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Recurrent Ventricular Tachycardia due to Multiple Etiologies

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Abstract

Background: Ventricular tachycardia (VT) is a life threatening arrhythmia in which an identification of the correct cause and its timely treatment is vital for the patient. *Case Report*: We present a case report of a 66 year man with recurrent episodes of sustained and non-sustained ventricular tachycardia (VT) confirmed through telemetry on a background of excessive ventricular ectopic activity. Surprisingly, he had different triggers for his ventricular tachycardia (VT) on each presentation to the hospital which settled with anti-arrhythmic drugs and treating the underlying cause. *Conclusion*: The reason for reporting this case is to highlight rare triggers of VT (a life threatening arrhythmia) and that it is important to keep on looking for other aetiologies especially if VT recurs after satisfactory treatment of an earlier cause as in this case.

Keywords: Anti-Arrhythmia Agents, Telemetry, Ventricular Tachycardia, Ventricular Premature Complexes.

Introduction

Ventricular tachycardia (VT) is a life threatening/malignant arrhythmia with a myriad of underlying etiologies. Overall VT accounts for 5.6% of all mortality. However in patients with ischemic cardiomyopathy and non-sustained VT, sudden death mortality approaches up to 30% in two years. Morbidity from VT is associated with hemodynamic collapse. In this fatal arrhythmia an identification of the correct cause and its timely treatment is vital in the longer term management and prognosis of patients with this condition.

Case Report

A 66 year old retired gentleman had recently completed a course of neo-adjuvant chemo-radiotherapy with capecitabine for his colorectal cancer a month prior to his first presentation. He was a non-smoker and consumed about 15 units of alcohol (red wine) per week. He also had a long-standing history of hay fever. He did not take any regular medications and had no known drug allergies. Two years prior to his first presentation, he was seen by cardiology for intermittent palpitations. Exercise tolerance test and echocardiogram were normal. 24 hour Holter ECG, apart from excessive ventricular ectopic activity of around 11% of total duration of recording, did not show any other sustained arrhythmia at that point. A cardiac MRI performed to investigate for any structural heart disease in view of excessive ventricular ectopic activity was normal.

First Presentation:

His first presentation was through emergency department with one week history of diarrhea and two days' history of palpitations and dizziness. On examination, he appeared alert but mildly dehydrated. His pulse was irregular with HR of 70-120 bpm, blood pressure (BP) was 135/75 and rest of cardiovascular examination was unremarkable. His routine bloods (including serum electrolytes, renal functions, thyroid function tests) and urinary catecholamine levels were within normal range. His 12 lead ECG **[Fig.1]** showed unifocal ventricular ectopics with 3-6 beat runs of non-sustained

ventricular tachycardia. We then requested serum calcium, magnesium and phosphate levels and this revealed severe hypophosphatemia with serum phosphate levels of <0.21 mmol/L (reference range 0.74-1.5 mmol/L) and mildly raised Troponin I of 0.06 ng/mL (cut-off <0.01 ng/mL). His inpatient echocardiogram again revealed normal biventricular size and function. In view of his mildly raised Troponin I and ventricular arrhythmia a coronary angiogram was performed to exclude any underlying coronary artery disease which was reassuringly normal.

He was diagnosed as VT secondary to hypo-phosphatemia. He was treated with intravenous (IV) amiodarone and IV phosphate infusions. Within 12 hours of starting the regimen his VT episodes settled. An exercise tolerance test to look for any further stress induced arrhythmias was normal. His palpitations and dizziness had significantly improved and he was discharged home on sotalol 80 mg BD. A month following discharge from the hospital he underwent uncomplicated elective anterior resection for his colorectal cancer with loop ileostomy.

