

Case Report

BIOPESTICIDE- A RARE CAUSE OF METHAEMOGLOBINAEMIA

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ABSTRACT

Methaemoglobinaemia is a rare diagnosis in patients who have cyanosis and dyspnoea that are unrelated to a cardiopulmonary cause. These patients remain unresponsive to oxygen therapy. We present a case of methaemoglobinaemia following ingestion of a biopesticide in an attempt of deliberate self-harm. Methaemoglobinaemia was suspected in our patient because of cyanosis and oxygen saturation gap. He was saved from life threatening emergency by mechanical ventilation, oral/ intravenous methylene blue, ascorbic acid and supportive measures. On literature search no case report of methaemoglobinaemia caused by ingestion of biopesticide was found. We report this because of its rare occurrence.

Key Words: Biopesticide, Methaemoglobinaemia, Methylene Blue

INTRODUCTION

Biopesticide, labelled to be eco-friendly unlike other pesticides, are generally considered safe. In our patient methaemoglobinaemia occurred following ingestion of a biopesticide (containing oligosaccharides, bio emulsifier and filter substance) in an attempt at deliberate self-harm. The rarity of a biopesticide causing methaemoglobinaemia is being highlighted in this case report, though conventional chemical pesticides causing the same have been reported.

CASES

A 40 year old male came with alleged history of consumption of biopesticide (50ml). He was taken to a nearby hospital where he received a stomach wash and was intubated. He was referred to our hospital after two days of treatment.

On arrival at the emergency department, the patient was restless and drowsy (GCS-9/15). On examination he had generalized cyanosis. Vital signs revealed a temperature of 101F with tachycardia (126/min), tachypnea (30/min) and SpO₂ of 84% with FiO₂ 50%. Blood pressure was 130/70mm Hg. Systemic examination revealed no other findings.

On examination in Intensive Care Unit, the patient continued to be drowsy with mid-dilated pupils reacting equally. He was continued on mechanical ventilation. His blood was noticed to be muddy brown in colour, hence methaemoglobin levels were done. Arterial blood gas (ABG) analysis showed pO₂ of 240.3mmHg and SaO₂ of 99.7% with FiO₂ of 100%, pH=7.45, pCO₂=27.9mm Hg and HCO₃=19.2mmol/L. Methaemoglobin was 50.6%. Methaemoglobin was measured using Cobas-b blood gas analyser. Serum pseudo-cholinesterase level was normal. Complete blood counts showed total leukocyte count 24.2X10³/ul and haemoglobin level of 10.4gm/dl. Urine analysis, liver function tests, renal function tests, serum electrolytes and coagulation profile were normal. Chest X ray and ECHO were normal. Ultrasound abdomen showed mild hepatomegaly.

On day one in our hospital, patient was diagnosed to have methaemoglobinaemia. Injection ascorbic acid 500mg in 100ml normal saline (NS) over 30 minutes was given 6th hourly. Due to unavailability of methylene blue (MB), he was managed with supportive and symptomatic treatment. On day two and three patient's status remained the same with persistently high methaemoglobinaemia.

On the night of day four when oral MB became available, first dose was given through Ryle's tube as 100mg in 500ml of 5% dextrose over 30 minutes. Prior to administration methaemoglobin was 57.6%.

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Within two hours, SpO₂ improved to 89% with FiO₂ of 70% and methaemoglobin level was 27.5%. Second dose of oral MB was given on the morning of day five, following which methaemoglobin level dropped to 18.7%. On availability of intravenous MB, it was given on day six as 100mg in 100 ml NS over one hour followed by maintenance of 60mg in 100ml NS intravenously over one hour twice a day for two days. Methaemoglobin level further dropped to 9.7%. SpO₂ increased to 93% with FiO₂ of 50%. On day nine patient's methaemoglobin level was 2.1%. SpO₂ improved to 95% with FiO₂ of 40%. Patient was extubated on day ten and was maintaining SpO₂ of 96% with oxygen 1lit/min through nasal prongs. Patient was shifted to ward on day 12 and discharged well on day 15.

