Outcomes of rescue cardiopulmonary support for periprocedural acute hemodynamic decompensation in patients undergoing catheter ablation of electrical storm

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BACKGROUND In patients with ventricular tachycardia or ventricular fibrillation (VT/VF) electrical storm (ES) undergoing catheter ablation (CA), hypotension due to refractory VT/VF, use of anesthesia, and cardiac stunning due to repeated implantable cardioverter-defibrillator shocks might precipitate acute hemodynamic decompensation (AHD).

OBJECTIVE We evaluated the outcomes of emergent cardiopulmonary support with extracorporeal membrane oxygenation (ECMO) to rescue AHD in patients undergoing CA of ES.

METHODS Between January 1, 2010 and December 31, 2016, 21 patients with ES (VT in 11 and premature ventricular complex–triggered VF in 10) were referred for CA and had periprocedural AHD requiring emergent ECMO support.

RESULTS In 14 patients, AHD occurred a mean of 1.5 ± 1.7 days before the procedure. In the remaining 7 patients, AHD occurred during or shortly after the procedure. ECMO was started successfully in all patients. Ablation was performed in 18 patients (9 with VF and 9 with VT). In patients with VF, premature ventricular complex suppression was achieved in 8 of 9 (89%). In those with VT, noninducibility was achieved in 7 of 9 (78%). After a median follow-up of 10 days, 16 patients died (13 during the index admission). Death was due to refractory VT/VF in 4 patients, heart failure in 11, and noncardiac cause in 1 patient. Seven patients survived beyond 6 months postablation; 5 remained free of VT/VF and 3 ultimately received a destination therapy (heart transplantation in 2 and left ventricular [LV] assist device in 1).

CONCLUSION In patients with ES undergoing CA, the outcomes of ECMO support as rescue intervention for AHD are poor. The majority of these patients die of refractory heart failure in the short-term. Strategies to prevent AHD including preemptive use of hemodynamic support may improve survival.

KEYWORDS Extracorporeal membrane oxygenation; Ventricular tachycardia; Electrical storm; Catheter ablation; Periprocedural acute hemodynamic decompensation

Introduction
Electrical storm (ES) is a life-threatening syndrome consisting of repetitive episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) over a short period of time, requiring termination by appropriate implantable cardioverter-defibrillator (ICD) interventions. The development of ES is associated with poor outcomes, with an up to 3-fold increased risk of mortality.

Catheter ablation (CA) has become an integral tool in the management of ES, with an efficacy of >80% in controlling recurrent ES and a potential favorable effect on cardiac mortality. In patients presenting with ES, significant competing risks related to the concomitant presence of advanced heart failure syndromes and associated comorbidities pose substantial periprocedural and postprocedural challenges. In particular, the occurrence of periprocedural acute hemodynamic decompensation (AHD) is a major concern because of its significant association with postprocedural mortality. Periprocedural AHD can be...
triggered by hypotension due to recurrent VT/VF, use of anesthesia, and cardiac stunning due to repeated ICD shocks. When AHD occurs, rescue circulatory support with mechanical devices such as extracorporeal membrane oxygenation (ECMO) can be performed, although outcome data of this intervention are lacking. In the present study, we evaluated the outcomes of rescue ECMO support for periprocedural AHD in patients with ES undergoing CA.

Methods

Study population
Between January 2010 and June 2016, 21 patients (19 men; mean age 62.6 ± 11.3 years) with drug-refractory ES undergoing CA had periprocedural AHD requiring emergent ECMO support.

ES was defined as the occurrence of ≥3 VT/VF episodes in a 24-hour period, requiring electrical cardioversion or defibrillation. Periprocedural AHD was defined as (1) cardiac arrest requiring cardiopulmonary resuscitation; or (2) cardiogenic shock, defined as systolic blood pressure of <90 mm Hg for at least 30 minutes or the need for supportive measures to maintain a systolic blood pressure of >90 mm Hg.

The study protocol was approved by the local institutional review board.

