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RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CLINICAL CASE

Brugada Syndrome Unmasked by Kratom Use in a Young Man

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ABSTRACT

BACKGROUND Brugada syndrome is an inherited cardiac channelopathy causing malignant ventricular arrhythmias and sudden death in structurally normal hearts, which may be unmasked by medications or recreational substances in susceptible individuals.

CASE SUMMARY A 28-year-old man collapsed after smoking a hand-rolled cigarette later confirmed to contain *Mitragyna speciosa* (kratom). Bystander cardiopulmonary resuscitation and 2 automated external defibrillator shocks restored circulation within 5 minutes. Electrocardiography demonstrated a type 1 Brugada pattern. He achieved neurologic recovery after intensive care. Cardiac imaging showed no structural abnormalities, and the family history revealed sudden death in an identical twin. Brugada syndrome unmasked by kratom exposure was diagnosed, and referral was made for subcutaneous implantable cardioverter-defibrillator implantation and genetic testing.

DISCUSSION Kratom possesses opioid activity and ion channel-modulating properties that may precipitate malignant arrhythmias.

TAKE-HOME MESSAGES Kratom is a psychoactive substance with potential arrhythmogenic effects. Increased awareness of its cardiovascular risks is important for both clinicians and public health. (JACC Case Rep. 2026;31:107173)

CASE PRESENTATION

A 28-year-old man without any known medical history experienced an abrupt loss of consciousness after smoking a hand-rolled cigarette at an outdoor arcade. Bystanders began chest compressions, and an automated external defibrillator delivered 2 shocks, with return of spontaneous circulation in <5 minutes.

In the emergency department, he was intubated for airway protection. Before sedation, he demonstrated purposeful movement. His temperature was 93 °F (33.9 °C), and active rewarming was initiated. Laboratory studies showed lactic acid 6.8 → 3.6 mmol/L, creatine kinase 713 U/L, high-sensitivity

TAKE-HOME MESSAGES

- Kratom is a psychoactive substance with potential arrhythmogenic effects.
- Increased awareness of its cardiovascular risks is important for both clinicians and public health.

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ABBREVIATIONS AND ACRONYMS

BrS = Brugada syndrome

ECG = electrocardiogram

hERG = human ether-à-go-go-related gene

S-ICD = subcutaneous implantable cardioverter-defibrillator

troponin 38.6 → 511 ng/L, alanine aminotransferase 401 U/L, aspartate aminotransferase 240 U/L, and creatinine 1.63 mg/dL; urine toxicology was benzodiazepine-positive. The chest radiograph and noncontrast head computed tomography were unremarkable.

There was transient tonic activity during resuscitation. Continuous electroencephalogram later demonstrated a normal background without epileptiform discharges, consistent with convulsive syncope in the setting of cardiac arrest. The initial electrocardiogram (ECG) (Figure 1) demonstrated coved ST-segment elevation ≥2 mm in V_2 with T-wave inversion, diagnostic of a type 1 Brugada pattern, which was validated by cardiology and electrophysiology.

He was treated in the intensive care unit with targeted temperature management, during which time he was extubated on hospital day 2 after spontaneous awakening and breathing trials. After extubation, he experienced brief vomiting and marked anterograde amnesia; magnetic resonance imaging was deferred as symptoms resolved clinically. Echocardiogram and other imaging findings did not demonstrate any structural cardiac abnormalities.

On further history, the patient reported using kratom to "boost energy and mood," especially as a result of his twin's recent sudden death. He was counseled to avoid kratom, cocaine, antipyretic nonuse during febrile illness, and other sodium-channel-blocking or vagotonic agents known to precipitate ventricular arrhythmias in Brugada syndrome (BrS).

Given the out-of-hospital cardiac arrest, spontaneous type 1 ECG, and family history, BrS was diagnosed. Sodium voltage-gated channel alpha subunit 5 testing and screening for first-degree relatives were recommended.

An ECG obtained before subcutaneous implantable cardioverter-defibrillator (S-ICD) placement (Figure 2) demonstrated sinus rhythm with coved-type ST-segment elevation and T-wave inversion in V_1-V_2, consistent with a type 1 Brugada pattern. Because of his age and lack of pacing indications, an S-ICD was implanted before discharge to prevent secondary events.

