ORIGINAL RESEARCH PAPER

Medicine

Volume - 7 | Issue - 2 | February - 2017 | ISSN - 2249-555X | IF : 3.919 | IC Value : 79.96

A RARE CASE OF ACQUIRED METHEMOGLOBINEMIA

KEYWORDS

Archana devi

M.D post graduate, Department of general medicine, Sri ramachandra medical college and research institute - Corresponding author

Dr. Nandhini

M.D post graduate, Department of general medicine, Sri ramachandra medical college and research institute

Dr. Madhavan

Associate professor, Department of general medicine, Sri ramachandra medical college and research institute

ABSTRACT There are two types of methemoglobinemia : Congenital and acquired.Acquired methemoglobinemia typically results from ingestion of specific drugs or agents that causes increase in the production of methemoglobin. It can be fatal diseases. The following case describes a patient with accidental exposure of bromoacetanilide powder inhalation. Patient developed methemoglobinemia and was treated with methylene blue. Since methemoglobinemia is life threatening if not treated early, knowledge about causes, clinical manifestations and management is essential.

Introduction

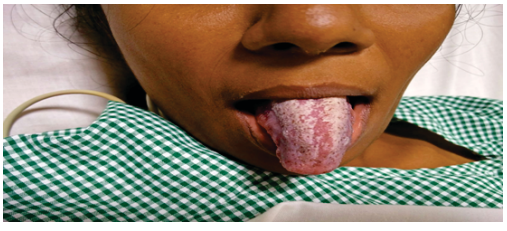
Methemoglobin (MeHb) is hemoglobin (Hb) containing oxidized (rather than oxygenated) iron. Normal hemoglobin contains four iron atoms in the ferrous (Fe+2) state. Oxygen binds to the ferrous ions and is transported to tissues where it is released in response to a lower oxygen gradient. If the ferrous ion loses an electron to another drug or chemical and is oxidized to the ferric (Fe+3) state, it can no longer bind oxygen. As well, if even one of the iron atoms becomes oxidized the release of bound oxygen from the other iron atoms in the haemoglobin molecule is impaired. So methemoglobin reduces oxygen carrying capacity and reduces oxygen release to tissues. Carbon dioxide transport from the tissues to the lungs for elimination is also impaired.

Case report

22 year old female who is a student, came to our hospital with alleged history of accidental exposure to Bromoacetanilide powder in chemical lab on 10/01/16 at around 12:30 PM. She had 5-6 episodes of vomiting following the event. History of giddiness and breathlessness was present following the event. On examination, Patient was Conscious, oriented, Afebrile, pulse rate was 120/min , blood pressure was 120/70 mmHg. Oxygen Saturation was 76% in room air by pulse oximeter and improved to 80% with 12 liters of oxygen. Central cyanosis was present.(Figure 1).

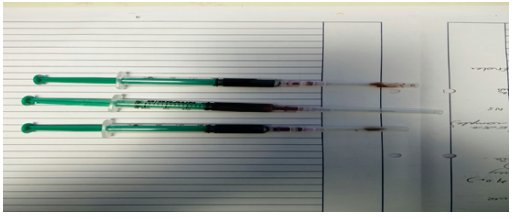
Systemic examination was normal.

Figure 1



ABG done was dark brown in color and even on exposure to oxygen there was no improvement in color, but ABG values showed saturation of 98% with PAO2 130 with 4 liters of oxygen.(Figure 2). Baseline investigations done showed hemoglobin of 6.9 g/dL, MCV of 64.3, total count of 16,000 cells/cu.mm, platelet of 2.50 lakhs/cu.mm. Renal function test, liver function test and serum electrolytes were within normal limits. Chest X-ray done was normal. ECG done showed sinus tachycardia.

Figure 2:



• Patient was started on IV fluids 100 ml/hour, Inj.Hydrocort 100 mg IV stat was given following which Inj. Methyl prednisolone 1 gm IV od was started for 3 days for suspicion of chemical pneumonitis. Methemoglobin level was sent for analysis in view of suspicion of methemoglobinemia. Inj. Methylene blue 40 mg in 200 ml NS over 10 minutes was given and patient had improvement in saturation 92% in room air, after 1 hour of administration of methylene blue. Patient was also started on Vitamin C TDS. Repeat methylene blue was not given since patient had improvement in symptoms. For anaemia, 2 unit packed cell transfusion was done following which her hemoglobin improved to 10.9g/dL . Patient cyanosis also reduced with improvement in oxygen saturation following transfusion. Serum methemoglobin level was 2.7g/dL. It was done by spectroscopic method.

DISCUSSION

Methemoglobin is an alternative state of hemoglobin in which the ferrous (Fe+2) irons of heme are oxidized to the ferric (Fe+++)state .The ferric hemes of methemoglobin are unable to bind oxygen affinity of any remaining ferrous hemes in the hemoglobin tetramer is increased. [(1)]As a result , the oxygen dissociation curves will be shifted to left . The net effect is that patients with acutely increased concentrations of methemoglobin have a functional anemia (ie, the amount of functional hemoglobin is less than the measured level of total hemoglobin). The circulating methemoglobin-containing hemoglobin molecules are unable to deliver oxygen and the remaining oxyhemoglobin has increased oxygen affinity, resulting in impaired oxygen delivery to the tissues. Those with chronically increased methemoglobinemia and functional anemia may develop compensatory erythrocytosis.

Acquired causes: Most cases of methemoglobinemia are acquired, resulting from increased methemoglobin formation by various exogenous agents. These may include medication overdoses or poisoning but may also occur with medications given at standard

Volume - 7 | Issue - 2 | February - 2017 | ISSN - 2249-555X | IF : 3.919 | IC Value : 79.96

doses, particularly in individuals with partial deficiencies of cytochrome b5 reductase. Infants and premature infants are particularly susceptible to the development of methemoglobinemia because their erythrocyte cytochrome b5 reductase activity is normally 50 to 60 percent of adult activity.

There are two pathways for reduction of methemoglobin back to hemoglobin:

- The only physiologically important pathway is the NADH-dependent reaction catalyzed by cytochrome b5 reductase (b5R).

- An alternative pathway that is not physiologically active utilizes NADPH generated by glucose-6-phosphate dehydrogenase (G6PD) in the hexose monophosphate shunt. However, there is normally no electron carrier present in red blood cells to interact with NADPH methemoglobin reductase. Extrinsically administered electron acceptors, such as methylene blue (MB) and riboflavin are required for this pathway to be activated [(3)]This non-physiologic pathway becomes clinically important for the treatment of methemoglobinemia.

Conclusion

- Bromoacetanilide is a rare compound which can cause methemoglobinemia.
- Clinical diagnosis by central cyanosis, normal arterial pO2 in arterial blood gas analysis should prompt the start of treatment with methylene blue early. Confirmation can be done by assessing the methemoglobin levels.
- Methylene blue is the drug of choice. It should not be given if G6PD is suspected. Blood transfusion can also be helpful if methylene blue is not available.

References

1. Darling, R, Roughton, F .The effect of methemoglobin on the equilibrium between oxygen and hemoglobin .Am J Physiol. 1942; 137:56.
2. Warang PP, Kedar PS, Shanmukaiah C, Ghosh K, Colah RB
Clinical spectrum and molecular basis of recessive congenital methemoglobinemia in India.Clin Genet. 2015 Jan;87(1):62-7. Epub 2013 Dec 20.
3. Yubisui T, Takeshita M, Yoneyama Y Reduction of methemoglobin through flavin at the physiological concentration by NADPH-flavinreductase of human erythrocytes. JBiochem. 1980;87(6):1715.