Electrophysiology study and ablation
General anesthesia was used in all the cases. Mapping and ablation were performed with a 3.5-mm irrigated catheter (ThermoCool or NaviStar, Biosense Webster, Inc., Diamond Bar, CA), and a 3-dimensional mapping system (CARTO, Biosense Webster, Inc.) was used. For mappable VTs, activation and entrainment mapping were performed to identify critical sites of the VT reentrant circuit, as previously described. For hemodynamically unstable VTs, substrate modification was performed with linear or cluster lesions targeting sites identified by pace mapping and late/fragmented/split abnormal potentials.7 Radiofrequency energy was delivered with power ≤50 W with a targeted impedance drop of at least 10% from the baseline values. After ablation, ventricular programmed stimulation from the right ventricle (RV) was performed with single, double, and triple extrastimuli delivered to ventricular refractoriness at drive trains of 600 and 400 ms. In 4 patients with VT storm, programmed stimulation was also performed from the LV. Procedural success was defined as lack of inducibility of the clinical VT(s) and of all the mappable and unmappable nonclinical induced VTs with cycle length >250 ms.

In patients with premature ventricular complex (PVC)-triggered VF, the clinical PVCs were mapped by activation and/or pace mapping. If no PVCs were present at baseline, isoproterenol (2–20 μg/min) infusion and/or burst pacing from the RV apex and right atrium were performed to provoke ventricular arrhythmias. PVCs were classified to originate from the Purkinje network if a sharp potential preceded the ventricular electrogram both in sinus rhythm and during PVC. Radiofrequency applications were delivered to the site with the earliest activity during spontaneous PVCs and/or site demonstrating best pace map. Successful PVCs and/or site demonstrating best pace map. Successful ablation in these cases was defined as the absence of spontaneous or inducible PVCs after ablation.

Venoarterial ECMO support
The ECMO circuit consisted of a centrifugal pump (Rotaflow, Maquet Cardiovascular LLC, Wayne, NJ), a polymethylpentene gas exchanger (Quadrox iD Adult, Maquet Cardiovascular LLC), a heat exchanger, tubing, and various sizes of cannulas for venous and arterial cannulation. The femoral artery and vein were cannulated using the Seldinger technique to insert the retrograde arterial and venous cannulas under fluoroscopy guidance. A small antegrade sheath was placed in the superficial femoral artery to provide distal flow and prevent limb ischemia. In 1 patient, right axillary artery cannulation (instead of femoral) was used for arterial perfusion. ECMO was started at 4.0–4.5 L and adjusted according to patient’s hemodynamics. During support, heparin was administered to a target activated clotting time of >250 seconds.

Follow-up
Clinical recurrences, ICD therapies, procedural complications, and mortality were recorded for all patients included in the cohort. For patients followed outside our institution, follow-up data were acquired through telephone interview of the referring physician and patient. Mortality data were confirmed via queries of the Social Security Death Index.

Statistical analysis
Continuous data were reported as mean ± SD and categorical data as number and percentage. The burden of associated comorbidities and baseline patient risk profile were assessed using the PAINESD score, as previously reported.6 Differences between categorical variables and frequencies were compared using the χ² test. Differences between continuous variables were compared using the Student t test. Mortality was analyzed using the Kaplan-Meier method, and differences between groups were assessed using the log-rank test. All tests were 2-sided, and a P value of <.05 was considered statistically significant. Statistical analyses were performed with GraphPad Prism (GraphPad Software Inc., La Jolla, CA, USA).

Results

Patient characteristics
The baseline characteristics of the 21 patients included in the study are detailed in Table 1. The presenting arrhythmia was monomorphic VT in 11 patients and PVC-triggered VF in 10 cases. Eight patients presented with ES in the context of an acute event (post–acute myocardial infarction [MI] in 6 and post–coronary artery bypass graft [CABG] in 2). Reversible ischemia was ruled out in each patient before the procedure with a left heart catheterization study. In the 6 patients post-acute MI, ES presented after a mean of 8.6 ± 8.7 days (range 1.5–25 days), whereas in the 2 patients post-CABG, ES presented 3 and 5 days after the
surgery, respectively. A history of remote MI was present in 4 patients, occurring 4 months to 19 years earlier. In 9 patients with ischemic cardiomyopathy, there was no clear history of an acute coronary event.

