Case Report

Management of Ventricular Storm with Thoracic Epidural Anesthesia

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Abstract

The incidence of recurrent ventricular arrhythmias is increasing these days. Ventricular electrical storm can be of three types as follows: monomorphic ventricular tachycardia (VT), polymorphic VT, and ventricular fibrillation. The mechanism of ventricular storm is complex, and its management is quite a challenge for the clinicians due to its life-threatening consequences. We report a case of ventricular storm in whom all the conventional methods for the management of arrhythmias were ineffective, and the case is managed effectively with thoracic epidural anesthesia (TEA). A 60-year-old male patient was admitted to recurrent ventricular arrhythmias. He received defibrillator shocks and other antiarrhythmic drugs, but he was not responding to the treatment. We managed to revert the ventricular arrhythmias to the sinus rhythm with TEA. Ventricular storm is a challenging complication, which can be managed effectively with timely diagnosis and effective management.

Keywords: *Epidural anesthesia, monomorphic VT, ventricular storm*

Introduction

The term electrical storm was introduced in 1990s. It is defined as the occurrence of two or more hemodynamically unstable ventricular tachycardia (VT) and/or ventricular fibrillation (VF) in 24 h, requiring electrical cardioversion or defibrillation.[1] However, nowadays with the widespread use of implantable cardioverter defibrillators (ICDs), the most widely accepted definition of ventricular electrical storm in clinical practice is "occurrence of ≥2 separate VT/VF episodes or ≥3 appropriate ICD therapies for VT/VF in a 24 h period."[2] Incidence is about 10%–20% in patients who have an ICD for secondary prevention of sudden cardiac death[3] and lower when ICDs are placed for primary prevention.[4] In the MADIT II study, 4% of patients developed electrical storm on an average of 20.6 months.[5] The etiology of ventricular electrical storm is fairly broad. Potential causes include enhanced sympathetic tone, ischemic heart disease, electrolyte imbalance (potassium and magnesium), genetic abnormalities (such as Brugada syndrome and long QT syndrome), iatrogenic (often in the presence of ICDs), and endocrine disorders (thyroid disorders and pheochromocytoma).[6] The mechanisms of electrical storm are quite complex and not well understood. It has been postulated that cellular and molecular alterations can increase intracellular calcium overload and changes of action potential duration and morphology that lead to the onset of electrical storm.[7] Determining the cause and good knowledge of mechanism are essential for effective management of electrical storm because treatment must target the underlying mechanism. We report a case of a patient who was referred to our hospital for the management of ventricular storm.

Case Report

The patient was a 71-year-old man who got admitted to private clinic with the complaints of chest pain and breathlessness. There he was diagnosed with VT and got reversed with direct current (DC) shock. However, he presented with multiple episodes of VT after that he required DC shocks to revert it to sinus rhythm. Hence, he was transferred to our hospital for further management of ventricular storm. VT was with pulse. Over 48 h duration, he had received around 100 DC shocks, and every time biphasic defibrillation was done with DC shock of 200 J. Meanwhile, he became hemodynamically unstable so got intubated, and mechanical ventilation was started. When we saw the patient, he was receiving injection amiodarone, injection heparin, injection

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fentanyl, injection midazolam, and injection vecuronium. His pulse rate was 210/min and BP was 190/130 mmHg. On evaluation, 12 lead electrocardiogram (ECG) showed monomorphic VT [Figure 1]. Complete blood count was normal; however, serum biochemistry revealed deranged electrolyte, liver enzymes, and creatinine (Sr K^+ – 6 mEq/L, SGOT/SGPT – 106/126 IU/L, Creatinine – 1.7 mg/dl, and BUN – 38 mEq/L). Rest of the biochemical profile including Sr Na^+, Ca^{++}, and Mg^{++} were normal. Serum potassium was corrected with intravenous potassium supplementation. His two-dimensional echocardiography (2D ECHO) showed ischemic heart disease with global left ventricular (LV) hypokinesia, mild mitral regurgitation, no pulmonary artery hypertension, E/o LV diastolic dysfunction and severely compromised LV systolic function, and ejection fraction (EF) 15%–20%. Even though 2D ECHO was suggestive of global hypokinesia with reduced EF (15%–20%), doing coronary angiography was not possible due to persistent VT, and hence, coronary ischemia could not be ruled out. Electrocardiographic differentiation of VT from supra VT (SVT) with aberrancy is difficult. It is important to differentiate SVT with aberrancy from VT because SVTs usually respond well to atrioventricular (AV) nodal blocking drugs whereas patients with VT may precipitate cardiac arrest.[8] In our case, ECG was showing very broad complexes with AV dissociation and absence of typical right bundle branch block or left bundle branch block, so SVT was ruled out. Furthermore, QT interval was normal when rhythm reverted to sinus [Figure 2]. Hence, we ruled out prolonged QT syndrome. In view of refractory, VT and unavailability of catheter ablation facility at our center, we decided to manage this case with thoracic epidural catheter. The patient was receiving unfractionated heparin 5000 IU thrice a day. According to ASRA guidelines, before placing epidural catheter, we checked activated partial thromboplastin time levels which were normal (patient value – 27.8 s and control – 30.3 s). We placed epidural catheter 6 h after the last dose of heparin and next dose was planned >1 h after placing. Under all aseptic precautions, with the patient in left lateral position, an 18 G Tuohy epidural needle was inserted into T1–T2 interspace using a standard loss of resistance technique through a median approach. After confirming negative aspiration for cerebrospinal fluid (CSF) and blood epidural catheter was advanced 5 cm beyond the needle tip into the epidural space and secured. Negative aspiration of CSF and blood was performed to rule out inadvertent intrathecal and intravascular placement. Initially, 1 ml of 0.25% bupivacaine with 1 ml of 2% lignocaine was injected epidurally, followed by continuous infusion of 0.25% bupivacaine @ 2 ml/h. Epidural catheter was in place for 7 days. Patient experienced two episodes of VT during those 7 days which were reverted to sinus rhythm with DC shocks. However, after removal of epidural catheter, he sustained VT and succumbed to death after 24 h.

