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Case Report

Preoperative Preparation and Anaesthetic Management of Patient with Asymptomatic Methemoglobinemia for Thoracoabdominal Aortic Aneurysm Resection: A Case Report

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Abstract: Acquired methemoglobinemia in the absence of a triggering factor is uncommon but should be suspected when saturation gap is detected between SpO_2 and SaO_2. Hypothermia during cardiopulmonary bypass and certain drugs used during cardiac surgery can worsen the condition resulting in tissue hypoxia, energy failure and subsequent end organ damage. Preoperative correction of the condition ensures adequate oxygen delivery at the tissue level. We describe a case of methemoglobinemia without any symptoms posted for thoracoabdominal aortic aneurysm resection diagnosed preoperatively and subsequent management.

Background: Methemoglobinemia (Methb) is a rare condition, clinically not routinely encountered where Fe^{2+} moiety ofhemoglobin is oxidised to Fe^{3+} . This results in impaired oxygen carrying capacity and delivery. It is characterised by low pulse oximeter readings despite of supplemental oxygen. It can be triggered by certain drugs or can be idiopathic as described in our case. Written informed consent for publication of this case was taken from the patient.

CASE DESCRIPTION

A 41-year-old gentleman presented with chief complaints of shooting pain in the upper back since 2 months with no other comorbidities. He was a tobacco chewer, not an alcoholic and not on any medications. On general examination, conjunctiva, tongue and fingers appeared pale, but no icterus, clubbing, cyanosis, lymphadenopathy or edema was seen. He was conscious, coherent, vitals were normal. CT angiography revealed an aneurysm in the descending thoracic aorta extending into the proximal abdominal aorta. He was admitted, started on tab. Metoprolol 25 mg, tab. Pregabaline 75 mg and posted for aneurysm resection. Preoperative hemoglobin was 15 g%, haematocrit was 48%. All other investigations were within normal range. Patient was kept fasting for 8 hours, routine premedication with tab. Lorazepam 0.5 mg and tab. Pantoprazole 40 mg per oral was given and taken up for surgery. After shifting to OR, standard pulse oximetry and five electrode electrocardiogram was connected. Oxygen Saturation was 88% on the monitor. Under local anaesthesia, right radial artery was cannulated. Blood was unusually dark red. Line was connected to transducer to rule out venous cannulation. Arterial trace was confirmed and blood pressure was 136/80 mm of Hg. Sample was sent for arterial blood gas (ABG) analysis which showed saturation of 96% and pO_2 of 81.5 mm Hg on room air with no acidosis. Patient was pre-oxygenated with 100% oxygen by spontaneous ventilation through leak proof mask for 5 minutes. Oxygen saturation on the monitor remained 88%. Arterial blood sampled again which was still dark red in colour. Blood gas analysis showed saturation of 100% and pO2 of 370 mm of Hg with no acidosis. Abnormal hemoglobin was suspected due to the discrepancy in saturation in the monitor and in the ABG analysis. Sample was sent for hemoglobin analysis by advanced ABG machine which revealed oxyhemoglobin percentage of 65%, carboxy hemoglobin 0.7%, methb 33.4%. Preoperative drug therapy and family history was reviewed.No other causative factor for methemoglobinemia was identified. Patient was shifted to the ICU. Inj. methylene blue i.v. was started at a dose of 3 mg/kg (150 mg) over 3 hours while supplementing oxygen through mask @ 10 litres/min. Pulse oximeter saturations improved gradually and reached 100% in three hours.Blood gas analysis was repeated at the end of three hours which showed methb of 0.9%, oxyhemoglobin of 97.6%, saturation of 99.9%,pO_2 320 mm Hg, no acidosis.Pulse oximeter saturation remained 100% overnight on room air. Other vital parameters were within normal range. Urine output was normal except for green colour. He was taken up for surgery the next day. ABG analysis was done prior to induction of anaesthesia which showed metHb of 1.1%, oxyHb of 97.3%, saturation of 99.6%, pO_2 of 216 mm of Hg, no acidosis. Pulse oximeter saturation was 100%. Patient was induced with midazolam 5mg, fentanyl 500mcg, propofol 60 mg, vecuronium 10 mg and pancuronium 4 mg. He was intubated and maintained on FiO_2 0.6 and isoflurane 0.8 – 1 MAC. Initiation of cardiopulmonary bypass (CPB) was uneventful. Resection and interposition graft was done under deep hypothermic circulatory arrest. Blood gas analysis was done after re-initiation of CPB which did not show any acidosis or methb. Patient was rewarmed adequately. Use of nitroglycerin infusion during rewarming was avoided and weaned off CPB using inotropes dopamine and noradrenaline. Surgery lasted for 9 hours. Three units of packed red blood cells, four units of platelets and two units of cryoprecipitate were transfused after coming off CPB. Pulse oximeter saturation was 99 – 100% throughout. Vital parameters were stable. Postoperative course was uneventful and patient was extubated on 2nd postoperative day. Methb levels were checked on 4th postoperative day and found to be 0.9%.

