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Recurrent Electrical Storm in a Patient with Chronic Heart Disease: A Case Report

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Abstract

Background: Electrical storm (ES) is a life-threatening cardiac emergency characterized by recurrent, sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) episodes that are often refractory to standard therapies. It predominantly affects patients with structural heart disease - most notably those with ischemic cardiomyopathy and chronic heart failure with reduced ejection fraction (HFrEF).

Case Presentation: We describe a 75-year-old man with ischemic cardiomyopathy and HFrEF admitted for sustained VT unresponsive to conventional management. His history included multiple catheter ablations, prior myocardial infarction, and an implanted ICD placed for recurrent ventricular arrhythmias. Despite additional ablations and antiarrhythmic medication, he continued to experience electrical storms. Definitive rhythm control was achieved via a multidisciplinary strategy comprising left stellate ganglion block, upgrade to cardiac resynchronization therapy-defibrillator (CRT-D), personalized ICD reprogramming, and initiation of flecainide in combination with propranolol. Device interrogation at follow-up confirmed effective biventricular pacing and complete suppression of sustained ventricular arrhythmias.

Conclusion: This case underscores the complexity of managing ES in patients with advanced structural heart disease and multiple comorbidities. Individualized device programming, innovative pharmacological regimens, and targeted interventional techniques can stabilize patients refractory to standard approaches. A holistic, team-based model of care is essential to optimize outcomes and improve quality of life in this high-risk population.

Keywords: electrical storm, ventricular tachycardia, ischemic cardiomyopathy, CRT-D, stellate ganglion block, ablation, flecainide, depression, anxiety

INTRODUCTION

Electrical storm (ES) is a life-threatening cardiac emergency characterized by the occurrence of three or more episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) within 24 hours, requiring interventions such as antitachycardia pacing (ATP), cardioversion, or defibrillation [1,2,3]. It most commonly occurs in patients with structural heart disease - particularly ischemic cardiomyopathy and chronic heart failure with reduced ejection fraction (HFrEF) [4] - and affects approximately 10-20 % of implantable cardioverter-defibrillator (ICD) recipients, being associated with increased mortality, rehospitalization, and markedly impaired quality of life [5,6]. Pathophysiologically, ES reflects an unstable arrhythmogenic substrate from extensive myocardial scarring, electrical remodeling, fibrotic changes, or microinfarcts, with dynamic triggers such as sympathetic overactivity, acute ischemia, electrolyte disturbances (hypokalemia, hypomagnesemia) and worsening heart failure sustaining ventricular arrhythmias [7]. Management requires a comprehensive, stepwise approach: first-line antiarrhythmic drugs and β-blockers to terminate VT/VF are combined with correction of reversible precipitants - such as ischemia, electrolyte imbalances, renal dysfunction, infection or device-programming issues - to reduce recurrence risk. Sedation and intensive hemodynamic support are critical, often necessitating ICU-level monitoring to detect recurrent arrhythmias promptly and guide optimization of circulatory support, with mechanical circulatory assistance (e.g. ECMO, IABP) used as a bridge in unstable patients [8]. In drug-refractory cases, class IC agents may be cautiously considered, though their use in structural heart disease demands careful risk assessment. Catheter ablation targeting the arrhythmic focus can provide durable suppression but depends on substrate location and hemodynamic tolerance, whereas neuromodulation - primarily left stellate ganglion block (SGB) - offers transient sympathetic attenuation, and in extreme scenarios surgical cardiac sympathetic denervation may be employed [9-11]. Given the multifactorial nature of ES, optimal outcomes hinge on a multidisciplinary team of cardiologists, electrophysiologists, anesthesiologists and critical care specialists coordinating tailored therapies to achieve effective arrhythmia control and improve prognosis. We report the case of a 75-year-old man with ischemic cardiomyopathy and HFrEF who experienced recurrent, sustained VT refractory to multiple ablations and antiarrhythmic regimens. Stabilization was achieved through a combined strategy of SGB, upgrade to cardiac resynchronization therapy-defibrillator (CRT-D), individualized ICD reprogramming, and

initiation of flecainide with propranolol, resulting in effective biventricular pacing and suppression of sustained ventricular arrhythmias. This case underscores the complexity of ES management in advanced structural heart disease and highlights the critical role of innovative pharmacological, interventional and device-based interventions within a multidisciplinary framework.

Case Presentation

A 75-year-old man with a history of ischemic cardiomyopathy and chronic heart failure with reduced ejection fraction (HFrEF) was admitted with sustained ventricular tachycardia (VT) refractory to standard therapy. The patient had previously been hospitalized multiple times for electrical storm and had undergone catheter ablations targeting arrhythmogenic substrates in the inferior and posterior walls of the left ventricle. His medical history included chronic coronary syndrome with prior myocardial infarction, coronary artery bypass grafting (CABG), percutaneous coronary interventions (PCI), descending aortic aneurysm, recurrent deep vein thrombosis, hypertension, hyperlipidemia, and benign prostatic hyperplasia. Several years earlier, a VVI-mode implantable cardioverter-defibrillator (ICD) had been implanted due to recurrent ventricular arrhythmias.

