ECG Quiz 72

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
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ANSWER

1. OVERVIEW OF THE ECG

This is a fast (250/min), regular, wide QRS (190 ms) tachycardia. Therefore, the default diagnosis is VT (Table I), even in a young boy. However, the other options must be considered.

TABLE I: Differential diagnosis of regular, wide QRS tachycardias

- Ventricular tachycardia
- SVT with bundle branch block
- SVT with non-specific intraventricular conduction delay
- Antidromic AV re-entry tachycardia
- Pre-excited SVT
- Paced rhythm

AV: atrioventricular, SVT: supraventricular tachycardia, WPW: Wolff–Parkinson–White

More detailed analysis of the ECG

The key to differentiating VT from SVT with bundle branch block lies in the QRS morphology in the chest leads. V1–V3 are negative, consistent with LBBB. However, the onset of the QRS to the nadir in V1–V2 is 115 ms (Figure 1). This excludes typical

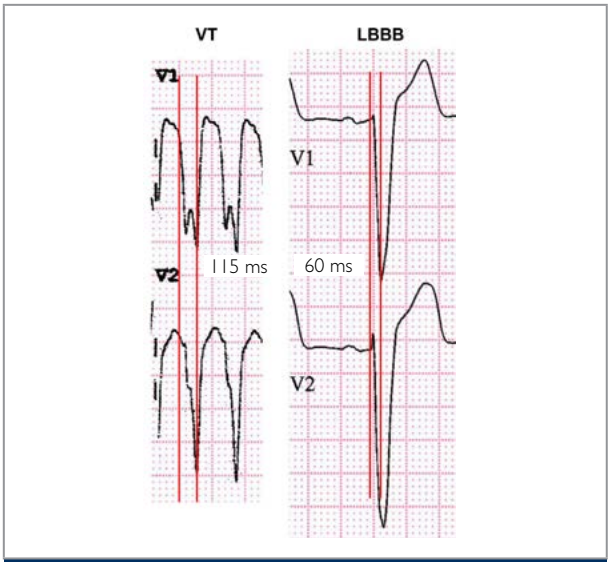

FIGURE 1: The marked delay in the downstroke of the QRS in this tachycardia (115 ms) excludes LBBB (< 70 ms).

LBBB: left bundle branch block, VT: ventricular tachycardia.

