An avoidable cause of life-threatening arrhythmia

A strange ECG

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Case presentation

The 84-year-old patient, who lived abroad, was known to have ischaemic heart disease with an old posterobasal transmural infarction. He had been treated for 12 months with oral verapamil for supraventricular ectopic beats with left bundle-branch block. His cardiologist had recently changed his antiarrhythmic medication after documenting an increase in the prevalence of ectopic supraventricular beats. Two weeks later, the patient suddenly lost consciousness during a walk.

On site, a tachycardia of 135 bpm was documented with haemodynamic instability, and the patient was admitted to the emergency department. The ECG on arrival is shown in figure 1.

Question

What can explain this arrhythmia?

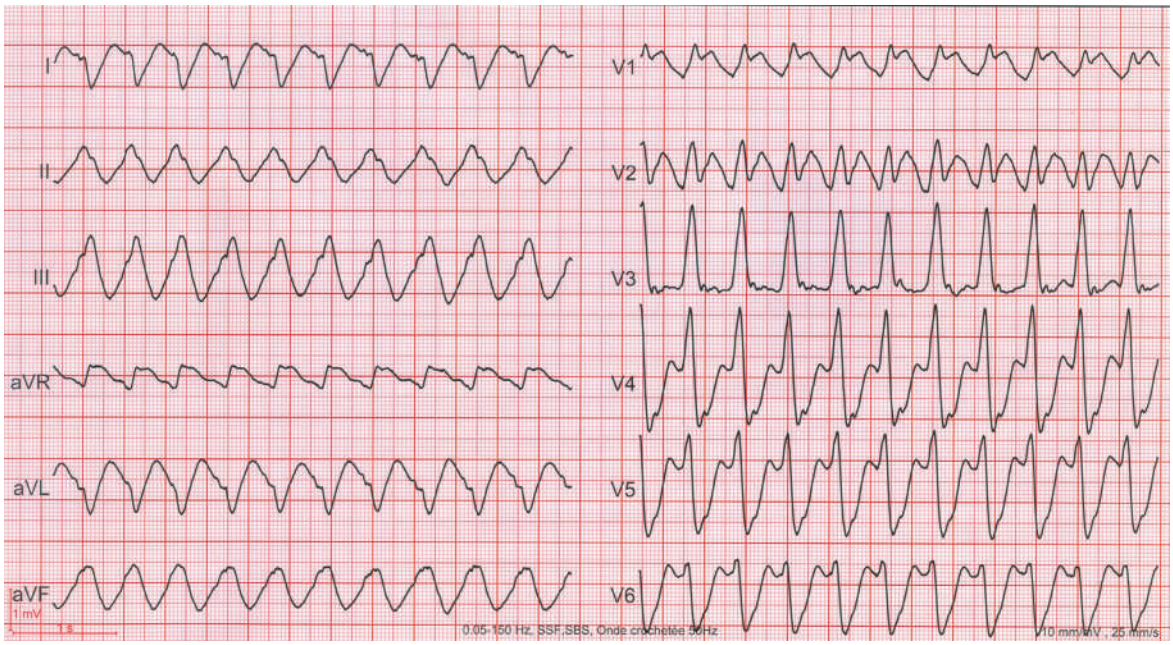

Figure 1: The 12-lead ECG on admission.

Comment

The ECG on arrival in the emergency department (fig. 1) shows a very wide QRS tachycardia, regular at 130 bpm (QRS 200 ms; right axis deviation). The QRS complexes have an undefined aspect (neither right nor left bundle branch morphology); QRS beginning and end are difficult to pinpoint precisely (a sinusoidal-like pattern); there is no visible P wave.

Such large and deformed QRS complexes should first suggest either severe hyperkalaemia or the effects of antiarrhythmic drugs. The patient's wife was able to inform the medical team that the medication introduced recently by the cardiologist was flecainide 2× 100 mg/day. Thus, the diagnosis was ventricular tachycardia due to a proarrhythmic effect of flecainide. The laboratory results were not relevant.

The patient was cardioverted electrically and the ECG immediately thereafter shows atrial fibrillation with a slow ventricular rate, a wide left bundle-branch block (190 ms) and left axis deviation (fig. 2).

