Nigerian Journal of Paediatrics 2006, 33 (2) 60-63

Naphthalene Poisoning in Children: a Report of Two Cases

A Nte*, I Anochie, F Eke+**

Summary

Nte I, Anochie I, Eke F. Naphthalene Poisoning in Children: a Report of Two Cases. *Nigerian Journal of Paediatrics* **2006; 33: 60.** Poisoning by naphthalene which is contained in *'Camphor'*, a commonly used household insecticide in Nigeria, may occur by ingestion, by skin or eye contact, by inhalation, or by transplacental transfer. Toxic effects vary from individual to individual. The chemical is particularly dangerous in children since its absorption occurs rapidly, causing haemolysis. We report two cases of naphthalene poisoning in toddlers seen within a period of two months. The patients who accidentally ingested unquantified amounts of naphthalene ball (*Camphor)* developed haemolyic anaemia and haematuria necessitating repeated blood transfusions in one case. In addition, oliguria and impaired renal function occurred in one case. The first case improved after treatment, his renal function normalised and he was discharged home ten days after hospitalization. The second case was also stable and was discharged against medical advice on the third day by the mother. Physicians need to be reminded that this commonly used agent can be dangerous; its use should therefore be discouraged.

Key words: Naphthalene poisoning, haemolytic anaemia, haematuria, acute renal failure.

Introduction

NAPHTHALENE ($C_{10}H_8$) is a natural component of fossil fuels such as petroleum, diesel and coal.[1] It is widely used in industries and households as insecticides and lavatory deodorizers. It is found in significant proportion in *Camphor* balls. Naphthalene poisoning is common in children who suck or chew mothballs.[2-5] It may also occur by skin and/or eye contact, inhalation, skin absorption or by transplacental transfer.[6,7] Naphthalene is especially dangerous in children in whom absorption occurs rapidly. Acute intravascular haemolysis may occur within three days of exposure.[8-11] Various systems of the body may be affected by naphthalene poisoning with the affected child presenting with haematuria, oliguric acute renal failure (ARF) due to dehydration, haemoglobinuria and neurological complications due to cerebral anoxia which may occur within 3-4days.[2,3,4,8-12] To our knowledge, naphthalene poisoning occurring after the neonatal period has hitherto, not been reported in our environment. This communication therefore presents two cases of such poisoing involving two toddlers who presented within a period of two months.

University of Port Harcourt Teaching Hospital, Port Harcourt

Department of Paediatrics

* Reader

** Senior Lecturer

+ Professor

Correspondence: Dr Ifeoma Anochie.
Email: anochieify@hotmail.com

Case Reports

Case 1

IC was an 18-month-old boy who presented to our emergency ward with a history of '*Camphor*' ingestion three days before presentation. He had developed fever, restlessness and weakness one day after this, and had been vomiting intermittently for about two hours before presentation. He was said to have been an active and playful child who had accidentally ingested half a ball of *Camphor*. He was given a few spoonfuls of palm oil to drink and some drugs purchased from the chemist. The following day, he developed a high grade fever, became restless, and developed non-projectile, non-bilious, non-bloody vomiting. He had no history of previous hospitalization, and had had an uneventful neonatal period. There was no history of neonatal jaundice. The first child of a married couple, his developmental milestones were normal for the age.

On examination, he was a well nourished boy with a weight of 11kg. He was somnolent, moderately pale, anicteric and pyrexial with a temperature of 38.2°C. His chest was clinically clear, cardiovascular system

was normal, and the liver edge was palpable only 2cm below the right costal margin. He was admitted as a case of suspected naphthalene poisoning and possible malaria. He was commenced on intravenous fluid using 4.3 percent dextrose saline; in addition he was placed on an antimalarial, namely *Coartem* one tablet (20mg artemether/120mg lumefantrine) twice daily for three days. A day after admission, he developed jaundice and started passing bloody urine (Fig. 1), and dark stools. At this time, he had become severely pale, his packed cell volume (PCV) had dropped from 27 percent to 20 percent and he was in anaemic heart failure. His urinalysis showed protein 100mg/dl, blood 3+, urobilinogen 12mg/dl, pH 6.0, specific gravity 1.010. Initial serum urea and creatinine were normal. His haematology profile showed normal white blood cells and platelet counts. He had no malaria parasite in the blood film. Glucose-6-phosphate dehydrogenase (G6PD) level could not be done due to lack of facilities for assaying the level. He was transfused with 15mls/kg of packed red blood cells (PRBCs), and he also received intravenous (IV) hydrocortisone 25mg 6 hourly for 48 hours, had alkaline diuresis with iv 8.4% sodium bicarbonate 2mls/kg six hourly for 48 hours and frusemide 2 mg/kg/day.

