ORIGINAL ARTICLE

Eastern Journal of Medicine

East J Med 27(3): 472-478, 2022
DOI: 10.5505/ejm.2022.44520

Management of Electrical Storm With Different Treatment Strategies Under Mechanical Support; A Single-center Case Series

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ABSTRACT

Electrical storm is a lethal clinical situation that consists of ventricular tachycardia or ventricular fibrillation attacks, which are usually resistant to pharmacologic or electrical cardioversion. In patients with heart failure; refractory ventricular tachycardia attacks worsen organ perfusion and precipitate acute decompensation, multi-organ failure, and mortality. Extracorporeal membrane oxygenation is very successful for managing periprocedural acute decompensation and it facilitates radiofrequency catheter ablation by adequate mapping and more substrate ablation.

Five patients were enrolled whose electrical storm had been treated with either catheter ablation under extracorporeal membrane oxygenation support or other approaches (device setting changes, stellate ganglion blockade, or sympathetic denervation).

One patient had a catheter ablation history and was treated with only implantable cardioverter-defibrillator setting changes on this admission. Three of the patients were under veno-arterial extracorporeal membrane oxygenation support, one patient was under left ventricular assist device support. Two patients under extracorporeal membrane oxygenation also needed sympathetic denervation after radiofrequency ablation and stellate ganglion blockade was performed on one of them, before sympathetic denervation.

Electrical storms should be managed with multidisciplinary and various treatment approaches. Although radiofrequency catheter ablation is central in treatment, the use of additional modalities including sympathetic denervation may be beneficial. Furthermore, radiofrequency catheter ablation under mechanical support especially with extracorporeal membrane oxygenation may be quite effective, and lifesaving.

Keywords: Electrical Storm, Catheter Ablation, Extracorporeal Membrane Oxygenation

Introduction

Electrical storm (ES) is a lethal clinical situation that consists of ventricular tachycardia (VT) or ventricular fibrillation (VF) attacks, which are usually resistant to pharmacologic or electrical cardioversion (1). Radiofrequency catheter ablation (RFCA) is an effective treatment modality for refractory VT and ES episodes. It also has significant benefits for lowering hospitalization and mortality rates (2). In patients with systolic heart failure, prolonged tachyarrythmias and especially refractory VT attacks worsen the target organ perfusion and precipitate acute decompensation, multi-organ failure, and mortality (3,4).

In hemodynamically unstable patients, vasopressor agents may be temporarily effective, but mechanical support devices are usually indicated in case of unresponsiveness. Extracorporeal membrane oxygenation (ECMO) is very successful in managing periprocedural acute decompensation and it facilitates RFCA by adequate mapping and more substrate ablation (5,6,7).

In this article, we aimed to present our experience with different treatment strategies in patients with ES, third of them were under mechanical support with veno-arterial ECMO (VA-ECMO) and one

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Received: 18.01.2022, **Accepted:** 01.06.2022

of them was under left ventricular assist device (LVAD).

Materials and Method

Five patients were enrolled in the study whose ES had been treated with either RFCA under mechanical support (ECMO or LVAD) or other approaches (device setting change or sympathetic denervation). After approval by the Local Ethics Committee (Decision number: 2021/14/544), the patient's records were evaluated retrospectively between June 2020 and April 2021.

Four patients were followed up during the procedure with invasive arterial monitorization and a 5-lead electrocardiogram (ECG). Only one patient was followed up with non-invasive monitoring. The anesthetic induction was performed with intravenous (IV) fentanyl (5 to 10 mcg/kg), propofol (1-2 mg/kg), and orotracheal intubation was performed following rocuronium (0,5 mg/kg).

During the procedure, the anesthesia was continued with an intermittent IV bolus of fentanyl and rocuronium.

The right femoral artery and vein were used for ECMO cannulation. Under scope guiding, 16-18 Fr arterial and 22-25 Fr venous cannulas were inserted percutaneously.

The blood flow was set at 1,5 L/min at least and as high as 3-4 L/min during the episodes of VT or VF for preserving hemodynamic stability and adequate organ perfusion.

Unfractionated heparin (UFH) was used for anticoagulation before the procedure and the patients under VA-ECMO support were followed with bivalirudin according to activated clotting time (ACT) levels.