Second Presentation:

He had a routine review in cardiology clinic two months following his discharge from hospital. He mentioned further symptoms of palpitations and was re-admitted to CCU for observation and investigations to rule out any sustained ventricular arrhythmia. His physical examination was unremarkable and all bloods including phosphate were within normal limits. ECG **[Fig.2]** showed unifocal interpolated VE's in trigeminal pattern with normal QTc. Whilst an inpatient he had further episode of sustained VT and given his recurrent VT, sotalol was switched to amiodarone. He was given a loading dose of IV amiodarone followed by oral amiodarone in reducing regimen with long term maintenance at 200 mg once daily. His palpitations improved, however, on that admission the cause for his ventricular arrhythmia remained unclear.

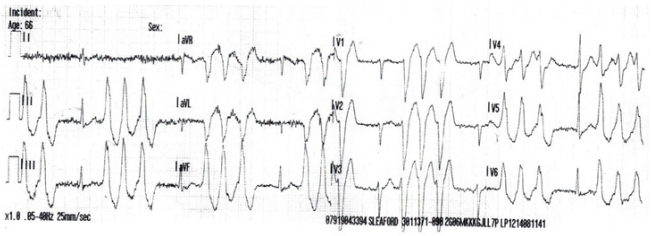

Fig.1: *ECG on first admission. Triplets (non-sustained VT) secondary to hypophosphatemia due to malabsorption.*

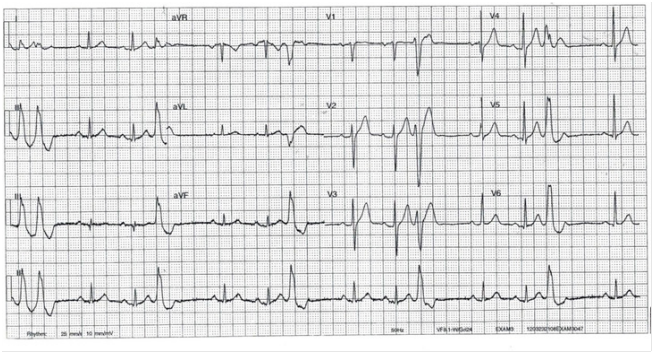

Fig.2: *ECG on second admission. ECG showed trigiminy pattern. No obvious cause of his VT was found on this admission.*

He was discharged home and a repeat outpatient 24 hour tape was reassuringly normal without any ventricular ectopic activity. On further outpatient follow up, the patient remained stable and decided to come off amiodarone due issues of long term side-effects.

Third Presentation:

Three month following cessation of amiodarone patient was readmitted with diarrhoea and weight loss for 4 weeks and palpitations for one week. His ECG **[Fig.3]** was again suggestive of frequent triplets and non-sustained VT. On blood tests this time he was found to have hyperthyroidism with very low TSH of 0.01 mU/L, high T4 at 35.5 pmol/L and T3 at 9.73 pmol/L. He had mild hypo-phosphatemia with a level of 0.55 mmol/L. The thyroid peroxidise antibodies were negative but ultrasound scan was suggestive of thyroiditis. He was treated as non-sustained VT secondary to hyperthyroidism/thyroiditis. This was exacerbated by co-existing mild hypo-phosphatemia due to

malabsorption secondary to his bowel malignancy. His arrhythmia settled with bisoprolol 2.5 mg once daily which was up-titrated to 5 mg OD before discharge and was also commenced on carbimazole for thyrotoxicosis. His hypo-phosphatemia was initially corrected with IV phosphate and then maintenance with oral phosphate replacement. He made a good recovery and was discharged home with further cardiology and endocrinology reviews as outpatients. His final diagnosis from endocrine team was primary thyrotoxicosis which eventually settled with carbimazole. Since amiodarone was stopped at least 3 months prior to developing symptoms of thyrotoxicosis, it was not thought to be directly contributing to this. However some studies have shown that its side effects can manifest several months after stopping it.

Patient had further one admission with chest pain and this time investigations were not suggestive of any arrhythmia or other cardiac event and his subsequent outpatient dobutamine stress study confirmed absence of stress related arrhythmia and ischemia. Regarding the excessive ventricular ectopic activity it was suggested that the gentleman routinely suffers from hay fever (which can lead to systemic histamine release). This combined with red wine that also contains histamine compounds could be a trigger of his frequent ventricular extra systoles. He is now on regular antihistamine medication and has not had any further VT episodes since.

