ECG Quiz 52

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

ANSWER

OVERVIEW OF THE ECG

This is a wide complex, regular tachycardia with a ventricular rate of 234bpm. The QRS complexes are wide (about 150ms) and monomorphic. The QRS axis is -90 degrees. The differential diagnosis of a wide complex, regular tachycardia includes the usual suspects: (1) monomorphic ventricular tachycardia (VT); (2) supraventricular tachycardia (SVT) with a bundle branch block; (3) SVT with a non-specific intraventricular conduction abnormality; (4) pre-excited tachycardia; and (5) pacemaker tachycardia.

MORE DETAILED ANALYSIS OF THE ECG

The first step in the analysis of this ECG requires a detailed analysis of the morphology of the QRS complexes in V1 and V6. The QRS complex in V1 starts with a small initial q wave, followed by a r wave and then a delayed R'. The onset of the QRS is difficult to identify in V1. In V2, the onset of the QRS begins with a distinct q wave, which is at least 30ms wide and 1.5mm deep. The QRS complex in V6 starts with a r wave (30 to 40ms wide), followed by a deep S wave. These complexes do not resemble a typical right bundle branch block (RBBB) pattern. SVT with RBBB and left anterior fascicular block can therefore be excluded.

Differentiating VT from a pre-excited tachycardia can be challenging, as ventricular activation begins outside the normal intraventricular conduction system in both tachycardias. A useful next step is to try and identify specific features for VT, which include signs of AV dissociation (dissociated P waves, fusion beats or capture beats). There are also specific QRS morphologies, which cannot occur with a pre-excited tachycardia – which include QS complexes in V4 to V6 (as depolarisation of the left ventricle starts at the base of the heart at the site where the accessory pathway inserts into the ventricle and never at the apex). Other QRS morphologies that cannot occur with a pre-excited tachycardia, include qR complexes in any lead from V2 to V6.[1] In this ECG, P waves are not easily seen, except in V3. In V3, P waves can be seen distorting the ST segments with each QRS complex (no dissociated P waves are seen), and rS waves are seen in V4 to V6. However, qR complexes are seen in V1 to V3, which cannot occur with a pre-excited tachycardia. A pre-excited tachycardia can therefore be excluded with reasonable certainty based on these criteria.

A SVT with an intraventricular conduction abnormality is a possibility, but is very unlikely. An intraventricular conduction abnormality with marked left axis deviation would be most unusual in a 17-year-old man with a structurally normal heart. The absence of an intraventricular conduction abnormality in sinus rhythm would be useful to exclude the diagnosis. A pacemaker tachycardia is excluded, as QRS complexes do not resemble a paced rhythm and a pacing rate of 234bpm is highly unlikely.

VT is therefore the diagnosis. The dominant R wave in V1 indicates that depolarisation is towards the right ventricular lead V1 from the left ventricle – indicating a left ventricular origin.

The correct answer is: (4) Ventricular tachycardia arising from the left ventricle

The axis is -90 degrees, which suggests an origin near the left posterior inferior septal region. In a young man with a structurally normal heart, the most likely diagnosis is an idiopathic left posterior fascicular VT.

DISCUSSION

Idiopathic fascicular left ventricular tachycardia accounts for 10% - 15% of idiopathic ventricular tachycardias (VTs).[2] Left posterior fascicular ventricular tachycardia (LPFVT) accounts for 90% - 95% of fascicular VTs. LPFVT typically presents in young adults (15 - 40 years) and males are predominantly affected, and may result in an underlying tachycardia-induced cardiomyopathy. This tachycardia is calcium-dependent and is not cAMP-medicated triggered activity, as in the case of the more common idiopathic outflow tract VTs.

Electrocardiographically, LPFVT is characterised by an atypical right bundle branch block (RBBB) pattern with left axis deviation, as the exit site of the VT is located in the left ventricular inferior posterior septum. The re-entrant VT circuit consists of an antegrade limb (abnormal Purkinje tissue, which has slow decremental conduction that is verapamil-sensitive) and a retrograde limb that consists of part of the left posterior fascicle (see Figure 1). A much rarer form of idiopathic LV VT is

left anterior fascicular VT, which has an atypical RBBB morphology with right axis deviation, and involves the left anterior fascicle.

LPFVT is frequently misdiagnosed as a supraventricular tachycardia (SVT) with RBBB and left anterior fascicular block, because they both occur in young patients with normal hearts, are well tolerated clinically, and are verapamil-sensitive. Although the presence of AV dissociation will confirm the diagnosis of LPFVT, AV dissociation is not always seen, as in this case.

In a recent study that compared ECG characteristics of LPFVT with SVT with RBBB and left anterior fascicular block, the authors identified 4 features that suggested LPFVT.[3] These include: (1) atypical RBBB morphology in V1; (2) QRS width <=140ms, as part of the ventricle is activated via the normal His Purkinje system; (3) V6 R/S ratio<=1; and (4) positive aVR. Patients with 3 of 4 features had a high probability of LPFVT, whereas patients with <=1 feature always had a diagnosis of SVT with RBBB plus left anterior fascicular block. Our patient displayed 3 of 4 features of LPFVT.

