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Case Report

Anaesthesia Section

Naphthalene Poisoning following Ingestion of Mothballs: A Case Report

TANVEER SINGH KUNDRA[1], VIKAS BHUTANI[2], RIMPLE GUPTA[3], PARMINDER KAUR[4]

ABSTRACT

Naphthalene is a widely used industrial and household chemical in the form of mothballs. But it has rarely been an agent of poisoning worldwide. We describe a case of ingestional naphthalene poisoning with a good outcome after proper management. A 29-year-old girl ingested 8 mothballs, and presented two days later with haemolysis and methaemoglobinaemia. She was given intravenous methylene blue, N-acetylcysteine and ascorbic acid, besides supportive treatment. Renal replacement therapy in the form of SLED of 8 hours was done on a daily basis. She was discharged after ten days on twice a week outpatient follow-up haemodialysis.

Keywords: Ascorbic acid, Hemolysis, Methemoglobinemia, Methylene blue, N-acetylcysteine, Renal replacement therapy

CASE REPORT

A 29-year-old female patient presented 72 hours after oral ingestion of 8 naphthalene balls with suicidal intent. She did not have any past medical history. She complained of vomiting and decreased urine output a few hours after the ingestion and was taken to a local hospital for treatment. On the third day, she was referred to our hospital for further management.

On presentation, patient was drowsy. Clinically she was afebrile, had a pulse rate of 117/minute, BP 110/70 mm Hg, respiratory rate 30/minute, spo_2 75% on oxygen at 6 l/min via simple face mask. She was pale and jaundiced. Heart sounds were normal. The lungs were filled with crepts. The abdomen was soft with no guarding. No organomegaly was noted. On neurological examination, there was no focal neurological deficit. Pupils were bilaterally normal size reacting to light.

Foley's catheterization was done and urine was black-coloured. ECG was suggestive of sinus tachycardia. ABG was done which was suggestive of severe metabolic acidosis with a pH of 7.059, HCO3 of 2.9 and a BE of -25.1mmol/L with a methaemoglobin of 11.1%.

She was admitted to the Intensive Care Unit (ICU) for further management. Subsequently, the patient was intubated and put on full ventilatory support. Endotracheal tube got filled with pink frothy sputum soon after intubation which was suggestive of pulmonary oedema. Gastric lavage was not done with activated charcoal in view of late presentation.

Initial investigations revealed severe anaemia with haemoglobin of 3.3g/dL and haematocrit of 9.1%. There was leukocytosis with marked neutrophilia (Total Leukocyte Count of 86,000/µL with 80% neutrophils), platelet count =5,92,000 and deranged coagulation profile with INR =3.28. Intravascular haemolysis was suggested by clinical jaundice and total bilirubin measuring 5.30mg/dL with indirect hyperbilirubinaemia and urine bilirubin positive. Liver function tests were deranged with elevated liver enzymes (SGOT of 420). Renal functions were deranged with BUN of 70mg/dL and serum creatinine of 4.2mg/dL. Post-intubation Blood Gas Analysis showed severe metabolic acidosis with a pH of 6.917, HCO_3 of 7.7, BE of -24.7mmol/L, pO_2 of 114.8 mm Hg, lactates of 9.7 with methaemoglobin of 11.1%. Haemodialysis was done in view of severe metabolic acidosis and acute renal failure. Subsequently, after haemodialysis her acidosis improved with post-dialysis ABG suggestive of pH of 7.440, pO_2 of 63.3 mm Hg, HCO_3 of 23.3, BE of -0.5mmol/L.

Her methaemoglobin levels were falling with first day reading of 11.1%, which decreased to 2.2% on second day. Her haemoglobin increased to 12.5g/dL after 5 units packed red cell transfusion. I.v. methylene blue 75mg (1.5mg/kg) was prescribed on Day 2 of admission after checking for G6PD status. I.v. ascorbic acid 300mg and N-Acetylcysteine (NAC) 1.2 gm daily was started. Ionotropic support in the form of norepinephrine infusion had to be started to maintain a MAP >65 mm Hg. On Day 3 of admission, the haemoglobin was 10.8g/dL, and there was some improvement of methaemoglobin which decreased to 1.7%. Her urine output was nil and renal replacement therapy (RRT) in the form of haemodialysis was started on daily basis. Day 1 onwards, her haemoglobin was maintained without any transfusions.

