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CME Article

Electrocardiographical case. A case of wide complex tachycardia

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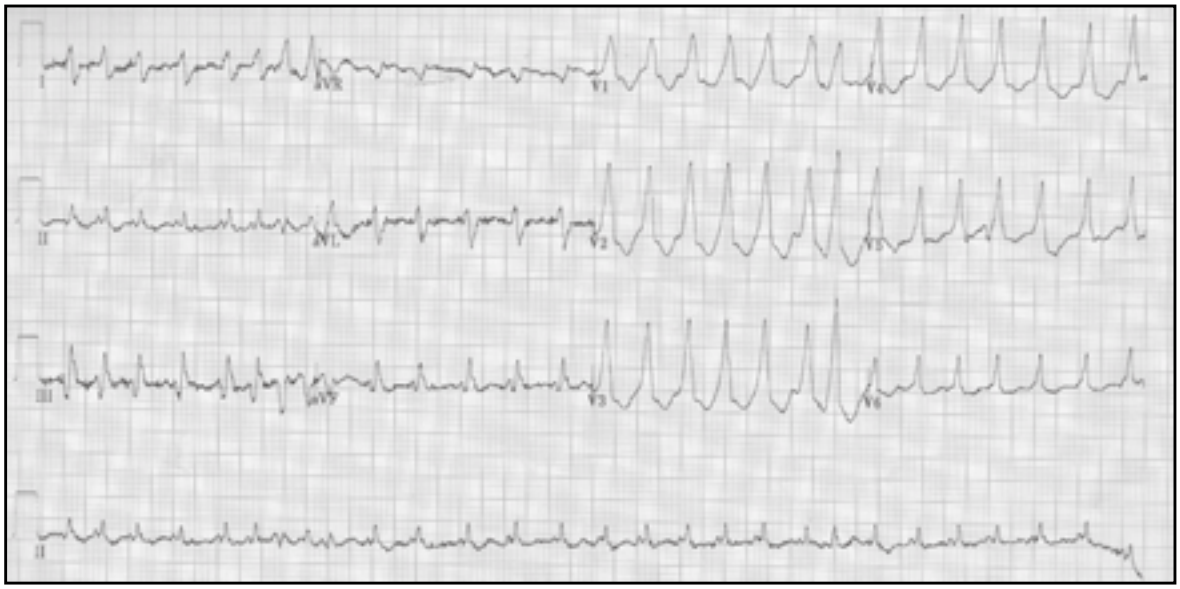

Fig. 1 12-lead ECG obtained at the emergency department.

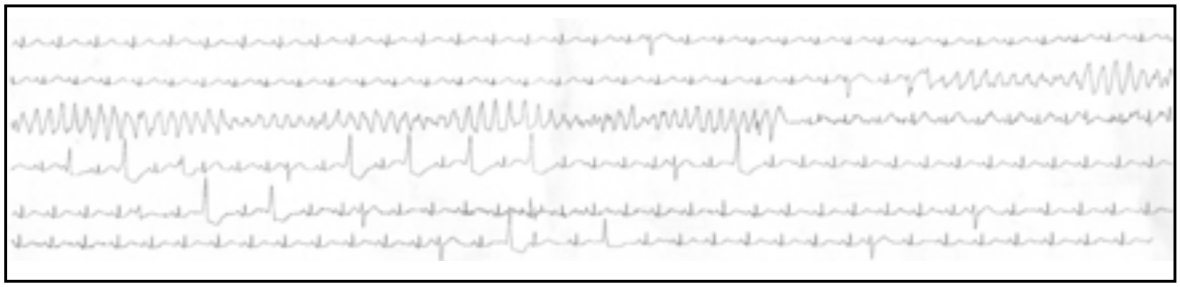

Fig. 2 Telemetry obtained when patient had transient loss of consciousness.

