BMC Pediatrics

CASE REPORT

Open Access

From cultural remedy to medical emergency: a case report of camphor toxicity in a one-year-old male patient

Yoalkris Elizabeth Salcedo[1*], Ashley Eliana Soriano-López[1], Aileen Almonte[1], Carlos Manuel Matos Florimón[1], Gillian Elaine Marcelino[1] and Gonzalo Antonio Güémez[2]

Abstract

Background Camphor (CMP) is widely used for cultural and medicinal purposes in regions like the Middle East, India, Nigeria, and Latin America. Despite its traditional uses, CMP presents serious toxic risks, particularly in infants and children, where even small ingested doses can lead to life-threatening symptoms. This case report highlights the risk posed by camphor ingestion in a pediatric patient, emphasizing its relevance to healthcare providers in regions where camphor is commonly used.

Case presentation.

A one-year-old male was admitted to the Dr. Hugo Mendoza Pediatric Hospital in Santo Domingo, Dominican Republic, after ingesting camphor tablets. He presented in a postictal state following generalized tonic–clonic seizures at home, with further seizures occurring in the hospital. The seizures were controlled with intravenous diazepam, and diagnostic tests, including a head CT scan, revealed no significant abnormalities. The patient received supportive care, including levetiracetam for seizure control, and was monitored in the pediatric intensive care unit. After clinical improvement, he was transferred to the general pediatric unit without further complications.

Conclusions This case highlights the dangers of camphor exposure, particularly in young children, and the need for healthcare providers to consider CMP poisoning in cases of unexplained seizures. It also underscores the importance of educating parents about the safe storage of camphor-containing products. Further research is needed to explore the long-term effects of camphor toxicity and develop effective public health measures to prevent such poisonings.

Keywords Camphor toxicity, Pediatric poisoning, Seizures, Neurotoxicity, Case report

Background

Camphor (CMP) is a cyclic ketone of the hydroaromatic terpene group, available as a solid white crystalline compound with a very intense odor that easily converts to a gas and as a liquid colorless substance with a strong smell and pungent taste [1–3]. CMP is naturally produced from the bark of *Cinnamomum camphora L.* trees that grow in Japan, China, Vietnam, Asia, Africa, Sri Lanka, Australia, Canada, and the United States (US), and synthesized from pinene, a hydrocarbon derivative of turpentine

*Correspondence:
Yoalkris Elizabeth Salcedo
ysalcedo@est.unibe.edu.do
[1] Universidad Iberoamericana, Santo Domingo, Dominican Republic
[2] Hospital Pediátrico Dr. Hugo Mendoza, Santo Domingo, Dominican Republic

oil [1–5]. It has been used for a long time in the Middle East's traditional medicine [5], in Indian religious ceremonies and for household fragrance [3], as a Nigerian aphrodisiac [1], contrary to Iran's traditional medicine, which suggests that CMP reduces sexual desire [5], and in Latin America. In the Dominican Republic, traditionally, CMP cubes have been used for household and baby clothing fragrance, to ward off evil spirits or negative energy, and its odor by the bed as a cold remedy [6]. It has also been reported that taxi drivers use CMP cubes to absorb car humidity, and many women mix a small dose of CMP with water and drink it to stop breastfeeding [6].

Despite its widespread use in vaporized or topical preparations, musculoskeletal anesthetic formulations, moth repellents, antimicrobial preparations, and herbal remedies for managing inflammation-related conditions such as rheumatism, sprains, bronchitis, and hemorrhoids, CMP has been associated with toxic effects when used in large quantities [3, 4]. This is particularly concerning in the pediatric population, where unintentional or accidental CMP poisonings have resulted in seizures, irritability, confusion, gastrointestinal distress, and other central nervous system-related disorders [1]. In cases of CMP oil toxicity, symptoms such as sweating and agitation may progress to more severe manifestations, including seizures, cardiac arrhythmias, and cardiopulmonary arrest [7]. In adults, ingesting up to 30 mg/kg of camphor has generally not posed a significant risk [3]. However, no consensus guidelines exist for toxic serum levels in the pediatric population [8]. CMP has been reported as neurotoxic at doses exceeding 50 mg/kg body weight and fatal at 4 g (500 mg/kg body weight) in adults, 0.5–1 g in children, and 70 mg/kg in infants [2, 3, 9].

