A case of post-STEMI electrical storm with multiple ICD shocks refractory to antiarrhythmic medications, treated successfully with bilateral sympathetic ganglionectomy.

HangYu Watson[1], Seher Berzingi[1], Emily Hendricks[1], Sittinun Thangjui[2], and Christopher Bianco[2]

[1]West Virginia University Department of Medicine
[2]West Virginia University Heart and Vascular Institute

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Abstract

Electrical storm (ES) is a life-threatening condition that requires a stepwise management approach, including antiarrhythmics, anxiolytics/sedatives, antiadrenergic, and hemodynamic support. In 88% of refractory cases, cardiac sympathetic denervation has proven effective in reducing ventricular tachycardia (VT) burden and ICD shocks. We present a patient with late-presenting ST-elevation myocardial infarction (STEMI), new reduced left ventricular ejection fraction (LVEF), post coronary artery bypass graft (CABG), who experienced recurrent monomorphic VT despite amiodarone, lidocaine, and left stellate ganglion block, who was successfully treated with bilateral video-assist thoracoscopy sympathetic ganglionectomy.

Introduction:

Electrical storm (ES), commonly defined as the occurrence of three or more ventricular arrhythmia or appropriate shocks from an implantable cardioverter-defibrillator within 24 hours, poses a significant risk of morbidity and mortality [1][2]. The MADIT-II sub-study highlighted that post-myocardial infarction (MI) patients with reduced LVEF developing ES experience heightened mortality risks, which persist for several months after initial storm events[3]. ES serves as an independent marker for subsequent death among ICD recipients, as highlighted by the AVID trial[4]. Common triggers of ES include myocardial ischemia, acute decompensation of heart failure, metabolic/electrolyte disorders, drug side effects, and increased sympathetic tone[5]. Managing ES demands a multidisciplinary, multimodality approach that may include antiarrhythmics and adrenergic blockade, sedation, anxiolysis, hemodynamic support, ICD reprogramming and, in selected cases, temporary mechanical circulatory support devices, and catheter ablation [6]. While antiarrhythmic drugs are crucial for the acute termination of ES, their efficacy in suppressing future arrhythmias is limited[5]. Post-MI patients with reduced LVEF commonly receive beta-blockers, lidocaine, and amiodarone. Mexiletine, a class IB antiarrhythmic, is considered for those refractory to high-dose amiodarone or intolerant to lidocaine due to neurological toxicity[7,8]. Stellate ganglion block has shown efficacy when standard measures fail [9]. In refractory cases, bilateral sympathetic ganglionectomy emerges as a viable option, demonstrating a high complete response rate (66.7%) and reduced ICD shocks in the year following surgery [10, 11]. We present a post-MI patient after CABG revascularization with reduced LVEF experiencing refractory ES despite multiple interventions. Conventional measures, including amiodarone, mexiletine, sedation, anxiolysis, and stellate ganglion block, proved insufficient. Mechanical support with Impella was initiated, and the patient underwent successful bilateral sympathetic ganglionectomy. This case highlights bilateral sympathetic ganglionectomy's role in the management of refractory ES, showcasing its potential efficacy when conventional interventions fail.

Case Description

A 40-year-old male presented with a two-week duration of intermittent retrosternal chest pain and was found to have a late presenting anteroseptal ST-elevation myocardial infarction (STEMI). Coronary angiography showed an occluded proximal left anterior descending (LAD) artery, along with diffuse left main and LAD disease. After a heart team discussion took place, the patient underwent coronary artery bypass grafting (CABG), utilizing left mammary artery (LIMA) to LAD and saphenous vein graft (SVG) to the diagonal branch. Pre-CABG transthoracic echocardiogram (TTE) disclosed a 35% ejection fraction with anterobasal, anterolateral and apical wall hypokinesis, and post-CABG TTE noted a 27% ejection fraction with severe global hypokinesis. Additionally, an EKG depicted Q wave anterior wall LV infarction and a prolonged QTc interval of 573 ms. Despite successful revascularization, the patient experienced a complex clinical course marked by Torsade de pointes (TdP)/polymorphic VT (PMVT) progressing to ventricular fibrillation (VFib) arrest. An urgent heart catheterization showed patent bypass grafts and unchanged native CAD. Aggressive measures were taken, including IV amiodarone and lidocaine, but the patient experienced ongoing PMVT, leading to cardiogenic shock with multiorgan failure, necessitating pressor support and intubation followed by Impella 5.5. A dual chamber Medtronic ICD was inserted on day 10 post-CABG, set to DDD mode with base rate originally set to 100 bmp, later decreased to 70 bmp. Although PMVT subsided for a brief period, the patient developed monomorphic VT (MMVT), requiring more than 10 ICD shocks, resulting in reintubation and sedation with propofol. Catheter ablation was contemplated, however, in the post-MI/cardiotomy setting, along with a high PAINESD score[12], it was thought that the risk outweighed benefits and the patient underwent a left stellate ganglion block with ropivacaine. Unfortunately, the patient continued to experience refractory ES.

