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Acute naphthalene toxicity presenting with metabolic acidosis: a rare complication

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ABSTRACT

Naphthalene moth ball poisoning in children can present with diagnostic and therapeutic challenges. A 2 year old boy who had accidentally consumed unknown number of moth balls presented 3 d later with vomiting, seizures, methemoglobinemia, hemolytic anemia and altered sensorium. He was managed with red blood cell transfusion, IV Methylene blue and Sodium bicarbonate. Clinical and laboratory parameters normalized. We describe this case as ingestional naphthalene poisoning with rare manifestation of metabolic acidosis, with a good outcome after treatment.

1. Introduction

Naphthalene is commonly found in moth repellent products such as mothballs in developing countries. It is well absorbed following oral, dermal and inhalation exposure. As little as one mothball can result in toxicity in children. Deaths have been reported following its ingestion in children[1,2]. We present a case of accidental moth ball ingestion in a 2 year old child who presented with hemolysis, methemoglobinemia, seizures and rare manifestation of metabolic acidosis, and review the management.

2. Case report

A 2 year old boy presented 72 h after accidental consumption of an unknown number of naphthalene balls. On the day of consumption, he had few episodes of vomiting which subsided later on. 3 d later he was noticed to have yellowish discoloration of eyes, altered sensorium, and 3 episodes of generalized seizures. On examination he was afebrile, his heart rate was 140/min, respiratory rate 28/min, blood pressure 106/60 mmHg, Oxygen saturation (SpO_2) 75% on pulsoximeter reading. He was pale, icteric and cyanotic. He was lethargic, with a GCS of 13/15. There was no focal neurological deficit. His heart sounds were normal, lungs were clear with good air bilateral entry. The abdomen was soft and non-tender.

Urine was dark brown in color and tested positive for hemoglobin and myoglobin, suggesting hemoglobinuria and myoglobinuria. Gastric aspirate revealed dark brown colored aspirate suggestive of upper GI bleed.

His hemoglobin (Hb) was 4.9 g/dL, total WBC count 20 100 cells/mm^3, Platelet count of 401 000 cells/mm^3. Peripheral smear showed hemolysis as evidenced by the presence of n RBCs, spherocytes and fragmented RBCs. His total bilirubin was 6.9 mg/dL, with the indirect fraction measuring 6.2 mg/dL. His liver enzymes, renal functions and coagulation profile were all normal. Methemoglobin level was 7.55%. Arterial blood gas analysis (ABG) revealed metabolic acidosis; pH 7.25, pCO_2 40.1 mmHg, HCO_3 16.8 mmol/L, base

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excess 8.8 mmol/L, pO_2 19.8 mmHg, sO_2 38.4%. His G6PD levels were normal.

He was started on Oxygen, IV fluids, bicarbonate infusion and was transfused with 2 units of packed RBC (total 200 mL). IV methylene blue was given at a dose of 1.5 mg/kg. He did not have any further seizures, his sensorium improved, saturation picked upto 100% and hemoglobin had increased to 7.5 g/dL on day 2 of admission. ABG repeated was normal. Renal functions monitored were normal. The urine color became lighter and was clear by day 5 of admission. However upper GI bleed persisted. Child was kept nil per oral, and Proton pump inhibitors were continued. Gastric aspirates became clear and oral feeds were started on day 7. He stayed for 9 d in the hospital. At discharge, he was asymptomatic, with hemoglobin of 8.7 g/dL. He had a history of pica and was started on oral iron supplementation at discharge.

