

Anti-flatulence treatment and status epilepticus: a case of camphor intoxication

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We describe a case of a young child who lived in Hong Kong who presented with a severe epilepticus status after a return flight to Paris. Routine laboratory tests failed to establish a cause. Upon further questioning, the parents reported that the nanny had given an abdominal massage to the child with an unlabelled solution reported to have anti-flatulence effects. Toxicological analysis of this solution revealed the presence of camphor. Although the highly toxic effects of camphor have long been established, the present case illustrates that camphor continues to be a source of paediatric exposure. This case highlights the importance of systematic questioning and recalls the extreme danger associated with camphor even when administered transcutaneously.

A 4-month-old previously healthy girl was admitted to our intensive care unit because of repetitive seizures with bradycardia and apnoea. She lived in Hong Kong with non-consanguineous normal parents. One week previously, the girl was hospitalised for vomiting and abdominal distension, without fever. During the return flight to Paris, eyelid myoclonus appeared. At the airport she had a generalised convulsion which was treated by intrarectal diazepam. Status epilepticus developed in the department of neurology after a series of generalised tonic-clonic seizures, despite intravenous treatment with clonazepam and phenytoin. The seizures stopped with 0.4 mg/kg/day of clonazepam associated with phenobarbital. An electroencephalogram showed a generalised convulsion and a slowed rhythm. Cerebral computed tomography was normal. Laboratory tests, including renal and liver function, cerebrospinal fluid examination (with culture, polymerase chain reaction for detection of herpes simplex virus, Epstein-Barr virus, and *Mycoplasma pneumoniae*) and search for metabolic abnormalities were normal. Upon further questioning, the parents reported that the nanny had given an abdominal massage to the child with a solution reported to have anti-flatulence effects. This solution, manufactured in the Philippines, exhibited the characteristic and penetrating odour of camphor, and toxicological analysis confirmed the presence of camphor. The infant was extubated after 3 days and her neurologic status improved within a week. At discharge, the neurological examination was normal.

DISCUSSION

Camphor has been used historically as an aphrodisiac, abortifacient, antiseptic, cardiac and central nervous system stimulant or as a remedy for colds and musculoskeletal pains.¹ Obtained originally by distillation of the bark of the

Cinnamomum camphora tree, camphor can now be prepared synthetically from turpentine oil. Camphor is a cyclic ketone in the hydroaromatic terpene group (fig 1). Once absorbed, it is oxygenated to produce the alcohol campherol which is then conjugated in the liver with glucuronic acid to become soluble in water. Most camphor is ultimately excreted in the urine. Camphor crosses the placental barrier which accounts for its embryotoxic effects. The toxic dose seems to range from 15–30 mg/kg bodyweight or above 500 mg.¹ Absorption from the gastrointestinal tract occurs rapidly (within 5–90 min from the time of ingestion). In the present case, the development of seizures 72 h after abdominal massage suggests delayed absorption of camphor via the transcutaneous route. The major manifestations of camphor intoxication involve the gastrointestinal tract. Severe poisonings are characterised by neurological symptoms including irritability, hyper-reflexia, tonic muscular contraction and seizures. The exact mechanism of camphor induced seizure activity is unknown. Nevertheless, young children seem particularly exposed because

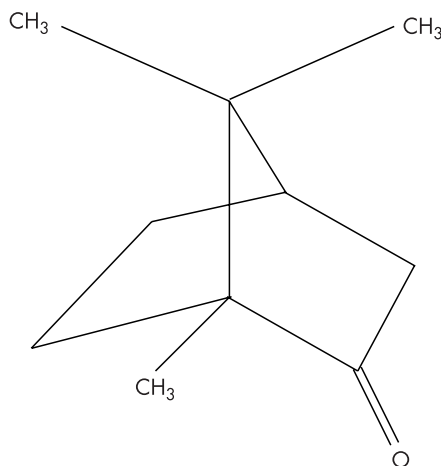


Figure 1 Molecular structure of camphor.

they lack the enzymes to hydroxylate and conjugate camphor that lead to accumulation of neurotoxic substances. Clinical toxicity generally resolves within 24 h. Seizures may persist for up to 24 h but this does not necessarily portend a poor prognosis.² Only one paediatric death has been reported in the literature since 1990.¹ Death results from respiratory depression or as a complication of status epilepticus.

There is no specific antidote to camphor poisoning, so it is treated symptomatically. Benzodiazepines are generally recommended as initial seizure treatment. As described in our case, management of seizures is often difficult and frequently requires multidrug treatments.^{2,3} From the medico-legal point of view, prescription drugs containing camphor should be labelled to indicate the toxicity of the substance. This was not the case with the present solution manufactured in the Philippines. Since 1983, medical products in the USA must contain <11% camphor.⁴ In France the regulation is less precise. Camphor is a frequently used industrial product, particularly in many cosmetics products. Drugs containing camphor are prohibited for use in children younger than 30 months of age, but few cosmetics containing camphor are on free access.

There are a number of points arising from this case that the authors feel should be considered. Firstly, the case highlights the importance of systematic questioning. Secondly, some solutions without labelling to indicate their composition but containing camphorated oil are still available. It is therefore probable that camphor intoxication is under recognised. Parents should be encouraged not to apply unlabelled home remedies to their children. Thirdly, if camphor toxicity most often results from oral ingestion, intoxication through skin contact continues to be a source of paediatric exposure, with a delay in the onset of symptoms. Camphor intoxication should be suspected in any child with seizures after application of an unlabelled solution.

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IMAGES IN EMERGENCY MEDICINE

Open dislocation of the elbow with ipsilateral fracture of the radial head and distal radius: a rare combination without vascular injury

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Open elbow dislocation is a rare injury often associated with neurovascular damage. Common fractures associated with elbow dislocation are of the medial epicondyle, lateral epicondyle, radial head and coronoid process. There is only one reported case in the literature of an elbow dislocation with fracture of the radial head and the distal radius.¹

We present an unusual case of an open posterolateral elbow dislocation associated with radial head and distal radial fracture. There was no neurovascular injury.

The most probable mechanism to produce this type of injury would be a fall on the outstretched hand. In this case the rebound forces from the ground were dorsal to the radius as the fracture was dorsally angulated. This force then

causes the radial head fracture due to the longitudinal impaction of the radius against the capitellum. This dorsal force would hyperextend the elbow causing the dislocation. This report increases the awareness of the presence of a double injury to the forearm.

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Figure 1 Radiographs showing posterolateral dislocation of the elbow with fractures of the radial head and distal radius.

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