

Clinical Case Reports

Camphor poisoning in an adult: Seizures manifesting as 'mis-purposed' drug effect

ANKIT KUMAR SAHU, SONIKA RATHOOR

ABSTRACT

Camphor, a common aromatic hydrocarbon, is known to be potentially hazardous due to its acute harmful effects primarily on the central nervous system. Contrastingly, camphor is an integral component of various indigenous medicinal potions owing to its medicinal value. Camphor neurotoxicity has been reported in children. However, accidental or voluntary ingestion in adults is rare. We report a patient with voluntary ingestion of camphor, in a relatively large dose for alleviation of a medical condition.

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INTRODUCTION

Originally a tree bark derivative of *Cinnamomum camphora*, camphor is a ubiquitous organic chemical compound with a characteristic odour used for various medicinal and non-medicinal purposes. It forms a customary ingredient of oils, inhalants, emollients, coryza remedies, balms, analgesic and anti-inflammatory ointments. In lower concentrations, it is also used as a part of traditional medicine prescription for osteoarthritis, haemorrhoids, pain relief and common cold. Non-medicinal uses involve its role as a pesticide, readily combustible substance used for religious prayers, etc.

However, owing to its easy availability over the counter or otherwise, camphor can be a household poison. The literature has multiple descriptions of neurological toxicity being caused by accidental poisoning in children.^{1–6} It is rapidly absorbed via the skin, gastrointestinal tract and respiratory mucosa. Toxicity affects predominantly the central nervous system and to some extent the gastrointestinal tract. The most commonly reported adverse effect is seizures, which are caused by oxidative abnormalities in neuronal mitochondrial Krebs's cycle due to camphor derivatives. The toxic dose ranges from 30 to 50 mg/kg. The lethal dose in children is estimated to be 1.0–1.5 g.

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We report an apparently healthy young man presenting to the emergency room (ER) with multiple episodes of seizures with baffling disclosure regarding the underlying aetiology for the apparently 'idiopathic' seizure.

THE CASE

A 38-year-old smoker, chronic alcoholic man of south Asian ethnicity presented to a tertiary care ER with complaints of multiple episodes of generalized tonic-clonic seizures for 4–5 hours. There were 3–4 episodes of involuntary irregular tonic-clonic movements of the body associated with tongue bite, up rolling of eyes, spontaneous micturition and transient loss of consciousness for 15–20 minutes followed by a period of post-ictal confusion. No prior history of seizure episodes, head injury, headache, vomiting, fever, cognitive impairment, any other focal neurological deficit including cranial nerve palsy, any chronic illness such as diabetes, hypertension, etc. or psychosocial or behavioural abnormality was present.

In the ER, the patient was given a bolus dose of phenytoin to terminate seizures followed by conservative management. Random blood sugar, oxygen saturation and other vital signs of the patient were normal. Routine blood investigations including calcium levels and customary toxicology screening were within normal limits. Electrocardiography, chest X-ray and gadolinium contrast MRI of the brain showed no abnormality. After stabilizing, the patient was transferred to the ward where he was put on an oral daily maintenance dose of phenytoin. Within 3–4 days phenytoin was tapered and the patient was discharged subsequently for follow-up in the surgical outpatient for further treatment of haemorrhoids.

The patient gave a history of haemorrhoids for the past 2–3 years, which were painless, non-prolapsing, associated with the passage of hard stools, constipation and was at the time of admission having fresh episodes of bleeding per rectum for the past 1 week. General physical and systemic examination was unremarkable except for the presence of grade 2 haemorrhoids. On further enquiring about the treatment that he took for the actively bleeding haemorrhoids, the patient admitted to taking about 5 g of camphor, on the advice of an ayurvedic practitioner by placing it in a split banana around 1–2 hours before the first episode of seizure.

DISCUSSION

Camphor poisoning is a well-documented entity in children with well-defined guidelines for its management.⁷ However, its occurrence in adults is rare.⁸ The most common presentation in both age groups is generalized tonic-clonic seizures. Rahimi *et al.*⁹ published a report of 30 patients presenting to the ER with camphor poisoning over 7 years. In this Iranian study, the mean (SD) ingested amount was 2.3 (1.3) g (range 0.7–6.7 g), nearly half compared to our patient, which could be responsible for the earlier occurrence of seizures and presentation to ER. The treatment of choice for camphor-induced seizures is benzodiazepines. Our patient presented with the first 'idiopathic' seizure episode without any obvious underlying explanation or aetiology. On questioning, the unusual cause of the presentation was discovered. This, therefore, makes a case for proper evaluation of a patient with a history of seizures and related

relevant details regarding unusual sources of poisoning especially when the individual presents with the first episode without any overt underlying cause. It also underlines the need for patient education and information for treatable common diseases so that they do not receive potentially harmful treatment.

Conclusion

Although voluntary, non-accidental ingestion of camphor in adults is rare, yet many individuals accept it as a part of alternative medicine for some medical conditions including haemorrhoids. This is potentially hazardous and preventable. Moreover, a possibility of camphor-induced neurotoxicity must be kept in mind if one encounters acute seizures in an otherwise healthy individual without any apparent cause.

Conflicts of interest. None declared

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