During the following days, the QRS progressively nar-

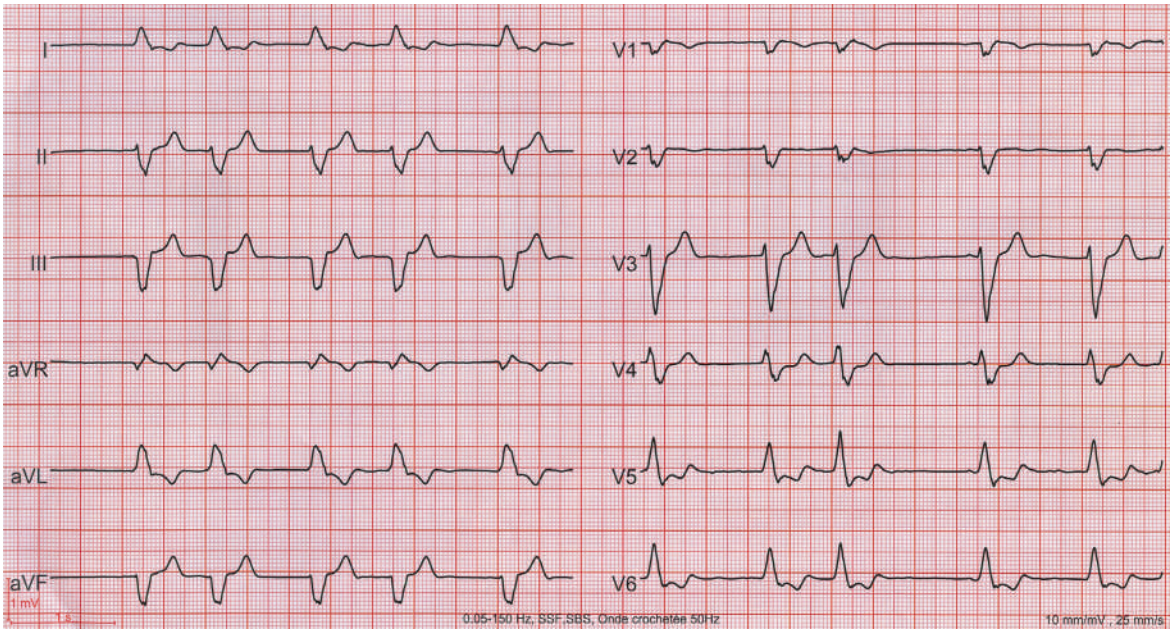

Figure 2: ECG after electrical cardioversion.

rowed to 120 ms and the patient spontaneously cardioverted to sinus rhythm. The investigations performed thereafter showed no active myocardial ischaemia; the left ventricular ejection fraction was measured at 40%. Our efforts to reassure the patient were ineffective: he lost confidence and remained extremely anxious. Despite his only moderate left ventricular dysfunction the patient was finally fitted with a cardioverter-defibrillator.

This case demonstrates once more the proarrhythmic effect of flecainide in patients with ischaemic cardiopathy. Flecainide, a class 1c sodium channel blocker, results in significant rate- and dose-dependent slowing of conduction, mainly in His–Purkinje and ventricular myocardial fibres and preferentially in ischaemic myocardium [1, 2]. On the ECG it prolongs the PR and QT intervals and the QRS complex.

In 1989, the Cardiac Arrhythmia Suppression Trial (CAST) showed excess mortality or nonfatal cardiac arrest rate among postmyocardial infarction patients treated with encainide or flecainide, as compared with placebo-treated patients [3]. Class 1c ventricular proarrhythmic effects facilitate the induction of reentry and can manifest as monomorphic sinusoidal wide QRS tachycardia (as in the present case), or as polymorphic ventricular tachycardia or fibrillation. Decreased left ventricular function, ventricular scar tissue, too high a dose and/or rapid dose increases are factors associated with proarrhythmia risk. Premonitory signs on ECG include excessive increases in QRS duration [2].

The CAST results rapidly led to a contraindication for the use of class Ic sodium channel blockers after myocardial infarction [4]. However, this warning was not respected in the present case, leading to a life-threatening proarrythmic effect.

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