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CASE REPORT
Arrhythmias/electrophysiology

A case report reappraising the usefulness of Valsalva manoeuvre in drug-refractory ventricular tachycardia

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Background Ventricular tachycardia (VT) is often misdiagnosed as supraventricular tachycardia with aberrancy. Twelve-lead electrocardiogram remains a key diagnostic tool to differentiate them while providing insights to aid localization of VT. The use of Valsalva manoeuvre (VM) in terminating VT is not conventionally recommended due to lack of robust evidence of its effectiveness and poor understanding of its mechanism in terminating VT.

Case summary A 74-year-old man with history of ischaemic heart disease was admitted with broad complex tachycardia. VT-1 was diagnosed following failed tachycardia termination by adenosine. Haemodynamic compromise necessitated synchronized cardioversion with successful reversion. However, a different VT-2 occurred after cardioversion. VM led to successful termination of VT-2. Subsequently, recurrent episodes of VT-2 occurred with consistent termination by VM. Transthoracic echocardiogram, cardiac magnetic resonance imaging, and a coronary angiogram were performed. Findings suggested that these are likely scar-related VT. VT-1 originated from an anteroseptal scar, whilst VT-2, responsive to VM, likely originated from the Purkinje fibres. Patient remained eurhythmic after Day 1 following amiodarone and beta-blocker initiation. An implantable cardioverter-defibrillator was implanted prior to discharge.

Discussion VM is one of the vagal manoeuvres which are commonly used as initial management of supraventricular tachycardia. Its role in management of VT is obscure. Anecdotal case series recorded its successful use for managing particular VT. Exact mechanism remains elusive although postulated to involve change in cardiac size during strain and release of acetylcholine.

Keywords Ventricular tachycardia • Vagal manoeuvre • Valsalva manoeuvre • Case report

Learning points

- Correct diagnosis of ventricular tachycardia (VT) is life-saving, requiring prompt electrocardiogram recognition facilitated by patient's history and clinical examination.
- Additional recognition of different VT morphologies provides insights not only in localization but also the mechanism of arrhythmogenesis.
- A structured treatment protocol will ensure success in most acute presentations of VT.
- Awareness of unconventional modalities of treatment such as Valsalva manoeuvre in the context of drug-refractory VT can at times be useful.

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Introduction

Ventricular tachycardia (VT) is a cardiac emergency exerting significant morbidity and mortality. Differentiation between VT and supraventricular tachycardia with aberrancy (SVT-A) can be challenging, necessitating awareness of the salient electrocardiogram (ECG) criteria[1] and at times, proven refractoriness to adenosine. Despite well-established guidelines and evidence-based anti-arrhythmic medications for VT management, the role of Valsalva manoeuvre (VM) as an effective treatment for VT remains controversial.[2,3] In this case report, we describe a patient who presented with multiple drug-refractory VTs, one of which repeatedly terminated by VM.

Timeline

1 week earlier	
Presentation to Emergency Department (ED) with palpitations and presyncope. No arrhythmia was diagnosed	Planned for outpatient Holter and transthoracic echocardiography (TTE).
Day 1: 0–1 h	
ED attendance after being found in broad complex tachycardia at outpatient TTE	Diagnosed as ventricular tachycardia (VT) (VT-1) following lack of tachycardia response with adenosine. Subsequent haemodynamic compromise in VT-1. Successful synchronized direct current cardioversion to sinus rhythm.
Day 1: 2–12 h	
Recurrence of multiple episodes of VT with different morphology (VT-2)	Haemodynamically stable VT despite ongoing intravenous (IV) amiodarone infusion with successful termination by Valsalva manoeuvre.
Day 2	
Patient remained stable in sinus rhythm	Conversion from IV to oral amiodarone.
Days 3 and 4	
No further VT episode	Investigated with TTE, coronary angiogram, cardiac magnetic resonance imaging.
Day 5	
Electrophysiology opinion was sought for a VT stimulation study; planning for secondary prevention implantable cardioverter-defibrillator (ICD)	VT stimulation study or electrophysiology study was deemed not indicated given the freedom from further VT. A dual-chamber ICD was implanted.
Day 6	
	Discharged from hospital with follow-up with heart failure and device clinic.
Day 180	
	No VT detected on routine ICD check.

