An Irregular Wide Complex Tachycardia

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ECG CHALLENGE

A 66-year-old woman with a history of hypertension and persistent atrial fibrillation was referred to the Arrhythmia Clinic. Before her visit, a routine 12-lead ECG was ordered (Figure 1). The patient was asymptomatic except for minor palpitations. Her medicine regimen included apixaban, flecainide 150 mg twice daily, lisinopril, loratadine, metoprolol, and simvastatin.

What is the cardiac rhythm and what should be the initial treatment?

Please turn the page to read the diagnosis.

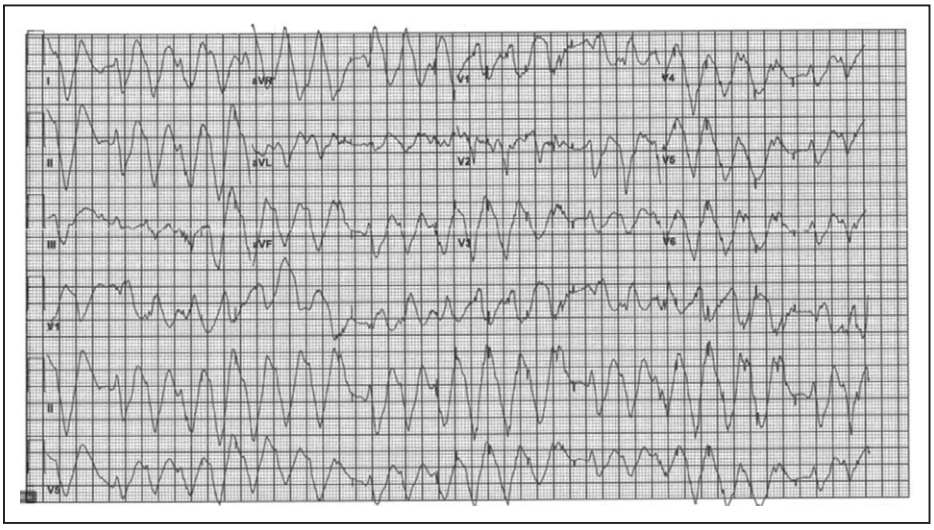

Figure 1. Routine 12-lead ECG recorded before a clinic visit to the electrophysiology clinic.

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Key Words: drug toxicity ■ flecainide ■ ventricular tachycardia

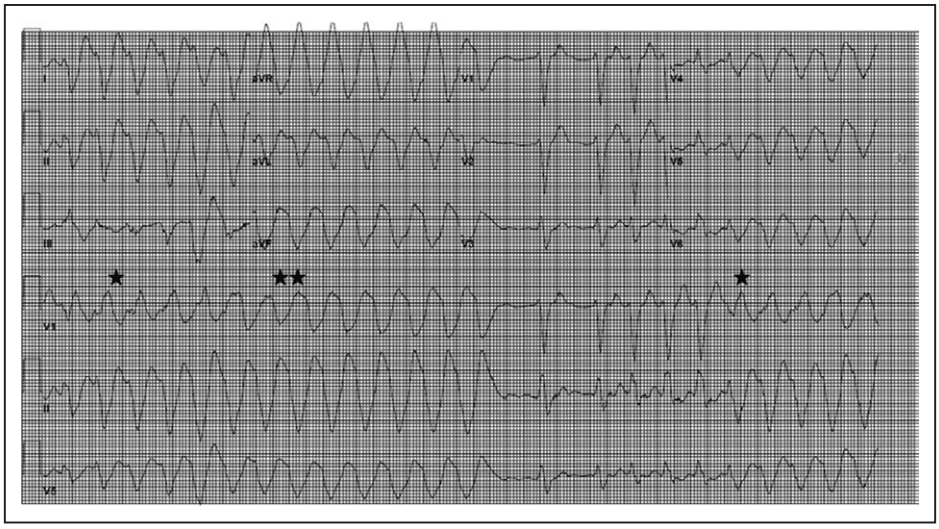

Figure 2. A 12-lead ECG recorded within minutes after the ECG in Figure 1, demonstrating ventricular tachycardia with an upright QRS in lead V_1 with variable morphology in lead III for 6 beats (*).
The ventricular tachycardia morphology spontaneously changes to an inverted QRS in lead V_1 for 7 beats (**). Following spontaneous termination of the ventricular tachycardia, the underlying rhythm is atrial fibrillation with an atypical left bundle-branch block conduction. This is followed by the occurrence of 3 beats of an upright QRS ventricular tachycardia in lead V_1 for 3 beats (*) that changes to an inverted QRS in lead V_1. The patient had only minor palpitations during this period. This ECG is consistent with a pleomorphic ventricular tachycardia that is a proarrhythmic complication of flecainide therapy.

RESPONSE TO ECG CHALLENGE

The ECG shows a very wide QRS complex tachycardia (QRS duration 280 ms) with variation in morphology. Following a pause of 700 ms, there are repeating sequences of 6 to 7 beats with cycle lengths of 400 to 415 ms. A second ECG demonstrated that the QRS morphology changed from a right bundle-branch block type pattern to a left bundle-branch block type pattern in lead V_1 (Figure 2). On interruption of the tachycardia, atrial fibrillation with an atypical left bundle-branch block was observed followed by resumption of the wide complex tachycardia. These features are consistent with flecainide-induced, pleomorphic ventricular tachycardia.

