

Acute Renal Failure Following Naphthalene Poisoning

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Many cases of naphthalene and naphthalene poisoning are reported in literature since the introduction of naphthalene in 1841 by Rossbach as an internal antiseptic in typhoid fever and an anthelmintic in 1842¹. Today, naphthalene poisoning occurs in suicidal attempts¹⁻³, or accidental ingestion by children^{2,4,5}. Naphthalene's relative insolubility in water and its poor absorption from the gut underestimates its toxicity⁶. The aim of this case report is to highlight the toxicity of this common household substance.

Case Report

A 22 years old male was hospitalized with acute abdominal pain, vomiting, diarrhoea, high grade fever and mental confusion. These symptoms developed half-an-hour after ingesting six naphthalene balls (8.6 grains), two days prior to admission, under a state of depression. On examination, the patient was a young man of average height and built in a state of confusion. His pulse was 130/min regular, of moderate volume, blood pressure 140/90 mm Hg and temperature 103°F. He was anaemic, jaundiced and dehydrated. Liver was palpable 2 cm below the costal margin and tender. Chest auscultation revealed bilateral basal crepitations. Occular fundi were normal. Patient had noticed scanty black coloured urine 24 hours after taking naphthalene balls. Neck rigidity was absent. On the day of admission his hemoglobin was 5 gm/dl and total leukocyte count 19000/mm³ with polymorphs 75%, lymphocytes 23% and monocytes 2%. Reticulocyte count was 8%. The peripheral blood smear showed microcytosis, anisocytosis, polychromatophilia and nucleated red cells. ESR was 33 mm for 1st hour, westergen. Bone marrow revealed erythroid hyperplasia. No malarial parasite was seen in blood smear or bone marrow. Urine contained albumin, free haemoglobin and red blood cells. Blood urea was 184 mg/dl and serum creatinine 7.4 mg/dl. Electrolytes, calcium, phosphate, serum proteins, prothrombin time, fasting blood sugar and ECG were within normal range. Liver function tests showed serum bilirubin 12 mg/dl, ALT 40 IU/L (normal up to 35) and alkaline phosphatase 32 IU/L (normal 39-117). X-ray chest revealed haziness in both lower zones. Blood and urine cultures were negative. Glucose-6-phosphate dehydrogenase (G6PD) level was normal.

The patient was managed conservatively and was transfused 3 pints of blood. On the day of admission, he passed 100 ml black coloured urine followed by anuria for 3 days. The urine output then increased steadily and on the 16th day it was 5000 ml in 24 hours. The blood urea was raised to 264 mg/dl and creatinine to 11.2 mg/dl on the 4th day. The levels remained very high for 18 days and then declined. On the 25th day, blood urea was 110 mg/dl and creatinine 5.6 mg/dl. On the 30th day blood urea, serum creatinine and creatinine clearance were within the normal range.

Discussion

Naphthalene balls are composed of hydrocarbon naphthalene which is a silvery white crystalline compound obtained from coal tar. It is used as a deodorant in lavatories, as a moth repellent and in insecticides, flea powders and vermifuges^{4,7,8}. Naphthalene is metabolized into alpha and beta naphthol. Alpha naphthol acts directly upon the erythrocytes and produces acute hemolysis⁹, especially in patients with G6PD deficiency². Our patient despite having normal levels of G6PD had severe

hemolysis. Other workers found normal levels of G6PD in 9 out of 21 children who developed acute hemolysis after naphthalene poisoning³. Naphthalene passes in urine in the form of glucuronate and ethyl sulphate. Its lethal dose in children is 2 gms and in adults 5-15 gms⁶. Manifestations of naphthalene poisoning are variable depending upon age, dose, route of administration and character of toxic response. It is toxic by inhalation of vapours, ingestion and skin contact⁶. Naphthalene ingestion with a suicidal purpose is rare. This case presents many typical features of naphthalene poisoning. During first 24 hours of ingestion nausea and vomiting of varying intensity are noted. On the second day, fever appears which lasts for several days. Pallor, prostration, haemolysis, jaundice and haemoglobinuria appear on third day. The urine assumes a characteristic portwine colour. Spleen may be enlarged and renal failure may ensue. Albuminuria, haematuria and granular casts are seen in the urine. Naphthol may be demonstrated in urine at this time. After 5-6 days haemolysis stops but there is still leukocytosis, oliguria and anuria due to lower nephron nephrosis. Blockade of renal tubule with haemoglobin may supervene or convalescence may begin which in the absence of complications is rapid. Cerebral oedema, confusion, convulsions, coma and death may occur^{1,2}. Laboratory findings are often comparable with acute hemolytic anaemia. Heinz bodies appear before haemolytic process becomes evident and hence has a prognostic value for development of haemolysis¹⁰. There is no specific treatment for naphthalene poisoning. The management is symptomatic with packed red cell transfusions and monitoring of fluid and electrolyte balance. Induced vomiting may help removing moth balls from the stomach¹⁰. Alkalies have been recommended in the presence of haemoglobinuria to prevent its deposition in renal tubules¹¹. Administration of milk, fats and oils should be avoided as they may enhance absorption of naphthalene¹².

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