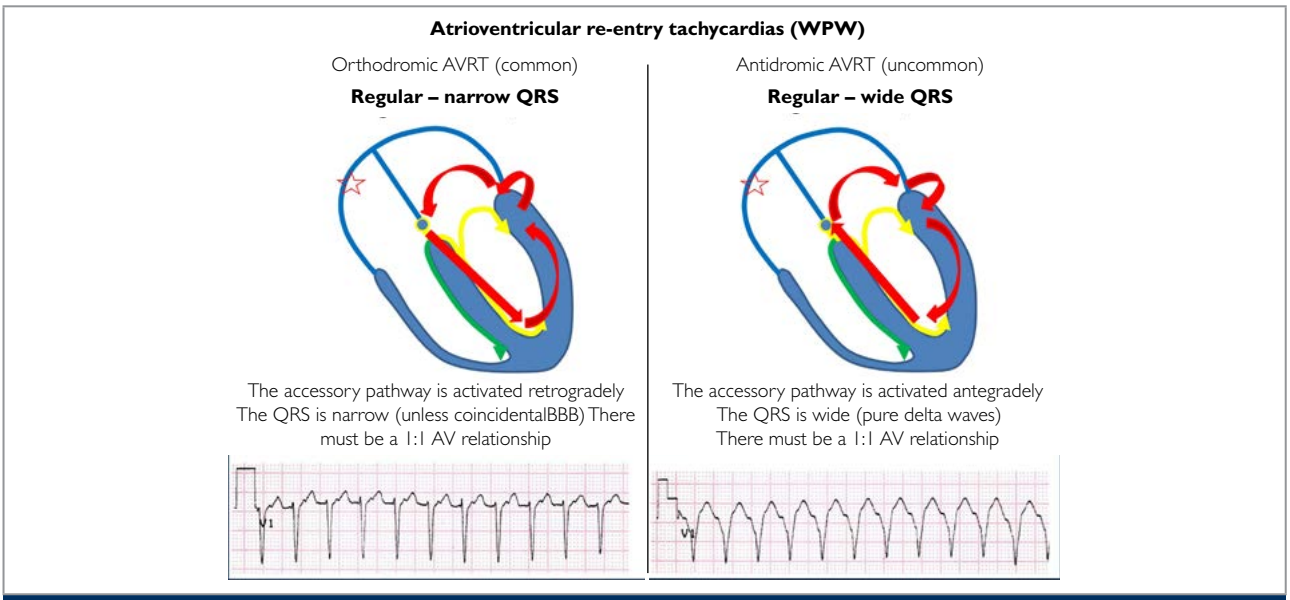

FIGURE 2: Orthodromic AVRT (wherein the accessory pathway functions as the retrograde limb and the normal conduction system as the antegrade limb of the re-entry circuit) is by far the most common tachycardia in patients with WPW syndrome. Antidromic AVRT (wherein the accessory pathway forms the antegrade limb) constitutes < 10% of clinical tachycardias in WPW syndrome. The QRS is wide because the accessory pathway inserts into the ventricular muscle at the base of the heart and conducts slowly cell-to-cell until it reaches the His-Purkinje system and is conducted retrogradely to the atrium. In both cases, the atria are an obligatory part of the circuit.

AV: atrioventricular, AVRT: atrioventricular re-entry tachycardia, BBB: bundle branch block, WPW: Wolff–Parkinson–White.

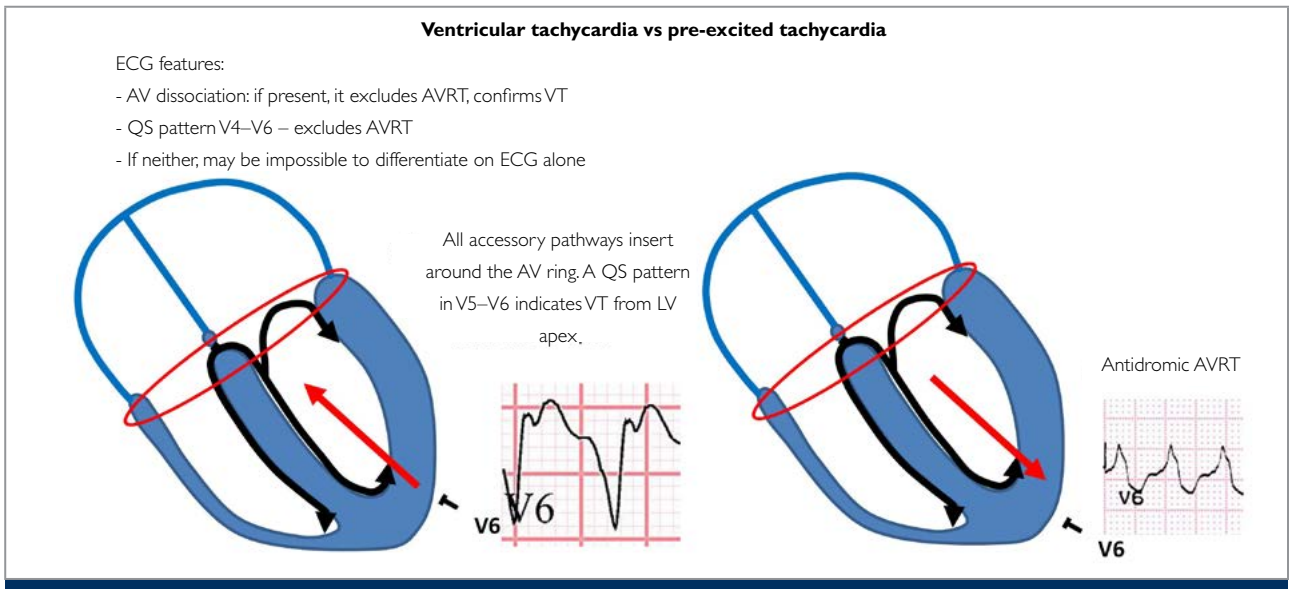

FIGURE 3: The differentiation of pre-excited tachycardia from VT depends on the QRS morphology and the presence or absence of AV dissociation, as above.
AV: atrioventricular, AVRT: atrioventricular re-entry tachycardia, ECG: electrocardiogram, LV: left ventricular, VT: ventricular tachycardia.

LBBB, which should not exceed 70 ms. Therefore, a diagnosis of SVT with LBBB is not tenable. An SVT with non-specific intraventricular conduction delay is a remote possibility. This only occurs in patients with severe left ventricular (LV) dysfunction due to extensive fibrosis. No clinical details or prior sinus rhythm ECGs (which would show the conduction delay) were given, but it would be very unusual at this age.

