Refractory ventricular tachycardia: a challenge in an emergency setting. A case report

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Abstract

The incidence of electrical storms or Ventricular Tachycardia (VT) storms is increasing due to the growing use of Implantable Cardioverter Defibrillators (ICDs), higher rates of ischemic heart disease, non-compliance with anti-arrhythmic medications, and misuse of other drugs. Timely recognition, accurate rhythm identification, and prompt intervention are critical in saving lives. We present a case of a middle-aged male with a prior diagnosis of idiopathic VT who arrived at the Emergency Department (ED) with palpitations. During his ED stay, he experienced refractory VT, which was managed through anti-arrhythmic medications and multiple synchronized cardioversions. Further investigation led to a diagnosis of cardiac sarcoidosis, and he was successfully treated.

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Introduction

Ventricular Tachycardia (VT) is a potentially life-threatening arrhythmia originating in the ventricles, defined by three or more consecutive beats at a rate exceeding 100 beats per minute.[1] Ventricular arrhythmias occurring three or more times within 24 hours (separated by at least 5 minutes), each requiring termination by intervention, are classified as electrical storms.[2] If left untreated or if the underlying cause remains unidentified, VT carries a poor prognosis. Common symptoms include palpitations, dizziness, lightheadedness, exercise intolerance, syncope, or sudden cardiac arrest.[3] Working on a sympathetic drive, refractory VT may be triggered by ischemic heart disease, electrolyte imbalances, medication misuse, drug toxicity, non-adherence to therapy, substance abuse, thyrotoxicosis, sepsis, myocarditis, and infiltrative cardiomyopathies like cardiac sarcoidosis or amyloidosis.[4]

We report a case involving a middle-aged male with a prior idiopathic VT diagnosis who presented with refractory VT and was later diagnosed with cardiac sarcoidosis.

Case Report

A male in his 40s, employed in the Information Technology sector, presented to the ED with palpitations persisting for two days. He was alert and oriented, with no notable family or substance use history. Ten years earlier, he had been diagnosed with idiopathic VT and treated with radiofrequency ablation and anti-arrhythmic drugs, but had recently become non-compliant with his medication.

Upon presentation, his vital signs were stable: temperature normal, blood pressure 110/70 mmHg, respiratory rate 20/min, and oxygen saturation 98% on room air. An Electrocardiogram (ECG) revealed Right Ventricular Outflow Tract (RVOT) VT with a rate of 190 bpm (Figure 1). Systemic examinations were unremarkable.

A vagal manoeuvre was attempted without success. Subsequently, 6 mg of intravenous adenosine was administered, which converted the rhythm to non-sustained VT with Ventricular Premature Complexes (VPCs)(Figure 2) and a blood pressure drop to 90/60 mmHg. A 2D echocardiogram showed a Left Ventricular Ejection Fraction (LVEF) of 55%, a dilated left ventricle, and an Inferior Vena Cava (IVC) diameter of 21 mm with less than 50%

collapsibility. Troponin I and BNP levels were elevated - 0.05 ng/ml and 468 pg/ml, respectively (normal values – 0.02 ng/ml and 100 pg/ml respectively), while other laboratory tests, including electrolytes, were normal.

Fifteen minutes later, he developed monomorphic VT (Figure 3) with hypotension (80/50 mmHg), prompting immediate synchronized cardioversion with 100 joules, after which he reverted to sinus rhythm. Vasopressor support was initiated, but recurrent VT episodes required additional cardioversions.

In consultation with cardiology, the patient received a loading dose of 300 mg IV Amiodarone, followed by an infusion of 1 mg/min for six hours and then 0.5 mg/min for the following hours in the ED.[5] His rhythm changed to ventricular trigeminy (Figure 4), and he stabilized. After a 13-hour ICU stay, he was transferred to the cardiology department. Further workup, including cardiac MRI, FDG-PET CT, and endomyocardial biopsy, confirmed cardiac sarcoidosis. He was treated with oral prednisone and amiodarone, remained stable, and was discharged on day 14. A summary of his hospital course is provided in Figure 5.

Discussion

Idiopathic VT occurs in structurally normal hearts and generally carries a better prognosis than VT associated with structural abnormalities. However, it may still cause significant morbidity and mortality. Idiopathic VTs often originate from the outflow tracts, mitral/tricuspid annuli, or left bundle branch fascicles. Outflow tract VTs, especially those originating from the RVOT, are most common and typically affect young individuals.[6] RVOT VT usually presents with a Left Bundle Branch Block (LBBB) morphology and an inferior axis on ECG (as seen in our first ECG), where the anterior site shows a dominant Q-wave or a qR complex in lead I and a QS complex in aVL. Pacing at the posterior sites produce a dominant R-wave in lead I, QS or R-wave in aVL and an early precordial transition (R/S = 1 by V3).[7] Cyclic AMP-mediated, calcium-dependent triggered activity is believed to be the mechanism.[8] These VTs usually respond to adenosine,[9] although beta-blockers and calcium channel blockers are more commonly

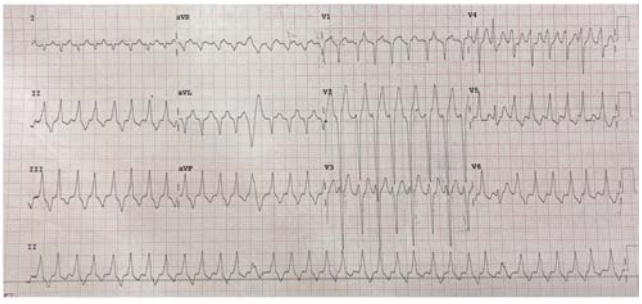

Figure 1. Representation of right ventricular outflow tract ventricular tachycardia as shown in his initial ECG.

