JACC: CASE REPORTS VOL. 30, NO. 5, 2025
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RHYTHM DISORDERS

CLINICAL CASE

Kratom Cardiotoxicity

Reversible Brugada Pattern and QTc Prolongation

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ABSTRACT

Kratom is derived from *Mitragyna speciosa* and contains active alkaloids that bind opioid, alpha-2 adrenergic, and 5-HT2A receptors. In vitro studies show kratom can inhibit myocardial potassium channels. We present a patient who developed a reversible Brugada pattern and QT prolongation after self-treating his attention-deficit/hyperactivity disorder with kratom. (JACC Case Rep. 2025;30:103109) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 25-year-old male patient was chronically self-medicating for anxiety and attention-deficit/hyperactivity disorder with kratom. He presented to an academic tertiary care emergency department after experiencing witnessed seizure-like activity. He reported ingesting kratom daily for the past 4 years. He estimated that his recent daily intake was approximately 84 g to 100 g. Thirty minutes after his last dose of kratom, he experienced approximately 2 minutes of witnessed seizure-like activity. Emergency medical services was called, and he was transported to the emergency department. His initial heart rate was 97 beats/min and blood pressure was 122/86 mm Hg. His initial electrocardiogram revealed a type 1 Brugada pattern with QRS of 160 ms and QTc of 654 ms (**Figure 1**). He had a potassium of 3.2 mmol/L and calcium of 8.4 mg/dL (**Table 1**), undetectable ethanol level, and negative urine drug screen. His physical exam was notable only for a small tongue abrasion. His neurologic and cardiac exams were normal, and he did not have evidence of an ongoing toxidrome.

TAKE-HOME MESSAGES

- Kratom use can lead to conduction and repolarization abnormalities through potassium channel inhibition.
- This case highlights that a Brugada-like pattern can be one of the manifestations of these kratom-induced abnormalities.

PAST MEDICAL HISTORY

The patient had a history of anxiety and attention-deficit/hyperactivity disorder. He engaged in vaping menthol nicotine using electronic cigarettes but denied any changes to his vaping habits or use of other recreational drugs. There was no family history of

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received July 12, 2024; revised manuscript received September 30, 2024, accepted October 21, 2024.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2024.103109

significant cardiac disease, unexplained drownings, unexpected sudden death, or dysrhythmias.

DIFFERENTIAL DIAGNOSIS

The patient's presentation was concerning for effects from kratom ingestion. We were also concerned a contaminant in the kratom ingested caused his symptoms, as he had recently purchased kratom from a new vendor. We also considered additional medical causes for his presentation, such as genetic channelopathy, myocarditis, myocardial ischemia, electrolyte abnormality, arrhythmogenic right ventricular cardiomyopathy, hypertrophic obstructive cardiomyopathy, and epilepsy.[1] There are a wide range of xenobiotics that cause abnormal QRS complex and QT prolongation, which include bupropion, tricyclic antidepressants, antipsychotics, diphenhydramine, and kratom.[2-6] Drug-induced Brugada syndrome is rare, but has been reported with administration of tricyclic antidepressants, lithium, first-generation antihistamines, cocaine, bupivacaine, verapamil, propofol, and kratom.[2]

INVESTIGATIONS

During his admission, serial electrocardiograms showed progressive shortening of his QRS and QT/QTc intervals, and resolution of the Brugada pattern (**Figure 2A-C**). His only previous electrocardiogram, performed 8 years before this presentation (**Figure 3**), demonstrated an incomplete right bundle branch block and normal intervals (QRS 94 ms and QTc 417 ms) but no Brugada pattern. A transthoracic echocardiogram showed no structural abnormalities. He had normal left ventricular size, normal wall thickness, no wall motion abnormalities, and an ejection fraction of 60%. His pulmonary arterial pressures were normal with an estimated peak systolic pressure of 13 mm Hg. There was no significant valvular stenosis or regurgitation.

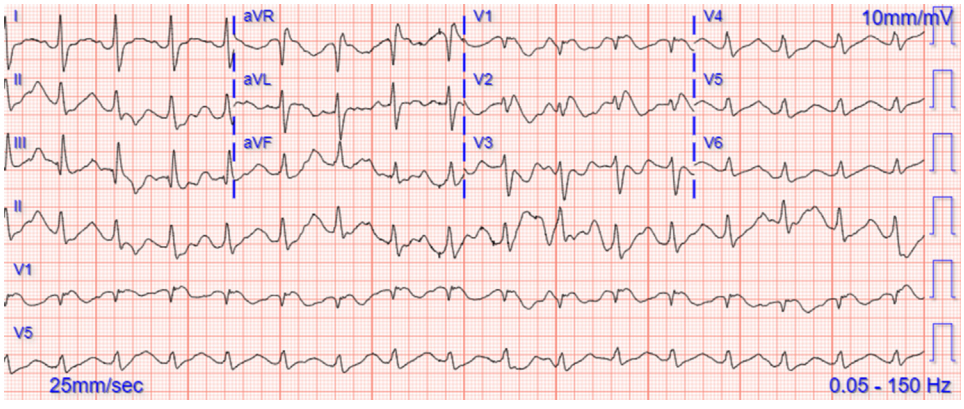
Brain magnetic resonance imaging revealed no intracranial abnormalities. He did not experience any additional seizure-like episodes, and an electroencephalogram did not capture any epileptiform discharges suggesting the seizure-like activity witnessed was related to cardiac syncope.

