ECG Quiz 46

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
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ANSWER

OVERVIEW OF THE ECG

The ventricular rate is 96bpm. The QRS complexes are narrow (80ms) and appear to be regular, except for the last R-R interval which appears to be slightly shorter. There appear to be underlying flutter waves with a regular atrial rate around 288bpm. Sinus rhythm is therefore excluded.

All the flutter waves appear to be identical in morphology with 3:1 AV block (apart from the last complex). Atrial fibrillation is a "chaotic rhythm" characterised by irregular fibrillatory waves (usually 400 - 600bpm) which have variable morphology with irregular conduction to the ventricles. This results in an irregular ventricular rate. Atrial fibrillation is therefore excluded.

Closer inspection of the morphology of the flutter waves is the next step. The morphology of the flutter waves can be described as "continuous", "wavelike" or having a "sawtooth" appearance. Flutter waves have no beginning and no end with no isoelectric baseline between successive flutter waves. This suggests that at any point in time, a region of the atrium is being depolarised. Electrophysiologists refer to this as a macro-reentrant circuit and hence, atrial flutters are referred to as macro-reentrant atrial tachycardias.

The term "atrial tachycardia" is sometimes confusing and is a "general term" to describe both macro-reentrant atrial tachycardias and focal atrial tachycardias. Generally, when electrophysiologists use the term "atrial tachycardia", they mean a "focal" atrial tachycardia. In contrast to macro-reentrant atrial tachycardias, focal atrial tachycardias arise from a discrete focus in the atrium with subsequent centrifugal spread of atrial activation from that site. Think about the common analogy of dropping pebbles in a pond. The entry point of the pebble represents the site of origin of the tachycardia and the subsequent wave represents atrial depolarisation radiating centrifugally from this site. There is a beginning and an end to each atrial depolarisation (causing a P wave on the ECG) with a resultant period of atrial "silence" between atrial depolarisations. This results in distinct P waves separated by an isoelectric baseline (similar to sinus tachycardia, which mechanistically can be viewed as an atrial tachycardia) (See Figure 1). Focal atrial tachycardia rates are usually slower than atrial flutter (atrial flutter rates can vary between 240bpm and 360bpm). Focal atrial tachycardia can sometimes be irregular and often conduct 1:1 to the ventricle. Depending on AV nodal conduction, focal atrial tachycardia can cause both long and short RP tachycardias. The morphology of the P wave (P wave axis) can sometime be useful (if not obscured by the T wave) to determine the site of origin of the atrial tachycardia. In view of the atrial rate (288bpm) and the flutter wave morphology, focal atrial tachycardia is excluded.

The patient therefore has an atrial flutter with 3:1 AV block (3:1 AV block is uncommon and AV block usually occurs in multiples of 2:1 [most common], 4:1 and 6:1). The next step is to try and determine the location of the atrial flutter circuit by inspecting the flutter wave morphology. Flutter waves are usually best seen in the inferior leads (II, III and aVF) and lead V1. The terminology used to describe the different types of atrial flutters can be confusing. In summary, there are only 2 types of atrial flutters: (1) typical atrial flutter and (2) atypical atrial flutter.[1]

Typical atrial flutter refers to a macro-reentrant right atrial tachycardia rotating counterclockwise, or clockwise, around the tricuspid annulus AND uses the cavotricuspid isthmus as an essential part of the flutter circuit. Atypical flutters refer to all atrial flutters that are not typical atrial flutters, some of which arise in the left atrium. Typical atrial flutter can be further divided into 2 types: (1) Counterclockwise flutter or (2) Clockwise flutter (also called reverse typical flutter). See Figure 1.

