CME Article

What is the cause of the regular wide QRS complex tachycardia?

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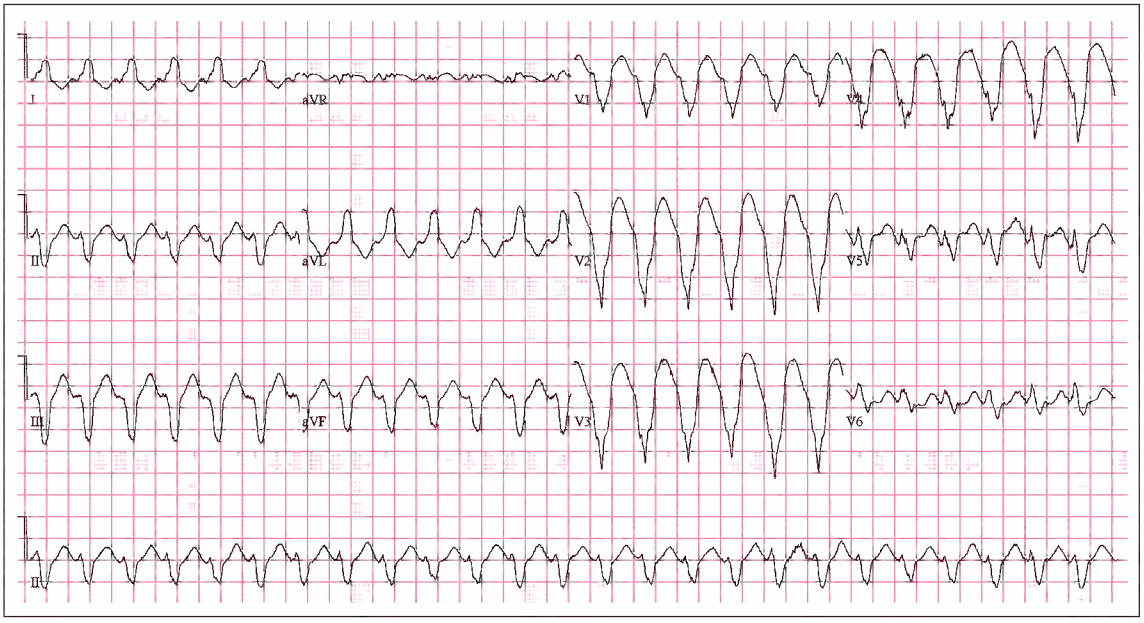

Fig. 1 12-lead ECG shows broad complex tachycardia with a rate of 150 bpm. There is left axis deviation. The QRS complexes measure 200 ms, and no atrioventricular dissociation is observed. There is negative concordance, which shows that the monomorphic ventricular tachycardia arises from the anterior ventricular wall.

CASE 1

CLINICAL PRESENTATION

An 88-year-old man presented with a one-week history of shortness of breath and intermittent palpitations. He had a history of ischaemic heart disease with no prior revascularisation. What does the electrocardiogram (ECG) done on arrival at the emergency department (Fig. 1) show?

ECG INTERPRETATION

Fig. 1 shows a broad QRS complex tachycardia, with a rate of 150 beats per minute (bpm). The QRS complexes measure 200 ms and are identical. There is no atrioventricular dissociation seen. The axis is left (approximately –60°). The QRS complexes in the praecordial leads are predominantly negative (negative concordance). This shows monomorphic ventricular tachycardia (VT). The presence of negative concordance correlates with tachycardia originating from the anterior ventricular wall. ECG done post cardioversion (Fig. 2) shows deep and wide Q waves in leads V1–V3. Smaller Q waves are also seen in leads V4 and V5. The ECG changes indicate an old anterior myocardial infarct.

