Heart Sci J 2023; 4(1): 40-43

Contents list available at www.heartscience.ub.ac.id

Heart Science Journal

Journal Homepage : www.heartscience.ub.ac.id

Case Report

Ventricular Tachycardia Storm Management in Acute Cardiac Care: Prompt response to life-threatening conditions

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ARTICLE INFO

Keywords:
VT Storm
Cardioversion;
ICD;
life-threatening.

ABSTRACT

Background: Ventricular Tachycardia (VT) storm or electrical storm (ES) is defined as cardiac electrical instability and refers to the occurrence of three or more ventricular tachyarrhythmias (VT and or ventricular fibrillation (VF)) in a 24-hour period, or VT recurring soon (within five minutes) after termination of another VT episode, or sustained or no sustained VT with total ectopic beats greater than sinus beats in a 24-hour period. The frequency of VT storms varies on population. When ICDs are implanted for primary prevention (4 percent), it is lower than when they are implanted for secondary prevention (20 percent).
Case Illustration: We presented patient with Ventricular Tachycardia (VT) storm. A 63-year old woman was admitted to emergency room with chief complaint frequent episodes of palpitation. She was found to have monomorphic VT with unstable hemodynamic. Then she got cardioversion 100 Joule, continued with lidocaine drip and VT reverted to sinus rhythm. Patient admitted to intensive cardiovascular care unit, but she had refractory VT. Although she had got complete revascularization for coronary artery before, but the episodic VT still occurred accompanied with cardiogenic shock (CS) and pulmonary edema. She got cardioversion, amiodarone iv and inotropes. She was observed at ICVCU. After the condition stable, she was discharged and planned for ICD insertion at the next admission.
Discussion: We discuss the various available treatment options for VT storm and practical challenges faced in management of hemodynamically unstable VT storm. Initial management involves identifying and correcting the underlying ischemia, electrolyte imbalances, or other inciting factors.
Conclusion: Unfortunately, after angioplasty or stenting, primary patency is poor. If there is recurring stenosis, the procedure can be repeated.

1. Introduction

Recurrent occurrences of ventricular arrhythmias are a life-threatening illness known as an electrical storm. Three or more sustained episodes of ventricular tachycardia (VT), ventricular fibrillation (VF), or appropriate implantable cardioverter-defibrillator (ICD) shocks in a 24-hour period in which lasts 30 seconds, is associated with hemodynamic deterioration, and requires intervention planning until the problem solved.[1] Electrical storms are linked to high rates of illness and mortality, with fatality rates as high as 14% in the first 48 hours. When compared to control patients with ICDs, studies have consistently shown that individuals with ES have a higher death rate. According to one research, the risk of mortality with ES was 2.4 times greater than with isolated VT/VF, and climbed to 5.4 times in the first three months.[2,3]

The prognosis for electrical storms has been proven to be bad on several occasions, owing to a high short-term death rate (10–20 percent after 48 hours) (particularly when no medicinal answer can be identified), which rises to 20–35 percent at prolonged follow-up.[4]

Electrical storms can happen in a number of ways, and successful therapy requires a thorough knowledge of the mechanism that causes repeated arrhythmias.[3] Acute electrical storm management entails a number of therapies or measures that must be administered simultaneously and urgently.[4] Electrical storm management is difficult, however identifying patients based on the kind of recurrent arrhythmia (monomorphic VT or polymorphic VT/VF) and whether or not they have structural heart disease would help differential diagnosis and allow for more targeted therapy.[3]

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https://doi.org/10.21776/ub.hsj.2023.004.01.7
Received 9 October 2022; Received in revised form 30 November 2022; Accepted 15 December 2022
Available online 1 January 2023

