Case Report

Methemoglobinemia as a result of accidental lacquer thinner poisoning

Ranju Singh, Stalin Vinayagam, Homay Vajifdar

Abstract

Lacquer thinner, commonly used for removing household paints, is known to contain a mixture of various aromatic hydrocarbons, halogenated hydrocarbons and naptha; if ingested, it may cause methemoglobinemia. We report two cases who presented to us with a history of accidental ingestion of paint thinner. Both the patients had very high levels of methemoglobin and were treated with methylene blue (MB), but did not respond to the MB therapy. One of them received an exchange transfusion followed again by MB and survived. Unfortunately the other patient succumbed to the poisoning.

Keywords: Methemoglobinemia, methylene blue, thinner intoxication

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Introduction

Lacquer thinner, commonly used for removing household paints, if ingested, may cause methemoglobinemia.[1] Acute methemoglobinemia often presents as a medical emergency requiring immediate treatment. We report two cases of accidental ingestion of thinner.

Case Reports

Case 1

A 26 year old male was admitted to the emergency with alleged history of accidental ingestion of thinner. He had consumed about 200–250 ml of thinner, mistaking it for water in the darkness of the night. He was brought within half an hour of thinner ingestion with complaints of vomiting, headache, weakness and dizziness. On examination, the patient was conscious, with a dusky discoloration of tongue and nails along with tachycardia and mild tachypnoea. Other vital parameters and systemic examination were within normal limits. Blood withdrawn for investigations was found to be chocolate brown in color. Because of the color of the blood, severe unexplained cyanosis and alleged history of thinner consumption, a clinical diagnosis of methemoglobinemia was made. Blood investigations revealed Hb = 15.5 gm%, platelet count = 2.1 lac/mm^3, blood sugar = 112 mg%, blood urea = 18 mg%, serum creatinine = 1.0 mg%, serum bilirubin = 1.1 mg%, S. Na+ = 143 mEq/L and S. K+ = 3.6 mEq/L. Chest X-ray was normal and arterial blood gas (ABG) analysis revealed mild hypoxemia and metabolic acidosis (pH = 7.23, pO_2 = 91 mmHg, pCO_2 = 31.5 mmHg, HCO_3 = 14.3 mEq/L, BE = 13.2 mEq/L). Oxygen therapy was given and vitals monitoring was carried out. Methemoglobin levels in blood using the absorption spectrophotometry method were performed, which were 32.61% of total hemoglobin. He was given supplemental oxygen and a gastric lavage was done. Inj. methylene blue (MB) 100 mg along with vitamin C 1 g and dextrose containing fluids were administered intravenously. As there was no improvement in the cyanosis, another dose of MB 100 mg IV was repeated after 1 h. An ABG performed at this point showed pH = 7.32, pO_2 = 117 mmHg, pCO_2 = 30.7 mmHg, HCO_3 = 17.4 mEq/L, BE = 8.7 mEq/L. Methemoglobin levels continued to increase and were 49.7% after 6 h and 71.7% after 8 h. ABG revealed pH = 7.32, pO_2 = 60.2 mmHg, pCO_2 = 37.4 mmHg, HCO_3

From:
Department of Anesthesiology and Critical Care, Lady Hardinge Medical College and Associated Hospitals, New Delhi, India

Correspondence:
Dr. Ranju Singh, B-2/532 Ekta Gardens, I.P.Extension, Patparganj, Delhi - 110 092, India. E-mail: ranjusingh1503@yahoo.com

= 19.3 meq/L, BE = 7.0 mEq/L. Patient's trachea was intubated and he was mechanically ventilated with 100% oxygen. He became hemodynamically unstable and was put on vasopressor support with inj. dopamine 15 µgkg/1min/1. Even after 12 h, his methemoglobin levels remained significantly high (74.9%) and, therefore, an exchange transfusion was planned. After the exchange transfusion, the methemoglobin levels decreased to 40.2% (ABG revealed pH = 7.40, pO_2 = 98.4 mmHg, pCO_2 = 32.8 mmHg, HCO_3 = 21.1 mEq/L, BE = 4.8 mEq/L). The patient had two episodes of bradycardia in the next couple of hours and about 6 h later; he had a cardiac arrest, but was successfully revived. Vasopressor support had to be increased (inj. noradrenalin 2-3 $\mu gmin^{-1}$) and mechanical ventilation was continued. The next day, he was given another dose of MB (100 mg) and the methemoglobin levels decreased to 8.60%. Supportive therapy was continued, and the patient was gradually weaned off ventilatory support after 4 days. The SpO_2 increased to 95% on day 3 and to 96–97% on day 5. The patient's trachea was subsequently extubated on day 5 and continued to maintain a good gas exchange on face mask oxygen (5L/min). The patient was discharged from the intensive care unit on day 7.

Case 2

A 20 year-old male was brought to the hospital after he was found unconscious outside his room. On questioning, there was an alleged history of consumption of unknown quantity of thinner. The patient was a chronic alcoholic who had allegedly consumed thinner as no alcohol was available to him that day. On examination, he was unconscious, not responding to deep painful stimuli and severely cyanosed with mottled skin. Pulse rate was 142/min, blood pressure was 80/50 mmHg, respiratory rate was 36/min and SpO_2 on air was 80%. His trachea was intubated and mechanical ventilation was started with 100% oxygen. ABG analysis showed severe hypoxemia and metabolic acidosis (pH = 7.21, pO_2 = 49 mmHg, pCO_2 = 31.5 mmHg, HCO_3 = 16.8 mEq/L, BE = 17.2 mEq/L). Blood withdrawn for investigations was chocolate brown in color. Investigations revealed Hb = 13.6 gm%, platelet count = 2.6 lac/mm3, blood sugar = 97 mg%, blood urea = 20 mg%, serum creatinine = 1.1 mg%, serum bilirubin = 0.9 mg%, S. Na+ = 146 mEq/L, S. K+ = 3.8 mEq/L. Dopamine infusion was started at 15µgkg/1min/1 He received MB 100 mg IV as a provisional diagnosis of methemoglobinemia had been made. As there was no improvement in the cyanosis, another dose of MB 100 mg IV was repeated after half an hour. Methemoglobin levels came out to be 78.6% of total hemoglobin. The patient had a cardiac arrest within 3 h of arrival in the hospital and could not be revived.