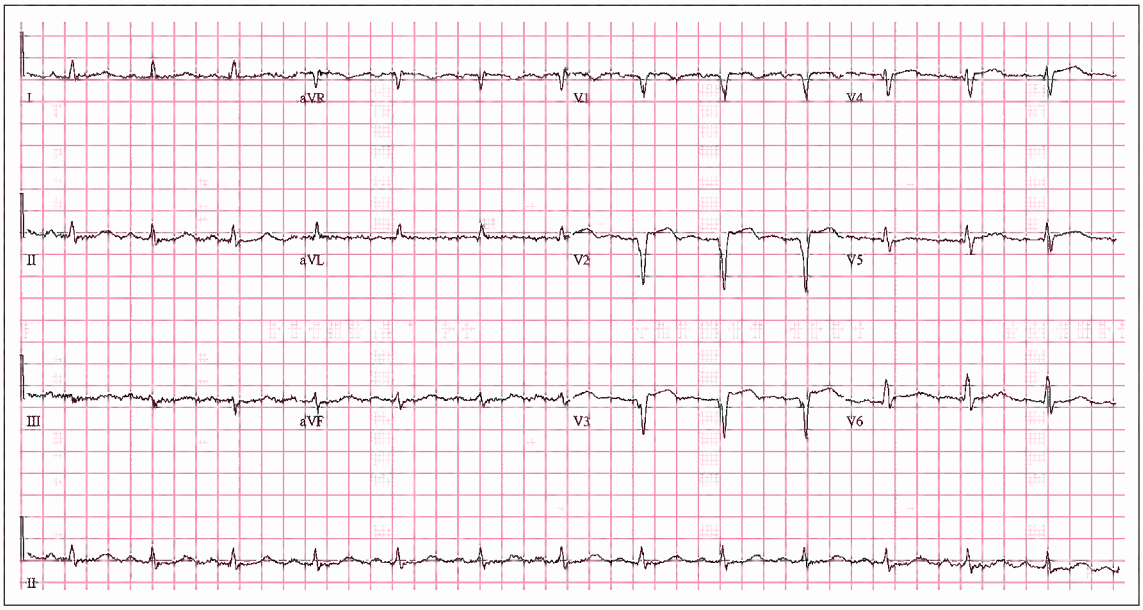

Fig. 2 12-lead ECG shows sinus rhythm with a heart rate of 80 bpm. There are Q waves in leads V1–V3.

