Case Report

Neurological Dysregulation in a 4-Year-Old Child Caused by Unintentional Cannabis Intoxication: A Case Report and a Mini-Review of the Literature

Vasiliki-Maria Kymioni *, Elena Chatzikalil, Maria Mandrapilia, Irini Karatsoli, Spyridon Karageorgos, Stavros Antonopoulos, Konstantinos Kakleas

Posted Date: 27 August 2026

doi: 10.20944/preprints202608.1953.v1

Keywords: cannabis; child; unintentional intoxication; mental status; reduced consciousness

 doi:10.20944/preprints202608.1953.v1

Case Report

Neurological Dysregulation in a 4-Year-Old Child Caused by Unintentional Cannabis Intoxication: A Case Report and a Mini-Review of the Literature

Vasiliki-Maria Kymioni *, Elena Chatzikalil, Maria Mandrapilia, Irini Karatsoli, Spyridon Karageorgos, Stavros Antonopoulos and Konstantinos Kakleas

First Department of Paediatrics Aghia Sophia Children's Hospital, Athens, Greece

* Correspondence: vmariakymioni@gmail.com

Abstract

Background: The increasing incidence of cannabis consumption and marijuana legalization in many countries worldwide has led to a growing exposure of the pediatric populations, leading to a steady increase in the number of unintentional cannabis intoxications in children and infants. Edible cannabis formulations have gained popularity, sometimes being 10-times stronger compared to the old ones, leading to neurological defects (dizziness, altered mental status, and generalized neurological dysregulation) in vulnerable pediatric individuals. Case presentation: We report a case of unintentional cannabinoid intoxication in a child experiencing altered mental status. After elimination of differential diagnoses, a toxic origin was considered, and biological testing led to the diagnosis of cannabis intoxication. Conclusions: Reporting similar cases is of great importance for increasing awareness among healthcare providers, thereby facilitating rapid diagnosis and therapeutic evaluation, especially in vulnerable populations.

Keywords: cannabis; child; unintentional intoxication; mental status; reduced consciousness

Introduction

Over the recent years, the legalization and widespread availability of cannabis products—particularly high-potency edibles—have been linked to a marked increase in unintentional pediatric exposures, especially among children under 6 years of age. In the United States, reports of edible cannabis ingestion in children increased by over 1,300% between 2017 and 2021, with similar sharp rises seen in France and other countries over the past decade [1,2].

Clinically, pediatric cannabis intoxication typically presents with decreased level of consciousness, ataxia, hypotonia, and respiratory compromise. Tachycardia, nausea, mydriasis, and seizures have also been reported [3]. Lethargy occurs in nearly three-quarters of cases, with hypotonia, tachycardia, and mydriasis also being common findings [3]. Young children are particularly susceptible to cannabis intoxication, as their low body mass and immature metabolism can lead to greater drug absorption and prolonged side effects—even with small doses [4]. Additionally, modern edible products often contain much higher concentrations of tetrahydrocannabinol (THC) compared to older formulations, increasing the risk of severe intoxication even with minimal accidental ingestion [5]. The delayed symptom onset and overlap with other acute neurologic or infectious diseases often result in prolonged and sometimes extensive diagnostic evaluations [5]. Early recognition of cannabis intoxication remains challenging, especially when no ingestion history is reported, and symptoms are non-specific.

We present the case of a previously well 4-year-old girl who arrived via ambulance with a sudden onset of reduced level of consciousness, which was eventually attributed to cannabis ingestion. This case underscores the crucial importance of maintaining a broad differential, even in the absence of an initial history of exposure or specific clinical indicators.

 doi:10.20944/preprints202608.1953.v1

Case Presentation

With parental consent, we report a 4-year-old girl who presented to the emergency department with acute altered consciousness. Approximately three hours before presentation, her mother noticed that she was minimally responsive to verbal stimuli and generally lethargic. There was no history of fever, head trauma, seizure-like activity, or accidental ingestion of medication or toxic substances.

