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Cannabis-Induced Cardiac Arrest in a Young Adult: A Case Report

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Patient: Female, 26-year-old
Final Diagnosis: Cardiac arrest and vfib due to thc use
Symptoms: Cardiac arrest
Clinical Procedure: —
Specialty: Cardiology • General and Internal Medicine

Objective: Unusual clinical course

Background: Tetrahydrocannabinol (THC), the psychoactive compound in cannabis, is increasingly used recreationally, especially among young adults. Although often perceived as harmless, emerging evidence links THC to serious cardiovascular complications, including QTc prolongation, ventricular arrhythmias, and sudden cardiac arrest, even in individuals without underlying heart disease.

Case Report: A 26-year-old woman with a history of chronic cannabis use was brought to the Emergency Department (ED) after having a sudden cardiac arrest. Her initial rhythm was ventricular fibrillation, and return of spontaneous circulation was achieved following advanced cardiac life support. An initial electrocardiogram revealed sinus tachycardia with a QTc interval of 483 ms, and a urine drug screen was positive for THC. Cardiac catheterization performed 4 weeks later revealed normal coronary arteries. Her left ventricular ejection fraction, initially reduced at 25-30%, had normalized by the time of catheterization. She sustained hypoxic-ischemic brain injury, requiring prolonged rehabilitation.

Conclusions: This case highlights the potential for cannabis use to induce life-threatening cardiac arrhythmias, including QTc prolongation, and ventricular fibrillation, even in young adults with no prior cardiovascular risk factors. Clinicians should maintain a high index of suspicion for cannabis-induced cardiotoxicity in similar presentations, particularly as cannabis use becomes more widespread.

Keywords: Cannabis • Cardiac Electrophysiology • Marijuana Use • Ventricular Fibrillation

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/950077

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Introduction

Cannabis is the most widely used recreational drug globally, with over 200 million users reported in 2022 alone, and its use continues to rise sharply, particularly among adolescents and young adults. In the United States, approximately 18% of individuals aged 12 and older reported past-year use of cannabis, with rates steadily increasing following legalization in several states [1]. Cannabis is often perceived to be harmless or even therapeutic; however, emerging evidence has revealed a wide range of adverse effects linked to THC, the principal psychoactive compound in cannabis. These include psychiatric disturbances, impaired cognition, respiratory effects, and increasingly, cardiovascular complications.

The first documented case of cannabis-induced cardiac arrest was published by Sattout and Nicol in 2009, describing a 15-year-old boy who collapsed after cannabis consumption and progressed from asystole to ventricular fibrillation [2]. Since then, multiple case reports and small studies have identified cannabis as a potential arrhythmogenic agent.

Kariyanna et al conducted a scoping review of 27 cannabis-associated arrhythmia cases, most involving young males with an average age of 28 years. The most frequently reported arrhythmias were atrial fibrillation (26%) and ventricular fibrillation (22%). Alarmingly, 11% of these cases were fatal—underscoring the severity of cannabis-induced cardiac events [3].

Cannabis has also been implicated in QTc prolongation, which can predispose patients to dangerous ventricular arrhythmias such as Torsades de Pointes and ventricular fibrillation [4-7]. Proposed mechanisms include increased sympathetic tone and inhibition of cardiac ion channels like the hERG potassium channel, which delays repolarization. Even in the absence of traditional cardiac risk factors or structural heart disease, cannabis can unmask latent arrhythmogenic conditions or directly induce electrical instability.

This case is especially noteworthy as it involves a young, previously healthy woman with no structural heart disease, no family history of cardiac disease, and no other toxicologic or metabolic abnormalities, who had a life-threatening arrhythmia shortly after heavy cannabis use. Her QTc was prolonged, and her cardiac function fully normalized during follow-up, making a strong case for THC as a reversible trigger. Unlike most published reports, this case also documents the patient's recovery from severe hypoxic brain injury after prolonged cardiac arrest, highlighting the dual cardiologic and neurologic impact of cannabis toxicity.

We aim to raise clinical awareness about cannabis as a potential cause of sudden cardiac arrest and encourage further investigation into its proarrhythmic mechanisms.