After the procedure, the patients were followed up in the coronary intensive care unit (CICU) for at least twenty-four hours as intubated and sedated, under ECMO support. The ECMO was removed surgically after resolution of arrhythmia or maintenance of hemodynamic stability which was guided by transthoracic echocardiographic (TTE) assessment of cardiac functions.

Patient 1: A 50-year-old male patient was admitted to our emergency department with dyspnea and chest pain. His ECG on admission showed ST-segment elevation in V1-2-3 leads and TTE revealed segmentary wall motion abnormality in the apex and anterior with an ejection fraction (EF) of 35%. Past medical history was remarkable for hypertension (HT), diabetes mellitus (DM), previous myocardial infarction (MI), and an implantable cardioverter-defibrillator (ICD) implantation for primary prevention two years ago. The patient was immediately transferred to the catheter laboratory and a coronary angiogram (CAG) was performed by femoral artery approach. An acute 90% stenosis with extensive thrombus burden was detected in the left anterior descending artery (LAD). A drug-eluting stent (DES) was implanted in the LAD after thrombus aspiration. Unfortunately, a no-reflow phenomenon developed and the flow could not be restored despite intracoronary adenosine and verapamil. The patient was followed up in the CICU, but VF and ES developed in a few hours. Laboratory and arterial blood gas analyses revealed no electrolyte disorder and the ECG showed a normal QT interval. Despite IV magnesium, lidocaine, and amiodarone infusions, the ES did not cease and because of repetitive ICD shocks he was intubated electively and VA-ECMO was implanted percutaneously.

The battery of ICD ran out because of shocks, within twenty-four hours and, additional multiple external electrical cardioversions were performed. Reassessment of the ECGs and monitor recordings of the patient revealed that the last beat immediately before VF was a premature ventricular contraction (PVC) on the peak point of the T wave which was considered the trigger of the arrhythmic insult. PVC with a right bundle branch block and inferior axis morphology was revealed (Figure 1).

Emergent RFCA was planned and the patient was transferred to the electrophysiology laboratory. By an En Site-NavX electro-anatomical mapping system (St. Jude Medical, USA), it was attempted to map the frequent PVC, triggering the VF, the frequent malignant arrhythmia during the procedure; therefore, after the failure of the 3D mapping system due to shocks, a focal PVC region was determined approximately 40-45 msec early and fragmented according to the reference QRS using fluoroscopy-guided conventional mapping. RFCA was applied to this area. At the 15th second of RFCA, PVC that caused the R on T phenomenon disappeared and after 30 minutes, neither PVC nor VT/VF was observed. Also, no arrhythmias were induced by programmed extra stimuli and burst pacing after RFCA (Figure 2 A,B,C,D). The ICD battery, which expired on the same day was replaced, and the patient was extubated 24 hours later, ECMO was removed, and he was discharged 10 days after the procedure.

Fig. 1. The PVC Was With A Right Bundle Branch Block and Inferior Axis Morphology Indicated By The Red Arrow

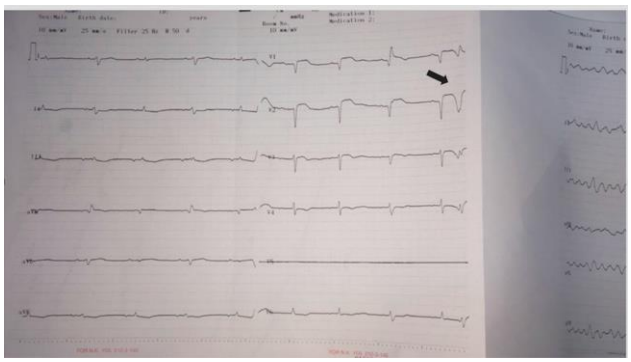

Fig. 2. Fluoroscopy Record (PVC, the cause of VF) Was Successfully Treated With RFCA, and Sinus Rhythm is Restored

