



Post Cardiac Surgery Ventricular Electrical Storm, A Successful Management - Case Report and Discussion

Mohd Shahbaaz Khan^{1*}, Eliyas Patwary² and Rajesh Sharma¹

¹Department of Pediatric cardiac surgery, Fortis Escorts Heart Institute, India

²Fellow in Department of Pediatric cardiac surgery, Fortis Escorts Heart Institute, India

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***Corresponding author:** Mohd Shahbaaz Khan, Department of Pediatric cardiac surgery, Fortis Escorts Heart Institute, New Delhi, India.

Abstract

Background: ventricular electrical storm is not so uncommon these days. It is a life threatening complication in post operative period which physicians have to face sometimes during practice. Management of electrical storm is very complex and challenging. Ventricular electrical storm can be of three types- Monomorphic Ventricular tachycardia, Polymorphic ventricular tachycardia and Ventricular fibrillation.

Case report: This was an 8-year-old boy admitted for redo- aortic valve replacement. After surgery in immediate post operative period in ICU he developed severe ventricular electrical storm. This was a polymorphic ventricular tachycardia following aortic valve replacement surgery. This patient received 45 defibrillatory shocks and other anti-arrhythmic medicines and then full sedation and paralyzing agents to revert to normal sinus rhythm. Outcome was very satisfactory with normal sinus rhythm and no residual neurological deficit or any other abnormality.

Conclusion: Ventricular electrical storm is severe life threatening complication. It needs early detection and intervention to control the event. It can be controlled by defibrillations and combination of multiple intravenous anti-arrhythmic drugs.

Keywords: Ventricular electrical storm; Defibrillation

Abbreviations: ES: Electrical Storm; VT: Ventricular Tachycardia; ECG: Electrocardiogram; LVEF: Left Ventricular Ejection Fraction; ABG: Arterial Blood Gas; ACLS: Advanced Cardiac Life Support

Introduction

TElectrical storm is not so uncommon these days and it's a life threatening complication in cardiac surgery. Electrical storm has been infrequently reported in children. The term electrical storm (ES) was introduced in the 1990s to describe a state of electrical instability of the heart characterized by a series of malignant ventricular arrhythmias in a short period of time [1]. Electrical storm is defined as the recurrence of hemodynamically unstable ventricular tachycardia and/or ventricular fibrillation, twice or more in 24 hours, requiring electrical cardioversion or defibrillation [2]. With the arrival of the implantable cardioverter defibrillator, this definition was broadened, and electrical storm is now defined as the occurrence of 3 or more sustained episodes of ventricular tachycardia, ventricular fibrillation, or appropriate shocks from an implantable cardioverter-defibrillator within 24 hours. Sustained VT lasts 30 seconds, involves hemodynamic compromise, or requires intervention to terminate the episode. The episodes of VT must be separate, meaning that the persistence of ventricular

tachycardia following inefficacious intervention is not regarded as a second episode [3]. By contrast, a sustained ventricular tachycardia that resumes immediately after (≥ 1 sinus cycle and within 5 minutes) efficacious therapeutic intervention by the defibrillator is regarded as a severe form of electrical storm [3].

This condition has been described in patients with post-infarction ischemic heart disease, various forms of cardiomyopathy, valve disease, corrected congenital heart disease and genetically determined heart diseases with no apparent structural alteration, as for example in Brugada syndrome [4]. The mechanisms of electrical storm are quite complex and not well understood. Each case of electrical storm may represent different underlying cause and electrophysiologic mechanism. It has been postulated that cellular and molecular alterations can increase intracellular calcium overload and changes of the action potential duration and morphology that lead to the onset of electrical storm [5]. Effective management of electrical storm needs good knowledge of mechanism of

electrical storm, available treatment options, and ICD and emergency techniques to treat refractory cases.