Case Report

A 26-year-old woman with a history of chronic cannabis use was brought to the Emergency Department following a witnessed sudden cardiac arrest. Her fiancé reported hearing an unusual noise from the bathroom, after which he found her unresponsive. No bystander cardiopulmonary resuscitation (CPR) was performed. Emergency medical services (EMS) arrived 10 minutes later and found the patient in ventricular fibrillation (**Figure 1**). Advanced cardiac life support was initiated, and return of spontaneous circulation was achieved after 2 defibrillation shocks, 3 doses of epinephrine, and 2 doses of amiodarone.

Upon arrival, her vital signs were blood pressure 127/84 mmHg, heart rate 122 bpm, and respiratory rate 22/min. Laboratory values on admission are shown in **Table 1**. She was intubated and had a Glasgow Coma Scale score of 3/15. The initial electrocardiogram showed sinus tachycardia and a prolonged QTc of 483 ms (**Figure 2**). Urine drug screen was positive for THC and negative for other substances. Troponin levels peaked at 475 ng/mL. Brain MRI on hospital day 2 demonstrated hypoxic-ischemic injury. Echocardiography showed a reduced ejection fraction (EF) of 25-30%, with no regional wall motion abnormalities. Genetic testing for long QT syndrome and arrhythmia panels was negative.

The patient underwent targeted temperature management and supportive critical care. Over the course of her hospitalization, her neurological status gradually improved. Repeat echocardiography at 5 weeks showed complete normalization of EF to 55-60%. Her QTc also normalized (443 ms) 1 week after cardiac arrest (**Figure 3**). Cardiac catheterization performed 4 weeks later revealed normal coronary arteries. She was ultimately discharged to a skilled nursing facility with a wearable cardioverter-defibrillator.

Discussion

Case Novelty and Clinical Context

This case highlights a rare but critical presentation of cannabis-induced cardiac arrest in a young, previously healthy woman. Unlike most published reports that focus on older adults or those with underlying cardiac abnormalities, our patient had no structural heart disease, genetic predisposition, or metabolic derangement. The temporal relationship between heavy cannabis use and the onset of ventricular fibrillation, QTc prolongation, and cardiac arrest makes this case particularly valuable for clinician awareness and public health discussions.

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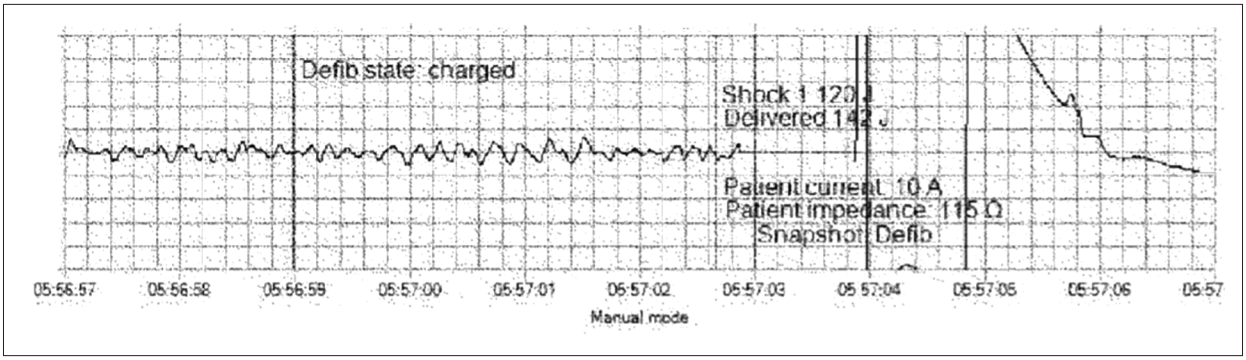

Figure 1. Telemetry strip by EMS prior to defibrillation, revealing ventricular fibrillation.

Table 1. Laboratory results on admission following cardiac arrest.

Test	Result	Normal range
Troponin (ng/mL)	27 → 291 → 395 → 475	<0.04
Bicarbonate (mmol/L)	12	22-28
Anion gap	27	8-16
Lactic acid (mmol/L)	8.3	0.5-2.2
Potassium (mmol/L)	Normal	3.5-5.1
Phosphorus (mg/dL)	Normal	2.5-4.5
Calcium (mg/dL)	Normal	8.5-10.5
Magnesium (mg/dL)	Normal	1.7-2.2
D-Dimer (ng/mL)	6544	<500
White blood cell count (×10^9/L)	12	4-11

