RESEARCH PAPER

Blood Blues: Pesticide-induced methemoglobinemia and its outcomes: Experiences from a Tertiary Care Center

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Abstract:

Acquired methemoglobinemia is a dyshaemoglobinemia that results from exposure to various oxidizing agents, resulting in impaired oxygen delivery to the tissues and can be potentially fatal if untreated. Methemoglobinemia is not the first among the differential diagnoses that crosses a clinician's mind when assessing a cyanotic and hypoxemic patient. Deliberate ingestion of certain herbicides, insecticides, and pesticides may produce this condition. We report 8 cases of methemoglobinemia due to intentional ingestion of pesticides, which were marketed as safe and contained only biological extracts and fillers. Methylene blue, ascorbic acid, blood transfusion, and exchange transfusion are the various modalities of treatment.

Key words: Biological extracts, pesticides, methemoglobinemia

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Introduction:

Methemoglobinemia refers to the oxidation of hemoglobin iron from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state, which is incapable of binding with the oxygen molecule, resulting in tissue hypoxia and functional anemia. It has two types: a) Congenital, b) Acquired, with the latter more frequently encountered due to exposure to nitrates, nitrites, aniline derivatives, dapsone, local anaesthetics, or agrochemicals such as pesticides and herbicides. The ferric hemes of methemoglobin (MetHb) do not bind oxygen, and the ferric heme in the hemoglobin tetramer also causes the remaining normal ferrous hemes within the same tetrameric hemoglobin have increased oxygen affinity, which produces a left shift of the hemoglobin oxygen dissociation curve, which in turn leads to decreased oxygen delivery to the tissues and results in cyanosis. The MetHb for normal adults will be approximately less than 0.14-0.17 and 0.12-0.15 mg/dl in males and females, respectively. In Methemoglobinemia, MetHb level is >1% of the Hb level in the blood.

Clinical features of methemoglobinemia depend on the levels of MetHb, some people may tolerate MetHb level up to 10%–20%, cyanosis may occur at 15% MetHb , at 20%–45% MetHb people may develop shortness of breath, coughing, headache, lethargy, tachycardia, tissue hypoxia, weakness, and dizziness, at level exceeds 45%, people will develop dyspnea, arrhythmias, metabolic acidosis, seizures, congestive cardiac failure, altered mental status, coma. Severe methemoglobinemia is life-threatening, needs immediate medical attention, and treatment.

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We hereby present a case series of methemoglobinemia, which may help clinicians to suspect methemoglobinemia in unexplained hypoxia in poisoning cases.

Case 1:

A 25-year-old woman presented to the emergency department with an alleged history of poisoning with pesticide (composition: Biological extracts-2%, Stabilizers-8%, and fillers-90%) with suicidal intent within 40 minutes after consumption. She was found unresponsive and was intubated and mechanically ventilated. At presentation, she was cyanotic with a pulse rate of 90 beats/min; respiratory rate, 28 breaths/min; and blood pressure, 130/80mmHg.Bilateral breath sounds were clear on auscultation of her chest. Bilateral pinpoint pupils with sluggish reaction to light were noted. She was initially treated with gastric lavage and activated charcoal. Within 3 hours after admission, she became hemodynamically unstable and developed generalized tonic-clonic seizures. Oxygen saturation measured by pulse oximetry (SpO2) was 67 % at ambient air and 88% with 100% oxygen. Her blood samples appeared chocolate brown (Fig1). Arterial blood gas (ABG)

analysis showed a partial pressure of O2 (pao2)467.5mmHg, lactate 8mmol/L, and an arterial O2 saturation of 99% (SaO2). In view of the "saturation gap'' (difference between SpO2 measured by pulse oximetry and SaO2 measured by ABG), chocolate brown colored blood, and cyanosis in the presence of normal pao2, we suspected methemoglobinemia. CO-oximetry revealed MetHb of 78%, which was further confirmed by absorption spectrometry. G6PD levels were normal. She was treated with 1% methylene blue, ascorbic acid, blood transfusion, antiepileptics, and inotropic support, but despite treatment, she expired on day3.