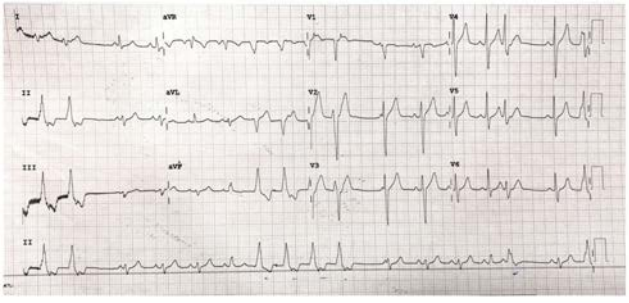

Figure 2. Non sustained ventricular tachycardia with ventricular premature complexes.

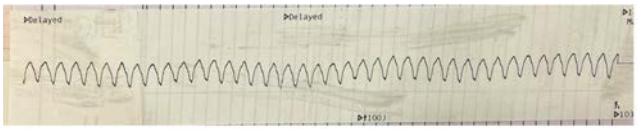

Figure 3. Monomorphic ventricular tachycardia.

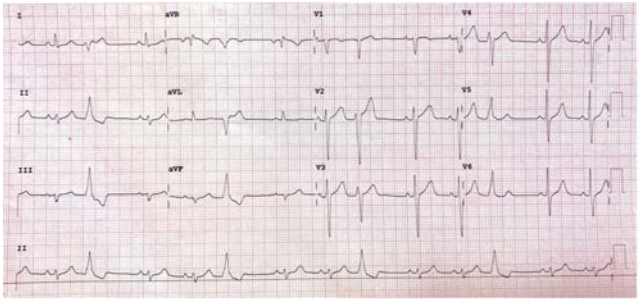

Figure 4. Ventricular trigeminy.

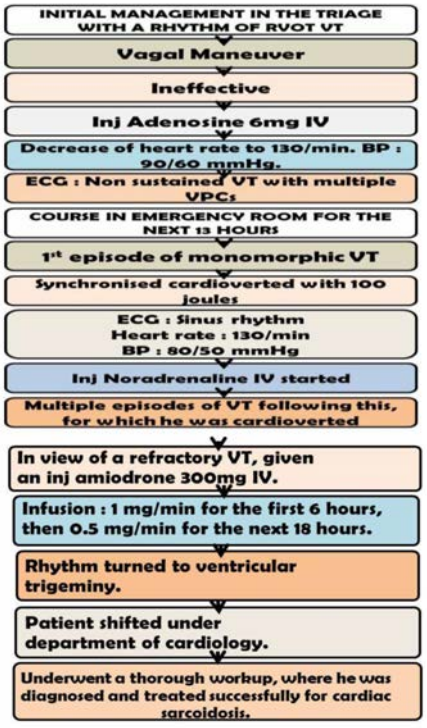

Figure 5. Summary of management in the Emergency Department.

used to prevent recurrence.[10] Hemodynamically unstable cases require immediate synchronized cardioversion. Radiofrequency ablation has shown to give the best outcomes, with or without a combination of calcium channel blockers.[11] RVOT tachycardia can be difficult to distinguish from SVT with LBBB, but they might be differentiated by factors including AV dissociation, concordance, duration of the initial part of the QRS complex, and axis. Adenosine, an endogenous nucleoside, slows AV nodal conduction and is effective for terminating most AV node-dependent Supraventricular Tachycardias (SVTs). However, it is less effective for arrhythmias not involving the AV node. It has a short half-life (0.6–10 seconds) and a favourable safety profile.[12]

VT mechanisms include reentry, enhanced automaticity, and triggered activity. Management of unstable refractory VT includes synchronized cardioversion and anti-arrhythmic agents such as amiodarone, lidocaine, procainamide, sotalol, and magnesium.[5] Amiodarone, a class III anti-arrhythmic, blocks potassium channels, prolonging cardiac repolarization. Despite its long half-life and risk of side effects (e.g., bradycardia, hypotension, torsades de pointes), it remains a mainstay in VT management.[13]

Synchronized cardioversion delivers timed electrical shocks to restore normal cardiac rhythm in patients with life-threatening or unstable tachycardic arrhythmias and a pulse, distinguishing it from defibrillation. It has been shown to be the most effective resuscitation measure for many arrhythmias.[14]

Cardiac Sarcoidosis (CS) is an infiltrative cardiomyopathy characterized by granulomatous inflammation, often leading to VT through reentry circuits or abnormal automaticity. CS commonly presents with conduction abnormalities, heart failure, or sudden cardiac death.[15] LVEF is the key prognostic factor. Triggers of CS include environmental, infectious, genetic, and immune factors.[16] Treatments include corticosteroids, methotrexate, and anti-arrhythmic agents. Patients may require implantable cardioverter defibrillators for arrhythmia prevention (including those with atrial fibrillation) or permanent pacing. Other immunosuppressants like azathioprine, cyclophosphamide, leflunomide, adalimumab, infliximab, and rituximab are considered in refractory cases.[17]

This case highlights the critical need for ED physicians to identify and manage life-threatening arrhythmias like VT rapidly. Given the rarity and complexity of refractory VT, early stabilization and cardiology involvement are essential for successful outcomes.

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