Patient 2: A 65-year-old male with a history of coronary artery bypass surgery after MI, was referred to our CICU as intubated due to recurrent VT and VF episodes refractory to anti-arrhythmic drugs. The patient had been followed up under treatment with amiodarone infusion (1.0mg/min) after 300 mg loading dose, lidocaine infusion (1.0mg/min), magnesium infusion, and general anesthesia with propofol. There was no electrolyte imbalance detected in laboratory tests. TTE revealed a globally hypokinetic and dilated left ventricle with an EF of 20%. After numerous attempts of defibrillation, the patient was immediately transferred to catheter laboratory. Due to recurrent VF episodes causing hemodynamic deterioration, a VA-ECMO was implanted. The CAG revealed patent coronary bypass grafts. After excluding ischemia and other reversible causes of arrhythmia, urgent RFCA was planned. Frequent torsades de pointes degenerating to VF was the mechanism of arrhythmia, so scar homogenization became the initial strategy. The mapping procedure started with 3D isochronal late activation (ILAM) and scar mapping of the left ventricle using CARTO 3

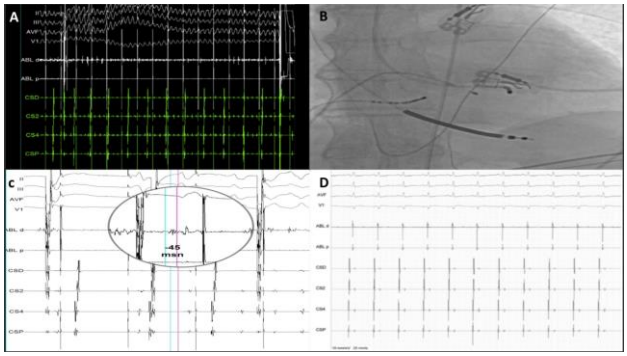

Fig. 3. EGM Records of The Patient In Sinus Rhythm

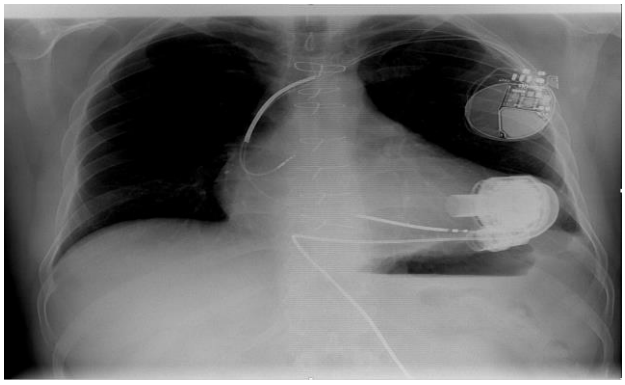

Fig. 4. The LVAD Is Seen In The Patient's Telecardiogram

and Penta Ray catheter (Biosense-Webster, Diamond Bar, CA, USA). RFCA and scar homogenization was performed on late potential areas localized on the posterior basal wall of the left ventricle with Smart Touch Surround Flowcatheter (Biosense-Webster, Diamond Bar, CA, USA) at 40 Watts and 50 degrees. The procedure was terminated after demonstrating no triggered VPCs with programmed and burst pacing. Normal sinus rhythm was restored with stable hemodynamic parameters, and the patient was free of VA recurrence. ECMO was removed successfully on the next day of the procedure. General anesthesia was carried on, and oral propranolol treatment was started. A single lead ICD was implanted on the following day but at the time of extubation, repetitive ICD shocks occurred and ES relapsed. The patient was sedated again and a percutaneous stellate ganglion blockade was planned. Bilateral stellate ganglion blockade was performed under ultrasound guidance using 5mL of 0.5 % bupivacaine. Sinus bradycardia developed and no VAs were detected on post-procedural ECG. No VAs were observed in the next 24-hours period in CICU. Stellate ganglion blockade was complemented with video-assisted thoracoscopic cardiac sympathetic denervation. After the last procedure, sinus rhythm was maintained; the patient was

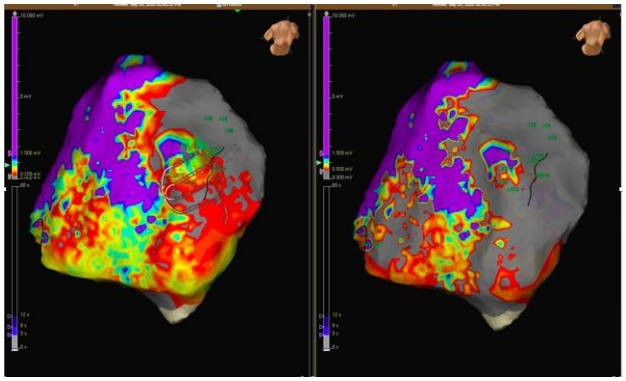

Fig. 5. Scar Mapping and ILAM Map of the Region Causing VT are Shown With 3D Mapping

hemodynamically stable and extubated successfully.