In counterclockwise typical atrial flutter (common type), the activation wavefront propagates superiorly up the septal side of the tricuspid annulus towards the crista terminalis and advances inferiorly along the lateral wall of the right atrium to reach the lateral tricuspid annulus after which it advances through the cavotricuspid isthmus (the tissue separating the inferior vena cava from the tricuspid valve). The entire flutter circuit is confined within the right atrium and counterclockwise, as seen in the LAO view from the ventricular side of the tricuspid annulus. Left atrial activation occurs as a bystander across the fossa ovalis and Bachmann's bundle. Flutter waves resemble a negative sawtooth pattern in the inferior leads. This consists of a downsloping segment (A=isthmus conduction), followed by a sharper negative deflection (B=septal conduction and LA conduction) and then a sharp positive deflection with a positive overshoot (C=lateral wall conduction) leading to the next downsloping plateau (see Figure 2).[2] The relative size of each

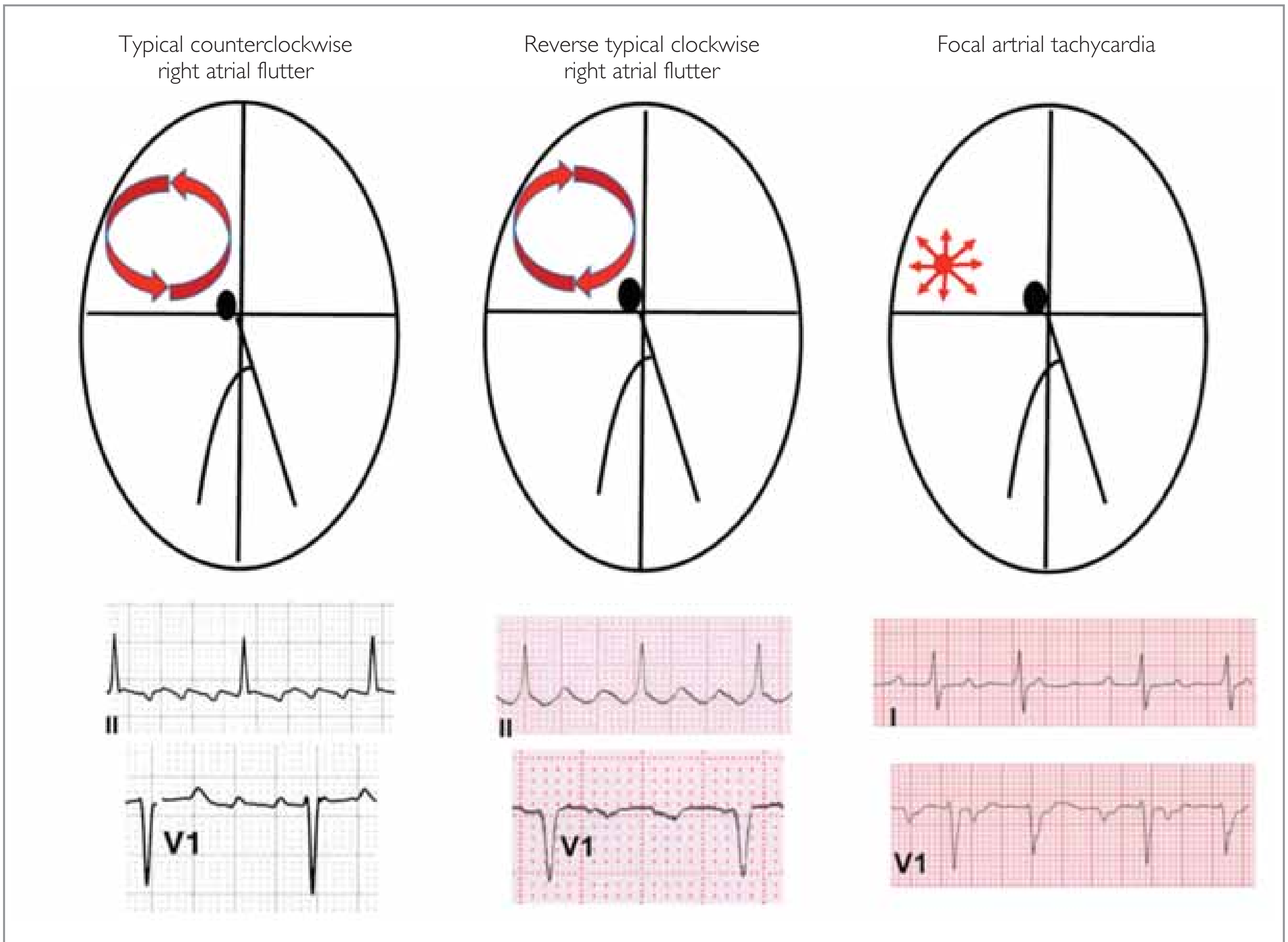

FIGURE 1: Mechanisms of atrial flutter and atrial tachycardia.

of the components can vary. Other variations include pure negative deflections in the inferior leads or negative and then positive deflections that are equal in size. Flutter waves are usually positive or biphasic in V1 and negative in V6.

