CASE REPORT

Neuraxial Modulation in Electrical Storm: A Case Report

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Abstract

Aims and background: Management of unstable and refractory electrical storm (ES) requires a multidisciplinary approach in an intensive care unit (ICU). A dysregulated autonomic system is understood to be the cause of perpetual arrhythmogenesis.

Case description: We present a case of refractory ES precipitated by myocardial ischemia (MI), which was promptly managed with primary percutaneous coronary intervention (PPCI). Despite successful reperfusion with PPCI, the patient continued to have arrhythmias for a prolonged period than expected. Due to refractory arrhythmia, escalation of antiarrhythmic drugs (AADs) along with sequential sympatholytic therapy was undertaken.

Conclusion: The use of neuraxial modulation [initially stellate ganglion block (SGB) followed by surgical sympathectomy] may lead to the control of arrhythmia and hemodynamic stability in this difficult subset of patients.

Clinical significance: Unstable ES is due to inappropriate sympathetic system activation and there is mounting evidence in support of neuraxial modulation as the main modality rather than escalating traditional AADs.

Keywords: Antiarrhythmic agents, Case report, Electrical storm, Mexiletine, Myocardial ischemia, Neuraxial modulation, Stellate ganglion block, Surgical sympathectomy, Sympatholytic therapy, Ventricular arrhythmia.

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Introduction

Refractory ventricular tachycardia (VT) is a challenge for cardiologists and intensivists alike globally. Refractory ventricular arrhythmia, or electrical storm (ES), is defined as a state of cardiac electrical instability that is identified by three or more episodes of sustained VT, ventricular fibrillation (VF) or three or more appropriate shocks from an internal cardiac defibrillator (ICD) during a 24-hour period.[1] The management of patients in ES has been varied and predominantly driven by antiarrhythmic drugs (AAD) (Flowchart 1). Prolonged ES may be associated with significant hemodynamic instability and the need for advanced cardiac life support (ACLS); this is a subgroup wherein altered autonomic nervous system activation in response to acute stress, such as ischemia or heart failure, is thought to be causative. Apart from the use of AADs, addressing the underlying mechanism, like ischemia, will help with outcomes and earlier resolution of ES. With a better understanding of pathophysiology in recent years, neuraxial modulation is being increasingly applied with improved outcomes.[2–4]

Case Description

A 76-year-old male was received at a tertiary care center for advanced cardiac evaluation. This gentleman with a background of longstanding diabetes mellitus, hypertension, and ischemic heart disease with moderate left ventricular (LV) dysfunction (LV ejection fraction of 45%) underwent coronary angioplasty 3 years before the index admission. VT was noted during an ECG evaluation at a local center where amiodarone and magnesium were administered as initial management for the arrhythmia. Upon arrival at the tertiary care center, he exhibited monomorphic VT with a systolic blood pressure of 100 mm Hg. Following a repeat dose of amiodarone (150 mg), he was initiated on infusion of amiodarone for arrhythmia prevention. Upon resolution of VT, ECG noted anterior wall ST-elevation myocardial infarction (Killips class 4), which prompted an emergent percutaneous coronary intervention (PCI). After a stent deployment to the left anterior descending artery (recanalization of thrombosis of old stent thought to be the culprit lesion), he was transferred to the intensive care unit (ICU) for further management as he required oxygen and low-dose vasopressors support during the procedure along with frequent sustained and ill-sustained episodes (<30 seconds) of VT. After the procedure, he was noted to have a reduction in LV ejection fraction (35%), though ST-segment elevation was resolved. Despite the improved ECG, recurrent episodes of sustained VT continued during the initial 8 hours. Due to significant hemodynamic fluctuation and pulmonary edema and to facilitate deep sedation and minimize sympathetic stimulation, the patient was initiated on mechanical ventilation. Other supportive measures included anti-platelet therapy, correction of