On the third day of admission, he still continued to haemolyse, with the PCV dropping further to 17 percent, at which point he received a second blood transfusion. He later developed oliguria with elevated serum urea 7.3 mmol/L (normal 2.2-6.6 mmol/L) and creatinine 180 μmol/L (normal 60-120 μmol/L). His blood pressure however remained normal. As his urine pH was now 8.0, sodium bicarbonate infusion was discontinued. The deranged renal function was managed conservatively by fluid restriction and monitoring of fluid input and output. He made a progressive recovery within one week, his urine colour became amber and the output increased. His repeat serum urea and creatinine levels normalized to 5.0 mmol/L and 70 μmol/L respectively, and he was discharged home after ten days of hospitalization.

Case 2

DO was a 12-month-old boy who presented to the children's emergency ward with a four-day history of ingestion of *Camphor* ball, fever for three days, pallor of the hands and feet for two days, passage of dark coloured urine for two days and convulsion on the day of presentation. The seizure was his first and was generalized tonic-clonic in nature, lasted for two minutes, and aborted spontaneously. There was no history of oliguria. Pregnancy and delivery were normal. He had developed neonatal jaundice on the second day of life; this had lasted for four days. The jaundice was managed by exposure to sunlight at home. His G6PD status was unknown. He was the second child of a married couple. The sibling, a female, had no history of neonatal jaundice.

Fig 1: Serial urine samples of Case 1 showing haematuria which subsequently cleared

On presentation, he was severely pale, mildly icteric, febrile with a temperature of 39.5°C, dyspnoeic, and had altered sensorium. His Glasgow coma scale was 11/15, the pupils were reactive to light, and there was global hypotonia, and hyporeflexia. The chest was clear on auscultation. He had gallop rhythm and tender hepatomegaly of four cm below the right costal margin. He was admitted as a case of suspected naphthalene poisoning and severe malaria in anaemic heart failure. As the PCV was seven percent, he was transfused with 15mls/kg of PRBCs. Lumbar puncture done showed a clear, colourless cerebrospinal fluid (CSF), with normal analysis and biochemistry. He was commenced on iv quinine for malaria. Urinalysis showed the presence of blood, with pH 6. The child became fully conscious within eight hours of admission. His clinical condition remained stable with no further evidence of haemolysis. Thereafter, the mother refused further investigations, and discharged the child against medical advice by the third day.

Discussion

These cases illustrate some morbidities attributed to naphthalene which is contained in *Camphor*, a common household agent in Nigeria. *Camphor* is a white solid substance at room temperature but some have attractive colours. It is very volatile, and sublimates easily into a gas which has a characteristic odour and unpleasant aromatic taste.[1,2] Despite this, it is often ingested accidentally by unsupervised toddlers.[10] Naphthalene is erratically absorbed when ingested and rapidly absorbed when inhaled. The dermal absorption especially in infants may be significantly enhanced by prior application of oils.[1,2] It is metabolized in the liver to yield a variety of hydroxyl and methylthio derivatives which are excreted as glucuronide conjugates in the urine. Naphthalene dihydrodiol may be further converted in the eye to yield 1,2-naphthoquinone, a known cataractogenic agent.

Naphthalene poisoning may cause haematological, renal and neurological effects.[2,3,4,8-12] The degree of systemic damage is related to the chemical's absorption by the small intestine, and the functional state of the liver. Naphthalene itself has no haemolytic properties but its oxidative metabolite, alpha-naphthol possesses potent haemolytic activity to which patients with red cell enzyme defect primarily, glucose-6-phosphate dehydrogenase (G6PD) deficiency are more susceptible.[8,11] After ingestion, naphthalene causes abdominal cramps with nausea and vomiting as was seen in one of our patients. Acute intravascular haemolysis is the most characteristic sign of the poisoning, and this occurred in our patients. Patients may develop acute oliguric renal failure due to dehydration and haemoglobinuria.[3,4] Neurological complications of the intoxication usually occur within 3-4 days and are due to cerebral anoxia.[8,12]

