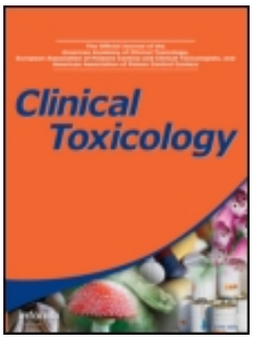

Clinical Toxicology

ISSN: 1556-3650 (Print) 1556-9519 (Online) Journal homepage: informahealthcare.com/journals/ictx20

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To cite this article: L. Vlaanderen, L. C. Martial, E. J. F. Franssen, P. H. J. van der Voort, E. Oosterwerff & G. A. Somsen (2014) Cardiac arrest after ibogaine ingestion, Clinical Toxicology, 52:6, 642-643, DOI: 10.3109/15563650.2014.927477

To link to this article: https://doi.org/10.3109/15563650.2014.927477

Published online: 18 Jun 2014.

Clinical Toxicology (2014), **52**, 642–643

ISSN: 1556-3650 print / 1556-9519 online
DOI: 10.3109/15563650.2014.927477

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LETTER TO THE EDITOR

Cardiac arrest after ibogaine ingestion

To the Editor:

We would like to address our concerns regarding the safety of ibogaine, after a case of a cardiac arrest following ibogaine use in a young, previously healthy man admitted to our hospital.

Ibogaine is an indole alkaloid derived from the West African plant *Tabernanthe Iboga*.[1] It is used by indigenous people for hallucinogenic properties and to reduce hunger and fatigue. There is special interest for its presumed anti-addictive properties. Various intoxications with ibogaine have been described, some with a fatal outcome.[2]

In our hospital, a previously healthy 26-year-old man (74 kg) presented after an out-of-hospital cardiac arrest based on ventricular fibrillation, approximately 5 h after taking 2400 mg of ibogaine for a spiritual experience. Basic life support was initiated immediately and he was defibrillated. Return of spontaneous circulation occurred after 20 min. After arriving into hospital he remained comatose (Glasgow Coma Scale [GCS] score 3, E1M1V1[tube]) on midazolam and propofol. He received therapeutic hypothermia, mechanical ventilation and fluid resuscitation. The physical examination revealed no abnormalities besides decreased GCS score. His potassium serum level was 4.0 mmol/l [ref 3.5–5.0]. No other abnormalities in blood tests were found. His ECGs showed a prolonged QTc interval from admission up to 32 h, with a maximum up to 663 ms, 18 h after his cardiac arrest. A recent ECG, performed before ibogaine ingestion, showed a normal QTc.

Toxicological screening revealed benzodiazepines (received during resuscitation in the ED) and ibogaine. The serum level of ibogaine was 948 ug/L 5 h after ingestion as analyzed using LC-MS/MS (Fig. 1).

Earlier case reports on ibogaine intoxications described a wide range of serum concentrations (i.e. 360–10800 ug/L).[3,4]

In ICU he developed seizures for which he was treated with antiepileptics (phenytoin, valproic acid and levetiracetam). A cerebral CT showed no abnormalities. After 8 days, he was transferred to another hospital with a GCS score of 4 (E1M2V1[tube]) without sedation. Several weeks later he was awake and able to communicate, but permanent cognitive and neurologic deficits (loss of vision) remained, most likely due to post anoxic encephalopathy.

We propose a direct relationship between the ventricular fibrillation and the ingestion of ibogaine, because our patient was previously healthy with a normal cardiology exam prior to the ibogaine use. There was no suspicion of other drugs/medication taken by the patient. The QTc prolongation might in part have been caused or exacerbated by propofol or hypothermia; but ibogaine is well known for its QTc prolongation.[5] Recent findings suggest that hERG channel blockade by ibogaine as partial opioid agonist may be the pharmacological mechanism of QTc prolongation.[6] The hERG channel is a potassium channel involved in repolarisation of the cardiac action potential. Mutations or blockade of this channel by pharmacologically active compounds can result in prolonged QTc.[7,8]

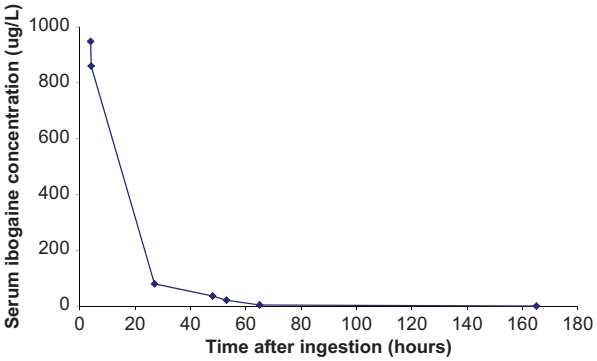

Fig. 1. Serum concentration–time curve of ibogaine in our patient (colour version of this figure can be found in the online version at www.informahealthcare.com/ctx).

Ibogaine is available in different pharmaceutical forms (powders and oral capsules in more or less purified forms). Its use is not wide spread, however, the effects can be devastating and its use should therefore be strongly discouraged.

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Declaration of interest

The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper.

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Received 25 November 2013; accepted 15 May 2014.

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