All patients had failed treatment with at least 1 antiarrhythmic drug (range 1–5), including amiodarone in 95%, lidocaine in 81%, procainamide in 19%, mexiletine in 10%, flecainide in 5%, sotalol in 5%, and quinidine in 5%.

The underlying cardiomyopathy etiology was ischemic in 19 patients, cardiac amyloidosis in 1 patient, and idiopathic VF in 1 patient. Severe LV dysfunction was present in all cases (mean LV ejection fraction 21.1% ± 13.2%), except the patient with idiopathic VF (LV ejection fraction 75%).

The prevalence of hypertension was 71%, diabetes mellitus 43%, and chronic kidney disease 52%. The New York Heart Association functional class before ES was I in 10 patients, II in 6, and III in 5. No patient was being considered for advanced therapies before the VT/VF event. The mean PAINESD score was 21.6 ± 4.1.5

Five patients with VT storm and 1 patient with VF storm had undergone a prior CA procedure (range 1–3 procedures; mean time 14.5 ± 15.9 months before the index procedure and range 2 days to 50 months).

**Occurrence of periprocedural AHD and rescue ECMO support**

In all cases, ECMO was started emergently as a rescue intervention after the occurrence of AHD. In 14 patients, AHD occurred a mean of 1.5 ± 1.7 days before the ablation procedure in the context of refractory VT/VF storm with hemodynamic collapse. In the remaining 7 patients, AHD occurred during the ablation procedure; 5 of these patients were immediately connected to ECMO, while in the other 2 patients a percutaneous mechanical support device (intraaortic balloon pump in one case and Impella device [Abiomed, Inc., Danvers, MA, USA] in another case) was initially implanted and transition to ECMO was performed within 24 hours. The total duration of circulatory support was 4.9 ± 2.6 days. In 11 patients, decannulation was performed because of clinical and hemodynamic stability, and in 4 patients, ECMO was continued as a bridge for a permanent LV assist device. The remaining patients died before weaning (n = 2) or were decannulated after decision to withdraw care (n = 4).

**Electrophysiology study and CA**

Ablation was performed in 18 patients (9 with VT and 9 with PVC-triggered VF), while in the remaining 3 patients the procedure was aborted before radiofrequency was delivered. In patients with VT storm (n = 9), the clinical VT was mapped to the LV in 8 cases and to the RV in 1 patient (cardiac amyloidosis). The number of VTs targeted was 3.4 ± 2.5 per patient (range 1–9 VTs). In patients with VF storm (n = 9), triggering PVCs were mapped to the left Purkinje system in 4, LV papillary muscles in 2, RV moderator band in 1, and other LV low-voltage areas in 2 (Figure 1).

In all patients, both activation mapping of the clinical PVC and pace mapping were performed. Ablation was successful in all except 1 patient with RV moderator band PVCs, in which ECMO was started at the beginning of the case, but the procedure had to be stopped because of worsening biventricular function and increasing requirement of vasopressors.

Overall, acute success was achieved in 15 of 18 cases (83%). In patients with VF storm, elimination of the
triggering PVC was achieved in 8 of 9 patients (89%). In 7 of 9 patients with VT storm (78%) in whom ablation was performed, programmed ventricular stimulation was repeated at the end of the procedure and showed no VT inducibility. The mean duration of the ablation procedure was 4.7 ± 1.8 hours (range 2.9–9.9 hours). The fluoroscopy time was 26.6 ± 17.9 minutes (range 11–64 minutes).

Complications
Three patients had significant bleeding from the ECMO vascular access sites (axillary in 1 and femoral in 2) requiring transfusion (2–5 units of red blood cells). One patient presented thrombosis and occlusion of the ECMO cannula with compromise of oxygenation and another patient developed an LV thrombus with embolism and transient ST-segment elevation.

Follow-up
After a median follow-up of 10 days (range 1 day to 27 months) from the procedure, 16 patients (76%) died (Figure 2). Of these 16 patients, 13 (81%) died during the index admission within 4.9 ± 4.6 days of the procedure. Death was due to refractory VT/VF in 4 cases, heart failure in 11 cases, and a noncardiac cause (gangrenous cholecystitis) in 1 case (Figure 3).

