Supraventricular or ventricular tachycardia?

A 30-year-old Bosnian man presented to casualty with a 24-hour history of persistent palpitations. He had no significant past medical history and was not taking any non-prescription medication. On arrival in accident and emergency his pulse was 130/min and his blood pressure was 120/80 mmHg. He was initially administered intravenous (IV) adenosine up to a dose of 24 mg which had no effect on the tachycardia. IV lignocaine 200 mg failed to cardiovert the patient. Therefore, three attempts at DC cardioversion were made to a maximum of 360 J delivered anteroposteriorly. This was ineffective. What is the diagnosis on the electrocardiogram (ECG) in *Figure 1* and what drug would successfully cardiovert this patient?

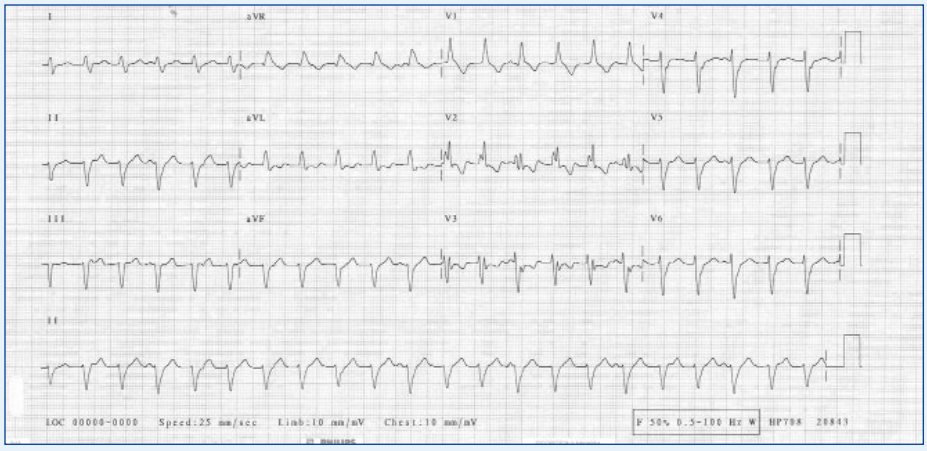


Figure 1. The electrocardiogram on presentation.

Discussion

The ECG demonstrates a broad complex tachycardia with a northwest axis (264°). The key diagnostic feature in this ECG is that of ventriculoatrial (VA) dissociation – there are clear P waves marching through the QRS complexes at rate of 72/min (*Figure 2*). The patient also had obvious canon waves on clinical examination as one would expect with VA dissociation. Therefore, the diagnosis is ventricular tachycardia (VT). However, one can be more specific. The QRS width is relatively narrow with a right bundle–branch block (RBBB) morphology and left superior axis deviation. These are typical features of fascicular tachycardia.

Fascicular tachycardia occurs in young patients (between 15 and 40 years of age) with a structurally normal heart. Approximately 60–80% of patients are male. The tachycardia may be incessant as appeared in this presentation and if prolonged over months can lead to a reversible tachycardia-induced cardiomyopathy. Sudden cardiac death is not usually associated with this form of VT.

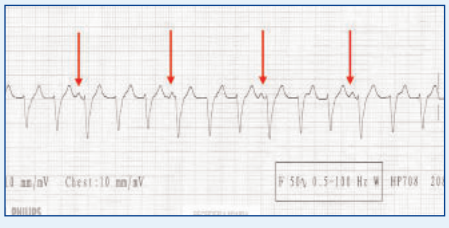
The tachycardia has a RBBB, left superior axis morphology in 90–95% of cases, indicating that the exit site of the re-entrant circuit is in the region of the left posterior fascicle, near the inferoposterior left ventricular septum. The remainder of patients with fascicular VT have a RBBB, right inferior axis morphology as the circuit exits in the region of the left anterior hemifascicle. Typically, they respond to adenosine but are exquisitely sensitive to verapamil. In this case IV verapamil 5 mg cardioverted the patient on the end of the needle.