DISCUSSION

Methaemoglobinaemia means excess accumulation of methaemoglobin in RBC. Methaemoglobin is a form of haemoglobin in which iron in the haem complex is oxidized from ferrous to ferric state. Due to this change oxygen (O₂) cannot combine with haemoglobin and O₂ will not dissociate in tissues. Methaemoglobin in our body is reduced through nicotine adenine dinucleotide phosphate (NADPH) dependent methaemoglobin reductase system and nicotine adenine dinucleotide (NADH) enzyme system which is catalysed by cytochrome b5 reductase (Kakhandki *et al.*, 2012; Saxena and Saxena, 2010).

Methaemoglobinaemia can be hereditary due to deficiency of enzymes required for methaemoglobin reduction or acquired due to nitrates, aniline dyes, dapsone, sulfonamides, primaquine, benzocaine (Abu-Laban *et al.*, 2001; Tantisattamo *et al.*, 2011).

Levels of methaemoglobin in blood directly correlate with symptoms seen (Weichert, 2011 and Pasternack, 2004).

1-2%	Normal
<10%	No symptoms
10-20%	Cyanosis
30-50%	Anxiety, dyspnoea, headache, nausea, palpitations, tachypnea, fatigue, giddiness, confusion
50-70%	Seizures, acidosis, coma, arrhythmias
>70%	Death.

Our patient had levels close to 50% and manifested the respective symptoms.

Diagnosis is based on the presence of cyanosis incompatible with degree of respiratory distress with symptoms related to methaemoglobin concentration. Typical finding is the pulse oximeter (SpO₂) reading of 85% not improving with 100% oxygen and a higher O₂ saturation (SaO₂) on ABG. This saturation gap (SaO₂- SpO₂) of more than five is suggestive of methaemoglobinaemia (Young, 2008). The blood has a chocolate brown colour not disappearing with O₂ therapy and fails to turn bright red colour on shaking. Our patient had both these features.

Pulse oximeter yields information about differential light absorption at two wavelengths (660nm and 940nm) by tissues. At 660nm, methaemoglobin has a similar absorbance pattern as that of haemoglobin. However at 660nm methaemoglobin has greater absorbance pattern than that of oxyhaemoglobin and exceeds absorbance pattern of both oxyhaemoglobin and haemoglobin at 940nm (Pasternack, 2004). So methaemoglobin having increased light absorbance at two different wavelengths produces an alteration in the calorimetric measurement by pulse oximeter. Here comes the importance of CO-oximetry study, an in vitro spectrophotometric method which measures oxyhaemoglobin, deoxyhaemoglobin, carboxyhaemoglobin and methaemoglobin separately (Haymond *et al.*, 2005). We have used CO-oximetry to determine methaemoglobin level.

The major goal in the treatment of methaemoglobinaemia is to improve the dissociative capability and oxygen carrying capacity of haemoglobin. The cornerstone in the treatment of methaemoglobinaemia is administration of MB which acts as a cofactor for reduced NADPH methaemoglobin reductase and increases the reduction of methaemoglobin. The mode of administration depends on the available form of MB locally. Typical doses of 1-2 mg/kg body weight are given intravenously over 5-10 minutes and can be repeated after 20 minutes (Pasternack, 2004; Saxena and Saxena, 2010) preferably to be administered

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with dextrose to increase NADPH formation (Singh *et al.*, 2012). Response to treatment occurs within one hour. The aqueous oral form of MB has higher absolute bioavailability, making it equally effective as IV form. Oral form of MB is absorbed from the gastrointestinal tract, reduced to leucomethylene blue in tissues and slowly excreted mainly in the urine (Kakhandki *et al.*, 2012). Other forms of treatment include administration of ascorbic acid, N-acetylcysteine and exchange transfusion (Singh *et al.*, 2012 and Bradberry, 2003). Patients with methaemoglobin level >50% or those who do not respond to standard treatment are ideal candidates for administration of hyperbaric oxygen (Jansen *et al.*, 2003).

This case is being reported for its rarity of a biopesticide causing methaemoglobinaemia. No such case was found on a literature search. Emphasis is also being made on the effectiveness of oral MB when intravenous MB is not available to treat this life threatening condition.

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