Discussion

Pathogenesis

Etiologies of methemoglobinemia can be broadly characterized as genetic or acquired, with the latter being much more common than the former. The majority of methemoglobinemia cases result from exposure to exogenous oxidizing agents [Table 1].[2] Thinner compounds, which are useful in removing household paints, are known to cause methemoglobinemia, as happened in both our cases. These products mainly contain various concentrations of toxic aromatic hydrocarbons (benzene, toluene and xylene) or halogenated hydrocarbons (carbon tetrachloride, methylcellulose and trichloroethylene) along with P naphtha.[1]

Diagnosis

Diagnosis is dependent on clinical suspicion and a history of medication/toxin intake. Direct observation may reveal blood that is chocolate brown in color. The

Table 1: Selected agents capable of inducing methemoglobinemia[2]

Acetanilid
Aniline dyes
Benzene derivatives
Benzocaine
Chlorates
Chloroquine
Clofazimine
Dapsone
Dimethyl sulfoxide
Dinitrophenol
Ferricyanide
Lidocaine
Methylene blue*
Metoclopramide
Naphthalene
Nitrites
Nitrates
Nitrobenzene
Nitroglycerin
Nitroprusside
Paints
Phenacetin
Phenazopyridine
Phenytoin
Prilocaine
Primaquine
Resorcinol
Rifampin
Smoke inhalation
Sodium valproate
Sulfasalazine
Sulfonamides
Toluidine

*Both a cause and treatment of methemoglobinemia

presence of definite cyanosis in an individual with little or no dysponea and a normal PaO_2 level may also suggest the diagnosis.[3] Arterial blood gas analysis will typically reveal a normal arterial oxygen tension (PO_2) and may reveal a metabolic acidosis proportional to the severity and duration of tissue hypoxia. Because of the spectral properties of methemoglobin, oxygen desaturation recorded by pulse oximetry may not correlate well with the actual percentage of methemoglobin in the blood. Determination of the calculated versus measured arterial "saturation gap" using co-oximetry helps in diagnosis. A saturation gap of more than 5% suggests the presence of methemoglobin, carboxyhemoglobin or sulfhemoglobin.[4]

Clinical symptomatology varies depending on the methemoglobin concentration. Cyanosis begins to develop at levels greater than 1.5 g/dL and at levels greater than 7.5 g/dL, acidosis, bradycardia, seizures, coma, arrhythmia and death may ensue. However, the onset of symptoms does not always correlate with particular methemoglobin concentrations.[3]

Treatment

Treatment of methemoglobinemia is dependent on the aetiology and time course. For acute methemoglobinemia due to drug exposure, treatment includes discontinuation of the offending agent. Treatment is with slow intravenous injection of MB (the recommended dose is 2 mg/kg for infants, 1.5 mg/kg for older children and 1 mg/kg for adults) diluted in 1% sterile aqueous solution infused over 5 min.[4] The drug is available as an ampoule of 1% solution containing 10 mg/ml. MB powder can also be reconstituted into a 1% solution and sterilized for intravenous use. Relief is rapid and methemoglobin levels are generally brought below 10% within 30 min. The dose can be repeated after an interval of 1 h if the cyanosis has not cleared, till a maximum dose of 7 mg/kg over 24 h can even be repeated twice or thrice daily.[5] MB exerts its reductive effects by activating the dormant but volatile hexose monophosphate (HMP) shunt to regenerate NADPH. Dextrose should be co-administered in order to increase NADPH formation. If MB therapy is ineffective and life-threatening shock is imminent, exchange transfusion should be initiated. Ascorbic acid, part of the minor reduction pathway of methemoglobin, may be useful in patients in whom MB therapy is contraindicated. Additionally, activated charcoal may be useful in instances of dapsone-induced methemoglobinemia [3] *N*-acetylcysteine, a mucolytic agent, is believed to restore intracellular glutathione and may be capable of serving as a glutathione substitute capable of directly reducing oxidized agents.[6] Hyperbaric oxygen is another modality recommended, but is reserved only for those patients who have a methemoglobin level >50% or those who do not respond to standard treatment.[7]

Acquired methemoglobinemia has been reported to be due to accidental, [8, 9] suicidal [5,10] or occupational causes,[11] many of the patients being children. On literature search, case reports on thinner intoxication as a cause of methemoglobinemia were found to be few and far in between. The present cases are among the very few that highlight methemoglobinemia as a manifestation of thinner intoxication in adults. Collison reported the death of a 38-year-old Caucasian male whose autopsy revealed thinner in his stomach.[12] Verma reported a case of a 3-year-old child who had accidentally ingested two to four teaspoons of thinner. The child was managed conservatively and had a complete recovery.[2] Saxena et al. reported acute poisoning with nitrobenzene in a 16-year-old female where they used repeated infusions of MB to treat the patient successfully.[13]

Conclusion

Acute methemoglobinemia constitutes a medical emergency and may result in significant mortality inspite of treatment, if it is severe. Cases of suspected toxin-induced methemoglobinemia should be treated with prompt administration of MB and an early exchange transfusion if there is no response to the administration of MB. Repeat dose of MB can also be used after the exchange transfusion to bring down the methemoglobin levels.

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