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Case Report

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ACUTE METHEMOGLOBINEMIA FOLLOWING UNKNOWN COMPOUND EXPOSURE: TWO CASE REPORTS

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Methemoglobinemia is a rare cause of cyanosis in clinical practice while exposure to oxidizing chemicals is common in rural areas. Acquired methemoglobinemia may present as a serious medical emergency. The diagnosis is mainly clinical and because of its potential lethal nature, a very high degree of clinical suspicion is important. Herein we present two cases of unknown compound poisoning with unexplained cyanosis which ultimately led to the diagnosis of methemoglobinemia on physical findings and blood gas analysis.

CASE REPORTS

- Acute methemoglobinemia is a common hazard caused due to exposure to a wide range of chemicals and often presents as a medical emergency requiring immediate treatment 1.

The diagnosis is mainly clinical and because of its potential lethal nature, a very high degree of clinical suspicion is important. Herein we present two cases of acquired methaemoglobinemia following unknown compound poisoning

CASE 1

- A 16 year old girl was brought to casuality following consumption of 50 ml of unknown compound used for gardening with suicidal intent. She had no notable history of any systemic illness. On examination, smell of organophosphate (OP) compound and cyanosis were present. Vital signs at presentation were as follows: Pulse rate of 90 beats/min, respiratory rate of 28 breaths/min, blood pressure of 100/60 mmHg and SpO2 of 70% with 8Litre/min of oxygen by mask. On auscultation of lungs, bilateral crepitations were present. In view of not maintaining SpO2, patient was intubated and connected to mechanical ventilator with FiO2 of 100%. The patient was treated for OP poisoning with Inj Atropine and Inj PAM. Saturation failed to improve despite ventilation. Arterial blood sample appeared chocolate brown which failed to change colour on exposure to air. Arterial blood gas (ABG) analysis showed a partial pressure of O_2 of 140mmHg (80 to 105 mmHg). Pseudocholinesterase levels, ECG, 2D ECHO and chest radiographs were within normal limits. Co-oximetry revealed methaemoglobinemia). Child was treated with I.V. methylene blue (1mg/kg) and oxygen saturation increased to 94%. Child gradually improved and was stable and discharged.

- CASE 2

A 6 year old boy was brought to paediatric ICU with history of consumption of unknown compound poisoning. Child was sick and smell of OP compound was present. Vitals at the time of admission to ICU were as follows: pulse rate of 80/min, SpO2 of 70% at room air , respiratory rate of 28 breaths per minute and blood pressure of 100/60mm of Hg. On inspection, cyanosis was present. The patient was managed as per protocol with Inj Atropine and Inj PAM. As the child was not maintaining saturation with oxygen support (8Loxygen/min) he was intubated and connected to mechanical ventilator with Fio2 of 100%. Oxygen saturation failed to improve despite mechanical ventilation. ABG revealed partial pressure of oxygen of 204 mm of Hg. Pseudocholinesterase level was normal. Patient's arterial blood sample drawn was chocolate brown in colour arousing a suspicion of methaemoglobinemia.

Co-oximetry could not be done due to financial constraints. Hence filter paper test, bubble oxygen test and saturation gap tests were done. On Filter paper test, patient's failed to change colour even on exposure to air. On Bubble oxygen test, there was no change in the colour of patient's blood on exposure to 100% oxygen. ABG revealed saturation gap. A diagnosis of methaemoglobinemia was entertained based on the above. Intravenous methylene blue was given and SpO2 improved within half an hour. After 2 hours child was

extubated and was maintaining a Spo2 of 95 % at room air. Child gradually improved and was discharged.

DISCUSSION

Acute toxic methemoglobinemia may represent a serious medical emergency because of loss of oxygen carrying capacity of blood and shift of oxygen dissociation curve to the left. Patients may present with cyanosis when the methemoglobin concentration reaches levels of approximately 10% of the total haemoglobin level.
The diagnostic clues for methemoglobinemia are:
(a) Dark chocolate color of arterial blood which fails to change colour on exposure to air.
(b) Cyanosis unresponsive to 100% oxygen.
(c) Oxygen saturation gap i.e., low saturation on pulse oximetry with high saturation on routine arterial blood gas analysis.The saturation gap should alert the physician and the diagnosis should be confirmed by CO - oximetry. In resource limited settings, physicians may not have access to sophisticated laboratory equipment (with advanced co-oximeter), which contributes to the high death rates after poisoning. In a time-pressured situation, some bedside investigations may be useful in guiding specific therapy like methylene blue in a cyanosed patient with suspected poisoning.

Blood samples with a methemoglobin concentration greater than 20% have an evident chocolate-brown color. A quick and easy bedside test to distinguish deoxyhemoglobin from dyshemoglobin is to bubble 100% oxygen in a tube that contains the dark blood. The blood that remains dark likely does so because of the presence of methemoglobin. Another simple test is Filter paper test. Place one to two drops of blood on a white filter paper, and then evaluate for the color change upon exposure to oxygen. The chocolate-brown appearance of methemoglobin does not change with time. In contrast, deoxyhemoglobin appears dark red initially and then brightens after exposure to atmospheric oxygen[2].

Furthermore, the cooking test consists of placing the clotted blood sample in a boiling water bath. After cooking and cooling, the blood sample with methemoglobin will turn pink whereas normal blood will appear dark brown[3].

Shihana and colleagues had developed a color chart which can facilitate more rapid diagnosis of methemoglobinemia and can estimate the percentage of methemoglobin present in a blood sample taken from a poisoned patient. This is of particular value in settings that lack the analytic instruments capable of performing this task[4].

Ideally, the treatment of methemoglobinemia should be guided by measurements of methemoglobin levels, and the instruments needed to measure methemoglobin are not readily available.

Clinicians should be trained on these simple bedside diagnostic tests, so that methemoglobinemia may be diagnosed and treated even at the primary care level.

Methylene blue is indicated as the first-line antidotal therapy for patients with methemoglobinemia. Methylene blue, 1-2 mg/kg administered as a 1% solution undiluted as direct IV over 3-5 minutes, repeated at 1 mg/kg in 1 hour as necessary to control fluctuating symptoms. Methylene blue is also known to cause erroneous SpO2 levels and the antidote is toxic at doses more than 7mg/kg which can cause dyspnea, chest pain, and hemolysis[5,6,7].

Methylene blue is contraindicated in patients with G6PD (glucose-6-phosphate dehydrogenase) deficiency leading to severe haemolysis and it can swap its action causing methemoglobinemia at higher doses. Adjuvant treatment includes ascorbic acid an antioxidant,free radical scavenger which reduces the NAD+ at doses of 0.5-1gm given 8th hourly[8,9]. RBC exchange transfusion and hyperbaric oxygen therapy are usually reserved for patients who are resistant to standard treatment and for those with severe symptoms[10].

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.

CONCLUSION

The treatment of poisoning caused by an unknown compound is a challenge and the situation becomes graver when the patient does not respond properly on treatment. Acute Methemoglobinemia is usually associated with high mortality,hence an early aggressive management of poisoning should be attempted [6,11].

Methylene blue and ascorbic acid are the drugs of choice, while RBC exchange transfusion and hyperbaric oxygen therapy are usually reserved for patients who are resistant to standard
treatment. The authors wish to point out that the non availability of sophisticated laboratory should not be a hindrance for starting treatment.

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