Case 2:

A 25-year-old man, with no premorbid illness, was brought to the emergency with an alleged history of consumption of about 100ml of pesticide called Deside (composition: biological extract 5%, stabilizers 5%, fillers 90%) with suicidal intent, after 90 minutes of ingestion. At presentation, he was conscious, coherent, and cyanotic. His vital signs were as follows: pulse rate, 120/min; blood pressure, 130/80 mmHg; respiratory rate, 26 breaths/min. SpO2 by pulse oximetry was 70% at ambient air and 86% with oxygen supplementation of 6L/min. Bilateral breath sounds were clear. No abnormality found on cardiac auscultation. Bilateral pinpoint pupils with sluggish reaction to light were noted. He was initially treated with gastric lavage and activated charcoal. Within 30 minutes of hospitalization, he became unconscious and desaturated (SpO2, 60%), for which he was intubated and mechanically ventilated. ABG analysis showed severe metabolic acidosis(pH:7.10) with lactic acidosis (lactate,15.4mmol/L);Pco2 24.7mmHg; pao2 ,621.9mmHg; and bicarbonate,7.7mmol/L.CO – oximetry analysis showed MetHb,80.1%, which was reconfirmed by spectroscopic analysis. G-6PD deficiency was excluded by the enzyme analysis method.

He was administered 1% methylene blue 1mg/kg diluted in 100ml of normal saline, infused over 5 mins. Green colored urine was noted after giving Methylene blue (Fig2). CO-oximetry after 1 hour showed MetHb of 50 %. A single unit of exchange transfusion was done. He gained consciousness and became oriented. Two doses of 1% methylene blue were repeated with an interval of 12 hours to prevent rebound methemoglobinemia and MetHb was 6.2% after 3rd dose. In addition, he received an intravenous ascorbic acid 1000mg in 5%dextrose once daily. He was extubated on the 4th day and discharged after 7 days of admission. MetHb was 2% at the time of discharge.

Case 3:

A 38-year-old man, with no premorbid illness, was brought to the emergency with an alleged history of consumption of about 40ml of pesticide (composition: biological extract 2%, stabilizers 8%, fillers 90%) with suicidal intent, two hours after ingestion. At presentation, he was conscious, coherent, and cyanotic. His vital signs were as follows: pulse rate, 86/min; blood pressure,120/70mmHg; respiratory rate,24 breaths/min. SpO2 by pulse oximetry was 78% at ambient air and 85% with oxygen supplementation of 6L/min. Bilateral breath sounds were clear. No abnormality found on cardiac auscultation. Pupils were normal in size and reacted to light. ABG analysis with CO –oximetry showed pao2, 214mmHg; SaO2, 98%, lactate 4.1 mmol/L and MetHb, 40%. The electrocardiogram showed a prolonged QTc interval. He was initially treated with gastric lavage and activated charcoal, followed by 1% methylene blue (1mg/kg), intravenous ascorbic acid 1000 mg in 5% dextrose was administered. Magnesium sulfate was given intravenously for a prolonged QTc interval. He was discharged after 5 days of observation.

Case 4:

A 45-year-old male, with no premorbid illness, was brought to the emergency with an alleged history of consumption of about 30 ml of pesticide (carbendazim 12% + Mancozeb 63%) with suicidal intent mixed with alcohol one hour after consumption. Immediate gastric lavage was done, and activated charcoal was given. At presentation, he was irritable and cyanotic. His vitals were as follows: pulse rate 105/min, BP 140/100 mmHg, RR 26/min. SpO2 by pulse oximetry was 82% on room air and 92% with O2 supplementation at 8L/min. Basal crepitations were present on auscultation of the chest. Pupils were normal in size and reacting to light.

ABG with CO-oximetry pH 7.34, PaO2 234mmHg, lactate 2.2 mmol/L, SaO2 96% and MetHb 22.4%. He was administered with 1% Methylene blue 1 mg/ kg over 15 min and ascorbic acid 1 g intravenously in 5% dextrose. On the third day, he was discharged with a MetHb of 2%.

