Case Report

Electrical storm caused by sertraline overdose: case report

Vraj Patel[1], Mina Rismani[2]^, Fakhra Sultan[2], Amr Essa[3], Pascha Schafer[3]

[1]School of Medicine, Medical College of Georgia, Augusta, GA, USA; [2]Department of Medicine, Wellstar Medical College of Georgia Health, Augusta, GA, USA; [3]Department of Cardiology, Wellstar Medical College of Georgia Health, Augusta, GA, USA

Contributions: (I) Conception and design: All authors; (II) Administrative support: All authors; (III) Provision of study materials or patients: All authors; (IV) Collection and assembly of data: All authors; (V) Data analysis and interpretation: All authors; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Mina Rismani, MD. Department of Medicine, Wellstar Medical College of Georgia Health, 1120 15th Street, Augusta, GA 30912, USA. Email: mrismani@augusta.edu.

Background: In cases of electrical storm, identifying the etiology is essential, as patients with reversible causes do not benefit from implantable cardioverter defibrillator (ICD). Given the diversity of pharmacologic and nonpharmacologic management tools available for hemodynamically unstable patients in electrical storm, all must be considered and tailored to each individual patient.

Case Description: This report describes a 36-year-old female without prior cardiac history who presented in ventricular fibrillation (VF) electrical storm. While she lacked significant electrolyte abnormalities or ischemia to explain etiology of electrical storm, she incidentally had variant coronary anatomy noted on angiography. After thorough consideration of possible etiologies of storm, selective serotonin reuptake inhibitor (SSRI) intoxication was the most highly suspected etiology. Regarding management of her hemodynamically unstable electrical storm, she was treated with lidocaine, amiodarone, as well as mechanical circulatory support devices including extracorporeal membrane oxygenation (ECMO) and intra-aortic balloon pump (IABP). The patient ultimately was decannulated from ECMO, had IABP removal, and achieved recovery of ejection fraction (EF) to baseline. She was not offered ICD as the etiology of her cardiac arrest was reversable. She was discharged with recommendation to discontinue SSRI and follow up with psychiatry regarding SSRI overdose.

Conclusions: Identification of electrical storm etiology is crucial as reversible causes do not warrant ICD placement. Selection of appropriate pharmacologic and nonpharmacologic management in the hemodynamically unstable electrical storm patient is important given the wide range of available options.

Keywords: Ventricular fibrillation (VF); electrical storm; case report

Received: 02 May 2024; Accepted: 13 September 2024; Published online: 18 November 2024.

doi: 10.21037/acr-24-93

View this article at: https://dx.doi.org/10.21037/acr-24-93

Introduction

Electrical ventricular storm is defined as the occurrence of three or more episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) within a 24-hour period (1). Common etiologies of electrical storm include ischemia, electrolyte abnormalities, structural heart disease, and drug intoxication. While drug toxicity has been reported, there is little information on selective serotonin reuptake inhibitor (SSRI) toxicity implicated in electrical storm. This report reviews a novel case of SSRI toxicity precipitating electrical storm, and the pharmacologic and nonpharmacologic tools available for management of in the hemodynamically

^ ORCID: 0000-0002-4943-143X.

unstable patient. We present the following case in accordance with the CARE reporting checklist (available at https://acr.amegroups.com/article/view/10.21037/acr-24-93/rc).

