pRVAD. The total time spent in VT was 30 minutes, 30 seconds. The procedure ended with no complication.

The patient was transferred to the cardiac intensive care unit in stable condition. He had venous bleeding around the pRVAD insertion site while on systemic anticoagulation that required a total of 4 units of packed red blood cells. The pRVAD was successfully weaned and decannulated on postoperative day (RA 7 mm Hg; pulmonary artery pressure 23/11 mm Hg; PCWP 11 mm Hg) 1; a mattress suture secured hemostasis at the jugular vascular access site. The patient was discharged home on postoperative day 6 in ambulatory condition on metoprolol and mexiletine. He has had no recurrent VT after 4 months of follow-up.

Discussion

To our knowledge, this is the first reported case of pRVAD-supported VT ablation and highlights the challenges and

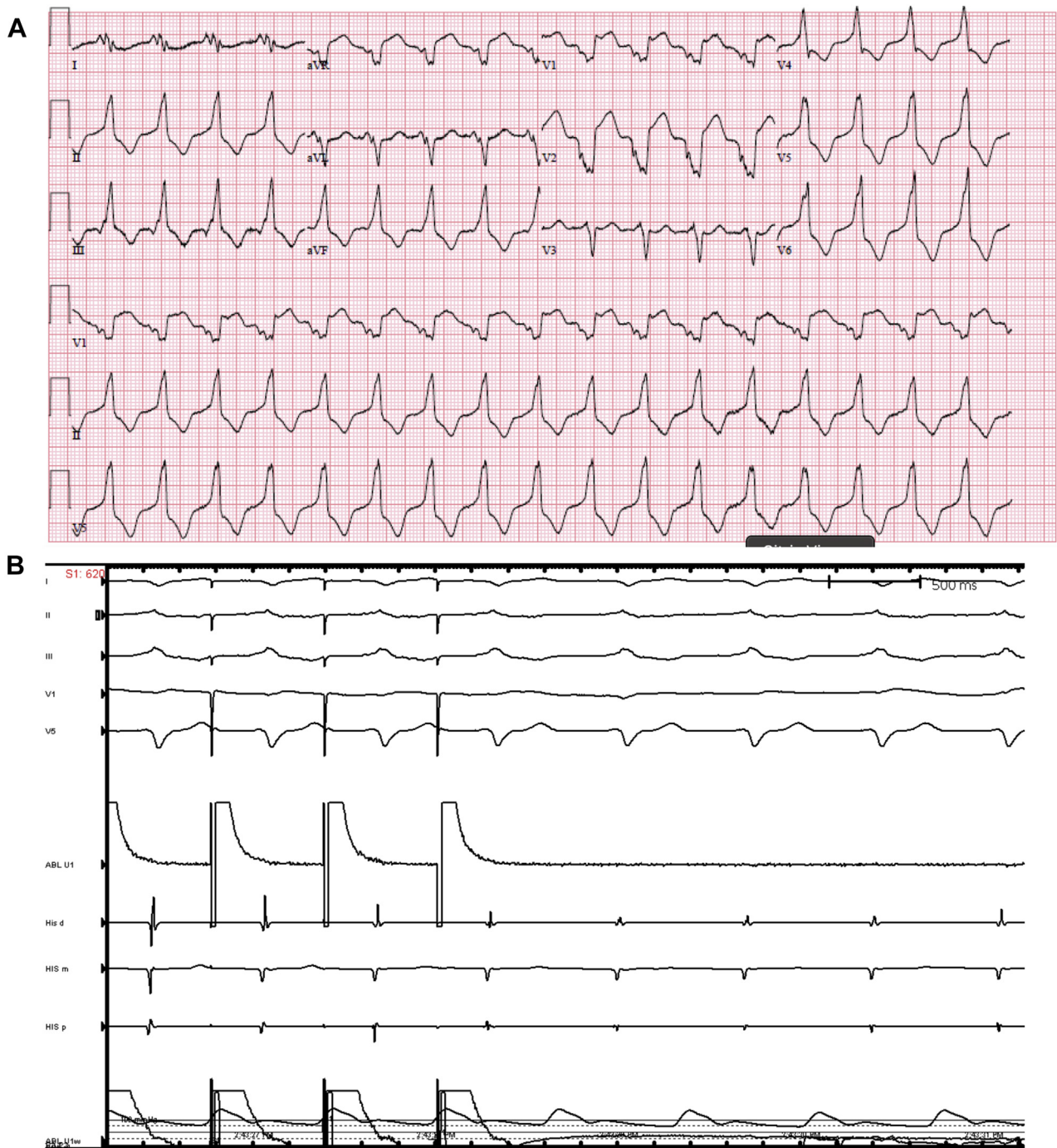


Figure 2 A: A 12-lead electrocardiogram of the patient's clinical tachycardia at a cycle length of 590 ms with left bundle/inferior axis configuration. B: Intra-procedural recording showing entrainment with concealed fusion for the clinical ventricular tachycardia; the arterial line tracing demonstrates stable blood pressure during tachycardia.

opportunities for the catheter-based treatment of VT in patients with severe RV dysfunction. The bulk of the published experience with the TandemHeart/Protek Duo has been in patients with post-LVAD RV dysfunction or acute RV failure.^{1,2} There is 1 published report of an Impella RP-supported atrial arrhythmia ablation in a patient with a Mustard operation and another reporting biventricular percutaneous-supported VT ablation in a patient with giant cell myocarditis.^{3,4}

VT ablation presents an important hemodynamic challenge to patients with structural heart disease. Repetitive activation/entrainment mapping and prolonged substrate mapping can negatively impact end-organ perfusion, increasing procedural morbidity and mortality.⁵ Potential benefits of percutaneous mechanical circulatory support (pMCS) for VT ablation include mapping/ablation of unstable VTs, maintenance of end-organ perfusion during prolonged substrate-based mapping/ablation, reduced

