ECG Quiz 71

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ANSWER

OVERVIEW OF THE ECG

This is a regular narrow complex tachycardia with a ventricular rate of 186 bpm. P waves are visible in II, III, and aVF that distort the ST segment (Figure 1). This is a 1:1 short RP tachycardia (RP time ~ 90 ms). The QRS duration is borderline wide (100 ms). AVNRT, atrial tachycardia, and AVRT are possibilities. The ventricular rate of 186 bpm in the setting of a 1:1 short RP tachycardia is unusual for an atrial flutter with 1:1 conduction, unless the patient has a markedly enlarged right atrium, or if the atrial flutter has been slowed by an antiarrhythmic drug. In this case, an antiarrhythmic drug was given, so a 1:1 atrial flutter must also be included in the differential diagnosis.

ANSWER

The correct answer is: 5. All the above are possible.

The rhythm strip with carotid sinus massage (before antiarrhythmic drug administration) shows atrial flutter with 2:1 atrioventricular (AV) block with an atrial rate of 250 bpm (ventricular rate 125 bpm). The typical sawtooth pattern of right atrial counterclockwise flutter can be seen in II and aVF. Carotid sinus massage induces AV block, which reveals continuous atrial flutter waves. The antiarrhythmic drug, therefore, slowed the atrial rate from 250 bpm (in 2:1 atrial flutter) to 186 bpm (in 1:1 atrial flutter). This resulted in a paradoxical increase in the ventricular rate from 125 bpm to 186 bpm.

The final diagnosis is 1:1 atrial flutter.

The most likely oral antiarrhythmic drug the patient received slows the atrial flutter rate but has minimal effect on the AV node. Class Ic antiarrhythmic drugs, like flecainide and propafenone, are likely culprits. Disopyramide, a class Ia antiarrhythmic drug, can also slow the atrial flutter rate and has vagolytic effects, which may increase AV node conduction and cause the same. This patient received oral flecainide without an AV nodal blocker. Another clue that the patient received flecainide is the mild QRS prolongation of 100 ms in the 12-lead ECG. A wider QRS duration is seen in II and aVF on the ECG compared with the rhythm strip performed before flecainide administration.

DISCUSSION

Flecainide, a class Ic antiarrhythmic drug, blocks cardiac sodium (Na) channels and slows atrial conduction (by prolonging the atrial action potential) with minimal effect on AV node conduction. Flecainide, taken with an AV nodal blocker, is commonly used in the acute setting to terminate atrial fibrillation ("pill in the pocket" or intravenous infusion) and in the chronic setting to prevent atrial fibrillation episodes. However, flecainide should NOT be used in the acute or chronic management of atrial flutter.[1]

Atrial flutter is a macro-re-entrant atrial tachycardia, which usually depolarises the atria at a rate between 240 bpm and 360 bpm. Atrial flutter usually occurs with 2:1 AV block, resulting in a ventricular rate of 120–180 bpm (usually ~ 150 bpm). Flecainide slows atrial conduction and can reduce the atrial rate by about one-third.[2] A reduction in atrial rate can facilitate 1:1 AV conduction, resulting in a very fast ventricular rate (as flecainide has a minimal effect on the AV node). A 1:1 atrial flutter is therefore a serious complication of flecainide and has no role in the acute or chronic treatment of atrial flutter. Occasionally, 1:1 atrial flutter can conduct with a rate-related bundle branch block, which can be misdiagnosed as ventricular tachycardia. Flecainide may convert atrial fibrillation into atrial flutter; it is therefore advised to prescribe flecainide with an AV nodal blocker (like a beta blocker), which reduces the likelihood of 1:1 AV conduction with atrial flutter.

Clinicians need to be aware of the other pro-arrhythmic effects of flecainide. Flecainide can cause monomorphic ventricular tachycardia and is associated with increased mortality, heart failure, and cardiac arrest in patients with prior myocardial infarction and impaired left ventricular function. It is advised to avoid flecainide in patients with ischaemic or structural heart disease, including significant left ventricular hypertrophy.[1] In the Cardiac Arrhythmia Suppression Trial (CAST), flecainide was

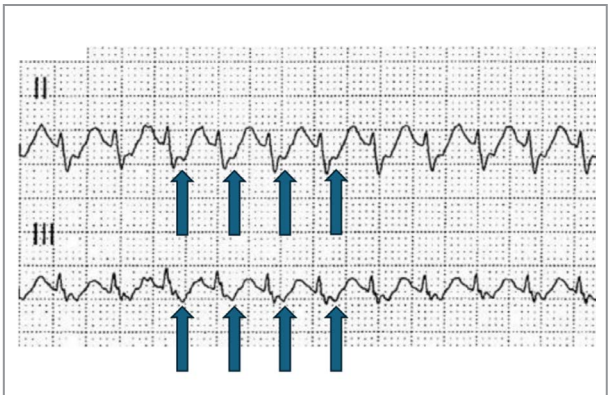

FIGURE 1: Arrows show P waves in II and III; this is a 1:1 short RP tachycardia with a wide differential diagnosis that includes atrioventricular-nodal re-entrant tachycardia, atrioventricular re-entrant tachycardia, atrial tachycardia, and 1:1 atrial flutter.

associated with an increased risk of death due to arrhythmias when given to suppress ventricular ectopy post-acute myocardial infarction.[3]

Flecainide also prolongs the action potential duration of ventricular muscle and, therefore, prolongs QRS duration. If the QRS widens by > 25% from baseline, it is advised to reduce the dose or to discontinue the drug. These drugs are not advised for patients with a baseline QRS > 120 ms due to the risk of excessive conduction delay, especially in those with left bundle branch block (LBBB) or bifascicular block. Flecainide must not be prescribed to patients with congenital long QT syndrome because of the risk of torsade de pointes. Flecainide may also unmask the Brugada ECG pattern and should be avoided in patients with the Brugada syndrome.

CONCLUSION

Flecainide without an AV nodal blocker may convert atrial fibrillation to atrial flutter with 1:1 AV conduction, which can be a life-threatening complication. Flecainide should also be avoided in the acute and chronic treatment of atrial flutter because of the dangers of 1:1 AV conduction. Atrial flutter with 1:1 AV conduction can cause very fast ventricular rates > 200 bpm, which can be difficult to distinguish from AVNRT, AVRT, or atrial tachycardia. Clinicians need to be aware of the potential pro-arrhythmic effects of flecainide, which can be fatal.

REFERENCES

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