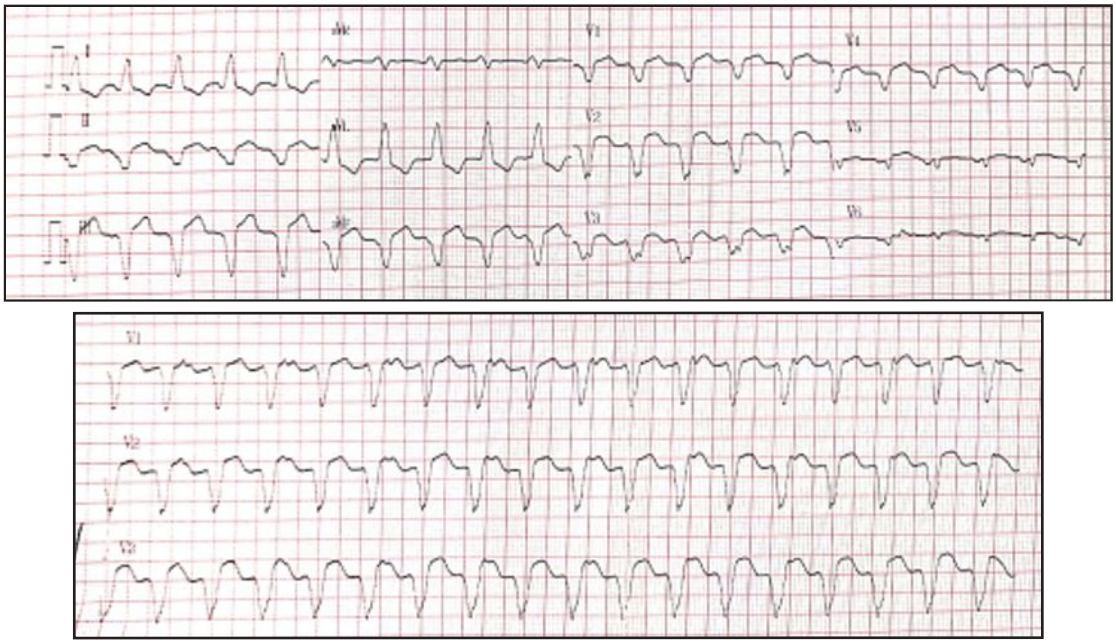

Figure 1. Electrocardiography at Emergency roomprocedure.

2. Case Illustration

In this case, we presented patient with Ventricular Tachycardia (VT) storm. A 63-year old woman was admitted to emergency department with chief complaint frequent episodes of palpitation. The patient's presenting vital signs were BP 90/50 mmHg on Dobutamine 12 mcg/kg/min and NE 1 mcg/kg/min; HR 120 bpm, RR 20 tpm and pulse oximetry 99% on nasal cannula 4 lpm. Patient's hemodynamic state was unstable with the initial rhythm upon emergency was noted to be monomorphic VT. Then she got cardioversion 100 Joule, followed by lidocaine drip and VT convert to sinus rhythm. Laboratory findings in emergency room were normal with kalium 4.07 mmol/L, bicarbonate 16 mmol/L, Magnesium 2.1 mg/dL, ureum 41.8 mg/dL, and creatinine 1.08 mg/dL. Cardiac enzyme also within normal limit. Chest radiograph showed cardiomegaly and no obvious acute cardiopulmonary process. Due to ongoing hemodynamic decompensation, patient admitted to intensive cardiovascular care unit. During hospitalization, we planned for staging PCI. She had history of PCI with implantation of 1 DES at mid to distal LAD and 1 DES at distal LCx. In this period of hospitalization the interventionist decided to PCI at proximal to distal RCA. She got complete revascularization for coronary artery. The Echocardiography showed dilatation of LA and LV, decrease of LVEF 38% by teich, diastolic dysfunction grade II, and global hypokinetic. After the condition stable, she was moved to ward and planned for discharge.

On the 7th day treatment, she had refractory VT and admitted to intensive cardiovascular care unit. In ICVCU, the patient's presenting vital signs were BP 70/40 mmHg on Dobutamine 5 mcg/kg/min; HR 140 bpm, RR 20 tpm and pulse oximetry 98% on nasal cannula 4 lpm. The episodic of VT occurred with cardiogenic shock (CS). She got cardioversion 100 Joule. The condition of VT still occurred three times and terminated after cardioversion then continue with drip Lidocaine 2 mg/minute. The ECG was converted to sinus rhythm. Laboratory test result indicated significant metabolic disorders including hypokalemia to 2.7 mmol/L and hypomagnesemia to 1.4 mg/dL. Aggressive intravenous repletion of potassium (50 mEq) to achieve target kalium 4.0 – 4.5 mmol/L and bolus MgSO4 to achieve target Magnesium 1.6 - 2.4 mmol/L.