Case presentation

This is the case of a 36-year-old female with a past medical history of depression on sertraline who presented to the emergency department (ED) after out-of-hospital cardiac arrest. In the field, the patient had witnessed seizure-like activity followed by cardiac arrest and received bystander cardiopulmonary resuscitation (CPR) and one automated external defibrillator shock delivery, with return of spontaneous circulation (ROSC) achieved within a few minutes. Emergency Medical Services transported the patient to the ED, where the patient was noted to have seizure activity progressing to status epilepticus. Admission vitals included heart rate 85 beats per minute, respiratory rate 20 breaths per minute, blood pressure 106/93 mmHg, and oxygen saturation 93% on room air. Electrocardiogram (EKG) initially normal sinus rhythm without ST changes, QTc 423 ms, QRS duration 97 ms. Initial labs significant for potassium 3.8 mmol/L, serum bicarbonate 20 mEq/L, lactic acid 3.1 mmol/L. Magnesium was 2.4 mg/dL and all other labs were within normal limits. She was intubated for airway protection and admitted to the neuro intensive care unit (ICU) for status epilepticus management. While in the neuro ICU, she had another episode of VF arrest requiring multiple rounds of CPR and shock delivery prior to ROSC, though she subsequently had multiple additional episodes of VF consistent with ventricular storm. The cardiology team was called to bedside, and she was subsequently transferred to the cardiovascular ICU (CVICU) for further management. This patient meets the criteria for electrical storm as she had over three occurrences of VF events within 24 hours.

As a result of the VF electrical storm, the patient went into severe cardiogenic shock requiring four pressors which included levophed, epinephrine, vasopressin, and dopamine. The sole electrolyte abnormality that was noted was mild hypokalemia. Of note, the patient received multiple boluses of potassium, magnesium, and calcium during the code event. She was first bolused with amiodarone and then put on an amiodarone drip. Shortly after, she was bolused with lidocaine and put on a lidocaine drip as well. She was subsequently put on venous-arterial (VA) extracorporeal membrane oxygenation (ECMO) (cannulated via femoral artery-femoral vein) and intra-aortic balloon pump (IABP) for refractory VF.

To further investigate precipitants of electrical ventricular storm, the patient underwent work-up including transthoracic echocardiogram (TTE) and cardiac catheterization to assess for structural disease and ischemic disease, respectively. Many patients who present with VT/VF storm have underlying structural heart disease and scarring post-myocardial infarction (MI) (2). During heart catheterization, the patient required multiple shock deliveries for recurrent VF episodes. In all, the patient had more than 25 episodes of VF arrest during the catheterization. An EKG obtained in between one of these episodes reveled augmented vector right (aVR) elevation and diffuse ST depressions. In total, the patient received 60 shocks for VF storm.

The results from the left heart catheterization (LHC) and right heart catheterization (RHC) were as follows: left ventricular end diastolic pressure 14 mmHg, right atrial pressure 12 mmHg, right ventricular pressure 24/12 mmHg, pulmonary artery pressure 36/10 mmHg, mean pulmonary artery pressure 18 mmHg, postcapillary wedge pressure 16 mmHg. Coronary angiography revealed a large left main artery and left anterior descending artery without obstructive coronary artery disease (CAD). Branches from the left anterior descending artery included the right ventricular marginal artery. The circumflex artery was found to be a large dominant vessel and it gave off the posterior

Highlight box

Key findings

- Selective serotonin reuptake inhibitor (SSRI) overdose can precipitate ventricular fibrillation electrical storm.

What is known and what is new?

- Drug toxicity is known to precipitate electrical storm. These cases do not require implantable cardioverter defibrillator (ICD) placement as it is a reversible cause.
- This is the first case of SSRI toxicity causing electrical storm. Management requires selection of specific pharmacologic agents as well as nonpharmacologic interventions, including mechanical circulatory support devices, stellate ganglion blockade, and catheter ablation.

What is the implication, and what should change now?

- More research on minimum acute dosage of SSRI is warranted. Obtaining a thorough history of patients presenting with electrical storm is essential, as identifying a reversible cause mitigates necessity of ICD implantation and directs more appropriate management.

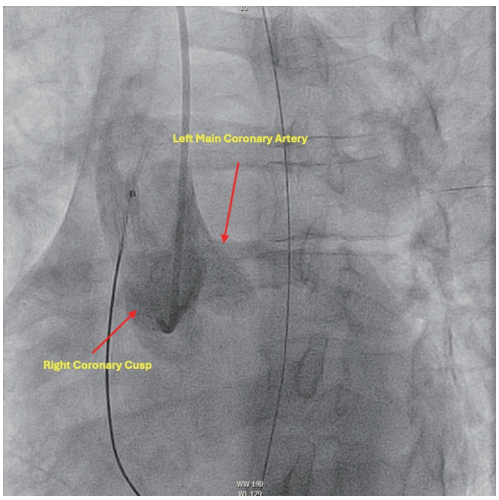

Figure 1 Aortogram showing absence of right coronary artery.