Case presentation

A 74-year-old Caucasian male with a history of remote myocardial infarction (MI) was admitted to Emergency Department (ED) with a haemodynamically stable broad complex tachycardia (BCT) of 180 b.p.m., having recently undergone an outpatient Holter for evaluation of syncopal palpitation. He reported dizziness and palpitation but no chest pain. His blood pressure was 102/78 mmHg. General clinical examination, in particular cardiovascular and respiratory examination, was normal. Twelve-lead ECG demonstrated BCT with classical findings suggestive of VT (VT-1, *Figure 1*), supported by its refractoriness to intravenous adenosine administered via a large cannula at the antecubital fossa. Successful administration of adenosine (6 mg, 6 mg, 12 mg) was validated by patient reporting short-living physiological side effects of flushing, chest tightness, dyspnoea, and 'sense of doom'. Patient was initially managed with intravenous amiodarone but subsequent haemodynamic compromise with a blood pressure of 80/60 mmHg necessitated an external direct current cardioversion.

Two hours later, patient had recurrent stable VT with a different morphology (VT-2, *Figure 2*) whist on telemetry. Patient reported symptoms similar to those of the haemodynamically compromised VT-1. A trial of VM led to immediate VT-2 termination. In the first 12 h, the patient had recurrent episodes of VT-2, all of which terminated by VM (*Figure 3*). Twelve hours after initiation of intravenous amiodarone, he had no further VT. He was transitioned to oral amiodarone and bisoprolol.

Routine blood tests were normal. Adjusted serum calcium was 2.39 mmol/L (2.20–2.60), serum magnesium was 0.93 mmol/L (0.7–1.0), and serum potassium was 4.7 mmol/L (3.5–5.3). His baseline ECGs showed sinus rhythm, normal axis, as well as Q waves in V1–V3 and T-wave inversion in V4–V6 (*Figure 4*). A recent outpatient 7-day Holter revealed 1122 VT episodes during which patient experienced dizziness. Coronary angiogram demonstrated single-vessel disease with chronic total occlusion (CTO) of the left anterior descending artery (LAD) (*Videos 1* and 2). Transthoracic echocardiography showed severe left ventricular (LV) systolic impairment (ejection fraction 31%) with akinetic mid-anterior and anteroseptal wall including LV apex. Cardiac magnetic resonance imaging (MRI) demonstrated transmural infarct at the mid-LAD territory implying non-viability and lack of benefit of revascularization of the LAD-CTO (Supplementary material online, *Figure S1*).

Based on these findings, the likely diagnosis is scar-related VT. Following discussion with cardiac electrophysiologists, a VT