The child was born at term via standard delivery with an uneventful perinatal period. She was fully immunized, with no significant medical history. She was achieving all developmental milestones for her age. The family history was unremarkable.

On presentation, she appeared somnolent, without rash or fever. Skin perfusion was normal. Cardiorespiratory examination revealed a regular heart rhythm and normal breath sounds bilaterally, without signs of respiratory distress. The abdomen was soft, mildly distended, non-tender, with present bowel sounds. No hepatosplenomegaly or palpable lymphadenopathy was noted. No nuchal rigidity or other signs suggestive of meningeal irritation were observed. Nevertheless, the child was still lethargic, with a GCS score of 10 . Pupils were equal and reactive to light. Deep tendon reflexes were symmetric and brisk, and the Babinski sign was positive bilaterally. There were no apparent neurological focal deficits or cranial nerve abnormalities.

The blood glucose was normal (100 mg/dl). Blood tests, including metabolic screening, blood gases, and infection markers, were within normal limits. (Table 1). The urgent brain CT and MRI scan showed no evidence of intracranial hemorrhage, space-occupying lesions, or structural abnormalities. Subsequently, a lumbar puncture was performed, and Cerebrospinal fluid microscopy and biochemistry were normal. Pending the results of cerebrospinal fluid PCR and cultures, as well as metabolic evaluation and autoantibodies testing, the patient was started on empiric treatment with intravenous ceftriaxone and acyclovir (Table 2). She was also admitted to the ward for continuous monitoring. Pediatric neurology and cardiology assessments showed no specific findings beyond the altered consciousness. Initial EEG demonstrated generalized theta and delta wave slowing, more prominent in posterior and right-sided leads, which was consistent with a widespread brain dysfunction that could be attributed to infection, metabolic abnormality, drug or toxin ingestion, and injury [6].

During the first hours of hospitalization, the child's responsiveness fluctuated and mainly remained lethargic. Gradually, her clinical status improved. By the end of the first day of admission, the patient had regained full consciousness. Given the rapid and spontaneous improvement in the neurological status, along with the absence of structural abnormalities on neuroimaging, normal cerebrospinal fluid and infectious workup, and unremarkable metabolic testing, the diagnostic focus shifted toward a potential toxicological cause. A urine toxicology screen returned positive for high concentrations. Upon further discussion, the parents disclosed that the child had likely consumed marijuana-containing jelly beans.

The diagnosis of acute cannabis intoxication was confirmed. Empiric antimicrobial and antiviral therapy was discontinued. The child remained clinically stable throughout the remainder of her hospital stay, with no recurrence of symptoms. Social services and the appropriate public health authorities were notified, given the nature of the exposure, and evaluated the family. The child was discharged in excellent clinical condition, with full neurological recovery and instructions for continued follow-up in the pediatric neurology outpatient clinics due to the EEG findings.

A chronological summary of the patient's clinical course, diagnostic evaluation, therapeutic interventions, and diagnostic reasoning is presented in Table 3. Given the unexpected nature of the diagnosis and its impact on the family, the parents' perspective on the clinical experience is presented. The patient's parents reported significant anxiety during the initial evaluation because the cause of their child's altered mental status was unknown. Following the diagnosis of accidental cannabis intoxication and the child's full recovery, they expressed gratitude for the multidisciplinary care provided and emphasized the importance of keeping cannabis-containing products out of children's reach. They agreed to share this case to help raise awareness of an increasingly recognized cause of acute neurological symptoms in children.

 doi:10.20944/preprints202608.1953.v1

Table 1. Laboratory and Diagnostic Findings at Admission.

