CASSA

CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA

A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

Answer to ECG Quiz 13

The ECG shows a regular tachycardia at 120bpm.

Before discussing the best management strategy, the exact ECG diagnosis must be made.

The **differential diagnosis** that must be considered includes:

1. **Sinus tachycardia:** After all, this patient is in heart failure and sinus tachycardia is a very common finding or response to decompensation.

2. **Ventricular tachycardia (VT):** Any patient with a cardiomyopathy is at risk of VT although this is less common in dilated cardiomyopathy than in ischemic cardiomyopathy. It would be unusual to have sustained for weeks without progressive deterioration of heart failure.

3. **Supraventricular tachycardias such as atrial tachycardia, atrial flutter or atrial fibrillation:** Each of these can be associated with heart failure: but this may be a "chicken vs. egg first" situation. Heart failure, probably mediated by atrial stretch, may precipitate any of these arrhythmias. On the other hand, it is well recognised that tachycardia may induce a cardiomyopathy or exacerbate an underlying less serious cardiac disorder. The relevance is that since tachycardia-induced cardiomyopathy is one of the few cardio-myopathies that may be reversible, it must always be excluded in every patient with heart failure and any tachycardia.

Observations

The first step is to differentiate between ventricular and supraventricular tachycardia. Analysis of any tachycardia should answer 2 questions:

- Is the rhythm regular?
- Are the QRS complexes wide?

If the answers to both are "Yes", the default diagnosis is VT. It is important to note that this diagnosis is not primarily dependent on P waves or atrial activity, although their analysis may have significant secondary supportive value.

Systematic analysis of the ECG reveals

- **QRS width:** The widest QRS needs to be measured. Normal width is up to 100ms or 2.5 little blocks. The first QRS complex in both V5 and V6 is easiest to measure: just over 2.5 blocks, around 110ms: so not exactly narrow but not definitely wide either (>120ms). This patient has an underlying cardiomyopathy which may account for some slowing of normal conduction and widening of QRSs. It is also likely that if he had VT, the QRS would be definitely wide and not borderline. Are there any other clues for VT vs. more normal conduction?

- **QRS morphology:** In the chest leads there is obvious poor R wave progression. In V1 to V3 there are in fact no R waves and QS waves are noted. This would be quite compatible with an anterior myocardial infarction pattern, but we have been told that this man does not have ischemic heart disease. Such patterns can occur in dilated cardiomyopathy. A closer look at the QS waves is useful because one can observe that the initial component is very rapid: from onset of the QRS to the nadir of the QS wave is 50ms or just over 1 little block. If one had any concern that this ECG could be a narrowish VT rather than a conducted rhythm, this finding of this rapid conduction <70ms dispels that.

But, clearly, these conducted QRS complexes are most abnormal. There is intraventricular conduction delay with double components in the limb leads. So is this Left Bundle Branch Block (LBBB)?

The criteria for LBBB

1. QRS duration ≥ 120ms

2. Broad monophasic R wave in leads I, V5 and/or V6, usually with notching

3. Absence of the normal tiny septal q waves in leads I, V5 and V6.

4. Delay in the intrinsicoid deflection (time from onset to peak of R) (>40ms) in V5 and V6.

Are any of these criteria present? The answer to all 4 is "No".

- **QRS axis:** approximately minus 75° (normal in adult is -30° to +90°). This may be explained by 2 mechanisms: left anterior hemiblock and loss of inferior myocardium, which most commonly occurs with inferior myocardial infarction.

The conclusion thus far is that this is a conducted SVT albeit with abnormal QRS complexes.

Now we can turn our attention to the atrial activity: The regularity excludes atrial fibrillation. Examination of V1 suggests P waves possibly stuck at the end of the T waves which would then give an apparent long PR interval. Is this sinus tachycardia? This needs analysis of the limb leads: is the atrial activity compatible with sinus node origin on 2 accounts: axis and morphology? The positive atrial activity in V1 that may be interpreted as a P wave occurs simultaneously with negative activity in Leads II and III and flattening in Lead I (Figure 1). Sinus P waves would be positive in Lead II. Morphology is also not typical: it is not possible to define the beginning or end of the "P waves" in this ECG.

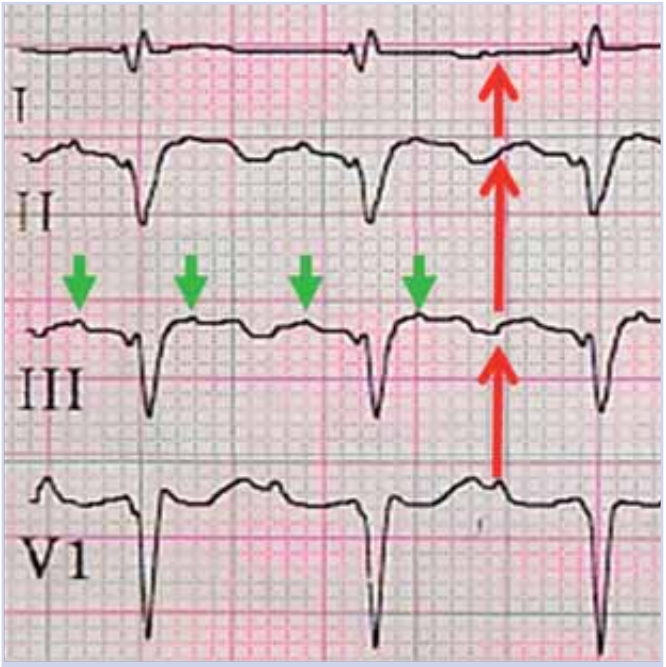

FIGURE 1: Leads I, II, III and V1: The red arrows show that simultaneously with the positive deflection of atrial activity in V1 there is absence of normal sinus P waves and instead flattening in I and inverted waves in II and III. The green arrows show recurring deflections occurring at double the QRS rate, suggesting atrial tachycardia or atrial flutter.

