Long-QT Syndrome Induced by the Antiaddiction Drug Ibogaine

TO THE EDITOR: Anecdotal evidence suggests that ibogaine alleviates drug craving and relapse of drug use in humans, as has been confirmed for animals.[1] Ibogaine is currently used as an antiaddiction drug in alternative medicine. In 1993, the Food and Drug Administration approved a clinical trial in humans to study those effects. The National Institute on Drug Abuse decided not to fund this study because of safety issues.[2,3]

Ibogaine is a naturally occurring alkaloid with hallucinogenic and psychedelic effects, derived from the bark of the root of the West African *Tabernanthe iboga* plant and is used in Gabonian initiation ceremonies. At least 11 sudden deaths were described after ibogaine use, in which the cause of death remained unclear even after autopsy.[4] It was hypothesized that ibogaine might dysregulate the autonomic nervous system, causing sudden death.[5]

A 31-year-old American woman was admitted to our emergency department because of a seizure-like attack after she had taken a single dose of 3.5 g of ibogaine 15% (usual dose, 2 to 6 g). She had not taken any other drugs or alcohol concurrently with the ibogaine. Her medical history was unremarkable, and there was no family history of cardiac-rhythm abnormalities. Besides nausea, she had no specific symptoms to report. She had come to the Netherlands to receive ibogaine as an alternative medicine for treatment-resistant alcohol addiction.

Electrocardiography showed a severe prolonged QT interval of 548 msec (QT interval corrected for the heart rate, 616 msec) and ventricular tachyarrhythmias during prolonged monitoring (Fig. 1). Laboratory findings revealed mild hypomagnesemia (magnesium level, 0.49 mmol per liter [1.2 mg per deciliter]; reference range, 0.70 to 1.00 [1.7 to 2.4]), hypokalemia (potassium level, 3.2 mmol per liter [12.5 mg per deciliter]; reference range, 3.8 to 5.0 [14.9 to 19.5]), and a normal serum osmolal gap (3.1 mOsm per kilogram; reference value, <10). Despite rapid correction of the electrolyte levels, the QT interval remained prolonged. During admission to the intensive care unit, with no further doses of ibogaine given, the QT interval normalized at 42 hours after presentation. The patient was subsequently discharged, in good condition.

In this case, ibogaine use was associated with severe lengthening of the QT interval and ventricular tachyarrhythmias, which normalized after 42 hours. These findings are suggestive of a causal relation. The electrolyte imbalance may also have played a role. The previously described sudden deaths may thus have been caused by

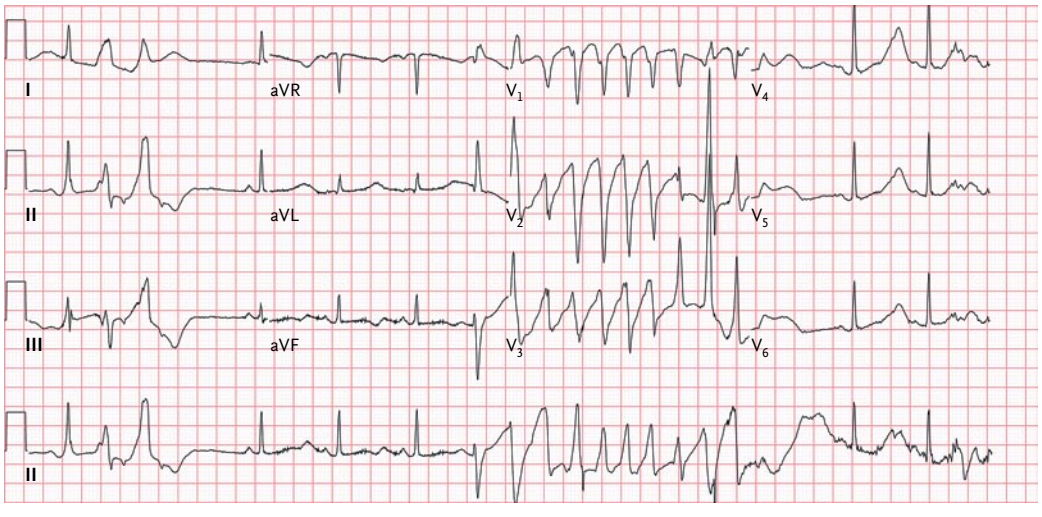

Figure 1. Electrocardiogram of the Patient, Showing Ventricular Tachyarrhythmias and a Prolonged QT Interval.

cardiac-rhythm abnormalities induced by QT-interval lengthening, ventricular tachyarrhythmias, or both.

At the doses currently used, ibogaine can lead to serious cardiac-rhythm abnormalities. The use and possible future trials of the drug should be permitted only under strict medical observation and continuous electrocardiographic monitoring.

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