



Ibogaine. A new nightmare for cardiologists in tourist cities?

Ibogaina. ¿Un nuevo reto para los cardiólogos en ciudades turísticas?

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Palabras clave:

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Substance abuse is a growing global health problem, and with it, the need to find medications or other substances that aid in the detoxification process or relieve symptoms associated with withdrawal syndrome. The abuse of kratom is gaining interest in the search for treatments that facilitate detoxification, marketed as a «natural» product for pain management or for reducing opioid use itself; it is a substance derived from a tropical tree native to Southeast Asia that appears to have properties similar to opioids, its main psychoactive constituents, mitragynine and 7-hydroxymitragynine, produce stimulant effects at low doses and sedative and analgesic effects at higher doses.

Prolonged kratom use may lead to tolerance, physical dependence, and withdrawal syndrome.¹ Currently, there are kratom detoxification tourism clinics at vacation points, utilizing ibogaine, a psychoactive alkaloid derived from *Tabernathe iboga*, used for its anti-addiction effects in various substance use disorders. Ibogaine and its primary metabolite, noribogaine, have anti-addiction effects by dual inhibition of the vesicular monoamine transporter-2 and the serotonin transporter, both of which are synaptic reuptake inhibitors.

Additionally, they exert modulatory effects on multiple neurotransmitter systems and exhibit micromolar affinity for the N-methyl-D-aspartate receptor and the κ -, μ -, and sigma-2 opioid receptors. Ibogaine also interacts with the acetylcholine, serotonin, and dopamine

systems. It alters the expression of proteins such as substance P, brain-derived neurotrophic factor, early growth response-1, and the proto-oncogene c-Fos, with a risk of cardiotoxicity, including QT interval prolongation and the onset of potentially fatal arrhythmias such as torsades de pointes.

After oral administration, ibogaine undergoes rapid and extensive metabolism, primarily via cytochrome P450 2D6 (CYP2D6), to its main active metabolite, noribogaine (12-hydroxyibogamine), which persists at clinically relevant concentrations for days after ibogaine plasma clearance.² The cause of the cardiotoxicity lies in the effect on specific types of channels. It acts by blocking the human ether-à-go-go-related gene potassium channels in cardiac myocytes, which are essential for the repolarization phase of cardiac action potentials, delaying ventricular repolarization, prolonging the QT interval on the electrocardiogram resulting in increased dispersion of ventricular repolarization, predisposing to polymorphic ventricular tachycardias and sudden cardiac arrest,³ even at therapeutic doses and in individuals with no history of cardiovascular disease; therefore, the focus should be on finding a safe way to administer this type of medication.

In the last month, we attended two ibogaine-intoxicated patients at Cozumel COSTAMED/ The Clinics of the Heart, both men aged 30-40 years, derived from kratom-detoxification clinics; the first one, a 34-year-old, after

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successful resuscitation from cardiac arrest, with evident recurrent torsades de pointes and QT interval > 600 ms, and the second with QT interval > 600 ms without arrhythmia; the first case with requested voluntary discharge still on long QT and the second with confirmed normal QT after 72 hours.

Efforts are underway to enhance ibogaine safety, including optimized dosing strategies, continuous electrocardiographic monitoring during administration to detect arrhythmia risk, and the development of analogs that maintain anti-addiction efficacy without cardiotoxic effects in preclinical models. These initiatives aim to provide safer detoxification options, address clinicians' concerns about adverse cardiac events, and guide future clinical practice.⁴ Meanwhile, it looks like kratom-ibogaine detox tourism may become a new nightmare for cardiologists in tourist destinations.

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