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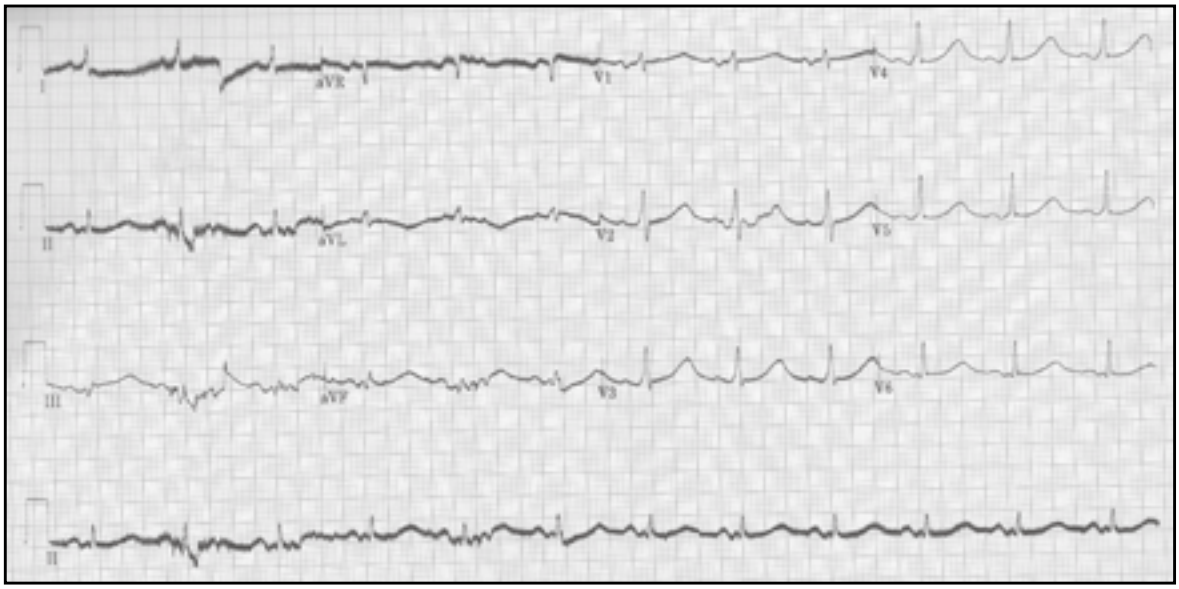

Fig. 3 12-lead ECG obtained one day after admission.

CLINICAL PRESENTATION

A 43-year-old Chinese woman presented to the emergency department complaining of a one week history of palpitations. These were irregular in nature, and associated with chest discomfort and shortness of breath. She was haemodynamically stable with a blood pressure of 154/95 mmHg. Her heart rate was more than 160 beats per minute (bpm). She was conscious, alert but diaphoretic. Cardiovascular examination was normal. A 12-lead electrocardiogram (ECG) was performed (Fig. 1). What is the diagnosis?

On admission to the coronary care unit, the patient's vital signs remained stable. Attempted direct current cardioversion (DCCV) under sedation failed to restore sinus rhythm. Pharmacological cardioversion with intravenous procainamide was attempted. Two hours later, the rhythm converted to a sinus rhythm. The procainamide infusion was continued overnight and she remained in sinus rhythm.

The next morning she developed a transient loss of consciousness lasting about 20 seconds. The telemetry captured the rhythm (Fig. 2). A 12-lead ECG was also obtained (Fig. 3). What had happened?