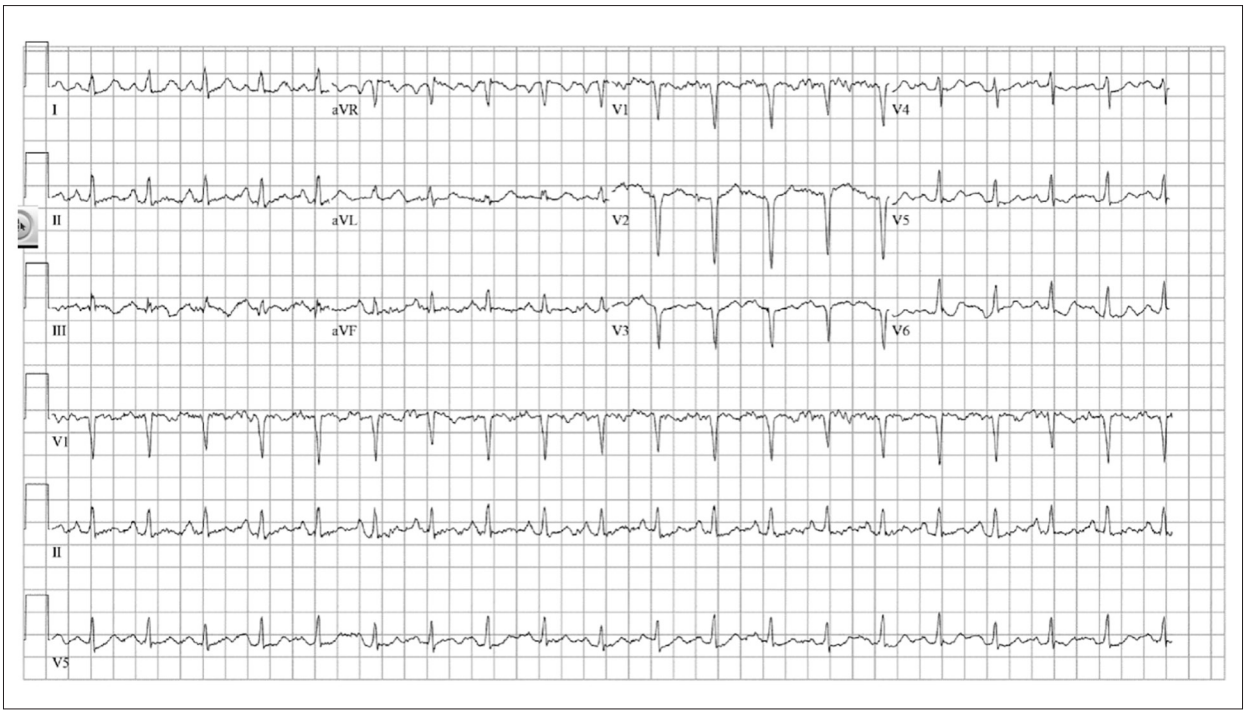

Figure 2. EKG on presentation shows sinus tachycardia 119 bpm, transient Q waves, and QTc of 483 ms.

