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MANAGEMENT OF A CASE OF METHEMOGLOBINEMIA DUE TO UNKNOWN SUBSTANCE

General Medicine

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ABSTRACT

Methemoglobinemia is a disorder caused by formation of an abnormal hemoglobin in the form of methemoglobin (MetHb). Its is a type of functional anemia as despite of normal oxygen content in the blood, this abnormal hemoglobin, i.e. MetHb is unable to deliver oxygen to tissues leading to hypoxia. Methemoglobinemia can occur either congenitally or after exposure to toxic substances like aniline, drugs, oxidant agents, etc. We present a case of a patient presenting in emergency department with cyanosis and profound hypoxia that failed to respond to high flow oxygen. Bedside diagnosis of blood sample along with ABG confirmed the diagnosis. Patient was successfully treated with IV methylene blue after immediately shifting to ICU. Clinicians must diagnose such cases immediately to aid in early treatment and avoid any further complication.

KEYWORDS

Methemoglobinemia (Methb); Methemoglobin (MetHb); Methylene Blue

Methemoglobinemia is a functional anemia where there is an abnormal level of methemoglobin (MetHb) formation causing inability to release oxygen effectively to body tissues[1, 2]. This is mainly because Methb is an altered state of hemoglobin where there occurs oxidation of ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}). This impairs the ability of hemoglobin to carry oxygen to tissues leading to hypoxia. Methemoglobinemia is said to be present when MetHb level is >1% of the Hb level in the blood[3, 4, 5]. The most common cause of methemoglobinemia is due to exposure to an oxidizing agent[6]. We present case of acute methemoglobinemia due to unknown compound poisoning presenting in emergency department and its successful management in ICU.

A young male of 27 years who worked near a chemical factory in a vehicle repair shop presented at our hospital in emergency department with contact of unknown liquid substance. At presentation, the patient was breathless, tachypneic, restless and irritable with cyanosis of tongue, lips and peripheries. He was conscious and oriented with a pulse rate of 124 bpm and blood pressure of 110/70mmHg. Rest of the systemic examination was normal. Patient was hypoxic with SpO2 of 84% despite high flow oxygen therapy. Patient was immediately taken to intensive care unit for further management. Despite the high flow oxygen patient showed no signs of improvement. A blood sample was collected which characteristically showed a 'chocolate brown' colour and sent for an arterial blood gas analysis. Having a 'chocolate brown' coloured abg sample gave a clue to presence of some hemoglobin abnormality most likely methemoglobinemia induced by some unknown toxin. Immediately another sample was sent for a G-6-PD and methylene blue was administered intravenously at a dose of 1mg/kg (30mg) in 500cc D5 solution given over a period of 30 min. After administration of 1st dose of methylene blue, the patient showed dramatic improvement. His saturation improved from 84% to 93% on high flow oxygen in the subsequent hour. A second dose of methylene blue at a dose of 20mg in 500cc D5 solution was administered intravenously over a period of 30 min. This was given 2 hours after the first dose. On administration of second dose patient improved clinically, his saturation improved to 98% with oxygen. Patient was monitored for 48 hours in ICU and later events were uneventful in hospital. Patient was later discharged after 3 days.

MetHb is formed by the deoxygenation of Hb. This occurs due to reactive oxygen species (ROS) like superoxide and/or peroxide where the iron in the heme group of the Hb is oxidized from ferrous state (Fe^{2+}) to ferric state (Fe^{3+}). This type of hemoglobin can carry oxygen, but it is not able to release it effectively to body tissues. Methemoglobinemia may be congenital or may result from exposure to toxins, oxidant drugs, chemical, local anesthetic drug.

Hemoglobin can transport or accept oxygen only when iron is in its ferrous form. Normally, levels of methemoglobin remains below 1% and symptoms are proportional to the level of methemoglobin. A slight discoloration (eg, pale, gray, blue) of the skin may be present when methemoglobin level is between 3-15%.

At the levels of 15-20% patient may only have mild cyanosis whereas at levels of 25-50% patient experiences headache, dyspnea, confusion, palpitations or chest pain. A level of 50-70% or more may lead to profound acidosis, cardiovascular arrest or delirium. Severe methemoglobinemia may lead to death if not treated immediately[2].

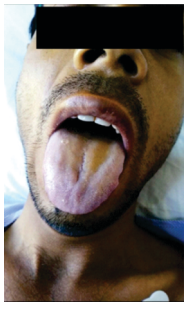

Fig. 1 (left) : central cyanosis as evidenced by bluish discolou rati on of lips and tongue before starting treatment.

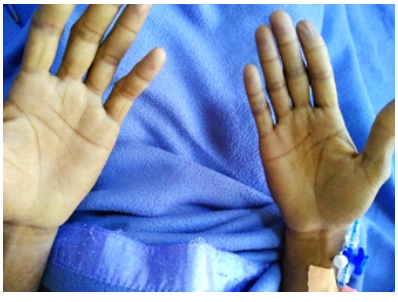

Fig. 2 (down) : peripheral cyanosis in fingers.

Our patient had persistent cyanosis (Fig. 1 and 2) and hypoxia on pulse oximetry despite high flow oxygen. Although patient clinically had cyanosis, his serial arterial blood gas analysis showed normal PaO_2. Also the blood drawn for ABG sample collection was characteristically 'chocolate brown'. Both these characteristics lead to immediate bedside diagnosis of methemoglobinemia in our patient and early initiation of treatment. The serial ABGs of our patient as shown in [Table 1] depicts that there was at no point any decrease in PaO_2 were as on treatment initiation, there was dramatic decrement of methemoglobin levels.

Table 1 : serial ABG of patient during hospital stay

	On admission	3Hrs after Icu admission	7 Hrs after Icu admission	24 Hrs after Icu admission
pH	7.450	7.364	7.391	7.393

pO_2 (mmHg)	74.3	87.1	220	200.6
pCO_2 (mmHg)	36.0	37.8	34.4	35.4
HCO_3(mmol/L)	24.5	21.0	20.4	21.1
SaO_2 (%)	95.5	96	99.7	99.2
MetHb (%)	70.1	63.1	7.5	0.2

As oxygen content is normal in the circulating blood but due to impairment of its delivery to tissue, hypoxia is observed as measured by a pulse oximeter. This is evident by our case where the PaO_2 or say, oxygen content in the blood was normal but due to the presence methemoglobin, patient showed hypoxia.

After initial early clinical suspicion and confirmation of MetHb level by an ABG, immediate treatment was started in the form of methylene blue. . It is given in a dose of 1-2 mg/kg (up to a total of 50 mg) in the form of 1% solution in IV ssaline or D5% solution over 10-15 min. Dose administration may be repeated at 1 mg/kg after 30 minutes-1 hour depending upon the symptoms. in methemoglobinemia, the ferrous form of iron (Fe^{2+}) is converted to ferric state (Fe^{3+}) leaving Hb electron deficient and hence oxygen delivery to tissue is hampered. Methylene blue is an oxidizing agent which provides an artificial electron to MetHb thereby reducing it to its ferrous form. This occurs via the NADPH-dependent pathway. This is the reason why methylene blue should not be administered in G-6-PD deficient patients. There have been cases where parenteral ascorbic acid was also used for successful treatment of methemoglobinemia when methylene blue was unavailable[8-10].

This case is presented in order to sensitize the clinicians regarding early suspicion of a patient presenting with hypoxia with cyanosis and immediate initiation of treatment on basis bedside tests so as to avoid any complication of the disease.

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