Parameter	Result	Interpretation
Hemoglobin	9.9 g/dL	Mild anemia
WBC	6.13 ×10³/μL	Normal
Lymphocytes	69%	Relative lymphocytosis
CRP	7 mg/L	Not suggestive of bacterial infection
Procalcitonin	0.02 μg/L	Negative for bacterial infection
Glucose	103 mg/dL	Normal
pH	7.41	Normal acid–base status
pCO_2	35.1 mmHg	Normal
HCO_3^-	22.7 mmol/L	Normal
Lactate	0.8 mmol/L	No lactic acidosis
CSF WBC	28 /μL	Mild pleocytosis
CSF RBC	28–30 /HPF	Likely traumatic tap
CSF Glucose	54 mg/dL	Normal
CSF Protein	<20 mg/dL	Normal

CRP, C-reactive protein; CSF, cerebrospinal fluid; WBC, white blood cells.

Table 2. Immunologic, Metabolic, and Toxicologic Investigations.

Category	Test	Result	Interpretation
Autoimmune / Encephalitis panel	Anti-NMDAR, CASPR2, AMPAR1/2, LGI1, GABAB	Negative	No autoimmune encephalitis
	Anti-MOG, Anti-AQP4	Negative	No demyelinating disease
	ANA	1:160 (fine speckled)	Non-specific finding
	Anti-dsDNA	Negative	No SLE evidence
Metabolic evaluation	Plasma amino acids, urine organic acids, acylcarnitine profile	Normal	No inborn error of metabolism
CSF immunology	IgG index, oligoclonal bands	Normal / Absent	No intrathecal inflammation
Toxicology screen	Cannabinoids	**Positive (548.8 μg/L)**	Consistent with cannabis exposure
	Other substances	Negative	—

ANA, antinuclear antibodies; CSF, cerebrospinal fluid; MOG, myelin oligodendrocyte glycoprotein; NMDAR, N-methyl-D-aspartate receptor.

Table 3. Timeline of the patient's clinical course.

Time	Event	Diagnostic Reasoning
Admission	GCS 10, lethargy	CNS infection, encephalitis, metabolic disease, intoxication considered
CT/MRI normal	Structural lesion less likely	
CSF normal	Infectious meningitis/encephalitis less likely	
EEG slowing	Non-specific encephalopathy	
Toxicology positive	Cannabis intoxication confirmed	

 doi:10.20944/preprints202608.1953.v1

Discussion

To the best of our knowledge, this is the first case of accidental cannabis intoxication reported in a child from Greece, where cannabis is illegal for recreational use but has been legalized for medical purposes since 2017 [7]. Acute cannabinoid intoxication in the pediatric population is a rising public health issue, especially in countries where the legalization of cannabis coincides with a child-neglectful environment. Data from the United States show a substantial increase in pediatric visits to the Emergency Department, related to cannabis exposure [5]. There are a few other cases of accidental cannabis intoxication in children [3,4,7–13] (Table 4).

Table 4. A summary of reported cases of unintentional cannabis intoxication.

Country of Origin [Ref]	Age (years)	Clinical manifestations	Screening method	Therapeutic evaluation	Time to resolution	Outcome/ Comments on intoxication severity
Worldwide [3]	<6	Lethargy, Ataxia, Hypotonia,Tachycardia, Hypertension	Urine drug screen	Supportive care	24-28 hours	18% ICU,6% intubated, 100% survived
France [8,9]	<5	Lethargy, Ataxia, Coma, Tachycardia, Hypertension, Bradypnea	Urine drug screen	Supportive care, benzodiazepines in case of seizures	24-72 hours	Potential link between the increased incidence of comas and increased THC concentration
Colorado [10]	<12	Vomiting, Tachycardia, Bradycardia	Urine drug screen	Supportive care, ventilation in case of seizures	24-48 hours	The severity of symptoms resulted from combination of the age, the form (edible), and dose
San Francisco [11]	1-4	Coma, Hypotonia, Seizure, Tachycardia, Bradycardia	Serum drug screen	ICU, ventilation	48-72 hours	More severe symptoms and longer hospital length of stay compared to adult studies
Columbia [12]	2-6	Vomiting, Lethargy, Bradycardia. Tachycardia, Hypertension	N/A	Activated charcoal	N/A	N/A
Worldwide Most cases from Canada and USA [13]	<4	Lethargy, Hypotonia, Ataxia, Vomiting, Bradycardia. Tachycardia	Urine drug screen,Serum drug screen	Supportive care, benzodiazepines in case of seizures	24-48 hours	N/A
USA[14]	0-15	Hypotension, Hypoventilation, Coma, Tachycardia	Urine drug screen, Serum drug screen	Supportive care, oxygen supplementation	6-48 hours	Because of the new higher concentrated products our understanding of the effects due to ingestion and inhalation is still not