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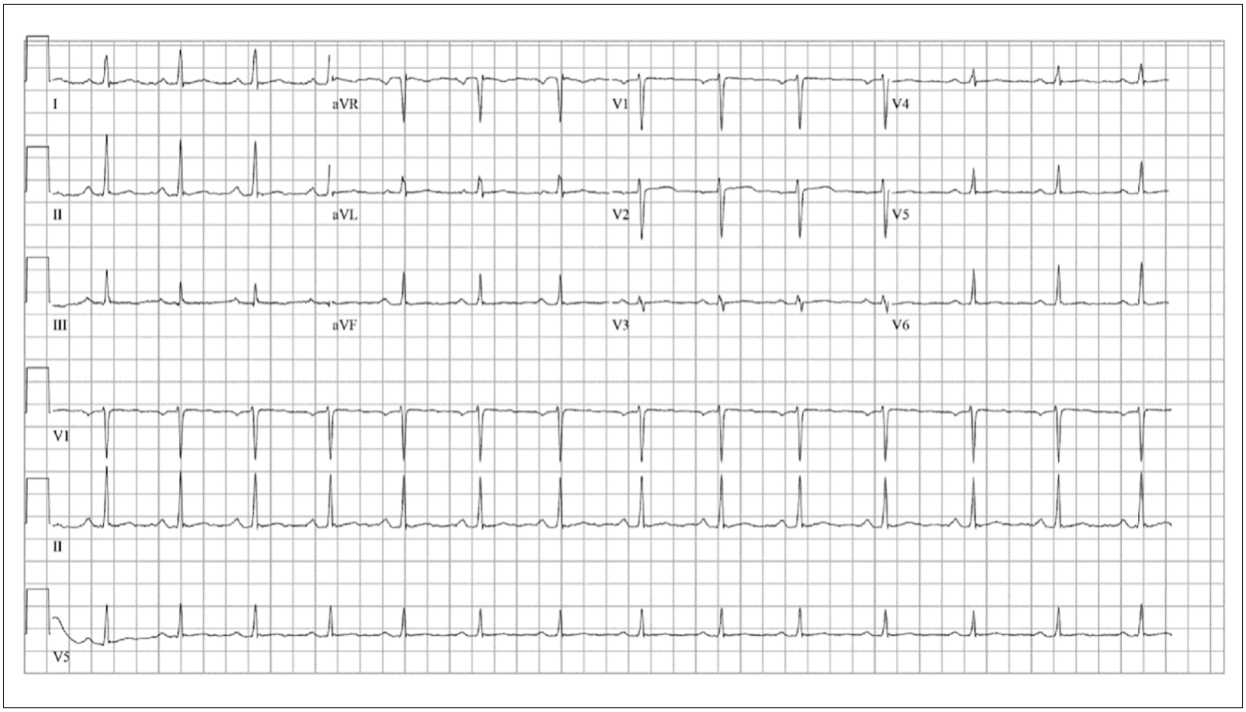

Figure 3. EKG after 1 week after cardiac arrest, with normalization of Q waves and QTc of 443 ms.

Table 2. Summary of case reports of patients aged 15-35 with cardiac arrest following cannabis use.

Case	Age	Type/amount of cannabis consumed	Time before arrest	Presumed mechanism leading to arrest	Reference
1	15	Smoked cannabis; amount not specified, shortly before event	Shortly before collapse	QTc prolongation, torsades de pointes	[2]
2	24	High-potency THC (dabbing); frequency not specified, within 1 hour of event	Within 1 hour	Coronary vasospasm, myocardial ischemia	[14]
3	30	Cannabis concentrate; heavy use reported, ~30 min before event	~30 minutes	Ventricular arrhythmia, hyperadrenergic state	[15]
4	30	Cannabis concentrate; heavy use, within 1 hour of event	Within 1 hour	Ventricular fibrillation due to IKr inhibition	[15]
5	24	Cannabis resin; repeated use, few hours prior	A few hours	Acute myocardial ischemia, arrhythmia	[15]

Comparison with Existing Literature

Several reports have linked cannabis use to adverse cardiovascular outcomes, including arrhythmias, myocardial infarction, and sudden cardiac death. Sattout and Nicol first reported a cannabis-associated cardiac arrest in a 15-year-old boy, and subsequent reports described a variety of arrhythmias, including atrial fibrillation and ventricular fibrillation in cannabis users [2,3]. However, many prior cases involved patients with coexisting risk factors or polysubstance use (Table 2). Our case contributes uniquely by documenting isolated cannabis use in a structurally normal heart with complete cardiac and neurological recovery following prolonged cardiac arrest.

Pathophysiology and Mechanistic Insight

Cannabis, also known as marijuana, contains over 100 active ingredients, the most potent of which is delta-9-THC. Other notable compounds include cannabidiol (CBD) [8]. THC is metabolized by the liver to 11-hydroxy-THC, a potentially more psychoactive metabolite. Due to its lipid solubility, THC accumulates in adipose tissue, reaching peak levels within 4-5 days

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and exhibiting an elimination half-life of approximately 7 days. It can remain detectable in the body for up to 30 days after a single dose, with chronic users accumulating higher systemic levels [9].

CB1 and CB2 cannabinoid receptors mediate the effects of THC. CB1 receptors are highly expressed in the central nervous system, while CB2 receptors are found in immune and peripheral tissues [10]. Cardiovascular effects are mediated largely through CB1 receptor activation, which promotes sympathetic stimulation, tachycardia, and vasospasm, and can inhibit cardiac ion channels like hERG, potentially resulting in QTc prolongation and malignant arrhythmias [6,11,14]. Our patient's QTc was initially 483 ms, without electrolyte imbalance, medication use, or underlying channelopathy. The interval normalized to 443 ms within 1 week, suggesting a reversible THC-mediated electrophysiological effect.