Ultimately, cardiothoracic surgery performed a video-assisted thoracoscopic bilateral sympathectomy, resecting T2-T4 sympathetic ganglia. This procedure led to substantial stabilization of the patient, marked by the absence of further MMVT. The patient was discharged five days post-surgery with optimized heart failure medications and oral amiodarone. He had no recurrent VT since the procedure at 6 months follow-up. This case highlights the critical role of bilateral sympathetic ganglionectomy in managing refractory ventricular arrhythmias in a challenging clinical scenario.

Discussion:

ES, characterized by three or more ventricular arrhythmias (VA) or appropriate ICD shocks within 24 hours, represents a critical and life-threatening condition [1]. The transition from recurrent VT to electrical storm can be influenced by various factors such as the underlying cardiac pathology, comorbidities, and the efficacy of antiarrhythmic therapies. The exact rate of recurrent VT to ES is unclear. In addressing ES, the European Society of Cardiology recommends a multifaceted approach, involving antiarrhythmic medications, adrenergic blockades, sedation/anxiolysis, and hemodynamic support [12, 13]. Current therapeutic recommendations include the use of antiarrhythmic medications, such as amiodarone and lidocaine, to stabilize cardiac rhythm. Concurrently, adrenergic blockade is employed to modulate sympathetic activity, crucial in managing the heightened sympathetic tone often observed in ES. Sedation and anxiolysis play a pivotal role, ranging from benzodiazepine to general anesthesia with propofol and opioids. These measures collectively contribute to alleviating the electrical and hemodynamic disturbances [13]. Despite these interventions, cases of refractory ES pose a significant challenge. In such scenarios, percutaneous stellate ganglion block has been explored as an additional therapeutic option. While providing immediate relief in up to 92% of cases with a 50% reduction in VA burden, long-term follow-up data reveal a notable recurrence rate[14]. This prompts the consideration of more definitive interventions to address the underlying dysregulation of the sympathetic nervous system. Cardiac sympathetic denervation (CSD) through bilateral sympathetic ganglionectomy, has emerged as a promising strategy for managing persistent, refractory ES[15]. This surgical procedure involves the removal of terminal cervical and thoracic sympathetic ganglia, effectively reducing sympathetic discharge to the heart [16, 17]. Studies have demonstrated significant reductions in VT recurrence and ICD shocks, particularly in patients with ischemic and nonischemic cardiomyopathy [15, 18]. Notably, bilateral CSD has shown superiority over left-only procedures in terms of longer transplant-free survival and ICD-

shock-free time[18]. Although the surgical approach commonly involves resecting one-third to one-half of the stellate ganglion with the sympathetic chain down to the fourth rib, the optimal extent of resection remains a subject of investigation [15]. The application of CSD in patients who have had a MI with structural heart abnormalities or ES due to other cardiomyopathies needs more investigation. Further research is required in order to assess the efficacy, safety, and long-term outcomes of CSD in these specific patient populations [19]. While concerns about perioperative risk and efficacy have limited the widespread adoption of CSD, most do not experience severe complications. One study showed a 7% perioperative death attributed to refractory arrythmia and decompensated heart failure, and 88% of patients were alive after 6 months of operation [15]. Potential complications include pneumothorax, Horner's syndrome, and hemothorax[20]. Some patients have reported hypohidrosis in upper extremities, hyperhydrosis in lower extremities, neuropathic pain, and sensitivity in specific regions following CSD[16]. Our case study contributes to the growing body of evidence supporting the potential efficacy of CSD in managing refractory ES in post-MI patients with reduced LVEF. Our patient, unresponsive to multiple antiarrhythmic medications and left stellate ganglion block, exhibited successful resolution of VT storms following bilateral sympathetic ganglionectomy, with favorable tolerance and no complications. While promising, the broader application of CSD in treating refractory VA necessitates further study. Further study will be crucial for elucidating the specific role of CSD, especially in the context of post-MI patients with structural heart abnormalities or ES secondary to other cardiomyopathies. Long-term follow-ups are imperative for assessing potential complications and evaluating the sustained efficacy of CSD, shedding light on its impact on long-term mortalities in this complex patient population.

