

# Severe Haemolytic Anaemia Due to Ingestion of Naphthalene (Mothball) Containing Coconut Oil

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## ABSTRACT

Naphthalene, a widely used industrial and household chemical, has rarely been an agent of poisoning worldwide. Severe haemolysis from naphthalene poisoning is rare and can be a challenge to clinicians. We report a 22-year-old female, who accidentally ingested naphthalene mixed coconut oil and got admitted with recurrent vomiting, headache and passage of dark urine. Severe intravascular haemolysis with hypotension and neutrophilic leukocytosis was detected. She was treated with red blood cell transfusions, intravenous saline infusion and ascorbic acid.

**Key words:** *Naphthalene. Poisoning. Haemolysis. Methaemoglobinaemia.*

## INTRODUCTION

Poisoning is a serious threat in Bangladesh considering it comprises around 44% of all deaths among adult female and around 8 – 10% of overall mortality in medicine wards of tertiary health care settings.<sup>1</sup> Common types of poisoning encountered in the community include pesticide poisoning, kerosene poisoning, poisoning by unknown sedative substances for stupefying purpose and occasional reports of methanol, aluminium phosphate, copper sulphate and puffer fish poisoning.<sup>2</sup>

Naphthalene poisoning has not been reported in Bangladesh probably because most cases are of mild toxicity and resolve at primary care. Naphthalene is well absorbed following oral exposure; it can also be absorbed through the skin route or following inhalation. The most characteristic sign of naphthalene toxicity is headache, vomiting, diarrhea, abdominal pain, fever, altered mental status and consequences of acute intravascular haemolysis leading to anaemia, leukocytosis, haematuria, jaundice and liver and kidney dysfunction.<sup>3,4</sup> The fatal dose of naphthalene for human is still unknown, but as little as a single mothball can cause death in children.<sup>5</sup>

We present here a 22 years old patient who developed severe haemolytic anaemia and hypotension following accidental ingestion of two naphthalene containing mothballs mixed with coconut oil.

## CASE REPORT

A 22 years Bangladeshi female student was admitted to the hospital 3 days after accidental ingestion of about 2 powdered naphthalene mothballs mixed in about 5 ml of coconut oil kept for household use. She experienced profuse vomiting half an hour after ingestion with upper abdominal ache. She developed headache with fever after 6 hours and noticed dark coloured urine after 12 hours of ingestion. She was treated in primary health care settings with intravenous infusion of 5% dextrose, anti-emetic, proton pump inhibitor and intravenous hydrocortisone without improvement before referral. She did not have any past medical history or family history of note.

On admission, she was in a good general condition, fully conscious but complained of weakness, lower abdominal discomfort and continued passage of dark urine. There was marked pallor and mild jaundice but no cyanosis. Her blood pressure was 80/50 mmHg, the pulse rate was 100 beats/minute, temperature was 37°C and respiratory rate was 20 breaths/minute. The abdomen was mildly tender in the suprapubic region, but soft and not guarded. No organomegaly was noted. The neurological and other systems revealed no abnormality. Urinary catheterization collected dark-brown urine, which tested positive for blood on dipstick. She did not bring any remaining mothball for examination.

Initial investigations revealed haemoglobin (Hb) 3 g/dl, MCV 75 fl; white blood cell count (WBC) 23 x 10<sup>9</sup>/l with neutrophilia, platelet count was 215 x 10<sup>9</sup>/l and ESR 105 mm in 1<sup>st</sup> hour. Peripheral blood film showed dimorphic picture with anisopoikilocytosis, nucleated RBC, neutrophilic leukocytosis and normal platelet distribution. Serum total bilirubin was 3.05 mg/dl and urine showed no RBC. On the basis of history and clinical findings our provisional diagnosis was intravascular haemolysis and haemoglobinuria secondary to naphthalene toxicity.

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Received September 21, 2010; accepted April 23, 2012.

Further investigations proved haemolysis by a raised reticulocyte count of 6% and reduced haptoglobin level of 0.3 gm/l. Haemoglobinuria was confirmed with positive urine for haemoglobin. Serum LDH (5442 U/l) and CPK (634 U/l) was raised. All liver enzymes, prothrombin time, serum creatinine, blood urea and electrolytes were within normal limits. Methaemoglobin level could not be measured. G6PD assay and haemoglobin electrophoresis were normal.

