Fatal Cannabis Resin Intoxication in a 10-Month-Old Infant with Severe Hyperkalemia: A Case Report and Literature Review

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Case Report

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Abstract

Fatal cannabis intoxication in infants is exceptionally rare. We report a case of lethal cannabis resin ingestion in a previously healthy 10-month-old male infant with confirmed toxicological evidence of Δ9-tetrahydrocannabinol (Δ9-THC) exposure and severe hyperkalemia. The child was admitted one hour after the suspected accidental ingestion, presenting with somnolence, axial hypotonia, fixed gaze, hypotension, and intermittent apnea. Laboratory testing revealed a serum potassium level of 6.8 mEq/L, and toxicological analysis confirmed Δ9-THC exposure. He was admitted to the pediatric intensive care unit and received endotracheal intubation, mechanical ventilation, intravenous fluid resuscitation, vasoactive support, and anticonvulsant therapy. Despite maximal supportive measures, he developed refractory seizures and progressive cardiovascular collapse, leading to death within 24 hours of admission. This case highlights the potential lethality of cannabis ingestion in infants and suggests that severe hyperkalemia may contribute to cardiovascular collapse in this setting. Early recognition, intensive management, and preventive strategies—including caregiver education and child-resistant packaging—are essential to mitigate the risk of pediatric cannabis intoxication.

Introduction

Accidental cannabis ingestion in infants is an increasingly recognized public health concern worldwide, even in regions where cannabis use remains illegal, such as Tunisia [1–3]. Despite its rarity, fatal pediatric cannabis intoxication cases have been documented, often associated with ingestion of high-potency cannabis resin, edibles, or polysubstance exposures [4–6]. Prevention relies on secure storage, caregiver education, and clinician awareness for early diagnosis and management.

Data from poison control centers report a sharp increase in unintentional pediatric cannabis exposures in recent decades, with rising admissions to hospitals and ICUs [7]. The clinical spectrum of acute cannabis intoxication in children varies widely and can include somnolence, agitation, hypotonia, bradycardia, hypotension, respiratory depression, seizures, and, in severe cases, coma or death [8–10]. Infants are particularly vulnerable due to immature hepatic metabolism, reduced cytochrome P450 and UGT enzyme expression, increased brain exposure due to immature blood-brain barrier, and higher oral bioavailability of cannabinoids [11–12].

Moreover, the type of cannabis product ingested significantly influences the toxicity profile; edibles, with delayed absorption and high THC content, are frequently implicated in more severe pediatric cases [12]. Herein, we report a fatal case of accidental cannabis resin ingestion in a 10-month-old infant, distinct for severe hyperkalemia and rapid progression, contributing valuable data to the limited literature on pediatric cannabis fatalities.

Case Presentation

A previously healthy 10-month-old male infant was brought to the emergency department approximately one hour after the sudden onset of somnolence.

The infant presented with somnolence, axial hypotonia, a fixed gaze, hypotension and intermittent apnea. According to the parents, the child had been playing in the living room and accessed a small piece of cannabis resin belonging to a household member

The infant had no notable past medical history, no known genetic disorders, and the family history was unremarkable. There was no prior history of neurological or metabolic conditions. Psycho-socially, the child lived in a household where cannabis resin was stored, which may have been easily accessible. There were no prior medical interventions relevant to this case. The current presentation was the first acute event prompting emergency care.

Clinical Examination and Important Findings

On admission, the infant's vital signs were: heart rate 85 beats/min, blood pressure 70/40 mmHg, respiratory rate 28 breaths/min with intermittent apnea and oxygen saturation at 92% on room air.

Physical examination revealed generalized hypotonia, fixed gaze accompanied by intermittent inappropriate smiling, equal and reactive pupils, and no signs of trauma. Neurological assessment showed decreased responsiveness, minimal spontaneous movements and absent protective reflexes.

Investigations and Diagnostic Testing

Initial investigations showed a complete blood count (CBC) within normal limits. Electrolyte analysis revealed **severe hyperkalemia**, with serum potassium measured at 6.8 mEq/L, while the remaining electrolytes were within normal ranges. Renal and hepatic function tests were normal,

and blood glucose was measured at 85 mg/dL. Arterial blood gas (ABG) analysis demonstrated a pH of 7.32, $PaCO_2$ of 48 mmHg, and PaO_2 of 70 mmHg. Toxicological screening confirmed exposure to Δ9-tetrahydrocannabinol (THC), with no evidence of other toxic substances. No diagnostic limitations were reported. Differential diagnoses included other causes of altered consciousness and hypotonia, but toxicological confirmation supported cannabis intoxication.