The toxic effects of camphor (CMP) have been recognized since the nineteenth century. In 1983, the U.S. Food and Drug Administration (FDA) established an 11% limit on the concentration of CMP permitted in products [10]. Despite these regulations, pure CMP cubes and products, often lacking proper labeling of CMP content, continue to be imported into the U.S. from countries such as India and China. These products are readily available in ethnic grocery stores and bodegas [10]. This widespread availability contributed to over 10,000 cases of CMP exposure via topical agents in the U.S. in 2011 alone [10].

Toxic effects of CMP typically manifest within 5 to 90 min after ingestion, following first-order kinetics, with a half-life of approximately 15 h [10]. CMP is initially metabolized by cytochrome P450 enzymes, followed by oxidation by alcohol dehydrogenase and aldehyde dehydrogenase in the liver, after which it is conjugated with glucuronic acid to facilitate water-soluble urinary excretion [10]. In pregnant women, oral ingestion of CMP can lead to high concentrations of CMP in the fetal brain, liver, kidneys, and blood [10]. This can potentially result in spontaneous abortion, as fetuses lack the necessary enzymes to hydroxylate and conjugate CMP with glucuronic acid [10]. In cases where abortion did not occur, CMP was detectable in maternal blood 15 min post-ingestion but undetectable after 8 h [9]. However, CMP may still be present in the amniotic fluid, cord blood, and fetal tissues, including the brain, liver, and kidneys, at delivery 36 h later. Notably, no teratogenic effects have been reported following topical exposure (FDA Pregnancy Category C) [9, 10].

Case presentation

This case report presents a one-year-old male patient who presented to the Dr. Hugo Mendoza Pediatric Hospital in Santo Domingo, Dominican Republic, after ingesting a camphor tablet on September 11th, 2024, at 02:00 AM. The patient was brought to the emergency department by his mother. The mother reports that the patient ingested an unknown quantity of camphor tablets 4 h prior to their arrival at the hospital. At home, prior to their arrival, the patient was witnessed to experience episodes of generalized tonic–clonic movements that lasted approximately 2 min, which prompted their transition to the hospital.

On arrival, the patient was unconscious and in a postictal state. On the physical exam, the patient was well-developed, eupneic, and afebrile. The patient's anthropometric measurements at the time of presentation were as follows: weight of 22 pounds, height of 28 inches, and a body surface area (BSA) of 0.45 m^2. Lungs were resonant to percussion with vesicular breath sounds throughout peripheral lung fields. No rales, rhonchi, wheezes, or rubs. His abdomen had normal bowel sounds, with no pain on superficial or deep palpation. The extremities had a capillary refill time of 3 s, with positive distal pulses and no edema. Vital signs included a temperature of 37 degrees Celsius, a heart rate of 112 beats per minute, a respiratory rate of 28 breaths per minute, and a blood pressure of 90/60 mmHg.

The patient has no pertinent past medical history, and both his parents also have no pertinent medical histories. His mother reports that the patient's vaccine schedule was up to date with the Dominican Republic's vaccination scheme.

In the emergency room, the patient experienced 2 episodes of vomiting of alimentary content and 2 episodes of generalized tonic–clonic convulsions, which were controlled with IV diazepam. An arterial blood gas, hemogram, comprehensive metabolic panel, radiograph of the thorax, and computed tomography (CT) of the head were ordered, with the results shown in the Tables 1 and 2 and Figs. 1, 2 and 3. The patient was started on an IV mixed

Table 1 Laboratory studies realized on September 11th, 2024

Biological Parameter	Results	Reference Range
Hematocrit (Hct)	32%	37—47%
Total hemoglobin (tHb)	10.8 g/dL	12—16 g/dL
Oxygen saturation (sO2)	71%	94.0—98.0%
Blood urea nitrogen (BUN)	20.4 mg/dL	7—20 mg/dL
Creatinine (Cr)	0.51 mg/dL	0.5—1.1 mg/dL
Aspartate transaminase (AST)	23 U/L	10—40 U/L
Alanine transaminase (ALT)	24 U/L	7—56 U/L
Sodium (Na+)	135 mmol/L	135—145 mmol/L
Potassium (K+)	3.0 mmol/L	3.5—5.0 mmol/L
Calcium (Ca++)	10.3 mg/dL	8.5—10.2 mg/dL
Magnesium (Mg++)	1.76 mg/dL	1.7—2.2 mg/dL
Chloride (Cl-)	104 mmol/L	96—106 mmol/L