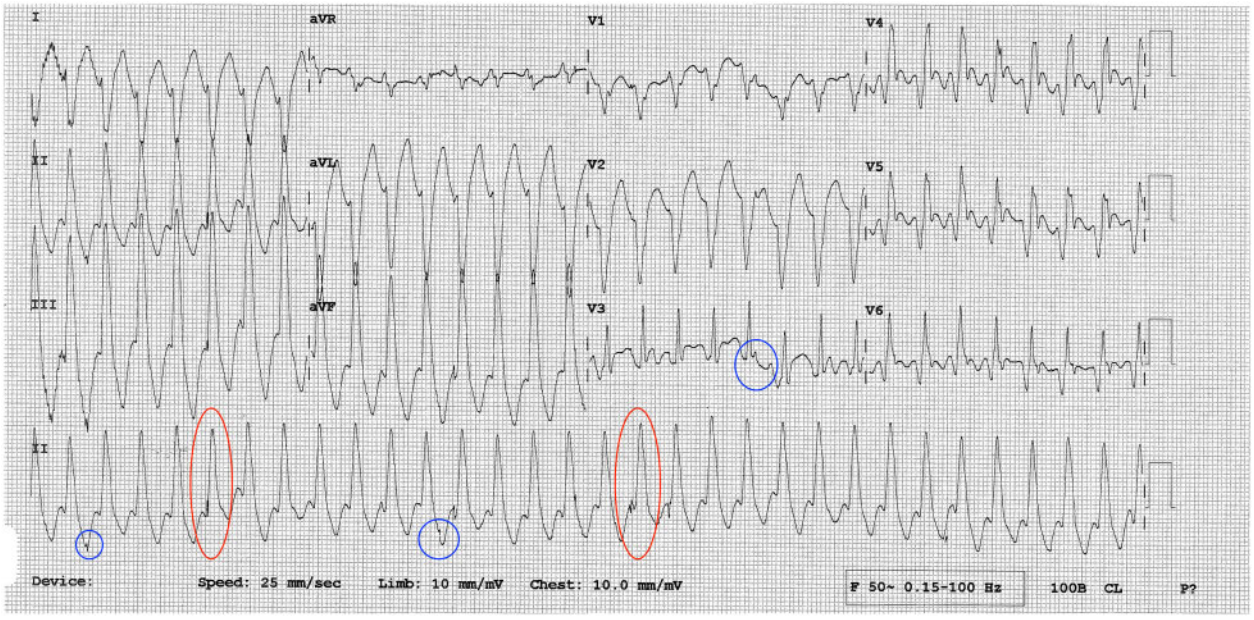


Figure 1 Atrioventricular dissociation (blue circles), dominant S in V1, notching and slurring of S wave (Josephson's sign) in V2, qR pattern in V6, and fusion beats (red circles) in Lead II favours ventricular tachycardia rather than supraventricular tachycardia with aberrancy. Left bundle branch block morphology, inferior axis with QS in V1 and V2 suggest a ventricular tachycardia with an anteroseptal exit (ventricular tachycardia-1).

stimulation study was deemed unnecessary given freedom from VT 12 h after admission. Prior to patient's discharge, an implantable cardioverter-defibrillator (ICD) was inserted for secondary prevention of ventricular arrhythmias. He was followed up in the heart failure clinic and device clinic. There is no ventricular arrhythmia detected on routine ICD monitoring since ICD implantation. The patient remains clinically well to this date.

Discussion

Multiple algorithms have been published to aid differentiation of VT from SVT-A, attesting the challenging diagnostic dilemma faced by physicians for these tachyarrhythmias.[1] In this case report, the ED physician chose to administer adenosine in the first instance given the patient's initial stable haemodynamic status. Adenosine is a relatively safe drug for establishing the diagnosis in 90% of SVT-A cases.[4] However, a survey of the patient's background would have provided the much needed clue. VT is more likely in a patient aged >35 years old presenting as BCT.[5] A history of ischaemic heart disease in the form of a remote MI is 90% predictive of ventricular origin of a BCT.[5]

A conscientious review of ECG reveals important clues of ventricular origin of a BCT. Presence of atrioventricular dissociation, capture beats and fusion beats strongly favours VT. The availability of a baseline ECG for comparison is of paramount help as features such as a change of ECG morphology and axis by >40°, axis of -90° to -180° (otherwise known either as the 'Northwest axis' or 'No man's land'), and a positive QRS amplitude in lead aVR are suggestive of VT.[6,7] Most of these features, namely fusion and capture beats as well as atrioventricular dissociation were present in the ECGs for VT-1 (*Figure 1*) and VT-2 (*Figure 2*) in our case. Another method of identification of VT is based on certain features in the context of right bundle branch block (RBBB) and left bundle branch block (LBBB) morphologies (*Table 1*).[5,8,9] Using these criteria, both BCTs in our patient fulfils the VT diagnosis for his LBBB tachycardia (VT-1) and RBBB tachycardia (VT-2).