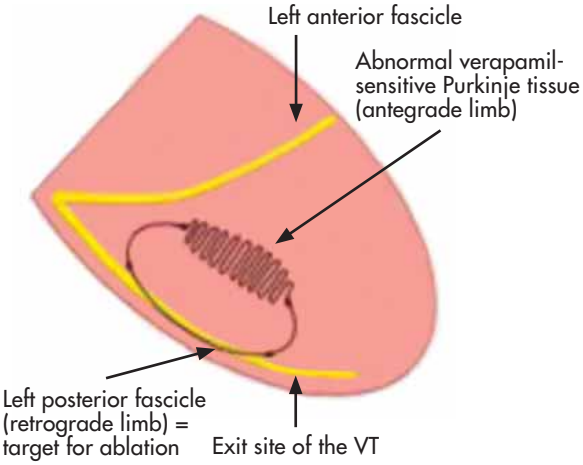

FIGURE 1: The VT circuit of left posterior fascicular ventricular tachycardia comprises abnormal Purkinje tissue (antegrade limb) and the left posterior fascicle (retrograde limb).
Exit site of the VT in the left posterior inferior region produces the characteristic atypical right bundle branch block morphology with left axis deviation. The figure is modified from reference 2.

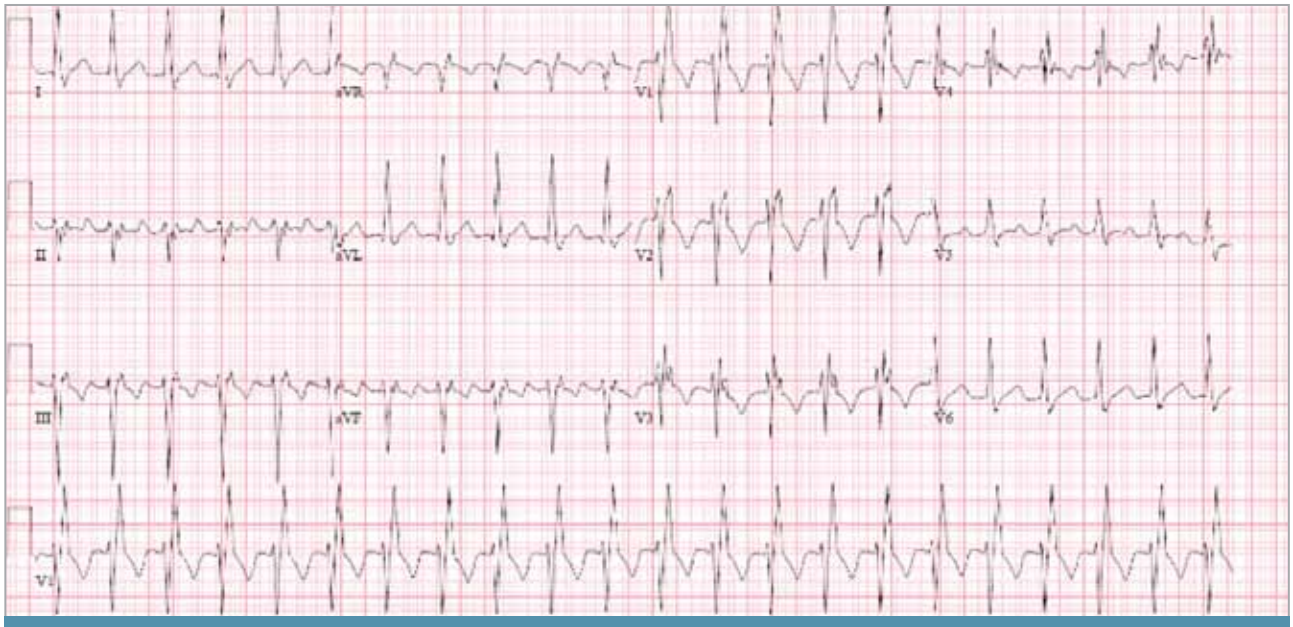

FIGURE 2: Atypical atrial flutter with 2:1 AV block, with right bundle branch block and left anterior fascicular block (see text for details).

An ECG of a patient with repaired infundibular pulmonary stenosis with a SVT with RBBB and left anterior fascicular block, is shown in Figure 2. The patient has an atypical atrial flutter with 2:1 AV block. This patient has typical features of RBBB in V1 and V6. Using the above criteria, the patient has no features of LPFVT. In addition to the typical RBBB morphology in V1 and V6, the QRS width was 160ms, the V6 R/S ratio was >1 and aVR was predominantly negative.

Intravenous verapamil can be successful in terminating LPFVT. Verapamil should never be administered to a wide complex tachycardia. LPFVT is a rare exception where verapamil can be administered for acute termination – but this should only be done in stable patients with a confirmed diagnosis of LPFVT. Termination with adenosine, vagal manoeuvres and beta-blockers is very rare, as the tachycardia is not cAMP-mediated. Chronic management involves oral verapamil, which can be useful to prevent recurrent episodes. Radiofrequency ablation of the LPFVT can be effective for drug-refractory cases.

The correct answer is: (4) Intravenous verapamil.

CONCLUSION

Left posterior fascicular ventricular tachycardia (LPFVT) is an idiopathic VT with an atypical right bundle branch morphology with left axis deviation.

Distinguishing LPFVT from a SVT with RBBB and left anterior fascicular block can be made by careful inspection of the morphology of the QRS complexes in V1, V6, aVR and QRS width.

Intravenous verapamil can terminate a LPFVT, and should only be administered in patients with a confirmed diagnosis.

REFERENCES

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Conflict of interest: none declared.

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