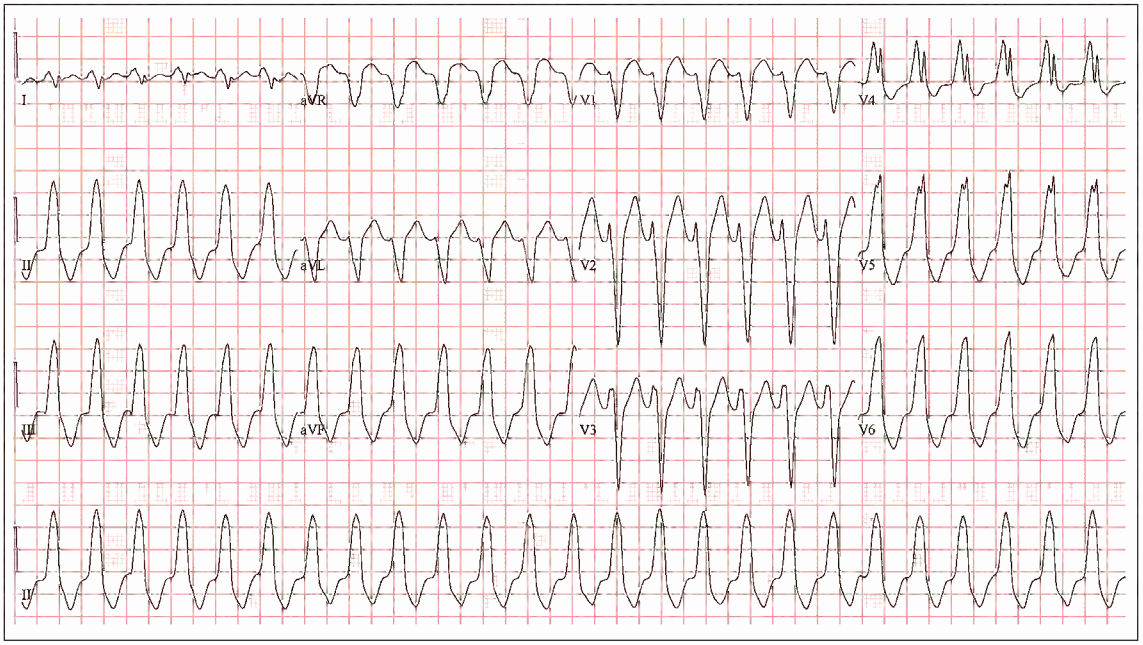

Fig. 3 12-lead ECG shows a broad complex tachycardia with a rate of 156 bpm. There is a left bundle branch block morphology with an inferior axis, which is typical of a right ventricular outflow tract ventricular tachycardia.

CLINICAL COURSE

The patient underwent electrical cardioversion, as he was hypotensive at presentation. He was then commenced on oral amiodarone and had further runs of VT in the ward requiring intravenous lignocaine. Bisoprolol was added. Transthoracic echocardiogram performed showed moderate left ventricular systolic dysfunction with regional wall motion abnormality at the anterior walls. Implantation of an implantable cardioverter-defibrillator (ICD) was considered in this patient. However, the patient and his family decided against the procedure due to his age and poor premorbid condition. He has been asymptomatic since.

CASE 2

CLINICAL PRESENTATION

A 26-year-old man presented with a one-day history of palpitation associated with chest tightness. This was the first time he experienced such symptoms. There was no prior medical history of any illness. An ECG was done at the emergency department. What does it show (Fig. 3)?

ECG INTERPRETATION

Fig. 3 shows a broad QRS complex tachycardia with a rate of 156 bpm. The QRS complexes measure 160 ms and are identical. The axis is inferior. The QRS complex shows a left bundle branch block (LBBB) morphology. There is no atrioventricular (AV) dissociation. rS complexes are seen in leads V1-V3 with broad r waves in V2 and V3. These and the presence of a QRS LBBB morphology and inferior axis indicate a VT originating from the right ventricular outflow tract (RVOT-VT).[1]

CLINICAL COURSE

The patient was given intravenous amiodarone at the emergency department. Post cardioversion, the ECG was normal, showing normal sinus rhythm with a heart rate of 78 bpm. Transthoracic echocardiogram performed showed normal chamber sizes, preserved left ventricular systolic function, mild mitral valve prolapse involving the anterior leaflet, and a slightly dilated aortic root. Magnetic resonance imaging of the heart revealed normal biventricular systolic function, normal chamber sizes and no imaging features of arrhythmogenic right ventricular cardiomyopathy. The patient subsequently underwent an electrophysiology study, which showed inducible arrhythmia at the anteroseptal region of the RVOT. Radiofrequency ablation was done with no inducible arrhythmias after the ablation. He has been asymptomatic since.

DISCUSSION

VT refers to any rhythm that is faster than 120 bpm arising distal to the bundle of His. The rhythm may originate from the distal conduction system and/or the ventricular myocardium. VT may be self-terminating, but is described as "sustained" if it lasts longer than 30 seconds.[2] It can be monomorphic or polymorphic. In monomorphic VT, the arrhythmia originates from a single focus, with identical QRS complexes. It may be generated from increased automaticity of a single point in either ventricle, or is due to a re-entry circuit within the ventricle. Polymorphic VT has beat-to-beat variations in morphology, and most commonly appears as a cyclical progressive change in the cardiac axis.

This article focuses on monomorphic VT, as is reflected in the cases presented. Monomorphic VT is most commonly seen in patients with underlying structural heart disease. Causes include previous myocardial infarction, primary cardiomyopathy, surgical myocardial scar, hypertrophic myocardium and infiltrative diseases of the heart. The tachycardia is usually initiated by an extrasystole and involves two pathways of conduction with differing electrical properties. The re-entry circuits that support VT often occur in the zone of ischaemia or fibrosis surrounding the damaged myocardium.