The majority of VTs in patients with structural heart disease are due to scar-related re-entry mechanism. Twelve-lead ECG can crudely predict the site of origin of scar-related VT coinciding with scar border.[10] A general guide to aid ECG localization of the origin of VT is provided in *Figure 5*. Using these simple rules, VT-1 typified by LBBB morphology and inferior axis (*Figure 1*), is localized to an anteroseptal exit, corroborated by cardiac MRI findings of scarring in this region. The second VT with RBBB morphology and inferior axis (*Figure 2*) hinted at a more basal location, and is potentially a Purkinje VT,[10] with the sharp initial QRS deflection suggesting the VT arising from the His-Purkinje conducting system. Indeed, it is conceivable that VT-2 is an interfascicular VT utilizing the left anterior fascicle as the antegrade limb.

The type of VT can guide suitable pharmacological approach. Idiopathic outflow tract VT responds to beta-blockers, while adenosine can successfully terminate monomorphic right ventricular outflow tract (RVOT) VT.[2,11] American Heart Association/American College of Cardiology/Heart Rhythm Society (AHA/ACC/HRS) Guideline recommends Procainamide as a preferred option over Amiodarone.[12] However, Amiodarone is traditionally used as first option based on clinicians experience.[11] VM, a vagal manoeuvre, however, is not recommended by convention as part of VT

