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Acute Methemoglobinemia with Hemolytic Anemia Following Bio-Organic Plant Nutrient Compound Exposure: A Case Report

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Abstract: A middle aged adult male patient was referred to our hospital with complaints of pain abdomen and vomiting following consumption of bio-organic plant nutrient extract with a suicidal intent . On examination patient had pallor and cyanosis of mucus membranes. Pulse oximetry showed oxygen saturation of 52% on room air. Even with supplementation of 10L of oxygen, saturation increased to 66% and cyanosis failed to improve.Arterial blood gas analysis was normal, thus suggesting a‘ saturation gap’.Methemoglobinemia was suspected based on above findings. Patient was then started on IV preparation of methylene blue , following which patient showed a dramatic improvement in saturation as monitored through pulse oximetry. On evaluation patient had signs and symptoms of hemolysis which was later confirmed through laboratory investigations. Patient eventually improved with blood transfusion and supportive treatment. Patient was discharged from hospital after 15 days without any sequelae.
Keywords: Bio-organic compound, Haemolytic anemia, Methemoglobinemia, Methylene blue, Saturation gap

INTRODUCTION

Methemoglobinemia is a disorder which occurs when red blood cells contains methhemoglobin at levels higher than 1%. This results in reduced capacity of the blood to carry oxygen to tissues. It is caused as a result of administrating oxidizing agents with the associated oxygen–hemoglobin dissociation curve therefore shifted to the left. Methemoglobinemia can be congenital, but its acquired form is more often [1]. The acquired form might be seen after consumption of some foods and additives, and exposure to certain chemicals and drugs. Symptoms are proportional to the methemoglobin level and include skin color changes and blood color changes at levels up to 15%. As levels rise above 15%, neurologic and cardiac symptoms arise as a consequence of hypoxia. Levels higher than 70% are usually fatal [8]. Herein we report a case of a bio-organic plant nutrient compound poisoning with unexplained cyanosis and hemolysis, which ultimately led to diagnosis of methemoglobinemia on physical findings and blood gas analysis.

CASE REPORT

A 35 year old male patient was brought to our emergency room with complaints of pain abdomen and 3 episodes of vomiting. On admission, patient’s relatives gave alleged history of consumption of about 50ml of bio-organic plant nutrient (growth enhancer) containing nitrogen based compound (SARAS) (Fig.1) with suicidal intent, 3 days before arrival to hospital. Patient was initially treated at a local hospital. In view of persisting pain abdomen and vomiting patient was referred to our hospital. Previously patient had no notable history of any systemic illness. On examination , patients mucus membranes were cyanosed (Fig.2) and had icterus. Vitals on arrival were Pulse rate – 80 beats/min , Blood pressure- 110/70 mm of Hg , Respiratory rate – 20 breaths/min . Respiratory and cardiovascular examinations revealed no abnormality. Saturation on room air was 52% and after supplementing with 10L of oxygen saturation increased only to 66%. An arterial blood gas analysis done at that time showed partial pressure of oxygen to be 121.6 mm of Hg and a hemoglobin oxygen saturation of 98.8%. ECG and CXR were within normal limits.

Fig. 1: SARAS: Containing nitrogen based compound

Fig. 2: Cyanosis of tongue

Fig. 3: Haemoglobinuria

Since patient presented after 3 days of consumption of the compound gastric lavage and activated charcoal was deferred. Patient was treated symptomatically with supportive treatment and oxygen supplementation through face mask. However no significant improvement was noticed in saturation despite arterial blood gas analysis showing normal partial pressure of oxygen. As patient had no respiratory distress patient was not put on mechanical ventilator. Antidote therapy with 50mg of methylene blue diluted in 500ml of NS over 4 hrs was given. Patient showed reduction in cyanosis and gradual improvement in saturation over a period of 3 days. Blood investigations, liver function tests and urine analysis showed hemolytic picture (Fig. 3). Since patients hemoglobin percentage was drastically dropping , patient was transfused with 4 units of packed red blood cells. He was also given 500mg of ascorbic acid IV OD for 5 days followed by oral supplementation. After 15 days patient was discharged with no cyanosis, increased hemoglobin values , other systems being unremarkable.

DISCUSSION

Hemoglobin has functions besides carrying oxygen to the tissues, and regulates vascular tone and inflammation via a redox couple with methemoglobin. Hemoglobin has iron in the reduced valance Fe(II) and methemoglobin has iron in the oxidized valance Fe (III), with a free energy capable of producing water from oxygen. In generating methemoglobin the couple functions as a nitrite reductase. The degree of oxidation of hemoglobin senses the oxygen level in the blood and uses its ability to produce nitric oxide from nitrite to control vascular tone, increasing blood flood when the proportion of oxygenated hemoglobin falls. Additional cardiovascular damage is produced by methemoglobin mediated oxidation of light density lipoproteins, accelerating arteriosclerosis. In addition, the release of heme from methemoglobin is an important factor in inflammation. The symptoms of methemoglobinemia can range in severity from dizziness to coma. Patients may present with cyanosis when the methemoglobin concentration reaches levels of approximately 10% of the total hemoglobin level.

Methemoglobinemia should be suspected when following clues are present:

a) History of exposure to nitrogen based compounds.
b) The presence of methemoglobin can falsely elevate the calculated oxygen saturation when arterial blood gases (ABGs) obtained, resulting in "saturation gap." This occurs when there is a difference between the oxygen saturation measured on pulse oximetry and the oxygen saturation calculated on the basis of ABG results.
c) Dark chocholatecolor of arterial blood which fails to change colour on exposure to air [2].
d) Persisting cyanosis which fails to improve despite giving 100% oxygen.

Methylene blue is first-line antidotal therapy for patients with severe methemoglobinemia. It increases the activity of nicotinamide adenine dinucleotide (NADH)-methemoglobinreductase in red blood cells (RBCs), assisting in the conversion of ferric

(Fe3+) to ferrous (Fe2+) iron [8]. It is available as a 1% solution (10 mg/mL)[5]. The initial dose of methylene blue is 1 to 2 mg/kg intravenously. If symptoms of hypoxia fail to subside, the same dose may be repeated within an hour usually symptoms subside within one hour[6]. Although, successful treatment with plasma exchange therapy, hyperbaric oxygen therapy and ascorbic acid has also been reported, these therapies should be considered as second-line treatments for patients unresponsive to methylene blue.As expected, the agents producing methemoglobinemia may also produce oxidant induced hemolysis and hence a combination of methemoglobinemia and hemolytic anemia may occur, as seen in our patient. Methylene blue may trigger hemolysis in patients with G6PD deficiency [7]. Acquired methemoglobinemia most commonly results from exposure to oxidizing chemicals such as nitrites. Our patient had exposure to bio-organic plant nutrient compounds, one of them being a nitrogen-based compound.

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