An intravenous bolus of sodium bicarbonate was given in the clinic with return to atrial fibrillation (Figure 3). Prior medical records showed QRS duration of 84 ms before initiating flecainide with a normal echocardiogram and nuclear stress test. On admission to the hospital, the serum flecainide level measured 1.8 μg/mL.

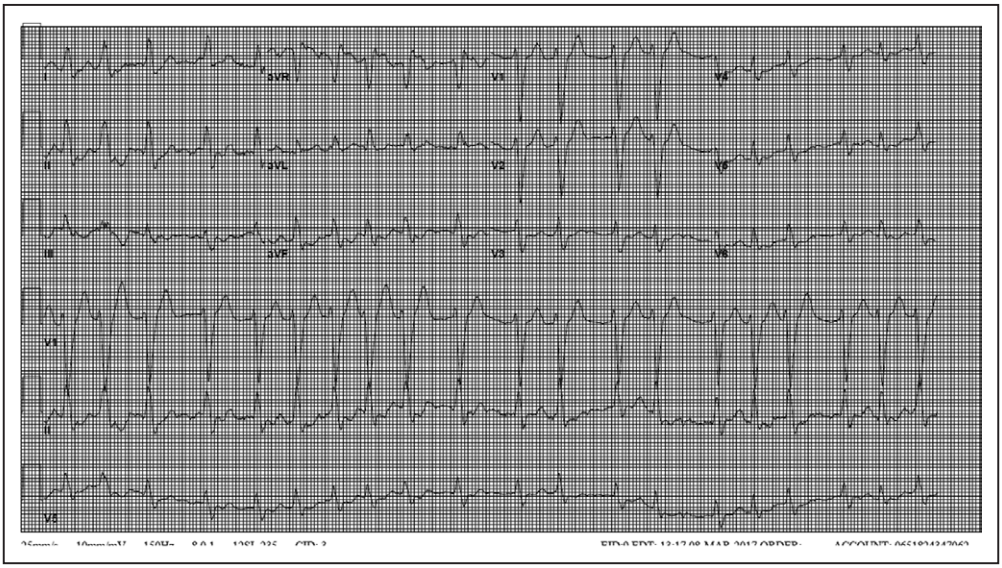

Figure 3. The 12-lead ECG recorded immediately following an intravenous bolus of sodium bicarbonate.
The underlying rhythm is atrial fibrillation conducted with left bundle-branch block. The QRS complex remains prolonged with a duration of 180 ms, probably because of the effect of flecainide on conduction in the left bundle branch because the QRS had been normal (84 ms in duration) before initiation of flecainide.

Flecainide has a high affinity for open-state Na^+ channels with slow unbinding kinetics. Flecainide also blocks the rapid component of the delayed rectifier current (I_{Kr}) and reduces spontaneous sarcoplasmic reticulum Ca^{2+} release by inhibiting ryanodine receptors. The mechanism of flecainide-induced ventricular tachycardia is uncertain. In canine studies, ventricular tachycardia (VT) was inducible with programmed stimulation after flecainide loading in 4 of 13 healthy dogs (31%) in comparison with 15 of 19 dogs after myocardial infarction.[1] Spontaneous VT occurred in 8 of 19 dogs with prior myocardial infarction but in no healthy dogs.[1] Rate-dependent conduction block transverse to fiber orientation was observed.[1] In the present patient, the first and subsequent beats of each sequence of VT were similar in morphology, suggesting a focal mechanism, consistent with ECG and action potential recordings from isolated perfused guinea pig hearts treated with flecainide.[2] Flecainide prolongs action potential duration significantly more in the left than in the right ventricle, thereby increasing action potential duration dispersion. In animal models, the ECG morphology of the first and subsequent beats of spontaneous VT had a consistent morphology suggesting a focal mechanism.[2] In the present case, the spontaneous change from a right bundle-branch block to a left bundle-branch block QRS pattern during longer episodes of VT is consistent with either reentry or induction of a second focal mechanism.

VT induced by class 1C agents may be resistant to direct current cardioversion. Sodium bicarbonate is effective by increasing serum sodium concentration, thereby competing with the drug for negatively charged sodium channels. In addition, alkalinization shifts more of the drug to the negatively charged state, thereby reducing its entry into sodium channels.[3]

Our patient was treated by discontinuation of flecainide and catheter ablation of atrial fibrillation.

DISCLOSURES

None.

AFFILIATION

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FOOTNOTES

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REFERENCES

1. Ranger S, Nattel S. Determinants and mechanisms of flecainide-induced promotion of ventricular tachycardia in anesthetized dogs. *Circulation*. 1995;92:1300–1311.
2. Osadchii OE. Flecainide-induced proarrhythmia is attributed to abnormal changes in repolarization and refractoriness in perfused guinea-pig heart. *J Cardiovasc Pharmacol*. 2012;60:456–466. doi: 10.1097/FJC.0b013e31826b86cf.
3. Bou-Abboud E, Nattel S. Relative role of alkalosis and sodium ions in reversal of class I antiarrhythmic drug-induced sodium channel blockade by sodium bicarbonate. *Circulation*. 1996;94:1954–1961.

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