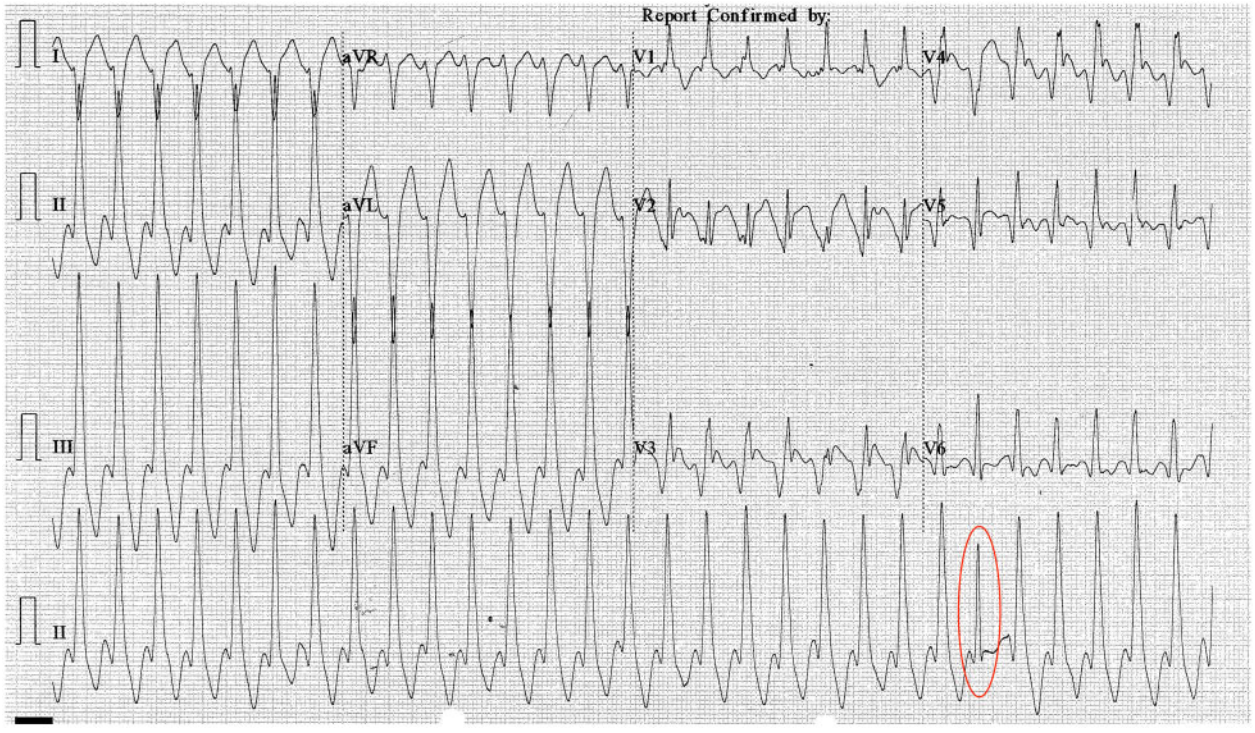


Figure 2 The significant difference from *Figure 1* can be appreciated in positive QRS amplitude in V1 and V2, i.e. right bundle branch block pattern (ventricular tachycardia-2). Inferior axis deviation remained the same. There is capture beat in lead II (red circle) together with positive concordance in chest leads V1 to V6 suggesting a more basal origin. Involvement of Purkinje system is suggested by the right bundle branch block morphology with sharp initial QRS deflection.

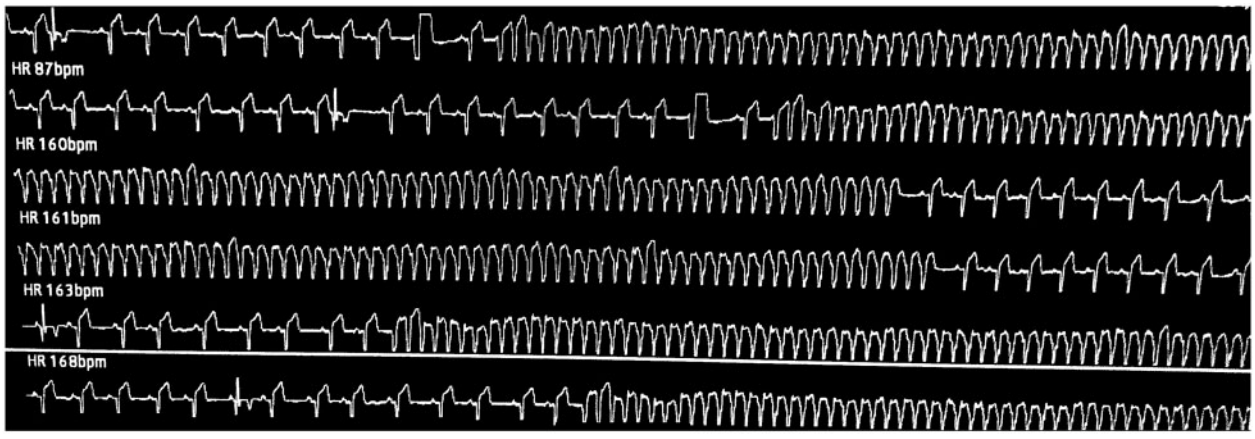


Figure 3 Telemetry tracing showing episodes of ventricular tachycardia (ventricular tachycardia-2) repeatedly terminated with vagal manoeuvre.

treatment, partly due to lack of understanding in its mechanism in terminating certain types of VT in anecdotal cases. In this case report, VT-1 possesses the same ECG morphologies as RVOT VT (i.e. a normal-heart focal VT) and yet failed to be terminated by adenosine. In the presence of structural heart disease, VT-1 turns out to be arising as a macro-reentrant VT from an anteroseptal exit, terminated by electrical cardioversion and suppressed by subsequent intravenous amiodarone.