A follow-up ECG (Figure 3) showed normal sinus rhythm with near-complete resolution of the prior coved-type ST-segment elevation in V_1-V_2, no longer meeting diagnostic criteria for a type 1 Brugada pattern. Rather, a saddleback or minimal r′ morphology with an isoelectric to mildly elevated ST segment and upright T waves was observed, consistent with a nondiagnostic or type 2-like Brugada pattern (normal variant).

DISCUSSION

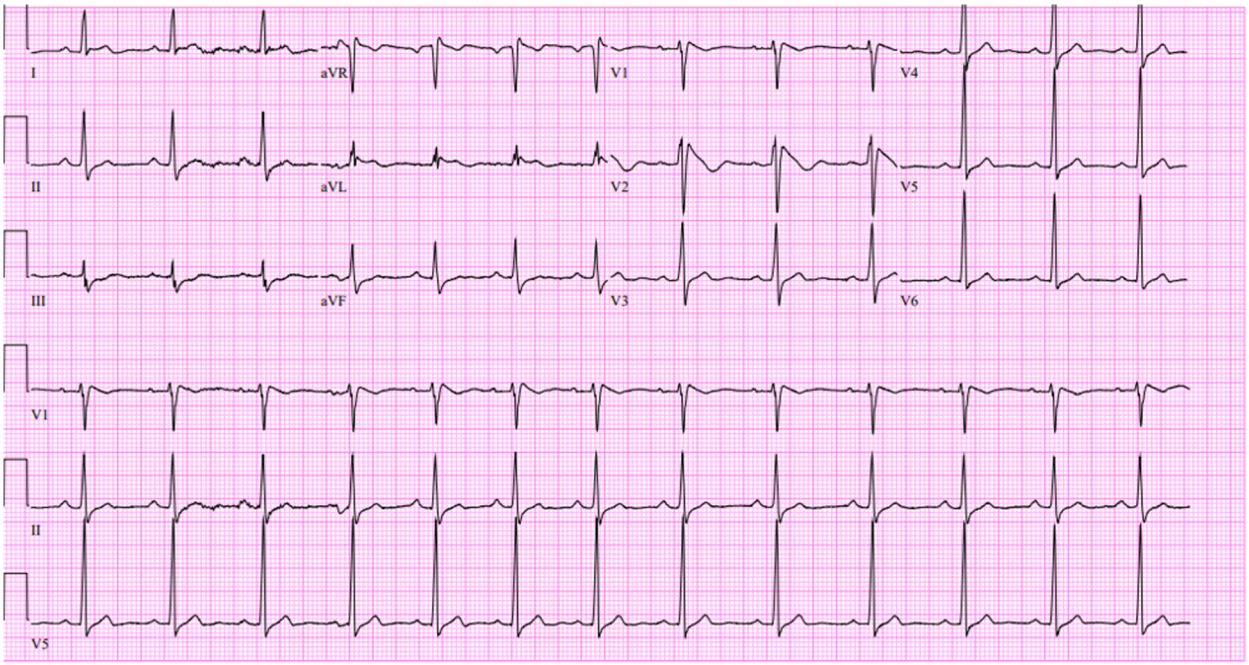
BrS is an inherited type of arrhythmia syndrome characterized by right precordial elevation of the ST segment in the absence of overt structural heart disease and increased risk for polymorphic ventricular tachycardia and sudden cardiac death,