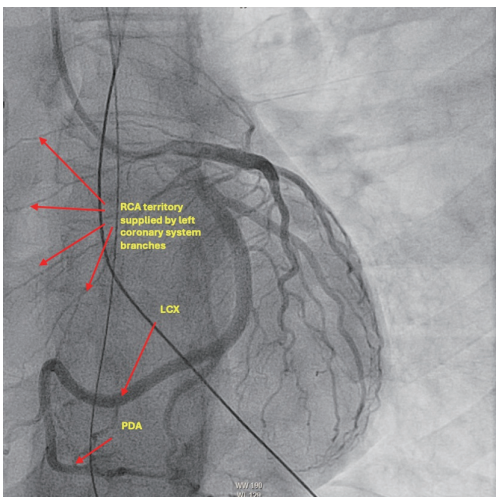

Figure 2 Coronary angiogram with PDA originating from the LCX with RCA territory supplied by branches from the left system. PDA, posterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

descending artery. The circumflex artery also gave rise to a distal left circumflex branch that wrapped around the atrioventricular groove. Of note, no right coronary artery (RCA) was identified during this procedure (*Figures 1-3*). TTE was remarkable for ejection fraction (EF) <15%, global hypokinesis, and no significant valvular pathology.

Based on the cardiac catheterization results, there was no evidence of obstructive CAD to cause ischemia, making myocardial ischemia a much less likely etiology

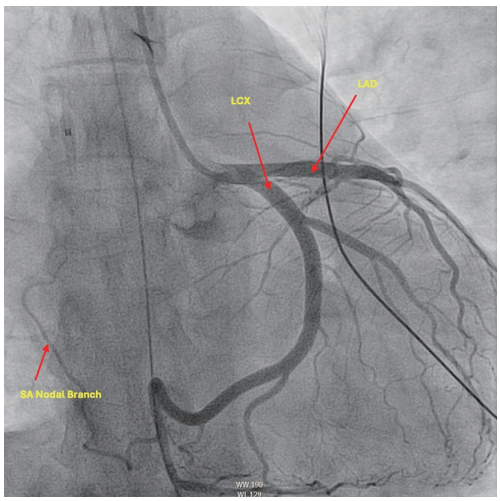

Figure 3 Coronary angiogram with SA nodal branch originating from the LCX. Also note LAD. SA, sinoatrial; LCX, left circumflex artery; LAD, left anterior descending artery.

of VF storm. A congenital coronary artery anomaly was considered as a cause for the arrest given that the patient did not have an RCA identified on angiography, however, her troponin peaked at 1.1 and a higher troponin leak would have been expected if an anomalous coronary artery precipitating ischemia was the etiology. In addition, the coronary anomaly did not have factors such as a malignant course or slit-like orifice typically associated with sudden cardiac arrest. At this point, the etiology of her arrest was still unknown.

The patient remained in the CVICU on pressors, amiodarone and lidocaine drips, ECMO, and IABP. At this point, she had been about 48 hours out from the last shock for ventricular arrhythmia. Due to the concern of lidocaine lowering the seizure threshold, it was discontinued, and her amiodarone drip was transitioned to an oral route. Antiepileptic drugs were subsequently stopped as her electroencephalogram (EEG) did not show concern for any seizure activity.

After four days of hemodynamic support, the patient was noted to have neurologic recovery with resolution of her cardiogenic shock. She was decannulated from ECMO and subsequently, the IABP was also removed and she was successfully extubated. With the patient back in normal sinus rhythm, an interval TTE was performed to reevaluate her cardiac function and look for any wall motion abnormalities. The patient was noted to have complete recovery of her cardiac function with EF of 55–60%,

without any regional wall motion abnormalities, diastolic dysfunction, or any valvular abnormalities. With these findings, she was transferred to the cardiology floor.