In addition, cannabis has been associated with myocardial ischemia and infarction. During the first hour after use, the risk of infarction increases approximately 6-fold. This is thought to be due to increased myocardial oxygen demand (tachycardia), reduced oxygen delivery (carboxyhemoglobin elevation), and coronary vasospasm. Bachs and Morland reported sudden deaths in 6 young individuals shortly after cannabis use, with no other substances found on toxicology [12]. Our patient's positive urine drug screen for THC and absence of serum toxicology limit confirmatory conclusions, but the temporal link supports a causative association.

THC's sympathomimetic effects can induce arrhythmias such as sinus tachycardia, ectopic beats, atrial fibrillation, and ventricular fibrillation [13], consistent with our patient's initial presentation in ventricular fibrillation.

Troponin Elevation

The patient's troponin levels peaked at 475 ng/mL, which may reflect global myocardial injury due to cardiac arrest and defibrillation rather than an ischemic infarction. Cardiac catheterization showed normal coronary arteries and no wall motion abnormalities. The transient Q waves likely represent global ischemia or post-resuscitation injury rather than an infarct pattern.

Differential Diagnosis and Diagnostic Approach

Differentials considered included congenital long QT syndrome, myocarditis, myocardial bridge, and coronary vasospasm. Genetic testing ruled out channelopathies, and cardiac catheterization revealed normal anatomy. Unfortunately, cardiac MRI was not available to exclude myocarditis or microvascular abnormalities. While a myocardial bridge or small-vessel spasm cannot be definitively ruled out, the clinical presentation and full cardiac recovery without recurrence support a toxicologic etiology.

Causality Assessment

Using the WHO-Uppsala Monitoring Centre (UMC) system and the Naranjo algorithm, the association between THC and cardiac arrest in this patient can be rated as "probable." This is based on the temporal relationship, absence of alternative explanations, and reversibility after cessation. However, confirmation through rechallenge was not ethically possible.

Public Health Implications

A prospective study involving 1913 adults over a 3.8-year follow-up period found a clear dose-dependent link between cannabis use and cardiovascular-related deaths. Additionally, the World Health Organization has reported multiple case studies indicating a heightened risk of cardiovascular events in younger individuals who otherwise have low baseline risk [15]. This case underscores the need for public awareness about the cardiovascular risks of cannabis use, particularly high-potency or chronic consumption. The absence of bystander CPR also contributed to our patient's neurologic injury, emphasizing the importance of widespread CPR training and emergency response education.

Limitations

This case report has several limitations. First, serum toxicology was not obtained, limiting definitive quantification of THC levels. Second, immediate cardiac MRI was not performed, which could have helped identify myocarditis or scar-related arrhythmia substrates. Third, this is a single case report, which inherently limits generalizability and cannot establish causation. Lastly, while the association between THC and cardiac arrest is strong, confounding variables such as undetected genetic predispositions or microvascular disease cannot be fully excluded.

Conclusions

Recreational cannabis use is steadily increasing among both adults and youth due to expanding legalization and easy accessibility, underscoring the urgent need for greater public and clinical awareness of its potential health risks. This case highlights the serious cardiovascular consequences of THC, including life-threatening arrhythmias and QTc prolongation, even in young individuals without structural heart disease.

Key takeaways from this case include:

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- Clinical awareness: Physicians should maintain a high index of suspicion for cannabis-induced QTc prolongation, cardiac arrest, and fatal arrhythmias, particularly in patients presenting with new-onset ECG abnormalities and no clear etiology.
- Diagnostic vigilance: Expanded toxicology panels should be considered to detect synthetic cannabinoids that may not be identified on standard urine drug screens but have significant arrhythmogenic potential.
- Public health initiatives: Delays in bystander CPR, as seen in this case, can worsen neurological outcomes. Widespread community CPR education and dispatch-assisted instructions are critical for improving survival and recovery after cardiac arrest.

We hope this report encourages further research into the cardiovascular effects of cannabis to inform evidence-based health policies, patient education, and development of clinical guidelines aimed at minimizing cannabis-related harm.

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Institution Where Work Was Done

WellStar Spalding Health System, Griffin, GA, USA.

Patient Consent

Written informed consent was obtained from the patient's legal representative for publication of this case report and any accompanying images. Written informed consent was obtained from the patient for publication of this case report.

Declaration of Figures' Authenticity

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