VISUAL SUMMARY **Brugada Case Timeline**

Day	Events
Day 0 (admission)	A young man was brought to the emergency department, having out-of-hospital cardiac arrest. CPR was administered by bystanders, and spontaneous circulation was achieved after 2 AED shocks. He was intubated to protect his airway. An initial ECG revealed a type 1 Brugada pattern with coved ST-segment elevation and T-wave inversion in leads V_1-V_2. CT head was normal. Laboratory investigations revealed mild acute kidney injury and leukocytosis. Targeted temperature management and supportive care were initiated.
Day 1	The patient was extubated and was alert, oriented, and hemodynamically stable. Neurologic recovery was consistent with no major neurologic deficits but some short-term memory loss. Neurology reviewed and thought compulsive syncope. Cardiology and electrophysiology were consulted for evaluation of Brugada syndrome. They advised to stay clear of kratom, cocaine, and other sodium-channel–blocking or vagotonic agents that may induce ventricular arrhythmias.
Day 2	Family history confirmed sudden death of his twin brother. Repeat ECG showed retained type 1 Brugada morphology. Genetic testing was advised for *SCN5A* mutation and screening of first-degree relatives. Echocardiogram showed a structurally normal heart with preserved systolic function.
Day 3	After a multidisciplinary discussion, S-ICD placement was scheduled in the hope of secondary prevention due to his cardiac arrest and spontaneous type 1 ECG. The patient was stable on telemetry without recurrences of arrhythmias.
Day 4	The patient underwent uncomplicated S-ICD implantation; appropriate sensing and impedance were observed. Preprocedural ECG demonstrated a persistent type 1 Brugada pattern (Figure 2).
Day 5 (discharge)	The patient was discharged home in stable condition. The follow-up ECG showed near resolution of the coved ST-segment elevation in a type 2–like Brugada morphology (Figure 3). He was counseled to refrain from fever, kratom, and medications that could worsen sodium-channel blockade. Sequential electrophysiology and genetic counseling follow-up were arranged at an outpatient facility.

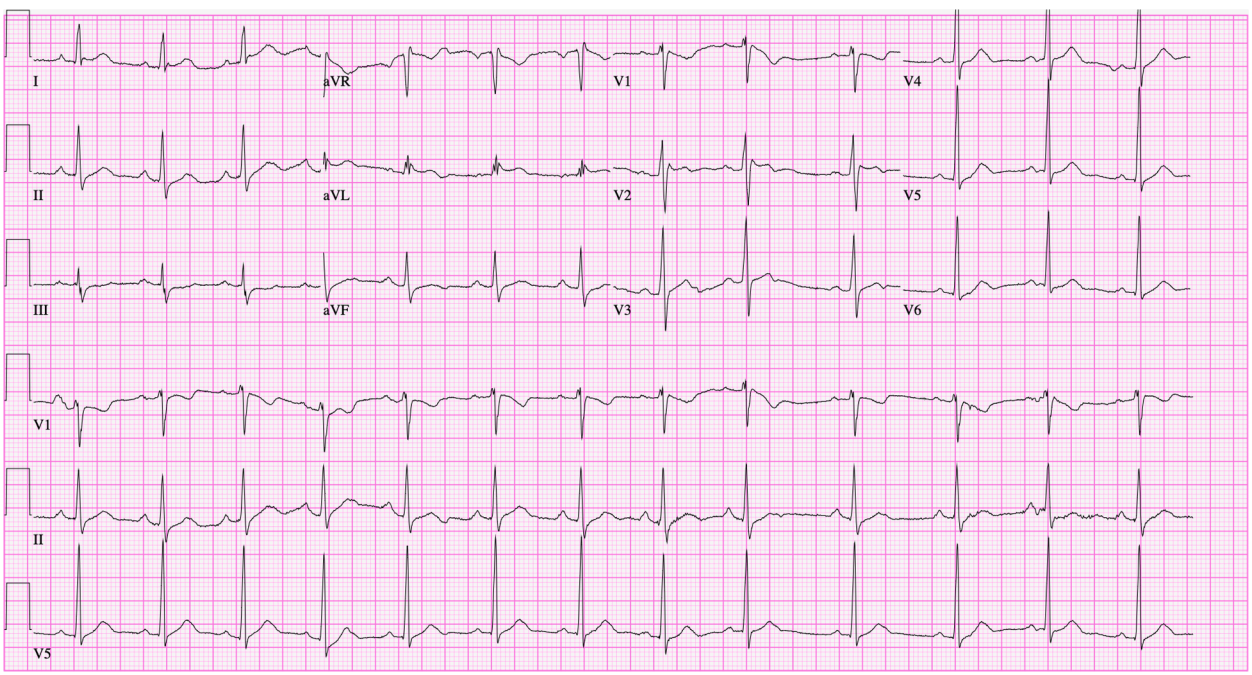
AED = automated external defibrillator; CPR = cardiopulmonary resuscitation; CT = computed tomography; ECG = electrocardiogram; SCN5A = sodium voltage-gated channel alpha subunit 5; S-ICD = subcutaneous implantable cardioverter-defibrillator.