After transfer, the cardiology floor team received collateral reports from family and coworkers concerning a social media post from the patient prior to the event that was suggestive of suicidal ideation due to a recent marital issue. Some close contacts suggested a possible sertraline overdose at the time of her arrest. Poison control was contacted; they suggested that there are no known case reports of sertraline toxicity leading to VF arrest.

Given the concern for sertraline overdose as a cause of the patient's cardiac arrest, psychiatry was consulted. Her status epilepticus at presentation further supported concern for SSRI overdose, as seizures have been reported as a clinical manifestation. While she was tachycardic at admission, she did not have myoclonus, fever, hyperreflexia, or tremor typical of serotonin syndrome. The patient's outpatient prescription was for sertraline 100 mg daily. She last filled 90 tablets two and a half months prior to her admission, and her prescription bottle found in her personal belongings was half empty. There were concerns regarding potential noncompliance and possibility that she consumed a toxic quantity at once. Her urine drug screen was negative at admission, but unfortunately a toxicology screen including sertraline level was not obtained. The patient persistently denied overdose; however, she was unable to recall the events surrounding her cardiac arrest.

Since the most likely etiology was an overdose of sertraline (a reversible cause of her cardiac arrest), implantable cardioverter defibrillator (ICD) implantation was not indicated. In cases of arrest where a reversible cause is identified, ICD implantation and antiarrhythmic medications are unlikely to provide any medical benefit in terms of preventing future episodes. ICD implantation for secondary prophylaxis is only indicated for hemodynamically relevant tachycardia diagnosed without a reversible cause (3).

At this point, her only inpatient medications were aspirin 81 mg daily and amiodarone 400 mg ter in die (TID). These medications were discontinued as sertraline overdose was determined to be the most likely precipitant of her arrest. She was discharged with explicit instructions to discontinue her home medication, sertraline. Due to the lack of any evidence of ongoing suicidal ideation or other safety concerns per the psychiatry team, the patient was not placed on involuntary commitment to a psychiatric facility during admission. She was discharged and provided instructions and resources for psychiatric follow up in the outpatient setting.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

Regarding pharmacologic interventions for electrical storm, this patient was put on amiodarone and lidocaine drips. Advanced Cardiovascular Life Support (ACLS) guidelines from the "2020 Handbook of Emergency Cardiovascular Care for Healthcare Providers" suggest intravenous antiarrhythmics and beta blockers as the initial primary treatment (4). However, in practice, amiodarone is typically used as first line therapy for electrical storm in both hemodynamically stable and unstable patients. Lidocaine is also frequently utilized as a secondary agent (5). Given that electrical storm pathophysiology involves catecholamine surge, beta blockers also have a role in therapy. While propranolol, metoprolol, and esmolol have been reported in the literature for treating electrical storm, propranolol has demonstrated superiority (1).

Nonpharmacologic interventions for electrical storm include mechanical circulatory support devices (ECMO, IABP), left stellate ganglion blockage, and catheter ablation. While catheter ablation and stellate ganglion blockade are both options for patients failing pharmacologic intervention for ventricular storm, the most important next step in this patient was mechanical circulatory support device given hemodynamic instability. Thus, she was put on VA ECMO for refractory VF. Early implementation of ECMO has been associated with successful outcomes including prevention of end organ damage and maintenance of sufficient cardiac unloading (6). An IABP was also placed as its counterpulsation for hemodynamic support has been frequently documented in literature for its role in suppressing VT storm (7). IABP is effective in treating electrical storm even outside the presence of ischemia, likely due to mechanisms of reducing afterload, left ventricle (LV) size, and wall tension (8,9).

Stellate ganglion block is an option for stabilizing ventricular rhythm in patients who have failed other therapies (deep sedation and general anesthesia to reduce

sympathetic output, antiarrhythmic drugs and beta blockers to suppress ventricular arrhythmias). This procedure involves ultrasound guided or fluoroscopy guided unilateral or bilateral block of stellate ganglion to suppress ventricular storm. One study reports stellate ganglion block effectively attenuating electrical storm in more than half of patients who failed other interventions without procedure-related complications (10). The mechanism behind stellate ganglion block suppressing electrical storm is related to direct targeting of the nerve fibers which innervate the myocardium and reduced adrenergic tone (11).