Dr Pier Lambiase *is Clinical Lecturer in Electrophysiology, Cardiology Department, The Heart Hospital, University College London, London W1M 8PH*

Treatment of fascicular VT

Fascicular VT is usually unresponsive to adenosine or beta blockade. Patients can be cardioverted with IV verapamil. There are limited data describing the long-term efficacy of oral verapamil, although this usually improve symptoms in patients who are moderately symptomatic. It has limited efficacy in those who are significantly debilitated by palpitations. Radiofrequency ablation has excellent results in this population, offering a 90% cure rate. Therefore, ablation is offered to patients with fascicular VT associated with presyncope or syncope, those with recurrent sustained tachycardia or patients intolerant or resistant to antidysrhythmic medication.

Figure 2. Atrioventricular dissociation. Arrows indicate P waves dissociated from the QRS complexes.



ECG features of VT

This case is an interesting example of normal heart VT. The diagnosis of VT is not always easy with the principal differential lying between supraventricular tachycardia (SVT) with aberrancy (bundle–branch block) and antidromic accessory pathway-mediated tachycardia. VT is seen in over 70% of patients before sudden death. There are many excellent reviews explaining how to differentiate between SVT and VT (Sager and Bhandari, 1991; Wellens, 2001). The key elements which help determine the diagnosis include the history, presence of structural heart disease and specific features on surface ECG. In this brief discussion the pertinent features to aid diagnosis will be highlighted.

History

A careful history often yields the diagnosis. A monomorphic broad complex tachycardia in a patient with coronary disease has an over 90% probability of being VT. The medication history is extremely important, principally looking for agents which prolong QT interval or recognized drug combinations with QT prolonging effects. A history of structural heart disease such as dilated cardiomyopathy raises the possibility of bundle–branch re-entry – a left bundle–branch block (LBBB) morphology of VT which is amenable to ablation. Patients with structurally normal hearts have a

greater propensity to develop either fascicular VT or VT arising from the right ventricular outflow tract. The latter have a LBBB morphology and an inferiorly directed axis (R waves in leads II, III and aVF).

ECG diagnostic features

Atrioventricular relationship: Careful inspection of the ECG for P waves at a slower rate marching through the QRS complexes will enable atrioventricular (AV) dissociation to be established. Occasionally the AV node can conduct rapidly in the retrograde direction and a 1:1 VA relationship occurs, making diagnosis difficult. The presence of fusion and capture beats are cardinal features of VT.

Axis: Extreme axis deviation is a further important feature. If the tachycardia has a LBBB morphology with right axis deviation or RBBB with left axis deviation, then VT is likely as there is extremely abnormal activation of the ventricle.

QRS width: The width of the QRS is a helpful guide to the probability of VT. A RBBB morphology with QRS >140 msec and LBBB duration >160 msec increases the probability of VT. This simply reflects the fact that the VT circuit is progressing through slowly conducting ventricular myocardium as opposed to specialized conduction tissue. VT circuits localized to the interventricular septum have a narrower QRS morphology. Since fascicular VT is a re-entrant rhythm involving the distal Purkinje fibres it also has a comparatively narrow QRS.

Chest lead: Chest lead concordance with R waves across leads V1–V6 or Q waves in leads V1–V6 is diagnostic. In the case of positive chest lead concordance with a structurally normal heart, one should also consider the possibility of a left-sided accessory pathway-mediated SVT in which case delta waves will be evident.

QRS morphology: Further clues to the diagnosis of VT lie in the morphology of the complex in leads V1 and V6. The right ventricle does not participate in initial ventricular depolarization during a normally conducted beat and thus the initial portion of the QRS complex is not affected by RBBB aberration. QRS patterns in V1 consistent with aberration therefore include rSr', rR, rsr' or rSR. However, if there is a dominant R, i.e. Rsr' pattern in V1 or a qR pattern, VT is the most likely diagnosis (*Figure 3*). Similarly a qR pattern in V6 with LBBB indicates VT.

Conclusions

The diagnosis of VT should start with careful examination of the rhythm strip to identify the AV relationship – if this can be determined with certainty then the diagnosis often can be made confidently. The identification of the above features been shown to significantly improve a physician's accuracy in diagnosing these frequently life-threatening arrhythmias. BJHM

Conflict of interest: none.