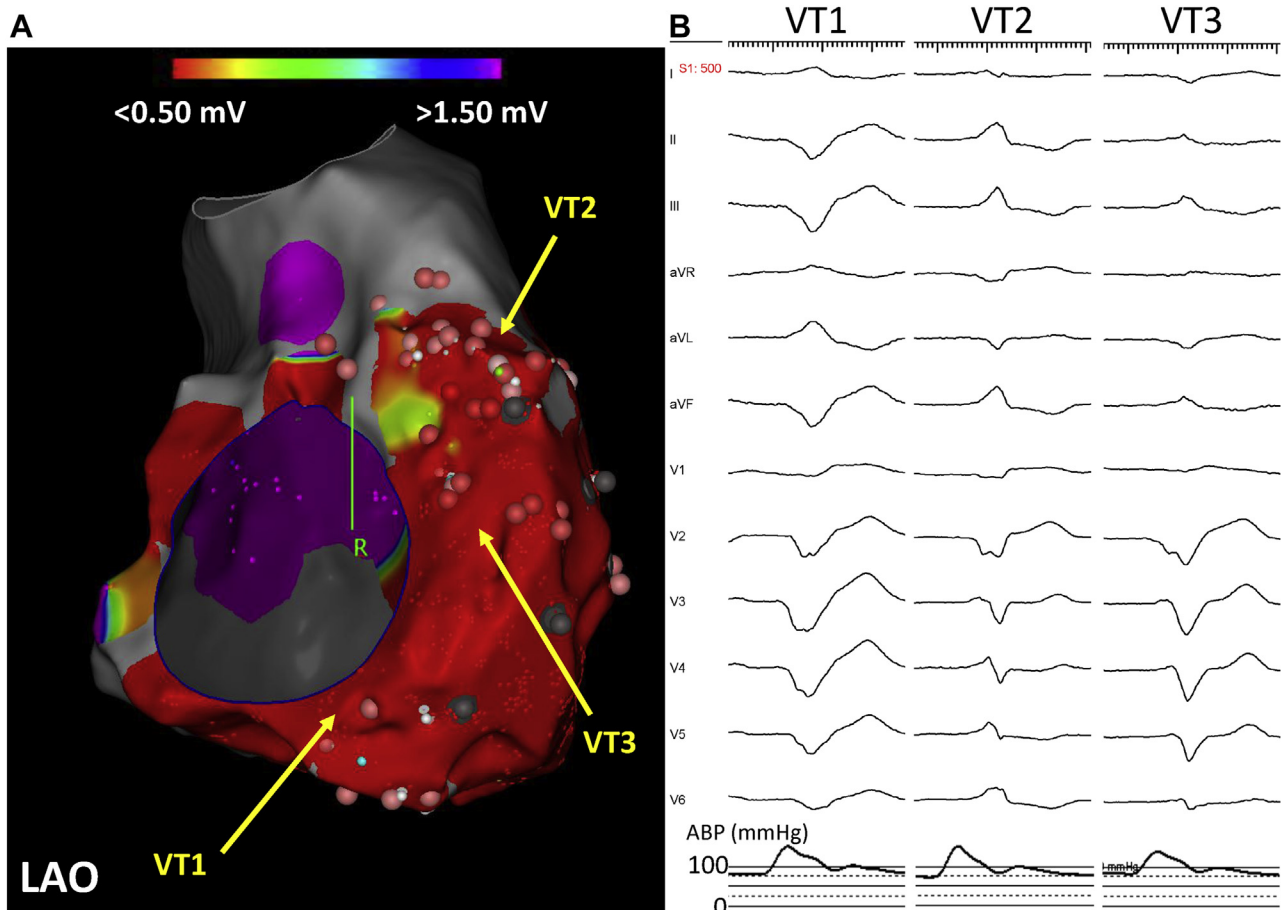


Figure 3 A: Left anterior oblique (LAO) view of the right ventricle voltage map demonstrating areas of low voltage around the tricuspid annulus (red) and the sites of successful ablation. B: The 12-lead configuration of the 3 tachycardias (VT1–VT3) targeted for ablation with arterial blood pressure (ABP) tracings.

periprocedural heart failure, and accelerated postprocedural recovery. However, these devices are associated with significant rates of vascular, embolic, and infectious complications in the range of 10%–15%. In addition, pMCS can interfere with catheter manipulation and electroanatomic mapping systems and increase the cost and complexity of the procedure.⁶

The majority of previous published experience with pMCS for VT ablation has been with percutaneous LVADs (pLVADs). Animal and human studies have shown that pLVADs do provide some support for hemodynamics during unstable rhythms.^{7,8} However, the net clinical benefit of their use is uncertain. Two recent meta-analyses reported similar findings in patients undergoing pLVAD-supported VT ablation. Luni and colleagues⁹ included 9 observational studies comparing 2572 patients undergoing pLVAD-supported vs non-pLVAD-supported VT ablation.