Case Report

Here we are presenting a case of an 8-year-old-boy, who was admitted to our hospital with complaints of progressive breathlessness and intermittent fever for one month. He was operated 1 year before for congenital aortic stenosis and had aortic valve replacement done with 21 mm sized St. Jude Bio-prosthetic valve. He was treated for suspected infective endocarditis in another hospital. After admission to our hospital proer examination and full investigations were done.

a) Electrocardiogram (ECG): Preoperative 12-lead electrocardiogram (ECG) showed sinus tachycardia with normal QTc interval (0.42 sec) and left bundle branch block pattern.

b) Echocardiography: A 2D echocardiography with color doppler study showed severe left ventricular dysfunction. Preoperative left ventricular ejection fraction (LVEF) was 25% only, with stuck aortic valve (no vegetations).

c) Laboratory investigations: Full septic screening was sent to rule out infective organism. His initial blood cultures were negative. He developed hemodynamically stable ventricular tachy-cardia after admission and was started on amiodarone Intravenous infusion.

d) Surgery: He underwent repeat aortic valve replacement with 19 mm sized TTK Chitra aortic mechanical tilting disc prosthesis. Intraoperative findings revealed stuck aortic valve with vegetations; valve tissue was sent for histological and microbiological study and it came as carbapenem resistant *Klebsiella pneumoniae*.

Post operative period

He was on inotropic and ventilator supports in ICU. He had sinus bradycardia. Amiodarone was tapered over 36 hours and he was maintained on overdrive AV sequential pacing. The patient was in low cardiac output state with fluctuating hemodynamics and blood pressure was maintained with adjusting inotropic support. Echocardiography was done in first post-operative day. Postoperative trans-esophageal echocardiography revealed biventricular dysfunction (LVEF 10-15%), and no residual gradient across aortic valve. On 2nd postoperative day he developed recurrent episodes of polymorphic ventricular tachycardia (Figure 1). Arterial blood gas (ABG) analysis showed normal electrolytes and acid base physiology. In next 8 hours, 45 DC shocks (up to 8J/kg) were delivered due to recurrence of VTs after transient reversion to sinus rhythm. He also received two boluses of intravenous (IV) amiodarone (5 mg/kg) and repeated doses of IV lidocaine (1 mg/kg) followed by their infusions. Since the patient was poorly responsive, he was also started on IV esmolol infusion after bolus.

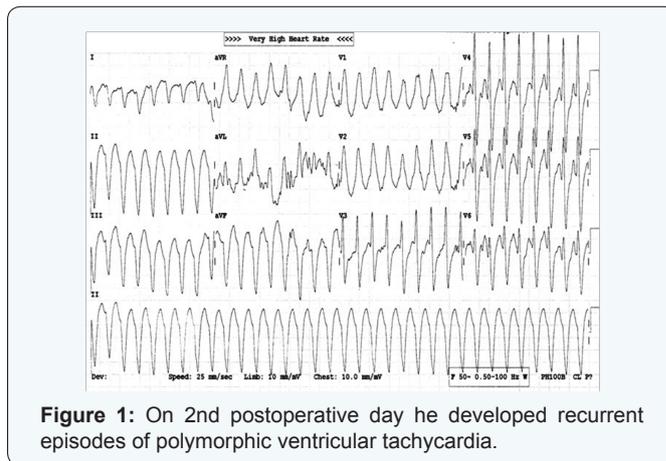


Figure 1: On 2nd postoperative day he developed recurrent episodes of polymorphic ventricular tachycardia.

There was no significant change in QTc interval despite multiple doses of amiodarone. Magnesium sulphate was given and electrolytes corrected. Finally, it was controlled with deep sedation and paralysis with fentanyl, midazolam and vecuronium, with infusions of lidocaine at 40 µg/kg/min, amiodarone at 20 µg/kg/min and esmolol at 100 µg/kg/min. Post-event, he had LVEF of 10% with septal and apical akinesis, borderline low blood pressure and high left atrial pressure. Inotropic support was reoptimized with dobutamine and milrinone, and ventilation was continued for next 72 hours. His left ventricular function gradually improved and he was extubated on 6th postoperative day with normal neurological status. He was continued on oral amiodarone, metoprolol and acetylcholinesterase inhibitors.

i. Follow up: At follow-up 14 days later, he was in sinus rhythm consistently.

