ECG Quiz 41

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

The ECG shows a wide complex, regular QRS tachycardia. The rate of the tachycardia is 264bpm. The QRS complexes are wide (140ms). The QRS axis of the tachycardia is +90 degrees. There is positive concordance in the chest leads.

Ventricular tachycardia (VT) is the default diagnosis in any wide complex, regular QRS tachycardia. Without further analysis, the diagnosis of VT will be correct 80% of the time. If a history of a prior myocardial infarction is obtained, the likelihood of VT increases to 90%. Other differential diagnoses of a wide complex, regular tachycardia include a supraventricular tachycardia (SVT) with typical right or left bundle branch block (BBB), a pacemaker tachycardia or a pre-excited tachycardia.

The next step in the analysis of the ECG is to exclude a SVT with BBB which accounts for 10 - 15% of wide complex tachycardias. In contrast to narrow complex tachycardias, identifying P-waves is not as useful as a 1:1 P:QRS relationship can exist in both VT (1:1 retrograde VA conduction) and SVT. While evidence of AV dissociation is specific for VT, this is not easily seen in rapid VTs. The clinician should compare the QRS morphology in V1 and V6 to typical right and left BBB. In this case, the QRS morphology in V1 and V6 does not resemble typical right BBB (Figure 1). SVT with RBBB can therefore be excluded. In V1, the initial part of the QRS complex is slow with an Rsr'pattern. In V6, there is no septal Q-wave and the R-wave has a slow upstroke. In typical RBBB, the initial part of the QRS is rapid because left ventricular (LV) activation is normal. This causes a rapid initial upstroke (r) followed by delayed right ventricular (RV) activation (R'). This is responsible for the typical rSR' pattern in V1. In V6, the septal Q-wave with a rapid R upstroke is present because LV activation is normal with a small slurred S-wave.

A pacemaker tachycardia can cause a wide complex, regular tachycardia. This can occur with dual chamber pacemakers when there is 1:1 tracking of P-waves or with single chamber pacemakers when the lower rate is increased by the sensor during activity. RV apical pacing causes an atypical LBBB morphology in V1 with left axis deviation. A pacemaker tachycardia is excluded as there are no pacing spikes visible (the absence of pacing spikes per se does not exclude a pacemaker tachycardia in digital ECG recording systems) and the QRS morphology is not consistent with RV apical pacing. Tracking of the P-wave does not occur at P-wave rates of 264bpm (mode switch rates to VVIR are typically around 180bpm) and the sensor rate in single chamber pacemakers is usually programmed around 120 - 150bpm.

Pre-excited tachycardias are uncommon tachycardias (1 - 5% of wide complex tachycardias) and can occur in patients who have an accessory pathway (Wolff-Parkinson-White syndrome). When both the accessory pathway and AV node are part of the tachycardia circuit in a wide complex tachycardia, the

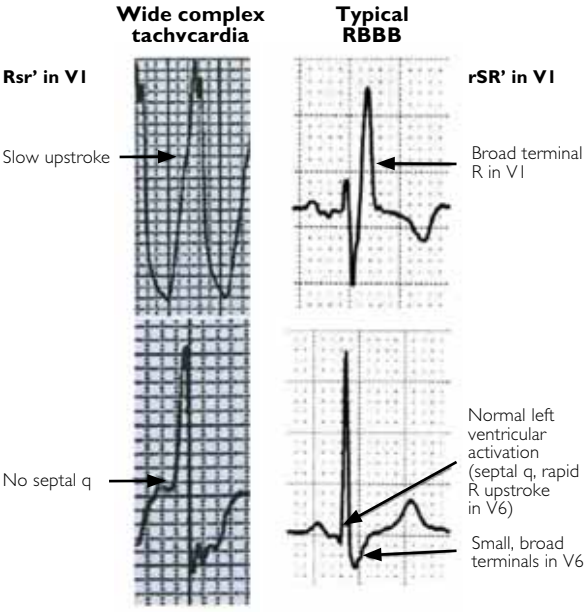

FIGURE 1: Comparison of QRS morphologies (V1 and V6) of the wide complex tachycardia with typical RBBB.