Interestingly, VT-2 with RBBB morphology and inferior axis was consistently terminated by VM, with its behaviour mimicking focal/triggered VT arising from Purkinje system. Vagal manoeuvres increase vagal parasympathetic tone and terminate sympathetic-driven

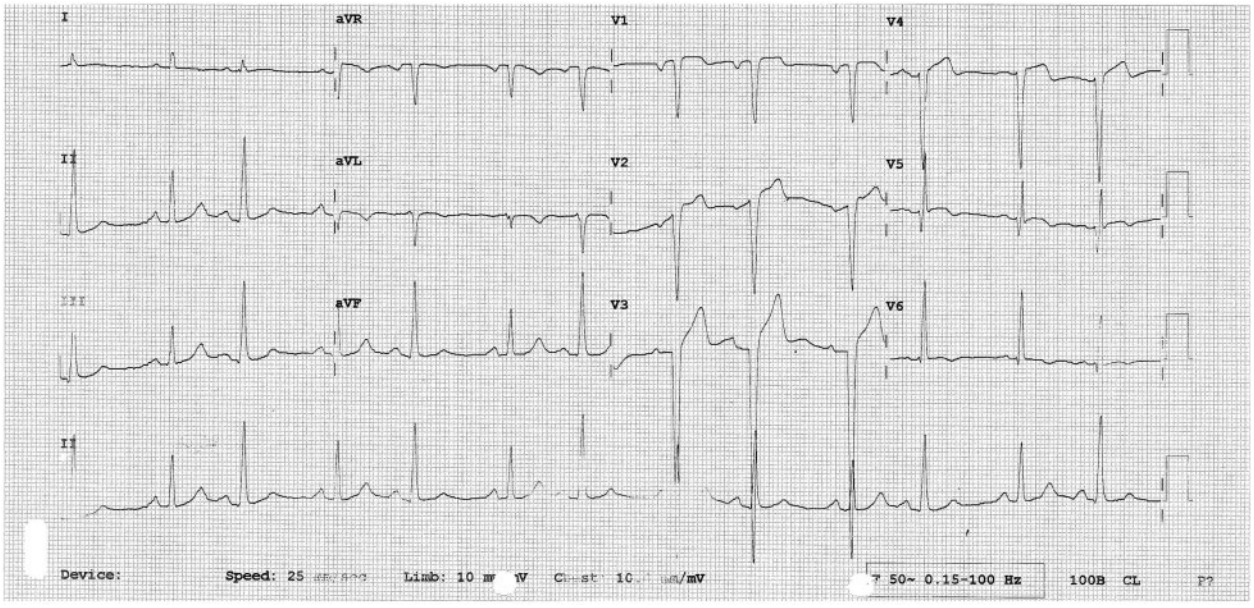
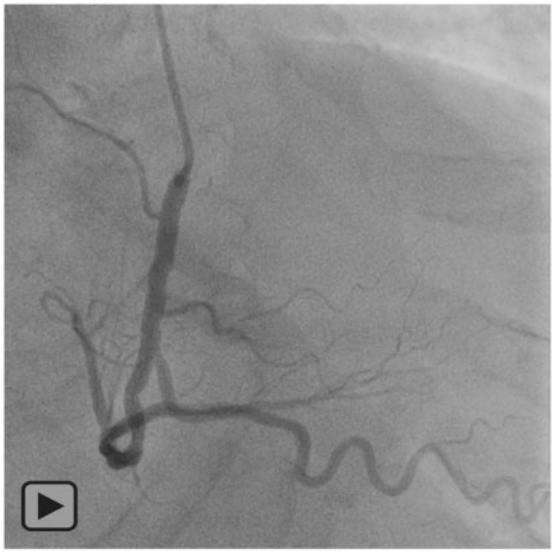
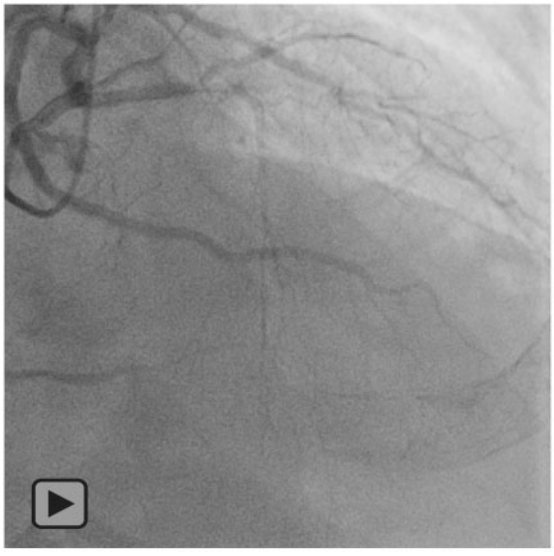


Figure 4 Baseline electrocardiogram of the patient demonstrated sinus rhythm with left ventricular hypertrophy voltage criteria, left ventricular strain pattern, and Q waves in V1–V3.