Antidromic AVRT is a serious consideration. It is the least common mechanism of tachycardia in patients with WPW syndrome, in which the accessory pathway forms the antegrade limb of the circuit. This results in slow cell-to-cell conduction (pure delta waves) and retrograde activation of the atrium via the normal conduction system (Figure 2). The vast majority of paroxysmal tachycardias in WPW patients are orthodromic AVRT, wherein the accessory pathway forms the retrograde limb, and the atrioventricular (AV) node forms the antegrade limb of the circuit. Therefore, QRS complexes are narrow, unless there is an incidental bundle branch block.

Differentiating antidromic AVRT from VT on the QRS morphology alone can be challenging. While some QRS morphologies, like negative QS waves in V4–V6, are specific for VT (Figure 3, all accessory pathways insert around the AV ring so the delta wave is always positive in V4–V6), this pattern is not present here, and antidromic AVRT cannot be excluded on QRS morphology alone. Looking for signs of AV dissociation now becomes crucial (Figure 3).

In AVRT (orthodromic and antidromic), there is an obligatory 1:1 ventriculo-atrial relationship. Tachycardia cannot occur in the presence of AV dissociation. Careful inspection of the SII rhythm strip reveals occasional dissociated P waves distorting the ST segments (Figure 4 arrows). This clinches the diagnosis of VT. Be aware that AV dissociation is not present or not visible in most VT ECGs, particularly at a rate this fast. A 1:1 retrograde VA conduction is common in VT and does not prove SVT diagnosis.

Therefore, the correct answer is (a): Sustained monomorphic VT.

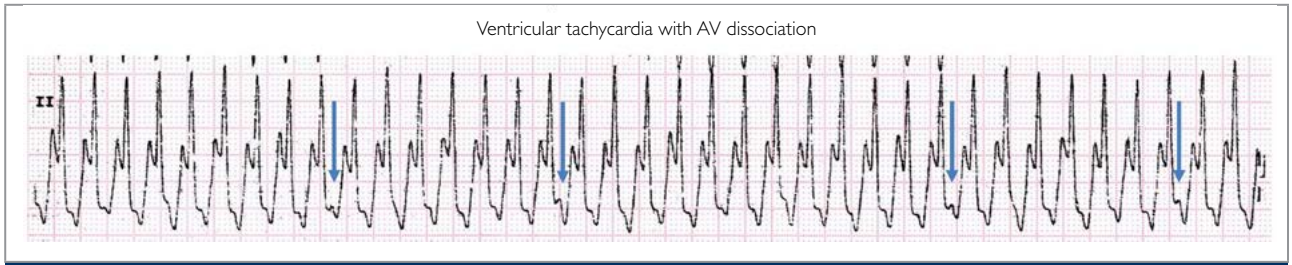

FIGURE 4: Dissociated P waves (arrows) are visible in the rhythm strip as high-frequency distortions of the ST segment. This excludes antidromic atrioventricular re-entry tachycardia and clinches the diagnosis of ventricular tachycardia.
AV: atrioventricular.