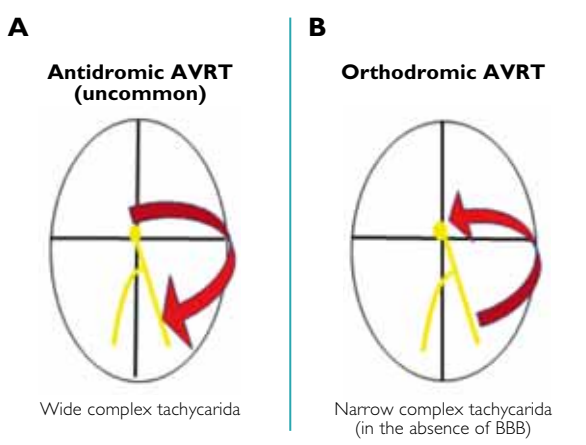

FIGURE 2: Comparison of antidromic AVRT with orthodromic AVRT. In antidromic AVRT, the accessory pathway is the antegrade limb and the AV node is the retrograde limb of the tachycardia circuit. In orthodromic AVRT, the AV node is the antegrade limb and the accessory pathway is the retrograde limb of the tachycardia circuit.

tachycardia is called antidromic atrio-ventricular re-entrant tachycardia (AVRT) where the accessory pathway is the antegrade limb and the AV node the retrograde limb of the circuit (Figure 2a). An accessory pathway can also be a passive bystander (not part of the tachycardia circuit) in any SVT (e.g. sinus tachycardia, atrial tachycardia, AVNRT). Conduction of the SVT can occur over the accessory pathway and/or the AV node in the antegrade direction. Orthodromic AVRT produces a narrow complex regular tachycardia in the absence of BBB because the antegrade limb is the AV node and the accessory pathway is the retrograde limb of the circuit (Figure 2b).

Differentiation between VT and a pre-excited tachycardia is challenging because ventricular activation begins outside the normal conduction system in both tachycardias. In antidromic AVRT, VT can never be excluded on the ECG alone because a VT that has its origin near the ventricular insertion site of the accessory pathway can cause a similar QRS morphology. Specific signs for VT need to be looked for:

- AV dissociation (fusion beats, capture beats, dissociated P-waves, more QRS complexes than P-waves) confirms VT and excludes a pre-excited tachycardia.
- Predominantly negative QRS complexes in V4 - V6 is highly specific for VT. Pre-excited tachycardias never produce negative QRS complexes in these apical leads in the absence of incorrect lead placement. Accessory pathways insert into the base of the LV along the mitral annulus and never at the apex. Therefore, LV activation never proceeds away from the apex (V4 - V6) and negative QRS complexes in these leads cannot occur.
- QR complexes in one or more leads (V2 - V6) also favours VT over a pre-excited tachycardia.

Although these ECG features are highly specific for VT, their absence does not exclude it. In this case, none of these features are present, so both pre-excited tachycardia and VT remain possibilities. Old or subsequent ECGs showing an identical baseline pre-excitation pattern in sinus rhythm compared to the wide complex tachycardia would strongly favour a pre-excited tachycardia.

The correct answer is therefore e) a and d are possibilities.

To distinguish between a) and d) a clinical history may be helpful. Structural heart disease or a history of myocardial