 doi:10.20944/preprints202608.1953.v1

						well studied or understood
Canada [4]	<6	Hypotonia, Seizures, Tachycardia, Hypertension	Urine drug screen	Supportive care, intubation	6-20 hours	N/A

ICU: intensive care unit.

Cases reported in medical literature indicate that children under 6 years of age are at significant risk of accidental ingestion, with ages ranging from <1 to 15 years. [4,8–14]. Cannabis consumption is occurring mostly through accidental ingestion of edibles rather than deliberate use [15]. Edible products include gummies, chocolates, cookies, and brownies that may contain high tetrahydrocannabinol levels with concentrations ranging from 15 to 91% and up to 500 mg tetrahydrocannabinol per item, according to the 2024 Canadian Cannabis Survey [16]. Children younger than 6 years of age are at a high risk of consuming entire products containing high tetrahydrocannabinol levels, primarily when products are marketed as appealing candies [15]. Doses of 1 to 5 mg are considered micro-doses, while concentrations above 100 mg are considered high [15].

The majority of cannabis intoxication cases occurred in America (mainly Canada, USA), although there are two studies from Europe (France) as well. Worth noting, in Colorado, cases of pediatric cannabis intoxication increased significantly and at a higher rate than the rest of the USA, with the number mainly growing in the 2 years after legalization, suggesting that legalization did affect the incidence of exposures [10]. Significant gaps remain related to evidence-based clinical strategies and social risk factors of acute cannabinoid intoxication. Cannabis legalization in the USA and Canada has removed some of the research barriers (previously restricted research materials and social desirability bias against participation). Further research on this field will contribute to increased awareness and improved clinical management of these cases.

Symptoms typically appear within a couple of hours and may last over 6 hours in severe cases, especially when children consume high quantities [15]. The most typical symptoms are tachycardia (73-83%), lethargy (71-83%), hypotonia (65% of exposed children), vomiting and/or nausea (20-30%) [8,17]. Our patient presented with hypotonia and lethargy. Coma is less frequent and is observed only in 10-18% of exposures, mainly after intoxication with edibles with high cannabinoid concentrations. [5,11].

Severe toxicity that involves significant cardiovascular complications (bradycardia, hypotension, sinus tachycardia requiring intravenous fluids, dysrhythmias), respiratory dysregulation, apnea, or severe neurological impairment (seizures, myoclonus, unresponsiveness, responsiveness only to painful stimuli) is not common [18]. Real-world evidence suggests that the severity of cannabis intoxication results from a combination of age, the form (edible), and the dose [10]. In pediatric patients, age-related differences in CYP enzyme expression affect tetrahydrocannabinol pharmacokinetics, resulting in higher central nervous system distribution and longer half-life [19]. Recent evidence also explains that expression of enzyme systems responsible for hepatic metabolism are closely linked to age, associating intoxication severity mostly to age rather than the concentration consumed [20,21]. Severe complications (intubation and death) occurred in the minority of cases. Regarding systematic research, less than 10% of the cases were intubated [3], while only one death occurred between the cases [22]. Specifically, a 3-year-old toddler died shortly after ingesting a cannabis substance, showing neurological and respiratory symptoms requiring intubation with fentanyl and ketamine, highlighting the dangers in cannabis ingestion in the pediatric population [22]. Long-term effects of cannabinoid intoxication in infants and children may include persistent neurodevelopmental changes, increased mental health risks, and respiratory or cardiovascular issues [23,24]. Thankfully, the patient we report did not have any long-term complications.