3. Discussion

Naphthalene is a bicyclic aromatic hydrocarbon with a molecular weight of 128 ($C_{10}H_8$). It is poorly soluble in water, and one mothball (depending on size) can contain between 0.5-5.0 g of naphthalene. The clinical consequence of ingestion may include headache, vomiting, diarrhea, abdominal pain, fever and altered mental status[3]. Oxidative stress from the hepatic metabolites causes hemolysis and methemoglobinemia. Hemolysis occurs through either hemoglobindefects (Heinz bodies formation) or cell membrane effects, particularly in patients with a low tolerance to oxidative stress (G6PD deficiency)[5,7]. Methemoglobinemia is caused by the oxidation of ferrous (Fe^{2+}) to ferric (Fe^{3+}) Hb. It renders the Hb incapable of carrying oxygen and shifts the oxyhemoglobin curve to the left[3].Clinical suspicion of methemoglobinemia should be raised when there is cyanosis that does not respond to high flow oxygen with no obvious cardiorespiratory causes, like right to left shunting. The neurologic symptoms of naphthalene ingestion include confusion, altered sensorium, listlessness, lethargy and vertigo. Muscle twitching, convulsions, decreased responses to painful stimuli, and coma occurred prior to death in individuals who ingested naphthalene[3]. The neurologic symptomatology resultsfrom cerebral edema, which is probably secondary to acute hemolysis. Rapid progression to coma and convulsions indicates poor prognosis[4,6]. Gastrointestinal bleeding is known to occur following ingestion[1,3,4]. A pubmed search did not reveal any association between acute naphthalene toxicity and metabolic acidosis. Our patient had metabolic acidosis with a pH of 7.2, HCO_3 of 16.8 mmol/L, and he improved after IV Bicarbonate.

In the absence of adequate supportive treatment, death may result from acute renal failure or kernicterus in infants[1]. Severe toxicity can occur following dermal and inhalation exposure, especially in neonates exposed to diapers and blankets that have been stored in naphthalene.

Standard treatment includes the use of methylene blue (1-2 mg/kg; 0.1 to 0.2 mL/kg of a 1% solution intravenously) and blood transfusion[1,3]. Methylene blue increases the rate of conversion of methemoglobinemia to Hb by accepting an electron (in the presence of NADPH and methemoglobinemia reductase) to form leucomethylene blue, which can then donate this electron to reduce methemoglobinemia. Hemolysis and hemoglobinuria are treated with IV hydration ensuring brisk urine output, urinary alkalinisation and packed RBC transfusion. Other than the specific therapies, management is mainly supportive. If the patient presents early, decontamination procedures like the use of activated charcoal, gastric lavage and whole bowel irrigation should be considered.

Naphthalene is widely used, and exposure can occur through inhalation, dermal absorption or ingestion. Hemolytic anemia with methemoglobinemia is the commonest presentation, and can be fatal especially in G6PD deficient patients. Paradichlorobenzene moth repellents are a safer alternative as it does not induce hemolysis and has a lower order of acute and chronic toxicity and has replaced naphthalene in developed countries. Both naphthalene and paradichlorobenzene are insecticides that kill moths and larvae when their vapors are sufficiently concentrated in a closed container. If clothes are dry cleaned (which removes eggs, larvae and the dirt and perspiration that larvae feed on) and then stored in any sealed container that can keep out the moths, they will be protected without the use of chemicals. Chemical moth repellants are thus unnecessary. Given their toxic potential and the large number of ingestions by children each year, they should be banned.

Conflict of interest statement

The authors declare that there is no conflict of interest.

References

[1] Earl Siegel, SumanWason. Mothball toxicity. *Pediatric Clin N Am* 1986; **33**: 369-374.

[2] Lim HC, Poulose V, Tan HH. Acute naphthalene poisoning following the non - accidental ingestion of mothballs. *Singapore Med J* 2009; **50**(8): 298-301.

[3] Kuffner EK. Camphor and moth repellants. In: Goldfrank LR, Flomenabum NE, Lewin NA, et al, eds. *Goldfrank's Toxicologic Emergencies*. 9th ed. New York: McGraw Hill, 1358-1363.

[4] Gidron E, Leurer J. Naphthalene poisoning. *Lancet* 1956; **1**: 228-233.

[5] Chusid E, Fried CT. Acute hemolytic anemia due to naphthalene ingestion. *Am J Dis Child* 1955; **89**: 612-614.

[6] Nash FL. Naphthalene poisoning. *BMJ* 1903; **1**: 251-259.

[7] Rieders F, Brieger H. Hemolytic action of naphthalene and its oxidation products. *Pediatrics* 1951; **7**: 725-727.