From Day 4, she started improving. Her haemoglobin was 9.9g/dL. Her total bilirubin dropped to 1.47mg/dL with SGOT showing a downward trend as well. Her renal functions were improving, although patient remained on frequent dialysis. Spo_2 was consistently >95% and patient was weaned gradually (her oxygen requirement decreased and ionotropic support was tapered off). On Day 5, she was extubated. Her post-extubation ABG was normal. Still her urine output was about 5-10ml on Day 6 and she was continued on daily haemodialysis. Her CT kidneys did not show any evidence of acute cortical necrosis. Her haemoglobin improved to 10.0g/dL. BUN decreased to 23mg/dL with serum creatinine of 2.5mg/dL. Her LFTs returned to normal. Progression of the biochemical parameters of the patient is shown in [Table/Fig-1].

Subsequently, she was discharged on day 10 and referred to dialysis clinic.

Day of admission	Day 1	Day 2	Day 3	Day 4	Day 5	Day 9
Haemoglobin (g/dL)	3.1	12.5	10.8	9.9	9.4	8.3
Methaemoglobin (%)	11.1	2.2	1.7	1.3	-	0.9
BUN (mg/dL)	70	12	10	12	40	12
S. Creatinine (mg/dL)	4.2	1.4	1.4	1.6	3.7	2.4
Urine colour	Black	Black	NA	NA	Clear	Clear

[Table/Fig-1]: Progression of biochemical parameters while the patient was admitted

DISCUSSION

Naphthalene mothballs are commonly used in households. It has rarely been an agent of poisoning worldwide [1]. Severe haemolysis from naphthalene poisoning is rare and can be a challenge to clinicians.

Naphthalene is a bicyclic aromatic hydrocarbon with a molecular weight of 128 ($C_{10}H_8$) [2]. The clinical features of naphthalene ingestion are mentioned in [Table/Fig-2] [3]. Studies have demonstrated that toxic manifestations of naphthalene may be due

Gastrointestinal Effects
Nausea, vomiting, abdominal pain, diarrhoea
Renal Effects
Increased creatinine level, increased serum urea nitrogen level, hematuria, renal tubular acidosis
Respiratory Effects
Congestion, Acute Respiratory Distress Syndrome (noted at 2ppm)
Neurologic Effects
Confusion, lethargy, vertigo, fasciculations, convulsions, anesthesia, cerebral oedema, coma (coma is noted at 0.05mg/kg body weight per day)
Hepatic Effects
Jaundice, hepatomegaly, elevated liver enzyme levels (noted at 0.02mg/kg per day)
Ocular Effects
Ocular nerve atrophy, bilateral cataracts with chronic exposure

[Table/Fig-2]: Systemic effects of naphthalene exposure [3]

to enhanced production of free oxygen radicals, resulting in lipid peroxidation and deoxyribonucleic acid damage [4]. Ascorbic acid acts as a free radical scavenger and hence may be useful in this situation [5].

Haemolysis occurs particularly in patients with G6PD deficiency, who have a low tolerance to oxidative stress. Renal failure as a complication of naphthalene-induced haemolysis and haemoglobinuria has been reported [6].

Methaemoglobinaemia commonly occurs in naphthalene poisoning. Methaemoglobin is an abnormal haemoglobin in which the iron moiety of unoxygenated haemoglobin is in the ferric (Fe^{+3}) state rather than the ferrous state (Fe^{+2}). Thus, methaemoglobin is the oxidized form of haemoglobin, which does not bind oxygen and increases the affinity of oxygen for the partially oxidized portion of haemoglobin [7]. Pulse oximetry is unreliable in the setting of methaemoglobinaemia. A high concentration of methaemoglobin causes the saturation to approximate 85%. When the patient is hypoxic (saturation 40-50%), the methaemoglobin artifactually increases the pulse oximeter reading to 85%. Conversely, if the oxygen saturation is 100%, the methaemoglobin spuriously decreases the pulse oximeter reading to around 85% [8]. Co-oximetry is the gold standard in these patients [7]. When the concentration of methaemoglobin in the blood is above 1.5%, the patient develops cyanosis [7].