Most cases reported reflecting real-world evidence use urine and serum blood tests to detect cannabinoid intoxication [8–11,13,14]. Medical literature suggests that urine analysis helps confirm

 doi:10.20944/preprints202608.1953.v1

intoxication, with monitoring the tetrahydrocannabinol to creatinine levels being proper in confirming recent consumption (ratio > or >= 0.5) [5,25]. Serum analysis is preferred for indicating acute effects after cannabis consumption; tetrahydrocannabinol serum levels can differentiate occasional users from regular consumers because of its long plasma half-life and accumulation in frequent users [25]. Hair analysis is suggested in literature; however, due to its low sensitivity and the requirement of specialized analytical techniques (mass spectrometry) it is not used in routine settings [25].

The standard treatment of acute cannabinoid intoxication is mainly supportive [14,26]. Treatment is guided by the nature of complications: neurological symptoms (e.g., seizures) are treated with benzodiazepines, cardiovascular symptoms (e.g., hypotonia) are treated with intravenous fluids or vasopressors [26]. Respiratory dysregulation may be treated with oxygen supplementation or mechanical ventilation [27]. In severe cases of cannabinoid toxicity, flumazenil is suggested [28]. Intoxicated children should be monitored for at least six hours to ensure symptom resolution. They may need hospital admission in cases of persistent mental status dysregulation, recurrent seizures, or unstable vital signs. Among the cases reported in the literature, the majority required mainly supportive care. At the same time, in most studies [7–9,12,13], ventilation and benzodiazepines were used in cases of seizures; one study [11] suggests the use of activated charcoal. One study [11] describes ICU admission for the severe cases (aged 1 to 4 years). Specifically, the reported pharmacotherapy treatments in the systematic studies presented [8–10,13,14] included: promethazine, atropine, bicarbonate, ipecac, diazepam, naloxone, physostigmine, flumazenil, dopamine, lorazepam, midazolam, dexmedetomidine, and antibiotics. Dexmedetomidine was used in a case of a lethargic infant who failed to tolerate benzodiazepine [29]. Flumazenil successfully treated cannabis intoxication-related coma in one case [30], while its use was proved unsuccessful in another case [31]. Long-term complications are not expected, as most cases are resolved within 24 to 48 hours, as was the case in our instance [1].

A diagnostic and therapeutic synopsis is presented in Figure 1.

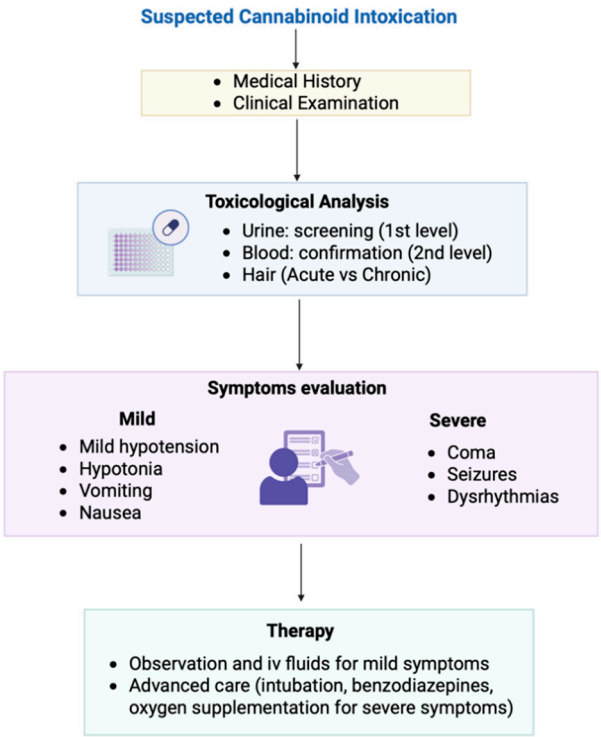

Figure 1. Diagnostic and therapeutic management of cannabinoid intoxication.

