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RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CASE REPORT: CLINICAL CASE

Ketamine Terminating Persistent Ventricular Tachycardia

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ABSTRACT

Ventricular tachycardia (VT) is an arrhythmia associated with sudden cardiac death. VT storm is a complication of persistent VT requiring immediate antiarrhythmic therapy. In refractory cases, adjunctive therapy includes sedation/mechanical ventilation or catheter ablation. This case highlights a patient with ischemic cardiomyopathy in refractory VT storm terminated by administration of ketamine. (JACC Case Rep 2024;29:102466) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Ventricular tachycardia (VT) is a wide complex tachycardia lasting ≥3 consecutive beats at a rate of more than 100 beats/min, arising from the ventricle.[1] Based on the duration, it is classified as sustained or nonsustained. Sustained VT lasts more than 30 seconds or requires intervention within 30 seconds due to hemodynamic instability, whereas nonsustained VT lasts <30 seconds without hemodynamic compromise.[1] Electrical storm (ES) is a life-threatening syndrome that involves recurrent episodes of sustained ventricular arrhythmias. If the patient has an implantable cardioverter-defibrillator, ES is defined as ≥3 separate episodes of VT or ventricular fibrillation leading to implantable cardioverter-defibrillator therapies within 24 hours via antitachycardia pacing (ATP) or cardioversion/defibrillation, thus excluding arrhythmias under the device detection threshold or that spontaneously terminated.[2] Early evaluation and management are crucial in preventing ES-induced cardiogenic shock. Triggers for ES are usually unidentifiable, and in 1 trial, a precipitating cause could be identified in only 13% of electrical storm cases.[3] Antiarrhythmic drugs (AADs) including class 1 (sodium channel blockers), class 2 (beta-blockers), and class 3 (potassium channel blockers) are used frequently in ES, whereas Class 4 AADs (calcium channel blockers) are typically not used in ES.[2] Amiodarone, lidocaine, and nonselective beta-blockers are first-line AADs for ES. Second-line therapy includes neuraxial modulation for intractable ES refractory to drug treatment, which is achieved by deep sedation.[4] Catheter ablation can also be considered when AADs insufficiently suppress ES.

LEARNING OBJECTIVES

- To understand the adjunctive therapies available for management of refractory VT and VT storm.
- To consider the potential antiarrhythmic effects of ketamine.

Ketamine, a phencyclidine derivative, possesses analgesic and anesthetic properties. It produces a state of altered consciousness while preserving hemodynamic stability with airway tone and respiration.[5] Ketamine's effects are dose-dependent. At lower doses (<0.5 mg/kg IV), it acts as an analgesic;

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ABBREVIATIONS AND ACRONYMS

AAD = antiarrhythmic drug

ATP = antitachycardia pacing

ES = electrical storm

VT = ventricular tachycardia

but at higher doses (1-4.5 mg/kg IV), it exhibits both analgesic and anesthetic effects. Ketamine causes mild stimulation of the cardiovascular system via increased sympathetic activity.[6] Despite its excitatory cardiac effects, significant ischemia is not seen with anesthetic use for sedation.[6] There are no previously documented clinical cases of ketamine disrupting an arrhythmia, and previous animal research on rabbits revealed that ketamine can induce a concentration-dependent lengthening of the R-R interval.[7]

HISTORY OF PRESENTATION

We present a case of a 62-year-old African-American man who presented for evaluation of palpitations and abdominal pain. Despite being tachycardic with beats per minute in the 130s on presentation, the patient denied any chest pain, dyspnea, or dizziness. On presentation, he was found to have a wide complex tachycardia with a heart rate of 130 beats/min but was otherwise hemodynamically stable (**Figure 1**). All labs, other than a high-sensitivity troponin level of 38/46/47 ng/L, were within normal limits.

PAST MEDICAL HISTORY

The patient had a past medical history of ischemic cardiomyopathy secondary to chronic total occlusion of the left anterior descending coronary artery, heart failure with reduced ejection fraction, American College of Cardiology/American Heart Association stage C, NYHA functional class III symptoms (ejection fraction of 30%), a St. Jude dual chamber implantable cardioverter-defibrillator (St. Jude Medical), VT with 3 endocardial ablations in the previous 6 months, hypertension, catheter-associated right upper deep vein thrombosis, and hypothyroidism. Medications before admission included amiodarone 200 mg twice daily, metoprolol succinate 25 mg daily, mexiletine 200 mg 3 times daily, sacubitril-valsartan 24 to 26 mg twice daily, spironolactone 12.5 mg daily, apixaban 2.5 mg twice daily, atorvastatin 40 mg daily, clopidogrel 75 mg daily, dapagliflozin 10 mg daily, and levothyroxine 50 μg daily.

DIFFERENTIAL DIAGNOSIS

Our differential diagnosis included acute coronary syndrome, VT, ventricular fibrillation, an acute electrolyte abnormality, hypoxia, ischemic cardiomyopathy, mesenteric ischemia, abdominal aortic aneurysm, and peptic ulcer disease.

INVESTIGATIONS

Initial electrocardiogram demonstrated VT without ST-segment elevation or depression. Chest x-ray did not demonstrate any acute abnormalities. Cardiology was consulted, and device interrogation did not reveal any defibrillations since his first ablation 6 months ago, however it did note multiple episodes of VT requiring ATP therapy.