Table 2 Laboratory studies realized on September 13th, 2024

Biological Parameter	Results	Reference Range
Hematocrit	32%	37—47%
Total hemoglobin (tHb)	10.9 g/dL	12—16 g/dL
Oxygen saturation (sO2)	97%	94.0—98.0%
Sodium (Na+)	138 mmol/L	135—145 mmol/L
Potassium (K+)	3.8 mmol/L	3.5—5.0 mmol/L
Calcium (Ca++)	9.56 mg/dL	8.5—10.2 mg/dL
Chloride (Cl-)	105 mmol/L	96—106 mmol/L

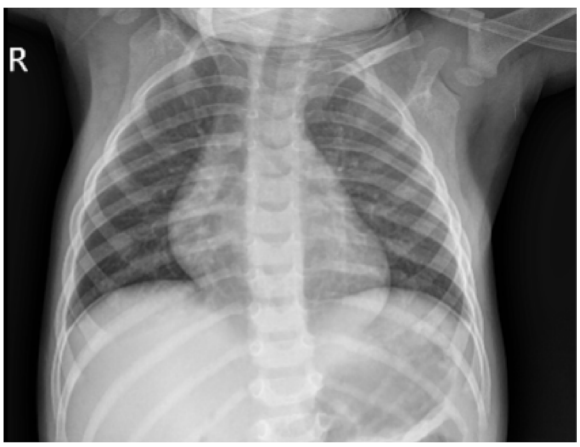

Fig. 1 Posteroanterior (PA) radiograph of the thorax. Patient had no relevant findings

solution of 0.33% sodium chloride and 5% dextrose at 1000 mL in 24 h, followed by 42 mL per hour.

The patient was then admitted into the intensive care unit at 03:00 AM on September 11th, 2024 with the diagnoses of intoxication by camphor and secondary

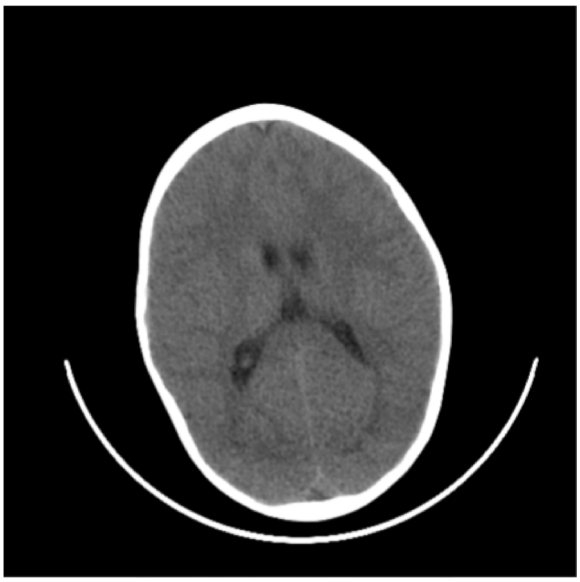

Fig. 2 Axial view computed tomography of the brain. Patient had no relevant findings

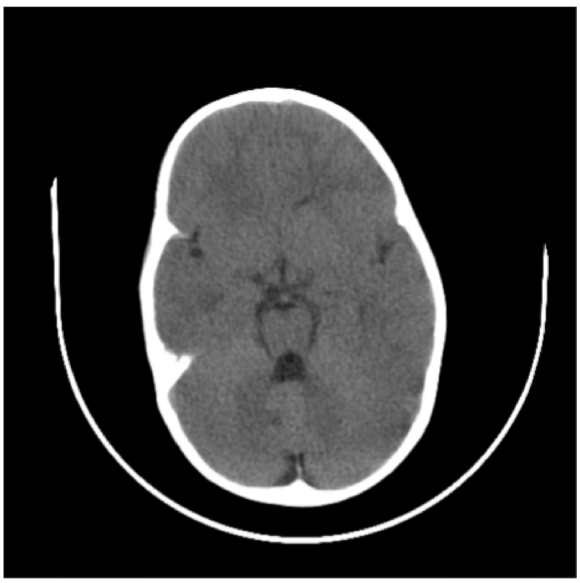

Fig. 3 Axial view computed tomography of the brain. Patient had no relevant findings

seizures. The medication regimen in the ICU included the following:

- Levetiracetam (10 mg/kg/day) use: 50 mg every 12 h IV diluted in 20 mL of 0.9% sodium chloride solution.
- Omeprazole (1 mg/kg/day) use: 10 mg every 24 h IV diluted in 10 mL of distilled water, passed in a slow bolus.

- Diazepam (0.2 mg/kg/dose) use: 2 mg IV PRN in the case of convulsions.

In the ICU, the management plan focused on monitoring hemodynamic, ventilatory, metabolic, renal, and neurologic parameters for any significant changes or abnormalities. Special attention was given to the hourly monitoring of vital signs, surveillance for any signs of bleeding, and proactive measures to prevent electrolyte imbalances. Throughout the remainder of the morning and the following day (September 12th), the patient experienced no significant events. Due to clinical improvement, the patient was transferred from the ICU to the general pediatrics unit on September 13th, 2024.

On September 14, 2024, the patient was discharged home in stable condition, accompanied by family members. During the hospital stay, the patient demonstrated good tolerance to oral intake and responded well to respiratory physiotherapy. The patient's family was advised to arrange follow-up care with outpatient neurology and their pediatrician.

Discussion

Camphor (CMP) is widely available over the counter in many pharmacies and grocery stores in the Dominican Republic. While CMP has various cultural uses, it can be fatal to infants and children when ingested, even in small doses. In cases of suspected CMP poisoning, clinicians should be alerted by the characteristic smell of camphor on the patient's skin, breath, or vomitus [11]. Seizures typically occur within the first two hours of ingestion [12]. Children exhibiting signs of CMP toxicity, particularly seizures, should be admitted to the pediatric intensive care unit for a minimum of 48 h [3]. No antidote for CMP poisoning exists, and enhanced elimination methods are not recommended [7]. Treatment is primarily supportive, with a focus on airway management and seizure control, utilizing benzodiazepines, phenobarbital, or phenytoin for uncontrolled seizures, and continuous infusions of midazolam or propofol for refractory status epilepticus [3].

CMP is a lipophilic neurotoxin capable of crossing the blood–brain barrier, potentially leading to excitation and convulsions through the GABAergic pathway, followed by central nervous system (CNS) depression due to the inhibition of catecholamine secretion via blockade of nicotinic acetylcholine receptors [3, 4, 7]. However, the precise pharmacodynamics of CMP remain unclear, and it is not yet established whether toxicity results from the parent compound, its metabolites, or both [7]. Somade [1] reports that high doses of CMP in rats have caused oxidative stress, histopathological changes in the cerebral cortex, hippocampus, liver, lungs, and kidneys, disruptions in male reproductive and thyroid hormones, and upregulation of inflammatory chemokines and cytokines via nuclear factor-kappa B activation.

Njan et al. [4] demonstrated that edible CMP directly antagonizes GABAergic neurotransmission in rats. At a dose of 300 mg/kg, it induces a transient elevation in blood glucose levels, normalizing within 24 h. Additionally, CMP significantly reduces the activity of antioxidant enzymes, including catalase and glutathione peroxidase, and depletes glutathione levels. The resultant accumulation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) leads to increased malondialdehyde levels, reflecting the severity of oxidative stress through lipid peroxidation. This oxidative damage impairs cellular function, damages biomolecules and organelles, and perpetuates a cycle of oxidative stress and brain inflammation.