The patient's kratom was analyzed by gas chromatography mass spectrometry and liquid chromatography quadrupole time-of-flight mass spectrometry at the Center for Forensic Science Research and Education (Horsham, Pennsylvania, USA).[7,8] Data processing against a large-in-house database containing more than 1,100 drugs was conducted. Only kratom alkaloids mitragynine, paynantheine, and speciogynine were detected; no other drugs or analytes were identified (**Figure 4**).

MANAGEMENT

In the emergency department, he received 50 mEq of 8.4% sodium bicarbonate for his prolonged QRS. He also received a sodium bicarbonate infusion for

FIGURE 1 Initial Electrocardiogram



The electrocardiogram obtained on presentation, 4 hours after last kratom ingestion, demonstrates sinus rhythm with a Brugada pattern present.

TABLE 1 Selected Labs 4 Hours After Kratom Ingestion

	Result	Reference Range
pH (venous blood gas)	7.336	7.350-7.450
Sodium	138 mmol/L	134-143 mmol/L
Potassium	3.2 mmol/L	3.5-5.1 mmol/L
Chloride	102 mmol/L	98-107 mmol/L
Carbon dioxide	24 mmol/L	21-31 mmol/L
Anion gap	12 mmol/L	3-15 mmol/L
Glucose	136 mg/dL	70-125 mg/dL
Blood urea nitrogen	8 mg/dL	8-30 mg/dL
Creatinine	0.92 mg/dL	0.70-1.10 mg/dL
Albumin	4.5 g/dL	3.7-4.8 g/dL
Aspartate aminotransferase	20 U/L	13-39 U/L
Alanine aminotransferase	16 U/L	7- 52 U/L
Alkaline phosphatase	81 U/L	38-120 U/L
Calcium	8.4 mg/dL	8.6-10.3 mg/dL
Magnesium	1.6 mg/dL	1.6-2.5 mg/dL
High-sensitivity troponin	10 ng/L	<20 ng/L
Thyroid-stimulating hormone	3.249 μIU/mL	

3 hours (150 mEq in 5% dextrose and water at 100 mL/h), 1 g of calcium gluconate, and 2 g of magnesium sulfate. The following day, he received 20 mEq of potassium chloride and 2 g of magnesium sulfate. He experienced mild withdrawal symptoms of muscle aches and anxiety from kratom cessation. He received oral clonidine with improvement of these symptoms. He was discharged home approximately 68 hours after his initial presentation.

DISCUSSION

Derived from *Mitragyna speciosa*, kratom is a plant native to Southeast Asia and Africa. It has been cultivated for several thousand years in Southeast Asia.[9] Kratom is traditionally used to treat conditions ranging from muscle pain to diabetes mellitus. More recently, kratom has been used for its effects as an aphrodisiac, opioid substitute, anxiolytic, antidepressant, stimulant, and sedative. Although the leaves of *Mitragyna speciosa* contain more than 25 alkaloids, there are 4 main pharmacologically active alkaloids: mitragynine, 7-hydroxymitragynine, corynantheidine, and speciociliatine. Mitragynine is structurally similar to yohimbine and is active at opioid receptors, and 7-hydroxymitragynine has central nervous system stimulant effects.[3,10-12] These compounds bind opioid, alpha-2 adrenergic, and 5-HT2A receptors to produce a combination of stimulant and analgesic effects.[3] Kratom is widely available in the United States as a resin, extract, powder, or dried leaves and can be purchased online or in stores.

In vitro studies have shown that kratom inhibits potassium channels in a concentration-dependent manner.[2] Mitragynine can prolong the action potential duration, which increases the risk for Torsades de Pointes.[9] Previous cases describe an association between kratom use and a variety of electrocardiogram abnormalities including prolonged QT intervals.[3] There are only 3 meeting abstracts reporting kratom use unmasking a Brugada pattern. In all 3 cases, no testing of the kratom to rule out contaminants was completed.[4-6] A small case-control study of Malaysian male individuals found that an average daily use of 434 mg of mitragynine was associated with increased odds of sinus tachycardia and having a borderline QTc interval.[12] The daily intake of this study population was significantly less than the daily estimated intake of 84 to 100 g in our patient.