Patient 3: A 68-year-old male patient with a history of coronary bypass surgery was hospitalized in another center due to acute coronary syndrome and malignant arrhythmia, and CAG revealed patent bypass grafts. Frequent arrhythmias despite medical treatment during the follow-up were observed, and the patient was referred to our hospital for RFCA with the diagnosis of ES. The patient was extubated on his admission, but due to frequent external defibrillations, elective intubation was performed and emergent RFCA was planned. Under VA-ECMO support, the mapping procedure was started with 3D ILAM and scar mapping of the left ventricle using CARTO 3 and Penta Ray catheter (Biosense-Webster, Diamond Bar, CA, USA). Late potentials in the left ventricle posteroseptal basal myocardium region and a scar area compatible with the frequently developing VT morphology were determined, and RFCA was applied to the critical isthmus in this region, and then scar homogenization was achieved. The patient, whose ventricular arrhythmia was not induced by programmed burst pacing after catheter ablation, was transferred to the CICU as intubated and hemodynamically stable. The patient developed non-sustained ventricular arrhythmias with multiple morphologies. These were considered to be caused by PVC with R on T phenomenon which is quite different from the initial ventricular arrhythmia treated by RFCA. He was consulted for thoracic surgery before extubation and permanent sympathectomy were performed. After the sympathectomy, ventricular arrhythmias disappeared completely. The patient was extubated 48 hours after RFCA. The patient, who was followed up in the CICU for 3 days free of VT/VF, was discharged on the 10th day of hospitalization.

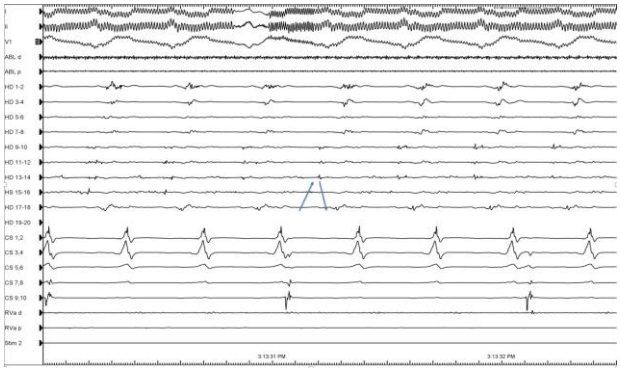

Fig. 6. With A High Density (Hd) Mapping Catheter, The Entrance, Isthmus, and Exit Regions of The Reentrant Arrhythmia Are Revealed (See Arrows), Respectively, During Tachycardia In Egm Records

Patient 4: A 44-year-old male patient who had an LVAD (Figure 4) due to heart failure developed after MI 3 years ago, and had also a single ICD for primary prevention from sudden death. The patient was immediately referred to the cardiovascular ICU because of palpitations, near syncope, and repetitive ICD shocks for the last 5 days. Assessment of ICD records demonstrated that he had frequent ventricular arrhythmias with appropriate ICD shocks. A wide-complex tachycardia was observed in his ECG. A TTE revealed moderate mitral and tricuspid regurgitations with a left ventricular EF of 20%. Electrolyte or laboratory findings were unremarkable. The patient, who received approximately 80 appropriate shocks despite medical treatment during his follow-up was intubated electively. He was transferred to the electrophysiology laboratory for RFCA. LV 3D mapping was performed using the En Site-Nav X electro-anatomical mapping system (St. Jude Medical, USA). After scar mapping, the estimated critical isthmus was determined with the ILAM (Figure 5). Clinical VT has been induced with TCL 320 msec with programmed ventricular extra stimulus. Activation mapping (TactiCath™ Contact Force Ablation Catheter, Sensor Enabled™, Abbott, USA) was performed using a high-resolution mapping catheter. The critical isthmus of the tachycardia in the LV posteroseptal basal region was determined. This region was ablated with 40W 50 degrees (Figure 6). Scar homogenization was then performed. After RFCA, no ventricular arrhythmia was detected during the 30 minutes of observation. Ventricular arrhythmia was not induced also by programmed stimulation (400/350/300) and burst pacing after ablation. The patient was transferred to the CICU in a hemodynamically stable state. He was extubated 12 hours after the procedure and

