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Interesting Cases

Acquired Methemoglobinaemia Secondary to Nitrobenzene Poisoning: A Case Report

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A B S T R A C T

Methemoglobinaemia occurs as a result of the oxidation of iron in haemoglobin from the ferrous state to the ferric state. Acquired methemoglobinaemia is one of the serious side effects occurring as a result of exposure to nitrate-containing compounds. Treatment of these patients usually involves the administration of methylene blue. Here, we have reported a 14-year-old adolescent with nitrobenzene poisoning.

Keywords: Poisoning, Methemoglobinaemia, Nitrobenzene, Methylene Blue

Introduction

Adolescent poisoning is a common problem encountered in a paediatric emergency. Nitrobenzene is a potentially fatal compound used as a paint solvent and dye, and in rubber synthesis. Consumption of nitrobenzene leads to methemoglobinaemia. Early diagnosis of methemoglobinaemia and administration of methylene blue prevents mortality in these cases. We report a 14-year-old adolescent child with nitrobenzene poisoning.

Case Report

A 14-year-old child came to our paediatric emergency room with a history of an alleged suicidal attempt by consumption of 20% nitrobenzene (Figure 1) 9 hours prior to presentation. She had presented with pain in abdomen and vomiting since then. There was no history of headache, chest pain, hurried breathing, drowsiness or convulsions.

On examination, she was conscious with a GCS of 14/15 with an oxygen saturation of 60% in room air which improved to 68% with 5 L of oxygen. She had an odour of bitter almond with peripheral cyanosis. Except for mild epigastric tenderness, rest of the systemic examination was fairly normal.

A stomach wash was given. Blood was drawn, which appeared dark in colour (Figure 2) and was sent for investigations including blood gas analysis and serum methemoglobin levels. In view of hypoxia, a chest X-ray was done which was normal. Blood investigations showed raised bilirubin levels (max - 2 mg/dl), a slight drop in haemoglobin and serum methemoglobin levels of 6.2% (Figure 3). Rest of the investigations were normal. Injection methylene blue 1 mg/kg was given intravenously over 20 mins. This showed a dramatic improvement in her saturation to 86-88% within an hour. Two repeat doses of methylene blue were given 8 hours apart. She also received 500 mg/day of oral Vitamin C for 1 week. The following day her saturation improved to 92-94% at room air. Over the course of the

next few days, she showed good clinical improvement. On HEADSS screening, she was found to have a history of excessive usage of electronic gadgets (cell phones) and episodes of emotional outbursts and impulsive behaviour. Psychiatric assessment and counselling were done and she was discharged within a week.

Figure 1.20% Nitrobenzene Solution

Figure 2.Dark Brown Coloured Blood

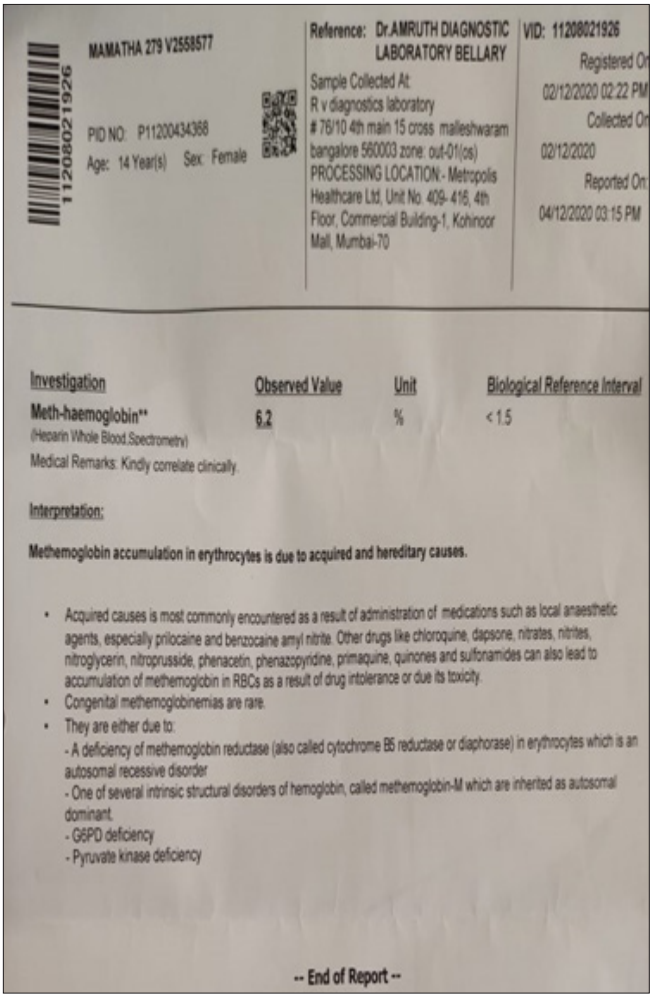

Figure 3.Serum Methemoglobin Levels

Discussion

Nitrobenzene is an important component in the synthesis of various solvents and flowering agents. It has a bitter almond odour and is yellowish in colour with an oily texture. The first case reporting poisoning due to nitrobenzene was seen in 1886.[1] Lethal dose of nitrobenzene is around 1 gm to 10 gm.[2]

It may be absorbed through the skin, respiratory tract, and gastrointestinal tract, and its highest concentration accumulates in the liver, brain, blood, and stomach due to its lipophilic nature.[2]

The ingestion of nitrobenzene results in a condition called methemoglobinaemia. This condition causes the oxidation of iron present in the blood from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state, which further leads to the blood turning brown, and the loss of the ability to transport oxygen.[3] Physiologically, a low level (< 1%) of haemoglobin is oxidised to methemoglobin. This low level is maintained due to the presence of two reductive pathways in the red blood cells, namely diaphorase pathways and HMP

pathway. In nitrobenzene poisoning, due to oxidative stress, enhanced quantities of methemoglobin are produced.[2] There are various mechanisms helpful in the reduction of methemoglobin, for example, catalysis by cytochrome b5 reductase and alternate pathway using NADPH-dependent methemoglobin reductase system.[4]

In case of less than 10%-15% of methemoglobin, only cyanosis seems plausible (as in our case), however, the child will be usually asymptomatic. Levels above 20% cause tachypnoea, headache, chest pain, and dyspnoea. 40-50% of methemoglobin causes lethargy and confusion. It might also cause metabolic acidosis further resulting in seizures, coma, bradycardia, hypertension, and ventricular dysrhythmia. Methemoglobin levels above 70% are lethal. Some other effects are haemolytic anaemia, altered liver functions, contact dermatitis, and hepatosplenomegaly. Severe symptoms are generally found in children with anaemia and G6PD enzyme deficiency.[5]

Methylene blue is an effective antidote for methemoglobinaemia. The advised dosage is 1-2 mg/kg (with a maximum of 50 mg dose in adults) given intravenously as a 1% solution over 5 minutes. If required, it can be repeated in one hour. At levels above 7 mg/kg, methylene blue acts as an oxidant and therefore may cause methemoglobinaemia in some individuals. For patients with methemoglobin levels of more than 30%, ascorbic acid (200-500mg/day) may be advised. The use of N-acetylcysteine in the treatment of methemoglobinaemia has been reported. Patients showing severe symptoms may be advised exchange transfusion. Cases with methemoglobin levels > 50% or those who are not responding to standard treatment are provided hyperbaric oxygen.[5]

Conclusion

Nitrobenzene poisoning is rarely encountered in clinical practice. Hence high index of suspicion in cases of unknown compound consumption and the above-mentioned features should prompt us to think of acquired methemoglobinaemia as administration of methylene blue can be life saving in these cases.

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