We believe that the kratom was the cause of our patient's cardiotoxicity based on the patient's negative family history of sudden cardiac death, previously unremarkable electrocardiogram, his presenting electrocardiogram, normalization of his electrocardiogram with kratom abstinence, lack of significant electrolyte derangements to explain his electrocardiogram abnormalities, and no structural abnormalities on echocardiography. In addition, there were no contaminants detected in the kratom sample provided by the patient and the urine drug screen was negative.

FOLLOW-UP

On a follow-up call, the patient reported he was doing well, abstaining from kratom, and had no recurrence of his symptoms.

CONCLUSIONS

Kratom can cause significant cardiac conduction and repolarization abnormalities in a patient without structural anomalies or additional confounding xenobiotics.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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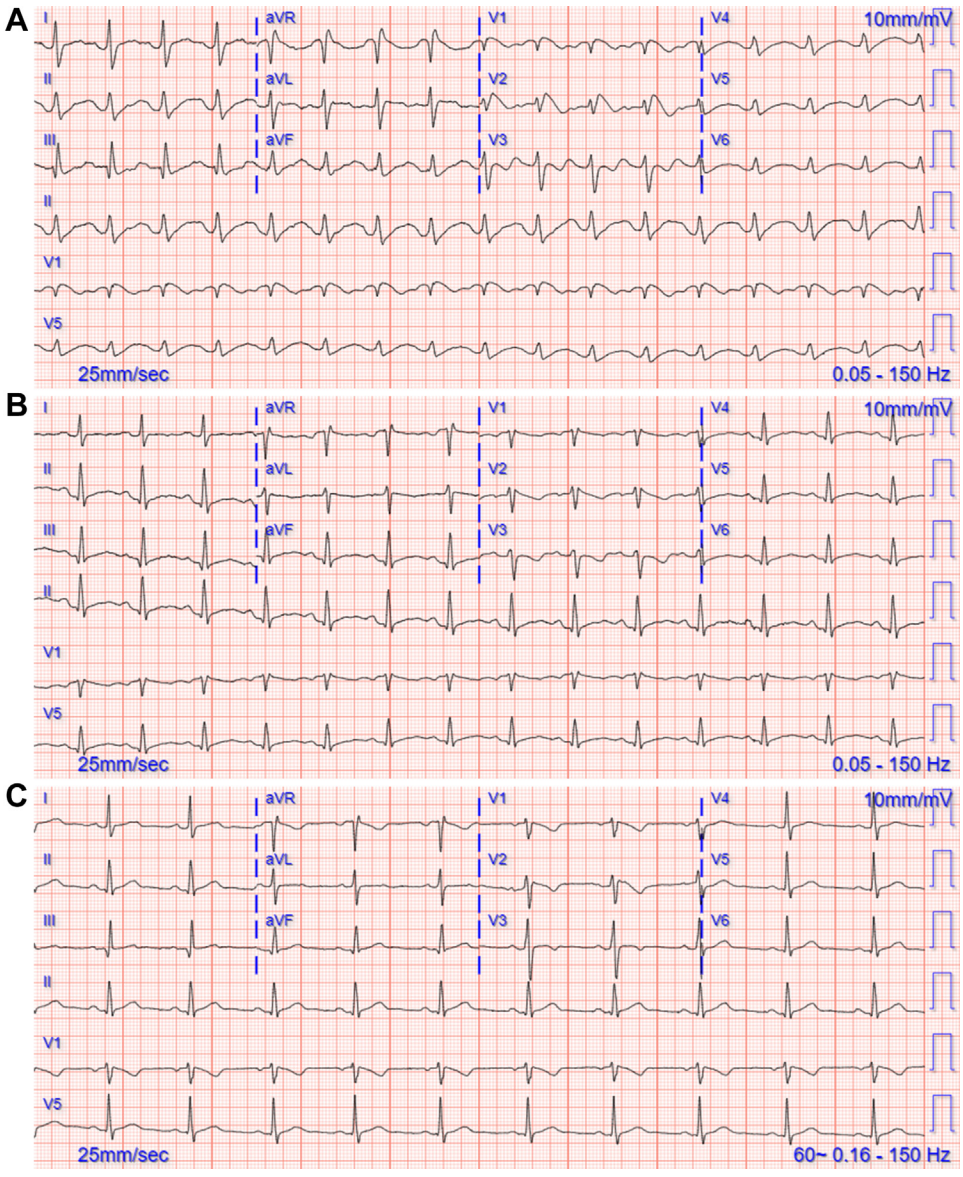
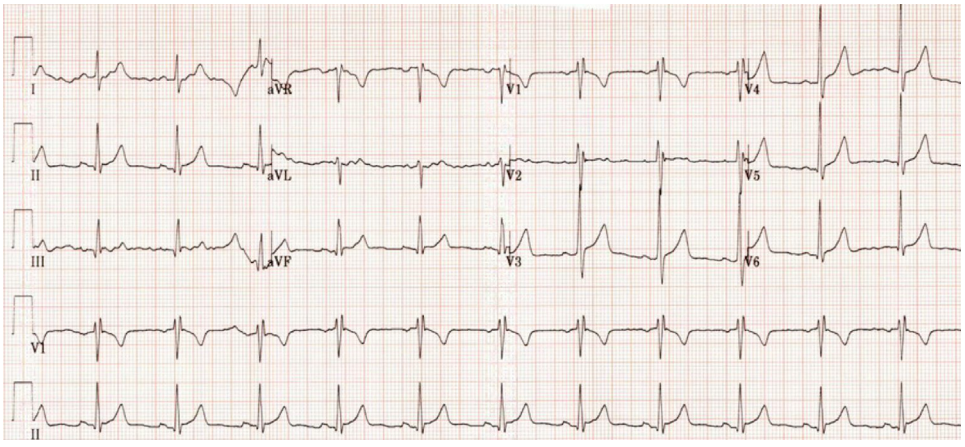
FIGURE 2 **Repeat Electrocardiograms**