Idiopathic VT concerns a small subgroup of patients with monomorphic VT, where there is an absence of structural heart disease. It is often exercise-dependent, and the clinical behaviour may be more consistent with triggered activity or abnormal automaticity. This type of VT is typically named after its site of origin. The most commonly involved site is the RVOT; other sites include the left ventricular septum and aortic root. RVOT-VT is a diagnosis of exclusion; structural heart disease should be adequately explored and ruled out. Although classically considered benign, sudden death may rarely occur despite the presence of a structurally normal heart.[3] In addition, idiopathic VT can cause tachycardia-induced cardiomyopathy.[4]

The diagnosis of VT is made based on the 12-lead ECG or a rhythm strip obtained from telemetry or 24-hour Holter ambulatory ECG monitoring. It may be difficult to differentiate between VT and supraventricular tachycardia (SVT) with aberrancy. A third possibility of a regular wide QRS complex tachycardia is antidromic atrioventricular re-entrant tachycardia occurring in a patient with Wolff-Parkinson-White syndrome. It is a re-entrant circuit that involves anterograde conduction via the accessory bundle of His and retrograde reduction via the AV node.

The 12-lead ECG in VT shows widened and bizarre-looking QRS complexes. The broader the QRS complex, the more likely the rhythm is ventricular in origin, especially if the QRS complexes exceed 160 ms. The rate of the tachycardia is usually 140–200 bpm. The points in favour of VT rather than SVT with aberrant ventricular conduction are: (a) AV dissociation; (b) Concordant pattern, where the polarity of all the QRS complexes in the praecordial leads is either positive or negative; (c) Sinus capture beats manifesting as beats with narrow QRS morphology in the midst of rapid and widened ventricular complexes; (d) Fusion beats; (e) A QRS axis in the north-west quadrant. In contrast, the ECG in SVT with aberrant ventricular conduction often shows a 1:1 P-QRS relationship and widened QRS complexes that frequently manifest as right bundle branch block morphology.

Although often mentioned as an important criterion for the diagnosis of VT, AV dissociation is, however, seen in only about 50% of VT.[5] Therefore, its absence by no means excludes this diagnosis, although its presence makes the tachycardia very likely to be a VT. Here, the sinus node continues to

initiate atrial contraction independent of ventricular activity, resulting in the dissociation of P waves from QRS complexes. However, even if AV dissociation is present, it is not always possible to recognise its existence in the 12-lead ECG. A concordance pattern, sinus capture beats and fusion beats are all seldom present (each in only about 5% of VT cases). Therefore, it has been recognised in recent years that it is of crucial importance to analyse the morphology of the ventricular complexes in a full 12-lead ECG (which should always be done), especially in leads V1 and V6. A ventricular complex with a right bundle branch block pattern, especially a triphasic pattern (i.e. rSR') in lead V1, favours SVT with aberrant ventricular conduction. On the other hand, a monophasic R wave in lead V1 or a rS complex in lead V6 both strongly favour the diagnosis of VT. In addition, there are several other important differences in the QRS morphologies between the two arrhythmias.[2,5,6]

Case 1 demonstrates monomorphic VT originating from scarred myocardium from a previous myocardial infarction. The QRS complexes are identical; broad QRS complexes of 200 ms and negative concordance favour a diagnosis of VT. In Case 2, broad QRS complexes of 160 ms, LBBB morphology in the precordial QRS complexes and an inferior axis suggest an RVOT origin of the tachycardia.

VT is often associated with haemodynamic compromise. During VT, cardiac output is reduced due to the rapid heart rate and lack of a properly timed atrial contraction. Diminished cerebral perfusion manifests as light-headedness and syncope. Chest pain may be due to ischaemia or arrhythmia. Haemodynamic collapse is more likely when underlying left ventricular dysfunction is present or with very rapid rates. With some exceptions, VT is associated with an increased risk of sudden cardiac death.