Treatment [7]

A) **Specific Treatment -** Specific treatment includes the use of methylene blue and exchange transfusion. Methylene blue increases the rate of conversion of methaemoglobin to haemoglobin by accepting an electron (in the presence of nicotinamide adenine dinucleotide phosphate [NADPH] and methaemoglobin reductase), to form leucomethylene blue, which can then donate this electron to reduce methaemoglobin [9]. Exchange transfusion is the treatment of choice in patients with G6PD deficiency as methylene blue itself may induce haemolysis and cause paradoxical methaemoglobinaemia in these patients [10,11]. NAC may be used in the treatment of methaemoglobinaemia as a reducing agent especially in patients with G6PD deficiency [11].

B) **Supportive Treatment –** Supportive treatment to maintain the airway, breathing and circulation (which may include endotracheal intubation, mechanical ventilation and use of inotropes).

CONCLUSION

In summary, naphthalene mothball ingestion can present with prolonged haemolytic anaemia and methaemoglobinaemia. Naphthalene poisoning is uncommon but can prove fatal, especially in patients who are G6PD deficient. But if managed properly, the patient can have a good outcome.

REFERENCES

[1] Rahman MM, Chowdhury FR, et al. Severe haemolytic anaemia due to ingestion of naphthalene (mothball) containing coconut oil. *Journal of the College of Physicians and Surgeons—Pakistan*. 2012;22(11):740-41.
[2] Kuffner EK. Camphor and moth repellants. In: Goldfrank LR, Flomenbaum NE, Lewin NA, et al, eds. Goldfrank's Toxicologic Emergencies. 7th ed. New York: McGraw-Hill, 2002: 1295-302.
[3] From US Environmental Protection Agency. Health and environmental effects profile, naphthalene EPA/600/X-86/241. Cincinnati (OH): Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development; 1988 and from EPA health effects notebook for hazardous air pollutants- draft, EPA-452/D-95-00, PB95-503579, December 1994. Available at http://www.epa.gov/ttn/atw/hapindex.html.
[4] Bagchi M, Bagchi D, Balmoori J, Ye X, Stohs SJ. Naphthalene induced oxidative stress and DNA damage in cultured macrophage J744A.1 cells. *Free Radic Biol Med*. 1998;25:137-43.
[5] Niki E. Action of ascorbic acid as a scavenger of active and stable oxygen radicals. *Am J Clin Nutr*. 1991;54:1119-24S.
[6] Chugh KS, Singhal PC, Sharma BK, et al. Acute renal failure due to intravascular haemolysis in the North Indian patients. *Am J Med Sci.* 1977;274:139-46.
[7] Nascimento TS, Pereira ROL, Mello HLD, Costa J. Methaemoglobinaemia: from diagnosis to treatment. *Rev Bras Anestesiol*. 2008;58:651-64.
[8] Kamat V. Pulse Oximetry. *Indian Journal of Anaesthesia*. 2002;46:261-68.
[9] Howland MA. Methylene blue. In: Goldfrank LR, Flomenbaum NE, Lewin NA, et al, eds. Goldfrank's Toxicologic Emergencies. 7th ed. New York: McGraw-Hill, 2002: 1450-51.
[10] Bradberry SM, Vale JA. Naphthalene and Paradichlorobenzene. In: Bateman N, Jefferson R, Thomas S, Thompson J, Vale A. Oxford Desk Reference: Toxicology. 1st ed. 2014: 254.
[11] Wright RO, Lewander WJ, Woolf AD. Methaemoglobinaemia: etiology, pharmacology and clinical management. *Ann Emerg Med*. 1999;34:646-56.

PARTICULARS OF CONTRIBUTORS:
1. Specialist Anaesthetist, Freelancer, Mohali, Punjab, India.
2. Additional Director, Department of Medicine, Fortis Hospital, Mohali, Punjab, India.
3. Consultant, Department of Anaesthesia, Mayo Hospital, Mohali, Punjab, India.
4. Specialist Anaesthetist, Freelancer, Mohali, Punjab, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Tanveer Singh Kundra,
Kothi No.184, Phase 4, Mohali, Punjab- 160055, India.
E-mail: tvskundra@yahoo.co.in

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