Discussion

Incidence

Table 1: Incidence of electrical storm varies according to the population of study and definition.

Author	Electrical storm definition	% Incidence
Wood [11]	>3 VT in 24 hours	10
Villacastin [12]	>2 shocks for single VT	20
Fries [13]	>2 VT in 1 hour	60
Credner [8]	>3 VT in 24 hours	10
Exner [7]	>3 VT in 24 hours	20
Arya [14]	>3 VT in 24 hours	14
Stuber [15]	>3 VT in 3 weeks	24
Gasparini [16]	>3 VT in 24 hours	7
Greene [17]	>3 VT in 24 hours	18
Gatzoluis [18]	>3 VT in 24 hours	19

Incidence of electrical storm varies according to the population of study and definition (Table 1) In a MADIT-II

substudy of 719 patients [6], 4% developed electrical storm over an average of 20.6 months. Electrical storm might be an independent risk factor for cardiac death. In the AVID trial [7] patients with electrical storm had an increased risk of non-sudden cardiac death (risk ratio, 2.4). In the Madit -II substudy, patients with electrical storm had a 7.4-fold higher risk of death than patients without electrical storm. Both studies showed that the risk of death was highest within the first 3 months after a storm. The prognosis remained poor for patients who survived the initial period of electrical instability. It is unclear whether electrical storm contributes directly to a poor outcome or is simply a result of advanced structural heart disease [8]. Recurrent VT or VF and ICD shocks may cause left ventricular (LV) systolic dysfunction and myocardial injury [9] which can lead to adrenergic neurohormonal activation and exacerbate heart failure [10].

Clinical Syndromes of Electrical Storm

Electrical storm can initially be classified on the basis of 3 gross electrocardiographic (ECG) surface morphologies: monomorphic VT, polymorphic VT, or VF.

Monomorphic ventricular tachycardia

Monomorphic VT occurs when the ventricular activation sequence is the same without any variation in the QRS complexes. Most monomorphic VT is due to electrical wavefront reentry around a fixed anatomic barrier which is most commonly scar tissue after MI. Monomorphic VT due to wavefront reentry does not require active ischemia as a trigger and it is uncommon in patients who have an acute MI [11-18].

Polymorphic ventricular tachycardia

Polymorphic VT occurs when the ventricular activation sequence on ECG consists of beat to-beat variations in the QRS complexes. For polymorphic complexes, multiple wavefronts must propagate throughout the heart or appear simultaneously in several parts of the heart [19]. Polymorphic VT can be associated with a normal or a prolonged QT interval in sinus rhythm. Polymorphic VT is most commonly associated with acute ischemic syndromes but can also be seen in organic heart disease, acute myocarditis or hypertrophic cardiomyopathy.

Ventricular Fibrillation

Ventricular fibrillation is usually fatal if it is not treated promptly. Even with defibrillation, VF may recur repeatedly and present as electrical storm. When this happens, mortality rates are between 85% and 97% [20]. Ischemia, which is the primary mechanism of VF storm, should be the focus of treatment

Mechanism of Ventricular electrical storm

The mechanisms of electrical storm are quite complex and not well understood. It has been postulated that cellular and membrane alterations can increase intracellular calcium overload, with altered action potential duration and morphology

leading to its onset [21]. The important role of increased sympathetic tone has been well documented. Many conditions including ischemia, surgery [22] and hyperthermia [23] can precipitate increased adrenergic output.

Pharmacologic Therapy for Electrical Storm

Adrenergic blockade: Epinephrin and vasopressin are recommended for pulseless VT and VF according to current guidelines for advanced cardiac life support. Studies have shown improved coronary blood flow and short-term survival after the administration of epinephrine [24], but Epinephrine makes the patient more susceptible to VF due to contribution to myocardial dysfunction [25].

β -Blockers: β -Blockers decrease the susceptibility for VT and VF. Although most of the β -Blockers are effective in decreasing susceptibility but most of the studies are done with Propranolol. The lipophilic nature of propranolol enables active penetration of the central nervous system and the blockade of central and prejunctional receptors in addition to peripheral β receptors [26]. Propranolol may effectively suppress an electrical storm even when metoprolol has failed [27]. Therefore, propranolol is the preferred β -blocker.

