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Camphor poisoning: An unusual cause of seizure in a toddler

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Abstract: Camphor is a common household product with unrecognized neurologic side effects, especially seizure. This report is aimed at raising awareness among healthcare providers of camphor poisoning as an unusual cause of seizure. A 23-month-old previously well female child, presented with vomiting and generalized seizures. Symptoms started about 30 minutes after inadvertent ingestion of camphor oil. Examination findings revealed a conscious child with no focal neurological deficit. Investigation findings

were within normal limits. A diagnosis of camphor poisoning with seizures was made. She was treated with phenobarbitone and other supportive management with no recurrence of seizure. Healthcare providers need high index of suspicion about this an unusual cause of seizure. There is also a need for community education and appropriate regulation of camphor use for children.

Key words: Camphor, childhood poisoning, seizure

Introduction

Poisoning due to accidental ingestion of commonly available household toxic substances is a recognized cause of morbidity and mortality among infants and young children in Nigeria.^{1,2} The incidence and type of substance ingested vary from place to place and over time.^{3,4} In developing countries like Nigeria, accurate data are difficult to come by, but it is estimated to be the fourth major cause of ill health and death.⁵ Common agents implicated include kerosene, organophosphates, drugs, caustic soda, hypochlorite, and herbal concoctions.^{3,6,7} The availability of these poisonous household agents, the socioeconomic pressure which leads to poor supervision of the child, coupled with the inquisitive and impulsive nature of children make them vulnerable to poisoning.^{3,8}

Some poisoning agents tend to cause grave illness or lead to death even if accidentally ingested in very small quantity. Camphor is one such common substances available in many households. It is a cyclic ketone of the hydroaromatic terpene group and a dose of about 3-5 mL of 20% camphor oil or >30 mg/Kg is potentially lethal.^{9,10,11} Unfortunately, its potentially fatal toxic effect is not very well-recognized. The report is aimed at highlighting the neurotoxic effect of camphor and raising an awareness of its unusual cause of seizure in children. We report the case of a 23-month-old who presented with seizures due to ingestion of camphor oil.

Case report

A 23-month-old girl, with no previous history of seizures or prior head trauma was presented to the Emergency Paediatric Unit (EPU) of our facility, Bingham University Teaching Hospital, Jos, with a history of convulsion which occurred 30 minutes prior to presentation. Convulsion was described as generalized tonic seizure, lasted 15 minutes, and aborted spontaneously with full regain of consciousness. She also had two bouts of vomiting. The characteristic strong aromatic odour of camphor from her breath and the vomitus raised the suspicion of ingestion of camphor. She was then given milk and a banana prior to presentation, in an attempt to counter any untoward effect of the agent. The child was reported to have ingested an estimated 10-15mls of camphor oil from a bottle in the grandmother's house where she had been playing when the incident happened. Shortly after arrival at the EPU, she had a generalized tonic clonic seizure which lasted about 1-2 minutes and self-aborted

On arrival at the hospital and while on admission, she was afebrile and other vital signs were stable. She was conscious, with no focal neurological deficit. Examination of other systems yielded nothing of note. Results of routine haematological and biochemical tests, including random blood sugar, electrolytes and urea were within normal limits. She was booked for brain neuroimaging but could not be done up until discharge. She was placed on tabs Phenobarbitone 4mg/kg in 12hourly divided doses.

By the following day, there was no more convulsion, and she was subsequently discharged home. In a telephone interview with the mother two weeks later, she reported that the child was doing well and had no additional seizures.

Discussion

Camphor has been used historically for several conditions as cold remedy, muscle and joint liniment, lactation suppressant, analeptic, cardiac and central nervous system stimulant and rodent repellent.¹² It was initially produced from the bark of the *Cinnamomum camphora* tree but is presently produced synthetically from turpentine and has a characteristic, penetrating odour and a pungent, aromatic taste.¹³ It is a component of many preparations available over the counter (Vicks VapoRub, BenGay, Eucalyptos oil)

Several sporadic cases of unintentional camphor poisoning resulting from gastrointestinal, pulmonary, and dermal absorption and with symptoms ranging from confusion, irritability, gastrointestinal upset, and seizures have been reported, particularly in children.^{14,15,16} Children are particularly vulnerable to toxicity, because camphor is highly lipophilic and is easily absorbed through the skin and mucous membranes. The use of camphor products to treat common childhood illnesses is particularly concerning as this makes it readily available in many households. There are reports of its use as nasal decongestant and cough suppressant,¹⁷ as well as antipruritic and counterirritant agent.¹⁸ It is also used as inhalants in form of camphorated oil, at combination of 19% or 20% camphor in a carrier oil, for the household management of colds.¹⁶ The camphor oil the index patient ingested was available in the house as muscle liniment.

