

Occurrence of Seizures with Cannabis Abuse: A Case Report

Running Title: Cannabis and Seizure

Sara Salarian¹, Bahador Bagheri², Ghazaleh Assar^{3*}

¹ Department of Intensive Care, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

² Cancer Research Center, Semnan University of Medical Sciences, Semnan, Iran.

³ Department of Clinical Pharmacy, Faculty of Pharmacy, Tehran University of Medical Sciences, Tehran, Iran.

ARTICLE INFO

Received: 02/08/2025

Accepted: 02/25/2025

*Corresponding author

Department of Clinical
Pharmacy, Faculty of
Pharmacy, Tehran
University of Medical
Sciences, Tehran, Iran
Tel: +989124316620

E-mail

Ghazaleh47@yahoo.com

Abstract

Introduction: *Cannabis sativa* is being medicinally used by humans and is well known for its recreational uses. Seizure is exceedingly rarely seen following cannabis use. Various cellular mechanisms have been suggested in the occurrence of seizures caused by cannabinoids.

Case: The patient was a 28-year-old man who complained of weakness, lethargy, and convulsions after consuming cannabis (hashish). The laboratory findings and brain CT were normal. The patient's toxicity panel was positive for hashish.

Conclusion: Physicians working in the emergency department should be aware that hashish may cause seizures. Although several investigations have shown anticonvulsant effects for cannabis.

Keywords: Cannabis, Hashish, Seizure, Emergency, Case report

Citation: Salarian S, Bagheri B, Assar G Occurrence of Seizures with Cannabis Abuse: A Case Report. *Adv Pharmacol Ther J.* 2025;5(1): 31-35.

Introduction

The cannabis plant has a long history of medicinal use, with over 100 phytocannabinoids and 200 terpenes identified (1). The 2021 World Drug Report estimates approximately 275 million people use cannabis, making it the most prevalent illicit drug (2). The primary psychoactive compound is Δ^9 -tetrahydrocannabinol (THC), while cannabidiol (CBD) is non-psychoactive and believed to have anticonvulsant properties, though its exact mechanism remains unclear (3, 4). THC has a complex relationship with seizure activity. This relationship is influenced by various biological mechanisms, receptor interactions, and individual patient factors. Clinical evidence regarding cannabis and seizure activity remains mixed (5). This report details a case of seizure induced by hashish.

Case

Approved by the local Ethics Committee (IR.SBMU.RETECH.REC.1401.880), this case involves a 28-year-old male presenting with weakness, lethargy, somnolence, and convulsions attributed to hashish use. The patient had consumed hashish about 10 hours before the convulsion. He had a history of occasional headaches but no significant medical or drug history, nor prior seizures. Three days before admission, he experienced a convulsion with loss of consciousness and incontinence. Upon admission, vital signs were stable (SpO₂: 96%, BP: 110/60, PR: 100), and pupils were mydriatic but reactive. Brain CT showed no structural abnormalities and electrolyte levels were normal. Sepsis was ruled out, and viral tests for COVID-19

were negative. Due to the variability and unknown nature of COVID-19 manifestations, as well as the persistence of certain complications observed even years after initial infection, it was necessary to conduct COVID-19 testing to ascertain whether the virus was responsible for these symptoms.

A toxicity panel confirmed hashish use. Fluid and Benzodiazepines were administered to address the acute phase of seizures, followed by the use of Levetiracetam for maintenance therapy. Additionally, comprehensive care in the intensive care unit (ICU) was implemented to ensure brain protection, which included maintaining normoglycemia, normocarbia, normothermia, normonatremia, and normal intracranial pressure. Therefore the patient's seizures subsided within 24 hours.

Discussion

Multiple case reports document acute cannabis intoxication leading to symptoms like ataxia and seizures in children (6). Adolescents may face heightened risks from cannabis (7). THC's inhibition of GABA (gamma aminobutyric acid) release could trigger seizures; its effects vary based on dosage (1). THC primarily exerts its effects through the activation of CB1 receptors, which are densely located in the central nervous system(8).The National Toxicology Program highlighted THC's proconvulsant effects in animal studies, although individual responses may differ due to receptor expression variations. CBD enhances GABAA (subtype A from gamma aminobutyric acid) receptor activity and may influence seizure thresholds (7).

