Declaration of interest

None declared.

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Growing menace of ibogaine toxicity

Editor—Ibogaine is an hallucinogenic alkaloid derived from a western African shrub. Although in recent years some degree of experimental evidence has emerged favouring the use of ibogaine in the management of substance abuse disorders, these developments have been overshadowed by concerns about its safety and several cases of unexplained sudden cardiac death have been associated with its use. Among its pleiotropic receptor effects, ibogaine has central 5HT-2A agonist activity, and through this mechanism it prolongs the QT interval.

Although ibogaine is not approved by any drug administration agency in the world, its unauthorized use in clandestine detoxification clinics is steadily growing worldwide. We describe the first case of severe ibogaine toxicity in the UK and highlight the complexities posed by its management.

A young Afro-Caribbean man was admitted to hospital with vomiting and agitation, after being found by his relatives in a state of confusion. He had ingested a total of 7 g of ibogaine to soothe the symptoms of heroin withdrawal. He denied ingestion of alcohol or any other drugs and he had not used heroin or methadone for >72 h. There was no family history of heart disease. Initial laboratory blood tests, arterial blood gas analysis, cardiac enzymes, and urine toxicology were unremarkable.

The patient was in sinus bradycardia, with marked prolongation of the QTc interval (600 ms). Several brief, self-terminating bursts of polymorphic tachycardia (VT) occurred. The VT was initially associated with tonic-clonic seizures and the patient rapidly deteriorated, losing cardiac output, and developing torsades de pointes cardiac arrest. Spontaneous cardiac output was initially restored by defibrillation (200 J), but pulseless torsades de pointes recurred despite treatment with i.v. magnesium (8 mmol), atropine (2 mg), epinephrine (4 mg), and isoprenaline (5 μg min⁻¹). The patient was defibrillated several more times and required tracheal intubation to secure his airway. Transcutaneous overdrive pacing immediately shortened the QT interval and controlled the ectopic ventricular activity. The transcutaneous pacemaker was promptly replaced with a temporary transvenous pacing wire. The patient was paced at a rate of 80 beats min⁻¹ for 48 h, without further episodes of VT. The patient’s bradycardia resolved and the QT interval spontaneously returned to 420 ms. The pacing wire was removed and the patient was weaned from mechanical ventilation. No further abnormalities were detected on subsequent ECGs and a transthoracic echocardiogram was normal. A review by a consultant cardiologist determined that no further electrophysiological testing was required. The patient had an uneventful recovery and was rapidly discharged.

This case summary represents the first report of ibogaine intoxication in the UK. In view of the growing use of this drug...
within the UK, it is important to consider ibogaine toxicity in the differential diagnosis of the acutely unwell intoxicated patient and in patients presenting with a prolonged QT interval and cardiac rhythm disturbance.

**Declaration of interest**

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**Sustained effect of spinal cord stimulation on pain and quality of life in painful diabetic peripheral neuropathy**

Editor—Painful diabetic peripheral neuropathy (PDPN) is a common complication of diabetes mellitus (DM) and may cause physical and emotional suffering with severe impact on quality of life (QoL).1 Pharmacological treatment often is only partially effective or unsuccessful due to unacceptable side-effects. Spinal cord stimulation (SCS) is considered a possible treatment modality. Recently, the short- and long-term results of SCS in PDPN were reviewed,2 showing sufficient pain relief in 15 out of 24 patients (63%) after 1 yr.3–5 After 2.5–3 yr, the percentage of patients who experienced pain relief remained 63%, although the number of patients decreased from 24 to 19.6–8 In view of these findings, we studied the long-term effectiveness of SCS on pain and QoL in patients with PDPN. The short-term results of SCS were reported earlier in a prospective open-label cohort study.7 Besides the effect of SCS on pain, effect on patients’ QoL was investigated. Complications and adverse events were registered. The study protocol was approved by the local Medical Ethics Committee, and all patients gave written informed consent.

Fifteen patients [eight male, mean age 59.9 yr (range 50–72)] with PDPN in the lower limbs met the eligibility criteria.7 A 2 week trial stimulation with an octapolar lead (Octad® lead, Medtronic, Minneapolis, MN, USA) was performed to evaluate sufficient pain relief before definitive SCS system implantation (Synergy Versitrel®, Medtronic) as described elsewhere.7 Eleven patients [seven male, mean age 60.9 yr (50–72)] received a definitive SCS system after positive trial stimulation. The primary outcome parameters were pain intensity, scored with a numeric rating scale, and patients’ global impression of change (PGIC) scale at 12, 24, and 36 months. Successful treatment was defined as ≥ 50% decrease in pain intensity at daytime and/or night-time, and/or peak pain and/or significant improvement of painful symptoms measured with the PGIC.7 Additionally, health-related QoL was assessed using the EuroQol-5 dimensions (EQ-5D) questionnaire.8–9

Four patients had mild neuropathy and seven had moderate-to-severe neuropathy.10 The duration of DM was 18.3 (SD 20.6) yr, the mean duration of neuropathy and of painful symptoms was 11.0 (SD 16.0) and 5.3 (SD 3.4) yr, respectively.

The group of 11 patients showed a significant pain reduction during daytime and night-time and peak pain at all time points compared with baseline, except for night-time pain after 24 months (Fig. 1). The majority of patients showed a significant

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**Fig 1** Scores for daytime pain intensity and QoL during follow-up. Presented are the median (50th percentile) and the interquartile range (IQR) (25th and 75th percentile).