Manoguerra et al. [13] reported a case of a 6-month-old boy who died after receiving a cumulative dose of 24.5 g (3,000 mg/kg) of CMP over five months in a whiskey-based remedy. Postmortem examination revealed diffuse cerebral edema with neuronal degeneration and necrosis, as well as hepatomegaly characterized by fatty infiltration and hepatocellular necrosis [13]. The authors [13] also described a case involving a 15-month-old boy who crawled through spilled CMP oil, subsequently developing ataxia followed by generalized convulsions that persisted for two days despite phenobarbital administration. In our case, the patient's seizures following CMP tablet ingestion ceased after the administration of diazepam. One year after being seizure-free, the 15-month-old boy described by Manoguerra et al. [13] was exposed to a 4.8% CMP ointment for respiratory relief, resulting in a single generalized convulsion and five years of phenobarbital treatment. In contrast, our patient only required levetiracetam during hospitalization and had no recurrence of seizures.

Manoguerra et al. [13] also reported a case of a 2-month-old girl who developed elevated serum transaminases after applying a 4.8% CMP ointment to her chest and neck three times daily for five days. Our patient, however, showed no significant changes in serum transaminases following the ingestion of pure CMP tablets. Similar to the case reported by Narayan et al. [11], our patient was afebrile, with stable vital signs and normal routine hematological and biochemical parameters, including blood glucose, serum electrolytes, and calcium levels. Additionally, our patient's imaging studies, including a head CT scan, had no abnormal findings, as observed in case reports by Santos et al. [10] involving a 25-year-old woman and a 15-month-old boy.

Mild anemia was identified in our one-year-old male patient. However, due to the lack of documented medical

history regarding anemia, it remains unclear whether the observed hemoglobin values are pre-existing. The most recent data from the National Micronutrients Survey (2014) [14], which evaluated preschool-aged children in the Dominican Republic, reported that 28.1% of children under five years of age were anemic. Moreover, no case reports on CMP toxicity have described anemia or low hemoglobin as a clinical sign of poisoning. Given that our patient's remaining biological parameters and imaging studies were within normal limits, it is not possible to determine whether pre-existing anemia influenced the clinical presentation or outcomes of CMP toxicity in this case.

Most case reports suggest that the toxic effects of camphor (CMP) typically resolve within 24 to 48 h after ingestion. However, Santos et al. [10], along with three additional case reports, have documented cases of persistent neurological effects lasting at least one week post-ingestion. In our case, the patient's symptoms resolved within 48 h, and he was discharged in stable condition. Despite this, outpatient follow-up is essential to monitor for any potential long-term neurological sequelae.

Based on these findings, we recognize that while camphor remains a valuable compound with diverse applications, its widespread availability and potential for toxicity, particularly in children, highlight the critical need for awareness, proper usage, and ongoing research to ensure its safe integration into modern practices. We strongly recommend advising parents to store CMP-containing products securely and out of reach of children, as even small amounts can be toxic. Understanding the risks associated with CMP exposure is essential for preventing accidental poisonings. Parents should be encouraged to carefully read product labels and seek immediate medical attention if exposure is suspected. Finally, studies evaluating the effectiveness of public health interventions to reduce accidental CMP poisonings could provide valuable insights for developing preventive strategies.

Conclusion

Camphor (CMP) has a wide range of cultural and medicinal applications worldwide, underscoring its long-standing significance across various traditions. However, despite its widespread use, over-the-counter CMP products carry significant toxic risks, particularly in pediatric populations, where accidental poisonings have resulted in serious complications.

Early consideration of CMP exposure as a potential cause of seizures in otherwise healthy children and prompt treatment of CMP toxicity by clinicians are crucial for improving patient outcomes. Finally, further research is needed into the long-term effects of CMP toxicity, particularly in vulnerable populations such as children and pregnant women.

Abbreviations

CMP	Camphor
CT	Computed Tomography
CNS	Central Nervous System
BSA	Body Surface Area
IV	Intravenous
FDA	U.S. Food and Drug Administration
GABA	Gamma-Aminobutyric Acid (refers to GABAergic pathways)
ROS	Reactive Oxygen Species
RNS	Reactive Nitrogen Species
AST	Aspartate Transaminase
ALT	Alanine Transaminase
Na+	Sodium
K+	Potassium
Ca++	Calcium
Mg++	Magnesium
Cl-	Chloride
pH	Potential of Hydrogen (acidity/alkalinity)
pO2	Partial Pressure of Oxygen
pCO2	Partial Pressure of Carbon Dioxide
sO2	Oxygen Saturation
BUN	Blood Urea Nitrogen
Cr	Creatinine
tHb	Total Hemoglobin
g	Grams
mg	Milligrams
mg/kg	Milligrams per Kilogram

Acknowledgements

N/A.