Despite rare reports of cannabis-induced seizures, adverse effects can include increased blood pressure, tachycardia, and respiratory depression (9). THC also activates the mitogen-activated protein kinase (MAPK) pathway, specifically through ERK (extracellular signal-regulated kinase) signaling, affecting neuronal functions like synaptic plasticity (10). The activation of ERK by THC has been shown to influence long-term neuronal plasticity and may contribute to the proconvulsant effects observed in some individuals. Studies indicate that THC-induced ERK activation is mediated by dopaminergic pathways, suggesting that interactions between cannabinoid and dopamine systems play a role in THC's effects on seizure susceptibility (11). Furthermore, THC's role in regulating mood and neuronal excitability underscores its complex pharmacology (12). The ratio of THC to CBD may significantly impact individual responses to cannabis (13-15). Prenatal THC exposure can affect offspring's motor performance and seizure susceptibility (16). CBD's influence on calcium channels also suggests potential implications for seizure management (17). Besides CB1 receptors, THC may also interact with other receptor systems that modulate neuronal activity. For example, THC can influence glutamate receptors, which are essential for excitatory neurotransmission. The interplay between cannabinoid and glutamatergic signaling may further exacerbate excitatory conditions conducive to seizures (18). The endocannabinoid system itself plays a complex role in regulating neuronal excitability. Endocannabinoids like 2-arachidonoylglycerol (2-AG) are produced during

seizures and can dampen excitatory signals through CB1 receptor activation. However, excessive THC consumption can overwhelm this protective mechanism, leading to heightened seizure risk due to disrupted homeostasis within the endocannabinoid system (19).

Thus, the relationship between cannabinoids and seizures remains contentious; while some studies indicate beneficial effects on seizure control, others highlight proconvulsant properties (20-26).

Conclusion

Hashish may cause seizures and its associated complications. Although it is rarely reported, physicians working in emergency departments should be aware of this untoward effect of hashish. Increasing awareness and education regarding the risks associated with cannabis use is imperative for protecting vulnerable populations, particularly adolescents and individuals with seizure histories. Healthcare providers are uniquely positioned to lead these efforts by educating patients and communities about the potential dangers of cannabis consumption.

Acknowledgment

We would like to express our sincere gratitude to the ICU staff of Tehran Imam Hossein Hospital.

Conflict of Interest: None

Funding: None

Ethical considerations: Approved by the local Ethics Committee (IR.SBMU.RETECH.REC.1401.880).

Authors' contribution: All authors had equal contribution to this paper. SS has managed the

patient, GA has drafted the manuscript and followed the patient, BB has designed the concept and revised the manuscript.

Table 1: Laboratory data upon arrival

WBC	11,000 /uL
PLT	323,000 /uL
Hb	14 g/dl
Na	144 meq/L
K	3.9 meq/L
Mg	2 mg/dl
P	4.3 mg/dl
Ca	8.8 mg/dl
Alb	3.5 g/dl
FBS	109 mg/dl
BUN	30 mg/dl
Cr	1.1 mg/dl
CRP	12 mg/L
Influenza A/B	Not detected
PCR for covid	Not detected

References

1. Thornton C, Dickson KE, Carty DR, Ashpole NM, Willett KL. Cannabis constituents reduce seizure behavior in chemically-induced and scn1a-mutant zebrafish. *Epilepsy Behav.* 2020;110:107152.
2. Gosetti F, Consonni V, Ballabio D, Orlandi ME, Amodio A, Valeria Picci M, et al. From the Streets to the Judicial Evidence: Determination of Traditional Illicit Substances in Drug Seizures by a Rapid and Sensitive UHPLC-MS/MS-Based Platform. *Molecules.* 2023;28(1):164.
3. Abdel-Salam OM, Sleem AA, Sayed MAE-BM, Khadrawy YA, Morsy FA. Cannabis sativa increases seizure severity and brain lipid peroxidation in pentylenetetrazole-induced kindling in rats. *Biomedical and Pharmacology Journal.* 2018;11(3):1187-97.
4. Devinsky O, Cilio MR, Cross H, Fernandez-Ruiz J, French J, Hill C, et al. Cannabidiol: pharmacology and potential therapeutic role in epilepsy and other neuropsychiatric disorders. *Epilepsia.* 2014;55(6):791-802.
5. Kaczor, E.E., et al., The potential proconvulsant effects of cannabis: a scoping review. *Journal of Medical Toxicology,* 2022. 18(3): p. 223-234
6. Emoto J, Weeks K, Kallail KJ. Accidental acute cannabis intoxication presenting as seizure in pediatrics patients. *Kansas journal of medicine.* 2020;13:129.