FIGURE 1 **Admission Electrocardiogram**



Electrocardiogram demonstrating a type 1 Brugada pattern with coved ST-segment elevation in V_2.

FIGURE 2 **Electrocardiogram Before S-ICD Implantation**



Electrocardiogram demonstrating sinus rhythm with coved-type ST-segment elevation and T-wave inversion in V_1-V_2, consistent with a type 1 Brugada electrocardiogram pattern. S-ICD = subcutaneous implantable cardioverter-defibrillator.

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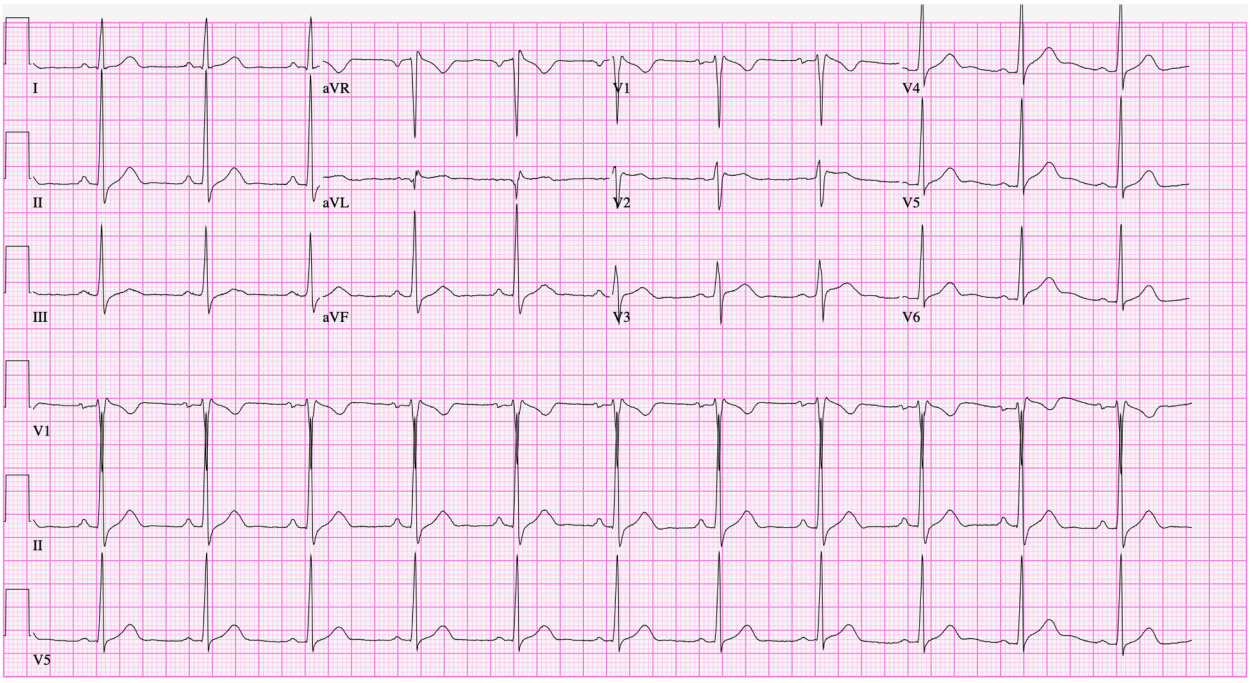
FIGURE 3 Electrocardiogram After S-ICD Implantation



Electrocardiogram demonstrating sinus rhythm with resolution of prior coved-type ST-segment elevation in V_1-V_2. Instead, there is a saddleback or minimal r′-wave morphology with an isoelectric or minimally elevated ST segment and upright T waves, consistent with a nondiagnostic or type 2–like Brugada pattern (simple normal variant). S-ICD = subcutaneous implantable cardioverter-defibrillator.

typically in young to middle-aged men.[1,2] However, loss-of-function variants in sodium voltage-gated channel alpha subunit 5, which encodes the cardiac sodium channel Nav1.5, as well as in additional genes that modulate calcium channel function, such as CACNA1C, CACNB2b, among others, have also been described.[2,3] However, most patients with BrS do not exhibit an identifiable genetic variant, underscoring the role of physiologic stressors and metabolic and pharmacologic triggers that unmask the phenotype.[1-3]