In clockwise (reverse typical) atrial flutter, the activation wavefront propagates in the opposite direction to that in typical counterclockwise atrial flutter. Clockwise flutter is much less common compared to counterclockwise flutter (9:1 counter-clockwise predominance). The ECG of clockwise flutter is more variable than counterclockwise flutter. Usually there are broad positive deflections in the inferior leads with characteristic notching with an inverted component preceding the upright notched component which results in an equal positive and negative deflection. The flutter wave in V1 is usually negative, wide and notched and positive in V6. The morphology of the flutter waves in the ECG is compatible with these findings.

The correct answer is therefore (6) Clockwise reverse typical right atrial flutter with variable AV block.

The term "atypical atrial flutter" is used to describe all other macro-reentrant atrial tachycardias other than "typical atrial flutter". Atypical atrial flutters can occur in the right or left atria. Atypical right atrial flutters usually occur in the setting of atriotomy scars (prior congenital heart disease surgery). Atypical left atrial flutters (perimitral flutter, roof dependent flutter) usually occur in the setting of prior catheter ablation for

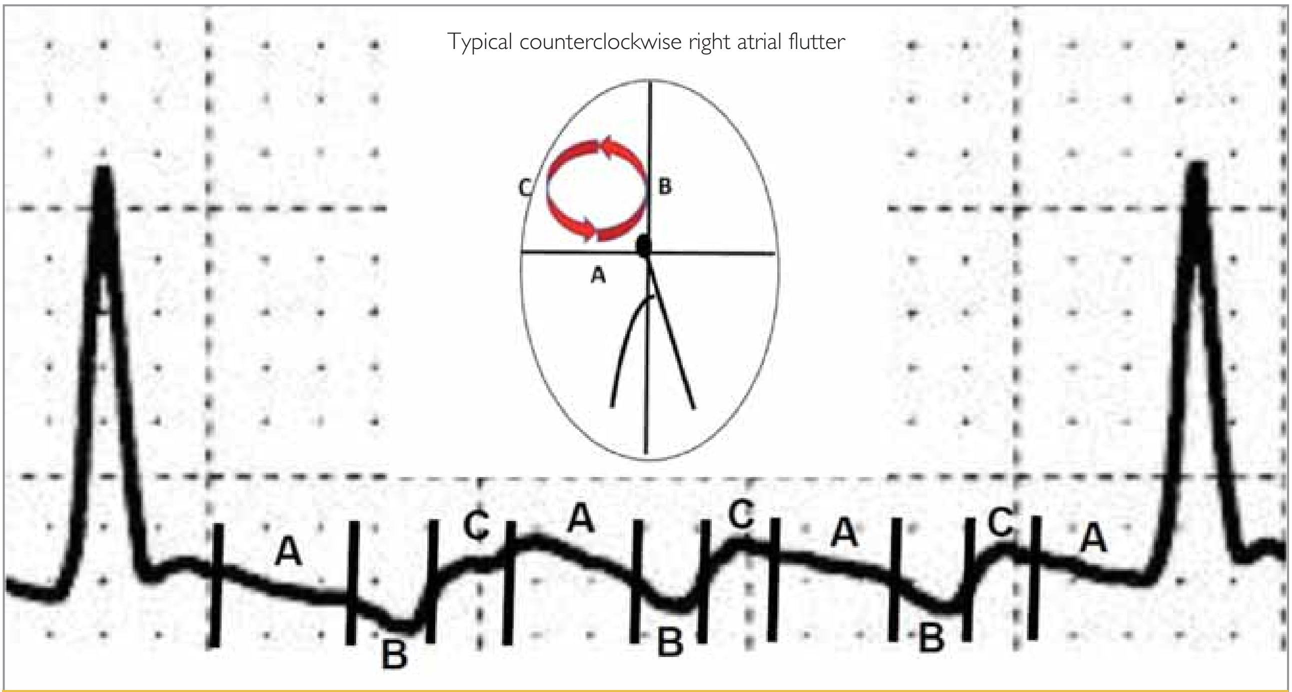

FIGURE 2: Different components of the typical atrial flutter wave (LEAD II).