The treatment of naphthalene poisoning is mainly supportive with measures to decrease absorption, and enhance excretion.[1-4,8] These measures include gastric lavage, intravenous fluids, diuretics with urine alkalinization, and blood transfusions.[1-3] If a significant amount of naphthalene is ingested and there is no convulsion, a cautious gastric lavage should be done. However, gastric lavage is not likely to be effective if more than two hours have lapsed since ingestion. Activated charcoal 1g/kg up to 50 gram should be administered following lavage. Gastric lavage was not carried out in our patients because they presented many days after naphthalene ingestion. Milk or fatty meals should be avoided for two to three hours after naphthalene ingestion because they may promote its absorption. This might partly have accounted for the deterioration of our first case who was given palm oil at home, causing prolonged haemolysis necessitating repeat blood transfusions, in contrast to the second case who did not receive palm oil. Repeat blood transfusions are required until haemoglobin concentration is 60-80 percent of normal.[1-4] The effect of corticosteroid therapy on the haemolytic process has been reported as beneficial in a few cases.[1] Sodium bicarbonate five gram orally or 1ml/kg of 8.4 percent solution intravenously should be given every 4-6 hours or as necessary to maintain alkaline urine. Fluid should be given up to 15ml/kg/hr with frusemide 1mg/kg to produce maximum diuresis and decrease injury to the kidney from haemoglobin products. Osmotic diuretic such as mannitol can be used for the same purpose. Dialysis has not been shown to be effective in removing the damaging metabolites from the blood except when required in renal failure. With regard to the use of antimalarials, both our cases did receive antimalarials because of the possibility that the infection, which is endemic, could have coexisted with the poisoning. The first case was conscious and had rapid haemolysis, he was therefore not given intravenous quinine. Unfortunately, we were unable for logistical reasons, to determine the serum level of G6PD in the two cases.

The prognosis of naphthalene poisoning depends on the degree of haemolysis and whether renal failure supervenes.[1,6-11] Although one of our patients had acute renal failure, he recovered with conservative measures. Convulsion and coma are said to be poor prognostic factors,[1,12] but this was not supported by this report.

In conclusion, naphthalene ingestion is an important cause of morbidity in children in our environment. Since naphthalene containing *Camphor* balls are widely used as household products without regulation, and

may be difficult to be keep away from toddlers in the home, we recommend removal of this product from homes where there are vulnerable groups.

Acknowledgments

The authors acknowledge all the staff of University of Port Harcourt Teaching Hospital who were involved in the management of these patients.

References

1. Fernando R,Nissanka S. Naphthalene. www: inchem.org/documents/pims/chemical/pim363.htm
2. Manchanda SS, Sood SC. Accidental poisoning in children with a case report of naphthalene poisoning. *Ind J Child Health* 1960;**9**:113-9.
3. Siegel E, Wason S. Mothball toxicity. *Pediatr Clin North Am* 1986; **33**:369-74.
4. Gidron E, Leurer J. Naphthalene poisoning. *Lancet* 1956; **270**: 228-31.
5. Bates N. Mothball poisoning. *Emerg Nurse* 2002;**10**:24-8.
6. Valaes T, Doxiadis SA, Fessas T. Acute hemolysis due to naphthalene inhalation. *J Pediatr* 1963;**63**:904-15.
7. Anziulewicz JA, Herman JD, Chiarulli EE. Transplacental naphthalene poisoning. *Am J Obst Gynecol* 1959;**78**:519-21.
8. Fradin Z, Stein GY, Varon M, Kanter P, Zeidman A. Naphthalene induced fatal haemolysis in G6PD deficiency. *Haema* 2005; **8**: 283-5.
9. Trevisan A, Rossi di Schio M, Pieno M. Haemolytic anaemia after oral self-giving of naphthalene-containing oil. *J Appl Toxicol* 2001;**21**:393-6.
10. Todisco V, Lamour J, Finberg L. Hemolysis from exposure to naphthalene mothballs. *N Eng J Med* 1991; **325**:1660-1.
11. Melzer-Lange M, Walsh- Kelly C. Naphthalene-induced hemolysis in a black female toddler deficient in glucose-6-phosphate dehydrogenase. *Pediatr Emerg Care* 1989;**5**:24-6.
12. Gupta R, Singhal PC, Muthusethupathy MA, Malik AK, Chugh KS. Cerebral oedema and renal failure following naphthalene poisoning. *J Assoc Physicians India* 1979;**27**:347-8.