MANAGEMENT

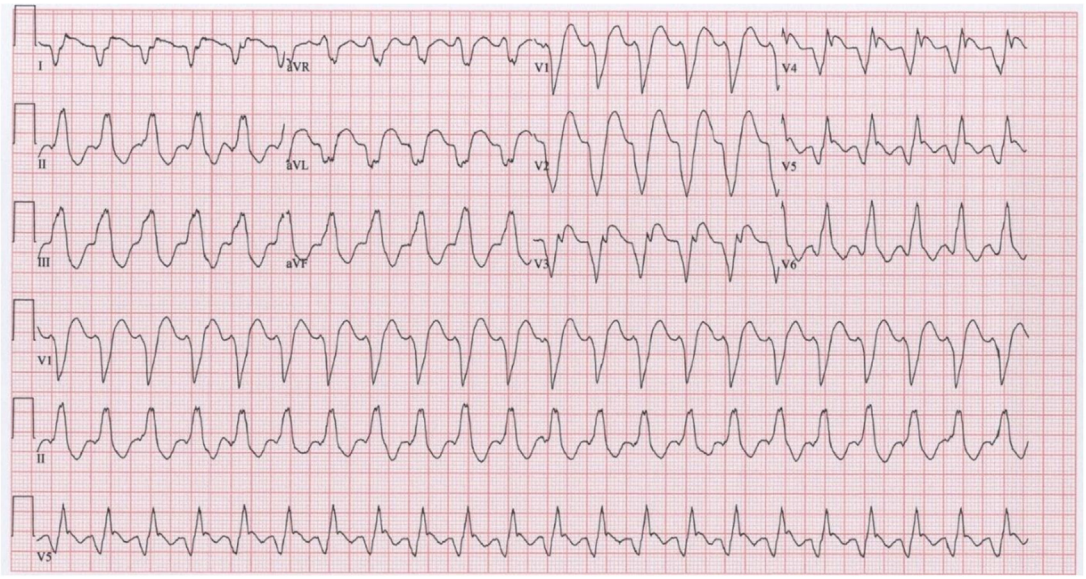
An amiodarone bolus in the emergency department converted the patient to a paced rhythm (pulse of 60 seconds) for a short period of time; however, he spontaneously converted back to sustained VT with a pulse of 130s. Amiodarone infusion was continued, and he was admitted to the intensive care unit. Cardiology recommended adding a lidocaine infusion (4 mg/min) to the amiodarone infusion for his persistent VT. With the addition of lidocaine, the patient experienced multiple episodes of spontaneous conversion of monomorphic VT to paced rhythm and back. A trial of esmolol failed to provide adequate control. Day 2 of hospitalization was complicated by an episode of unresponsiveness and seizure-like activity. The patient experienced myoclonus of the jaw and trauma to the tongue. Lidocaine was stopped, and lidocaine levels were ordered. Stopping lidocaine led to sustained monomorphic VT. At this time, the patient became hemodynamically unstable with failure of cardiac resynchronization therapy-defibrillator to perform successful ATP. The patient met criteria for VT storm. Cardiology recommended intubation and deep sedation to reduce sympathetic tone. Ketamine was selected for induction. The patient was successfully intubated with a single attempt via drug-assisted intubation with ketamine. A ketamine bolus of 90 mg (~1 mg/kg) was administered with immediate termination of his VT.

The patient was subsequently transferred to a tertiary care facility for higher level of care. Unfortunately, the day after transfer the patient experienced cardiac arrest requiring multiple rounds of cardiopulmonary resuscitation and multiple defibrillations for pulseless VT. A VT ablation on extracorporeal membrane oxygenation was performed and successful the following day. Heart transplant evaluation was initiated; however, the patient ultimately refused heart transplantation due to personal beliefs.

DISCUSSION

This patient presented with hemodynamically stable monomorphic VT that developed quickly into VT

FIGURE 1 Electrocardiogram of Ventricular Tachycardia on Admission



storm. The biggest risk factors for this patient were ischemic cardiomyopathy, a history of multiple VT ablations, heart failure with reduced ejection fraction, and medication noncompliance.[8] The next steps in treatment of VT storm refractory to AADs (amiodarone, lidocaine, and esmolol) include deep sedation with propofol, stellate ganglion blockade, and VT ablation. Although multiple publications have provided evidence for VT termination using deep sedation with propofol, to our best knowledge, there is no literature describing ketamine as the agent for deep sedation. A single bolus of ketamine delivered during induction for intubation immediately converted the patient's VT to a paced rhythm. We cannot definitively say that the ketamine was solely responsible for converting the patient's hemodynamically unstable ES to a stable rhythm, or if it was the cumulative effect of multiple AADs with the ketamine. However, there is some literature describing the antiarrhythmic properties of ketamine in animal models. One current ongoing trial is evaluating the neurological functioning of patients who received ketamine during cardiac arrest treatment (NCT04360070). Because there has been very limited research studying the antiarrhythmogenic effects of ketamine in humans, additional human studies need to be performed to analyze ketamine's effect on the cardiac conduction system.

FOLLOW-UP

At 6 months postablation, the patient was able to live alone, optimize his medical regimen, and continue to perform all independent activities of daily living.

CONCLUSIONS

This patient presents as a case that did not respond to typical therapy for VT storm. He therefore required intubation, and during the induction dose of ketamine, VT was terminated. Ketamine has known antiarrhythmic effects in murine models. However, further investigation is needed to clarify these effects in humans.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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