Table 1
Laboratory findings on admission

Parameter	Result	Reference Range	Units
Hemoglobin	11.5	11.0–14.5	g/dL
White blood cells	9200	6,000–17,000	/µL
Platelets	280000	150,000–450,000	/µL
Sodium (Na^+)	138	135–145	mmol/L
Potassium (K^+)	6.8	3.5–5.0	mmol/L
Chloride (Cl^-)	102	98–107	mmol/L
Bicarbonate (HCO_3^-)	22	22–28	mmol/L
Urea	18	10–40	mg/dL
Creatinine	0.4	0.2–0.5	mg/dL
Glucose	85	70–110	mg/dL
AST (SGOT)	25	10–40	U/L
ALT (SGPT)	20	10–40	U/L
ABG – pH	7.32	7.35–7.45	—
ABG – $PaCO_2$	48	35–45	mmHg
ABG – PaO_2	70	80–100	mmHg
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Management and Therapeutic Interventions

The infant was admitted to the pediatric intensive care unit (PICU). Endotracheal intubation and mechanical ventilation were initiated in assist-control mode, with a tidal volume of 6–8 mL/kg, respiratory rate set at 20–30 breaths per minute, positive end-expiratory pressure (PEEP) of 5 cm H_2O, and FiO_2 adjusted to maintain oxygen saturation above 92%. Intravenous fluid resuscitation was performed with isotonic crystalloids (0.9% saline), administered as an initial bolus of 20 mL/kg over 15–20 minutes to address hypotension, with additional boluses titrated according to the hemodynamic response. Maintenance fluids were administered with isotonic solutions adapted to the infant's weight and estimated requirements. Cardiac and respiratory parameters, including heart rate, blood pressure, oxygen saturation, and end-tidal CO_2, were continuously monitored.

Despite maximal supportive therapy, vasoactive drugs such as norepinephrine were required, initiated at 0.05–0.1 µg/kg/min and titrated up to 0.3 µg/kg/min. The patient developed refractory seizures, treated with benzodiazepines and other anticonvulsants, but his condition continued to deteriorate. Ultimately, the child progressed to cardiopulmonary arrest and died within 24 hours of admission (Table 1).

Table 2
Timeline of Clinical Course

Time	Clinical Findings	Interventions	Notes / Observations
0 h	Infant playing in living room; possible access to cannabis resin	—	First suspected exposure
~ 1 h	Sudden onset of somnolence	—	First symptom noted by parents
Arrival at ED	HR 85 bpm, BP 70/40 mmHg, RR 28/min (intermittent apnea), SpO_2 92%; generalized hypotonia, fixed gaze, minimal responsiveness	Initial stabilization	Pupils equal/reactive, no trauma
Initial tests	CBC normal; K^+ 6.8 mEq/L; renal/hepatic function normal; glucose 85 mg/dL; ABG: pH 7.32, $PaCO_2$ 48 mmHg, PaO_2 70 mmHg	Toxicology positive for Δ9-THC, no other toxins detected	Differential diagnoses considered; cannabis intoxication confirmed
PICU admission	Persistent hypotension, altered consciousness	Intubation, mechanical ventilation, IV fluids, continuous monitoring, initiation of vasoactive drugs	Initial stabilization in PICU
12–24 h	Refractory seizures, persistent hypotension	Anticonvulsants (benzodiazepines), norepinephrine up to 0.3 µg/kg/min	Progressive deterioration despite maximal supportive care
~ 24 h	Cardiopulmonary arrest	Advanced life support attempted	Fatal outcome within 24 hours of admission

AHR, heart rate; BP, blood pressure; RR, respiratory rate; SpO_2, oxygen saturation; CBC, complete blood count; ABG, arterial blood gas; $PaCO_2$, partial pressure of carbon dioxide in arterial blood; PaO_2, partial pressure of oxygen in arterial blood; Δ9-THC, delta-9-tetrahydrocannabinol; PICU, pediatric intensive care unit.

Outcome and Follow-up

There were no further interventions due to the rapid clinical deterioration. No further diagnostic tests were possible post-mortem.

This case illustrates a fatal outcome despite maximal intensive care efforts and highlights the severe prognosis associated with accidental cannabis ingestion in infants.

Discussion

The clinical presentation included profound somnolence, hypotonia, intermittent apnea, and severe hyperkalemia (K^+ 6.8 mEq/L), notably higher than levels typically reported in pediatric cannabis intoxication, where potassium is usually normal or low [13–16]. This hyperkalemia may reflect systemic toxicity, potentially secondary to renal impairment, cellular potassium shifts, or other disturbances following Δ9-THC exposure. Such findings are rare in humans and animals, highlighting the exceptional severity of this case [14–17].