TABLE 1: The differential diagnosis of the common supraventricular tachycardias in adults.

Supraventricular Tachycardias
Sinus Tachycardia
Atrial Tachycardia
Atrial Flutter
AV Junctional Re-entry Tachycardias (AVNRT; AVRT)
Atrial Fibrillation

AVNRT=atrioventricular nodal re-entry tachycardia
AVRT=atrioventricular re-entry tachycardia using an accessory pathway

In the list of differential diagnoses of SVT, we are left with atrial tachycardia, atrial flutter and atrioventricular junction re-entry tachycardia (Table 1). As the ventricular rate is 120bpm, if this is atrial flutter, which is commonly conducted 2:1, the atrial rate would be 240bpm. Typical atrial flutter is usually around 300bpm but can be slower if the atria are enlarged or conduction is slow or slowed by antiarrhythmic drugs. Examination of Leads II and III shows that there appears to be continuous electrical activity between the QRS complexes and the tiny positive notches are not only equidistant but occur at exactly half the RR intervals (Figure 1). This suggests atrial flutter or atrial tachycardia.

To confirm this diagnosis, it would be useful to record the atrial activity "uncontaminated" by QRS complexes. Carotid sinus massage was performed in this patient while recording the ECG (Figure 2). This did not terminate the SVT but increased AV block from 2:1 to 4:1 and higher and exposed the underlying continuous "saw-tooth-like" atrial activity of atrial flutter.

ANSWER TO ECG QUIZ 13

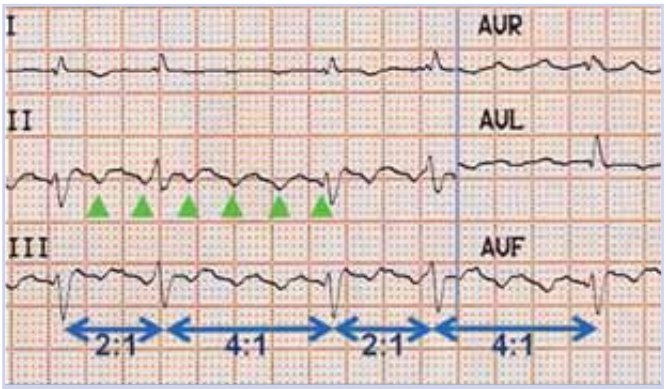

FIGURE 2: ECG during carotid massage: The vagal effect caused intermittent change from the baseline 2:1 conduction to 4:1 atrioventricular conduction and clearly exposed the sawtooth pattern of the underlying atrial flutter.

TREATMENT OPTIONS

Management of this patient with advanced cardiomyopathy requires exclusion / correction of any reversible causes. As this tachycardia is not just sinus tachycardia associated with bad heart failure, intensification of medical therapy and dobutamine (a) is inappropriate. Also, consideration of transplantation is premature (i). Is there a place for cardiac resynchronization therapy with a biventricular pacemaker? (b) No – firstly because a correctable arrhythmia has been identified and also because the patient does not have LBBB or sufficiently wide QRS complexes without which benefit is unlikely. Cardioversion (d), unless for an immediately life-threatening arrhythmia, is contra-indicated in this patient. The duration of this patient's atrial flutter is unknown and the risks of thrombo-embolism are similar to those in atrial fibrillation.

The best definitive treatment option with the greatest chance of achieving reversibility of any component of tachycardia-induced cardiomyopathy is to ablate this atrial flutter circuit and to cover him with warfarin (e). This was planned. However, as he had not been on prior anticoagulation and the risk of thrombo-embolism is identical whether cardioversion from atrial flutter to sinus rhythm is electrical, chemical/drug or by ablation, transesophageal echocardiography was done first. He was shown to have a left atrial appendage thrombus. The procedure has therefore been postponed until 3-4 weeks of therapeutic anticoagulation with warfarin has been achieved. In the interim, amiodarone for its AV conduction rate slowing effect may be considered. Standard beta-blockers used for rate control, e.g. atenolol are contra-indicated in this patient with "wet" heart failure. Low "commencement-dose" beta-blockers used for heart failure, such as carvedilol, are unlikely to achieve much ventricular slowing in atrial flutter.

The correct answer is (e).

CONCLUSION

- Since tachycardia-induced cardiomyopathy is a potentially reversible condition or superadded component in patients with cardiac dysfunction, all tachycardias in patients with heart failure require careful analysis and intervention when possible.
- Sinus tachycardia requires normal P wave axis and compatible morphology in especially Lead II.
- A vagal maneuver, such as carotid sinus massage, may be therapeutic or an easy diagnostic tool in difficult SVTs.
- Patients with atrial flutter are at similar risk of thrombo-embolism as those with atrial fibrillation and require the same precautions prior to cardioversion by any method.

ECG and **QUESTION** on page 186