Video 1 Coronary angiogram of the right coronary artery (RAO cranial view) demonstrates retrograde filling of the LAD.



Video 2 Coronary angiogram of the left system (RAO cranial view) demonstrates chronic total occlusion of LAD.

arrhythmias, especially SVT with a success rate of up to 40%.[13] Carotid sinus massage and VM are vagal manoeuvres, which increase the arterial pressure in the carotid sinuses and aortic arch triggering a baroreceptor reflex resulting in increased parasympathetic output to the heart via the vagus nerve with differential effect to the sinoatrial and the atrioventricular node respectively.[13]

Medical literature demonstrates the rare cases of vagal manoeuvres terminating VT. The successful use of VM in adults was described by Waxman *et al.*[2] and in a paediatric patient by Chaszczewski *et al.*[3] Waxman *et al.*[2] demonstrated that nine patients with recurrent VT were terminated repeatedly by VM. One of the postulated mechanisms is the sudden change in blood pressure and more importantly on cardiac size rather than the effect of parasympathetic vagal tone. Focal VT arises from Purkinje system by triggered automaticity[2] and is common in 20% of patients with post-MI septal scar.[14] It has been suggested that sudden changes in cardiac size by VM terminates

Table 1 BCT with right bundle branch block (RBBB) and left bundle branch block (LBBB) morphologies favouring diagnosis of VT

RBBB	LBBB
QRS complex duration > 140 ms	QRS complex duration > 160 ms
Positive concordance in precordial leads	Negative concordance in precordial leads
V1: qR, R, or RS complex; RSR' where R is taller than R', and S crossing baseline	V1: rS complex
V6: R/S ratio > 1	V6: QS (i.e. mostly negative) complex
Superior axis	

BCT, broad complex tachycardia; ms, milliseconds; VT, ventricular tachycardia.