Nademanee et al. [28] investigated the efficacy of sympathetic blockade in electrical storm by comparing propranolol, esmolol, and left stellate ganglionic blockade to combined lidocaine, procainamide, and bretylium therapy. Their patients had experienced a recent MI and more than 20 episodes of VT within 24 hours. Although the trial was nonrandomized, sympathetic blockade provided a marked survival advantage (78% vs 18% at 1wk, and 67% vs 5% at 1 yr). Despite the high doses of propranolol, heart failure was not exacerbated. These authors and others have suggested that the combination of amiodarone and propranolol improves survival rates and should be the mainstay of therapy in managing electrical storm.

In our patient, we used esmolol (predominantly a β -1 antagonist), which can be used as an infusion and dose can be easily titrated based on response.

Amiodarone: Amiodarone is widely used in the treatment of electrical storm [29]. In acute amiodarone therapy, rapid intravenous administration blocks fast sodium channels, inhibits norepinephrine release, and blocks L-type calcium channels. Amiodarone can be effective even when other agents have been ineffective. Levine et al. [30] examined 273 hospitalized patients who had electrical storm that was refractory to lidocaine, procainamide, and bretylium therapy. When amiodarone was given, 46% of the patients survived for 24 hours without another episode of VT, and another 12% responded after taking amiodarone plus another agent. Current Advanced Cardiac Life Support (ACLS) guidelines recommend amiodarone for cardiac arrest in children associated with shock-refractory VT/VF. Studies examining the effect of intravenous amiodarone in the management of electrical storm have reported its efficacy [1].

Class I Antiarrhythmic (Sodium Channel-Blocking) Agents

Lidocaine binds to fast sodium channels and binding increases under cellular conditions that are common in ischemic VT, such as a reduced pH, a faster stimulation rate, and a reduced membrane potential [31]. However, outside the setting of ischemia, lidocaine has relatively weak antiarrhythmic properties: conversion rates from VT to sinus rhythm range from 8% to 30%. If lidocaine is used, it should be administered as an intravenous bolus of 0.5 to 0.75 mg/kg that is repeated every 5 to 10 min as needed. A continuous intravenous infusion of 1 to 4 mg/min maintains therapeutic levels. The maximum total dose is 3 mg/kg over 1 hr. Procainamide- When given as a loading dose of 100 mg over 5 min; procainamide is a reasonable choice for terminating monomorphic VT. In patients with depressed systolic function, procainamide can cause hypotension or prolong the width of the QRS complex by more than 50%, which would necessitate discontinuation of the drug.

Anesthetic agents

All patients who have electrical storm should be sedated. Short-acting anesthetics such as propofol, benzodiazepines, and some agents of general anesthesia have been associated with the conversion and suppression of VT [32].

Non pharmacologic therapy

Mechanical assisted devices as Intra-aortic balloon pump, extra corporeal membrane oxygenator supports, left ventricular assisted devices can also be used as non pharmacological agents. These devices increase coronary perfusion pressure and can dramatically relieve the ischemic substrate.

Electrical Storm in ICD Patients

ICDs do not prevent arrhythmias and implanting an ICD is contraindicated in the acute phase of electrical storm. Intravenous analgesics and sedatives should be given early and aggressively to patients who sustain multiple ICD shocks [33]. If an ICD fails to convert a life threatening rhythm, external defibrillation pads should be ready for use. Being very unstable nature of the disease, electric storm often requires combination therapy. Manolis, *et al.* [34] reported a case using triple drug intervention with a beta antagonist, class III antiarrhythmic, and a class IB antiarrhythmic.

Conclusion

Ventricular electrical storm is a challenging situation. Despite repeated defibrillations and severe left ventricular dysfunction, our patient made a good recovery with aggressive supportive treatment. It is advisable that clinicians should be well versed with Pediatric Advanced Life Support guidelines to manage these challenging resistant arrhythmias.

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