⁹ They found that pLVAD-supported ablation allowed for a higher number of VTs to be mapped and ablated vs non-pLVAD-supported ablation (odds ratio [OR] 0.34 [0.19–0.49], $P < .00001$), but this did not translate into improved outcomes, as there was no difference in mortality (OR 1.05 [0.37–2.98], $P = .92$), acute procedural success (OR 1.10 [0.81–1.49],

$P = .54$) or VT-free survival (OR 1.01 [0.71–1.76], $P = .97$), with a higher number of procedure-related complications (OR 1.56 [1.10–2.20], $P = .01$).⁹ In the second meta-analysis, Turagam and colleagues¹⁰ included 5 observational studies comparing 2026 patients undergoing VT ablation with (284 patients) and without (1742 patients) pLVAD support. Again, pLVAD-supported ablation had similar rates of acute procedural success (risk ratio [RR] 0.95; 95% confidence interval [CI] 0.89–1.00; $P = .070$), VT recurrence (RR 0.94; 95% CI 0.66–1.34; $P = .740$), and mortality (RR 1.28; 95% CI 0.43–3.83; $P = 0.660$) vs non-pLVAD-supported ablation but with higher complication rate (RR 1.83; 95% CI 1.21–2.76; $P = .004$).¹⁰ The results of these meta-analyses of observational studies have to be interpreted with caution, as there is a high risk of bias toward use of pMCS in sicker patients.