 doi:10.20944/preprints202608.1953.v1

Establishing the diagnosis of cannabis intoxication in pediatric patients and especially in toddlers, as in our case, can be challenging due to the unreliable medical history reported by caregivers, who sometimes fear of legal or social consequences. Due to these difficulties, toxicological testing is essential to confirm exposure and to rule out the alternative differential diagnoses: atrial tachycardia, allergic and environmental asthma, anxiety disorder, major depressive and/or bipolar disorder, and intoxication syndromes from other substances (amphetamine, cocaine, benzodiazepine).

Strengths and Limitations

A major strength of this report is the detailed diagnostic evaluation leading to toxicologically confirmed cannabis intoxication in a previously healthy child. To the best of our knowledge, this is the first published pediatric case of accidental cannabis intoxication reported from Greece, supplemented by a focused review of the available literature. However, as a single case report, the findings cannot be generalized, and the exact amount of tetrahydrocannabinol ingested could not be determined.

Conclusions and Future Perspectives

The increasing prevalence of pediatric cannabis intoxication highlights the urgent need for targeted public health interventions and stricter prevention strategies, including child-resistant packaging and public education campaigns. Clinicians should consider cannabis exposure in differential diagnoses for pediatric patients presenting with unexplained neurological, immunological, or metabolic symptoms. Future research should focus on longitudinal studies to better evaluate the pharmacological long-term aspects of cannabis exposure at a young age, and its epidemiological association with child neglect/maltreatment. Bridging these gaps may necessitate coordinated efforts across medical, legal, and social sectors, with the further aim to protect vulnerable children from cannabis related risks.

Topic	Item No	Checklist item description	Reported on Page Number/Line Number	Reported on Section/Paragraph
Title	1	The diagnosis or intervention of primary focus followed by the words “case report”		p 1 , Title
Key Words	2	2 to 5 key words that identify diagnoses or interventions in this case report, including “case report”		P1 , Keywords section
Abstract (Structured summary)	3a	Background: state what is known and unknown; why the case report is unique and what it adds to existing literature.		P1, abstract section
	3b	Case Description: describe the patient’s demographic details, main symptoms, history, important clinical findings, the main diagnosis, interventions, outcomes and follow-ups.		P1, abstract case description
	3c	Conclusions: summarize the main take-away lesson, clinical impact and potential implications.		P1abstract conclusion-abstract section
Introduction	4	One or two paragraphs summarizing why this case is unique **(may include references)**		P2-3 introduction
Patient Information	5a	De-identified patient specific information		P 3, Case Presentation, Paragraph 1

 doi:10.20944/preprints202608.1953.v1

	5b	Primary concerns and symptoms of the patient	P 3, Case Presentation, Paragraphs 1–2
	5c	Medical, family, and psycho-social history including relevant genetic information	P 3, Case Presentation, Paragraphs 3
	5d	Relevant past interventions with outcomes	Not applicable
Clinical Findings	6	Describe significant physical examination (PE) and important clinical findings	P 3, Case Presentation, Paragraphs 2-3
Timeline	7	Historical and current information from this episode of care organized as a timeline	P13-14, Table 3 (Timeline of the patient's clinical course)
Diagnostic Assessment	8a	Diagnostic testing (such as PE, laboratory testing, imaging, surveys).	Pages 3–4; Tables 1 and 2 (Pages 12–13)
	8b	Diagnostic challenges (such as access to testing, financial, or cultural)	Pages 2, 4, and 8 Sections: Introduction; Case Presentation; Discussion
	8c	Diagnosis (including other diagnoses considered)	Pages 3–4 and 8 Sections: Case Presentation; Discussion
	8d	Prognosis (such as staging in oncology) where applicable	Page 4 Section: Case Presentation (complete neurological recovery)
Therapeutic Intervention	9a	Types of therapeutic intervention (such as pharmacologic, surgical, preventive, self-care)	Pages 3–4 Section: Case Presentation
	9b	Administration of therapeutic intervention (such as dosage, strength, duration)	Page 3 Section: Case Presentation intravenous ceftriaxone , acyclovir (intravenous ceftriaxone and acyclovir)