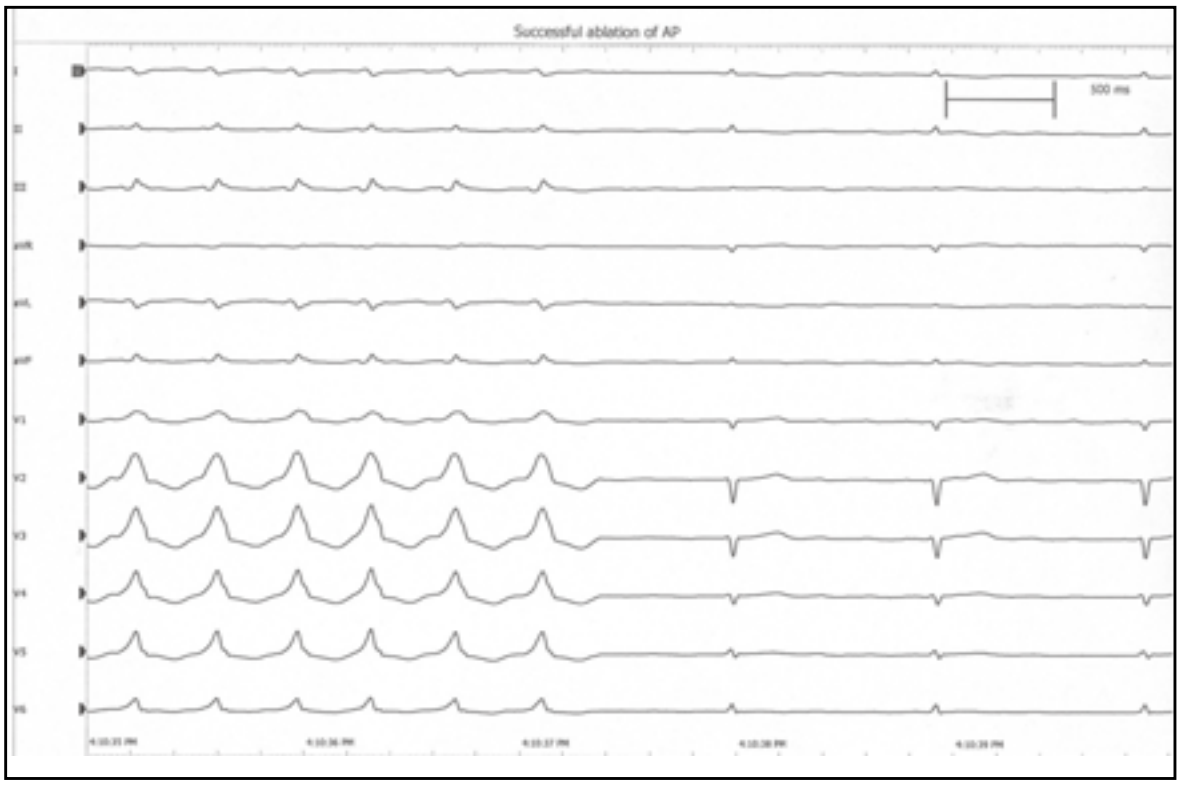

Fig. 4 12-lead ECG obtained immediately after successful ablation of the accessory pathway shows loss of pre-excitation.

ECG INTERPRETATION

The ECG (Fig. 1) revealed a wide complex tachycardia with a slightly irregular rhythm. There was slurred upslope of the QRS complexes. These ECG features should lead to an index of suspicion for pre-excited tachyarrhythmia. Conversion with intravenous procainamide led to sinus rhythm with drug-induced QT prolongation (Fig. 3) and hence torsades de pointes (Fig. 2).

DIAGNOSIS

Wolff-Parkinson-White (WPW) syndrome with pre-excited tachycardia, complicated by torsades de pointes as a result of drug-induced QT prolongation.

CLINICAL COURSE

The patient developed irregular wide complex tachycardia again, upon cessation of intravenous procainamide. Following informed consent, she underwent an electrophysiology study and radiofrequency catheter ablation. During the electrophysiology study, a single accessory pathway with only antegrade conduction properties was identified and located at the left posterolateral region. This was successfully ablated. The ventricular rate decreased to less than 100bpm. However, atrial tachycardia persisted.

Intravenous flecanide bolus was administered to slow the atrial rate following which the patient received direct current cardioversion (DCCV). Sinus rhythm was restored (Fig. 4). The patient was then given sotalol to maintain sinus rhythm. Her progress will be observed on an outpatient basis, with a view to further ablation in the event of atrial tachycardia recurrence.