She was treated with oxygen inhalation via face mask, rapid infusion with normal saline, three units of packed RBC and oral ascorbic acid. She came too late for gastric washout and activated charcoal administration. Her urine gradually became clear by the second day and apart from a blood pressure of 80/60 mm of Hg, she was sound clinically. On the second, third day and fifth day of admission her Hb improved to 7.4 g/l, 9.5 g/l and 10.0 g/l respectively and ESR gradually came down to 22 from 105 (Table I). Total WBC count was  $23 \times 10^9/l$  on 1<sup>st</sup> day which came down to  $3.5 \times 10^9$  on the fifth day. Serum bilirubin level also came down to 2.05 mg/dl from 3.0 mg/dl on day 5. So alkalinisation of urine, intravenous methylene blue or any renal replacement therapy was not needed. She stayed at hospital for 5 days without any renal or neurological sequelae.

**Table I:** Status of biochemical parameters of the patients during hospital stay.

Laboratory parameters	Hospital stays in days			
	1 <sup>st</sup>	2 <sup>nd</sup>	3 <sup>rd</sup>	5 <sup>th</sup>
Hb% (gm/dl)	3.00	7.4	9.5	10.0
S. Bilirubin (mg/dl)	3.05	3.0	2.5	2.0
ESR (mm after 1 <sup>st</sup> hour)	105	70	35	22
WBC (per liter)	$23 \times 10^9$	$17 \times 10^9$	$12 \times 10^9$	$3.5 \times 10^9$

## DISCUSSION

This is the first reported case of haemolytic anaemia occurring after accidental ingestion of coconut oil containing naphthalene from Bangladesh. Naphthalene is a bicyclic aromatic hydrocarbon with a molecular weight of 128 (C<sub>10</sub>H<sub>8</sub>).<sup>3,4</sup> It is poorly soluble in water, and one mothball (depending on size) can contain between 0.5 – 5 g of naphthalene.<sup>4</sup> After exposure, naphthalene is readily absorbed in systemic circulation, initially metabolized into a number of reactive epoxide and quinone metabolites by cytochrome P450 oxidation and then excreted in the urine as mercapturic acids, methylthio-derivatives and glucuronide conjugates.<sup>4</sup> Following liver metabolism, naphthol-alpha, the most potent derivative of naphthalene, causes haemolysis with severe anaemia and Heinz bodies formation.<sup>3</sup> There is often a concurrent leucocytosis and the degree of haematological involvement is more severe in patients with G6PD deficiency.<sup>4</sup>

The clinical consequences of naphthalene ingestion may include headache, vomiting, diarrhea, abdominal

pain, fever and altered mental status.<sup>4</sup> This patient showed the typical clinical and haematological features found in naphthalene intoxication. After ingestion, vomiting, diarrhea and fever appeared on day 1, followed by an acute haemolytic crisis from day 2 onwards evidenced by pallor, mild jaundice and pigmented urine with blood indices showing severe intravascular haemolysis and neutrophilic leucocytosis. After 5 – 6 days, the haemolytic process ended and then recovery was rapid. This trend was found in other reported cases too where the haemolysis process persisted around a week followed by rapid recovery.<sup>3,5</sup>

The treatment of naphthalene poisoning is mainly supportive, including gastric lavage, intravenous fluids, and diuretics with urine alkalinization, blood transfusions, and methylene blue as the antidote to methaemoglobinaemia. The effect of steroid therapy is still unclear. We found rapid improvement with conservative management only, most likely because there was no G6PD deficiency (but may be false negative as in this case) or renal toxicity and the haemolysis process were almost over at the delayed admission. Naphthalene induces toxic manifestations by enhanced production of free oxygen radicals, resulting in lipid per oxidation and deoxyribonucleic acid damage.<sup>3</sup> Ascorbic acid was given to counteract this effect. Elimination of toxin by any enhanced techniques like continuous renal replacement therapy (CRRT) could be considered, but is still inconclusive.

Safety issue is very important in this regard. In Bangladesh naphthalene is specially used as a cockroach or ant repellent. Proper packaging and regulation act is necessary to control its use. Some of the states in United States of America and Australia have this kind of regulation.<sup>6</sup> The health policy makers of our country must think about it.

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