 doi:10.20944/preprints202608.1953.v1

9c	Changes in therapeutic intervention (with rationale)	Page 3 Section: Case Presentation (intravenous ceftriaxone and acyclovir)

Ethical: Approval and Informed Consent. The authors confirm that all procedures complied with institutional ethical standards. Informed consent requirements were met in accordance with institutional policy. Written informed consent was obtained from the patient's parent/guardian for publication of this case report.

Disclosure: The authors declare no conflicts or financial interest in any product or service mentioned in the manuscript, including grants, equipment, medications, employment, gifts, and honoraria. Artificial intelligence–assisted language editing (ChatGPT, OpenAI) was used to refine the structure and grammar of the manuscript. The authors take full responsibility for the integrity and scientific accuracy of the manuscript. All authors attest to meeting the four criteria recommended by the ICMJE for authorship of this manuscript.

References

1. Tweet MS, Nemanich A, Wahl M. Pediatric edible cannabis exposures and acute toxicity: 2017–2021. Pediatrics.2023;151:e2022057761. doi:10.1542/peds.2022-057761.
2. Shi E, Minns A, Schwartz K. Mary and Jane: A case of severe marijuana intoxication in school-aged sisters. Clin Pediatr (Phila). 2022;61:490–492. doi:10.1177/00099228221081251.
3. Richards JR, Smith NE, Moulin AK. Unintentional cannabis ingestion in children: A systematic review. J Pediatr.2017;190:142–152. doi:10.1016/j.jpeds.2017.07.005.
4. Pepin LC, Simon MW, Banerji S, Leonard J, Hoyte CO, Wang GS. Toxic tetrahydrocannabinol dose in pediatric cannabis edible ingestions. Pediatrics. 2023;152:e2023061374. doi:10.1542/peds.2023-061374.
5. Malta G, Albano GD, Lavanco G, Brancato A, Cannizzaro C, Argo A, et al. Acute cannabis intoxication among the paediatric population. Front Toxicol. 2025;7.
6. Louis EKS, Frey LC, Britton JW, Hopp JL, Korb PJ, Koubeissi MZ, et al. Electroencephalography (EEG): An introductory text and atlas of normal and abnormal findings in adults, children, and infants. Chicago, IL: American Epilepsy Society; 2016.
7. Panagopoulos E, Koukoulis AN. The legal framework governing the production and use of medical cannabis in Greece – Greece embraces the therapeutic potential of medical cannabis. Eur Health Pharm Law Rev. 2022;6:81–86. doi:10.21552/ehpl/2022/2/7.
8. Claudet I, Mouvier S, Labadie M, Manin C, Michard-Lenoir AP, Eyer D, et al. Unintentional cannabis intoxication in toddlers. Pediatrics. 2017;140:e20170017.
9. Claudet I, Le Breton M, Bréhin C, Franchitto N. A 10-year review of cannabis exposure in children under 3 years of age: Do we need a more global approach? Eur J Pediatr. 2017;176:553–556.
10. Wang GS, Le Lait MC, Deakyne SJ, Bronstein AC, Bajaj L, Roosevelt G. Unintentional pediatric exposures to marijuana in Colorado, 2009–2015. JAMA Pediatr. 2016;170:e160971.
11. Vo KT, Horng H, Li K, Ho RY, Wu AHB, Lynch KL, et al. Cannabis intoxication case series: The dangers of edibles containing tetrahydrocannabinol. Ann Emerg Med. 2018;71:306–313.
12. Tsutaoka B, Araya-Rodríguez G, Durrani T. Edible marijuana labeling and packaging. Clin Pediatr (Phila).2018;57:227–230.
13. Gaudet LA, Hogue K, Scott SD, Hartling L, Elliott SA. Acute pediatric cannabis intoxication: A scoping review. J Child Health Care. 2024;28:196–214.
14. Takakuwa KM, Schears RM. The emergency department care of the cannabis and synthetic cannabinoid patient: A narrative review. Int J Emerg Med. 2021;14:10.
15. Zwiebel H, Greenky D, Goldman RD. Accidental cannabis ingestion in young children. Can Fam Physician.2025;71:161–163. doi:10.46747/cfp.7103161.