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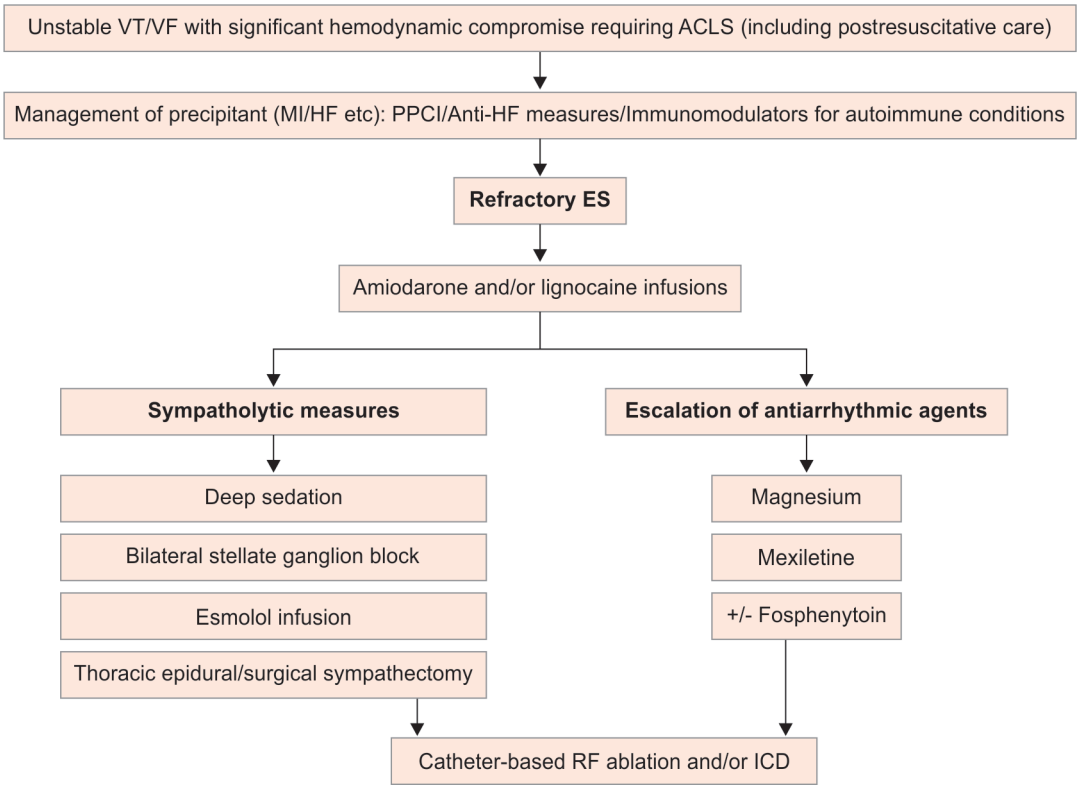
Flowchart 1: Proposed approach to management of ES; ES, electrical storm; VT, ventricular tachycardia; VF, ventricular fibrillation; ACLS, advanced cardiac life support; MI, myocardial ischemia/infarction; HF, heart failure; PPCI, primary percutaneous intervention; RF, radiofrequency; ICD, implantable cardioverter defibrillator



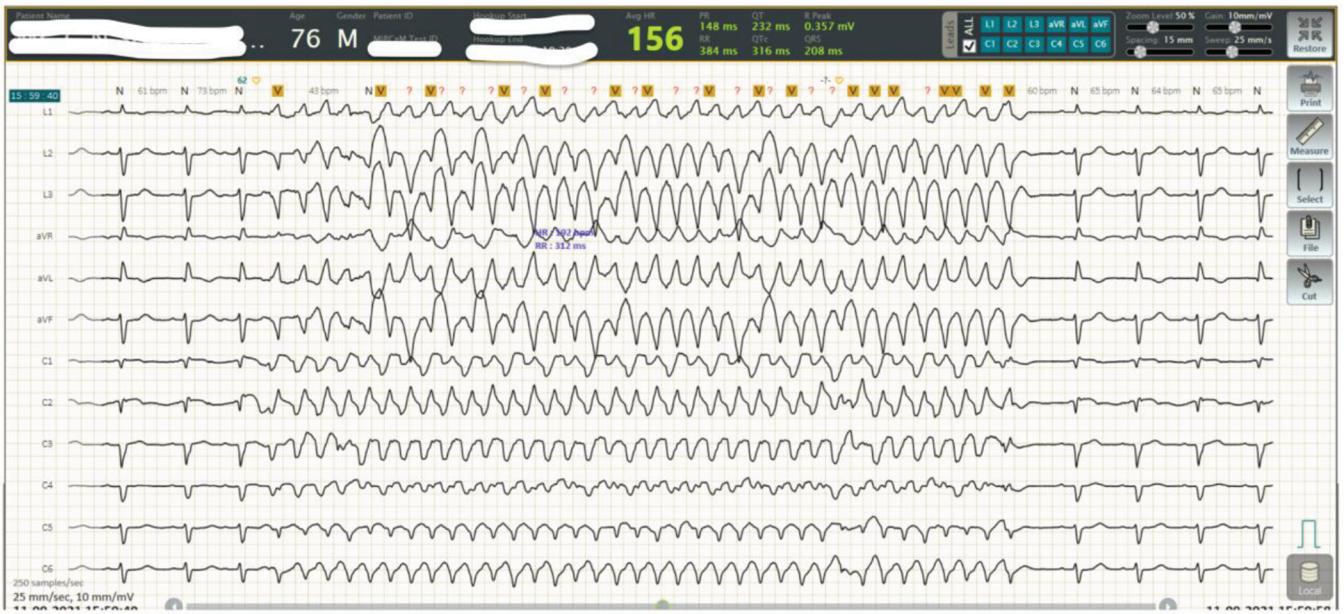


Fig. 1: Holter recording during the course of ICU stay (day 5)

electrolyte abnormalities, and management of hyperglycemia with insulin, vasopressors, mechanical ventilation with lung protection strategy, infection control, sedation, nutrition, and physiotherapy.