Authors' contributions

Y.E.S. and A.E.S.L. contributed to the conception of the case report and manuscript writing. A.A. and C.M.M.F. assisted with data collection and analysis. G.A.G.M. was directly involved in the clinical care of the patient and provided critical input for the manuscript revisions. G.E.B.M. contributed to the final manuscript revisions. All authors read and approved the final manuscript.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. This study was approved by the Ethics Committee of Dr. Hugo Mendoza Pediatric's Hospital. Written informed consent to participate in the study was obtained from the patient's legal guardians.

Consent for publication

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

Competing interests

The authors declare no competing interests.

Received: 21 September 2024 Accepted: 5 February 2025
Published online: 20 February 2025

References

1. Somade OT. Camphor toxicity: A review of recent findings. Proc Natl Acad Sci. 2022;93:775–90. https://doi.org/10.1007/s40011-022-01437-0.
2. Duda-Madej A, Viscardi S, Grabarczyk M, et al. Is camphor the future in supporting therapy for skin infections? Pharmaceuticals. 2024;17(6):715. https://doi.org/10.3390/ph17060715.
3. Kumar S, Kavitha TK, Angurana SK. Kerosene, camphor, and naphthalene poisoning in children. Indian J Crit Care Med. 2019;23(Suppl 4):S278–81. https://doi.org/10.5005/jp-journals-10071-23316.
4. Njan AA, Ogharanduku U, Ilesanmi O, et al. Mechanism and changes associated with edible camphor induced neurotoxicity. Pharmacol Toxicol Nat Med. 2022;2(2):37–49. https://doi.org/10.52406/ptnm.v2i2.29.
5. Rahimi M, Shokri F, Hassanian-Moghaddam H, et al. Severe camphor poisoning: A seven-year observational study. Environ Toxicol Pharmacol. 2017;52:8–13. https://doi.org/10.1016/j.etap.2017.03.009.
6. Perez X. El alcanfor hace daño. Listin Diario. 2010 Jun. Available from: https://listindiario.com/la-vida/2010/01/06/127080/el-alcanfor-hace-dano.
7. Jabbour SG, Mawlawi NA, Kuthbudeen MW, et al. Camphor oil toxicity: A case report. Cureus. 2023;15(10): e47412. https://doi.org/10.7759/cureus.47412.
8. Ramya R, Harish N, Sundari S. A case of camphor toxicity in a toddler: All on skin is not safe. J Clin Diagn Res. 2023;17(11):SD01–2. https://doi.org/10.7860/JCDR/2023/64443.18649.
9. PubChem Compound Summary for CID 2537, Camphor. Bethesda (MD): National Library of Medicine (US), National Center for Biotechnology Information; 2004. Available from: https://pubchem.ncbi.nlm.nih.gov/compound/Camphor.
10. Santos CD, Cabot JC. Persistent effects after camphor ingestion: A case report and literature review. J Emerg Med. 2015;48(3):298–304. https://doi.org/10.1016/j.jemermed.2014.05.015.
11. Narayan S, Singh N. Camphor poisoning—An unusual cause of seizure. Med J Armed Forces India. 2012;68(3):252–3. https://doi.org/10.1016/j.mjafi.2011.11.008.
12. Mathen PG, Sreekrishnan TP, Kumar KPG, et al. Camphor poisoning: A rare cause of acute symptomatic seizures in children. J Emerg Trauma Shock. 2018;11(3):228–9. https://doi.org/10.4103/JETS.JETS_21_18.
13. Manoguerra AS, Erdman AR, Wax PM, et al. Camphor poisoning: An evidence-based practice guideline for out-of-hospital management. Clin Toxicol (Phila). 2006;44(4):357–70. https://doi.org/10.1080/15563650600671696.
14. Iglesias Vázquez L, Valera E, Villalobos M, et al. Prevalence of Anemia in Children from Latin America and the Caribbean and Effectiveness of Nutritional Interventions: Systematic Review and Meta¯Analysis. Nutrients. 2019;11(1):183. https://doi.org/10.3390/nu11010183.

Publisher's Note