The diagnostic type 1 Brugada ECG pattern is defined by a coved ST-segment pattern: ≥2-mm ST-segment elevation in V_1-V_2 with T-wave inversion.[1-3] Type 2 demonstrates a saddleback configuration with ≥2-mm elevation, whereas type 3 resembles either morphology with <2-mm elevation.[4] Phenotype expression is dynamic. Fever, electrolyte abnormalities, heightened vagal tone, and sodium-channel-blocking agents all reduce and can convert a nondiagnostic tracing into type 1 BrS.[1-4] This is clinically important because it links bedside triggers to actionable management steps—antipyretics, drug avoidance lists, and careful interpretation of right precordial leads placed in higher interspaces when suspicion persists.[1-5]

Management in BrS targets sudden-death prevention. Survivors of ventricular fibrillation or cardiac arrest meet criteria for ICD placement for secondary prevention.[1] Given the patient's age and lack of pacing needs, a subcutaneous ICD was selected to minimize transvenous lead complications. Quinidine and radiofrequency ablation are reasonable adjuncts in selected patients with recurrent ventricular arrhythmias.[1,2] Universal measures include aggressive fever management, avoidance of sodium-channel blockers, and other listed agitators. Given the heritable nature of BrS, screening of first-degree relatives, genetic counseling, and testing are recommended.[2,3]

Kratom (*Mitragyna speciosa*) is a widely available herbal product used for energy enhancement, mood elevation, analgesia, and self-treatment of opioid withdrawal. Kratom is available in powdered, encapsulated, beverage-mixed, and smokeable forms.[6,7] Its principal alkaloids (mitragynine and 7-hydroxymitragynine) act as partial μ-opioid agonists but also interact with cardiac ion channels, with in vitro data

demonstrating inhibition of voltage-gated sodium current and human ether-à-go-go-related gene (hERG)-mediated potassium channels.[7-10] Mitragynine, the principal active alkaloid in kratom, has been associated with cardiac electrophysiological toxicity. Experimental studies demonstrate that mitragynine inhibits hERG potassium channels, reducing the rapidly activating delayed rectifier potassium current in human cardiomyocytes. This effect prolongs action potential duration and increases susceptibility to torsades de pointes, a potentially fatal ventricular arrhythmia. Mitragynine has also been shown to decrease expression of fully glycosylated hERG1a protein, suggesting impaired channel trafficking that may further enhance cardiotoxic risk. Although kratom-associated corrected QT interval prolongation and torsades de pointes have been reported, its effects on cardiac sodium channels and its potential to precipitate BrS remain poorly characterized and warrant further investigation.[10] The resulting QRS and QT effects mirror those seen with established Brugada-provoking agents. In our patient, the tight link between kratom exposure and cardiac arrest, spontaneous type 1 pattern on ECG, and a strong family history of sudden death together make kratom a biologically plausible trigger of a latent Brugada substrate.[11-13]

Kratom may also inhibit CYP2D6, increasing the proarrhythmic effects of commonly prescribed agents such as antiarrhythmics, antipsychotics, calcium channel blockers, β-blockers, and antidepressants.[11] Prior reports of kratom-associated Brugada patterns have largely involved chronic or high-dose use, sometimes with polysubstance exposure.[4,5,10] Notably, a JACC case report demonstrated reversible Brugada phenotype and corrected QT interval prolongation after kratom exposure, supporting a direct ion-channel-mediated mechanism.[12]

In contrast, our patient developed a spontaneous type 1 Brugada ECG pattern and ventricular fibrillation arrest after a first known exposure to a kratom-containing smokeable product, in the absence of fever, electrolyte abnormalities, or coingestants. This case demonstrates that even a single, nonchronic exposure may be sufficient to unmask BrS in genetically susceptible individuals.

As kratom use becomes more common and remains unregulated, clinicians should maintain a high index of suspicion and inquire about herbal supplements when evaluating unexplained syncope, aborted sudden death, or Brugada-like ECG changes. Future work is needed to define genetic susceptibility, dose-response relationships, and public health risk associated with increasing recreational kratom exposure.