Discussion

Ventricular storm is an emergent life-threatening clinical condition. The physical and emotional distress that the patient experience due to recurrent VT and DC shocks may increase the sympathetic tone and facilitate further arrhythmias.[9] The treatment can be broadly classified into pharmacological and nonpharmacological. Pharmacological treatment includes drugs such as Beta-blockers, amiodarone, dofetilide, and azimilide. Antiarrhythmic drugs reduce the number of ICD shocks, but they are associated with a relatively high incidence of side effects.[10] Thus, the decision to prescribe antiarrhythmic drugs in such patients should be individualized. When conventional pharmacotherapy fails, nonpharmacological modalities should be used, such as catheter ablation, pacing, sympathetic blockade by thoracic epidural anesthesia (TEA) and left stellate ganglion block, and heart transplant. Multiple reports have described successful ablation in patients with drug-refractory electrical storm.[11,12] In our case, automated ICD, radiofrequency ablation was kept in mind, but due to unavailability nothing could be done.

The autonomic nervous system continuously receives input from the heart, integrates them, and sends efferent signals to maintain cardiac function and arrhythmogenesis. Bourke *et al*. have studied the efficacy of both left cardiac sympathetic denervation and TEA in the management

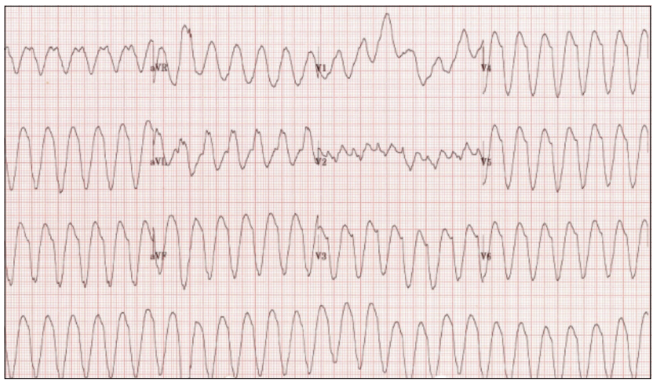

Figure 1: Preprocedure electrocardiogram showing ventricular tachycardia

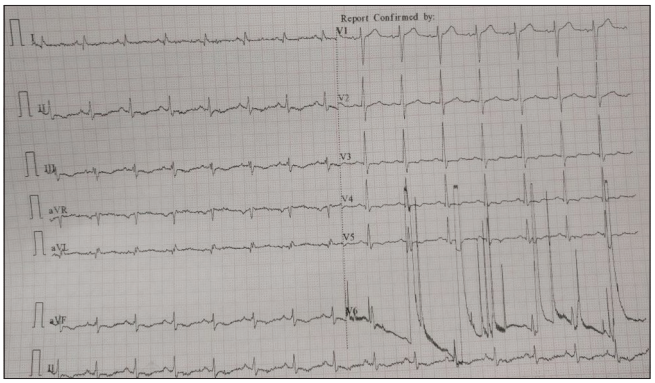

Figure 2: Postprocedure electrocardiogram showing normal QT interval after reverting to sinus rhythm

of electrical storms.[13] TEA was well tolerated by all the patients in their series, and 6/8 patients had a >80% reduction in arrhythmia after TEA. No adverse effects were reported. Stellate ganglion block provides transient abolition of ventricular storm, which can be utilized as the therapeutic test for sympathetic denervation and later on cardiac sympathetic denervation. Due to unavailability of ultrasonography machine at our set up, we opted for thoracic epidural catheter.

TEA involves the application of local anesthetic directly onto the sympathetic chain which results in almost immediate sympatholysis. Furthermore, the effects of TEA on hemodynamic parameters including heart rate, mean arterial pressure, cardiac index, and central venous pressure are minimal.[14]

Our case report highlights the value of TEA in the management of ventricular storm and also its added advantage in conjunction with other pharmacological agents. After starting epidural infusion, the frequency of VT has significantly reduced in our patient with stable vital parameters.[13] We removed the epidural catheter after 7 days of placement because of the chances of complications of catheter such as kinking, migration in subarachnoid space, and breakage. Radiofrequency catheter ablation would have been the best possible intervention in this patient and would have had a high possibility of arrhythmia control. Unfortunately, because of nonavailability of the facility of catheter ablation in our hospital, this patient died a day after removal of epidural catheter.

Conclusion

Sympathetic hyperactivity is an important modulator of ventricular storm; therefore, neuraxial modulation with TEA is an attractive option for arrhythmia management. This treatment modality can be considered when standard treatments fail; this may be used as a bridge to cardiac surgery or catheter ablation procedures. Large prospective randomized studies are needed to further define the clinical role of TEA or other neuroaxial methods in the future.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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