Conclusion:

Electrical storm is a life-threatening condition and can be treated effectively by bilateral sympathetic ganglionectomy in post-MI patients with structural heart abnormality when the ventricular arrhythmias are refractory to multiple antiarrhythmic drugs and ICD reprogramming. Further prospective studies are needed to evaluate bilateral sympathetic ganglionectomy's long-term effects and complications in treating electrical storms in selected patients.

References

1. Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). J Am Coll Cardiol. 2006;48(5).
2. Di Fusco S, Pignalberi C, Spinelli A, et al. In-hospital management and outcomes of electrical storm: single center retrospective study. Eur Heart J Suppl. 2022;24(Supplement_C). doi:10.1093/eurheartj/suac011.009
3. Sesselberg HW, Moss AJ, McNitt S, et al; MADIT-II Research Group. Ventricular arrhythmia storms in postinfarction patients with implantable defibrillators for primary prevention indications: a MADIT-II substudy. Heart Rhythm. 2007;4(11):1395-1402. doi:10.1016/j.hrthm.2007.07.013
4. Exner DV, Pinski SL, Wyse DG, et al; AVID Investigators. Electrical storm presages nonsudden death: the antiarrhythmics versus implantable defibrillators (AVID) trial. Circulation. 2001;103(16):2066-2071. doi:10.1161/01.cir.103.16.2066
5. Dinov B, Darma A, Nedios S, Hindricks G. Management of patients with electrical storm: an educational review. Eur Heart J Acute Cardiovasc Care. 2023;12(1):69-73. doi:10.1093/ehjacc/zuac160
6. Jentzer J, Noseworthy P, Kashou A, et al. Multidisciplinary critical care management of electrical storm: JACC state-of-the-art review. J Am Coll Cardiol. 2023;81(22):2189-2206. doi:10.1016/j.jacc.2023.03.424
7. Deyell MW, Steinberg C, Doucette S, et al. Mexiletine or catheter ablation after amiodarone failure in the VANISH trial. J Cardiovasc Electrophysiol. 2018;29(4):603-608. doi:10.1111/jce.13431
8. Daraz YM, Abdelghffar OH. Lidocaine infusion: an antiarrhythmic with neurologic toxicities. Cureus.

2022;14(3)
9. . doi:10.7759/cureus.23310
10. Tian Y, Wittwer ED, Kapa S, et al. Effective use of percutaneous stellate ganglion blockade in patients with electrical storm. Circ Arrhythm Electrophysiol. 2019;12(9)
11. . doi:10.1161/CIRCEP.118.007118
12. Ajijola OA, Lellouche N, Bourke T, et al. Bilateral cardiac sympathetic denervation for the management of electrical storm. J Am Coll Cardiol. 2012;59(1):91-92. doi:10.1016/j.jacc.2011.09.043
13. Vaseghi M, Barwad P, Malavassi Corrales FJ, et al. Cardiac sympathetic denervation for refractory ventricular arrhythmias. J Am Coll Cardiol. 2017;69(25):3070-3080. doi:10.1016/j.jacc.2017.04.035
14. Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al; ESC Scientific Document Group. 2022 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2022;43(40):3997-4126. doi:10.1093/eurheartj/ehac262
15. Jentzer J, Noseworthy P, Kashou A, et al. Multidisciplinary critical care management of electrical storm: JACC state-of-the-art review. J Am Coll Cardiol. 2023;81(22):2189-2206. doi:10.1016/j.jacc.2023.03.424
16. Savastano S, Baldi E, Compagnoni S, et al; STAR study group. Electrical storm treatment by percutaneous stellate ganglion block: the STAR study. Eur Heart J. 2024. doi:10.1093/eurheartj/ehae021
17. Elliott IA, DeJesus M, Dobaria V, et al. Minimally invasive bilateral stellate ganglionectomy for refractory ventricular tachycardia. JACC Clin Electrophysiol. 2021;7(4):533-535. doi:10.1016/j.jacep.2020.12.001
18. Dusi V, De Ferrari GM, Pugliese L, Schwartz PJ. Cardiac sympathetic denervation in channelopathies. Front Cardiovasc Med. 2019;6:27. doi:10.3389/fcvm.2019.00027
19. Barwad P, Lokhandwala YY, Kumar B, et al. Surgical cardiac sympathetic denervation in patients with VT storm: long term follow-up data. Eur Heart J. 2020;41(Supplement_2)
20. .0751. doi:10.1093/ehjci/ehaa946.0751
21. Vaseghi M, Gima J, Kanaan C, et al. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: intermediate and long-term follow-up. Heart Rhythm. 2014;11(3):360-366. doi:10.1016/j.hrthm.2013.11.028
22. Yagishita A, Goya M, Takahashi Y, et al. Bilateral cardiac sympathetic denervation of a recurrent refractory ventricular tachycardia occurring after catheter ablation of atrial fibrillation and outflow tract premature ventricular contractions. J Arrhythm. 2019;35(2):287-289. doi:10.1002/joa3.12159
23. Chihara RK, Chan EY, Meisenbach LM, Kim MP. Surgical cardiac sympathetic denervation for ventricular arrhythmias: a systematic review. Methodist Debakey Cardiovasc J. 2021;17(1):24-35. doi:10.14797/QIQG9041
24. Muser D, Castro SA, Liang JJ, Santangeli P. Identifying risk and management of acute haemodynamic decompensation during catheter ablation of ventricular tachycardia. Arrhythm Electrophysiol Rev. 2018;7(4):282-287. doi:10.15420/aer.2018.36.3