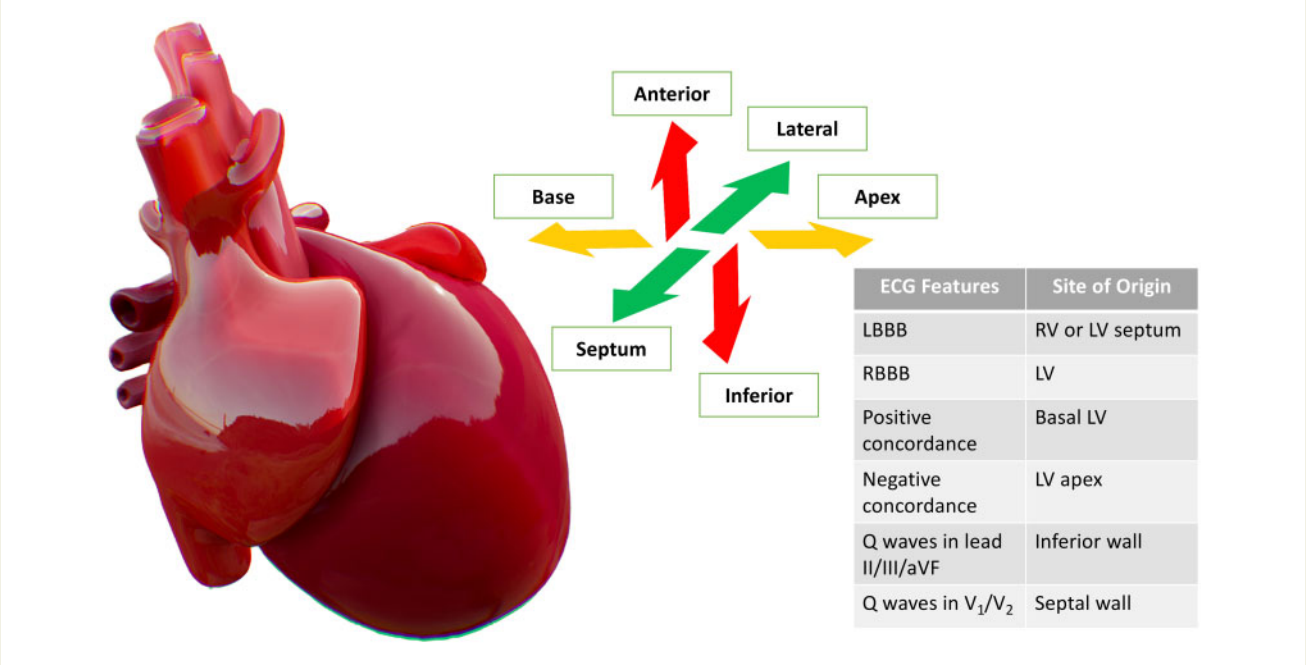


Figure 5 Crude electrocardiogram localization for site of origin for ventricular tachycardia. Q waves usually indicates the myocardial region where ventricular tachycardia is originating. Hence an inferior axis implies an origin *opposite* the inferior wall, i.e. the anterior wall. LBBB, left bundle branch block; LV, left ventricle; RBBB, right bundle branch block; RV, right ventricle.

Purkinje VT by reducing Purkinje fibre stretch.[2] Another hypothesis is that VM leads to vagal-mediated acetylcholine release, which in turn decreases cyclic adenosine monophosphate and β-adrenergic stimulation of the ventricular myocardium, an effect akin to that by adenosine.[3] Our case gives credence to the hypothesis proposed by Waxman *et al.* with evidence of Purkinje VT responding to VM.

In comparison to aggressive antiarrhythmic medications, VM can be easily and quickly utilized. Hence, further mechanistic studies for the use of VM is warranted. This case study highlighted the effectiveness of the VM in patients with specific VT, and as such it should not be discounted as a potential acute treatment whilst more definitive treatment of chemical or electrical cardioversion is sought.

Limitation

We acknowledge that deduction of site of origin of VT based on ECG localization provides important anatomical and mechanistic insight, but 12-lead ECG remains a crude tool with many limitations.[15] As such, these VT trigger/substrates should be confirmed through an electrophysiology study. However, given the suppression of both VTs following adequate amiodarone loading, there is no indication for an electrophysiology study. Ultimately, the patient would require a secondary prevention ICD implantation due to haemodynamic compromise during VT-1. In the case of VT-2 which was consistently terminated by VM, no adenosine was used but assessment of VT-2 by adenosine may provide further insights into the mechanism of this VT.

Patients perspective

I entered the A/E feeling very poorly and had been having dizzy spells for a week causing blackout on one occasion. Drugs were

administered and my heart rate was 188/min. The drugs were administered several times with no effect. I was then asked after several hours to hold my nose and blow hard. This reduced my heart rate to below 100/min. This was done several times and had a most positive effect. The result was I felt much better and my heart rate stabilized to 60–65.

Lead author biography

Tin Sanda Lwin graduated in the University of Medicine (1), Yangon, Myanmar in 2009. She finished MRCP in 2014. She worked as a cardiology registrar in Khoo Teck Puat Hospital, Singapore for 3 years. She moved to UK and worked as an acute medicine trust grade registrar in Kettering General Hospital in 2018 and then as a trust grade cardiology registrar in the same hospital from 2019 to 2020. She has been offered a national traning number in cardiology and currently working in the Castle Hill Hospital as a cardiology specialist trainee year 3.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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