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NAPHTHALENE TOXICITY PRESENTED WITH HEMOLYSIS AND METHEMOGLOBINEMIA - A CASE REPORT

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Abstract : Naphthalene is a widely used in industries and houses as a chemical in the form of mothballs. But it has been used rarely as an agent of poisoning worldwide. We describe a case of ingestional naphthalene poisoning with a good outcome after appropriate management. A 23-year-old girl ingested 10 mothballs, and presented three days later with haemolysis and methaemoglobinaemia. She was given intravenous methylene blue, N-acetylcysteine and ascorbic acid, as additional medication to routine supportive treatment. Renal replacement therapy in the form of Sustained low-efficiency dialysis [SLED] of around 7-8 hours was done on a daily basis. She was discharged after ten days on twice a week outpatient follow-up haemodialysis.

Index Terms Ascorbic acid, Hemolysis, Methemoglobinemia, Methylene blue, N-acetylcysteine, Renal replacement therapy, Sustained low-efficiency dialysis

CASE REPORT

A 23-year-old female patient presented 72 hours after oral ingestion of 10 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 clinic for treatment. On the third day, she was referred to our hospital for the further management.

On presentation, patient was little drowsy. Clinically she was afebrile, had a pulse rate of 112/minute, BP 108/70 mm Hg, respiratory rate 28/minute, spo2 74% on oxygen at 6 l/min via simple face mask. She was looking pale and jaundiced. Heart sounds were normal. The lungs were filled with crepts bilaterally. The abdomen was soft with no guarding present. No organomegaly was noted. On neurological examination, there was absence of focal neurological deficit. Pupils were bilaterally normal in size, reacting to light.

Foley's catheterization was done and urine was black in colour. ECG showed sinus tachycardia. ABG was was suggestive of severe metabolic acidosis with a pH of 7.055, HCO3 of 2.8 and a BE of -24.1mmol/L with a methaemoglobin of 11.2%.

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

Initial investigations revealed severe anaemia with haemoglobin of 3.4g/dL and haematocrit of 9.2%. There was leukocytosis with marked neutrophilia (Total Leukocyte Count of 87,000/µL with 78% neutrophils), platelet count =5,88,000 and deranged coagulation profile with INR =3.30. Intravascular haemolysis was suggested by clinical jaundice and total bilirubin measuring 5.40mg/dL with indirect hyperbilirubinaemia and urine bilirubin positive. Renal functions were deranged with BUN of 68mg/dL and serum creatinine of 4.1mg/dL. Liver function tests were deranged with elevated liver enzymes (SGOT of 450). Post-intubation Blood Gas Analysis showed severe metabolic acidosis with a pH of 6.916, HCO3 of 7.8, BE of -24.6mmol/L, pO2 of 110.8 mm Hg, lactates of 9.8 with methaemoglobin of 11.2%. 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 7.450, pO2 of 64.3 mm Hg, HCO3 of 23.2.

Her methaemoglobin levels started falling with first day reading of 11.2%, which decreased to 2.3% on second day. Her haemoglobin increased to 12.5g/dL after 5 units packed red blood cell transfusion. I.v. methylene blue 75mg (1.5mg/kg) was prescribed on the 2nd day 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 was started to maintain a MAP >65 mm Hg. On the 3rd of admission, the haemoglobin was 10.8g/dL, and there was some improvement of methaemoglobin which decreased to 1.8%. Her urine output was absent and renal replacement therapy (RRT) in the form of haemodialysis was started on daily

basis. After packed Rbc transfusion, from 2nd day onwards her haemoglobin was maintained without requirement of any further transfusions.

From 4th day onwards, she started improving. Her haemoglobin was 9.9g/dL. Her total bilirubin dropped to 1.46mg/dL with SGOT started showing a downward trend. Her renal functions also started improving, although patient remained on frequent dialysis. Spo2 was consistently >95% and patient was weaned gradually (her oxygen requirement decreased and ionotropic support was tapered off). On 5th day, she was extubated. Her ABG after post-extubation was normal. Still her urine output was about 5-10ml on the 6th day and she was continued on daily haemodialysis. Her CT kidneys did not show any evidence of acute cortical necrosis. Her haemoglobin improved to 10.1g/dL. BUN decreased to 22mg/dL with serum creatinine of 2.4mg/dL. Her LFTs returned to normal. Progression of the biochemical parameters of the patient is shown in [Table-1].

Subsequently, she was discharged on the 10th day and was referred to dialysis center

DISCUSSION

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

Naphthalene is a bicyclic aromatic hydrocarbon with a molecular weight of 128 (C10H8) [2]. The clinical features of naphthalene ingestion are mentioned in [Table -2]. Several Studies had demonstrated that toxic manifestations of naphthalene might be due to enhanced production of free oxygen radicals, resulting in lipid peroxidation and deoxyribonucleic acid damage[3]. Ascorbic acid acts as a free radical scavenger and hence may be useful in naphthalene poisoning [4]. Haemolysis occurs particularly in patients with G6PD deficiency, who had a low tolerance to oxidative stress. Renal failure as a complication of naphthalene-induced haemolysis and haemoglobinuria has been reported in some cases[5]. 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. Pulse oximetry also becomes unreliable in the setting of methaemoglobinaemia. A high concentration of methaemoglobin causes the saturation to show a approximate value of 85%. When the patient is hypoxic (saturation 40-50%), the methaemoglobin artifactually increases the pulse oximeter reading to around 85%. Conversely, if the oxygen saturation is 100%, the methaemoglobin spuriously decreases the pulse

oximeter reading to around 80% . In these patients Co-oximetry is the gold standard . Patients develop cyanosis, When the concentration of methaemoglobin in the blood is above 1.5%.

Treatment

A) Definitive Treatment - Definitive 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 . In patients with G6PD deficiency exchange transfusion is the treatment of choice as methylene blue itself may induce haemolysis and cause paradoxical methaemoglobinaemia in these patients[67]. NAC may be also be used in patients with G6PD deficiency for the treatment of methaemoglobinaemia as a reducing agent especially[7].

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

CONCLUSION

Naphthalene mothball ingestion can present with prolonged haemolytic anaemia and methaemoglobinaemia. Naphthalene poisoning is rare but can prove fatal, particularly in patients who are G6PD deficient. But if managed correctly, the patient prognosis can be improved.

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