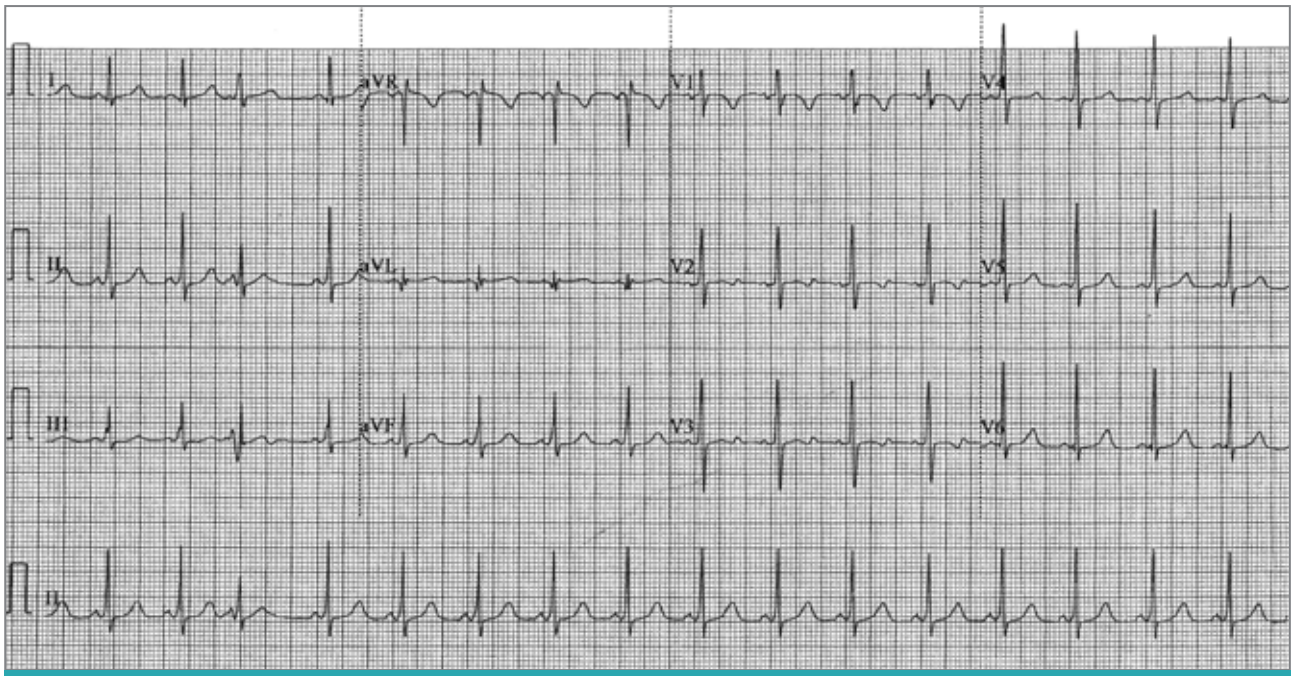

FIGURE 3: ECG shows sinus rhythm with evidence of pre-excitation consistent with a left lateral accessory pathway. Delta waves are best seen in II, III and aVF and V1.

