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ENDGAMES

CASE REVIEW

A case of palpitation and pre-syncope

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A 66 year old woman presented to the emergency department with episodes of palpitations, dizziness, and near fainting for a few hours. She had had a left atrial myxoma removed 15 years ago and had suffered from palpitations as a result of atrial tachycardia from time to time since then for which she took modified release verapamil 240 mg once daily. More recently she had started on sotalol 120 mg twice daily in addition to verapamil for better control of her tachycardia, and one week ago had undergone direct current cardioversion for persistent atrial tachycardia, which reverted her to sinus rhythm. She was admitted to the coronary care unit. Her ECG on admission is shown (fig 1). Subsequently an echocardiogram showed normal left ventricular systolic function and no recurrence of left atrial myxoma. Urea and electrolytes including magnesium were all within normal limits.

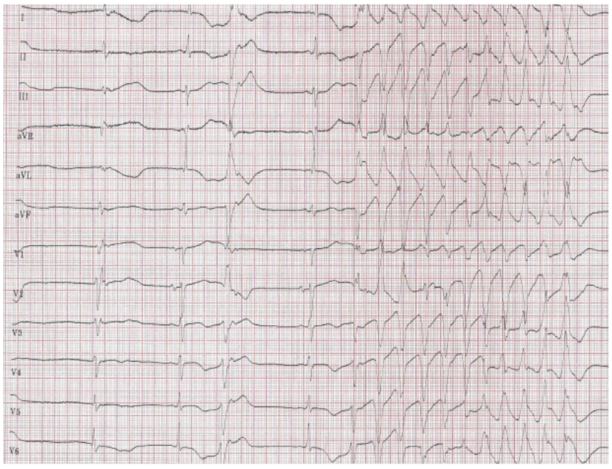

Fig 1 Electrocardiogram on presentation

Questions

1. What is the ventricular arrhythmia?
2. What is the likely cause and how common is it?
3. What are the steps to manage the most likely diagnosis?

Answers

1.

What is the ventricular arrhythmia?

Short answer

Torsade de pointes.

Discussion

This is typical torsade de pointes (polymorphic ventricular tachycardia with gradual change in QRS amplitude and twisting of QRS around the isoelectric line because of a prolonged QT interval). Bradycardia has likely increased the chance of sotalol induced torsade de pontes.

2.

What is the likely cause and how common is it?

Short answer

Drug induced QT prolongation. Sotalol is one of the commonest causes of torsade de pointes.[1] The incidence of torsade de pointes is 0.3% on 80 mg per day of sotalol and 3.8% on >680 mg per day of sotalol.

Discussion

Sotalol, as an anti-arrhythmic agent, has both class II (β blocking) and class III (action potential duration prolonging) effects. Substantial β blockade can happen with a dose as small as 25 mg, however the important class III effect only occurs at a dose of more than 160 mg per day.[2]

Sotalol prolongs ventricular repolarisation by blocking the rapid component of the delayed rectifier potassium current. It manifests on the ECG as QT prolongation and T-U wave abnormalities. The effect of action potential prolongation is more pronounced at a slower rate. A sequence of short-long-short RR intervals can facilitate torsade de pointes in drug induced cases of QT prolongation.

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Torsade de pointes is thought to result from triggered activity caused by an early after depolarisation (EAD) current generated during the abnormally prolonged phase of repolarisation. A long pause before the triggering beat helps to increase the amplitude of EAD and reach the necessary threshold to cause a ventricular ectopic. The varying delay of repolarisation in certain parts of the myocardium results in conduction of the ectopic beat being blocked in some directions but not others. This leads to re-entry, which sustains torsade de pointes.[3]

Drug induced QT interval prolongation leading to torsade de pointes is a serious but rare adverse reaction. A recent active surveillance study in Germany reported an age standardised incidence of drug induced long QT syndrome/torsade de pointes as 2.5 per million per year in men and 4 per million per year in women.[4]

The incidence of torsade de pointes with sotalol is higher in females, in renal dysfunction, and in patients with heart failure and sustained ventricular arrhythmia.[5]

3.

What are the steps to manage the most likely diagnosis?

Short answer

Review the patient's medications and stop drugs that might prolong the QT interval (eg, sotalol). Intravenous magnesium, isoprenaline, and temporary transvenous pacing can help.

Discussion

The main treatment of drug induced QT prolongation and torsade de pointes is to stop the relevant drug. Any associated dyselectrolytaemia should be corrected. Intravenous magnesium is a reasonable option irrespective of magnesium level to terminate torsade de pointes.[3] Temporary transvenous atrial or ventricular pacing is effective in preventing torsade de pointes in a patient with QT prolongation, particularly if there is notable bradycardia. Isoprenaline can be useful to prevent bradycardia and suppress torsades if temporary transvenous pacing is not immediately available or while waiting for temporary wire insertion.

Patient outcome

The patient's sotalol and verapamil were stopped on admission and after five days the QT interval normalised. She was treated with temporary transvenous ventricular pacing for three days (initially at a rate of 80 beats/min, which was gradually reduced to 60 beats/min) which prevented her bradycardia and torsades.

While in hospital the patient showed high grade atrioventricular block (2:1 and 3:1) when the temporary wire was taken out. This block persisted even after stopping sotalol and verapamil for five days. Hence she had a permanent pacemaker implanted. The decision to implant a permanent pacemaker was influenced by the fact that she would need anti-arrhythmics or rate controlling medication to treat her atrial tachycardia in future and it was thought that this would be difficult without a backup pacemaker.

I have read and understood BMJ policy on declaration of interests and declare that we have no competing interests.

Provenance: Not commissioned, externally peer reviewed

Patient consent obtained.

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