Diagnosis and management of ventricular tachycardia

Authors: John Whitaker, A Matthew J WrightB and Usha TedrowC

Ventricular tachycardia (VT) describes rapid heart rhythms originating from the ventricles. Accurate diagnosis of VT is important to allow prompt referral to specialist services for ongoing management. The diagnosis of VT is usually made based on electrocardiographic data, most commonly 12-lead echocardiography (ECG), as well as supportive cardiac telemetric monitoring. Distinguishing between VT and supraventricular arrhythmias on ECG can be difficult. However, the VT diagnosis frequently needs to be made rapidly in the acute setting. In this review, we discuss the definition of VT, review features of wide-complex tachycardia (WCT) on ECG that might be helpful in diagnosing VT, discuss the different substrates in which VT can occur and offer brief comments on management considerations for patients found to have VT.

Introduction

During normal atrioventricular (AV) conduction, supraventricular impulses are conducted through the cardiac conduction system (CCS) comprising the AV node, penetrating AV bundle, right and left bundle branches and fascicles, which subsequently activate Purkinje cells of the peripheral ventricular conduction system (PVCS), typically located in the subendocardial tissue of the ventricle, and then the contractile ventricular myocardium itself. The fascicular system comprises the penetrating AV bundle, which arises from the AV node and penetrates the aorto-ventricular membrane, or central fibrous body, following which it separates into the right and left bundles. The bundles subdivide into the distal fascicles, which, in the left ventricle (LV), can exhibit significant cross-linking (or arborisation) and anatomical variation. Fascicular tissue is likely to arise from myocytes within the developing interventricular septum and, in maturity, comprises electrically insulated, rapidly conducting cells, which can be distinguished from AV nodal tissue by their rapid conduction and expression of Connexin-40 (Cx-40) gap junction proteins. Purkinje cells of the PVCS, which also express Cx-40, are derived from trabeculations within the ventricular myocardium and connect to the fascicular tissue during development to complete the formation of the CCS.

Ventricular tachycardia (VT) is defined as an arrhythmia that originates from tissue below the penetrating AV bundle and results in a fast (>100 beats per minute; bpm) ventricular rate. These arrhythmias can arise from, or involve, specialised conducting tissue as well as ventricular myocardium. VT can occur in structurally normal or abnormal hearts and can be accompanied by an irregular rhythm. An irregular rhythm does not exclude VT as an underlying diagnosis.

Where there is any uncertainty, particularly in emergent situations, a wide-complex tachycardia should be treated as VT until proven otherwise and, ~80% of the time, the diagnosis will prove to be VT, especially in patients aged >35 years.

Most VT occurs in patients with underlying heart disease but ~10% occurs in those with structurally normal hearts, often arising from the outflow tracts.

Management of patients with VT can involve risk stratification for sudden cardiac death; the use of implantable cardioverter-defibrillators; suppression of VT through the treatment of any underlying myocardial disease; the use of anti-arrhythmic drugs; catheter ablation; and, in refractory cases, emerging approaches, such as cardiac stereotactic body radiation therapy.

Key points

VT is an arrhythmia with a rate >100 beats per minute arising from the ventricular myocardium or specialised conduction tissue below the penetrating atrioventricular bundle.

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haemodynamically stable or unstable. It is not possible to definitively distinguish between rhythms of ventricular or supraventricular origin solely based on ventricular rate, the presence or absence of structural heart disease, or whether a rhythm is haemodynamically tolerated.

**Diagnosis**

VT forms an important differential diagnosis for any patient presenting with a regular wide complex tachycardia (WCT), which is a regular heart rhythm demonstrating at least three consecutive beats with a ventricular rate >100 bpm and a prolonged QRS duration (>120 ms) on 12-lead electrocardiogram (ECG). In addition, VT can present as an irregular rhythm, although irregularity does not exclude VT as a diagnosis. A wide QRS complex indicates a delay in propagation of an electrical impulse throughout the ventricular myocardium and could be the result of slowed or absent conduction in any single portion of the CCS, or origin of a ventricular impulse from a location where it does not engage the CCS. In this case, propagation throughout the ventricles occurs at a slower speed, because of conduction via contractile myocardium as opposed to specialised conduction tissue. In addition to VT, the differential diagnosis for a WCT includes any supraventricular tachycardia (SVT) with associated bundle branch block, SVT that is conducted to the ventricles via an accessory atroventricular (or related) pathway (or ‘pre-excited’ SVT), pacemaker-mediated tachycardia, SVT conducted with a delay in myocardial propagation (often because of medication, including class 1A, 1C and class III anti-arrhythmic drugs) or systemic disturbances resulting in abnormal myocardial electrical propagation. When possible, comparison between a resting 12-lead ECG and the WCT is helpful, because it enables a comparison between the morphology of a conducted rhythm and the WCT.