Discussion

Ventricular tachycardia (VT) is responsible for most of the sudden cardiac deaths. VT refers to any rhythm faster than 100 or more beats/min, with three or more successive irregular beats, arising distal to the bundle of His. The rhythm could be generated by the ventricular myocardium, the distal conduction system, or both. Common symptoms of VT are palpitations, light-headedness, syncope or chest pain. However, patient can be asymptomatic or the symptoms may be related to

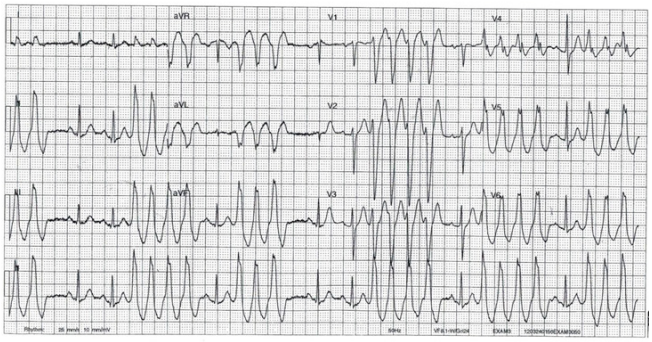

Fig.3: *Non-sustained VT secondary to thyrotoxicosis, further exacerbated by mild hypo-phosphatemia.*

associated therapy like an implantable cardioverter-defibrillator [ICD] shock. Electrocardiography (ECG) is the standard test for the diagnosis of VT, However in hemodynamically unstable patients the diagnosis is made and ECG rhythm strip if available. Broadly speaking, the causes of VT can be (i) structural for example old MI or non-MI scar [1,2], ischemia/ACS [3], cardiomyopathies/ARVC [4], heart failure [5.6], (ii) electrical: long QT/ short QT/ Brugada syndromes, RVOT- VT (inferior axis and LBBB), catecholaminergic polymorphic VT (CPVT) [7,8]. (iii) extra-cardiac like electrolyte abnormalities (including hypo-magnesaemia and hypo-phosphatemia) [9-11], endocrine disorders especially thyrotoxicosis [12-14], histamine compounds [15,16], and various drugs [17,18]. Amiodarone in some cases can cause delayed thyroiditis leading to thyrotoxicosis [19]. In terms of treatment this could be medical or interventional. In medical treatment, anti-arrhythmic medications like amiodarone are useful option for treating VT [20-22]. However, amiodarone should not be used for VT related to prolonged QT interval [23]. Beta-blockers like bisoprolol as in this case are useful in suppressing ventricular arrhythmic ectopic activity and also providing symptomatic relief [24-26]. Sotalol is another option which apart from beta-blocker activity also acts as an anti-arrhythmic agent [27,28] but is contraindicated in patients with VT secondary to long QT syndrome. For long-term treatment of most patients with left ventricular dysfunction, current clinical practice favours class

II (beta-blockers) and III antiarrhythmics (eg, amiodarone, and sotalol). As for as interventional treatment is concerned, various interventional approaches can be useful for useful for treating VT e.g. VT ablation is very effective for monomorphic/ unifocal VT [29-31]. Implantable cardioverter defibrillator (ICD) can be used to prevent VT related cardiac death for complex and congenital cases [32,33].

Conclusion

VT is a life threatening arrhythmia which has a number of underlying causes ranging from very common to rare and could be multi-factorial. Various episodes of VT in a single patient could be due various underlying causes on each presentation as in our case. Hence, a thorough assessment of patient on each presentation is vital as to not miss a treatable underlying cause as outcome could lead to grave consequences. Previously normal cardiac investigations do not exclude the possibility of future ventricular arrhythmia.

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