CASE

Transient Paralysis

A Novel Expression of Kratom Toxicity in Humans

Harold A. Matos-Casano, MD, and Sunanda Nanduri, MD

Neurology: Clinical Practice February 2021 vol. 11 no. 1 e28-e29 doi:10.1212/CPJ.0000000000000751

Correspondence
Dr. Matos-Casano
hcasano@
wyckoffhospital.org

Kratom (*Mitragyna speciosa*) use has become a growing public health concern. Its increasing popularity in Western countries has been concomitant with a rise in reports of adverse events, including some fatalities, which are possibly related to the use of adulterated formulations. Its pharmacology, clinical profile, testing, and the treatment of abuse-related complications have not been conclusively elucidated.[1] Kratom has been used as a muscle relaxant, an effect that could be mediated primarily by neuromuscular blockade.[2] It is possible that at toxic doses, kratom ingestion may result in transient paralysis through this mechanism. To our knowledge, this expression of kratom toxicity is rare.

PRACTICAL IMPLICATIONS

Intoxication with kratom, an emerging drug of abuse, may present with transient paralysis. Clinicians should be aware of this drug and its deleterious effects.

Case

A 39-year-old woman with no previous medical conditions reported experiencing lightheadedness, palpitations, and generalized weakness, which worsened over the course of minutes. She then turned pale and was completely unresponsive to external stimuli. A witness described the patient as being paralyzed or in a "catatonic" state, and with a pulse greater than "130" beats per minute. The patient reported being fully aware of her surroundings but unable to execute any movements or speak. The condition resolved spontaneously after 1 hour. The patient reported experiencing self-resolving flu-like symptoms over the 3 days preceding these events and denied using any medications, trauma, toxic habits, and past similar occurrences.

At the hospital, she was mildly nauseous but otherwise asymptomatic. Mild diaphoresis and a pulse of 121 beats per minute were the only notable findings on physical examination. The neurologic examination was unremarkable.

Except for a potassium of 3.3 mmol/L, calcium of 8.0 mg/dL, and lactic acid of 2.4 mmol/L, the rest of her laboratory test results were normal. These included thyroid-stimulating hormone, beta-human chorionic gonadotropin, toxicology (screening for methadone and other opiates, benzodiazepines, amphetamines, phencyclidine, and barbiturates), urinalysis, erythrocyte sedimentation rate, herpes simplex virus, HIV, hepatitis, influenza, and CSF studies. Electrocardiogram revealed sinus tachycardia. Noncontrast CT of the head, CT angiogram of the head and neck, and chest x-ray were normal. Rapid Group A *Streptococcus* testing was positive.

On further questioning, the patient admitted to using kratom within 1 hour of becoming paralyzed. She began using it a few months earlier as an herbal supplement and changed to a new formulation sometime before these events. She was discharged after a period of observation and supportive treatment.

Department of Internal Medicine (HAM-C), Wyckoff Heights Medical Center, Brooklyn, NY; and Neurology Division (SN), Wyckoff Heights Medical Center, Brooklyn, NY.

Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

Discussion

Kratom has been used traditionally in Southeast Asia for its stimulant and medicinal properties. In the West, it is consumed recreationally for the self-treatment of opioid dependency and other uses.[1] Some products may have inconsistent or artificially elevated alkaloid concentrations.[1,3] Others may be contaminated with toxic metals or *Salmonella*.[4] These facts raise serious concerns about the consumption of kratom products, as their dose and quality are often unknown. Kratom is mostly consumed by middle-aged, middle class individuals.[3]

Mitragynine and 7-hydroxymitragynine are kratom's principal psychoactive alkaloids, acting on neuronal Ca^{2+} channels, α-2 adrenergic, μ-opioid, 5-hydroxytryptamine, and other receptors.[5] The drug reaches maximum plasma concentration approximately 1 hour after ingestion.[6]

The diagnosis of kratom intoxication may be difficult without a proper history, as it presents most commonly with nonspecific complaints. Tongue numbness has been suggested as a marker of ingestion.[6] Jerky limb movements, diaphoresis, liver injury, seizures, and tachycardia may be observed as well. Standard drug screens cannot detect its metabolites, precluding laboratory confirmation of its use. Treatment involves using drugs that target the mechanism underlying a given toxic effect.[1]

The use of a kratom compound on a rat diaphragm preparation found that its alkaloids can block the neuromuscular junction, decrease muscle twitch, and impair nerve conduction.[2] It is unclear at which doses these effects could be replicated in humans. Apparently, the patient continued to breathe normally while paralyzed. A differential in sensitivity between the laryngeal and diaphragmatic muscles as compared to more peripheral ones has been observed with other neuromuscular blockers and may underlie this phenomenon. Size discrepancies between nerve fiber and motor endplate, higher acetylcholine receptor density, and other variations may account for this differential in sensitivity to neuromuscular blockade.[7]

Unfortunately, we were unable to identify the exact source of kratom in this case, as the information was not revealed on request. Nonetheless, the likelihood of accidental exposure to higher doses of the drug after changing formulations, the temporal relationship between drug ingestion and paralysis, absence of other suspicious exposures, and a mostly unremarkable workup suggest that kratom intoxication is the most likely cause of the clinical picture described in this report.

Study Funding

No targeted funding reported.

Disclosure

The authors report no disclosures relevant to the manuscript. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

Publication History

Received by *Neurology: Clinical Practice* July 9, 2019. Accepted in final form September 12, 2019.

Appendix Authors

Name	Location	Contribution
Harold A. Matos-Casano, MD	Wyckoff Heights Medical Center, Brooklyn	Participated in the drafting of the work and contributed to the acquisition, analysis, and interpretation of data for the work and gave final approval for the version to be published
Sunanda Nanduri, MD	Wyckoff Heights Medical Center, Brooklyn	Participated in the drafting of the work and contributed to the acquisition, analysis, and interpretation of data for the work and gave final approval for the version to be published

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