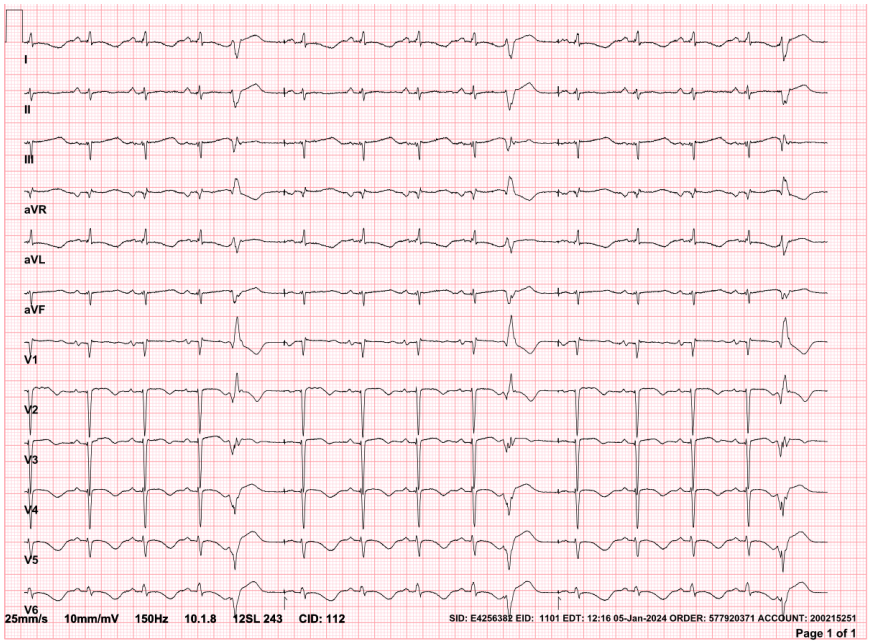

Figure 1: 12-lead EKG showing PVC on initial presentation

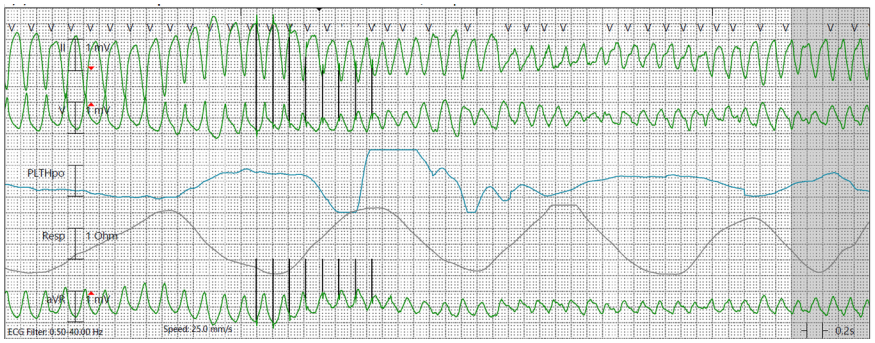

Figure 2: Torsade de pointes (TdP) progressing to ventricular fibrillation (VFib)

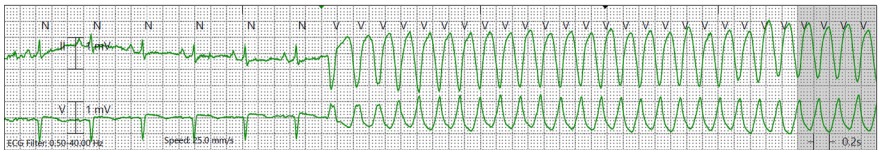

Figure 3: Monomorphic ventricular tachycardia