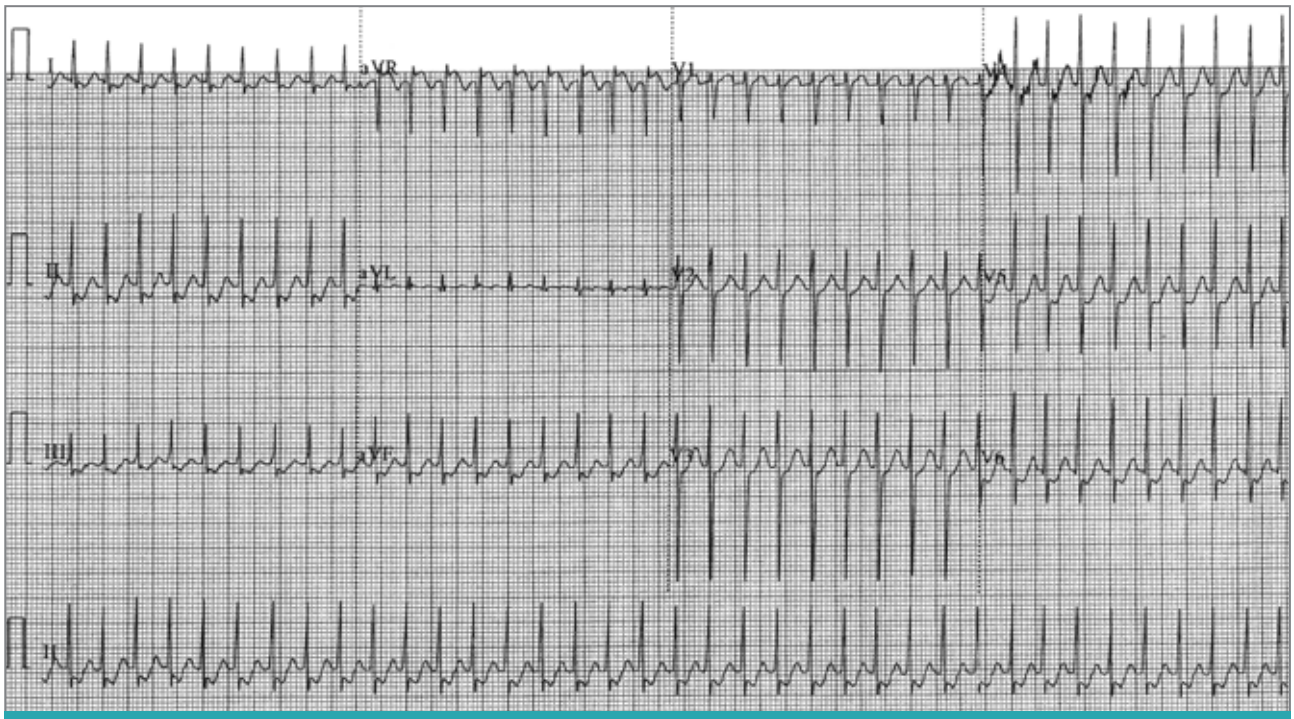

FIGURE 4: ECG shows a narrow complex, regular tachycardia. Retrograde P-waves are seen in the ST segment in the inferior leads consistent with an orthodromic atrio-ventricular re-entrant tachycardia (AVRT).

infarction strongly favours VT as a diagnosis. Signs of AV dissociation on clinical examination (cannon A-waves) can be sought to confirm VT but can be very difficult at rapid ventricular rates and prominent A-waves can be seen in some SVTs.

Carotid sinus massage can be a useful diagnostic manoeuvre. VTs are unaffected by carotid sinus massage although this may slow the atrial rate or block VA conduction and can help expose AV dissociation. If the tachycardia terminates, an AV nodal dependent tachycardia (antidromic AVRT) is confirmed. The ventricular rate can transiently slow during an AT or AFL because of AV block which can reveal underlying P-waves. This is, however, unlikely in a pre-excited SVT, as the accessory pathway is not affected by vagal stimulation. Neither verapamil nor adenosine should ever be given to a wide complex tachycardia because they can cause severe haemodynamic deterioration in VT and can provoke cardiac arrest or haemodymamic collapse.

In this case, carotid sinus massage had no effect and the patient was cardioverted back into sinus rhythm. The post cardioversion ECG is shown in Figure 3. A short PR interval and delta waves are best seen in leads II, III, aVF with a dominant R-wave in V1 consistent with a left lateral or anterior-lateral accessory pathway. This pre-excitation pattern is similar to the wide complex tachycardia confirming a pre-excited tachycardia. The most likely diagnosis is an antidromic AVRT although an AT or AFL with 1:1 conduction over a bystander accessory pathway is a remote possibility.

Interestingly, a narrow complex regular tachycardia, consistent with orthodromic AVRT, was also documented in this patient (Figure 4).

SUMMARY

Pre-excited tachycardia is an uncommon cause of a wide complex, regular QRS tachycardia.

Pre-excited tachycardia can be indistinguishable from ventricular tachycardia on an ECG.

The clinical background of the patient and the resting ECG in sinus rhythm provides important additional information.

REFERENCE

1. Issa, Miller and Zipes. Clinical Arrhythmology and Electrophysiology. Elselvier Inc. 2012.

Conflict of interest: none declared.

ECG and **QUESTION** on page 34