Case 5:

A 26-year-old male was brought to the emergency with an alleged history of consumption of an unknown quantity of insecticide (Emamectin benzoate 1.9 W/V, Sodium N methyl taurate 7.5%, Sodium alkyl naphthalene sulfonate 1%W/W, lactose anhydrous) mixed with alcohol with suicidal intent, approximately 3 hours after consumption. At presentation, he was unconscious, cyanotic, immediately intubated, and mechanically ventilated. His vital signs were as follows: Pulse rate 110/min, BP 130/90 mmHg, respiratory rate 28/min. His SpO2 is 77% on room air and 88% with FiO2 of 100%. Lungs were clear and cardiac auscultation was normal. Pupils were bilateral pinpoint but reacting to light. ABG analysis with CO-oximetry showed PH of 6.9 with lactic acid levels 22 mmol/L, PaO2 385mmHg, SaO2 99%, Hco3- 9 mol/L, MetHb 85%. He was treated with gastric lavage, activated charcoal, 1% Methylene blue 1 mg/kg given twice with a gap of 3 hours, a single unit of exchange transfusion, intravenous ascorbic acid 1000 mg in 5% dextrose, and Sodium bicarbonate intravenously. ABG analysis was repeated on the next day showed pH 7.31, PaO2 264mmHg, MetHb 18.2%, and was given third dose of 1% Methylene blue (1mg/kg). He was discharged on day 5 with stable vital signs.

Case 6:

A 50-year-old patient, who was diabetic and hypertensive, was brought to the emergency with an alleged history of consumption of 200ml of herbicide (GLORY N: seaweed extract 2%, chelating agents, stabilizers, buffers, 98%) with suicidal intent, within 1 hour of consumption. He was conscious, oriented, and complained of diffuse abdominal pain. Both pupils were normal in size, reacting to light, both lungs were clear, and cardiac auscultation was normal. At presentation, his vitals were as follows: Pulse rate 112/min, BP 100/80mmHg, respiratory rate 25/min.

SpO2 in room air was 88% and 92% with Oxygen supplementation of 8litres/min. ABG analysis with CO-oximetry showed pH 7.38, PaO2 295 mmHg, lactate 1 mmol/L, SaO2 99%, and MetHb 16.3%. He was treated with gastric lavage, activated charcoal, 1% Methylene blue (1 mg/kg) in 100 ml saline over 15 min, and ascorbic acid 1000 mg in 5% dextrose. He was discharged on day 3 with MetHb 1.8%.

Case 7:

A 60-year-old male with no premorbid illness was brought to the emergency with an alleged history of consumption of an unknown quantity of an unknown compound at his agricultural fields with suicidal intention. On presentation, he was unresponsive and cyanotic. His vital signs at presentation were as follows: Pulse rate 105/ min, BP 140/70 mmHg, respiratory rate 24 breaths/min. Blood glucose levels by glucometer showed 52 mg/dl. On examination, both pupils were small and reacted to light, both lungs were clear, and cardiac auscultation was normal. He was given 25% dextrose immediately and was intubated and mechanically ventilated. His SpO2 was 75% on room air and 80% on FiO2 100%.ABG analysis showed pH 7.42, PaO2 551mmHg, lactate 2 mmol/L, SaO2 99.96%, and MetHb 44.5%. He was initially treated with gastric lavage and activated charcoal, followed by antidote 1% Methylene blue 1 mg/ kg once a day for 3 days, and ascorbic acid 1000 mg in 5%dextrose was given. Despite clinical improvement, the patient developed hemolytic jaundice with a drop in hemoglobin. Two PRBC transfusions were done. G6PD deficiency was suspected in view of hemolytic jaundice, but levels were found to be normal. He was treated symptomatically for 1 week. After one week, the patient was discharged in a stable condition with MetHb of 1.5%.