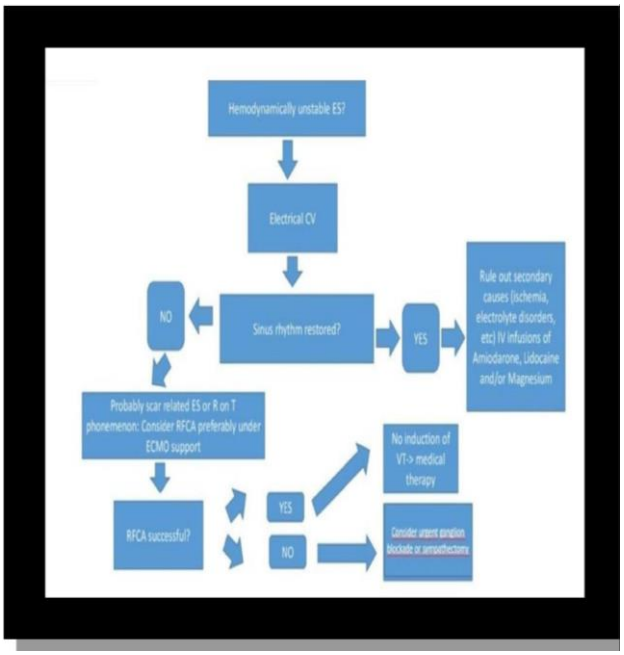

Fig. 7. Our clinical algorithm for the management of ES

discharged in the first week of the hospitalization.

Patient 5: A 44-year-old female patient with ICD had been followed in an outpatient clinic for about 3 years due to PVC. TTE showed 40% EF with mild tricuspid regurgitation. Approximately, 10000 PVCs were detected in the 24-hour Holter recording. Previously, successful RFCA was performed with 3D mapping to the posterolaterally localized PVC of the tricuspid annulus (Biosense-Webster, Diamond Bar, CA, USA). A year after RFCA, she was re-admitted to our center with palpitations. Rhythm Holter recording revealed 2000 PVCs per day. The patient was initially followed up with oral beta-blocker therapy. During the follow-up, unfortunately, the patient was admitted to the emergency department with an ES (14 ICD shocks in 24 hours). The ECG revealed PVCs with R on T phenomenon. Emergent RFCA was planned and she was transferred to the catheter laboratory. 3D mapping with the CARTO 3D mapping system (Biosense-Webster, Diamond Bar, CA, USA) was performed due to rare PVC, and the region similar to the previously ablated localization was isolated by RFCA. Amiodarone was added to the patient's medical therapy and she was discharged. Six months after the discharge, she was again admitted to our emergency department with ES and was interned in the CICU. In ICD device examinations; PVCs similar to the ones observed during the previous RFCA, caused short-term non-sustained VT and Torsades de pointes secondary to the R on T phenomenon were detected.

However, it was recognized that the settings were programmed to shock in short-term tachycardias. The program was switched to overdrive pacing mode with 130 bpm pacing in VVI mode. The shocks ceased and the patient was followed for 24 hours in CICU free of arrhythmias. After 24 hours, the device records revealed that PVCs are not followed by ventricular arrhythmia. Then the device was set at 70 bpm in VVI mode and the patient was discharged on the third day uneventfully.

Discussion

Malignant ventricular arrhythmias and/or ES are life-threatening clinical situations that are frequently encountered in patients with systolic heart failure. The management of ES particularly in those with ICD or in patients with heart failure in the early phase of MI requires a multidisciplinary approach. In this article, we aimed to summarize our multidisciplinary approach in a case series with successful results in patients admitted to our clinic with ES.