It is generally accepted that the default diagnosis of a WCT on ECG should be considered VT until proven otherwise, particularly in urgent situations. This consideration is based on the importance of avoiding administration to patients with VT of medication commonly reserved for the treatment of SVT, which can commonly be associated with haemodynamic deterioration when administered during VT.

**Pre-test probability**

When attempting to diagnose the cause of a WCT on ECG, it is important to acknowledge the pre-test probability of different aetiologies that might be responsible. It is has been reported that ~80% of presenting WCTs are ultimately diagnosed as VT. In the context of a WCT, structural heart disease (including previous myocardial infarction, angina or heart failure) is associated with a positive predictive value (PPV) of >95% for VT; age over 35 confers a PPV of 85% for VT; and age <35 confers a PPV of 70% for SVT. Although these observations should not prevent a careful examination of the ECG and the clinical context in which a tachycardia occurs, they can be helpful when considering management options when uncertainty persists despite consideration of the available evidence.

**Diagnosis of ventricular tachycardia**

A definitive diagnosis of VT requires the demonstration of ventriculoatrial dissociation (ie ventricular activation that is independent of atrial activity). With extremely rare exceptions, this can be considered the case when the number of spontaneous ventricular activations exceeds the number of atrial activations. Alternatives features that can be taken as evidence of AV dissociation include the presence of fusion and capture beats, which are characterised on 12-lead ECG as early beats (ie occurring before the next expected beat of WCT would be seen) with a typical conducted QRS morphology (capture) compared with a 12-lead ECG in sinus rhythm or an intermediate morphology between the WCT and a conducted beat (fusion). AV dissociation can also be demonstrated on 12-lead ECG through cardiac electrical implantable devices (CIED) or by invasive electrophysiology study (Fig 1). When this phenomenon is present, an arrhythmia can be considered ventricular in origin, which could be helpful for guiding immediate and subsequent therapy.

Proof of ativoventricular dissociation is sometimes not possible based on available data when a patient is being assessed and, therefore, a judgement about the likely origin of an arrhythmia must be made instead. Such a judgement can be based on the clinical context in which the arrhythmia occurs, the myocardial substrate that an arrhythmia occurs in and electrocardiographic evidence that might be suggestive, even if not diagnostic, of a ventricular origin for the rhythm. It might be easier to prove that an arrhythmia is not ventricular in origin compared with proving that it is. In the absence of contra-indications, vagal manoeuvres or adenosine can be diagnostically helpful in that they can slow AV conduction, proving SVT, and arrhythmias might terminate following the administration of adenosine, which would be suggestive of a supraventricular arrhythmia, whereas slowing of ventriculoatrial conduction could expose VA dissociation, proving VT.

There are ECG features that are suggestive of VT as opposed to SVT, with some mechanism of QRS broadening. Several algorithms have been proposed to systematically distinguish between VT and SVT (Fig 2). They typically depend on several principles to indicate the likely origin of a WCT, which include:

- Similarity of QRS morphology to a typical pattern of aberrant conduction: if the QRS complex has a ‘typical’ bundle branch or fascicular block appearance, there is a higher likelihood that the arrhythmia is an aberrantly conducted SVT. If the appearance is not consistent with any combination of bundle branch or fascicular blocks, then the diagnosis is most likely that of pre-excited SVT or VT.
- Ventricular activation speed: this can be considered as the speed of initial ventricular propagation, as defined by the steepness of the initial QRS deflection. A rhythm that is conducted via the CCS will usually be associated with rapid early deflection in the QRS complex. By contrast, impulses arising from a ventricular focus remote from the CCS will usually propagate slowly initially, giving rise to a less steep initial portion of the QRS complex. This has been quantified as an RS interval in any precordial lead of >100 ms (Brugada algorithm) or an R-wave in V1 of >30 ms or >60 ms from QRS onset to the nadir of the S-wave in V1 or V2 (Kindwall criterion). In addition, overall QRS duration >140 ms in a RBBB configuration or >160 ms in a LBBB configuration favours VT.
- Relative rapidity of the early portion of ventricular activation compared with the late portion of ventricular activation: this is based on the principle that, during aberrantly conducted SVT, the initial ventricular activation would be rapid, with conduction delay occurring later in the ventricles, whereas, in VT, the initial
Fig 1. Electrocardiographic diagnosis of ventricular tachycardia (VT). (a) 12-lead electrogram (ECG) showing wide complex tachycardia with right bundle branch block configuration. Diagnosis is made from the regular P-waves, which are dissociated with QRS complexes and indicated by red arrows. (b) Electrograms from an implantable cardiac device (ICD; in this case, a biventricular ICD). Top line (blue) shows nearfield electrical signal on atrial channel, middle line (magenta) shows nearfield electrical signal on right ventricular (RV) channel and bottom line (green) shows far field electrical signal between RV lead tip and left infra-clavicular generator, which can be thought of as a single lead ECG and is used as a discriminator channel to differentiate conducted (ie supraventricular tachycardia; SVT) from ventricular rhythms. On the left-hand side of the panel, there are more signals on the RV channel than on the atrial channel, which is seen to be dissociated from the signal on the ventricular channel, a pattern diagnostic of VT. The device delivers a high-energy shock (indicated) following which the VT terminates and there is a single conducted sinus beat (Vs) followed by resumption of biventricular pacing (BiV). The difference in morphology on the discriminator channel between both the spontaneous beat (Vs) and biventricular paced beats (BiV) can be seen. (c) Data from an electrophysiology study of a patient undergoing VT ablation for recurrent shocks in the context of a non-ischaemic cardiomyopathy. The image demonstrates a catheter entering the left ventricle (LV) through the mitral valve. Colours on the LV shell indicate the measured bipolar voltage and are used as an indication of the health (or otherwise) of the tissue, with purple corresponding to a voltage in the normal range, and red representing severely reduced voltage, found in diseased tissue. A 12-lead ECG of the VT being treated is shown on the right side of the panel. This patient was successfully treated for VT with an ablation procedure and remained free from further shocks during follow-up, which, at the time of writing, was 25 months.
Ventricular tachycardia