The acute emphasis is on achieving an accurate diagnosis from the ECG and arrhythmia conversion. The severity of clinical symptoms determines the urgency of treatment. When the rhythm diagnosis is in question, resuscitative therapy should be directed toward VT.[6] Pulseless VT is treated with immediate defibrillation. Haemodynamically unstable VT requires immediate electrical cardioversion with standard advanced cardiac life support protocol. In a haemodynamically stable patient with no evidence of coronary ischaemia or infarction, rhythm restoration in monomorphic VT can be achieved via medical cardioversion; intravenous amiodarone or lignocaine are appropriate anti-arrhythmic agents. If medical therapy is unsuccessful, synchronised cardioversion is appropriate. Electrolyte abnormalities, in particular, hypokalaemia and hypomagnesaemia, should be corrected.[7]

Following conversion of VT, the clinical emphasis shifts to determining the severity of heart disease, the prognosis and the best long-term management plan. Options include medications, ICD implantation and catheter ablation. The therapies depend on the severity of symptoms and the degree of structural heart disease. A combination of these therapies is often used when structural heart disease is present. Class III anti-arrhythmics (e.g. sotalol) and beta blockers are preferred in most patients with left ventricular dysfunction. ICD implantation is recommended for patients at risk of recurrent VT and/or ventricular fibrillation. Endocardial catheter ablation is used early in idiopathic monomorphic VT.[8] Ablation should be recommended for patients who present with syncope and who also remain symptomatic despite optimal medical therapy with a well-tolerated drug. It should also be considered in patients with myocardial ischaemia who are not candidates for revascularisation. In addition, it can be used to reduce arrhythmic burden in the presence of cardiomyopathy.[9]

In general, the prognosis for VT is best predicted by left ventricular function. Some exceptions would include long QT syndrome, right ventricular dysplasia and hypertrophic cardiomyopathy, where the patients are at increased risk of sudden death despite relatively preserved left ventricular ejection fraction.

ABSTRACT

Regular broad QRS complex tachycardias may be ventricular in origin or due to supraventricular tachycardia with aberrancy. Antidromic atrioventricular re-entrant tachycardia occurring in Wolff-Parkinson-White syndrome is a third possibility. The electrocardiogram is a key tool for distinguishing these tachycardias, which have differing causes, prognoses and treatment strategies. Ventricular tachycardia may be monomorphic or polymorphic. The management of ventricular tachycardia depends on clinical symptoms and is influenced by the presence of structural heart disease.

Keywords: diagnosis, management, monomorphic, regular broad complex tachycardia, ventricular tachycardia

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 201106A)

	True	False
Question 1. These features in the ECG of broad complex tachycardia favour a diagnosis of ventricular tachycardia:		
(a) Fusion and capture beats.	☐	☐
(b) Atrioventricular dissociation.	☐	☐
(c) Right bundle branch block morphology with triphasic pattern in lead V1.	☐	☐
(d) Concordant pattern.	☐	☐
Question 2. If the broad complex tachycardia has a left bundle branch block morphology, a ventricular origin is suggested by the following features on ECG:		
(a) QRS complexes with duration > 160 ms.	☐	☐
(b) An rS complex in V1 with a wide r wave (> 0.3 sec).	☐	☐
(c) Negative concordance in the praecordial leads.	☐	☐
(d) Normal axis.	☐	☐
Question 3. The following are causes of monomorphic ventricular tachycardia:		
(a) Hypertrophic obstructive cardiomyopathy.	☐	☐
(b) Antidromic atrioventricular re-entrant tachycardia in Wolff-Parkinson-White syndrome.	☐	☐
(c) Old myocardial infarction.	☐	☐
(d) Arrhythmogenic right ventricular cardiomyopathy.	☐	☐
Question 4. The following symptoms may be manifestations of ventricular tachycardia:		
(a) Syncope.	☐	☐
(b) Palpitations.	☐	☐
(c) Sudden cardiac death.	☐	☐
(d) Angina.	☐	☐
Question 5. The following therapies are used in rhythm restoration in ventricular tachycardia:		
(a) Intravenous amiodarone.	☐	☐
(b) Intravenous verapamil.	☐	☐
(c) Intravenous lignocaine.	☐	☐
(d) Intravenous esmolol.	☐	☐

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