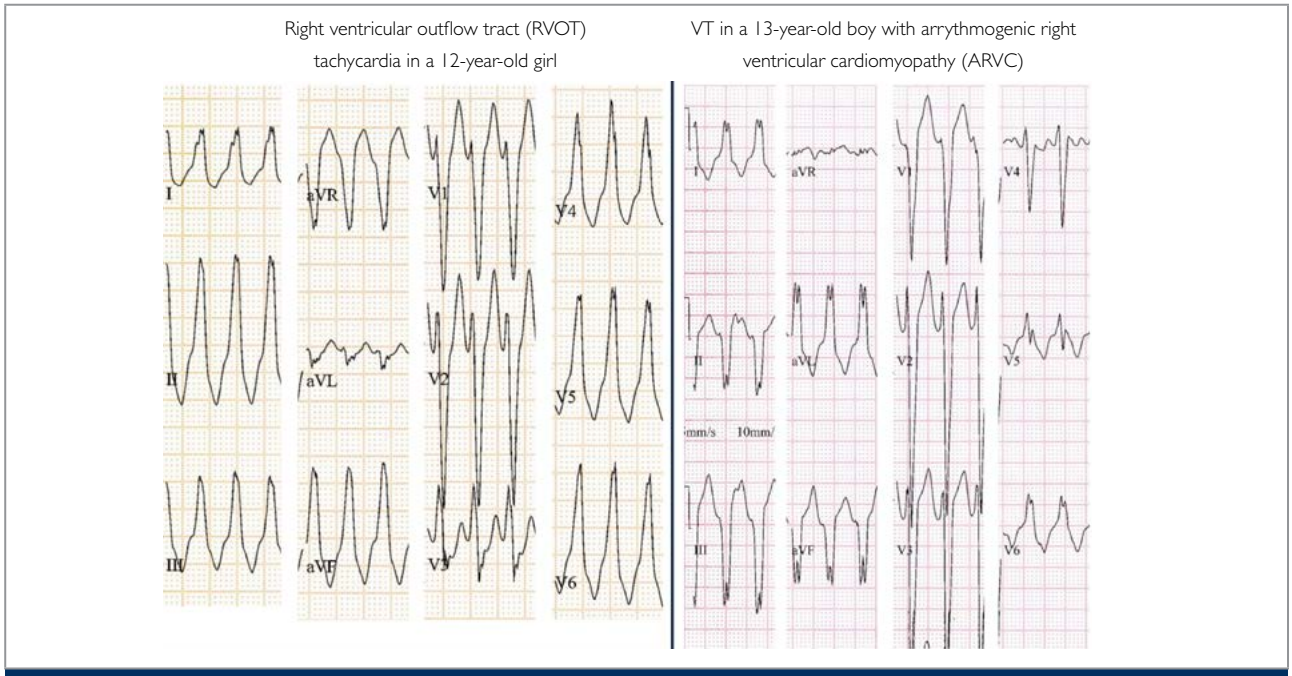

FIGURE 5: Comparison of proven idiopathic RVOT tachycardia with VT caused by ARVC. The RVOT tachycardia has an inferior QRS axis, with only a small amount of notching and transition by V3. The ARVC VT has a left axis, indicating origin from the right ventricular body, notching in most QRS complexes, and a transition after V5.
ARVC: arrhythmogenic right ventricular cardiomyopathy, RVOT: right ventricular outflow tract, VT: ventricular tachycardia.

2. WHAT IS THE MOST LIKELY CLINICAL DIAGNOSIS?

VT in children and young adults has various possible causes. The ECG in VT is not specific to the cause – clinical information and further cardiac imaging are now needed. Nonetheless, there are some helpful features. This tachycardia has a pseudo-LBBB pattern, suggesting a likely RV origin. The axis is about +60 °, suggesting it comes from the RVOT. Is it, therefore, idiopathic RVOT tachycardia, a condition of young people? Contrary to this diagnosis is the very wide QRS with marked notching (best seen in V1), indicating inhomogeneous ventricular activation. This is suggestive of scar-related re-entry, which is not a feature of RVOT tachycardia (Figure 5).[1]

ARVC is an important cause of VT arising in the right ventricle. However, the VT site is usually from the inflow tract or body, which would produce a superior QRS axis. Rarely, VT can also arise from the outflow tract, mimicking this ECG. The notched QRS complexes (best seen in the limb leads) would be compatible with this diagnosis and are probably related to extensive areas of fibrosis and fatty infiltration, which form the substrate for re-entry (Figure 5).

Non-sustained (< 30 seconds) VT is common in HOCM, but sustained monomorphic VT is less so. One mechanism for sustained VT in HOCM is an LV apical aneurysm.[2] Since HOCM predominantly involves the left ventricle, one would expect the VT to have a pseudo-right bundle branch block (RBBB) pattern

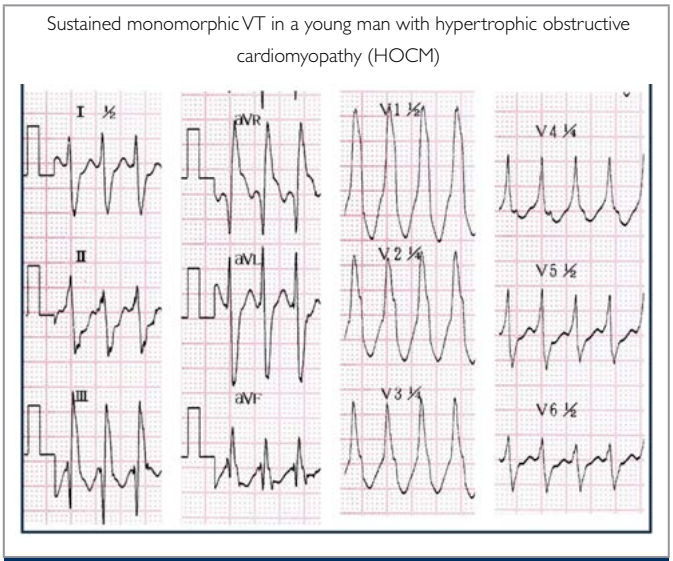

FIGURE 6: This VT has a pseudo-RBBB morphology, indicating an origin from the left ventricle.
RBBB: right bundle branch block, VT: ventricular tachycardia.

