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Case Report of A Patient With Methemoglobinemia

KEYWORDS

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Haemoglobin contains iron in ferrous[Fe2+] form,which is oxidised to ferric[Fe3+] form in methemoglobin.This form has decreased ability to bind with oxygen.However the ferric iron has increased affinity for bound oxygen.Thebindin of oxygen to methemoglobin results in an increased affinity of oxygen to three other heme sites (that are still ferrous)within the same haemoglobin unit.This leads to an overall reduced abilty of red blood cell to release oxygen to tissue.

When methemoglobin level is higher than1% it is called methemoglobinemia.

It can be congenital or acquired.

There were two types of acquired cases in our hospital,one was 'inhalational' and another was 'ingestional'.The case of inhalational toxicity was saved and the case of suicidal ingestional toxicity died.

This is a case report of a 16 year old male patient who was working in textile factory.He was brought to the hospital ,at 10 pm, with

- *Vomiting*
- *Giddine*
- *Headach*
- *generalized weakness*

developed after ingestion of substance which was used in his factory for paint removal,before an hour.

The low level of methemoglobin is maintained through 2 important mechanisms.1.the hexose-monophosphate (HMP)shunt pathway within the erythrocyte. Through this pathway, oxidizing agents are reduced by glutathione.

2. More important mechanism involves 2 enzyme systems, diaphorase I and diaphorase II, which requires nicotinamide adenine dinucleotide (NADH) and nicotinamide adenine dinucleotide phosphate (NADPH), respectively, to reduce methemoglobin to its original ferrous state.

NADH-dependent methemoglobin reduction (diaphorase I pathway) is the major enzymatic system involved.[6] Cytochrome b5 reductase plays a major role in this process by transferring electrons from NADH to methemoglobin, an action that results in the reduction of methemoglobin to hemoglobin. This enzyme system is responsible for the removal of 95-99% of the methemoglobin that is produced under normal circumstances.

The NADPH-dependent methemoglobin reduction pathway can be accelerated by exogenous cofactors such as **methylene blue** to as much as 5 times its normal level of activity.

Signs and symptoms depends on methemoglobin .Patientss with methemoglobin level up to 20% may be relatively asymptomatic apart from mild cyanosis.

Methemoglobin levels of 50-70% can cause the following:
Cardiovascular - Abnormal cardiac rhythms

CNS - Altered mental status; delirium, seizures, coma

Metabolic - Profound acidosis

He had no past history of any major medical illness.

He had habbit of alcohol and coccain.

On examination,cyanosis were present in hands,feets,trunk and lips.on on auscultation crepitations were present bilateral.oxygen saturation was 72% with oxygen mask and ecg monitor was showing sinus tachycardia.

Patient was drowsy.He was intubated and put on ventilator support with volume a/c mode.

All routine investigations was within normal limit on admission.

*S.methemoglobin level was **57.24** on admission.*

IV methelene blue was given a/c to weight over 5 minutes. it was repeated twice during night.

On next day S.Methemoglobin level was still 52.17 Than he was treated with exchange transfusion with three unit whole blood two times.

And thanS.methemoglobin was 31.54 Up to third day the patient was improving.On third day during night he suddenly developed bradychardia and became drowsy.Elec-

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trolytes and acid-base correction was done and inotrophic support given.The patient died at that time unfortunately.

Another case of 32 year male patient(Majidkhan) who came with history of inhalation of dye used in his factory for paint-removal,and developed complain of giddiness and vomiting.Cyanosis was present in lips,hands and feet.

Patient was vitally stable.O2 saturation on monitor was 82% on room air.

Serum methemoglobin level in this patient was **37.27** and all other routine investigation was within normallimit.ABGA was s/o mild metabolic acidosis which was corrected.

This patient was treated with IV Methylene blue a/c to weight single time and other supportive management(Oxygen,IV fluids and antibiotics).No ventilator support was required in this patient.

After treatment with IV Methylene blue,cyanosis reduced gradually and O2 saturation increased to 99% on room air. Patient was totally asymptomatic on next day.

In methemoglobinemia IV methylene blue is the primary emergency treatment given in a dose of 1-2 mg/kg(up to 50 mg)as 1 % solution I n IV saline over 3-5 minutes.May be repeated at 1 mg/kg every 30 minute as necessary to control symptoms. Methylene blue is itself an oxidant at doses greater than 7 mg/kg and thus may cause methemoglobinemia in susceptible patients; hence, careful administration is essential.

Methylene blue is contraindicated in patients with G6PD deficiency. Because it requires G6PD to work, it is ineffective in G6PD-deficient patients with methemoglobinemia. Additionally, methylene blue administration may cause hemolysis in these patients, and it is also not effective in patients with hemoglobin M (Hb M). Other conditions in which methylene blue may be ineffective or even deleterious include the following:

- Nicotinamide adenine dinucleotide phosphate (NADPH) methemoglobinreductase (ie, diaphorase II) deficiency
- Sulfhemoglobinemi

Exchange transfusionis for who are unresponsive to methylene blue and G6PD deficient severely symptom,atic patients

Gastric lavage followed by charcoal administration may decrease this prolonged drug effect.

Hyperbaric oxygen treatment is another option for situations where methylene blue therapy is ineffective or contraindicated. This approach permits tissue oxygenation to occur through oxygen dissolved in plasma, rather than through hemoglobin-bound oxygen.

REFERENCE 1) Medscape dictionary of medicine | 2) Harrison's principle of internal medicine (18th edition)