Ingestion of a dose of 30mg/kg and above is more likely to cause severe side effects, while consumption of about 50mg/kg will trigger neurological side effects, seizure being the most common manifestation seen within the first 90 minutes and can persist for 24hours.¹⁴ A previous study however showed that as little as 0.5 teaspoon (18.5mg/kg) caused toxicity in a 6-year-old child with mumps.¹⁹ The precise volume ingested by a child may be difficult to ascertain but, the clinical presentation can guide the clinician in estimating the dose of camphor the child is exposed to. A 5-ml dose approximately represents 1000 mg camphor (50–225 mg/kg).¹⁶ The index patient developed the toxic effects (convulsion and vomiting) within 30 minutes of ingestion and within two hours of exposure had had two episodes of convulsions. The minimum plausible dose she would have ingested would be 5mLs. It is also important to note that side effects of exposure to the agent are unlikely to occur if they do not develop in the first four hours of exposure.

Seizure, as seen in the index patient, is a common neurological manifestation of camphor poisoning. Other neurological presentations of camphor poisoning are confusion, agitation, myoclonus, ataxia, visual hallucination,

delirium, hyperreflexia, lethargy and even coma.^{14,16,20} Camphor is highly lipid soluble and primarily a neurotoxin with a chemical structure that allows for easy crossing of the blood-brain barrier.²¹ The exact mechanism of camphor induced seizures is not clear, but it is believed to occur at neuronal level where there is disruption of the oxidation cycle of the cytochrome oxidase system leading to rapid oxidation and depletion of high energy phosphorous compounds.^{21,22} Its toxic effect on the central nervous system (CNS) ranges from mild CNS excitation to generalized seizures. Other reported manifestations of the irritant property of camphor on mucosa following ingestion include nausea and vomiting,^{16,23} as experienced by the index patient, oral and oesophageal burning sensation,¹⁶ urinary incontinence,²⁴ tachycardia and myocarditis.²⁵ Chronic low dose dermal exposure has been reported to cause hepatic granulomatous hepatitis.¹⁶ A previous study documented postmortem findings of diffuse edema in the brain with neuronal degeneration and necrosis, as well as hepatomegaly with fatty infiltration of the liver and hepatocellular necrosis.²⁶

Diagnosis of camphor induced seizure can be made on clinical findings. However, in the presence of a febrile illness, it may be difficult to link camphor as the cause of seizure, as a diagnosis of febrile seizure may prevail. Similarly, in recurrent seizures, chronic exposure to camphor will require good clinical acumen for the diagnosis not to be missed. A high index of suspicion is therefore needed to make the diagnosis of camphor induced seizure. Toxicology test is usually reserved for medicolegal reasons, as these studies are not readily available in many facilities.²³ Nonspecific laboratory findings of camphor toxicity include leukocytosis, proteinuria, and transient elevation in hepatic transaminases.²⁷ In the index patient, there was no evidence of any febrile illness, hypoglycemia, or dyselectrolytemia, being the possible cause of seizures. Camphor ingestion was the best possible explanation for the seizures occurring in her.

Treatment is mainly supportive, giving attention to airway management and seizure control. Several reports have reported improvement in neurological symptoms following treatment with benzodiazepines or barbiturates.^{24,28,29} Benzodiazepines like diazepam, lorazepam or barbiturate like phenobarbitone can be used to manage the seizures, as in our case, phenobarbitone was used and seizure was controlled without any recurrence. Our patient did not experience any respiratory depression following administration of the anticonvulsant as has been reported in previous reports after administration of sedative-hypnotic drugs.^{29,30} The patient's caregivers attempted to reduce the toxic effect of camphor by giving milk and banana. However, camphor is rapidly absorbed after ingestion from the gastrointestinal tract, so gastrointestinal decontamination like use of activated charcoal, gastric lavage, ipecac syrup, salt water, milk, castor oil, magnesium sulfate and other emetics, cathartics or evacuants is not helpful.¹⁶ Given the

high propensity of seizure occurrence, these maneuvers may rather prove harmful for the patients. Patients who remain asymptomatic after four hours can be observed at home safely.

This report also brings to fore the importance of child safety in relation to harmful household products. Unsafe storage of medicines, unsafe storage of household chemicals, inadequate supervision of the child, mother employment during the daytime, non authoritative parenting styles, primary level education in the mother, poisonous plants in the home garden, and parental concern of lack of family support are some factors identified to risk accidental poisoning.^{3,8,31} Among these factors, inadequate supervision of the child,³¹ has been found to be one of the strongest causes of accidental poisoning.

In conclusion, this case highlights that camphor toxicity remains an important but under-recognized cause of seizure among children. There is a need to strongly discourage the use by community education and appropriate regulation of camphor use for children. Health care providers need to be aware about camphor toxicity when

evaluating seizures in paediatric age group with no other risk factors, as camphor products are readily available in many household and exposure could be acute ingestion, chronic dermal or inhalational. Regulatory bodies like Standard Organization of Nigeria (SON), National Food and Drug Administration Commission (NAFDAC) need to ensure that safe levels of camphor are contained in camphor containing agents and that there is proper labeling of these agents. More importantly there is a need for strict policies on secure packaging and prescription of medications as well as storage of dangerous household chemicals to prevent accidental poisoning. This report also raises a need for a scaling up of information given to parents and care givers on poison prevention and other child care practices that ensure child safety.

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