Case 8:

A 40-year-old male who was diabetic, brought to the emergency with an alleged history of consumption of an unknown quantity of herbicide (Alfa naphthalene acetic acid, navica plant extract) with suicidal intent, within 3 hours of consumption. On presentation, he was unconscious and cyanotic. Capillary blood glucose was 112 mg/dl. His vital signs were as follows: Pulse rate 98/min, BP 150/110 mmHg, respiratory rate: 28/ min. SpO2 was 77% on room air and 82% with FiO2 of 100%%. No abnormality found on auscultation of the chest. No focal neurological deficit was identified. ABG analysis with CO-oximetry showed pH 7.38, PaO2 386 mmHg, SaO2 99%, PaCo2 40 mmHg, lactate 1.1 mmol/L, and MetHb levels of 70%. The patient was intubated and mechanically ventilated in view of low GCS. He was treated with gastric lavage, activated charcoal, antidote Methylene blue 1mg/kg, a single unit of exchange transfusion, and ascorbic acid 1000mg in 5% dextrose, but the patient was discharged against medical advice during the middle of hospitalization.

Table 1: Clinical features of cases at the time of presentation

	Clinical features	Frequency (n)
1	Cyanosis	7
2	Low oxygen saturation on pulse oximetry (<92%)	8
3	Pinpoint pupils	4
4	Altered mental status	5
5	Hypoglycemia	1

Table 2: Representing Methemoglobin levels on CO-oximetry

	Methemoglobin levels (%)	Frequency (n)
1	Normal (0-1)	
2	Asymptomatic methemoglobinemia (2-20)	1
3	Mild methemoglobinemia (21-50)	2
4	Moderate methemoglobinemia (51-70)	1
5	Severe methemoglobinemia (>70)	4

Discussion:

In India, methemoglobinemia is not uncommon as of thought because most of the cases were unrecognized by the physicians. Methemoglobinemia (MetHb>2%) is an altered state of hemoglobin that results when there is overwhelming oxidative stress exceeding the normal protective mechanisms. Acquired methemoglobinemia is induced by exposure to various oxidizing agents, most commonly due to nitrates and nitrites (1, 2). Several cases of acquired methemoglobinemia due to suicidal intent have been reported from India (3, 4, 5, 6, 7, 8, 9)

Under normal circumstances, methemoglobin is reduced by enzymatically, mostly through NADH-dependent reactions rather than NADPH-dependent reactions, and nonenzymatically by either ascorbic acid or reduced glutathione, the latter being one slow and quantitatively less important.
The clinical manifestations (Table 3) of methemoglobinemia are due to impaired oxygen delivery to the tissues and hence correlate with severity of methemoglobinemia.

Table 3: Levels of methemoglobin and associated clinical features (10)

Methemoglobin %	**Signs and symptoms**
< 15%	Asymptomatic
20-30%	Cyanosis, headache, fatigue, mental status changes, syncope, dizziness, and exercise intolerance
30-50%	Shortness of breath and headache
50-70%	Lethargy, stupor, dysrhythmias, seizure, coma
>70%	Death