After the condition stable, this patient was discharged and planned for ICD insertion at the next admission as a last resort. She got amiodarone 1x200 mg and Bisoprolol 1x2.5 mg as anti-arrhythmia drug

3. Discussion

Electrical storms continue to be linked to significant mortality and morbidity, as well as poor long-term consequences. The episodes that occur in certain individuals with shock-refractory VT/VF, which are typically seen during cardiopulmonary resuscitation, might be caused by a severe sort of electrical storm. Because the prevalence of electrical storms is not low (10–28 percent in patients with an ICD inserted for secondary prevention and 4% for primary prevention), it is critical to enhance the treatment strategy.[3] The most prevalent form of electrical storm is monomorphic VT storms linked with structural heart disease. The majority of monomorphic VTs are caused by reentry. Conduction and repolarization anomalies are found in scarred myocardium produced by diverse etiologies of fibrosis and collagen deposition. Focal mechanisms are normally critical in beginning reentry, but they can also help keep the VT going, especially in individuals with non-ischemic cardiomyopathy.[3,4] The most common symptom of an electrical storm is a persistent monomorphic VT that is linked to structural heart problems. Monomorphic VT is likely to develop when there is similar ventricular activation sequence without any change in the QRS complexes. The foremost reason of monomorphic VT events is reentry of electrical wavefront around a specific anatomic barrier, in particular scar tissue following MI. Wavefront reentry-induced monomorphic VT does not require active ischemia as a trigger, and it is unusual in individuals with an acute MI. The susceptible substrate for reentry in ischemic or nonischemic cardiomyopathy includes diverse regions of damaged myocardium. The heart experiences structural alterations after an acute MI or as nonischemic cardiomyopathy proceeds. Scarring occurs as a result of fibrosis, resulting in regions of conduction blockage. Bundles of myofibrils, on the other hand, can survive, especially towards the scar's edge. Slow conduction offers a channel for electrically stable reentry across these locations.[1]

Management of ventricular tachycardia storm approach can be done step by step. Step 1 management is stabilizing patient

hemodynamically. Regardless of the genesis of the electrical storm, it is routinely treated according to a treatment algorithm, which is commonly the Advanced Cardiovascular Life Support (ACLS) protocol. In critical patient care, when a rapid reaction to life-threatening circumstances is required, such a pre-specified algorithmic method is extremely successful.[3] Step 2 finding the triggers for electrical instability and try to reverse. Knowing and understanding basic knowledge of the arrhythmia mechanism and possible reversible etiologies is crucial in determine of electrical storm's management.[5] Step 3 is administering Antiarrhythmic drugs (AADs). Various classes of AADs are commonly used in setting of VT storm and for long term management to prevent recurrences.[6]

In our case, this patient admitted to emergency room with chief complaint frequent palpitation and the electrocardiography showed monomorphic ventricular tachycardia with unstable hemodynamic. We performed cardioversion 100 Joule, continued with lidocaine drip and VT convert to sinus rhythm. Based on ACLS Tachycardia algorithm, patient with initial electrocardiography monomorphic VT and unstable hemodynamic, the initial recommendation for wide regular tachycardia is synchronized cardioversion 100 joule. After that, we gave continuous lidocaine drip 2 mg/minute. Antiarrhythmic drug regimen should be considered in the acute phase to suppress further ventricular tachyarrhythmias.[7]

The fundamental management for VT storm can be classified into two modalities: pharmacological and non-pharmacological. The pharmacological treatment such as sympathetic blockade, antiarrhythmics, and combination therapy. Non-pharmacological treatment for example is catheter ablation, overdrive pacing, intraaortic balloon pump or extracorporeal life support, and heart transplant.[8] The objective in acute management for VT storm were patient's stabilization, prevention of the arrhythmic recurrences, sedation and containment of the adrenergic tone.[9] Intravenous amiodarone injection is considered the most effective first choice for pharmacological management.[10] Lidocaine is second choice drugs when there are contraindications for amiodarone and beta-blockers administration. In this case, lidocaine could be first choice of anti-arrhythmic drug because the patient had a history of long QT interval after amiodarone administered. Lidocaine is a Class IB antiarrhythmic agent that exerts its action by sodium channel blockade. Lidocaine preferentially works in ischemic myocardium.[9,11] In a use-dependent manner, lidocaine binds to fast sodium channels in which increase incident of ischemia VT when cellular circumstances such as a lower pH, a quicker stimulation rate, and a lower membrane potential are present.[12] Lidocaine, on the other hand, has only moderate antiarrhythmic capabilities outside of ischemia, with conversion rates from VT to sinus rhythm ranging from 8% to 30%. Only 12 percent of patients who were randomized to receive lidocaine survived until hospital admission in one research of 347 patients with out-of-hospital, shock-resistant VT or VF, compared to 23 percent of patients who were randomized to receive amiodarone.1 Lidocaine dose recommendation is bolus 1.0 to 1.5mg/kg IV, repeat dose 0.5 to 0.75 mg/kg IV with maximum dose 3 mg/kg.2 After an electrical storm has passed, the attention should move to maximizing heart failure treatment, potential revascularization, and preventing recurring ventricular arrhythmias.