Contrary to pLVADs, pRVADs represent a recent management option for patients with RV failure. The clinical experience to date has been in patients with RV failure in the setting of acute myocardial infarction, myocarditis, or LVAD-induced RV dysfunction. Currently available options for pRVADs include (1) the Impella RP (Abiomed, Danvers, MA), (2) the TandemHeart (LivaNova, London, UK), and (3)

veno-arterial (V-A) extracorporeal membrane oxygenation (ECMO). The Impella RP is inserted via a single 22F femoral venous access and placed across the pulmonary valve to support the RV with up to 4.8 L/min.¹ Limitations of the Impella RP include hemolysis, the inability to concurrently deliver oxygenation support, restricted patient mobility, and an infectious risk because of femoral vascular access. Moreover, the Impella RP can fall back from the PA into the RV outflow tract, especially in the setting of severe RV dilatation, requiring repositioning or removal of the device. The TandemHeart centrifugal-flow pump is an extracorporeal system that classically required 2 femoral venous access sites for a RA inflow and a PA outflow cannula.^{1,11} More recently, the Protek Duo (LivaNova, London, UK), a dual-lumen cannula, was approved for use in combination with the TandemHeart pump. The TandemHeart/Protek Duo is inserted via a single 29–31F right internal jugular venous access and can deliver up to 4.0 L/min of RV support. An oxygenator can be spliced into the circuit to effectively provide venovenous ECMO support. The TandemHeart/Protek Duo is associated with less hemolysis than axial-flow pumps and may have a lower infectious risk and allow for patient ambulation, as the femoral sites are spared.¹ Finally, V-A ECMO provides full cardiopulmonary support but can be complex to implement, and there is also potential concern that a tenuous patient may not be able to be successfully weaned from the circuit, which can present challenging decisions if the patient is not a candidate for a durable VAD or transplantation.

In our case, the decision to proceed with pRVAD-supported VT ablation was made on the basis of clinical and hemodynamic markers of RV failure in a patient with refractory VT with a recent history of ablation complicated by pulseless electrical activity arrest. The specific pRVAD was carefully selected on the basis of the patient's support requirements, anatomy, and procedural objectives. The patient had preserved left ventricular and pulmonary function; he therefore did not require full cardiopulmonary support with V-A ECMO with its associated complications. The Impella RP and TandemHeart/Protek Duo were 2 viable options. The TandemHeart/Protek Duo was favored because of its lower rate of hemolysis in our local experience, as well as enhanced postprocedural patient mobility. Moreover, the Protek Duo cannula is more stable in severely dilated RV, as was the case in our patient, than the Impella RP. Finally, the Impella RP, because of the intracardiac location of the axial pump, could interfere with electroanatomic mapping of the RV. The pRVAD appeared to have positively contributed in maintaining hemodynamics, allowing for fairly detailed entrainment mapping. Whether pRVAD-supported VT ablation will improve clinical outcomes in systematic study remains to be determined.

Periprocedural anesthesia also needs to be carefully administered in patients with severe RV dysfunction in order to minimize acute changes to RV preload and afterload. Fluid

administration was kept at a minimum, including catheter irrigation. Preemptive pLVAD insertion is associated with better outcomes than rescue insertion, with 1 study reporting a significant mortality benefit of preemptive insertion (30-day mortality 58% vs 4%; $P = .003$).^{7,12} In our experience with this case, preemptive pRVAD insertion was viewed to have contributed to the positive outcome in our patient. Prospective identification of patients who may benefit from preprocedural pRVAD insertion is important and a clinical challenge.

Our case highlights the challenges and opportunities in the catheter-based treatment of VT in patients with significant RV dysfunction undergoing VT catheter ablation. These complex patients require coordinated multi-specialty care in which pRVADs may also prove to be a useful supportive therapeutic modality. More data are needed to better define the role and indications for these devices.

Conclusion

This is the first reported case of isolated pRVAD-supported ablation for refractory RV VT in the setting of severe RV dysfunction. More data are needed to better define the role and procedural indications for these devices.

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