A: Isthmus conduction, B: Septum conduction, C: Lateral wall conduction.

atrial fibrillation or after surgical procedures like the MAZE procedure. Occasionally, right and left atrial flutters can occur without prior history of surgery or catheter ablation. The ECG morphology of the atrial flutter waves can be highly variable and even resemble typical atrial flutter or focal atrial tachycardia with an isoelectric baseline. The ECG is less useful in predicting the atrial flutter circuit in patients who have had a prior AF ablation or who have scarred atria. Generally, concordant P wave polarity in V1 and the inferior leads (either negative or positive) is suggestive of an atypical flutter. This observation may help in distinguishing atypical atrial flutter from reverse typical atrial flutter which sometimes can be difficult.

MANAGEMENT

After taking a history and performing an examination, the clinician must assess the symptom burden and whether there is underlying structural heart disease. An echocardiogram is often needed. The rest of the discussion will focus on the management of atrial flutter per se.

The diagnosis of reverse typical atrial flutter can be made from the 12 lead ECG. Carotid sinus massage might increase the degree of AV block but will not provide any additional diagnostic information and will not terminate the atrial flutter. Adenosine should not be given to patients with atrial flutter as the reflex sympathetic discharge shortly after adenosine administration can precipitate 1:1 AV conduction and can result in haemodynamic compromise.

The patient has had palpitations for a few weeks and one must assume that the atrial flutter has been present for a similar length of time. The patient is haemodynamically stable. The thromboembolic risk of atrial flutter is widely regarded as being similar to atrial fibrillation. In patients with atrial flutter of unknown duration, or who have atrial flutter >=48 hours, it is essential to exclude left atrial appendage thrombus prior to performing a DC cardioversion, with a transoesphageal echocardiogram, because of the risk of thromboembolism.[1]

The next step in the acute management of atrial flutter is to control the ventricular rate (so-called rate control strategy). Oral or intravenous (if available) beta-blockers should be tried first. It is more difficult to control the ventricular rate for atrial flutter compared to atrial fibrillation and beta-blockers should be used with caution in patients with heart failure. Oral calcium channel blockers can sometimes be used when beta-blockers are contraindicated (like asthma) in the absence of heart failure. Intravenous or oral amiodarone should only be used for the acute control of ventricular rate when beta-blockers are contraindicated or ineffective in patients with systolic heart failure. Although unlikely, amiodarone may result in the acute cardioversion of atrial flutter to sinus rhythm so it should not be given in patients with atrial flutter >=48 hours who are not on anticoagulation. The $CHA2DS_2VASc$ score can be used in atrial flutter to assess the need for chronic long-term anti-coagulation. In patients with a $CHADS_2VASC$ score of ≥2, oral anticoagulation is advised.

The correct answer is therefore (5) Rate control with beta-blockers and consider anticoagulation.

In most patients with atrial flutter, a rhythm control strategy (restoring sinus rhythm by means of catheter ablation) is the preferred long-term strategy. Since rate control is seldom satisfactory with AV nodal blockers, referral to an electro-physiologist to consider catheter ablation is required. Anti-coagulation for 3 - 4 weeks followed by DC cardioversion or catheter ablation can be performed. Post cardioversion, recurrences are likely and referral to an electrophysiologist to consider catheter ablation is often needed. Since typical atrial flutter is curable by ablating the cavotricuspid isthmus, this is the treatment of choice.

This patient had confirmed reverse typical atrial flutter at EP study. A cavotricuspid isthmus ablation was performed which terminated the atrial flutter.

CONCLUSIONS

Atrial flutter is a macro-reentrant atrial tachycardia and can be classified as typical or atypical.

Typical or reverse typical atrial flutter involves the cavotricuspid isthmus.

Catheter ablation is the treatment of choice for typical and reverse typical atrial flutter.

Conflict of interest: none declared.

REFERENCES

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