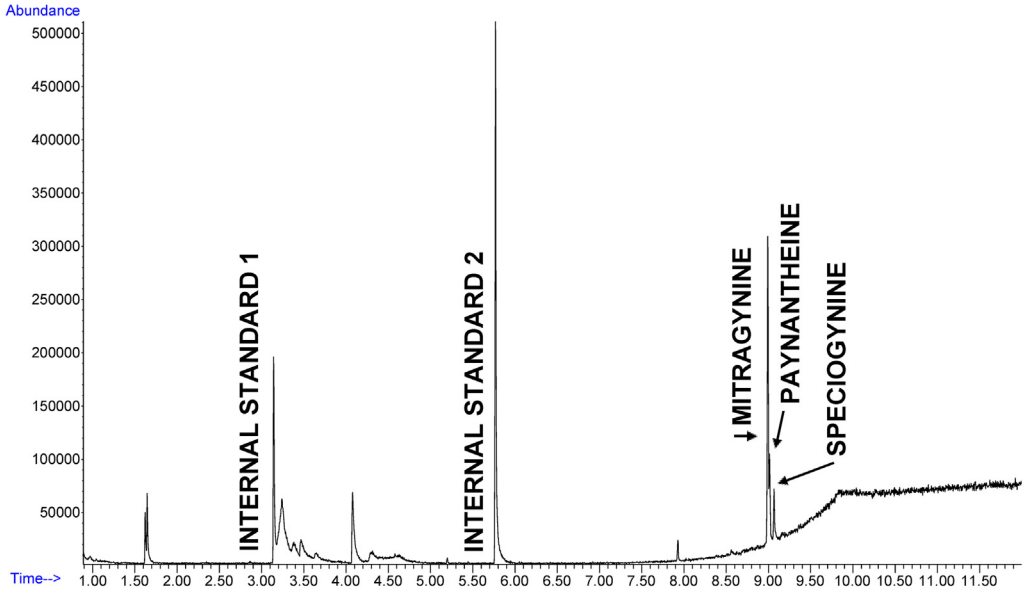
(A) Electrocardiogram obtained five hours following last kratom ingestion demonstrates sinus rhythm with continued Brugada pattern (QRS 164 msec, QTc 552 msec). (B) Repeat electrocardiogram 24 hours following last kratom ingestion demonstrates sinus rhythm with a resolving Brugada pattern (QRS 122 msec, QTc 556 msec). (C) Electrocardiogram 31 hours after last kratom ingestion demonstrates a resolving Brugada pattern (QRS 108 msec, QTc 450 msec).

FIGURE 3 Baseline Electrocardiogram



The patient's previous electrocardiogram demonstrates sinus rhythm and no Brugada pattern.

FIGURE 4 Kratom Sample Chromatogram



The patient's kratom was analyzed by gas chromatography mass spectrometry and liquid chromatography quadrupole time-of-flight mass spectrometry and only kratom was identified in the sample.

JACC: CASE REPORTS, VOL. 30, NO. 5, 2025
MARCH 5, 2025:103109

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KEY WORDS **Brugada, kratom, mitragynine, QRS prolongation, QT prolongation**