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Research Paper

Anaesthesia for Right Total Knee Arthroplasty in Patient With Methemoglobinemia: A Case Report

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I. INTRODUCTION

High amounts of methemoglobin, which come from hemoglobin's oxidation of ferrrous (Fe2+) to ferric state (Fe3+), is the cause of methemoglobinemia, a hemoglobinopathy. Methemoglobinemia is an uncommon but potentially serious haemoglobin disorder in which oxygenation product of haemoglobin, i.e., methemoglobin accumulates in erythrocytes in large amounts. It is not capable of carrying oxygen and thus interferes with oxygen delivery to the tissues[1]. Methemoglobinemia symptoms are the results of inadequate oxygen transport. The diagnosis confirmed by co-oximetry. Here, we report successful anaesthetic management of a patient with methemoglobinemia who underwent right total knee arthroplasty under subarachinoid block.

II. CASE REPORT

60-year old female with primary osteoarthritis of bilateral knee joints(R>L) with genu valgum deformity of right knee, posted for right knee arthroplasty surgery. She had history of pulmonary tuberculosis 30 years back and also underwent open cholecystectomy under general anesthesia 14 years back which was uneventful. Patient was conscious, moderately built and nourished with a pulse rate of 72 beats per minute, blood pressure 130/80mmHg, respiratory rate 14/min, oxygen saturation by pulse oximeter was 88% on both fingers. She had cyanosis of lips, tongue and fingers. Preoperative complete blood count showed hemoglobin 15.7 g/dl and hematocrit 45.7%. Further cardiopulmonary work-up, including echocardiography, pulmonary angiography were normal.ABG analysis showed saturation gap. The difference between oxygen saturation by arterial blood gas analysis and oxygen saturation by pulse oximeter was presumably due to dyshemoglobin. For confirmation, arterial blood gas analysis and hemoglobin analysis using a co-oximeter was ordered. Arterial blood gas analysis showed normal findings and hemoglobin analysis showed methemoglobin at 35.7%.From the history and investigations a diagnosis of severe methheglobinemia was made. Ascorbic acid 500 mg thrice daily was administered and methylene blue 1 mg/kg was intravenously injected slowly over 3 minutes. IV Methylene blue 50mg – 4 doses to attain normal methhemoglobin levels were used. Serial co oximetre values after treatment showed decline in methemoglobin level to 23.9%, 6.5% and 2.1% .Her cyanosis of lips and fingers improved and was posted for the surgery. Standard NPO guidelines were followed, written informed high-risk consent obtained, IV Methylene blue kept ready and 2 large bore IV cannulae and arterial line secured under local anaesthesia. Supplemental oxygen via face mask given at 5L/min. ECG, HR, NIBP, SpO_2, urine output were monitored. Preoperative vitals of heart rate 74 bpm, blood pressure 160/80 mmHg noted. The intraoperative blood pressure and pulse rate were stable, oxygen saturation by pulse oximeter was 93-96%(room air) .Subarachnoid block given with 2ml 0.5% bupivacaine (heavy) and 20µg fentanyl using 25G Quincke needle, with preferential blockade to right lower limb. Epidural anaesthesia was avoided to limit the use of local anaesthetics. Surgery lasted for 1.5 hours. Intraoperative period uneventful. Inj. Tranexamic acid 1g and Inj. hydrocortisone 100mg given intraoperatively. Co-oximetry done 20 minutes after tourniquet release showed methemoglobin level of 5.8%.Postoperative pain managed with opioids and NSAIDs. After the surgery, the patient was transferred to the intensive care unit (ICU). In the ICU, oxygen 6 L/min was administered by a face mask. The oxygen saturation by pulse oximeter was 94%. She was discharged without complications on postoperative day 3.

III. DISCUSSION

Methemoglobin is formed when Fe ion in haemoglobin oxides from ferrous (Fe^{2+}) to ferric (Fe^{3+}) state. In healthy adults, a small amount of hemoglobin oxides to methemoglobin and then it is rapidly converted back to hemoglobin such that methemoglobin levels remain below 1%. This balance is maintained by the intracellular enzyme systems, NADH cytochrome-b_5 methemoglobin reductase and NADPH methemoglobin reductase[2] .Methemoglobinemia occurs when the methemoglobin level exceeds 1-2%. Methemoglobin has very high oxygen affinity and virtually no oxygen is delivered to the tissues.

Congenital methemoglobinemia arises from mutations that stabilize iron in the ferric state or from mutations that impair the enzymes nicotinamide adenine dinucleotide (NADH) b5 reductase and nicotinamide adenine dinucleotide phosphate flavin reductase that reduce methemoglobin to hemoglobin. Acquired methemoglobinemia is more common and occurs on exposure to oxidising agents such as aniline dyes, nitrobenzene, nitrate, nitrite, benzocaine, prilocaine, dapsone, pyridium, nitric oxide, nitrous oxide and naphthalene due to overwhelming of capacity of reducing enzyme

The symptoms appear depending on methemoglobin level in blood. Chocolate colour cyanosis presents at levels of 5-15% and appears early in anaemic patients. Even at least 5 mg of reduced haemoglobin must be present for cyanosis to be detected. At 30-40%, weakness, headache, dyspnoea, tachycardia and dizziness occur. Patient will be lethargic, stuporous, confused and comatose at concentration of 55-60% and at about concentration of 70% circulatory collapse occurs[3]

OxyHb absorbs infrared light with a wavelength of 990 nm, and deoxyhemoglobin (deOxyHb) absorbs red light with a wavelength of 660 nm. The accuracy of pulse oximetry decreases in patients with methemoglobinemia as it measures oxygen saturation using infrared and red light. Methhemoglobinemia is suspected when arterial blood gas reveals the classical 'saturation gap', with normal PaO_2 despite cyanosis[4.] A co-oximeter is considered to be an accurate device for measuring methemoglobin and is very useful for the diagnosis of methemoglobinemia. It measures absorbance at over 100 wavelengths to construct a continuous absorption spectrum from 450 to 700 nm[5]. It detects the presence as well as quantifies methemoglobin level

Injection methylene blue is the antidote and the dose is 1-2 mg/kg IV over 3-5 min. It forms reduced methylthioninium chloride - acts as an electron donor to reduce Fe^{3+} back to Fe^{2+}. Produces leucomethylene blue which increase rate of conversion MetHb to Hb. Vitamin C in perioperative period should be considered which may help by increasing the level of NADH methemoglobin reductase, which helps in conversion of ferric ion to ferrous ion.

Anaesthetic drugs, which induce methemoglobinemia, are local anaesthetics example (lidocaine, benzocaine etc), metoclopramide, nitrous oxide, and nitroglycerine and sodium nitroprusside. They should be avoided or may be used on titrated doses[6] .The anesthesiologist should avoid the use of oxidizing agents and maintain O2 carrying capacity of the arterial blood by ensuring high O2concentration in the inhaled gas in patients with congenital methemoglobinemia during general anesthesia.

IV. CONCLUSION

In conclusion, congenital methemoglobinemia is rare but fatal clinical condition to be encountered during perioperative period. The dissociation between low SpO_2 and near normal PaO_2 should raise the suspicion of Methemoglobinemia and the presence of dyshemoglobin should be confirmed by performing a hemoglobin analysis with a co-oximeter. Planning and preference for mode of anaesthesia needs to be evaluated thoroughly as it being a rare disease.

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