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Uncommonly, positively concordant QRS complexes can occur in the context of pre-excited SVT because of a ventricular insertion around the mitral annulus. Negatively concordant QRS complexes in the precordial leads are highly suggestive of VT. Careful examination of the 12-lead ECG during tachycardia is of value for determining the aetiology of the arrhythmia. Of the algorithms that have been published and validated, the most well known is the Brugada algorithm.18 Although these algorithms can be extremely helpful, all are subject to misclassification, and all can be ambiguous under some circumstances. Most of these have been validated almost exclusively in patients undergoing electrophysiology study with interpretation by cardiac electrophysiologists outside of the acute clinical setting in which the tachycardia presented.18 This is important because it gives an indication of why, despite the excellent reported sensitivity and specificity of these algorithms, clinicians in the real world continue to face significant diagnostic uncertainty when interpreting ECGs of WCT. Ultimately, it is likely to be most helpful to apply the principles that the algorithms are derived from rather than to learn the

Ventricular activation: when ventricular activation arises from the apex, the ventricular impulse will spread away from this focus, resulting in a QRS axis in the precordial leads between –90 and –180°, or ‘northwest’. This feature on 12-lead ECG is strongly suggestive of VT. Other QRS axis features that favour VT include left-axis deviation in RBBB morphology tachycardias and right axis deviation in LBBB tachycardias.18 The polarity of the lead aVR can also be helpful in differentiating the aetiology of a tachycardia. During conducted rhythms, the initial ventricular activation is septal and subsequent ventricular activation proceeds away from the lead aVR, resulting in a predominantly negative QRS complex in the lead aVR. Therefore, rhythms with an initial R wave in the lead aVR (indicating activation toward that lead) strongly suggest VT.9 Finally, a significant (>40°) change in axis between sinus rhythm and WCT suggests VT.9

Precordial lead concordance: both positive and negative concordance in the precordial leads are suggestive of VT. Concordance describes uniform polarity of the QRS complexes in the leads in question; for example, positive concordance describes QRS complexes that are positive throughout leads V1–V6. Uncommonly, positively concordant QRS complexes can occur in the context of pre-excited SVT because of a ventricular insertion around the mitral annulus. Negatively concordant QRS complexes in the precordial leads are highly suggestive of VT.

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the emerging wave meets excitable tissue, following which this wave can ‘re-enter’ the isthmus, and the process is repeated. As such, an ‘endless loop’ circuit is established that continues until it is interrupted. Myocardial fibrosis promotes the emergence of re-entry through the slowing of conduction as well as the electrical isolation of myocardial cells that can then behave as an electrically insulated isthmus through which re-entry can occur.

In addition to fibrosis, conditions associated with primary electrical abnormalities, in the absence of structural changes, have been associated with ventricular arrhythmias. These conditions comprise channelopathies, including catecholaminergic polymorphic VT (CPVT), and long-QT syndromes, as well as others. These are important because, unlike it often is in overt structural heart disease, the mechanism of VT associated with channelopathies is less likely to be re-entry, given that they are not typically associated with fibrosis. Management should be undertaken in specialist centres with experience in these conditions.

