

(A) OVERVIEW OF THE ECG

This is a regular wide QRS tachycardia at 192/minute with a pattern resembling left bundle branch block (LBBB).

MORE DETAILED ANALYSIS OF THE ECG

The QRS is about 140ms wide with a LBBB pattern. The initial small R wave in V1-3 is just under 40ms wide and the onset of the QRS to the nadir of the S wave in these leads is 70ms. This is borderline, but compatible with LBBB. The axis is -20° .

There is no evidence of AV dissociation. There are sharp negative deflections distorting the ST segments in the inferior leads, compatible with retrograde P waves about 150ms after the onset of the QRS. The RP time is shorter than the PR (short RP tachycardia). There is no evidence of an extra P wave between those in the ST segments (1:1 ventriculo-atrial [VA] tachycardia).

DIFFERENTIAL DIAGNOSIS:

The default diagnosis for a regular wide QRS tachycardia is ventricular tachycardia.

However, the relatively brisk initial depolarisation in the right chest leads is compatible with LBBB and renders right ventricular tachycardia and antidromic AVRT unlikely.

Atrial flutter is also unlikely with a ventricular rate of 192/minute (too fast for flutter with 2:1 block and too slow for flutter with 1:1 conduction) in a structurally normal heart.

AV junctional re-entry tachycardia with LBBB is possible, either orthodromic AVRT or atypical AV nodal re-entry tachycardia. However, the leftward axis would be unusual in a young woman with a structurally normal heart, even in the presence of rate-related LBBB.

This pattern is typical of a Mahaim tachycardia, a rare form of antidromic AVRT where antegrade conduction occurs over an atypical long accessory pathway which, in the majority of cases, connects the right atrial free wall to the distal fibres of the right bundle branch (RBB) and retrograde conduction occurs over the AV node (Figure 1). The origin of the pathway is usually at the lateral border of the tricuspid valve. It differs from the typical accessory pathways (which are short AV

accessory pathways responsible for the WPW syndrome) in that it has decremental properties similar to those of the AV node.⁽¹⁾ As a result, the sinus rhythm ECG is usually normal with no delta wave (Figure 2), but pre-excitation with a LBBB pattern is occasionally present. As the accessory pathway inserts into the distal RBB, the tachycardia has a typical LBBB morphology (QRS usually $<150\text{ms}$) with left axis deviation as ventricular activation begins in the distal right bundle near the RV apical region.

Mahaim tachycardia is also known as an atriofascicular tachycardia. Mahaim initially described fibres connecting the His Bundle to the ventricular myocardium (so called nodoventricular and nodofascicular fibres) and not atriofascicular fibres. Although the terms "Mahaim fibres" and "Mahaim tachycardia" are still commonly used, it is better to describe the accessory pathway and tachycardia involved e.g. atriofascicular accessory pathway/tachycardia. Pathways with Mahaim characteristics can be atriofascicular, atrioventricular, nodofascicular and nodoventricular, depending on their variable proximal and distal insertions. Tachycardias involving Mahaim pathways, other than atriofascicular, do occur but are considered rare.⁽¹⁾

Confirmation of the diagnosis requires invasive electrophysiological study (EPS). Atrial pacing at faster rates leads to pre-excitation with left bundle branch block morphology as a result of the slowing of AV nodal conduction exceeding that of the Mahaim pathway. As the A-H interval increases, the H-V interval decreases. During Mahaim (atriofascicular) tachycardia, the right bundle potential precedes His bundle activation.

The correct answer is therefore (c):

Mahaim tachycardia.

(B) INTERVENTIONS

Adenosine should be avoided in wide QRS tachycardias. If the tachycardia is ventricular (the majority are) the catecholamine surge following adenosine vasodilatation may provoke ventricular fibrillation. If it is atrial flutter, 1:1 conduction may occur after the brief AV nodal blockade wears off. If it is orthodromic AVRT, pre-excited atrial fibrillation may be induced⁽²⁾ which can degenerate into VF.

Intravenous amiodarone will only terminate a minority of VTs (about 20% - 30%) and may cause haemodynamic collapse.

Intravenous adenosine and amiodarone may terminate a Mahaim tachycardia but should not be the first choice of treatment.

Carotid sinus massage or the Valsalva manoeuvre may terminate a Mahaim tachycardia (AV node is part of the circuit) and AV junctional re-entry tachycardia – useful both therapeutically and diagnostically. Vagal manoeuvres are ineffective in VT, although they may induce VA block/AV dissociation which would confirm the diagnosis. They should preferably be performed with at least a 3 channel ECG running, to document the

mechanism of termination or the atrial mechanism during transient slowing.