On admission, he presented in a life-threatening condition with sustained VT at a rate of 120-130 beats per minute, unresponsive to antitachycardia pacing (ATP). Due to hemodynamic instability, emergency electrical cardioversion was performed. The patient was subsequently referred for repeat electrophysiological study (EPS) and ablation. During the EPS, an attempt was made to ablate arrhythmic foci in the inferior wall; however, no clinical benefit was achieved. A left stellate ganglion block (SGB) was then performed, followed by intraoperative termination of the arrhythmia using burst pacing therapy.

Because of bradycardia, the existing ICD system was upgraded to a cardiac resynchronization therapy defibrillator (CRT-D). Postprocedural monitoring revealed effective biventricular pacing with only a single episode of self-terminating VT. Given the ineffectiveness of prior antiarrhythmic regimens, the ICD was reprogrammed to deliver therapy only in the VF zone, with a high detection threshold. Pharmacological therapy was also modified by initiating flecainide in combination with propranolol.

Discussion

Management of electrical storm in patients with advanced structural heart disease remains a significant clinical challenge. Antiarrhythmic therapy is often limited by adverse effects or insufficient efficacy, particularly in the context of extensive myocardial scarring and autonomic imbalance. Although catheter ablation is the cornerstone of VT treatment, its success is constrained by arrhythmia location, substrate accessibility, and hemodynamic tolerance during the procedure [10].

In our patient, prior ablations failed to prevent recurrent arrhythmias, indicating a deeply entrenched arrhythmogenic substrate. This lack of success prompted exploration of alternative strategies. Stellate ganglion block, although infrequently used, has demonstrated promising results in refractory ventricular arrhythmias by modulating sympathetic tone [11], a mechanism likely responsible for the stabilization observed in this case.

The decision to upgrade the existing ICD to a CRT-D system was driven by the presence of bradycardia and the potential benefits of resynchronization therapy in HFrEF patients. Reprogramming the device to deliver therapy only for VF episodes represented a crucial measure to avoid unnecessary or ineffective shocks.

Despite general contraindications for class IC agents in structural heart disease, flecainide was considered in this particular scenario due to the absence of acute ischemia and the refractory nature of the arrhythmia. Sangpornsuk et al. emphasize that with careful patient selection and close monitoring, class IC drugs can be safely administered in select structural heart disease populations. In our patient, the combination of flecainide and propranolol produced a favorable short-term antiarrhythmic effect, underscoring the importance of individualized therapy and risk assessment in managing drug-resistant ventricular arrhythmias [12].

Electrical storm carries not only the risk of life-threatening cardiac events but also imposes a substantial psychological burden. A meta-analysis of 39,954 ICD recipients found that 22.6% experienced clinically significant anxiety, 15.4% exhibited depressive symptoms, and 12.4% met criteria for PTSD [13]. Among those who survived ES episodes, anxiety risk increased nearly fourfold (OR 3.92) and depression risk almost twofold (OR 1.87) compared with patients without ES [13]. Furthermore, anticipatory anxiety regarding future shocks independently predicts both heightened anxiety (OR 6.35) and depression (OR 2.29), regardless of the number

of therapies received [14]. Additional risk factors for psychological distress include Type D personality, prior anxiety or depressive episodes, lack of social support, and comorbid chronic diseases such as COPD or diabetes [15,16]. ICD shocks frequently deteriorate quality of life, leading to social isolation and reduced self-esteem in approximately 25-30% of patients [17]. Fortunately, psycho-cardiological interventions - such as structured psychoeducational programs and cognitive-behavioral therapy - have demonstrated tangible benefits, including a mean reduction of 4.2 points in HADS-A scores and a 15-point improvement in MLHFQ scores [18]. Therefore, a comprehensive management approach should encompass not only optimization of pharmacotherapy, ablation, and neuromodulation but also routine psychological screening and early psycho-cardiological intervention.

Conclusion

This case vividly illustrates the complexity of managing electrical storm refractory to standard therapies, particularly in patients with advanced structural heart disease and multiple comorbidities that further complicate treatment. In such scenarios, it is essential to move beyond conventional algorithms and employ innovative pharmacological approaches - including cautiously selected agents traditionally considered contraindicated - tailored to the patient's individual clinical profile. Equally important are neuromodulatory techniques such as stellate ganglion block, which can modulate autonomic tone and significantly enhance arrhythmia control.

Moreover, advanced device management - encompassing optimized ICD programming and adoption of contemporary pacing strategies - plays a pivotal role in preventing recurrent electrical storms. Delivering such high-specialty care demands close interdisciplinary collaboration among cardiologists, electrophysiologists, intensivists, and anesthesiologists to comprehensively assess the patient's dynamic needs and adapt therapy accordingly. Only through this holistic, personalized, team-based approach can optimal outcomes and improved quality of life be achieved in this high-risk population.

Finally, integrating psychological care into standard treatment protocols - with routine assessment for anxiety, depression, and PTSD, along with access to psycho-cardiological support - not only enhances patient well-being but also promotes treatment adherence, reduces hospitalization rates, and contributes to better overall prognosis.

Disclosure

Author's Contribution

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All authors have read and agreed with the published version of the manuscript.

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