Additionally, retrospective studies in patients who have failed medical therapy for VT storm show that catheter ablation is highly effective (12). One retrospective study demonstrates that catheter ablation was associated with greater ventricular arrhythmia-free survival, greater cardiac hospitalization-free survival, fewer total number of cardiac-related hospitalizations, less serious adverse events at 12 months after first electrical storm admission, and greater survival free from composite end point of death, cardiac transplantation, storm recurrence, and storm hospitalization compared to medical therapy (13). However, randomized control trials are necessary before making ablation first-line therapy for electrical storm management. Radiofrequency ablation is also indicated in recurrent polymorphic VT or VF when specific triggers such as monomorphic premature ventricular contractions (PVCs) can be targeted, but neither one of these cases applied to our patient. In these clinical scenarios, electrical storm has been well-suppressed in patients with ischemic and nonischemic cardiomyopathy (14,15).

In terms of etiologies of electrical storm, common causes include drug toxicity, significant electrolyte disturbances (hypokalemia, hypomagnesemia), new or worsening heart failure, acute myocardial ischemia, thyrotoxicosis, or QT prolongation (16). Other risk factors for VT storm include advanced age, male gender, a low left ventricular EF, New York Heart Association (NYHA) class III or IV heart failure, and chronic kidney disease (CKD) (17). None of these factors other than possible drug toxicity were relevant to our patient. As previously stated, she had a congenital coronary artery anomaly on angiography, however, a higher troponin leak or malignant features would have been expected if an anomalous coronary artery precipitating ischemia was the etiology. Thus, structural cause was a less likely etiology than SSRI toxicity. Additionally, her Schwartz Score was <1, and in the absence of family history of long QT syndrome, she did not undergo genetic testing for this syndrome.

Despite ubiquitous utilization of SSRIs, there is a lack of literature defining a minimum acute dosage of SSRIs. One study showed that for doses greater than five times the lowest adult therapeutic dose of a single SSRI (in the case of sertraline when prescribed for depression, 250 mg), patients should receive treatment (18). Most case reports of SSRI overdose describe an array of serotonergic symptoms such as dizziness, nausea, vomiting, blurred vision, central nervous system depression, and sinus tachycardia. In cases of SSRI toxicity, seizures and cardiovascular complications, including QT prolongation leading to malignant arrhythmias like VT are also reported. SSRIs induce QT prolongation by antagonizing myocyte potassium channels, potentially triggering fatal reentrant tachycardias like torsades de pointes and other malignant arrhythmias such as VT/VF (19). This risk increases with higher doses of citalopram, especially in vulnerable populations such as patients over the age of 60 years and those with a history of heart disease or other QT prolongation risk factors. Sertraline carries a lower risk of QT prolongation compared to citalopram and escitalopram, although this risk is more significant in patients with liver impairment. Therefore, for vulnerable patients suspected of overdosing on citalopram or escitalopram, EKG and continuous cardiac monitoring are recommended to promptly detect the onset of ventricular dysrhythmias and QT prolongation. Among SSRIs, citalopram is the most frequently cited offender of the class. With suspected overdose of citalopram or escitalopram specifically, EKG and cardiac monitoring are recommended to identify the development of ventricular dysrhythmias and QT prolongation (20).