Synchronised DC cardioversion is the treatment of choice for termination of a regular wide QRS tachycardia (default diagnosis – VT), whether haemodynamically stable or not. It will terminate most tachycardias, other than ectopic atrial tachycardia.

The answer to (B) is (c) Carotid sinus massage followed, if necessary, by DC cardioversion.

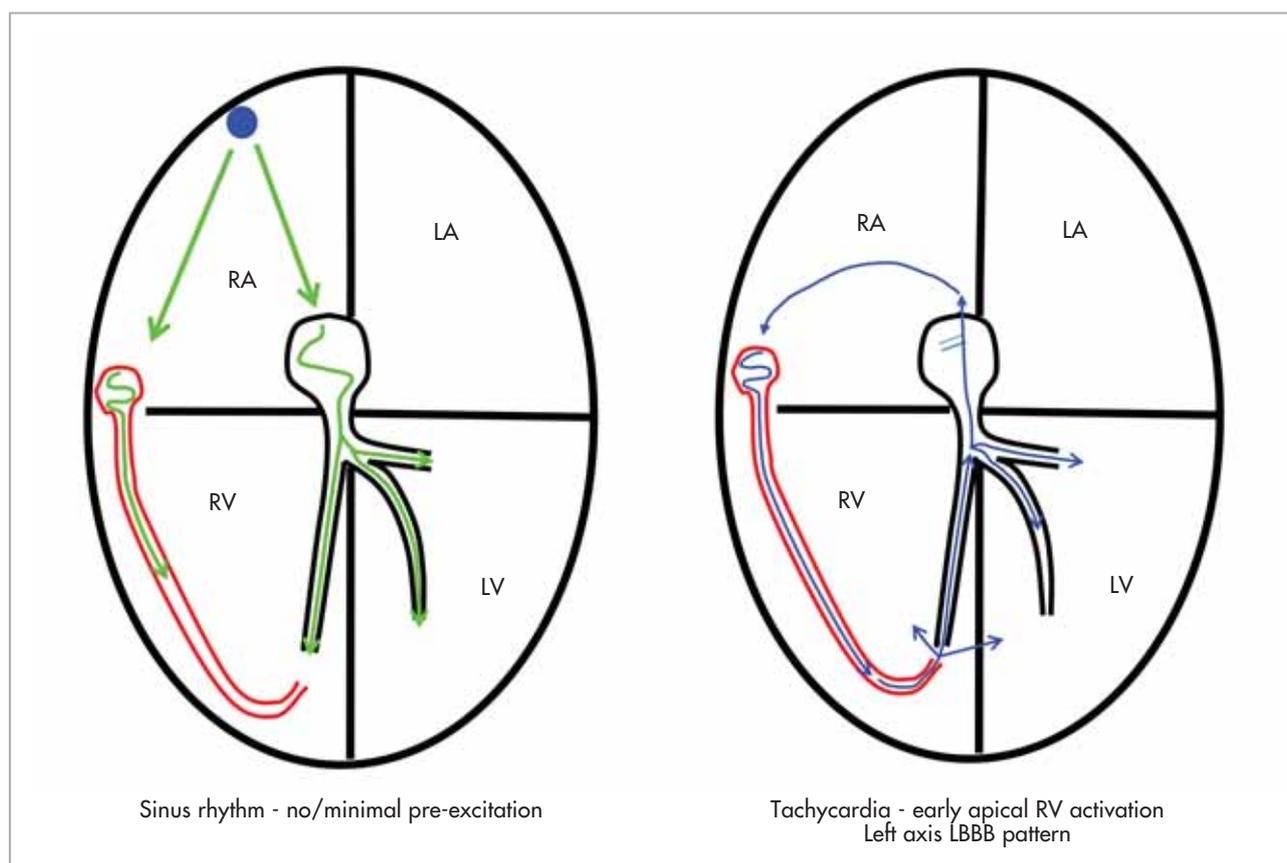


FIGURE 1: Diagrams showing the atriofascicular accessory (Mahaim) pathway connecting the right atrium at the lateral tricuspid annulus to the distal end of the right bundle branch.

On the left, during sinus rhythm, there is no pre-excitation as the impulse travelling via the AV node reaches the ventricles before the long, slowly conducting atriofascicular pathway.