VT in structurally normal hearts

Although the presence of structural heart disease confers a higher likelihood of VT when diagnosing an undifferentiated WCT, up to 10% of VTs occur in the absence of an identified structural abnormality of the heart. The most common site of origin of these rhythms is the ventricular outflow tracts. Outflow tract VTs are associated with characteristically tall QRS complexes in the inferior leads. A variety of features on 12-lead ECG can help differentiate a right- from left-sided origin. An example of a VT arising from the RVOT is shown in Fig 3. Although often benign, presentation with algorithms themselves and be prepared to trial several algorithms in the case of difficulty interpreting a particular algorithm.
outflow tract arrhythmias, in the form of premature ventricular contractions (PVCs) or VT, should prompt careful exclusion of the possibility of an arrhythmogenic cardiomyopathy (AC). Outflow tract arrhythmias in structurally normal hearts are often not associated with haemodynamic compromise. In some cases, they might be associated with tachycardia-induced myopathy or sudden-cardiac death. Outflow tract arrhythmias are frequently amenable to successful catheter ablation, which is a first-line therapy in symptomatic patients and, therefore, patients with symptomatic PVCs/VT with an outflow tract appearance should be referred for electrophysiology consultation to discuss the merits of invasive electrophysiology study. Another distinct arrhythmia encountered in structurally normal hearts is that of fascicular VT. This arrhythmia results from re-entry through a circuit including the fascicles and Purkinje cells in the LV. These VTs typically have a right bundle branch block morphology and characteristic narrow QRS complexes with an axis dependent on the fascicle involved in the tachycardia. These are usually haemodynamically well-tolerated rhythms that can be treated with catheter ablation as a first line. When identified, patients with these arrhythmias should be referred to an electrophysiologist for consideration of catheter ablation as a curative option.

Management

Acute management of VT is focused on assessing and then stabilising haemodynamic compromise resulting from the arrhythmia. The advanced life support algorithms offer a helpful and practical approach to managing WCT, and appropriate parameters for determining haemodynamic compromise. As has been noted, in cases of diagnostic uncertainty, arrhythmias should be treated as ventricular in origin and the administration of medication, such as verapamil, avoided. The most commonly administered anti-arrhythmic drug is amiodarone because of its efficacy and relatively safe short-term safety profile. An important aspect of acute management that might be crucial to guide future arrhythmia management is the acquisition (and storage) of documentary 12-lead ECG evidence of the arrhythmia. The absence of these data can present significant challenges for ongoing care, including decision making around the indication for secondary prevention implantable cardioverter-defibrillators (ICDs). Ongoing management of patients with VT includes an assessment of the ongoing risk of sudden cardiac death (SCD). In general, ventricular arrhythmias that occur outside of the context of significant systemic disturbance or a reversible cause, and which are associated with haemodynamic instability, represent an indication for consideration of secondary prevention ICD. Shared decision making is an important aspect of this process and it is important to discuss both quality of life and longevity alongside an individual patient’s goals of care when discussing the option of an ICD. Suppression of recurrent arrhythmia is another challenge that is important for symptomatic reasons. This might be required as a result of recurrent symptoms of palpitations or in the context of recurrent therapies from an ICD, which can have a profound and debilitating impact on patient wellbeing. The options for suppressing arrhythmias include: treatment of the underlying myocardial disease with guideline-directed medical therapy or of structural abnormalities, such as valvular or coronary disease; the use of anti-arrhythmic drugs; invasive catheter ablation procedures; autonomic modulation, including the use of catheter-based continuous sympathetic blockade or surgical sympathectomy; and emerging non-invasive therapies for VT, such as cardiac stereotactic body radiation therapy (cSBRT).

Conclusion

The diagnosis of VT is made primarily using the 12-lead ECG. Diagnostic algorithms exist to differentiate VT from other WCTs, but uncertainty often persists despite appropriate application of these algorithms. In this scenario, a WCT should be treated as VT until proven otherwise. In the acute setting, acquisition of a 12-lead ECG of the arrhythmia is vital to guide future management. Ongoing management of VT comprises an assessment of the associated risk of sudden cardiac death, the use of ICDs in appropriate cases, and subsequently suppression of VT through the treatment of any underlying myocardial disease, the use of anti-arrhythmic drugs, catheter ablation, and other emerging therapies. Ongoing management of VT can be complex and ideally involves an electrophysiologist. Early referral to an electrophysiologist is also helpful to allow appropriate patient selection for invasive treatment for VT.

References


Address for correspondence: Dr John Whitaker, School of Biomedical Engineering and Imaging Sciences, King’s College London, St Thomas’s Hospital, London, SE1 7EH, UK. Email: john.whitaker@kcl.ac.uk