Co-morbidities such as anemia, congestive heart failure, chronic obstructive pulmonary disease, pneumonia, sepsis or the presence of other dyshemoglobins amplify the clinical effects of methemoglobinemia. The onset of signs and symptoms in our cases was 30-60 minutes after pesticide ingestion. In case 6, patients had an asymptomatic range of methemoglobinemia and had tachypnea only. MetHb levels of >70% are usually fatal despite treatment, which also happened in the first case however, survival has been reported in a few cases (4,8). We observed bilateral pinpoint pupils in four out of eight cases and excluded organophosphate poisoning based on normal RBC and plasma cholinesterase levels. This finding of pinpoint pupils was seen in two cases reported by Geetika et al (7) with similar poisoning but not seen in cases described by George et al (4).
Common insecticides that induce methemoglobinemia include indoxacarb, aluminium phosphide, and paraquat. The pesticide consumed in 5 out of 8 case reports contains biological extracts, stabilizers and fillers which were marketed to be safe and have no mention of an antidote. Biological extracts are rich in nitrogenous products and hence can potentially cause methemoglobinemia (4). These agrochemicals containing alkyl naphthalene compounds that induce methemoglobinemia as seen in cases 5 and 8.(20)
Methemoglobinemia should be suspected clinically by the presence of clinical cyanosis in the presence of a normal arterial pO2 (PaO2) and chocolate-brown colored blood. Routine pulse oximetry may be inaccurate for monitoring oxygen saturation in the presence of methemoglobinemia and should not be used to make the diagnosis of this disorder. The presence of methemoglobin can be suspected when the oxygen saturation measured by pulse oximetry (SpO2) is significantly different from the arterial oxygen saturation (SaO2) from ABG analysis ("saturation gap"). This saturation gap between SaO2 and SpO2 greater than 5% is a diagnostic clue to the presence of MetHb (12). To confirm methemoglobinemia, carbon monoxide (CO)-oximetry is required. CO-oximetry is an in vitro spectrophotometric method that uses multiple wavelengths to determine the levels of oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin (13). Even COoximeters cannot distinguish between MetHb and sulfhemoglobin due to similar absorbance peaks at 630 nm (14). MetHb was detected by absorption spectrophotometry after the addition of sodium cyanide in the clinical biochemistry laboratory.
Management of acute methemoglobinemia includes discontinuation of the offending agent, gastric decontamination, and administration of activated charcoal. Methylene blue (MB) is the first-line antidotal therapy, which provides an artificial electron transporter for the reduction of MetHb via the NADPH-dependent methemoglobin reductase system. Methylene blue is indicated when MetHb exceeds 20-30% in an asymptomatic patient or when the patient exhibits symptoms of oxygen deficiency, such as dyspnea and alteration of consciousness as in our case 6 (15). The recommended dose of 1% methylene blue for adults is 1-2mg/kg diluted in 100ml of isotonic saline, infused intravenously over 5minutes. The response is usually rapid within 30minutes; the dose may be repeated in one hour if the level of methemoglobin is still high one hour after the initial infusion (16). Dextrosecontaining fluids should be co-administered to increase NADPH formation. Injection ascorbic acid (300 to 1000 mg/day) may be useful which activates the alternate minor pathway. Serial measurements of methemoglobin levels should be performed following treatment with MB, as rebound methemoglobinemia may occur up to 18 hours after MB administration, due to prolonged absorption of the implicated agent from topical or enteric sites. The dose can be repeated hourly up to a maximum of 7mg/kg over 24hours (17). Certain drugs, such as dapsone, produce MetHb over a prolonged biologic half-life because of ongoing formation of metabolites. In these situations, some clinicians prefer continuous infusions of MB titrated from a starting rate of 0.1 mg/kg/hour, rather than intermittent bolus therapy (18). As observed in one of our patients, symptoms of dyspnea and depressed mental status improved within 30 minutes of MB injection. Caution should be exercised to avoid overdosage (>7 mg/kg) because cumulative doses of MB can cause dyspnea, chest pain, hemolysis, and paradoxical methemoglobinemia in some susceptible

subjects, as occurred in our case 7 (19).In case Methylene blue should not be administered to patients with known glucose 6-phosphate dehydrogenase (G6PD) deficiency, since the reduction of methemoglobin by MB is dependent upon NADPH generated by G6PD.

Severe methemoglobinemia (MetHb >70%) is usually fatal, as evidenced in our first case though survival has been reported with a MetHb level of 80% in our case2 and 85% in our case 5 and previous reports (4, 8).

If MB therapy fails to relieve methemoglobinemia, one should consider the possibility of the need for additional doses of MB or inadequate decontamination of the gut or an incorrect diagnosis (sulfhemoglobinemia) or G6PD deficiency or congenital NADPH MetHb reductase deficiency.

Blood transfusion may be indicated to decrease MetHb levels that have escalated to near-fatal levels but should be done only after antidotal therapy with MB.

In case of unresponsive methemoglobinemia with MB, exchange transfusion, as done in our case 2 or hyperbaric oxygen may be beneficial (9,12).

Conclusion: Every physician should be cautious in cases of poisoning with an unknown chemical composition, which may potentially cause fatal complications like methemoglobinemia. A high index of clinical suspicion of methemoglobinemia is required in all cases of unexplained cyanosis. Acute methemoglobinemia is a medical emergency that can cause mortality if untreated. Methylene blue is the treatment of choice. An early exchange transfusion may be beneficial if there is no response to MB.

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