7. Wilson J, Freeman TP, Mackie CJ. Effects of increasing cannabis potency on adolescent health. *The Lancet Child & Adolescent Health.* 2019;3(2):121-8.
8. Whalley BJ, Lin H, Bell L, Hill T, Patel A, Gray RA, et al. Species-specific susceptibility to cannabis-induced convulsions. *Br J Pharmacol.* 2019;176(10):1506-23.
9. Espinosa-Jovel, C., Cannabinoids in epilepsy: Clinical efficacy and pharmacological considerations. *Neurología (English Edition),* 2023. 38(1): p. 47-53.
10. Walsh KB, Andersen HK. Molecular pharmacology of synthetic cannabinoids: delineating CB1 receptor-mediated cell signaling. *International journal of molecular sciences.* 2020;21(17):6115.
11. Thomas GM, Haganir RL. MAPK cascade signalling and synaptic plasticity. *Nature Reviews Neuroscience.* 2004;5(3):173-83.
12. Powles, T., et al., Cannabis-induced cytotoxicity in leukemic cell lines: the role of the cannabinoid receptors and the MAPK pathway. *Blood,* 2005. 105(3): p. 1214-1221.
13. Rosenberg EC, Tsien RW, Whalley BJ, Devinsky O. Cannabinoide und Epilepsie.
14. Boggs DL, Nguyen JD, Morgenson D, Taffe MA, Ranganathan M. Clinical and preclinical evidence for functional interactions of cannabidiol and Δ^9 -tetrahydrocannabinol. *Neuropsychopharmacology.* 2018;43(1):142-54.
15. Colizzi M, Bhattacharyya S. Does cannabis composition matter? Differential effects of delta-9-tetrahydrocannabinol and cannabidiol on human cognition. *Current Addiction Reports.* 2017;4:62-74.
16. Englund A, Freeman TP, Murray RM, McGuire P. Can we make cannabis safer? *The Lancet Psychiatry.* 2017;4(8):643-8.
17. de Salas-Quiroga A, Díaz-Alonso J, García-Rincón D, Remmers F, Vega D, Gómez-Cañas M, et al. Prenatal exposure to cannabinoids evokes long-lasting functional alterations by targeting CB1 receptors on developing cortical neurons. *Proceedings of the National Academy of Sciences.* 2015;112(44):13693-8.
18. Liu, C., et al., Cannabinoids promote progression of HPV-positive head and neck squamous cell carcinoma via p38 MAPK activation. *Clinical Cancer Research,* 2020. 26(11): p. 2693-2703.
19. Gordon, E. and O. Devinsky, *Marihuana: Effects on neuronal excitability and seizure threshold,* in *Marihuana and Medicine.* 1999, Springer. p. 619-629.
20. Cilio MR, Thiele EA, Devinsky O. The case for assessing cannabidiol in epilepsy. *Epilepsia.* 2014;55(6):787-90.
21. Hegde M, Santos-Sanchez C, Hess CP, Kabir AA, Garcia PA. Seizure exacerbation in two patients with focal epilepsy following marijuana cessation. *Epilepsy Behav.* 2012;25(4):563-6.

22. Breijyeh Z, Jubeh B, Bufo SA, Karaman R, Scrano L. Cannabis: A Toxin-Producing Plant with Potential Therapeutic Uses. *Toxins (Basel)*. 2021;13(2).
23. Keeler MH, Reifler CB. Grand mal convulsions subsequent to marijuana use. Case report. *Dis Nerv Syst*. 1967;28(7 Pt 1):474-5.
24. Consroe PF, Wood GC, Buchsbaum H. Anticonvulsant nature of marihuana smoking. *Jama*. 1975;234(3):306-7.
25. Mortati K, Dworetzky B, Devinsky O. Marijuana: an effective antiepileptic treatment in partial epilepsy? A case report and review of the literature. *Rev Neurol Dis*. 2007;4(2):103-6.
26. Maa E, Figi P. The case for medical marijuana in epilepsy. *Epilepsia*. 2014;55(6):783-6.