Figure 1  Ablation of ventricular fibrillation storm in 2 patients with triggering premature ventricular complexes mapped to the left posterior fascicle (A) and the right ventricular moderator band (B).

Figure 2  Survival curve with detail in the first 30 days postprocedure. The majority of patients (81%) died during the index hospitalization.
Seven patients survived >6 months postablation; 5 of these remained free of VT/VF and 3 ultimately received destination therapy (heart transplantation in 2 and LV assist device in 1).

When comparing patients who died with those who survived, the only difference between the 2 groups that appeared was the presence of chronic kidney disease (62.5% vs 0%, respectively; \(P = .0351\) for comparison) (Table 2). Overall, despite similar baseline clinical features and risk profile (Supplemental Table 1), survival appeared better in patients with ES after an acute event (acute MI or CABG) than in the remaining patients, although the difference did not reach statistical significance (6-month survival 50% vs 23%, respectively; \(P = .2037\)).

**Discussion**

This study reports the outcomes of emergent ECMO support to rescue periprocedural hemodynamic collapse in patients undergoing CA of ES and documents an overall dismal prognosis with a high short-term mortality. Most patients died during the index hospitalization, predominantly because of refractory heart failure. The clinical implications of these findings are significant.

CA has emerged as an important therapeutic strategy in patients presenting with ES to achieve effective VT/VF suppression and long-term arrhythmia control.\(^3\)–\(^5\) In these patients, sustained hypotension and cardiac stunning due to repetitive VT/VF with multiple ICD interventions together with several procedural aspects such as induction of anesthesia, programmed ventricular stimulation, spontaneous and/or mechanically induced VT/VF, need for multiple electrical cardioversions, and fluid overload during mapping and ablation may precipitate AHD. In a prior study from our group, we reported an overall 11% incidence of periprocedural AHD in a consecutive series of 193 patients undergoing CA of VT in the setting of structural heart disease.\(^6\) In that study, presentation with ES was strongly associated with an increased risk of periprocedural AHD, which was defined as acute hemodynamic impairment prompting premature procedure discontinuation and/or placement of a percutaneous mechanical support device. The latter included intraaortic balloon pump or percutaneous LV assist devices.

The present study expands our prior findings by reporting the outcomes of patients who experienced periprocedural AHD and underwent rescue cardiopulmonary support with ECMO, which allowed temporary hemodynamic stabilization and completion of the ablation procedure in most cases. However, despite hemodynamic stabilization and effective acute arrhythmia suppression in most patients, the prognosis remained poor. These findings provide additional evidence supporting the concept that prevention of periprocedural AHD in patients at risk is crucial and has the potential of reducing mortality.

In this context, in a recent observational study, Mathuria et al\(^7\) compared the outcomes of preemptive and rescue use of percutaneous LV support devices (Impella, Abiomed, Inc., Danvers, MA, and TandemHeart, CardiacAssist, Inc., Pittsburgh, PA) in patients with structural heart disease undergoing VT ablation. The authors reported a strikingly higher 30-day mortality rate in patients who underwent rescue hemodynamic support device placement (58%) than in those with an otherwise similar risk profile who underwent preemptive placement of hemodynamic support devices. It is important to point out that the devices used in the study by Mathuria et al provide only LV support and whether the results could

Table 2  Comparison of patients who died and those who survived

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Death (n = 16)</th>
<th>Survival (n = 5)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>61.2 ± 10.1</td>
<td>67.2 ± 14.9</td>
<td>.4359</td>
</tr>
<tr>
<td>Sex: male</td>
<td>14 (87.5)</td>
<td>5 (100.0)</td>
<td>.5440</td>
</tr>
<tr>
<td>Ischemic etiology</td>
<td>14 (87.5)</td>
<td>5 (100.0)</td>
<td>.5440</td>
</tr>
<tr>
<td>Presentation after an acute event</td>
<td>5 (31.2)</td>
<td>3 (60.0)</td>
<td>.2479</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (68.8)</td>
<td>4 (80.0)</td>
<td>.6269</td>
</tr>
<tr>
<td>Diabetes</td>
<td>8 (50.0)</td>
<td>1 (20.0)</td>
<td>.2367</td>
</tr>
<tr>
<td>Kidney disease</td>
<td>10 (62.5)</td>
<td>0 (0)</td>
<td>.0351</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>21.6 ± 14.9</td>
<td>19.5 ± 5.7</td>
<td>.6385</td>
</tr>
<tr>
<td>PAINESD score</td>
<td>24.0 ± 5.1</td>
<td>23.4 ± 3.9</td>
<td>.7886</td>
</tr>
<tr>
<td>Arrhythmia type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td>9 (56.2)</td>
<td>2 (40.0)</td>
<td>.5254</td>
</tr>
<tr>
<td>VF</td>
<td>7 (43.8)</td>
<td>3 (60.0)</td>
<td></td>
</tr>
<tr>
<td>Acute success</td>
<td>10 (62.5)</td>
<td>5 (100.0)</td>
<td>.2621</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD or as n (%).