On the right, during antidromic atrioventricular (Mahaim) tachycardia, conduction via the accessory pathway activates the right ventricle near the distal end of the right bundle from where the impulse travels retrogradely via the normal conducting system back to the right atrium. As a result, the ECG shows left axis deviation and LBBB.

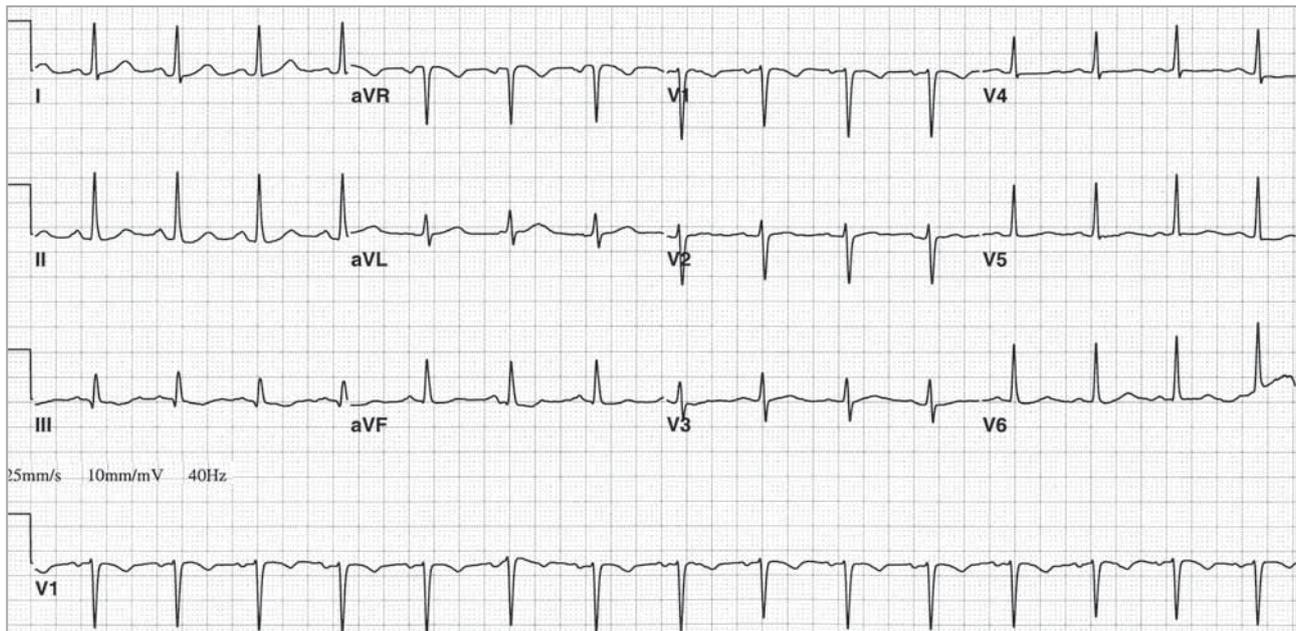


FIGURE 2: A subsequent ECG in sinus rhythm with no evidence of pre-excitation.

In addition to confirming the diagnosis, EPS forms part of long-term management in that the accessory pathway can be located and ablated with a high degree of success and low risk. This young woman's pathway was located at the lateral tricuspid annulus by recording accessory pathway potentials and consequently successfully ablated, curing her tachycardias.

LESSONS AND CONCLUSIONS

- While most (80%) regular wide QRS tachycardias are ventricular, other mechanisms should be considered when the QRS pattern is typical for left or right bundle branch block.
- Mahaim tachycardia is a rare form of antidromic AVRT in which an accessory pathway with decremental properties, similar to the AV node, connects the right atrium to the right bundle branch (atriofascicular accessory pathway).
- The tachycardia has a pattern of typical LBBB, usually with left axis deviation, but sometimes a horizontal or normal axis, depending on the site of ventricular insertion.
- The ECG in sinus rhythm is usually normal but may show different degrees of pre-excitation with LBBB morphology.
- The accessory pathway can be located at EPS and ablated with radiofrequency via the catheter.

REFERENCES

1. Katritsis DG, Wellens HJ, Josephson ME. Mahaim accessory pathways. *Arrhythm Electrophysiol Rev.* 2017;6:29-32.
2. 2019 ESC guidelines for the management of patients with supraventricular tachycardia. *Eur Heart J.* 2019;00:1-65.

Conflict of interest: none declared.