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RESEARCH ARTICLE

INTRAVENOUS METHYLENE BLUE- LIFE SAVING IN METHEMOGLOBINEMIA DUE TO NITROBENZENE POISONING

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ABSTRACT

Nitrobenzene is a nitrite compound often used in polishes or solvents. Its toxic effects are due to its ability to induce methemoglobinaemia. The clinical presentation of this poisoning varies according to the concentration of methemoglobin level in blood. The importance of early identification of the compound on the basis of clinical suspicion corroborative with methemoglobin levels with timely intervention is required to prevent fatal outcome (Agrawal *et al*., 2011). Our case presented with history of consumption of unknown compound followed by vomiting,mild cyanosis and altered sensorium. On investigation, ABG was suggestive of decreased SpO_2, methemoglobinemia and severe metabolic acidosis. The urgent institution of methylene blue as the specific antidote saved the patient's life.

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INTRODUCTION

Acute poisoning with nitrobenzene causing significant methaemoglobinaemia is a life threatening emergency. Early aggressive management of severe poisoning, strongly suspected on clinical grounds may change the outcome of the patient (Saxena, 2010).

CLINICAL FEATURES

A 50 year female patient was brought by relatives, with a history of consumption of an unknown compound 2 hours earlier followed by profuse vomiting, abdominal pain and altered sensorium. On examination, she was conscious but disoriented, had pallor, mild cyanosis, her SpO2 was only 70%, pulse- 96/min, blood pressure 130/90 mm of Hg but she was not tachypneic (RR-12/min).

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Blood sample drawn for ABC was chocolate brown in colour (as shown in Fig.1), which did not improve on 100% oxygenation and ABC was suggestive of metabolic acidosis with methemoglobin levels 22mg % (normally not detectable). She passed green coloured urine. Haemoglobin was 9g%.Complete blood counts, liver and renal function tests, serum electrolytes were within normal limits. On auscultation, chest was clear. A diagnosis of severe acute methemoglobinemia was made. She was immediately given gastric lavage, inj. Methylene blue was administered 60 mg (1mg/kg body weight) I.V. which improved her SpO2 to 89% over next 15 min. Inj.Vitamin C I.V. was also administered for 3 days, one unit packed cells was given. On the second day, she became conscious, oriented, SpO2 improved to 92%, ABG showed reduction of MethHb levels to 2mg% and she admitted to have consumed "Bloom flower" containing nitrobenzene compound. The bottle containing this poison consumed by her was subsequently brought by relatives (as shown in Fig.2).

Fig. 1.

Fig. 2.

She was discharged on the fourth day on oral iron, folate supplements and ascorbic acid.

DISCUSSION

Nitrobenzene is an oxidising nitrite compound. Acute ingestion of nitrobenzene leads to rapid development of methemoglobinaemia, a condition in which the iron within the haemoglobin is oxidised from the ferrous (Fe^{2+}) state to the ferric (Fe^{3+}) state, resulting in the inability to transport oxygen and causing brownish discolouration of blood. Once formed, methemoglobin can be reduced enzymatically either via an adenine dinucleotide (NADH)-dependent reaction, catalysed by cytochrome b5 reductase, or an alternative pathway using the nicotine adenine dinucleotide phosphate (NADPH)-dependent methemoglobin reductase system. Methemoglobin is normally present as less than 1% of the total haemoglobin under physiologic conditions. Levels above it are defined as methemoglobinaemia. The estimated lethal dose ranges from 2 to 6 g in adults; and doses less than 0.8 mg/kg/day does not normally cause methemoglobinaemia (Agrawal *et al*., 2011).
Acute intoxication is usually asymptomatic up to the level of 10 – 15% of methemoglobin, showing only cyanosis. Beyond 20% levels, headache, dyspnoea, chest pain and tachycardia develops. At 40 – 50% levels, confusion, lethargy, and metabolic acidosis occur leading to coma, seizures, bradycardia, ventricular dysrythmia, and hypertension. Fractions around 70% are fatal. Anemic or G6PD-deficient patients suffer more severe symptoms. Leukocytosis has been reported, with relative lymphopenia (Bradberry, 2003).

Arterial blood gas analysis is mandatory in severe poisoning and reveals an increased methemoglobin concentration and possibly a metabolic acidosis. Following decontamination, high-flow oxygen should be given to maximise oxygen carriage by remaining ferrous haem. No controlled trial of the efficacy of methylene blue has been performed but clinical experience suggests that methylene blue can increase the rate of methemoglobin conversion to haemoglobin some 6-fold. Patients with clinical features and/or methemoglobin concentrations of 30-50%, should be administered methylene blue 1-2 mg/kg/bodyweight intravenously (the dose depending on the severity of the features), whereas those with methemoglobin concentrations exceeding 50% should be given methylene blue 2 mg/kg intravenously. Symptomatic improvement usually occurs within 30 minutes and a second dose of methylene blue will be required in only very severe cases or if there is evidence of ongoing methemoglobin formation. Methylene blue is less effective or ineffective in the presence of glucose-6-phosphate dehydrogenase deficiency since its antidotal action is dependent on nicotinamide-adenine dinucleotide phosphate (NADP+).

In addition, methylene blue is most effective in intact erythrocytes; efficacy is reduced in the presence of haemolysis. Moreover, in the presence of haemolysis, high dose methylene blue (20-30 mg/kg) can itself initiate methemoglobin formation. Supplemental antioxidants such as ascorbic acid (vitamin C), N-acetylcysteine and tocopherol (vitamin E) have been used as adjuvants or alternatives to methylene blue with no confirmed benefit. Exchange transfusion may have a role in the management of severe haemolysis or in G-6-P-D deficiency associated with life-threatening methemoglobinaemia where methylene blue is relatively contraindicated (Bradberry, 2003).The first report of nitrobenzene poisoning came in 1886 and subsequently fatality reports followed (Virendra Goyal, 2014). In our experience of 7 other cases of methemoglobinaemia due to poisoning of suicidal intent, we could save 6 in whom methylene blue was administered within a few minutes of diagnosis. It is cheap - an ampoule of 100 mg costs about Rs.50/- and we recommend that it should always be available as an emergency drug in the casualty or intensive care unit or

even at the periphery. Any case of poisoning whose SpO_2 is low but does not have tachypnea or abnormal findings in the chest should be suspected to have methemoglobinemia. Investigations may not be possible at many centres, but in addition, if the blood appears chocolate brown in colour, greenish urine is passed, or the patient has consumed nitrobenzene, it is worth giving methylene blue immediately. Our ABG machine has the facility to estimate methemoglobin levels, so it simplified making the diagnosis.

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