Table 3
Summary of Severe or Fatal Pediatric Cannabis Intoxication Cases

Year	Author(s)	Age/Sex	Cannabis Product	Confirmed Toxicity	Key Clinical Features	Laboratory Findings	Outcome	Country/Setting	Comments
2025	Present Case	10 mo/M	Resin	Δ9-THC	Somnolence, hypotonia, intermittent apnea, refractory seizures	Severe hyperkalemia, K^+ 6.8 mEq/L	Fatal	Tunisia	First reported fatality with confirmed Δ9-THC and severe hyperkalemia in infant
2023	Cohen N. et al.	2 year/M	Edible	THC	Somnolence, seizures	Mild electrolyte changes	Survived	USA	Severe toxicity requiring ICU
2023	Pepin LC et al.	3 year/F	Edible	THC	Ataxia, hypotonia	Normal labs	Survived	Canada	High-dose exposure (> 1.7 mg/kg THC)
2022	Lim JS et al.	1 year/M	Resin	THC	Lethargy, hypotension	Not reported	Survived	South Korea	Post-legalization increase in severe exposures
2025	Malta G. et al.	4 year/F	Edible	THC	Lethargy, vomiting	Elevated liver enzymes	Survived	Italy	

Mortality directly attributable to acute cannabis exposure is exceptionally rare. Most pediatric deaths with detected cannabinoids involve polydrug use, trauma, underlying cardiac disease, or synthetic cannabinoids [18–19]. In England (1998–2020), only 4% of deaths with cannabinoids detected were associated with cannabis alone [19]. Contributory mechanisms include CB1 receptor–mediated CNS depression, autonomic disturbances, cardiac ion channel perturbation, and inflammatory/oxidative pathways, with synthetic cannabinoids often producing more severe toxicity.

Severe pediatric cannabis intoxication ranges from stupor and respiratory compromise to seizures and cardiovascular instability. High-potency products, edibles, and unintentional ingestion in young children are disproportionately associated with ICU admission. Documented risk factors include polydrug exposure, underlying cardiac conditions, high-potency products, synthetic cannabinoids, and oral ingestion.

Management remains primarily supportive, including airway protection, seizure control, fluid resuscitation, and cardiovascular stabilization. Despite maximal intervention, our patient progressed to refractory seizures, hypotension, and cardiopulmonary arrest. This case emphasizes the lethality of cannabis ingestion in infants and the importance of heightened clinical awareness, caregiver education, secure storage, child-resistant packaging, and public health measures.

Conclusion

This case highlights the potential for severe and fatal outcomes following accidental cannabis ingestion in infants. Severe hyperkalemia may serve as a marker of systemic toxicity and warrants further investigation. Even a single ingestion of cannabis resin can lead to profound neurological depression, refractory seizures, cardiovascular collapse, and death.

Management remains entirely supportive, emphasizing early recognition, vigilant monitoring, and aggressive intensive care. The rapid progression in this case illustrates that even maximal interventions may be insufficient.

Preventive strategies are critical to reduce morbidity and mortality. These include caregiver education, secure storage of cannabis products, child-resistant packaging, and public health initiatives to raise awareness of pediatric exposure risks.

This report contributes to the limited literature on fatal pediatric cannabis intoxication and underscores the need for continued surveillance, research, and preventive measures to protect children from inadvertent exposure.

Declarations

Informed Consent

Informed consent for publication of the case report was obtained from the patient's legal guardians in accordance with ethical standards.

Patient Consent

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient/parent/guardian/relative of the patient.

Trial registration number/date

N/A

Ethical statement: According to our institutional policies, ethical approval was not required for publication of a single case report.

Data availability

Underlying data

Figshare: Dataset supporting "**Fatal Cannabis Resin Intoxication in a 10-Month-Old Infant with Severe Hyperkalemia**: **A Case Report and Literature Review**

This project contains the following underlying data:

Table 1. Laboratory findings on admission

Table 2. Timeline of Clinical Course

Table3. **Table 3. Summary of Severe or Fatal Pediatric Cannabis Intoxication Cases**

Figshare: CARE checklist for "**Fatal Cannabis Intoxication in an Infant Following Accidental Ingestion: A Case Report and literature review**

DOI: 10.6084/m9.figshare.30273745

Maaroufi N. Dataset supporting ""**Fatal Cannabis Resin Intoxication in a 10-Month-Old Infant with Severe Hyperkalemia**: **A Case Report and Literature Review**". Figshare. 2025. DOI: 10.6084/m9.figshare.30273745

Competing interests

The authors declare no competing interests.

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Author contributions

- *Dr Neila Maaroufi : patient management, manuscript drafting, figure preparation*
- *Dr Safia Othmani: literature review, discussion drafting, manuscript editing*
- *Dr Amina Jebali: discussion drafting, manuscript editing*

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