DISCUSSION

Conventional pulse oximetry can provide inaccurate readings in the presence of abnormal hemoglobins – MetHb, sulfmethemoglobin and carboxyhemoglobin.[1,2] Other common causes of desaturation like lung related problems or intracardiac shunting should be ruled out. If no obvious etiology is identified, evaluation for abnormal blood hemoglobin is warranted. The "oxygen saturation gap" is the difference between the reading from a pulse oximeter and the calculated oxygen saturation from a standard ABG machine. If it is greater than 5%, it may point towards abnormal hemoglobin, representing carbon monoxide poisoning, methemoglobinemia or sulfhemoglobinemia.[3,4]In our patient, a saturation gap of 8% had been identified. This raised a suspicion of presence of abnormal hemoglobin but the blood was dark red colour which ruled out the possibility of carboxyhemoglobinemia and sulfhemoglobinemia.To confirm the diagnosis, hemoglobin analysis was performed which revealed methemoglobinemia. Ralston *et al.* reported that MetHb has approximately the same absorption coefficient at both 660nm and 940nm wavelengths and the absorbance ratio is approximately 1.0 when enough MetHb is present. When MHb levels reach 30%–35%, the

light absorbance reaches a plateau and the pulse oximeter reading becomes stable in the 82%–86% range.[1,5]This explains the reading of oxygen saturation on the monitor as 88% in our case.The arterial PO_2 is a measure of dissolved oxygen and does not directly correlate with oxygen molecules bound to hemoglobin. This explains the normal PO_2 values on room air in our patient which increased after supplemental oxygen.

Methb forms when hemoglobin's one or more Fe ion of the heme moiety converts from ferrous (Fe^{2+}) to ferric (Fe^{3+}). Conversion of iron from the ferrous to ferric state represents loss of an electron, i.e. it is an oxidative process.[6,7] It causes conformationchange of hemoglobin resulting in less oxygen carrying capacity and also causes a leftward shift of the oxygen dissociation curve, increasing the affinity of the hemoglobin for oxygen and reducing tissue oxygen release.[8] In healthy adults, methemoglobin levels remain below 1%. Cyanosis appears when meth levels reach 15-20% but not observed in our patient due to the dark skin colour.[9]Causes of methemoglobinemia are Congenital, occupational exposure to oxidising agents like aniline dye, nitrates, antibiotics like quinones, sulfonamide, dapsone, local anaesthestics like benzocaine, prilocaine, and other unknown causes.[10]

Treatment should be considered when the methemoglobin is 30% in an asymptomatic patient and 20% in a symptomatic patient.[11] The traditional first line therapy for drug induced methemoglobinemia is intravenous methylene blue administration. Methylene blue undergoes reduction to leukomethylene blue in the presence of nicotinamide adenine nucleotide phosphate (NADPH) within the red blood cells. In our case, no obvious cause could be identified, methylene blue infusion was started and given slowly to avoid sudden rise in methylene blue concentration which is an oxidising agent. Saturation on the monitor gradually increased from 88% to 100% over 3 hours and remained stable thereafter.Some drugs produce a rebound methb, in which methb levels increase 4 to 12 hours after successful methylene blue therapy but in our patient no rebound methb was observed.

Our case was planned to be done under deep hypothermic circulatory arrest. Hypothermia may exacerbate methemoglobinemia by slowing down the normal enzymatic processes which reduces methemoglobin back to hemoglobin under physiological conditions.[12,13]This might result in severe tissue hypoxia. So methb was treated before proceeding to the procedure.There are several drugs routinely used during cardiovascular surgeries which may exacerbate methemoglobinemia like nitroglycerine and lignocaine. All such drugs were avoided during the procedure.

CONCLUSION

Diagnosis of methemoglobinemia requires a high index of suspicion in a scenario of low pulse oximetry readings. Presence of saturation gap can be a key to the diagnosis of methemoglobinemia. Preoperative diagnosis and treatment can help in avoidance of adverse consequences during cardiopulmonary bypass and post operative recovery.

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