Sager PT, Bhandari AK (1991) Wide complex tachycardias, differential diagnosis and management. *Cardiol Clin* **9:** 595–618

Wellens HJ (2001) Electrophysiology: Ventricular tachycardia: diagnosis of broad QRS complex tachycardia. *Heart* **86:** 579–85

Further reading

Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou PJ (1988) Wide QRS complex tachycardia. Reappraisal of a common clinical problem. *Ann Intern Med* **109:** 905–12

Antunes E, Brugada J, Steurer G, Andries E, Brugada P (1994) The differential diagnosis of a regular tachycardia with a wide QRS complex on the 12-lead ECG: ventricular tachycardia, supraventricular tachycardia with aberrant intraventricular conduction, and supraventricular tachycardia with anterograde conduction over an accessory pathway. *Pacing Clin Electrophysiol* **17:** 1515–24

Baerman JM, Morady F, DiCario LA, de Buitleir M (1987) Differentiation of ventricular tachycardia from supraventricular tachycardia with aberration: value of the clinical history. *Ann Emerg Med* **16:** 40–3

Brugada P, Brugada J, Mont L, Smeets J, Andries EW (1991) A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* **83:** 1649–59

Clayton R, Murray R (1995) Objective features of the surface electrocardiogram during ventricular tachyarrhythmias. *Eur Heart J* **16:** 1115–19

Griffith MJ, Garrett CJ, Mounsey P et al (1994) Ventricular tachycardia as default diagnosis in broad complex tachycardia. *Lancet* **343:** 386–8

Hayes JJ, Stewart RB, Greene L et al (1991) Narrow QRS ventricular tachycardia. *Ann Intern Med* **114:** 460

Lau EW, Ng GA (2002) The reliable electrocardiographic diagnosis of regular broad complex tachycardia: a holy grail that will forever elude the clinician's grasp? *Pacing Clin Electrophysiol* **25**(12): 1756–61

Levitt MA (1988) Supraventricular tachycardia with aberrant conduction versus ventricular tachycardia: differentiation and diagnosis. *Am J Emerg Med* **6:** 273–7

Lobban JH, Schmidt SB, Rhodes LA, Jain AC (1994) Differential diagnosis of wide QRS tachycardias. *W V Med J* **90:** 232–4

Michelson EA, Brady WJ (2002) Emergency physician interpretation of the electrocardiogram (editorial). *Acad Emerg Med* **9:** 317–19

Morady F, Baerman JM, DiCarlo LA et al (1985) A prevalent misconception regarding wide complex tachycardias. *JAMA* **254:** 2790–2

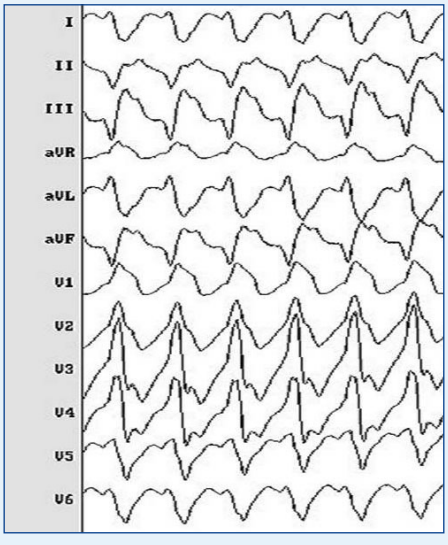
Steinman RT, Herrera C, Schluger CD et al (1989) Wide QRS tachycardia in the conscious adult: ventricular tachycardia is the most frequent cause. *JAMA* **261:** 1013–16

Steurer G, Gursoy S, Frey B et al (1994) The differential diagnosis on the electrocardiogram between ventricular tachycardia and preexcited tachycardia. *Clin Cardiol* **17:** 306–8

Stewart RB, Bardy GH, Greene HL (1986) Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med* **104:** 766–7

Tchou P, Young P, Mahmud R, Denker S, Jazayeri M, Akhtar M (1988) Useful clinical criteria for the diagnosis of ventricular tachycardia. *Am J Med* **84:** 53–6

Figure 3. Right bundle–branch block ventricular tachycardia (VT) morphology with dominant Rsr pattern in V1 and rS waves in V6 consistent with VT confirmed at electrophysiological study. Note the very broad QRS complexes which were over 160 msec in this case.



KEY POINTS

- The principal features of ventricular tachycardia are:
  - Ventriculoatrial dissociation.
  - Extreme axis deviation.
  - Chest lead concordance – positive or negative QRS complexes in all ventricular leads.
  - Fusion beats and capture beats.
  - Broad right bundle–branch block with left axis deviation or left bundle–branch block with right axis deviation.
- Any one of these features is diagnostic of ventricular tachycardia.