Patient had history of PCI before. Cardiac catheterization denoted that Left Main was normal; stent DES patent at mid-distal LAD and distal LCx; and diffuse stenosis from proximal to mid RCA. Cardiac electrical storm might occur even after the percutaneous coronary intervention in the culprit lesion if there is also another significant stenosis. Hence, we recommend cardiac interventionists to perform percutaneous coronary intervention in other significant stenosis during the hospitalization to achieve complete revascularization and to prevent the arrhythmias.[13]

By interfering with the resting membrane potential, raising threshold potential, and causing occasional automaticity, hypokalemia can render arrhythmia event. Hypokalemia correction with potassium chloride stabilizes the cardiomyocyte membrane by generating a change in the resting membrane potential to a less negative value, bringing the threshold potential closer to normal. Hypokalemia affects cardiac myocytes by hyperpolarizing the membrane electrical potential, causing Na channels to open but not K inflow channels to close. Hypokalemia also reduces the activity of repolarization-related rapid-activating delayed-rectifier channels.[14,15]

The wide range of pathophysiological mechanisms that underpin ventricular tachycardias may explain some of the discrepancies found in the primarily short research on magnesium in monomorphic tachycardias. North found that magnesium successfully stopped ventricular tachycardia in one-third of the patients in a randomized, double-blind trial.[16] The metabolism of magnesium and potassium appears to be intertwined. Mg deficiency leaves the cell unable to hold the K difference between intra- and extracellular space, resulting in intracellular K depletion, according to human and animal studies. Because the operation of the Na/K pump is dependent on Mg, this occurrence happens. The Na/K pump's ineffective activity causes K depletion and intracellular Na buildup. Whang et al reported that hypokalemia could only be restored when the Mg deficiency was rectified in 46 hypokalemic individuals with hypomagnesemia. Every patient with uncontrollable hypokalemia should have their Mg levels measured, and the depleted quantity of Mg should be given. In patients with cardiac illness, convulsions, significant hypocalcemia, hypokalemia, or hypomagnesemia of less than 1.4 mg/dL, intravenous magnesium repletion is warranted. The intravenous mode of administration is used in the inpatient situation because it is extremely efficacious, cheap, and generally well tolerated. MgSO4.7H20 is the usual preparation. 8 to 16 mEq (1 to 2 gr) intravenously during a 2-4 minutes period may be given to individuals who are actively seizing or have cardiac arrhythmia.[17]

After the initial VES episode is under control, the need for an ICD implantation should be considered as soon as possible if the patient does not already have one. Current recommendations recommend an ICD implantation for secondary prevention if no reversible reasons can be found. Cardiac rehabilitation in the hospital and in outpatient care is suggested and has been proven to prevent VES recurrence.[11]

4. Conclusion

VT storm is more common in patients with dilated cardiomyopathy, either ischemic or idiopathic, as an evolution of the arrhythmic substrate of the pathology. Treatment options for VT storm and practical challenges faced in management of hemodynamically unstable VT storm. Initial management involves identifying and correcting the underlying ischemia, electrolyte imbalances, or other inciting factors.

5. Declarations

5.1. Ethics Approval and Consent to participate
This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.6. Authors contributions
Idea/concept: PL, SA. Design: PL, SA. Control/supervision: SA, MSR, AR. Literature search: PL, SA. Data extraction: PL, SA. Statistical analysis: PL, SA. Results interpretation: PL, SA. Critical review/discussion: SA, MSAR, AR. Writing the article: PL. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

5.7. Acknowledgements
We thank to Brawijaya Cardiovascular Research Center.

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