The ES is defined as sustained VT, VF, or ICD shocks that occur at least 3 times in 24 hours, and inappropriate ICD shocks are excluded from this definition (8). The case series shared in the literature recently, points out that different strategies can be followed in the management of patients with ES. These strategies can be summarized as broad-spectrum treatment modalities, from classical drug use to RFCA of ES, sympathetic nerve blockade, or ablation with complex supportive treatments such as ECMO or any other mechanical support. The recent data shared by Alnsasra et al., emphasize the importance of adjusting the ICD settings to manage the malignant arrhythmia episodes secondary to the R on T phenomenon in patients with ES (9).

Similarly, one of our patients in this article had ES due to the recurrent R on T phenomenon, which recurred after the previous ablation with complex mapping, and it was managed by only adjusting the ICD settings and she was successfully discharged.

In recent years, mechanical support devices such as ECMO and LVAD have gained more importance, especially in cases of acute respiratory and cardiac failure. In addition to permanent LVAD; veno-venous and VA-ECMO are used in

life-threatening acute pulmonary pathologies or in cases of acute heart failure, myocarditis, or cardiogenic shock (10). Uribarri et al. used VA-ECMO successfully both in the treatment of ES patients and as a bridge treatment for RFCA in a study of 5 cases (11).

The sympathetic nervous system plays a critical role in the initiation, aggravation, and treatment of ES (12). Therefore; another approach in the management of ES is sympathetic nerve blockade, which has recently been reported in the literature.

Sympathetic ganglion blockade (SGB), has gained increased recognition in the treatment of recurrent ES. Sympathetic fibers arising from thoracic spinal segments synapse in stellate ganglion and send post-ganglionic fibers to targets in the head, neck, upper extremities, and heart. In animal models, left SGB prolonged the action potential duration (measured as 90% monophasic action potential duration) in all layers of the myocardial wall, reducing transmural repolarization heterogeneity, increasing the effective refractory period, and reducing the VF threshold (13). Therefore, in the case of ES, deep sedation, intubation, and sympathetic blockade are often required for the stabilization of the patients. Because of these effects, the management of patients with ES has been provided by sympathetic nerve blockade. In a case published by Aksu et al., a patient with ES had been treated with renal sympathetic denervation successfully (14). Similarly, Cardona Gureche et al. obtained successful results in the management of malignant arrhythmia by applying stellate ganglion blockade in a case series of 6 patients with ES (15).

As a result, the management and treatment of ES can traditionally be stepped as trigger identification, regulation of antiarrhythmic therapy, sedation, and RFCA preferably under mechanical support. A detailed examination and adjustment of the ICD records, if any, allows to rule out inappropriate shocks and to re-programming for avoiding these shocks.

In patients with suspected acute myocardial ischemia, CAG followed by revascularization should be performed quickly. Correction of the electrolyte disorders, if any, is simple but maybe life-saving. The first choice in antiarrhythmic medication in most cases is amiodarone unless it is contraindicated (16).

Beta-blockers can be used for second-line therapy considering their antiarrhythmic and antiadrenergic effects. But their use may be limited in patients with decompensated heart failure or hemodynamic instability. Recently, Ortiz et al., have reported that the use of procainamide is more effective compared to amiodarone (17). In patients with ES, only oral anti-arrhythmic drug use is almost always ineffective, and sedation, RFCA, mechanical support devices, and sympathetic blockade are usually required.

Mechanical support, especially with ECMO, is an excellent management method that had been in use more frequently for both hemodynamic stabilization and electrophysiologic studies in patients with ES. It allows for determining the source of ES (adequate scar mapping) and RFCA (treatment target) during the electrophysiological procedure. In this study, we aimed to share our treatment strategies in patients presenting with ES, the success and importance of ECMO support, and the clinical results of all mentioned methods (Figure 7).

ES is a complex type of life-threatening malignant arrhythmias, that should be managed with multidisciplinary and various treatment approaches. Although RFCA is central in treatment, the use of additional modalities including sympathetic denervation may be beneficial. Furthermore, RFCA under mechanical support especially with ECMO may be quite effective, and lifesaving.

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