\(LVEF = left\) ventricular ejection fraction; \(VF = ventricular fibrillation; VT = ventricular tachycardia.\)
be generalized to more effective biventricular support platforms such as ECMO is unclear.

The use of ECMO to assist CA has been previously reported by Carbucicchio et al in 19 patients (mean age 61 ± 6 years) with nontolerated recurrent VT. ECMO was started at the beginning of the case, and the flow was titrated after VT induction targeting a mean arterial pressure of 60–70 mm Hg. In this series, complete stabilization was achieved in 13 patients (68%). After a mean follow-up of 42 months, 4 patients died, 1 of them died of transplant-related causes. After a mean follow-up of 28 months, complete stabilization was achieved in 69% of cases. After a median follow-up of 21 months (range 13–28 months), VT recurrence was 33% and overall survival was 88%. At variance with our study, in the Milan experience ECMO was mainly used as a preemptive strategy for ablation of high-risk patients or hemodynamically unstable VTs, especially when a previous substrate modification performed in sinus rhythm had been ineffective. As such, a prophylactic use of ECMO support in patients deemed at high risk may provide substantial benefits by preventing AHD and allowing a safe completion of the procedure, with a potential for reducing postablation mortality.

**Study limitations**

This is a single-center observational study including consecutive patients with refractory ES undergoing CA and receiving ECMO support for periprocedural AHD. The number of patients was relatively small, which is linked to the relatively low prevalence of AHD and the specific study inclusion criteria. Given the small sample size, a detailed assessment of the possible benefits of rescue ECMO support in different subsets of patients was not possible. For instance, the outcomes appeared slightly better in patients who had ES after an acute event (eg, cardiac surgery or acute MI), although the analysis was not powered to detect statistical differences between subgroups. In addition, until recently, the use of ECMO at our institution has been limited to patients with AHD as a bailout strategy, and none of the cases included in the present series underwent prophylactic (ie, before the occurrence of AHD) ECMO support. As mentioned, the latter approach has been suggested beneficial in recent observational series. Finally, only the Rotaflow ECMO system was used in this study, and whether the observed results can be generalized to other ECMO devices warrants further investigation. Currently, 3 ECMO systems are available in our institution: the Rotaflow pump with the Quadrox oxygenator, the CentriMag (Thoratec Corporation, Pleasanton, CA, USA) pump with the Quadrox oxygenator, and the Cardiohelp (Maquet, Rastatt, Germany) integrated pump oxygenator. In our hospital we use the Cardiohelp system only for transport because of smaller size and priming simplicity. However, the device is ~7 times more expensive than the Rotaflow system. For emergency cases, we typically prefer the Rotaflow system because of simplicity of use and lower cost; of note, there is no evidence showing superiority of one system over the other. As such, we would not expect a difference in outcomes based on the type of device used.

**Conclusion**

In patients with VT/VF ES undergoing CA, the outcomes of rescue ECMO support for periprocedural AHD are poor. The majority of these patients die mostly of refractory heart failure during the index hospitalization, and few survive beyond 6 months postprocedure. Strategies to prevent AHD including preemptive use of hemodynamic support may improve survival.

**Appendix**

**Supplementary data**

Supplementary data associated with this article can be found in the online version at [https://doi.org/10.1016/j.hrthm.2017.09.005](https://doi.org/10.1016/j.hrthm.2017.09.005).

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