DISCUSSION

The atrioventricular (AV) node is the only electrical pathway connecting the atria to the ventricles in normal individuals. However, certain individuals have an accessory AV conduction pathway (Wolff-Parkinson-White syndrome) that forms a connection between the atria and the ventricles outside the normal conduction system of the heart. This pathway comprises myocardial cells and therefore bypasses the AV nodal protection which is important when atrial tachyarrhythmias, in particular atrial fibrillation/flutter, occur.

The accessory pathway allows a direct relationship between the atrial and ventricular rates. Therefore, rapid ventricular rates during atrial tachyarrhythmias can provoke ventricular fibrillation[1]. The shortest preexcited RR interval during atrial fibrillation gives an indication of the refractory period of the accessory pathway which determines the ventricular rate. Short refractory periods (<250ms) are associated with an increased risk of sudden death.

The important point in management is to recognise the underlying arrhythmia and administer the appropriate treatment. The presence of an irregular wide complex tachycardia should raise the possibility of conduction via an accessory pathway. A Class 1 antiarrhythmic agent such as intravenous procainamide is the pharmacological agent of choice to prolong the refractory period of the accessory pathway, failing which direct current cardioversion is administered, especially if haemodynamic compromise occurs.

Procainamide, an antiarrhythmic agent, also has the potential of increasing the QT interval causing tosades de pointes[2] as in this case. Digitalis, beta blockers and calcium channel blockers are contraindicated and can increase the ventricular rate and provoke ventricular fibrillation in atrial tachyarrhythmias with accessory pathways. Radiofrequency ablation should be strongly considered, particular for accessory pathways with short refractory periods.

ABSTRACT

A 43-year-old Chinese woman complained of a one week history of irregular rapid palpitations associated with chest discomfort and dyspnoea. Her heart rate was more than 160 beats per minute and blood pressure was 154/95 mmHg. 12-lead electrocardiogram (ECG) showed a wide complex tachycardia with a slightly irregular rhythm. Delta waves were also present. She was treated appropriately with intravenous procainamide but developed torsades de pointes secondary to prolonged QT interval. Electrophysiology study revealed atrial tachycardia with a left-sided accessory pathway which was successfully ablated.

Keywords: accessory pathway, long QT, procainamide, wide complex tachcardia, Wolff-Parkinson-White syndrome

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 200505B)

	True	False
Question 1. The appropriate pharmacotherapy for atrial fibrillation in Wolff-Parkinson-White syndrome is :		
(a) Verapamil.	❑	❑
(b) Atenolol.	❑	❑
(c) Procainamide.	❑	❑
(d) Digoxin.	❑	❑
Question 2. Which of the following drugs may cause prolonged QT interval?		
(a) Procainamide.	❑	❑
(b) Aspirin.	❑	❑
(c) Quinidine.	❑	❑
(d) Sotalol.	❑	❑
Question 3. The potential complication of drug-induced prolonged QT interval is:		
(a) Sinus tachycardia.	❑	❑
(b) Torsades de pointes.	❑	❑
(c) Atrial flutter.	❑	❑
(d) Atrial tachycardia.	❑	❑
Question 4. In patients with haemodynamically compromised atrial fibrillation with Wolff-Parkinson-White syndrome, the immediate treatment is:		
(a) Intravenous amiodarone.	❑	❑
(b) Intravenous adenosine.	❑	❑
(c) Intravenous verapamil.	❑	❑
(d) Direct current cardioversion.	❑	❑
Question 5. Inappropriate treatment for atrial fibrillation in Wolff-Parkinson-White syndrome would result in:		
(a) Ventricular fibrillation.	❑	❑
(b) Sinus tachycardia.	❑	❑
(c) Torsades de pointes.	❑	❑
(d) Atrial flutter.	❑	❑

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