CONCLUSIONS

This case illustrates a rare but clinically important instance of BrS unmasked by kratom use, leading to resuscitated out-of-hospital cardiac arrest in a young man with a family history of sudden death.[6-9,14] Unlike previously described cases, which predominantly involved chronic or high-dose kratom use, this patient experienced ventricular fibrillation arrest after a single exposure in the absence of fever, electrolyte imbalance, or coingestants. This highlights the importance of recognizing kratom as a potential sodium-channel-modulating trigger, capable of converting a subclinical Brugada substrate into a life-threatening phenotype. Clinicians should inquire about herbal and recreational substance use when evaluating unexplained cardiac arrest, particularly in the setting of a Brugada ECG pattern. Early identification, trigger avoidance, and ICD implantation remain the cornerstones of management.[10-12]

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REFERENCES

1. Krahn AD, Behr ER, Hamilton R, et al. Brugada syndrome. *JACC Clin Electrophysiol*. 2022;8(3):386-405. https://doi.org/10.1016/j.jacep.2021.12.001

2. Li KHC, Lee S, Yin C, et al. Brugada syndrome: a comprehensive review of pathophysiological mechanisms and risk stratification strategies. *Int J Cardiol Heart Vasc*. 2020;26:100468. https://doi.org/10.1016/j.ijcha.2020.100468

3. Watanabe H, Minamino T. Genetics of Brugada syndrome. *J Hum Genet*. 2016;61(1):57-60. https://doi.org/10.1038/jhg.2015.97

4. Holton K, Sanchez Corredera C, Haidar J, Valentin RG, Perez D, Boccio E. Acquired type 1 Brugada syndrome induced by chronic high-dose kratom use. *Cureus*. 2024;16(10):e72129. https://doi.org/10.7759/cureus.72129

5. Dodulík J, Plášek J, Handlos P, et al. Ventricular fibrillation during football training as a consequence of kratom and caffeine use in an adolescent: case report. *Eur Heart J Case Rep*. 2024;8(8):ytae364. https://doi.org/10.1093/ehjcr/ytae364

6. Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada syndrome: report of the second consensus conference. *Circulation*. 2005;111(5):

659–670. https://doi.org/10.1161/01.CIR.0000152479.54298.51

7. Priori SG, Blomström-Lundqvist C, Mazzanti A, et al. 2015 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J*. 2015;36(41):2793–2867.

8. Tungsanga S, Peerapanyasut W. Cardiac toxicity of kratom (*Mitragyna speciosa*): a comprehensive review. *Front Pharmacol*. 2022;13:866303. https://doi.org/10.3389/fphar.2022.866303

9. Lu J, Cottrell A, Han M, Shao X. Kratom and cardiac arrhythmia: mechanistic insights. *Toxicol Rep*. 2021;8:1587–1592. https://doi.org/10.1016/j.toxrep.2021.08.003

10. Abubaker M, Adrejiya P, Berhe L, Chennareddy S, Kanugula A. Chronic kratom use as a precipitant for acquired Brugada syndrome. *HeartRhythm Case Rep*. 2025;11(9):958–961. https://doi.org/10.1016/j.hrcr.2025.06.031

11. Leong Bin Abdullah MFI, Singh D. The adverse cardiovascular effects and cardiotoxicity of kratom (Mitragyna speciosa korth.): a comprehensive review. *Front Pharmacol*. 2021;12:726003. https://doi.org/10.3389/fphar.2021.726003

12. Miller AHF, Krotulski AJ, Walton SE, et al. Kratom cardiotoxicity: reversible brugada pattern and QTc prolongation. *JACC Case Rep*. 2025;30(5):103109. https://doi.org/10.1016/j.jaccas.2024.103109

13. Tay YL, Teah YF, Chong YM, et al. Mitragynine inhibits hERG1a/1b channels and reduces IKr current density in HEK293 cells. *Sci Rep*. 2019;9:18934. https://doi.org/10.1038/s41598-019-56106-6

14. Barajas-Martínez H, Hu D, Antzelevitch C. Brugada syndrome: current concepts and management. *J Am Coll Cardiol*. 2023;81(3):305–322.

KEY WORDS **Brugada syndrome, kratom, *Mitragyna speciose*, out-of-hospital cardiac arrest, S-ICD, ventricular fibrillation**