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LETTER TO THE EDITOR

Ibogaine-associated ventricular tachyarrhythmias

To the Editor:

Ibogaine is an alkaloid with psychedelic effects derived from the *Tabernanthe iboga* plant and is used in alternative addiction treatment and connected to some sudden deaths.^{1,2} We present ventricular tachyarrhythmias in an ibogaine-exposed patient.

A 33-year-old man ingested a single 600 mg dose of ibogaine after 2 days of cocaine, heroin and methadone abstinence. After 30 minutes, he lost consciousness after trying to urinate due to ventricular fibrillation (VF) which was defibrillated at 200 J DC. On admittance, his vital signs and laboratory results including electrolytes were normal. Electrocardiography showed a prolonged QTc-interval of 460 ms. During the first 10 hours, he had two more VFs, one after micturition, that were defibrillated with 200 J. Amiodarone infusion of 300 mg in 30 minutes followed by 1200 mg per day was started, and he developed sinus bradycardia, a QTc-interval prolongation (510 ms) and the 4th VF which was defibrillated. During transient amiodarone withdrawal he experienced the 5th VF and the QTc transiently shortened, but after amiodarone reintroduction, the QTc-interval prolonged up to 593 ms 42 hours post-ingestion, and he suffered three monomorphic VTs between 36 and 48 hours while trying to urinate or defecate, which were electroconverted with 100 J. Forty-eight hours post-ingestion, the QTc-interval began to decrease and amiodarone was stopped on the 4th day, but the QTc-interval did not normalize until the 9th day. Ibogaine and noribogaine were detected by LC-MS/MS in his blood from day 1 to 9. The highest ibogaine level was 0.68 mg/L at the time of the last VF. No other drugs were revealed except methadone (0.04 mg/L) without EDDP on day 1. The genetic syndromes of the long QT-interval and structural heart disease were excluded.

In this patient, ibogaine was associated with VF/VT and QTc-interval lengthening, which could explain the sudden death syndrome after ibogaine. The mechanism of VF is not known, but later episodes of VT were probably due to QTc-interval prolongation. The influence of the autonomic nervous system in ventricular tachyarrhythmia is probable because 2/5 VFs and 3/3 VTs appeared at micturition and defecation, which are vagal maneuvers prolonging the QTc-interval.^{2,3,4}

VF/VTs kept repeating for 2 days, and the QTc-interval was prolonged for 9 days, which coincided with an increased

ibogaine level due to its half-time of 48 hours.⁵ Amiodarone effect on the QTc-interval is probable regarding QTc-interval decrease during amiodarone discontinuation. Nevertheless, QTc-interval prolongation was primarily associated with ibogaine and its metabolite considering the observation that after 48 hours post-ingestion, the QTc-interval was decreasing despite ongoing amiodarone therapy, which could be related to progressive lowering of the ibogaine level. Furthermore, the QTc-interval normalized only 5 days after amiodarone withdrawal when ibogaine and noribogaine became undetectable, which is consistent with the role of ibogaine in QTc-interval prolongation. Methadone could have contributed only to a slightly prolonged QTc-interval on arrival.

In conclusion, ibogaine is associated with QTc-interval prolongation and VF/VT that might be provoked by vagal maneuvers. In ibogaine exposure, amiodarone did not appear effective in VF/VT therapy, and DC shock should be used first.

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