Recurrence of arrhythmia despite amiodarone infusion prompted the use of additional boluses of amiodarone and lignocaine (two boluses of 100 and 50 mg and later continued as an infusion). Following 12 hours of recurrent arrhythmias, with electrophysiologist input, neuraxial therapies were initiated as adjutants to AADs. He received bilateral stellate ganglion block (SGB) with bupivacaine 0.125% of 6–8 mL on each side under ultrasound guidance. Following the neural block, arrhythmia control was achieved for 16 hours. However, during the next 3 days, multiple episodes of sustained VT (initially monomorphic but later predominantly polymorphic) recurred (Fig. 1), requiring graded escalation of drug therapy apart

from repeated use of SGB. The patient received a total of 1500 mg of amiodarone during the initial 24 hours but was continued on infusion for the next 3 days at a rate of 0.5 mg/minute. Lignocaine infusion was used at a rate under 1.5 mg/kg/hour following the initial two boluses and continued for a period close to 72 hours. The use of esmolol as an infusion despite shock (low-dose vasopressor requirement) was the next escalation to maximize sympatholytic activity. Following prolonged parenteral use of amiodarone and lignocaine, oral mexiletine and amiodarone were added on day 4. The patient received fosphenytoin (1.5 gm for 2 consecutive days) as an additional AAD. During the ICU stay, he required cardioversion a total of 37 times apart from three episodes of ACLS measures, each needing multiple cycles of CPR. By day 7, breakthrough episodes of intermittent ill-sustained VT were noticed during periods when the neuraxial blockade was the least, making it easy to use thoracoscopic bilateral thoracic sympathectomy for sustained effect. Following surgery, he remained VT-free and could be liberated from mechanical ventilation and vasoactive medications. He underwent electrophysiological studies and insertion of an implantable cardiac defibrillator (ICD) before ICU discharge on day 10. At the time of discharge from the hospital on day 17, he was free of arrhythmia and progressively recovering from critical illness weakness and did not need support for activities of daily living.

Discussion

Unstable ES (protracted and usually associated with sustained ventricular arrhythmia) precipitated by myocardial needs to be differentiated from commonly occurring reperfusion arrhythmias (lasting for a few minutes to hours and predominantly ill-sustained). Initial management is based on ACLS guidelines apart from concurrently addressing the offending precipitant, followed by post-resuscitative care in an ICU setting.[5] It is mandatory for coordinated care involving specialties like electrophysiology, cardiology, and ICU teams with effective communication.[6] ES carries higher morbidity and mortality, and literature suggests that usual arrhythmia control over the cause of 3–7 days is based on the case series available.[7] AADs predominantly used in ES are parenteral amiodarone (class III) and lignocaine (class IB). Their use has seen a change in the last decade, with later recommendations proposing the use of lignocaine more than amiodarone during ACLS. The duration and dose of these two agents for ES are driven predominantly by experience and case series-based recommendations compared to powered studies that were done for their use during ACLS.[8] Amiodarone is deemed safe for parenteral use for a period longer than 14 days based on the product monograph (maximum of 720 mg/day), while the same can't be said for the use of lignocaine parenteral infusions.[9,10] The effectiveness of the combination of these medications in ES management is likely due to the combined blockage of sodium and potassium channels. Despite these beneficial effects in combination, they in themselves could precipitate ventricular arrhythmias, including Torsades de Pointes, when used in parenteral form at higher doses and for prolonged duration.[11] Oral mexiletine as a replacement for parenteral lignocaine and enteral amiodarone in favor of parental form is a usual change during ES care. Fosphenytoin is investigated for myocardial infarction-induced ventricular arrhythmias and has been used extensively in the past. This is another avenue for escalation in AAD optimization for refractory ES.[12]

Noting the limitation of traditional AADs, neuraxial modulation may be considered as a main strategy to control autonomic dysregulation that perpetuates the arrhythmia. Sympatholytic agent of choice is a β-blocker agent (BB). However, in refractory ES requiring ACLS and hemodynamic instability, the use of BB is often limited. Based on our experience and scant literature, esmolol in parenteral form is well tolerated in patients needing low-dose vasopressors.[13] Given this handicap, neuraxial interventions are evaluated for ES management. Literature noted the use of SGB, thoracic epidural anesthesia, and surgical cardiac sympathetic denervation with significant success, reducing the demand for traditional AADs. SGB is a bedside intervention that is easy to learn and can be repeated safely under ultrasound guidance. The use of agents with long half-life agents like bupivacaine could allow for sympathetic control for a period over 12–16 hours. Clinical improvement and noting Horner's syndrome on clinical examination could be surrogates for the effectiveness.[14–17,18] Surgical cardiac sympathetic denervation has been shown to reduce short and long-term recurrence of arrhythmia and could be undertaken ahead of catheter ablation therapies.[7,19] Finally, catheter ablation techniques would map the offending areas before ablation and find appropriate ICD and pacemakers for arrhythmia control in case of a late breakthrough.

Conclusion

Unstable VT/VF leading to an ES is due to inappropriate sympathetic system activation. Evidence in support of neuraxial modulation as the main modality of therapy for ES is mounting, in particular when higher doses of traditional AADs themselves are likely to worsen ventricular arrhythmias. SGB, as an intervention, is an easy bedside skill to learn for intensivists. A multidisciplinary approach to arrhythmia and protocol-based ICU care is essential for good outcomes.

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