(Figure 6), unlike the ECG in this quiz. Consequently, HOCM is unlikely.

VT may complicate TOF, usually after surgical repair, which involves a right ventriculotomy in the RVOT and resection of muscle to relieve obstruction. The risk factors for VT include late repair, time after surgery, a QRS duration ≥ 180 ms,

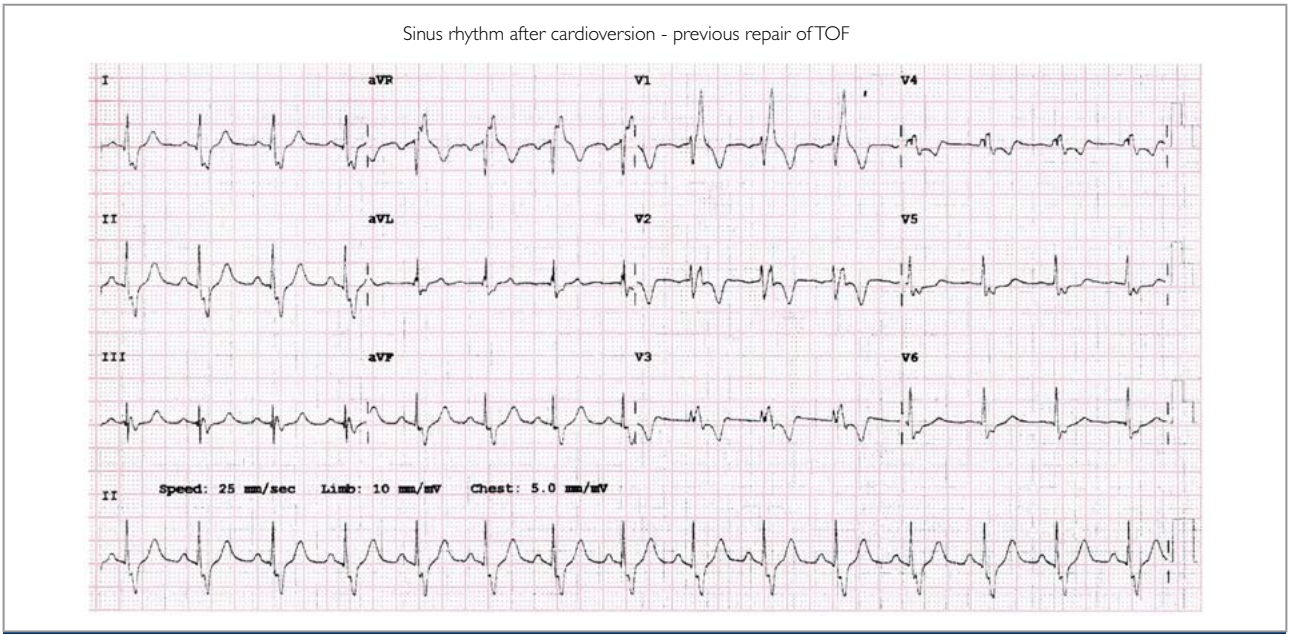

FIGURE 7: The sinus rhythm electrocardiogram is typical of post-repair of TOF. Right bundle branch block is inevitable if there is a ventriculotomy in the right ventricular outflow tract.
TOF: tetralogy of Fallot.

ventricular dysfunction, and atrial tachyarrhythmias.[3,4] The mechanism is re-entry around the surgical scar.[5] While most patients with VT related to surgery are teenagers or young adults, it is likely that this child had surgery as an infant (date unknown), as the ECG with VT was recorded in 2017. The post-cardioversion ECG (Figure 7) is typical of previously repaired TOF. There is complete RBBB with a QRS duration of 180 ms.

The most likely clinical diagnosis is (d): TOF.

Note that in the absence of clinical information, clinical imaging, or a post-conversion ECG, ARVC is also a possible diagnosis with an uncommon outflow tract VT.

LESSONS AND CONCLUSIONS

- VT is the most likely cause of a regular, wide QRS tachycardia, even in children.
- AV dissociation, if visible, is diagnostic.
- The QRS morphology is useful to reveal the potential origin site of VT and may point to the cause.
- While surgical repair of TOF is essential for survival and quality of life, it is not a cure, so patients need to be followed up for life.
- Patients with post-operative TOF are at risk of developing VT and sudden death due to scar-related re-entry.

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