 doi:10.20944/preprints202608.1953.v1

16. Health Canada. Canadian cannabis survey 2022: Summary. Ottawa: Government of Canada; 2022.
17. Harvey T, Gomez R, Wolk B, Ozcan A, Harvey TN. Varied presentations of pediatric patients with positive cannabinoid tests. Cureus. 2022;14.
18. King-Stephens D. Seizures and cardiac dysrhythmias: And the beat (sometimes) goes on. Epilepsy Curr.2022;23:153575972211359.
19. Lu H, Rosenbaum S. Developmental pharmacokinetics in pediatric populations. J Pediatr Pharmacol Ther.2014;19:262–276.
20. Kearns GL, Abdel-Rahman SM, Alander SW, Blowey DL, Leeder JS, Kauffman RE. Developmental pharmacology—drug disposition, action, and therapy in infants and children. N Engl J Med. 2003;349:1157–1167.
21. Dotta A, Chukhlantseva N. Ontogeny and drug metabolism in newborns. J Matern Neonatal Med. 2012;25:75–76.
22. Favretto D, Cirnelli A, Pertile R, Stimamiglio R, Cestonaro C, Cuman O, et al. Infant death due to cannabis ingestion. Drug Test Anal. 2025. doi:10.1002/dta.3904.
23. Bourque J, Potvin S. Cannabis and cognitive functioning: From acute to residual effects, from randomized controlled trials to prospective designs. Front Psychiatry. 2021;12:596601.
24. Muheriwa-Matemba SR, Baral A, Abdshah A, Diggs BNA, Gerber Collazos KS, Morris KB, et al. Cardiovascular and respiratory effects of cannabis use by route of administration: A systematic review. Subst Use Misuse. 2024;59:1331–1351.
25. Musshoff F, Madea B. Review of biologic matrices (urine, blood, hair) as indicators of recent or ongoing cannabis use. Ther Drug Monit. 2006;28:155–163.
26. Kaczor EE, Mathews B, LaBarge K, Chapman BP, Carreiro S. Cannabis product ingestions in pediatric patients: Ranges of exposure, effects, and outcomes. J Med Toxicol. 2021;17:386–396.
27. Pélissier F, Claudet I, Pélissier-Alicot AL, Franchitto N. Parental cannabis abuse and accidental intoxications in children: Prevention by detecting neglectful situations and at-risk families. Pediatr Emerg Care. 2014;30:862–866.
28. Pianca TG, Sordi AO, Hartmann TC, von Diemen L. Identification and initial management of intoxication by alcohol and other drugs in the pediatric emergency room. J Pediatr (Rio J). 2017;93:46–52.
29. Cipriani F, Mancino A, Pulitanò SM, Piastra M, Conti G. A cannabinoid-intoxicated child treated with dexmedetomidine: A case report. J Med Case Rep. 2015;9:152.
30. Rubio F, Quintero S, Hernandez A, Fernandez S, Cozar L, Lobato IM, et al. Flumazenil for coma reversal in children after cannabis. Lancet. 1993;341:1028–1029.
31. Carstairs SD, Fujinaka MK, Keeney GE, Ly BT. Prolonged coma in a child due to hashish ingestion with quantitation of THC metabolites in urine. J Emerg Med. 2011;41:e69–e71.