Conclusions

This case highlights the diverse pharmacologic and nonpharmacologic approaches available for management of VT/VF storm. To our knowledge, this is the only case reported in the literature of SSRI overdose precipitating VF storm. This case also emphasizes the importance of obtaining a thorough history in patients presenting with VT/VF storm, as the etiology determines whether ICD implantation is appropriate or not. Lastly, more research on SSRI toxicity is warranted, especially in terms of cardiotoxicity and minimum acute dosages with different agents.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://acr.amegroups.com/article/view/10.21037/acr-24-93/rc

Peer Review File: Available at https://acr.amegroups.com/article/view/10.21037/acr-24-93/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://acr.amegroups.com/article/view/10.21037/acr-24-93/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

References

1. Dusi V, Angelini F, Gravinese C, et al. Electrical storm management in structural heart disease. Eur Heart J Suppl 2023;25:C242-8.
2. Villacastín J, Almendral J, Arenal A, et al. Incidence and clinical significance of multiple consecutive, appropriate, high-energy discharges in patients with implanted cardioverter-defibrillators. Circulation 1996;93:753-62.
3. Kovacs B, Reek S, Krasniqi N, et al. Extended Use of the Wearable Cardioverter-Defibrillator: Which Patients Are Most Likely to Benefit? Cardiol Res Pract 2018;2018:7373610.
4. American Heart Association. ACLS guidelines from the 2020 Handbook of Emergency Cardiovascular Care for Healthcare Providers. Handbook of Emergency Cardiovascular Care for Healthcare Providers. American Heart Association, Dallas, TX; 2020.
5. Sweeney M, Eldrich S. An Electrical Storm Is Coming!: Ventricular Storm Case Study. Adv Emerg Nurs J 2022;44:302-11.
6. Eifling M, Razavi M, Massumi A. The evaluation and management of electrical storm. Tex Heart Inst J 2011;38:111-21.
7. Chia PL, Loh SY, Foo D. Ventricular tachycardia storm: a case series and literature review. Med J Malaysia 2012;67:582-4.
8. Fotopoulos GD, Mason MJ, Walker S, et al. Stabilisation of medically refractory ventricular arrhythmia by intra-aortic balloon counterpulsation. Heart 1999;82:96-100.
9. Franz MR, Burkhoff D, Yue DT, et al. Mechanically induced action potential changes and arrhythmia in isolated and in situ canine hearts. Cardiovasc Res 1989;23:213-23.
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:e007118.
11. Eisenach JC, Tong CY. Site of hemodynamic effects of intrathecal alpha 2-adrenergic agonists. Anesthesiology 1991;74:766-71.
12. Nayyar S, Ganesan AN, Brooks AG, et al. Venturing into ventricular arrhythmia storm: a systematic review and meta-analysis. Eur Heart J 2013;34:560-71.
13. Huang K, Bennett RG, Campbell T, et al. Circulation: Arrhythmia and electrophysiology. Circulation: Arrhythmia and Electrophysiology 2022;15:803-12.
14. Kohsaka S, Razavi M, Massumi A. Idiopathic ventricular fibrillation successfully terminated by radiofrequency ablation of the distal Purkinje fibers. Pacing Clin Electrophysiol 2007;30:701-4.
15. Haïssaguerre M, Shoda M, Jaïs P, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circulation 2002;106:962-7.
16. Haegeli LM, Della Bella P, Brunckhorst CB. Management of a Patient With Electrical Storm: Role of Epicardial Catheter Ablation. Circulation 2016;133:672-6.
17. Gatzoulis KA, Andrikopoulos GK, Apostolopoulos T, et al. Electrical storm is an independent predictor of adverse

long-term outcome in the era of implantable defibrillator therapy. Europace 2005;7:184-92.
18. Nelson LS, Erdman AR, Booze LL, et al. Selective serotonin reuptake inhibitor poisoning: An evidence-based consensus guideline for out-of-hospital management. Clin Toxicol (Phila) 2007;45:315-32.
19. Edinoff AN, Akuly HA, Hanna TA, et al. Selective Serotonin Reuptake Inhibitors and Adverse Effects: A Narrative Review. Neurol Int 2021;13:387-401.
20. Fitzgerald KT, Bronstein AC. Selective serotonin reuptake inhibitor exposure. Top Companion Anim Med 2013;28:13-7.

doi: 10.21037/acr-24-93
Cite this article as: Patel V, Rismani M, Sultan F, Essa A, Schafer P. Electrical storm caused by sertraline overdose: case report. AME Case Rep 2025;9:22.